The Intergenerational Transmission of Eating Behaviour

by

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SUMMARY

Thesis Abstract

Approximately two thirds of the Australian population are either overweight or obese (ABS, 2015). Diet and lifestyle factors independently contribute to the risks for obesity and a range of prevalent chronic diseases (NHMRC, 2013b; Stewart & Wild, 2014) therefore, the Health Belief Model (HBM) was used to predict health motivation and behaviour change in a series of four studies. According to the model, the benefits of engaging in health enhancing, or the disadvantages of engaging in health compromising, behaviours depend upon an individual weighing up the perceived risk and severity of a potential disease with the likely benefits and barriers of taking any relevant health action (Conner & Norman, 1995; Harrison, Mullen, & Green, 1992).

It is also important to understand the dietary behaviours that contribute to overall food consumption, and to explore the social context of the family for any influencing and modifiable factors. To date, there is little in the published literature beyond bi-directional food influences between parents and children, hence Study 1 presents a qualitative study that, through semi-structured family interviews, explored food purchasing, preparation, and consumption, and examined the bi-directional influences that occurred between family members within three generations (N = 57). Results were consistent with previous research indicating mothers were dominant in the provision of family meals (Beydoun & Wang, 2009; Green et al., 2009). Less is known regarding grandmother influences on diet (Wroten, O'Neil, Stuff, Liu, & Nicklas, 2012) and Study 1 suggested that the female role of grandmother also plays the most dominant role in the grandparent household. Fathers were found to be more likely to relax the family food rules. The grandparent-child dyad shared a bi-directional influence pathway that bypassed the parent generation: children influenced grandparents by introducing new foods to the diet, and grandparents provided grandchildren with indulgent treats.

Study 2 used a correlational design to examine resemblances in healthy and unhealthy food consumption between three generation family relationship dyads, and explored whether 'Food Life

behaviours and attitudes' (Food Life Questionnaire Short-Form; Sharp, Hutchinson, Prichard, & Wilson, 2013) played a role in influencing healthy and unhealthy food consumption (N = 229). Results indicated both parents impacted upon the healthy food consumption of children. The mother's importance was acknowledged by mother-daughter fruit consumption resemblances, and mother-child resemblances in vegetable consumption. A novel finding suggests fathers' attitudes that diet can influence health and disease have positive influences on children's healthy food consumption. Suggested by the correlation between his healthy food attitudes and fruit consumption in sons, and vegetable consumption in daughters. Food Life behaviours and attitude correlations supported directional hypotheses with the total sample for healthy and unhealthy food consumption. Grandparent marital-ties shared a lower incidence of fast food consumption when compared to parent and child generations, however, shared strong resemblances in snack consumption.

Study 3 was a cluster randomized control trial investigating tailored family health history feedback using the Families Sharing Health and Risk Evaluation (SHARE) workbook (Koehly, Morris, Skapinsky, Goergen, & Ludden, 2015) modified for Australians, to observe dietary and screening behaviour over time, and also examined the influence of Food Life behaviours and attitudes within three generation families (N = 178). Fruit consumption showed the most promising result in response to the Families SHARE intervention as demonstrated by stage of change progression from 'precontemplation' to 'action' (i.e., TTM, the Transtheoretical Model Stage of Change; Prochaska, DiClemente, & Norcross, 1992) in the experimental group for fruit consumption. Vegetable consumption, on the other hand, showed little change on any of the variables, and consumption remained well below the NHMRC recommendations (NHMRC, 2013a). Food Life behaviours and attitudes made significant contributions to food consumption when a family member or mother was at risk of chronic disease. Correlations in self-reported dietary behaviours (i.e., diet-health oriented behaviours, DHOB subscale of the FLQ SF) were shared between parents and grandparents at baseline. At follow up, correlations between parents and children achieved significance. Except for vegetable consumption, the grandparent generation

showed greater dietary behaviour improvements than younger generations. Parent-child resemblance in attitudes that diet affects subsequent health or disease (i.e., diet-health/disease linked attitudes, DHLA subscale of the FLQ SF) observed at baseline differed at follow up with significant parent-grandparent correlations reported. This may suggest that the educational nature of the Families SHARE workbook motivated parents to talk with grandparents about the links between diet and disease and learn from this.

Finally, Study 4 used a mixed methods survey and family interview design to further evaluate the findings of Study 3 (N = 113). Results showed tentative support for the Families SHARE workbook as an effective family health history intervention tool that was particularly successful in engaging with families and promoting screening behaviours. Potential for dietary behaviour change was indicated by participant's intentions to improve fruit and vegetable consumption. The dissemination of information beyond the nuclear family showed promise for intervening at the intergenerational family level in motivating health behaviour change.

Conclusions from this series of studies suggest that a combination of psychological variables influence the healthy and unhealthy food consumption of children. These include co-existing parental sex-role modeling (Bandura, 1977b; Bussey & Bandura, 1984), parenting and feeding styles (Baumrind, 1991; Blissett, 2011), and attitudes that diet affects subsequent health and disease (Rozin, Fischler, Imada, Sarubin, & Wrzesniewski, 1999; Sharp et al., 2013). Future research directions suggest investigating how gender differences that exist between mothers and fathers' expression of co-existing parenting variables affect developing children's dietary behaviours.

DECLARATION

I, KATE RHODES, certify that this thesis does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any university; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text.

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CHAPTER 1: A REVIEW OF THE LITERATURE & INTRODUCTION

Title: Disease Prevention Starts at Home: How Food-related Decision-making and Behaviour within Families affect Healthy and Unhealthy Food Consumption

Prevalence of Overweight and Obesity

In 2015 the Australian Bureau of Statistics (ABS) reported that approximately two thirds of the adult Australian population were either overweight or obese in 2012 (ABS, 2015) with the combined rate rising from 56% in 1995 to 63% in 2012. While chronic disease risk rises with increasing BMI (BMI; weight in kilograms divided by height in metres squared (kg/m^2) (NHMRC, 2013b) the American Medical Association has recently classified obesity as a disease in itself (Katz, 2014).

Overweight and obesity arise as a consequence of energy imbalance. When energy-intake equals energy-expenditure the body will maintain an even weight (Whitney & Rolfes, 2007). Imbalance arises when energy intake through food and drink exceeds energy output expended through activity. Both input and output are modifiable behaviours and appropriate changes to these can not only decrease risk of chronic disease but reverse the current obesity pandemic (Popkin, Adair, & Ng, 2012). Although certain medical conditions and genetic predisposition to weight gain can cause obesity in some people (Anderson & Butcher, 2006; Block, Scribner, & DeSalvo, 2004) behavioural choices have a major influence. Diet and lifestyle factors independently contribute to the risks for a range of prevalent chronic diseases such as heart disease, type 2 diabetes (T2D) and a number of cancers (Bray, 2006; Calle, Rodriguez, Walker-Thurmond, & Thun, 2003; Finer, 2010; Hsueh & Deng, 2016; Lawrence & Kopelman, 2004; NHMRC, 2013b; Rowen, Milner, & Ross, 2010; Stewart & Wild, 2014; Vernarelli, Mitchell, Rolls, & Hartman, 2014). Of the two behaviours implicated, diet and activity, Binkley (1997) suggests excess energy-intake (i.e., diet) contributes more to obesity

than inadequate energy expenditure (i.e., activity).

Disseminating Dietary Guidelines as a Strategy for Disease Prevention

Diets that adhere to national guidelines are said to be disease-preventative because they are 'healthy' (e.g., high in fruit, vegetables and grains; low in meat, salt, trans- and saturated fats) and maintain energy balance (Deblinger, 2001; Levine et al., 2014; NHMRC, 2013a; Romaguera et al., 2012; Scarborough, Nnoaham, Clarke, Capewell, & Rayner, 2012). To date, there are few NHMRC guidelines for 'unhealthy' food consumption and Australian recommendations suggest consuming smaller amounts in proportion to healthier food types. Dietary behaviours linked to obesity such as the regular consumption of foods high in saturated fats, salt and sugar, or overeating (Gluckman, Hanson, Zimmet, & Forrester, 2011) vary between individuals and exist within distal environmental contexts as well as proximal social contexts that impact upon individual behavioural choices (Ajzen & Fishbein, 1980; Ashida, Wilkinson, & Koehly, 2012; Cullen, Baranowski, Rittenberry, & Olvera, 2000; Linke, Robinson, & Pekmezi, 2013). Addressing dietary behaviours is difficult due to the complexity of factors contributing to overall food consumption. Eating is often a social act and individual food choice occurs in contexts where one's environment, social roles, and family norms are all influential. Given that dietary choices made early in life may also affect whether chronic diseases will develop later (World Health Organisation (WHO) & Food and Agricultural Organization of the United Nations (FAOUN), 2003), it is important to consider how food choices increasingly contribute to overweight and obesity. Social and environmental considerations are gaining greater attention in the contemporary literature to seek solutions that target obesity prevention.

Changes in Societal Attitudes to Food and the Physical Environment

The rise in obesity in Western countries over the last 20-30 years has been so dramatic that it is thought that the reasons for it are more environmental than hereditary

(Block et al., 2004; Drewnowski, 2003). Rosenkranz and Dzewaltowski (2008) explored the overlapping environments that contribute to the development of obesity. These environments were not only physical built environments; on the contrary, they were conceptualized as a myriad of layers that create behavioural settings which together shape the dietary intake of individuals or groups of people. In relation to childhood obesity; political, economic, socio-cultural and physical components shape the overall home food environment in which children develop enduring dietary behaviours. Further, each of these determinants could be considered at the distal (macro) or proximal (micro) level. For example, food production at the distal level influences the availability and accessibility of food at the proximal level. Socio-cultural environments such as ethnic identity at the distal level influence parenting food practices and rules in the proximal level home environment (see Figure 1).

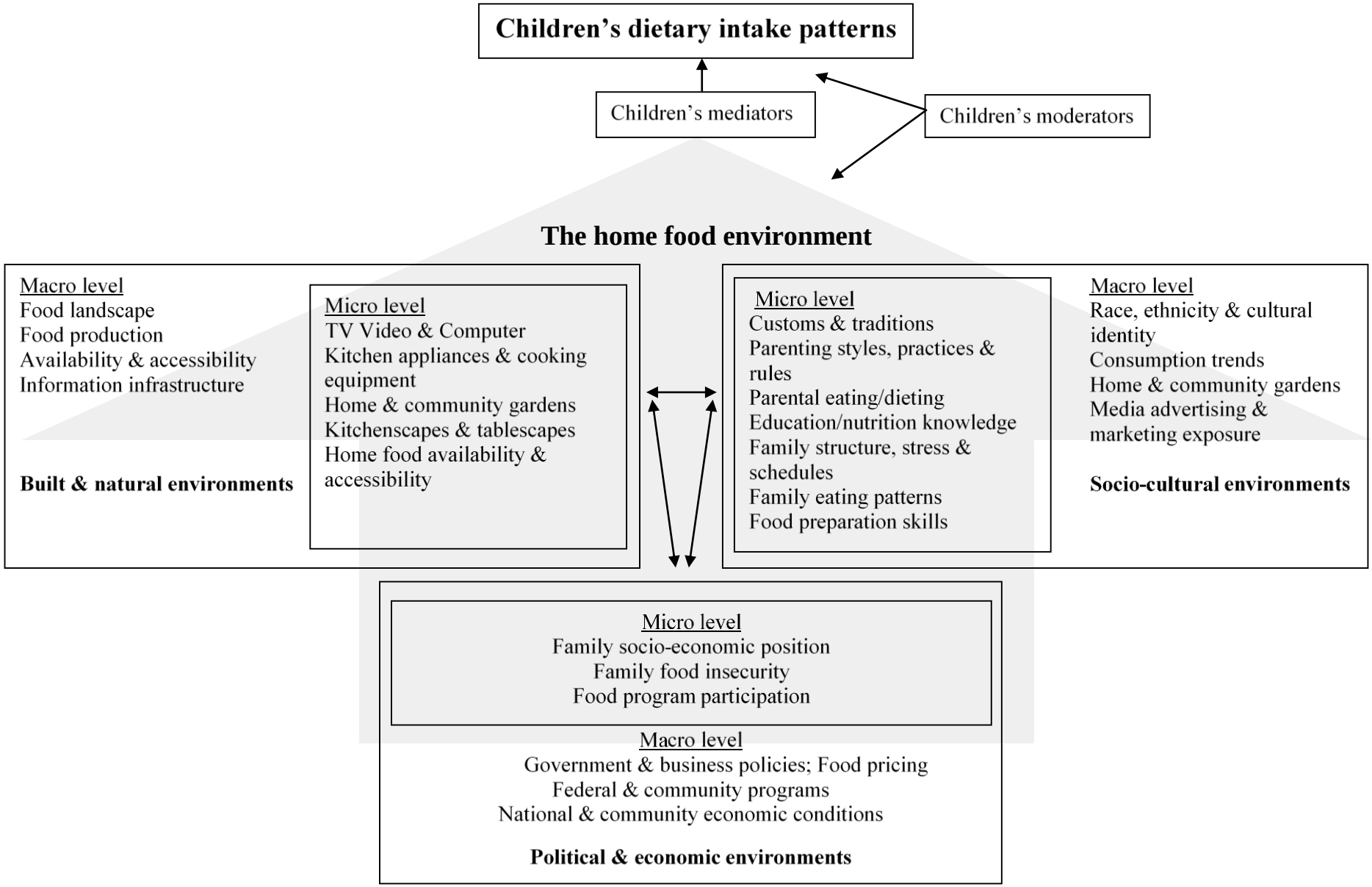

Figure 1. Macro and micro level determinants that impact on the home food environment. Adapted from "Model of the Home Food Environment Pertaining to Childhood Obesity," by Rosenkranz, R. R., & Dzewaltowski, D. A., 2008, *Nutrition Reviews*, *66*(3), p.128. Copyright.

Distal level environmental influences on societal food consumption. At the distal level, Anderson and Butcher (2006) considered a multitude of possible reasons for the obesity pandemic, which have been attributed to environmental and societal changes since the 1970s. For example, the sprawl of the urban environment has caused more people to travel by car thus lowering energy-expenditure dramatically. Other changes include the greater availability of energy dense foods and beverages in vending machines than there was 20 years ago, in both workplaces and schools. The sales of soft drinks containing high proportions of sugar has increased markedly since 1990 (Anderson & Butcher, 2006; Popkin et al., 2012), and the consumption of high-energy beverages is also associated with weight gain (Block et al., 2004; Fagherazzi et al., 2013). Societal and environmental changes contributing to energy imbalance have occurred over the same time period as the obesity pandemic.

***Obesogenic environments that impact on higher kilojoule consumption*.** Since the 1970's, developed countries have experienced a growth in the availability of fast food (Jeffery, Baxter, McGuire, & Linde, 2006), increasing portion sizes (Anderson & Butcher, 2006; Block et al., 2004; Kling, Roe, Keller, & Rolls, 2016), and more frequent eating occasions (Duffey & Popkin, 2011). Urban environments have provided greater access to processed foods that typically contain higher proportions of fat, sugar and salt compared to whole foods (Nestle et al., 1998). Research indicates that an extra 900 kilojoules[1] (kJ) per day are required to maintain an existing overweight status, whereas a steady positive energy imbalance of 30 kJ per day over a protracted period is required for average weight gain to occur from a baseline healthy weight (Hall et al., 2011). This suggests that with rising obesity rates over time, average kilojoule consumption has risen above the recommended daily intakes for many years and emphasises the necessity to intervene at the dietary level.

[1] 1 kJ = 0.2 Calories.

(Binkley, 1997; Duffey & Popkin, 2011; Raubenheimer, Machovsky-Capuska, Gosby, & Simpson, 2015; The Lancet Diabetes & Endocrinology Review, 2016).

Data suggests that there has been an increase in the number of people eating away from home and more fast food outlets are available than in the past, although the association between fast food outlets and overweight is not clear or consistent (Anderson & Butcher, 2006; Block et al., 2004). A review of studies looking at the connection between eating out of home and dietary intake confirms that the association between eating out is linked with increased energy and fat intake, and decreased micro-nutrient intake (Lachat et al., 2012). Research with school-aged children, similarly associated food eaten away from home with poorer nutrient quality (Bell & Swinburn, 2004). Energy dense food and beverages are commonplace in Australian schools, either brought from home (e.g., biscuits, snack bars and fruit cordial drinks) or purchased at school (e.g., fast food, packaged snacks, confectionary, and soft drinks) and it has been recommended by some that these food types should be replaced with fruit and water to combat obesity (Bell & Swinburn, 2004). In summary, changes in environmental exposure, lifestyle, and typical meal-time practices in the developed world from the twentieth century have been linked to increased risk of poor food choices.

Cost of energy dense food and consequences for food choice. The spread of obesity in Western countries has not been uniform with some demographic and socio-economic regions within these countries having higher rates of obesity than other local regions. Lower socio economic areas have higher rates of obesity and some have linked this to the lower cost of energy dense foods (Anderson & Butcher, 2006; Jeffery et al., 2006). Not only are these foods less expensive, they are also more palatable and convenient to purchase (Drewnowski & Darmon, 2005). Living in close proximity to fast food outlets has not necessarily been associated with higher BMI (Jeffery et al., 2006), yet access to foods containing lean meats,

fish, fresh fruit and vegetables recommended to provide a healthy diet can be comparatively costly (Drewnowski, 2004; Drewnowski, Monsivais, Maillot, & Darmon, 2007; White, 2007). Greater cost consequently precludes some people from consuming the recommended daily nutritional intake found in healthy foods. For example, in Britain, lower socio economic populations who shopped for food were shown to purchase fewer fruit and vegetables, more meat, more fat and sugar compared to higher socio economic groups (Block et al., 2004) implying that low energy density healthy foods are more expensive to purchase than energy dense food, and when deciding which healthy food to purchase, meat was prioritized over fruit and vegetables.

In Australia the incidence of fast food consumption is reported to be greater in households that have financial and physical barriers to purchasing healthier food such as bread and milk (Burns, Bentley, Thornton, & Kavanagh, 2015). Research also points out that the frequency of fast food consumption is associated with poor exercise habits, working outside the home, and with having children (Jeffery et al., 2006). Therefore, in addition to lower socio-economic position, families with children who have working parents may be at greater risk of exposure to fast food and to perpetuating poor food choices within the family environment. Other research indicates that a lack of nutritional knowledge and food advertising also play a role in influencing unhealthy food choices and purchasing (Anderson & Butcher, 2006). Therefore, empowering families to moderate their chronic disease risk through dietary change should increase knowledge of the links between nutrition and subsequent disease, while enabling greater access to improved low energy density food choices in an otherwise obesogenic environment. Food choice within the family context however, may not be an individual decision with responsibility for overall food choice being shared by a number of people within families (Dunn, Mohr, Wilson, & Wittert, 2011). Given that early socialisation within families is a powerful, developmental influence on habitual

dietary behaviour (Nestle et al., 1998), exploring how decision-making around food occurs within this context is critical.

What influences proximal level food availability and food choice in the family?

Typical food intake in families, including the energy density of diets, is associated with a constellation of food-related behaviours that include purchasing decisions, meal preparation strategies, and control of how, when and by whom food is accessed. Each of these behaviours, in turn, will be influenced by social and physical contexts with habitual food choice as an adult at least partly determined by behavioural patterns laid down through childhood and learnt in the family home. The 'conceptual model of the food choice process over the life course' (Sobal, Bisogni, Devine, & Jastran, 2006) provides a basis from which to understand the complexity of what shapes, and what behaviours operationalize, food choice. This model adds the dimension of time across the lifespan to the macro and micro level determinants impacting on childhood obesity as was outlined earlier by Rosenkranz and Dzewaltowski (2008). This conceptual model considers how life trajectories shaped by present contextual experiences, and by previous experience also contribute to food choice (see Figure 2).

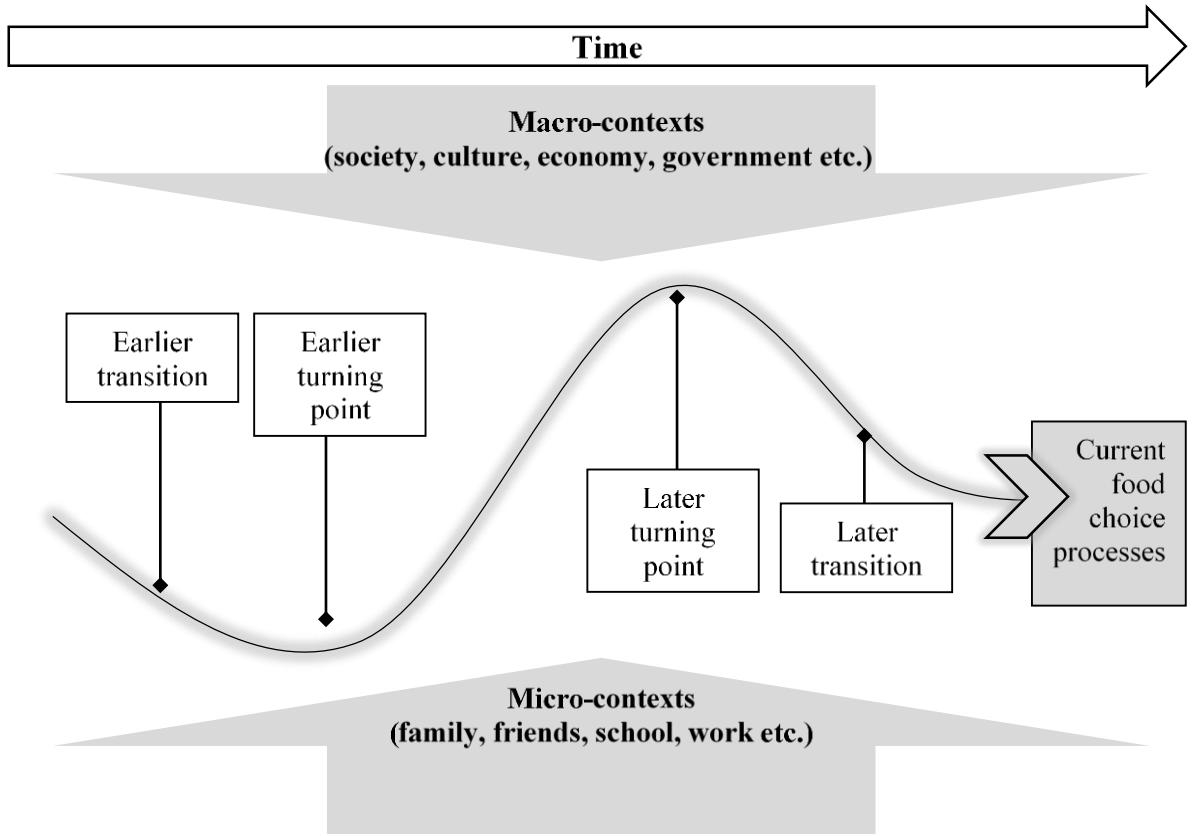

Figure 2. A conceptual model of how food choice is shaped by contexts over time to form a food choice trajectory. Adapted from Devine, Connors, Bisogni, & Sobal, (1998) by Sobal, J., Bisogni, C.A., Devine, C.M. and Jastran, M. (2006) in Chapter 1: "A Conceptual Model of the Food Choice Process over the Life Course" p. 4, in the book: R. Shepherd, M. Raats & Nutrition Society (Great Britain) (Eds.), *The psychology of food choice*. Wallingford, Oxford: CAB International. Copyright.

Consistent with the conceptual model of food choice, a systematic review of research on dietary development through childhood has identified the critical role of various forms of food familiarity; visual appeal, taste and context on children's food choices (Aldridge, Dovey, & Halford, 2009). Empirical evidence suggests that the development of infant preferences for different types of food will be influenced not only by food exposure, but by how the parent chooses to deal with feeding practices (Alm, Olsen, & Honkanen, 2015) as well as which foods are made "accessible" to children. It is likely that these practices reflect parental concerns about the role of food but also broader environmental issues like food security. For example, historically, encouragement to eat large portions was a practice used in times of food scarcity to prevent child starvation, however, this persists as a common feeding practice today despite excess food availability and over-consumption being commonplace (Birch, Savage, & Ventura, 2007; Blake, Wethington, Farrell, Bisogni, & Devine, 2011). Moreover, this feeding style may also be linked to a concern to minimise food "wastage", a common response from generations exposed to the Great Depression, and among immigrants fleeing starvation (Banwell, Broom, Davies, & Dixon, 2012; Wilson, Renzaho, McCabe, & Swinburn, 2010).

Over and above the influence of what food is purchased and brought to the house (i.e., availability) is the issue of what food is made "accessible", to which family members, and under what circumstances (Hearn et al., 1998; Story, Kaphingst, Robinson-O'Brien, & Glanz, 2008). Wider societal norms impact attitudes but social norms within the family that define "appropriate" or "acceptable" eating behaviours and food consumption are likely to be dominant (Ball, Jeffery, Abbott, McNaughton, & Crawford, 2010). Evidence suggests that families share social and behavioural norms for diet (Ball et al., 2010), as well as attitudes to food (Rasmussen et al., 2006; Shaikh, Yaroch, Nebeling, Yeh, & Resnicow, 2008) and associated health beliefs (Montgomery-Anderson & Borup, 2012). These findings highlight

the possibility that the family context is uniquely placed to serve as a target for behaviour change directed at a network of people; "the family".

As highlighted above, the importance of the family context for establishing appropriate behavioural choices is particularly important for children. For example, children aged as young as four already respond to cues in the social environment about food and are driven to eat by factors other than hunger alone (Patrick & Nicklas, 2005). Families that live together usually share the same social environment for a significant proportion of their lives, and this proximity has been shown to influence dietary behaviours (Chadwick, Crawford, & Ly, 2013). The mechanism of influence within the family is likely to include operant conditioning (i.e., reinforcement with punishment and/or reward that either decreases or increases dietary behaviours) but also extends to observational learning and modeling (Bandura, 1977b; Bussey & Bandura, 1984; Maccoby, 1992; Medin, 1992). Disease prevention in children has been considered important because food habits learnt in childhood are thought to track into adulthood, therefore campaigns targeting children focus on recommended fruit and vegetable intake (Resnicow et al., 1997). Underscoring the importance of the family unit as a social context within which healthy dietary choices may be made.

To date, empirical research has confirmed a strong link between behaviours promulgated within the family context and dietary outcomes. Thus family members shape the eating patterns and food quality of other family members through their sharing of beliefs to, and attitudes about food, the modeling of consumption choices, the constraining of food preferences by means of influencing both availability and accessibility, and the way that food is used within families (e.g., as both positive and negative reinforcement) including via restriction (Ajzen & Fishbein, 1980; Alm et al., 2015; Bandura, 1977b; Beech, Rice, Myers, Johnson, & Nicklas, 1999; Birch et al., 2007; Blake et al., 2011; Brown & Ogden, 2004). If

food choices learnt at home could be utilized in a way to impact chronic disease prevalence, understanding the family member best placed to influence others behaviour is crucial.

Identifying the key influencer of family diet and mechanisms by which dietary influences operate. Research suggests that certain family members are likely to play a more important role in shaping the dietary preferences of family members than others. As this chapter will outline further, the most commonly identified family member is the mother (Bouhlal, McBride, Ward, & Persky, 2015; Green et al., 2009; Jain et al., 2001; Johnson, Sharkey, McIntosh, & Dean, 2010; Longbottom, Wrieden, & Pine, 2002; MacFarlane, Abbott, Crawford, & Ball, 2010; McBride, Persky, Wagner, Faith, & Ward, 2013). The identified behavioural influence of mothers in the literature spans a number of different mechanisms, for example, modeling, parenting style, control of food availability and accessibility, and family mealtime practices and socialization. Bandura (1977a) drew particular attention to the behaviour of mothers and the potential for the imitation of modeled behaviours to impact children in the long term. Evans et al. (2011) found that when introducing healthy foods to the family diet, mothers have more influence than fathers and young children do. Although both parents have been shown to influence the eating attitudes, beliefs and behaviours of their children (Baker, Whisman, & Brownell, 2000; Cullen et al., 2000; De Bourdeaudhuij, 1997), overall, evidence suggests that the mother's influence predominates (Green et al., 2003; Green et al., 2009; Johnson, Sharkey, Dean, McIntosh, & Kubena, 2011; Johnson et al., 2010). For example, among Flemish children (N = 316), aged between two and seven years whose dietary behaviours were influenced by a number of parental behaviours including; verbal praise and food as reward (operant conditioning); intentionally abstaining from negative modeling; negotiation and discouraging sweets (authoritative parenting style); and parental food restriction (home food availability and parenting style), the mother was deemed more important (Vereecken, Keukelier, & Maes,

2004). In another study, mothers were found to shape taste preferences and food acceptance consistent with family food norms through modeling consumption and repeated exposure to certain food types in early childhood, (Chadwick et al., 2013).

The mechanisms by which a parent exerts influence on their children's behaviour are many and varied. They operate at a higher, potentially indirect level through mechanisms such as parenting style (e.g., Baumrind, 1991; Maccoby, 1992), socialization practices (e.g., Cullen et al., 2001), and at proximal more direct levels though the establishment of reinforcement schedules at home.

Proximal Level Parental Influences: Parenting Style and Socialization

Parenting styles have been categorized by Baumrind (1973) as authoritative (e.g., offering choice, negotiating), permissive (e.g., no restrictions), or authoritarian (e.g., insisting, using food as punishment or reward) (Nicklas et al., 2001). Neglectful (i.e., uninvolved) parenting has since been added as a parenting style by some researchers (e.g., Sleddens, Gerards, Thijs, de Vries, & Kremers, 2011). The authoritative style, using the combined principles of democracy and authority, has been described as the most conducive style to children's learning (Baumrind, 1973). This approach, when paired with recommended dietary choices, is thought to foster healthy eating habits in children (e.g., fruit and vegetable consumption; avoiding saturated fat or sugar consumption) (Sleddens et al., 2011; Vereecken et al., 2004). By contrast, authoritarian, permissive, or neglectful parenting, may be less successful in developing healthy eating habits, and reports suggest children parented in these ways develop negative health behaviours over time (Sleddens et al., 2011). For example, authoritarian parenting, which uses strong parental control, has been related to obesity and associated unhealthy eating behaviours in children (Cullen et al., 2000). In addition, children of authoritarian, permissive and neglectful parenting have been associated with higher BMI (overweight) and lower levels of positive health behaviours (e.g., nutritious

diets and adequate physical activity) when compared to children of authoritative parents (Sleddens et al., 2011).

Proximal, Direct Parental Influences on Children's Dietary Behaviour

The proximal (direct) influence of parents on children's food choices arises from the social learning that occurs within the family. Social cognitive theory (SCT; Bandura 1977a, 2001) describes a mechanism by which families promulgate dietary choices across generations. According to SCT, behaviours are transmitted to others by repeated opportunities for observation over time with some frequently observed behaviours internalized even before a child can talk (Bandura, 1977b). This theory, and how it relates to dietary behaviours, demonstrates the importance of research addressing the unintended and unanticipated consequences of habitual behaviours displayed by parents, including food selection, preparation, food preferences and consumption. The effect of modeling on children's food consumption has been confirmed in experimental studies (Cullen et al., 2001), with research showing that food preferences can be modified using modeling in combination with food exposure, to turn around children's existing food dislikes into food acceptance (Wardle & Cooke, 2008).

Other research suggests that social learning and the importance of modeling continues into late childhood and adolescence. For example, the important role of parents as models on children's dietary behaviours between the ages of nine and 13 has been evidenced by significant positive correlations between child and parental fruit, juice and vegetable consumption (Cullen et al., 2001). Hence, the adult role-model most responsible for family food decision-making plays a key function in the subsequent consumption of offspring.

The resemblance in food intake between family members. Obesity and chronic disease risk are two factors that appear to cluster in family networks (Koehly et al., 2009), and reasons for this may be due to the resemblances in food consumption between mothers

and children from early childhood (Feunekes, Stafleu, de Graaf, & van Staveren, 1997), through adolescence (Feunekes, de Graaf, Meyboom, & van Staveren, 1998) and into adulthood (Prichard, Hodder, Hutchinson, & Wilson, 2012). For example, Feunekes and colleagues (1998) used a food frequency questionnaire to assess fat consumption in 361 social networks comprising adolescents aged 15 (n = 347), their mothers (n = 309), their fathers (n = 270), their friends (n = 240), 79 friends of mothers, and 29 friends of fathers. Results within nuclear families demonstrated the intake of 76 to 94% of all foods were significantly correlated between parents and their adolescent children, and between marital pairs. Parents had significantly similar fat intake, and mothers had stronger associations of food consumption with their adolescent children than fathers did. These findings are consistent with earlier research with parents and younger children showing significant resemblances in family members' fat intake (Feunekes et al., 1997). In contrast, no significant resemblances were subsequently observed between the fat intake of adolescents and their friends (Feunekes et al., 1998) suggesting that the family is the more important social context for influencing dietary choices. The underlying mechanisms remain largely unknown although learning theory, as detailed earlier in this chapter, provides guidance on one of the likely explanations.

Further evidence shows the intake of young children's fruit, vegetables, snacks, sweets and overall energy consumption has been correlated with their mothers' intake of the same food types and kilojoule consumption (Prelip, Kinsler, Thai, Erausquin, & Slusser, 2012). In a large study of over 2,000 parents and 2,000 children aged 2 to 18 years, familial resemblance in dietary intake was examined (Beydoun & Wang, 2009). Due to the influences of certain foods and beverages on obesity and disease, the intake of specific nutrients (e.g., sugar, fats, calcium, fibre, sodium), food types (e.g., fruit, vegetables, sweets, dairy) and beverages (e.g., sweetened and unsweetened) were examined. The findings revealed

moderate parent-child resemblances in the intakes of all food types with stronger resemblances for fat intake. Stronger associations were also noted between mother-child and mother-daughter intake than father-child relationships between parents and younger children (Beydoun & Wang, 2009). This suggests that the mother's influence is stronger than fathers, and mothers influence daughters more so than sons.

Another recent study examined the relationship between adult daughters' food intake (energy dense food, fruit and vegetables) and their perception of their mothers' consumption of the same food types (Prichard et al., 2012). Results revealed a significant positive correlation, even where mothers and daughters no longer resided in the same home, suggesting that maternal influence on their daughter's food choices persist beyond childhood and into early adulthood. When relationship closeness between mother and daughter was examined as a moderating variable, the results were not significant, suggesting that learnt dietary behaviours endured regardless of how personally connected the women were. These resemblances in dietary intake have been well-established in the literature; however, the underlying behavioural mechanisms require further research to identify them. Similarly, investigations into any upward directional influences that may exist from children towards parents are also needed.

Compared to mothers, less is known about fathers' influence on children's diet (e.g., Wake, Nicholson, Hardy, & Smith, 2007). Although some research has attempted to examine the father's role with a particular focus on the impact on child weight status. Some fathers appear to adopt different feeding practices and parenting styles to mothers (Mallan et al., 2013). The consequences of these differences are yet to be clearly identified and may vary across different family groups (Mallan et al., 2013). For example, in a systematic review of paternal influence on childhood obesity, French fathers were shown to model healthier eating habits than did fathers in the United States; feeding practices that were associated with

children's lower and higher BMI respectively in both cultural contexts (Fraser et al., 2011; Musher-Eizenman, de Lauzon-Guillain, Holub, Leporc, & Charles, 2009). Any further known influences are limited largely because studies correlating parent and child behaviours most often focus on mothers, or combine the results of mothers and fathers. The literature has focused predominantly on the impact of mothers, parents, and the child-care environment on children's food consumption and associated dietary behaviours, however, few studies have compared differing *intra*-generational influences between mothers and fathers, or fathers' influence alone.

If one concludes from the current evidence that differences exist between parents, it is important to determine what mechanisms of influence are being practiced by mothers compared to fathers. This literature gap needs to be addressed with research into the area of both *intra*- and *inter*- generational influences within various family relationship dyads that have the potential to impact on the dietary behaviours and subsequent health outcomes of offspring. Furthermore, little is known about the intergenerational effects of grandparents on the food consumption of grandchildren in their care, and how this may differ from that of the children's parents (Eli, Howell, Fisher, & Nowicka, 2016). Overall, this represents an intergenerational literature gap worth investigating further for any bi-directional multigenerational family influences on dietary choice and is a key focus of exploration in this thesis.

Intergenerational family influences on dietary behaviours beyond parent-child generations. Although the transmission of dietary behaviours has been investigated, for the most part, between two generations, emerging evidence suggests influence across three generations exists. For example, in a study of 650 dyads involving mothers of pre-school children and, to a lesser extent, grandmothers (six percent of study participants), the resemblances in dietary intake of snacks, sweets, fruit and vegetables were examined (Wroten

et al., 2012). Findings revealed significant positive correlations between mother/grandmother and child intakes for all food types (with the exception of snacks) and total energy consumption. One drawback of the study however, was the grouping together of mothers and grandmothers possibly due to limited grandmother participation. It is worth investigating whether any different feeding styles exist between mothers and grandmothers, given that social learning continues until adolescence, since mothers and grandmothers can have an impact on children's lifelong dietary behaviours. Research that separately explores differences between mother-child and grandmother-child influences on dietary behaviours of children beyond pre-school age is also needed and any intergenerational mechanisms of family influence requires further exploration.

The potential for members of the "extended"[2] family (i.e., members beyond that of parent and child) to have a significant role in shaping food-related behaviours may be linked to the increasing proportion of mothers in paid employment over time. Anderson and Butcher (2006) reported an increase occurring in Australia over recent decades. The Australian Bureau of Statistics (ABS, 2014b) confirm a high proportion (i.e., 48%) of Australian children aged up to twelve years required some form of child care in 2014, with the main reason given by parents as "work-related". These same data demonstrate the role of grandparents with 23% of two parent families and 22% of one parent families who each received regular assistance with child care from a grandparent in 2014. In comparison with recent decades, for example, between the years of 1984 and 2014 there has been little variability in the percentage of children under 12 years in the part-time care of grandparents (i.e., 26% in 1984, 36% in 1996, 37% in 1999, 33% in 2002, 33% also in 2005, and 31% in 2014; ABS, 1988, 1997, 2000, 2003a, 2005; ABS, 2014b). Therefore, one quarter of

[2] Includes others in the family in addition to parents and children.

Australian children under 12 years of age have been in the regular care of grandparents for over three decades, yet few studies have examined the potential role of grandparents on influencing the dietary habits of Australian children.

Little is known about what influence the extended family and significant others may have on the dietary behaviours of infants, children, or adolescents. To date, data suggest that many different people, both inside and outside of the family, may have a role in providing food to very young children (Wasser, Thompson, Siega-Riz, Adair, & Hodges, 2013) and it is important to recognise this because of the potential for behaviours learned in childhood to continue to impact on dietary behaviours later in life (Contento, Williams, Michela, & Franklin, 2006; Savage, Orlet Fisher, & Birch, 2007). For example, it is possible that food of differing nutritional value are provided by different family members, or that meal times and rules vary, or that serving sizes and meal frequency differ between these groups of care givers (Wasser, et al., 2013). To date, there is little in the published literature beyond bi-directional food influences between parents and children, even though understanding influences on the development of childhood dietary behaviour is critical in addressing the present-day obesity pandemic (Gluckman et al., 2011).

Influences on dietary behaviours within families are bidirectional, not only "top-down" from adults towards children. Research has demonstrated that children can also influence family health (e.g., Montgomery-Anderson & Borup, 2012) and family diet (Anderson, Must, Curtin, & Bandini, 2012). For example, food preferences of children aged between five and fifteen years have been shown to influence parental decisions about family meals by introducing novel foods into the family diet (Anderson et al., 2012; Chavda, Haley, & Dunn, 2005; Green et al., 2003). Whether other family members have similar or other bidirectional influences with children is an important issue for research due to the implications for long-term health outcomes (Berge, Arikian, Doherty, & Neumark-Sztainer,

2012). It remains largely unknown whether children influence the diets of grandparents and if this influence is positive or negative in terms of overall health. For example, Green and colleagues (2003) undertook a series of family interviews and focus groups to determine the nature of exchanges about food in the extended family. They reported that children asked both parents and grandparents to prepare specific types of foods that had not previously been consumed and that, on some occasions, these were subsequently incorporated into the family diet.

Attitudes Toward the Connection that exists between Health Behaviours and Consequent Disease

As previously outlined, a number of chronic diseases such colorectal cancer, breast cancer, heart disease and type 2 diabetes can be prevented in part by minimising one's risk with adherence to modifiable health behaviours, such as consuming a healthy diet and maintaining a healthy weight (Finer, 2010; NHMRC, 2013a). However, attitudes toward the connection that exists between health behaviours and consequent disease may vary between individuals or family members; with females across several countries having shown greater food attitudes linking diet with health than males (Rozin, Bauer, & Catanese, 2003). Diet-health food attitudes are said to motivate diet-health related behaviours. For example, individuals who believe that diet has an effect on one's health or subsequent disease (e.g., beliefs that diet can have an effect on obesity, cancer and heart disease) are said to have *diet-health/disease linked attitudes (DHLA)* (Rozin et al., 2003). Similarly, individuals who modify their diet to reduce the consumption of fat and salt, regularly make food choices that prioritise nutrition rather than taste (e.g., by trimming the fat from meat) are said to have *diet-health orientated behaviour(DHOB)* (Rozin et al., 2003).

Food attitude research by Rozin and colleagues has shown strong support for beliefs in the link between diet and health in four culturally diverse countries (i.e., the USA, France,

Belgium, and Japan). Results revealed gender differences across all cultures; males were more pleasure oriented and females were more health oriented in their food attitudes. Females were also shown to have greater concern for the diet of others, and this concern was greater in adults than children and adolescents. It was suggested that the reason for this was because women, more so than men, take on greater responsibility for food choice and preparation in most countries (Rozin et al., 2003; Rozin et al., 1999).

More recent research by Sharp and colleagues (2013), demonstrated a significant but small negative relationship between diet-health/disease linked attitudes and fast food consumption; with higher diet-health/disease linked attitudes associated with lower levels of fast food consumption; and a small positive relationship was shown between diet-health oriented behaviour and healthy eating, with higher diet health-oriented behaviour associated with higher levels of fruit and vegetable consumption. They also found a significant strong negative relationship between diet-health oriented behaviour and unhealthy eating practices; with higher diet-health oriented behaviour associated with lower levels of fast food and snack consumption (Sharp et al., 2013).

Previous research has indicated that the main determinants of influence on food consumption (besides gender, age, and socio-economic position) have typically been taste, cost, convenience, nutrition, weight concern, food preferences, food availability, time costs, and parental intake influencing children (Glanz, Basil, Maibach, Goldberg, & Snyder, 1998; Kratt, Reynolds, & Shewchuk, 2000; Krolner et al., 2011). However, it is suspected from the evidence that diet-health/disease linked attitudes and any pre-existing diet-health behavioural orientation may also impact on healthy and unhealthy food consumption (Rozin et al., 2003; Rozin et al., 1999; Sharp et al., 2013). One of the limitations of previous research has been that the influence of intergenerational family relationships on diet and health food attitudes has not been examined. Therefore, research is needed to examine the potential impact of

Food Life behaviours and attitudes on the dietary behaviours of grandparents, parents, and children. This thesis will add to the research literature by examining associations between the Food Life behaviours and attitudes of family members within three generations and their own food consumption, in addition to associations between the food attitudes of parents and the consumption of their offspring. In doing so, this potential explanation for the mechanism underlying resemblances in food intake between family relationship dyads will be explored further.

The Role of the Family in Health Behaviour Management

An individual's dietary behaviours do not occur in isolation and are instead influenced by the decision making of others in the family. For example, an individual with heart disease may wish to reduce saturated fat and salt intake; however, another family member may be responsible for preparing all family meals. Therefore, an intervention for dietary behaviour change that is focused on the "at-risk" individual may be hard to sustain if the key influencers in the broader family are not contributing to any necessary behavioural change strategies. Family-focused interventions have not been extensively utilised in adult chronic disease management research. Research shows that stable, supportive family relationships contribute to shared responsibilities that ease an individual's emotional and behavioural burden of disease (Cousins et al., 1992; Rolland, 1987, 2005). Therefore, interventions aimed at changing dietary behaviours to prevent chronic disease (e.g., cancers, heart disease, and T2D) may be more successful by using a whole of family approach (Institute of Medicine (US) Committee on Health and Behavior: Research, 2001).

Dietary Behaviours can be influenced by Family Health History Information

Individual free will is a determinant of healthy dietary choice, however, collective agency can be utilised when a group of individuals are committed to shared intentions whereby individuals act interdependently to achieve healthy outcomes (Bandura, 2001). One

way to mobilise the family to engage in healthy dietary behaviours that prevent chronic disease may be to inform them of their familial risks for chronic disease through provision of family health history and disease risk information. Motivating healthy dietary behaviour change can be achieved by informing families of their familial health risks for chronic disease (e.g., Koehly et al., 2015; Ruffin et al., 2011). Evidence supports family health history as a way to motivate some people to adopt prevention measures such as healthy dietary behaviours or other health related behaviours such as disease screening. Within the family setting, family members can encourage each other to act in accordance with, and adhere to, recommended healthy dietary behaviours to prevent obesity or disease (Lykins et al., 2008).

Individuals who have a family history of chronic disease such as colorectal cancer, breast cancer, heart disease, or type 2 diabetes are said to be at higher risk for developing these chronic diseases themselves (Barrett-Connor & Khaw, 1984; Chang et al., 2011; Fuchs et al., 1994; Kahi & Lieberman, 2016; Koehly et al., 2009; Melvin et al., 2016; Seaborn et al., 2016; Wing, Venditti, Jakicic, Polley, & Lang, 1998). This is due not only to genetic risks but to the shared environment as well. As presented earlier in this chapter, families usually share the same environment for a significant proportion of their lives, and this proximity is likely to encourage the development of shared behavioural norms for diet as well as associated health beliefs and attitudes (Ball et al., 2010; Haslam, Jetten, Postmes, & Haslam, 2009; Rolland, 2005; Wardle, 1993). It follows that health beliefs that have developed in the family context can impact upon the way individuals respond to chronic disease diagnosis and disease management (Rolland, 1987). Health beliefs developed within the family context could therefore also impact upon one's responses to chronic disease risk and subsequent behavioural health actions.

Familial risk plays an important role in the assessment of chronic disease risk and is a method currently used to identify people who are at increased risk of a range of

chronic diseases (Butterly et al., 2010; NHMRC, 1999; NVDPA, 2012; Reifsnyder & Leiter, 2000; The Royal Australian College of General Practitioners (RACGP), Australian Government Department of Health and Ageing (AGDHA), The Cancer Council Australia (CCA), & Australian Cancer Network (ACN), 2008; Valdez, Yoon, Qureshi, Green, & Khoury, 2010). In some instances, asymptomatic individuals identified at increased familial disease risk (i.e., prior to acquiring any form of chronic disease), are reported to have engaged in preventative health behaviours (Chang et al., 2011). Recent research has demonstrated the effectiveness of family health history and any associated risk feedback as a motivational intervention for health behaviour change, including dietary change between mothers and young children (Koehly et al., 2015; McBride, Koehly, Sanderson, & Kaphingst, 2010; Ruffin et al., 2011). However, more research is needed to demonstrate its effectiveness with three generation family relationship dyads. To assist in understanding how family health history can be used as a motivational tool to change dietary behaviours, the underlying theory will be outlined to follow.

The Health Belief Model (HBM) as a predictor of individual dietary behaviour

The variables that influence the behavioural choices made by individuals that impact on health, including diet, are described in a number of different psychological health behaviour theories (Conner & Norman, 1995; Sheeran & Abraham, 1996). The Health Belief Model (HBM) developed in the 1950s, provides one such framework for understanding the factors that motivate health behaviour change and is one that remains widely used today (Glanz & Bishop, 2010; Janz & Becker, 1984; Leung, Wong, & Chan, 2016; Linke et al., 2013; McWhirter & Hoffman-Goetz, 2016; Rosenstock, 1974; Scarinci, Bandura, Hidalgo, & Cherrington, 2012). This model may be used to understand health motivation and behavioural change in chronic disease prevention. According to this model, the benefits of engaging in health enhancing, or the disadvantages of engaging in health compromising, behaviours

depend upon an individual weighing up the perceived risk and severity of a potential disease with the likely benefits and barriers of taking any relevant health action (Conner & Norman, 1995; Harrison et al., 1992; Linke et al., 2013; Sheeran & Abraham, 1996). In general, it is widely accepted that individual behaviour change can be predicted by using this model (Strecher & Rosenstock, 1997).

There are two components and four health beliefs comprising the HBM. The two components assume that firstly, an individual's goal is to avoid illness, and secondly, succumbing to illness can be avoided by adopting healthy behaviours (Janz & Becker, 1984). The four health beliefs comprise *perceived susceptibility* to illness, perceived *severity* of illness, and weighing up the perceived *benefits* and *barriers* of any preventative health actions (Conner & Norman, 1995; Janz & Becker, 1984). Briefly, according to the HBM, health outcomes depend on whether an individual sees illness as one that: may affect them, is severe, and risk reduction by health action comes at not too great a cost. For example, Food Life attitudes that support beliefs in the link between diet and consequent disease may explain one of the motivating factors that encourage adherence to healthy dietary behaviours and discourage unhealthy dietary behaviours. Evidence supports the use of the HBM with interventions designed to modify dietary behaviours in: families with obese children (Becker, Maiman, Kirscht, Haefner, & Drachman, 1977), dietary interventions for adults in the prevention of heart disease and cancer (Abood, Black, & Feral, 2003), and strategies that promote parents intentions to participate in parenting programs (Salari & Filus, 2016). Hence, the Health Belief Model is relevant today in health behaviour research that predicts actual and intended behaviour change relating to adults and children.

Following the HBM, each of the four beliefs (*italicised below*) are used to understand how family health history and disease risk information can motivate people to engage in healthy dietary behaviours. For example, the Families SHARE workbook implemented by

Koehly and colleagues (2015), infers *susceptibility* by delivering tailored familial disease risk feedback for four chronic diseases (i.e., colorectal cancer, breast cancer, heart disease and type 2 diabetes) on a tailored family pedigree diagram; it provides information on the *severity* of the four chronic diseases; and provides simple, accessible healthy guidelines (e.g., increase fresh fruit and vegetable consumption) prompting families to weigh up the *benefits and barriers of any health action* (see the concept diagram in Figure 3 below). The workbook in its entirety is a disease prevention strategy aimed at the family level (Koehly et al., 2015). If disease prevention interventions aim to change the long term incidence of chronic disease, starting with modifiable dietary behaviours directed at the whole family may prove successful (Hendriks, Gubbels, Jansen, & Kremers, 2012). Following from this, one of the aims of the current thesis is to test the impact of a Families SHARE workbook in an Australian population.

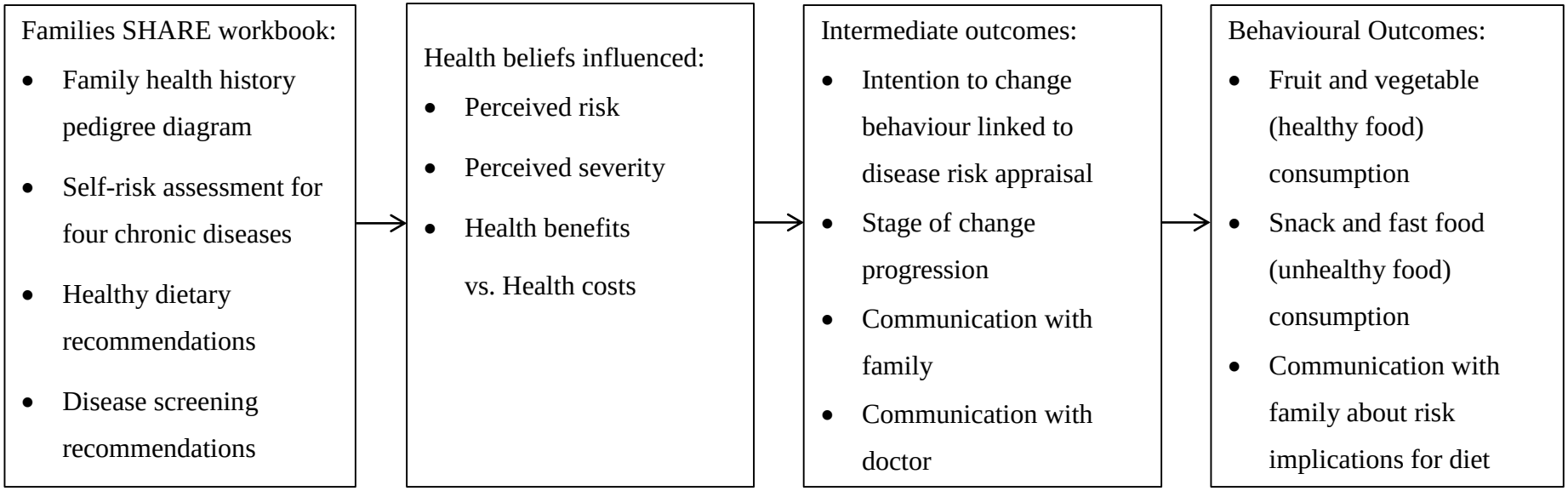

Figure 3. Conceptual model showing how tailored family health history, risk status and recommended risk reducing strategies would result in behavioural change.

Improving dietary choices within families utilizing the Health Belief Model to guide an intervention.

Disease diagnosis in close family members has also been shown to motivate healthy dietary behaviour in other family members (Beagan & Chapman, 2004)[3]. This body of research emphasises the important aspects of family influences on health behaviour change, not only in their shared biological disease risk histories, but also in shared psychological responses to any disease risks that act as the catalyst for subsequent health behaviour change. For children, family health history and disease risk information may be a key factor in reducing the health burden if families act early enough with appropriate dietary modifications that meet relevant health guidelines and recommendations. Yet to date, little is known of intergenerational family influences and how family relationships impact upon subsequent responses to familial disease risk information. Further research is needed to explore any effects beyond that of mother-child to include father-child, grandparent-child, and grandparent-parent dyadic relationships.

Exposure to familial disease risk information may provide the necessary stimulus to move individuals within the family to a more serious consideration of lifestyle change. However, not all families require health behaviour change since, due to prior experience of disease or for other health reasons, some people will already be engaged in behaviours that meet healthy guidelines. It is necessary therefore to take this into consideration when expecting behaviour change to occur over time in response to an intervention carried out under experimental conditions. Fortunately, stage of change theory can be applied to best capture any responses to motivational disease prevention interventions over time and the theory used to explain this concept is outlined to follow.

The Transtheoretical Model (TTM): Stage of change. Since only a proportion of families in any population will need to make the necessary dietary improvements to reduce their risk of

[3] Notwithstanding the potential for one's own life events and those of parents, family, and friends, with chronic disease to impact behaviour, research has demonstrated positive change in knowledge, beliefs, and behaviours related to fruit and vegetable consumption among Australian adults in response to the "Go for 2&5" fruit and vegetable campaign since its inception in 2002 (Pollard, Miller, Woodman, Meng, & Binns, 2009).

chronic disease, of those whose dietary behaviours could be improved upon, responses to family health history information may depend on their readiness to change behaviour. Readiness to change is a necessary precursor to any behaviour change and may be understood as a process of movement through a series of stages (i.e., 'stage of change', Transtheoretical Model (TTM); Prochaska et al., 1992). The TTM identifies an individual's readiness to engage in healthy behaviours. Stage of readiness to change progresses across five stages until behaviour change is successfully maintained (Sarkin, Johnson, Prochaska, & Prochaska, 2001). *Precontemplation* represents the first stage during which an individual has not thought about, and does not intend, taking any action to change health behaviour within the next six months. *Contemplation* represents the next stage where an individual seriously intends changing health behaviour within the coming three months, however, has not yet committed themselves to do so. This is followed by the *preparation* stage during which an individual intends changing health behaviour in the next month and has made behavioural steps towards doing so. *Action* represents the stage where an individual has already made changes to their behaviour but for less than six months and lastly, one enters the *maintenance* stage when healthy behaviour has been sustained for more than six months. Change is not always a linear progression through all five stages, rather a cyclical process whereby an individual may regress to earlier stages before attaining actual long term behaviour change (Prochaska et al., 1992).

The aim of providing tailored family health history and disease risk information to families is to motivate people who are at the precontemplation or contemplation stage, into the preparation for action, or the action stage of change. The advantage of this outcome measure is that it does not assume all individuals are at the same change stage at any one point in time (i.e., when baseline or follow up measures are administered). Individuals may be at any stage of change according to their own life experiences (e.g., experience of illness may change one's own dietary behaviour) or health beliefs (e.g., the degree that diet may have an effect on subsequent disease development or prevention) thus a range of stages are likely to be represented in any population sample at one single time point.

Conclusion

This introductory chapter has shown that overweight and obesity are modifiable risk factors for chronic disease which are partially influenced by an individual's lifestyle choices. These choices, in turn, are influenced by the broader contexts of one's circumstances such as the environment, socioeconomic position, healthful food availability in an obesogenic environment, dietary behaviours that develop throughout childhood: including immediate and extended family modeling, conditioning, and social norms. Moreover, engaging in healthy dietary behaviours is one way of reducing the risks for chronic disease. Since eating is a social behaviour, it makes sense to engage with influential individuals within the family context in order to initiate behavioural change. Accessing the benefits of family relationships and decision making may be one approach to facilitate successful dietary behavioural interventions. Through their collective agency, family relationships may be channelled into motivating others in the family to engage in healthy dietary behaviours (Bandura, 2001; Hendriks et al., 2012). By providing health history feedback to families and identifying those 'at-risk', it is anticipated that increased risk and the severity of four chronic diseases (i.e., breast cancer, colorectal cancer, type 2 diabetes, and heart disease) together will provide the impetus for three-generation families to see the benefits of disease preventative action (or intentions) by engaging in healthier dietary behaviours over time. This thesis aims to (1) explore the connections that exist between different family members in three-generation families and their perceptions surrounding food, (2) examine the effects that food attitudes have on healthy and unhealthy food consumption within three-generation families, and (3) test the efficacy of a family-based intervention (Families SHARE; Koehly et al., 2015) that targets disease risk factors to enhance health behaviours in three generation families.

Overview of Studies 1-4

The aims and objectives of this thesis are proposed to be achieved through a series of four studies that are outlined in more detail below (see Figure 4 below for a procedural flow diagram).

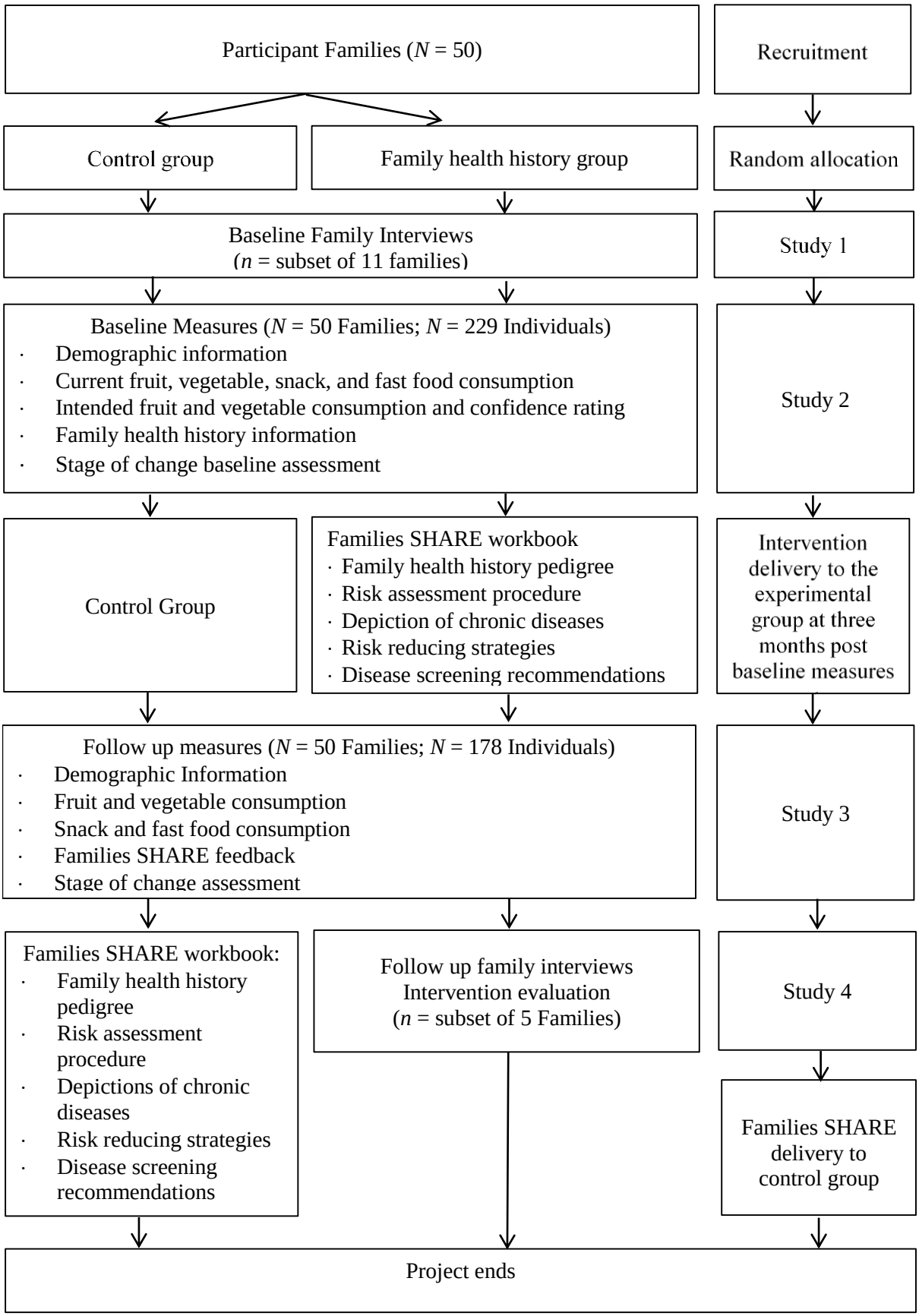

Figure 4. Procedural flow diagram with studies 1 – 4 represented alongside.

Four studies will comprise this thesis overall using a mixed methods project design. The reason for this is to quantitatively investigate relationships and causal mechanisms underlying dietary behaviours within families of three-generations, and to qualitatively understand the reasons why these behaviours occur from the participants own perspectives (Muntaner & Gómez, 2003).

Firstly, family interviews will be used in Study 1 to identify which family member (i.e., mothers, fathers, grandparents, or children) within three-generation families is most responsible for influencing the dietary behaviours of other family members, and explore what mechanisms of influence (i.e., behaviours) are operating. Interview studies are the most common form of qualitative health research methodology because of the important insights that can be gained from otherwise unexplored topics of research (Daly et al., 2007). Family interviews incorporate an interpretivist research method which follows inductive reasoning (Hennink, Hutter, & Bailey, 2011). In this way, the researcher works from the data collected in transcripts to determine patterns or themes that develop and support subsequent theories or hypotheses (Broom & Willis, 2007; Creswell, 2003; Saks & Allsop, 2012). Succinctly put, "*qualitative exploration into the background of a health issue may be embarked upon initially to tease out unknown factors which can then be tested for significance through quantitative measures*" (Macpherson, Bonita, Beaglehole, & Kjellström, 2008).

Study 2 will use a correlational design to examine the intra- and intergenerational associations that exist between family member dyads (e.g., mother-daughter) representing resemblances in dietary behaviours. Associations of attitudes to food as a potential mechanism of influence on dietary behaviours will also be examined. Food Life behaviours and attitudes will comprise two categories: self-reported healthy dietary behaviours, and attitudes that diet affects subsequent health or disease. A correlational study design is an appropriate method to examine the strength of associations between variables in health research (Cohen & Cohen, 1983). In science, correlations are based on law-like relationships indicating that one phenomenon occurs with another and can be explained by the shared variance between two (or more) variables (Ehrenberg, 1968).

Correlational designs have also been used in numerous research studies to develop theoretically based health behaviour interventions (e.g., Fishbein & Yzer, 2003) and this thesis will investigate the utility of a theoretically based health behaviour intervention in the subsequent study. Results of correlational studies do not imply causation, however in this thesis, will be used to establish the key associations that exist between family relationship dyads who share the same dietary behaviours (i.e., healthy and unhealthy eating), and Food Life behaviours and attitudes (Sharp et al., 2013).

Study 3 will be the intervention study investigating the effectiveness of a family health history and disease risk information workbook (Families SHARE) in motivating change in dietary behaviours over time. The Families SHARE workbook is an educational tool utilizing tailored disease risk information and healthy guidelines to increase user confidence in increasing healthy behaviours, including fruit and vegetable intake, with the aim of preventing chronic disease (Koehly et al., 2015). Study 3 will utilise unique family pedigrees to determine individual risk assessments for each family member. In doing so, dietary behaviour change over time will be examined in response to tailored family health history and disease risk feedback. A cluster randomised control trial (RCT) will be the method used to substantiate any significant findings. RCTs are considered one of the highest forms of evidence as has been shown on the hierarchy for evidence-based practice pyramid (Daly et al., 2007). If the aim of research is to ultimately have implications for health benefits, then the research needs to be of a high standard. More than any other research methodology, RCTs have a strong and well documented impact on evidence-based healthcare in the literature (Begg et al., 1996; Campbell et al., 2000).

Finally, Study 4 will evaluate the utility of the Families Sharing Health

Assessment and Risk Evaluation (Families SHARE; Koehly et al., 2015) workbook in motivating dietary behavioural change from the perspective of families participating in the research project. This will be achieved using a mixed methods study comprising family interviews and a separate survey. This research method uses triangulation methodology that refers to the use of two or more study methods to facilitate the validation of data. Validity is increased when the results

from two studies lead to the same conclusion (Given, 2008). By triangulating the research in this way, the results of one method complements the other and will provide a broader understanding that overviews the impact of family health history information on families from their own viewpoint.

CHAPTER 2: STUDY 1

Title: The intergenerational transmission of dietary behaviours: A qualitative study of Australian three-generation families[4][5]

Dietary intake is one modifiable risk factor that contributes to obesity and chronic disease. Dietary behaviours that develop over time are at least partly learnt by children through the social context of family life (e.g., Cullen et al., 2000). To date research has focused on the key unidirectional role of mothers who both model and reinforce specific dietary related behaviours to children (e.g., Feunekes et al., 1998; Feunekes et al., 1997; Green et al., 2009; Prichard et al., 2012). However, present-day family food choice is complex with a number of people sharing food choice and preparation responsibilities. To date, little research attention has focused on fathers' impact on the diets of children, or grandparents providing regular care of grandchildren who may also impact on children's dietary behaviours (e.g., Eli et al., 2016; Mallan et al., 2013), even though their feeding practices may differ from that of mothers. Evidence from other multicultural Western countries suggest that influences on food choice within families can operate bi-directionally and that influence may extend beyond two generations to also include extended family members (e.g., Forero & Smith, 2010; Green et al., 2003). It is important to understand what influence various family members may have on the diets of others within the family to better inform obesity and disease prevention initiatives that go beyond addressing the individual alone. The current study aims to address this gap in the literature to explore any intra- and intergenerational influences on dietary behaviours that exist, uni- or bi-directionally, within three generation Australian families.

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[4] Study 1 was presented as a poster at the International Congress of Behavioural Medicine in the Netherlands in 2014. The abstract was published in the Supplement titled: *Abstracts from the ICBM 2014 Meeting*. See Rhodes, K., Wilson, C., Prichard, I., Hutchinson, A., Coveney, J., & Ward, P. (2014). Dietary choices within multigenerational Australian families: Does the mother still play the most important role? *International Journal of Behavioral Medicine, 21*(Supplement 1), S28. doi:10.1007/s12529-014-9418-2.

[5] The research deployed in this chapter was later developed into a journal article, see Rhodes, K., Chan, F., Prichard, I., Coveney, J., Ward, P., & Wilson, C. (2016). Intergenerational transmission of dietary behaviours: A qualitative study of Anglo-Australian, Chinese-Australian and Italian-Australian three-generation families. *Appetite, 103*, 309-317. The Chinese-Australian and Italian-Australian participants were excluded from this dissertation.

The Present Study

The present study considers the uni- and bi-directional influences on food choices within families by conducting interviews with three generational Australian families. Thus, informing future family dietary interventions that reduce risks for obesity and chronic disease. Interview studies are the most common form of qualitative health research methodology because of the important insights that can be gained from otherwise unexplored topics of research (Daly et al., 2007). The study probes the following behaviours: purchasing decisions, food preparation, healthy and unhealthy dietary intake, with a focus on who makes or influences these decisions, the factors considered in this decision-making process, and the interactive nature of the influences. Any psychological mechanisms underlying these behaviours will also be identified and reported.

Aims

1. In order to inform family dietary interventions that reduce risks for obesity and chronic disease, Study 1 aims to identify which family members (e.g., grandmother, father, daughter) within three generational Australian families who most influence the dietary behaviours of other family members.
2. Using group family interviews this study aims to explore behaviours that contribute to mechanisms of influence within families, and the directions of influence between family members (i.e., uni-directional and bi-directional, intragenerational, and intergenerational).

Research Questions

To achieve these aims the following research questions will be examined:

1. Who are the key influencers in decision-making about food choice, preparation, and meal practices in three generational family networks?
2. What dietary behaviours are transmitted between generations and what direction do they take?

Method

Participants

The sample consisted of 11 three generation Australian families who were recruited from the community of Adelaide, South Australia (N = 50). Families were recruited between the 13th September, 2012 and the 17th September, 2013. Recruitment activities included placing flyers on community noticeboards, distributing invitation letters to schools, placing advertisements in newspapers, speaking on radio or to community centre groups, placing advertisements in social media and via email distribution lists through organisations such as the Cancer Council of South Australia and Flinders University. Inclusion criteria included having at least one child between the ages of seven to 18 years, one parent, and one grandparent. Additional family members could have included a second parent, sibling, a second grandparent or relative once removed (e.g., a great-aunt, uncle, cousin). Average family interview group size was four, with a range of three and six family members across three generations. All families identified themselves as Australian nationality and were of English speaking backgrounds. Families of ethnic backgrounds other than Australian and children under the age of seven were excluded from the study. All families were of middle class background and the parent generation in each family had some form of university education. The 50 participants in the present study comprised grandmothers aged 60 – 79, (n = 10; 20%), grandfathers, aged 71 – 80, (n = 4, 8%), mothers, aged 34 – 52 (n = 12, 24%), fathers, aged 35 – 51, (n = 6, 12%), daughters, aged 9 – 18, (n = 13, 26%), and sons, aged 7 – 18, (n = 5, 10%).

Research Method and Fieldwork Approach

A qualitative research method was used to identify intergenerational influences on food-related behaviour within Australian families. Rigour in qualitative research is underpinned by the appropriate use of qualitative research methodology, methods and procedures that design and carry out research (Hennink et al., 2011). Hence attention to detail and description has been used in the method section of this study.

Semi-structured family interviews used in the present study are a qualitative research method frequently used in health and social science research because they allow for the use of prepared questions whilst also allowing for some flexibility and participant elaboration (Liamputtong, 2013). Using a similar approach to that used in focus groups, an interactive discussion between participants (i.e., family members) about the topic of research interest is promoted to generate qualitative data (Krueger & Casey, 2009). This method allows data to be collected from the discussion points raised by the facilitator as well as from the dynamic interaction between family members that occurs when points are further refined and clarified, thus eliciting rich information (Hennink et al., 2011; Morgan, 1997). Family interviews incorporate an interpretivist research method which follows inductive reasoning (Hennink et al., 2011). Hence, working from the data collected in transcripts, the researcher determines what patterns or themes develop that support subsequent theories or hypotheses (Broom & Willis, 2007; Creswell, 2003; Saks & Allsop, 2012). The present study involved a series of family interviews undertaken within each family home at a time that was convenient for the participants.

Materials

Demographic information. Participants were asked to indicate their name, their age and each person's position in the family in relation to the 'nodal'[6] child (e.g., mother, grandfather). A node is an identifier within a family pedigree diagram, from which all family members can also be identified according to relationships with the node.

Procedure

Family interview discussion guide development. To facilitate discussion within interviews an 18-point family interview discussion guide (Appendix 2.A) was prepared rigorously using procedures described by Krueger and Casey (2009) with specific guidance as described by Krueger

[6] The node is an identifier from which other group members may be identified according to their relationship with the node.

(1998a).

Content. The discussion guide was based on the research questions that had been developed from a review of the literature. The content was developed encompassing eight topics that arose from these research questions. The topics were 1) Eating occasions, 2) Discussing food, 3) Meal planning and preparation, 4) Food purchasing, 5) Food types, 6) Multicultural food influences, 7) Family food rules, 8) Overview. Several individual discussion points were developed under each topic heading with the expectation that an interactive discussion between family members would ensue. The research questions were placed in a table and each discussion point was aligned against the most relevant research question. Drafted discussion points were reviewed by two experts in the field (John Coveney and Paul Ward) then shared with the research/supervisory team and further revised.

Design and facilitation. The discussion guide was designed to be flexible enough to encourage a conversation-like discussion, rather than a straight question and answer format. In this way, the moderator was able to establish rapport and gather data, some of which resulted from following up with supplementary questions when the conversation took a particular direction. The interview discussion guide began with questions that asked family members about their usual meal and snack times during the day. Participants were then asked to discuss: which family members were most responsible for family food purchases, meal planning and preparation, the types of food eaten and how this may have changed over time, and the family food rules. The discussion questions were pilot tested with a sample of participants who were not involved in the study. Over the course of the family interviews, best practice suggests that existing discussion points are refined as necessary, such that the clarity of subsequent family interviews improve (Hennink et al., 2011).

This is an iterative process; decisions were made during the research study to make changes that subsequently improved the research process (Hennink et al., 2011). In doing so, the research study evolved over the course of the interviews and the participant's narrative was elicited more naturally. For example, it became apparent in the first interviews that the opening discussion point

was confusing for participants, because clarification was requested (i.e., originally the first question was, "*First, let's talk through a typical weekday and the occasions that involve eating food. I'd be interested to hear about your typical eating occasions, such as meals, snacks, coffee breaks*?"). After revision of this discussion point, subsequent interviews began with a simplified and more conversational opening discussion point (i.e., "*First, let's talk through your usual weekday, and the times of day that involve eating food. For example, when would you usually have meals, snacks or coffee breaks*?"). The interviews that followed this change were understood immediately and were answered without requiring further clarification by the moderator. The brief family interview discussion guide is provided in Appendix 2.A, and the full version, inclusive of probing questions, is provided in Appendix 2.B.

Family interviews. Family Interview Information Sheets invited participation in Study 1 (see Appendix 2.C). Family members contacted the moderator directly to make an appointment. All interviews were conducted at the participants' preferred family home (either the parent's or the grandparent's) at their convenience. Prior to commencement a Letter of Introduction (see Appendix 2.D) was provided then Consent Forms (see Appendix 2.E) were obtained from each family member and parents signed on behalf of children who also provided verbal consent. All family interviews were audio-taped on two devices for transcription purposes. Interviews were moderated by the author (a fifty-year-old Anglo-Australian woman who had completed professional development in focus groups at Flinders University, South Australia) and included one of two note-takers (an Anglo-Australian woman in her forties who was a trained clinical psychologist, or a Chinese-Australian woman in her thirties) to facilitate each scheduled interview. The note-taker supported the moderator by allowing her to focus completely on the moderation task itself. The note-taker sat outside of the group circle and was therefore able to also inconspicuously write down her observations of non-verbal communication. Facilitation of the interviews was conducted according to focus group procedures and strategies as described by Krueger (1998b). Moderation procedures used in the present study included summarising participant responses to confirm that

content and meanings were clearly understood; asking participants to "think back" to facilitate thoughtful recollection; and probes were used as prompts or to encourage elaboration. Additional moderator techniques assured that all family members had equal opportunity to participate by purposely encouraging quiet participants and politely deflecting more dominant participants. For consistency, all family interviews were conducted by the same moderator (and using one of the two note-takers). Each family interview typically took between 40 and 60 minutes. Upon conclusion of the interviews individual participants each received a supermarket voucher to the value of ten dollars to compensate them for their time. As soon as practicable after each interview the note-taker and moderator met for a debriefing to share and discuss their insights (which were also noted).

Krueger and Casey (2009) have suggested that best practice requires focus groups (or group interviews) be continued until data "saturation" is reached. This is the point at which no new information is obtained (Hennink et al., 2011). Typically, this is operationalised by reaching a predetermined number of repeated interviews in which no new issues are raised (Glaser & Strauss, 1967) and, at this point, it can be assumed that all issues and perspectives have been fully identified and explored (Liamputtong, 2013). The number of interviews recommended varies between sources, with a range of anywhere between three and twenty group interviews that might be required to reach saturation (Glaser & Strauss, 1967; Hennink et al., 2011; Krueger & Casey, 2009; Liamputtong, 2013). Hennink et al. (2011) has described the variables that are likely to influence the rate at which saturation is achieved. These are: topic and research specificity (more specific topics are saturated more quickly); homogeneity of the study population (greater homogeneity, fewer interviews); extent of participant segmentation (more segments, slower saturation); and the requirements of past, similar research. The family interviews undertaken here involved a tightly defined topic area; English-speaking, Anglo-Australian families only; and segmentation by three generations. Past qualitative research with families on this topic has utilised ten families (Feunekes et al., 1998), with another study looking at social trends undertaken with three generations collected data from seven families (Banwell et al., 2012).

On the basis of the guidance provided by Hennink et al., (2011), the initial plan was to complete an estimated ten family interviews. Consistent with the advice of Francis et al., (2010), continuing beyond ten would only occur if new information was evident in each additional interview, and it is suggested that ceasing occurs at the third consecutive interview that elicits no new information. In the present study, data saturation was achieved at the 9th family interview with no new themes emerging in interview 9, 10 or 11. The sample size allowed for some diversity in family structure and function (e.g., single parent families, step-parent families, nuclear families, all with various extended family members), and in dietary choice (e.g., vegetarian, vegan, red meat, and restricted red meat consumers).

Data Analysis

Family interview data were analysed after first transcribing audio recordings verbatim. Transcripts were progressively imported into QRS NVivo™ computer software for coding and thematic analysis while data were being collected. Each of the transcripts was closely examined repeatedly during *first cycle coding* to name and label the research question concepts in a manner consistent with the literature reviewed in the introduction. This was undertaken using structural and simultaneous coding methods (Saldana, 2012). Any additional concepts that emerged from the interviews were also coded (using open and simultaneous coding methods) until transcripts were comprehensively analysed during the first cycle (Saldana, 2012). Using QRS NVivo™, coded segments of text passages were grouped together to form "nodes" (e.g., broad topics such as 'mother influence') and "child-nodes" (e.g., smaller sub-topics branching from the broader nodes, such as 'mother to child influence'). During the *second cycle coding*, similar codes were then grouped into themes and labelled (using focused coding) (Saldana, 2012). Similarities and differences in themes that emerged from the data were analysed across all transcripts and compared between selected participant family groups (e.g., grandmothers, grandfathers, mothers, fathers, daughters, sons) (Saldana, 2012).

Decision making around themes (thematic analysis) focussed on capturing important

data relating to the research questions that represented a pattern within the overall data set (i.e., across all, but not necessarily included in all, family interview transcripts) (Braun & Clarke, 2008). The methodology used an *interpretivist* approach, hence the data were analysed beyond simply reporting the number of instances a topic occurred or by other quantifiable methods (Hennink et al., 2011). Similarly, the data were not reported at face value (i.e., simply what was said), rather, analysis occurred by examining then interpreting patterns of behaviours from participant's descriptions in their own words. For example, a grandfather said, "*We won't dare say what we give the grandchildren to eat when they are at our place!*" Further exploration revealed that children were knowingly given more treats to eat at the grandparent's home, than would normally have been allowed at the parental home. By exploring the data in this way, strong insights into the data were gained, interpreted, and reported. In this instance, this theme depicted grandparental influence that bypassed parents and indulged grandchildren in treats and was labelled "grandparent to grandchild influence". A full exploration of influences between family roles will be reported in the results and discussed in the discussion sections.

Rigour in the present study was strengthened in several ways. Firstly, three of the moderator's transcripts were checked for accuracy by supervisors. The remaining eight (out of a total of 11) audio recordings were transcribed by a professional transcriptionist to save time. The moderator checked each of the eight transcripts thoroughly against the respective audio recording for accuracy. The moderator then coded all transcripts. In addition, several of the final transcripts were subsequently printed for review by a supervisor and the emerging themes were discussed with the moderator. Finally, once analysis was completed by the moderator, five printed transcripts were independently and systematically analysed by the note-taker who was present at each of those five interviews. On completion, the moderator and note taker then discussed their respective findings by simultaneously examining each of the five transcripts until any differences were resolved and agreement was reached on all themes.

Ethics

Ethical standards were met and approval for the study was granted through the Flinders University Social and Behavioural Research Ethics Committee. The moderator and note takers each obtained a National Police Certificate prior to commencement of the study as part of the ethical requirements of working with children.

Results

Familial and intergenerational influences on dietary behaviours and food choices were evident in a bi-directional manner across all family relationship dyads. Some notable differences existed between generations[7]. The results are described in turn according to the generational location of influence under the following themes: The Influence of the Great-grandparent[8] Generation; The Influence of the Grandparent[9] Generation; The Influence of the Parent Generation; and The Influence of the Child[10] Generation (see Figure 1). Where gender differences existed in the present study, these are presented separately under each relevant generational heading. Intra- and inter-generational differences are addressed separately under the existing headings. The discussion section then summarises the results in relation to theory by addressing and integrating the research questions, drawing conclusions from the findings and suggesting directions for future research.

[7] Generations are defined in terms of relationship to, and distance from, the "nodal" child
[8] Great-grandparents did not participate in the study
[9] Grandparent's children are referred to as parents or mothers and fathers throughout.
[10] Children are referred to as children of parents or as sons and daughters throughout.

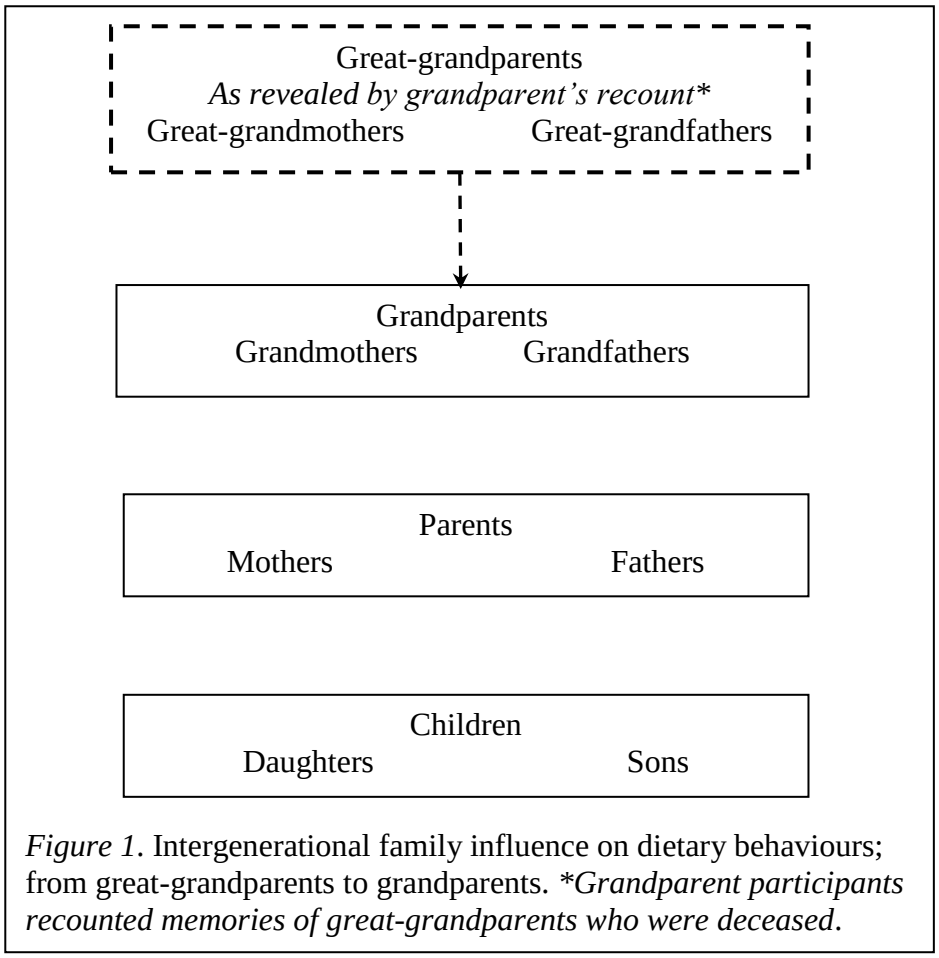

Figure 1. Intergenerational family influence on dietary behaviours; from great-grandparents to grandparents. **Grandparent participants recounted memories of great-grandparents who were deceased*.

The Influence of the Great-grandparent Generation (on Grandparents)

Great-grandparent generation influences were interpreted from *reports* made by the grandparent generation only. This generation was included because grandparents in the study described rich childhood memories of their own parents' influence on their food choices. Habits that may have been passed down the generations were considered important and therefore these data were retained. It is important to note that great-grandparental influences are limited by grandparents reported memories; great-grandparents were since deceased and it was impossible to obtain their independent verification of these verbal reports.

The older generation's diets were influenced by the effects of the Second World War (WW2) due to food rationing in Australia. Availability of foods high in saturated fats (e.g., bacon) were unrationed and other more "healthy" foods (e.g., dairy products) were rationed or prioritised for mothers and infants, therefore they were less available to the majority of people during that time

(ABS, 2002; Banwell et al., 2012). Families relied upon local seasonal foods and self-sufficiency practices such as owning chickens or growing fruit and vegetables. Growing home produce was typically a role of the men in the family in Australia at that time (Banwell et al., 2012). The grandparents in the present study recalled, as children, sitting at the table for meals and finishing what was on their plate thus minimising waste; they did not remember going hungry even though times may have been financially strained in those days.

Regional food scarcity affecting diet. As indicated above, contextual factors at play in the lives of grandparents made it essential for families to maximise the use of foods that were available to them at the time (e.g., grandfather, Colin, aged 79 cited: "*fried bread,*" and "*bread and dripping,"* because typical spreads such as butter were unavailable). Although the child and parent generation responded to some of the grandparent's descriptions of their typical childhood foods as "*yuck*" (daughter, Jade, aged 12), the grandparent generation described these foods as unhealthy but "*delicious*". This suggests that great-grandparent's regional food scarcity had an effect on food exposure and food acceptance by grandparent's during their childhood years. This example supports research that suggests childhood food exposure has implications for long-term food preferences that persist over the lifespan (Savage et al., 2007).

Some of the foods that were consumed by grandparents out of necessity, differed from, and were not accepted by subsequent parent and child generations. For example, grandparents Colin and Pam (both aged 79) recounted a time when Colin's mother cooked with saturated fat. Colin said: "*Well when you melt them* [pork fat] *down and you get two big pots of… uh lard like that. Oh, it's beautiful.*" When asked how it was typically eaten Pam said: "*You use it in cooking"* and Colin added: "*Spread it on baked bread or on your toast and more."* Lard was something they enjoyed at the time and they reminisced about it together. Pam recalls: "*Yeah, best we ever had."* Colin went on: "*And that's all we ever had in those days. But this was, you just put it in a saucepan – lumps of fat like that, it came out of the pig. You cut it all up, put it in the saucepan – big saucepan, and cook it, well melt it down. We used to have the lard from those. And it was beautiful. No good for*

you probably but it was beautiful."

Effects food scarcity had on subsequent food preferences. The grandparent generation also recounted memories of their time as children when they typically ate foods that might no longer be considered acceptable by younger generations, for example, brains, kidneys, and liver. Consumption of these was viewed as "not wasteful". Two sisters in the grandparent generation discussed eating some of these food types when they were young. Nancy (grandmother aged 67) started the topic with: "*And another thing, when we were kids we had to eat tripe* [stomach of a cow]." Her sister Audrey (great-aunt aged 69) said: "*Oh*." Nancy: "*Which I love, and Audrey can't stand it, and ah battered brains.*" Valerie (mother aged 43) interjected: "*Oh yuck*." Nancy continued: "*...which I loved. And, um I don't know if you* [Audrey] *like it?*" Audrey: "*Oh, I don't mind them but I remember we dished them up for Dad and told him they were fish fritters."* Steve (father 42): (*laughs*), Audrey: "*And he ate them and we all fell over…*" Facilitator: "*Laughing?*" Nancy: "*Lambs fry and bacon*." Audrey: "*I love it.*" Nancy: "*And that's quite nice, but mum used to…*" Audrey: "*I loved lambs fry* [liver] *and bacon."* Nancy: "*…but mum used to overcook the uh, lambs fry*." Then the discussion went on to finally include bacon and steak with kidney. The reactions of parents and children to grandparents' descriptions such as these examples above, suggest strong generational differences in food preferences. This also shows that different food types consumed as children have had long lasting effects on the food preferences of the grandparent generation.

Gender-based distribution of responsibility for food and health. Grandparents described their perception that their mothers (the great-grandmothers) had predominant responsibility for family meals. Fathers (the great-grandfathers) who came home after work were recalled as being served their evening meal and appeared to have little active role in family food related behaviours. Grandparents recounted memories that the great-grandmothers' role was to source all of the food, plan and then prepare all of the meals because great-grandfathers' worked. Grandmother Christine, aged 73, recalls the important role of her mother (i.e., the family's great-grandmother) in cooking

healthful foods: "*Yes. It did, we always had vegetables, yeah."* Facilitator: "*Yes, yeah*", Christine: "*Yeah which is a complete…* [meal]*"* Facilitator: "*And who in the family would have encouraged that do you think? Which of your parents, would that have been your Mum or your Dad?*" Christine: "*My mother because she was the cook*." This example demonstrates the mother's role, stemming from the great-grandparent's era, in taking care of family through her concern with healthy eating.

Grandparents recalled times when they were themselves children and their mothers urged them to eat fruit and vegetables. Christine (grandmother, aged 73) clarifies this: "*You just have to have fruit and vegetables, you know, it's something that I was brought up with, to have vegetables."* Facilitator: *"hmm okay."* Christine: "*I am not overly keen on vegetables, mind you, but you still have to eat them, don't you?"* Facilitator: *"So that has come down from your parents?"* Christine: *"Having to eat vegetables, yeah."* Facilitator: *"Yeah."* Christine: "*Yes. It did, we always had vegetables, yeah."* The previous two examples show that healthy food attitudes have been transmitted from great-grandmother to grandmother, who, despite disliking vegetables understood the importance of eating them nevertheless.

The Influence of the Grandparent Generation (on the Parent Generation)

Grandmothers made healthier food choices over time. Although grandparents reminisced about foods they enjoyed as children, and still liked, they were not opposed to changing to a healthier diet over time, which would have impacted on the parent generation as they were growing up (see Figure 2). For example, families discussed changes over time such as using olive oil instead of lard, steaming vegetables instead of pressure cooking them, as one father pointed out to grandparents Jennifer and Peter: Daniel (father, aged 45) stated: "*You don't fry as much now*", Jennifer (grandmother, aged 73) replied: "*Well I suppose, yeah*", Peter (grandfather, aged 76): "*You grill more don't you now?*", Jennifer: "*That's right, yeah*", Peter: "*Like we used to years and years ago, we used to always fry the chips, didn't you? If you did chips but,*" Jennifer: "*At one time, you know homes made fried chips for years at home*".

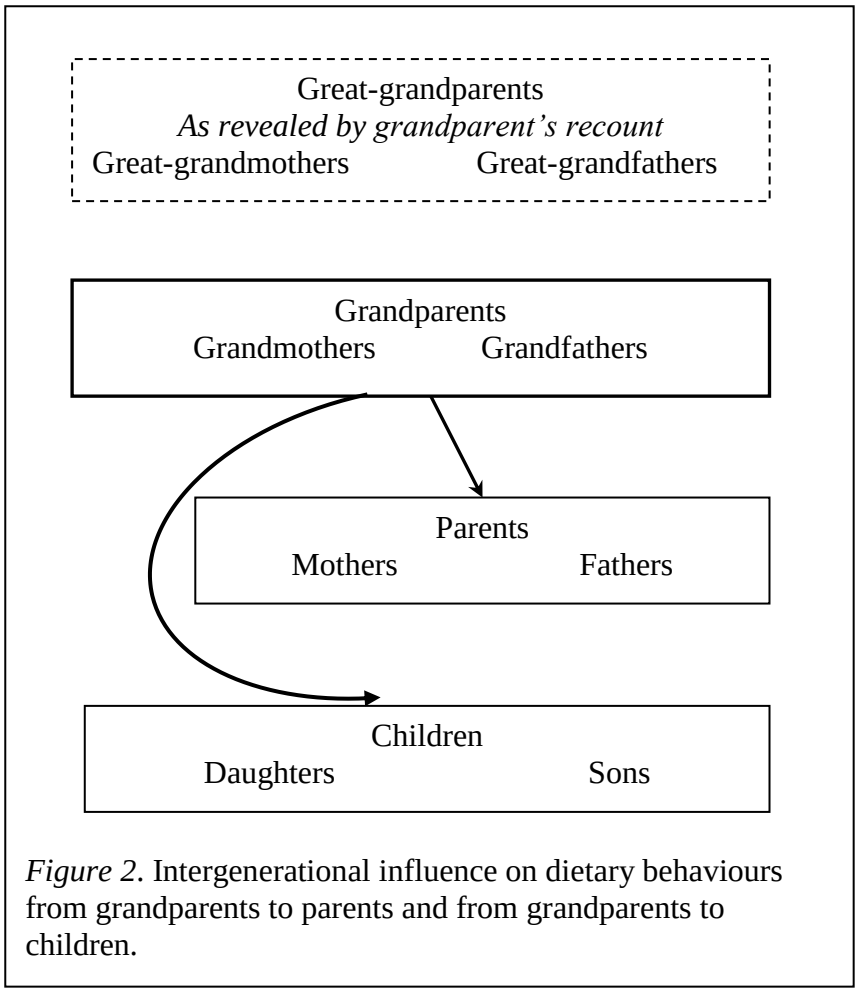

Figure 2. Intergenerational influence on dietary behaviours from grandparents to parents and from grandparents to children.

Grandparents preferences for plain and simple food persisted over time. Grandparents showed a preference for the plain food that they were exposed to as children, which they also prepared for the parent generation as children. For example, Barbara (grandmother aged 71) said: "*I'm very bland in what I eat."* and Brian (grandfather, aged 71) said: "*I don't go for anything too fancy. A plain food man I am*." Grandmother, Sandra aged 70, similarly said: "*You see, I do prefer bland food rather than spicy."* Grandparents in the present study denied eating fast foods. Christine (grandmother, aged 73) stated: "*I don't have takeaway, I don't really have takeaways, no.*" Grandparents had also refrained from much of the multicultural foods available in Australia over the same time period which the parent generation were more open to trying and providing for their children. For example, some grandparents would not eat pasta or rice because it was considered "foreign" food (of Italian or Asian origin). Peter, grandfather, aged 76, talked about his wife: "*Jennifer* (grandmother, aged 73) *doesn't like any foreign food at all, you know*." She replied: "*Not*

much." and Peter explained: "*She won't eat pasta or rice*," Facilitator: "*Ah, okay*," and Peter went on to say: "*Or anything, you know, only plain things.*" Another grandfather, Brian, aged 71, further elaborated on his preference for plain food. Brian: "*No, I'm not a spicy food man, so – not – European foods I don't take to, so*." Facilitator: "*English style cooking?*" Brian: "*English style cooking, yes, yeah.*" Facilitator: "*Yep. And that's stayed the same?*" Brian: "*Yeah, I'm boring.*" Gender differences noted in the grandparent generation are discussed separately to follow.

Grandmothers model mother's responsibility. Grandmothers had taught their children (the parent generation) the value of health and nutrition and how it was important to eat fruit and vegetables. Grandmothers in the present study were principally responsible for meal planning, preparation, and cooking. For example, Jennifer (grandmother aged 73) spoke with her daughter*: "But I probably decide more on food because I do the shopping for it."* Sarah (mother aged 41): *"And the cooking."* Jennifer: *"And the cooking yes."* Sarah: *"Yes."*

Grandmothers' primary influence on grandfathers' food consumption. Margaret (grandmother, aged 76) also stated: "*Well I mainly do the shopping. Sometimes Jack* [grandfather, not present] *will come and I just buy sort of, sort of what I think we need for the week and Jack will always ask about mid-morning what's for dinner at night (laughs). But as far as discussing meals he'll usually just sort of have what I cook and yeah."* Consistent with reported memories of their own parents' behaviour, grandmothers highlighted their dominant female role in food decisions and the comparatively passive role of the husband in accepting the meals prepared. An *intragenerational* influence was also apparent in this example because the direction of influence was transmitted from grandmother to grandfather (i.e., within the same generation) (see Figure 3).

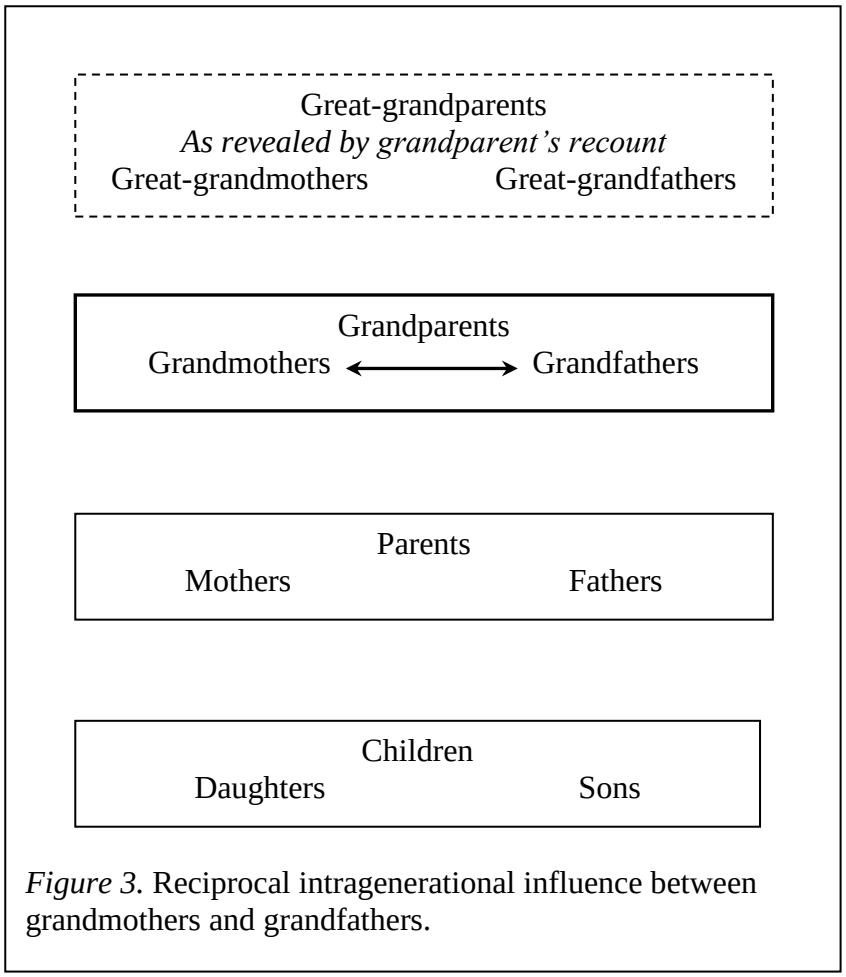

Figure 3. Reciprocal intragenerational influence between grandmothers and grandfathers.

Grandfathers did not object to, interject, or attempt to clarify, the family's assumption that his role was to unquestioningly accept whatever food he was provided with. "*He just accepts whatever* [food] *I give him"* (Pamela, grandmother, aged 79). Daniel (father, aged 45) called Jennifer's (grandmother, aged 73) attention to her husband's lack of involvement in food decisions. He said: "*There is no discussion about it is there? You say what you're going to eat, and then there it is!*" All: (*laughter*). In another family, Ailsa (grandmother, aged 60) stressed her authority in making food decisions; Facilitator: "*Oh, I didn't ask you* [grandmother] *about when you talk about food!*" Ailsa: "*I don't (laughs). I just eat it. My husband just eats what I put in front of him (laughs).*" One grandmother was the exception in the present study, indicating that she was tired of thinking about what foods to prepare and asked her husband for meal ideas.

Grandfather's food preferences influence grandmothers' purchasing and preparation. Most grandmothers however, over time, knew exactly what their own household's food preferences were, what meals were liked and disliked, and from this implicit understanding, seemed happy to

prepare the same types of foods for grandfathers over a long period of time (e.g., the Sunday roast and sometimes other foods eaten on the same day of each week). Overall, there was a strong grandmother to grandfather influence demonstrated regarding food decisions, planning and cooking. However, grandfathers' influence was reciprocated by his food preferences having been made known to grandmothers' over time.

Grandfathers model "father role" in facilitating self-sufficiency. Some grandfathers in the present study were involved in growing produce that was then used in meal preparation. These grandfathers shared with grandmothers' certain food preparation tasks such as pickling, freezing or preserving excess home-grown foods for later use. Some grandfathers cooked occasionally; meals that varied in complexity from the very simple, to quite complex, requiring reference to recipes. As reported earlier, however in an Australian historical context, like the great-grandfathers before them some of the men in the grandparent generation adopted the same self-sufficiency food practices as their fathers. Ailsa (grandmother, aged 60) recalled memories of her father keeping chickens: "*No. So, uh we did have our own chickens. I remember Dad doing them and me as a kid helping pluck them. I don't know how I ever did that…"* Pamela (grandmother, aged 79) and Andrea (mother, aged 47) talked about Colin's (grandfather, aged 79) vegetable garden;

Pamela: Broad beans. He's just had a…
Colin: Oh yeah.
Pamela: …heap of ripe broad beans.
Colin: Oh yeah.
Pamela: You've got beetroot in at the moment.
Colin: And deep freeze those for the rest of the year, yes.
Pamela: Tomatoes, lettuce.
Andrea: And you've got a good herb garden.
Pamela: Oh yes, good herb garden. Mm hmm.

The motivation for growing fruit and vegetables was generally economic; fresh produce was considered expensive for many families. For a summary of the influences associated with grandfathers, see Figure 4.

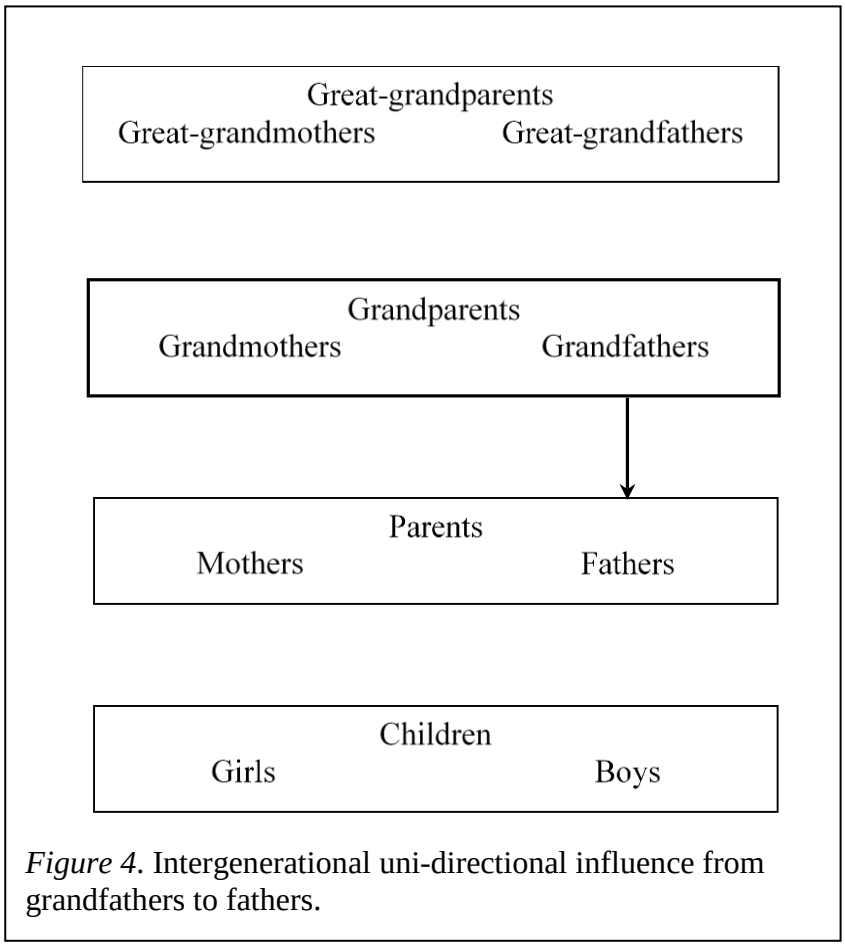

Figure 4. Intergenerational uni-directional influence from grandfathers to fathers.

The Influence of the Parent Generation

The direction of influence from parents, was intergenerational toward both the child and grandparent generations; and intragenerational between parents (see Figure 5). Mothers and fathers in the parent generation are discussed separately below in order to facilitate gender role-based contrast.

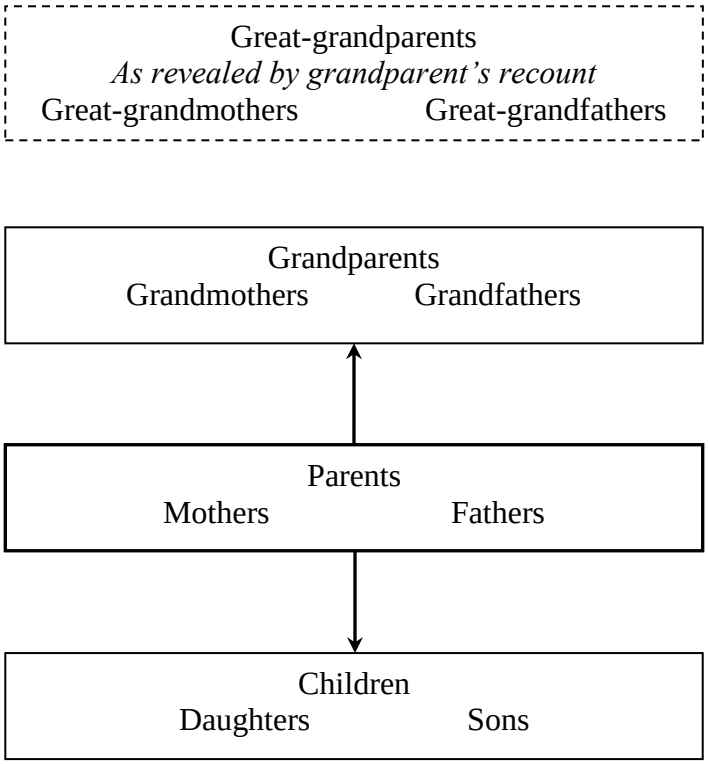

Figure 5. Intergenerational influence on dietary behaviours from parents to children, and from parents to grandparents.

Fathers facilitate ongoing self-sufficiency. As described earlier, influences were transmitted from grandfathers to fathers thus "self-sufficiency" home produce practices were then modeled to the children in the present study. Likewise, reports by grandfathers in the previous section indicated self-sufficiency was practiced by great-grandfathers also, suggesting influence over three generations of men. Simon (aged 48, the father in the same family as Colin, Pamela and Andrea mentioned in the previous grandparent section), talked about what he was growing in his vegetable garden;

Facilitator: So, some things are growing in the garden?

Simon: Yeah. Tomatoes, cucumbers, um lettuce.

Facilitator: Fresh foods.

Simon: Beetroot.

Facilitator: Hmm.

Simon: Stuff like that.

Although Cliff and Pamela were the maternal grandparents in the family above, Simon grew up on a farm and was influenced by the paternal grandfather's modelling and encouragement

to grow his own produce to be as self-sufficient as possible. The only difference noted was that the parent generation were motivated to grow their own produce for reasons beyond that of grandparents. Parents stated their reasons were also partly to do with thrift, although they placed much more emphasis on health-consciousness and broader global concerns that were not apparent in the grandparent generation. For example, health-conscious topics involved the importance of healthy and organic foods, avoiding the consumption of poor quality supermarket fruit and vegetables; and global concerns included reducing use of imported produce, waste reduction, and recycling leftover food. Simon (father, aged 48) and Renata (mother, aged 35) highlighted this point by saying, Simon: "*You don't waste money, but you don't…*" Renata: "*Well we don't throw anything* [food] *away. Things that we do end up throwing away go out to the chickens*."

Mum is in charge. Mothers in the parent generation strongly influenced the food consumption of the other family members in their households and, to a certain degree, beyond it to that of the grandparent household. In the present study, the important role of the mother was emphasised throughout by family members in each generation. For example, after presenting us with her monthly meal planner and spreadsheet, Kylie (mother, aged 45) concluded with: "*Yes, I do plan my meals. Yes, I do (laughs).*" Facilitator: "*So you're in charge of the planning…"* Kylie: "*Oh yes.*" Facilitator: "*…obviously.*" Kylie: "*Oh yes.*" Facilitator: *"Does anyone else?*" Kylie: "*Oh no.*" In another example, Andrea (mother, aged 47) stated: "*See it's mainly the female that organises the food.*" Jade (daughter, aged 11) argued: "*Oh Mum, come on.*" and Andrea justified further: "*It is. So, like I already know now… I always know what I'm going to have for tea* [evening meal] *the next night.*" Facilitator: "*Mm hmm*." Andrea: "*Talking to the aunties and mum. It was the females that organise it. The men do what we tell them to do*."

Most mothers, were similar to grandmothers who understood their husband's preferences, considered the likes and dislikes of the family as a whole in meal preparation, by preparing meals that they knew were most likely to be enjoyed by all members of the family. Lauren (mother aged 46) showed that she did not necessarily need to talk with family members to accomplish this.

Facilitator: "*Okay. So, when you're planning the meals for the whole family would you talk to Phil and Thomas about that – and Lily?*" Lauren: "*Not very much, because I sort of know what they like to eat and we kind of pretty much have a…*" Facilitator: "*So you don't need to?"* Lauren: "*No.*" Most mothers in this study also showed a preference for, and included, a variety of different foods. For example, Sarah (mother aged 41) said: "*Ah and also um, I don't know I like quite a lot of variety.*"

Dad cooks, but Mum remains in charge. In a few households, the fathers were responsible for the majority of the food shopping, preparation and cooking for the family, nevertheless, mothers still made a strong contribution to their family's meal content. One way this occurred was by mothers closely monitoring family fruit and vegetable consumption and reminding fathers to include more of these food groups in subsequent purchases or meals each week. For example, Nicole (mother, aged 44), and David (stepfather, aged 35), had explained earlier that David was the one responsible for most of the family meals, and the facilitator probed this further:

Facilitator [to David]: So if you are the one doing the planning, who talks with you most about food?
David: She [Nicole] tells me what we are having.
All: (laughter).
Nicole: Yeah, we'll talk about like, if we haven't had vegetables for a couple of nights…
David: Yeah.
Nicole: …or so, we will say we really need vegetables tonight, so we will pick something around…
David: Vegetables.
Nicole: Yeah around vegetables.

Another way that mothers remained involved was by preparing the shopping list that the father subsequently followed. For example, Daniel (father aged 45) illustrated how his wife Sarah (mother, aged 41) reminded him and placed food items on the shopping list, even though he was primarily responsible for the food shopping.

Daniel: You'll [mother] be reading a, a magazine article, or something about, how er broccoli is really good for you.
All: (laughter).
Daniel: And then.

Sarah: Put it on the shopping list or, yeah yeah.
Daniel: Or you will say, "We need to eat more broccoli." (laughter)
Sarah: Yeah.
Daniel: Ah… so that's, tends to be when it's discussed.

The Influences on Mothers

Health-consciousness transmitted from grandmothers evident in mothers' behaviour – was then transmitted to daughters. Mothers were influenced by the maternal grandmothers in the present study. An intergenerational influence from grandmother to mother was noted in relation to certain food types, for example, encouragement to eat nutritious foods such as fruit and especially vegetables. The mothers in the parent generation continued with the same focus on healthy nutrition for their own family. Health-conscious mothers in the parent generation also restricted the frequency of unhealthy snacks, confectionary, fast-food, or sugar-sweetened beverages to weekly or monthly; foods that were not as readily available during the grandparents' childhood era. For example, Nicole, (mother, aged 41): "*Yeah, we probably restrict soft drinks*"; and Sarah (mother, aged 44): "*Yeah and we, we have some rules around treats, don't we?*" Debra (mother, aged 43): "*Yeah, so… um… we don't, as much as I'd love it on a weeknight to come home and have takeaway once a week – we don't. It would probably be more like once a month I guess.*" The daily dessert provided by most grandmothers was not a practice carried on by the mothers in the parent generation who seemed to be more health-conscious and avoided sweetened foods or beverages (in one case, no food or drink besides water was allowed after the evening meal). Daniel (father, aged 45, who prepared most family meals) pointed out, not only his wife's (mother, aged 41) involvement, but also her healthy dietary orientation.

Facilitator: So, who would talk, be talking the most about food?
Daniel: That's you [mother] isn't it? In our family, yeah.
Sarah: Yeah, yeah.
Daniel: Because you're the one.
Sarah: Yeah, yeah.
Daniel: You do the most reading about diet and health.
Sarah: Health-conscious I think.
Daniel: Yeah you are, aren't you?

Alternatives to unhealthy food options offered by mothers were, for example, fruit for snacks and this, in turn, seemed to influence the child generation, some of whom consequently thought about, and were mindful of, healthy food choices when making their own decisions. Anne (daughter, aged 13) commented: "*Um Grandma and Mum like always remind us to eat healthy and if we go for snacks sometimes I will just have a piece of fruit instead*." A health-conscious attitude was more apparent in female family members, and was transmitted intergenerationally from grandmother to mother, then from mother to daughter (e.g., Christine, grandmother, aged 73: "*Well you just had to, didn't you?*"[Eat vegetables], Barbara, grandmother, aged 71: "*Finish what's on your plate*", Lauren, mother, aged 46: "*Because he's usually with fruit… yes, he would have an apple in his lunchbox and I'd say, 'Well, just eat your apple before you get anything else…*", Anne, daughter, aged 13: "…*if we go for snacks sometimes I will just have a piece of fruit instead*".

Mother's Influence on Others

Broadening tastes and raising health-consciousness by introducing new foods. Mothers in the present study influenced grandparents by introducing them to new multicultural foods and spicier tastes (e.g., chilli), or some of the "new" foods available including the 'so called' "superfoods" (e.g., quinoa, chia seeds, acai berries). These foods were offered at family meals in the parental home or provided pre-cooked for later use and delivered to the grandparents' home. Sandra (grandmother, aged 70), emphasised how her daughter Kylie (mother, aged 45), broadened her otherwise plain dietary experiences with foods that were new to her.

Sandra:	Oh yes, it's incredible what [foods] I've had (laughs)…
Kylie:	(laughs).
Sandra:	…when I've come here [mothers home] (laughs).
Facilitator:	You never would have normally made yourself?
Sandra:	No, I don't think so. No.

Sometimes the new foods were liked and appreciated and at other times they were disliked however, the mother, as though introducing a new food to a child, would cautiously serve a small amount for grandparents to try – with an alternative "plain" meal if it was not to their liking.

Mothers were shown to influence grandparents with their own health-consciousness by encouraging new, healthier foods, and discouraging unhealthy eating behaviours, such as the consumption of excess sugar or salt. Debra (mother, aged 43) discouraged the grandfather's salt consumption: "*Yeah, we always get a – we're always getting on to Dad* [grandfather] *about the salt (laughs).*"

The Influence on Fathers

As reported earlier in the sections detailing grandparent's and mother's influences on others, grandfathers influenced fathers intergenerationally by role modeling self-sufficiency food practices, and mothers influenced fathers intragenerationally with health-conscious food attitudes.

Father's Influence on Others

Father's relaxed food attitudes model less healthy food preferences. In comparison to mothers, fathers in the parent generation were more likely to treat themselves, and the children, to less healthful foods. Andrew (son, 17) mentions a television cooking program: "*Oh yeah, the cake bake-off*." Facilitator: "*Yeah bake-off, which is basically, just cakes*." Sally (mother, aged 52, agrees): "*Yep*" and John (father, aged 51), admits: "*That's my weakness*." Simon (father, aged 48) said if he cooks, he makes cakes: "*Yeah, I'll make cakes now and then, now and then. Not a lot.*" In another family, David (step-father, aged 35) responded to the grandmother's health-conscious comment about reducing salt in her diet with: *"I like salt."* All: (*laughter*) Facilitator: *"But has that stayed the same? It hasn't changed over time?"* David: *"No, I have always liked salt."* Facilitator: *"hmm,"* and most sons more so than daughters showed a preference for these food types themselves as Joshua (son in the same family, aged 15) shows: "*Well I eat a lot of really like snacky* [sic] *foods for recess at school – little biscuits and things like that.*" This suggests fathers modeling to sons and influencing them with their own preferences for 'unhealthy' food types (Bandura, 1977b). Men admitted consuming treats, fast, or snack foods with the family (or in the mothers absence) which was occasionally met with the mother's disapproval. Daniel (father, aged 45*)* mentions the mother's attitude towards snacks: "*…and it'll be quite often we will be having a snack or something and*

you'll [Sarah, mother, aged 41] *say, 'oh, oh, we shouldn't be eating, you know, this kind of thing in the evening we'll get into bad habits,' so.*" This demonstrates the difference between mothers and fathers level of health-conscious beliefs or attitudes and that mothers influence fathers *intragenerationally* by attempting to change their behaviour (see Figure 6).

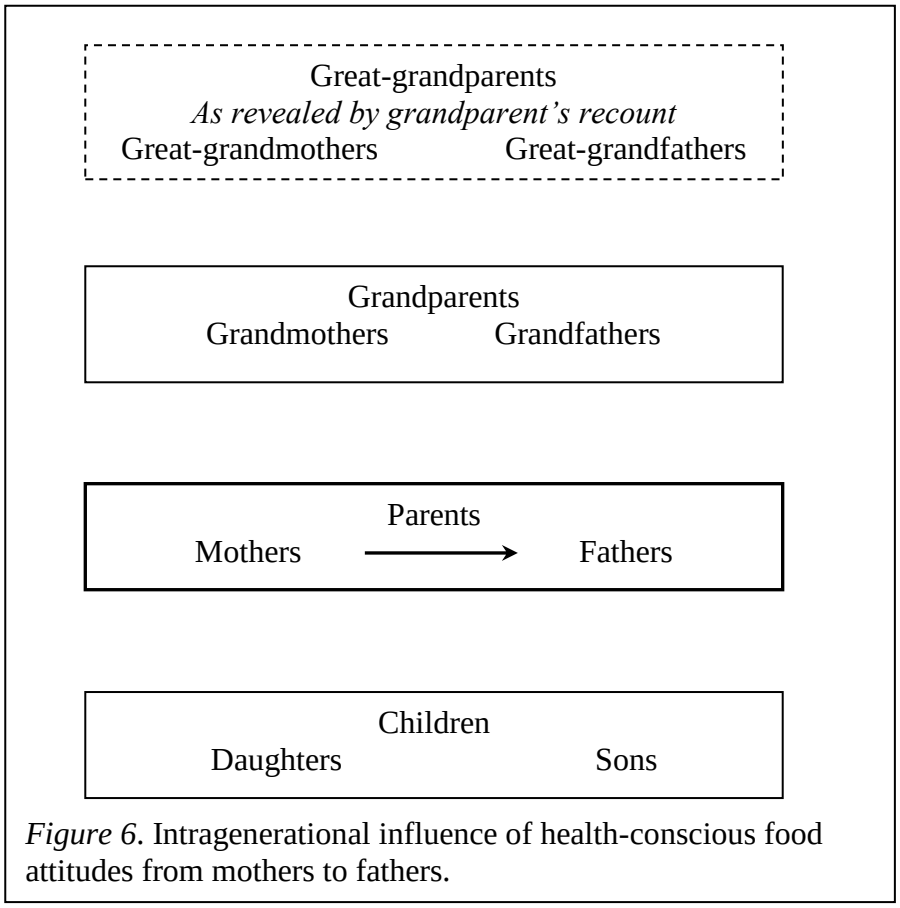

Figure 6. Intragenerational influence of health-conscious food attitudes from mothers to fathers.

Fathers share with children food preferences lacking variety. Consuming a variety of different foods from the available food groups are encouraged in the Australian recommendations for a healthy balanced diet (NHMRC, 2013a). Fathers, more so than other adult family members, expressed their preference for foods which lacked day to day variety, a theme that was also apparent amongst the child generation. For example, Isabella (daughter, aged 8): "*But all the time I usually have, Vegemite sandwiches*"; also Nicole (mother, aged 44) explains: "*Yeah, she's* [Courtney, daughter, aged 18] *to that point where we just can't, all she wants is the same thing over and over so we just make whatever we're gonna* [sic] *make now, and hope she eats it*."; and Sandra (grandmother, aged 70) said: "*Well see that's what children seem to be like*," Kylie (mother, aged

45) replied: "*Oh, they're* [the children] *quite happy with pizza every night*." Lisa (mother, aged 43) also said about her daughter (Katie, aged 11): "*You're pretty… well she's pretty routine with her lunch box, though aren't you? You like to stick to the same thing, and then suddenly after six months she'll go off it*." Lauren (mother, aged 46) also mentioned her son (Thomas, aged 9) and noticed the difference with her daughter (Lily, aged 15): "*Um, normally in the mornings when I'm making their lunch I'll ask them what they want and quite – yeah… Thomas usually has the same thing every day for lunch. But Lily, she takes different things*." Similar to fathers and most of the children in the present study, some grandparents who lived alone also expressed a lack of interest in cooking for themselves or in food variety. For example, Christine (grandmother, aged 73) commented "*Yeah but I would make, yeah I do good meals for the family yeah, when we eat* [together] *and, but I love tomatoes and eggs; I could just eat tomatoes and eggs."* Although the mothers in the present study, most likely coming from a health-conscious perspective, considered food variety as important, most grandparents living alone, fathers and children still preferred less day-to-day variety in their food choices. This could reflect differences in health-conscious food attitudes between mothers and fathers and between mothers and children. Health-consciousness was a prominent theme amongst female family members and, as a result of these findings, will be investigated further in Study 2.

The Influence of the Child Generation

The child generation in the present study had less influence than the older generations. In all probability, this may reflect non-participation in the food purchasing decisions and occasions. Intragenerational influences between siblings were not distinct in the present study, however, new themes did emerge from the data that demonstrated influence pathways between child-parent and child-grandparent generations (see Figure 7). The section to follow presents parental influences on children, then child influences on parents are described.

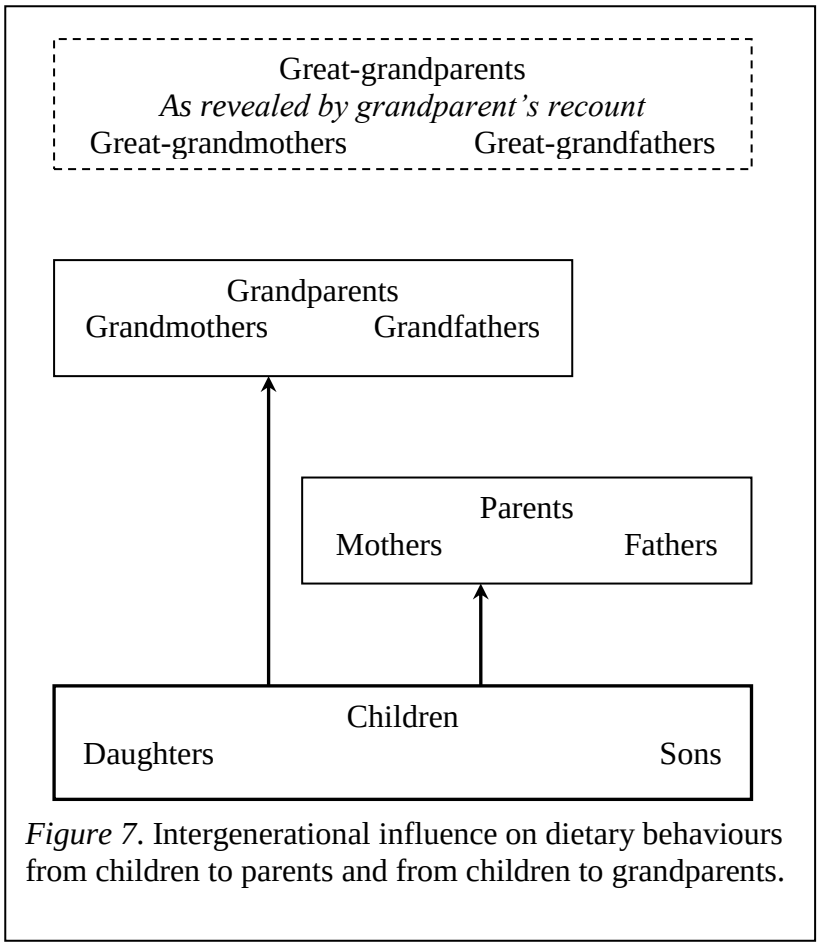

Figure 7. Intergenerational influence on dietary behaviours from children to parents and from children to grandparents.

The influence on children by parents. Child and parent influence was mainly limited to one direction for food choice (parent to child). This was because parents (predominantly the mother) decided for children what their day-to-day food choices would be, especially the evening meal. Most mothers catered to the tastes of the family as a whole by providing one evening meal that suited everyone, which at times, excluded their own food preferences. Some parents were the exception and catered to each individual child's food preferences when preparing the evening meal, by providing variations of the same meal which met the preferences of each person. Alternatively, entirely different meals were prepared for each person on the same eating occasion. This most usually occurred where a family member had special dietary requirements (e.g., gluten free, weight loss diet, sports nutrition). A small proportion of older children occasionally opted out of the family's evening meal and made their own alternative. Yet most children, even as adults living at home, ate what their parents provided.

The parents introduced their children to a wider variety of cuisines available from

multicultural influences and incorporated international foods into meals eaten at home or when dining out, or for takeaway meals. For example, mother, Nicole (aged 44): "*And if we go out to tea* [evening meal] *and we like something and David* [stepfather] *might think, 'Oh I am going to try and make that*.'"; Sarah (mother, aged 41): "*We tend to eat a lot of er, curries, Thai food, Indian food* [pause]*... and rice*"). Lisa (mother, aged 43) took pride in her children eating multicultural food; "*Italian, yeah. They're pretty good. Stephanie's 15, Katie's 11 but I think for their age compared to what I was like as an 11-year-old, they've got quite a broad taste range which is good. Because they will tolerate… Stephanie tolerates a really hot curry. Katie is getting there."* Kylie (mother, aged 45) similarly exposed her children to wider multicultural tastes: "*No. But I am trying to encourage them with like, things like massaman. You know massaman beef curry and that – it's very mild. So I'm trying to encourage them to explore different tastes, but… very slowly*." Finally, Isabella (aged 8) exclaimed: "*I like curries!*"

Some parents expressed disapproval of grandparents' food preferences from earlier times, which were favoured by some grandchildren today. This may have been because parents now considered these food types "unhealthy". For example, Sally (mother, aged 52) explains: "*In those days. But it was like, it was always – I mean my mum would make – you would have your hot dessert.*" Facilitator: "*Like a pudding or something?*" Sally: "*Like puddings, we would have that every night.*" Neil (son, aged 18): "*Oh!*" Sally: "*Or we'd have ice cream, or those sort of things, which when I had the children, we certainly didn't carry that on. We weren't having dessert every night of the week*." This shows a greater orientation towards health-conscious food attitudes than the previous generation had. The generational difference may be partly due to the contrasting food scarcity of yesteryear and food abundance that exists in Australia at the present time. Overall, a healthy nutrition focus was noted more so in the food attitudes of daughters than sons; some daughters refused to eat takeaway at all when the rest of the family did, whereas some sons were the only ones in the family that ate fast food at all on "takeaway night", and some daughters showed mindfulness when selecting food for themselves. Therefore, health-conscious food attitudes of

grandmothers, and especially mothers in the present study showed a trend towards an intergenerational transmission through the females in each generation.

The influence of children on parents. As would be expected, with advancing age children made more of their own decisions about food. This was demonstrated most frequently in the context of meals or snacks that could be prepared by themselves, for example, Courtney (daughter, aged 18) made requests of her parents to purchase easy to prepare meals at home: "*Yeah, or things like the packet food you just put it in the microwave and cook it.*" Some adult children still living at home made independent food choices; often fast, convenience foods purchased beyond the home environment. For example, in an otherwise health-conscious family, John (father, aged 51) said of his son aged 18: "*He's a takeaway addict.*"

Children make their preferences known by asking mother. Most parents in this study considered children's food preferences, for example, when they prepared packed lunches for school, often on a daily basis. Catherine (mother, aged 42) talked about this topic with her children: "*Yeah. Oh, we talk about what goes in your lunch box.*" Anne (daughter, aged 13): "*Yep, what we are going to have every day.*" Catherine: "*That's your choice though*." Leigh (son, aged 9): "*Bananas every day*." Most children chose their own breakfast and even the youngest in the study could prepare a simple bowl of their preferred cereal themselves. Child to parent influences beyond breakfast and lunch choices were demonstrated by children who made specific requests of parents for certain foods to be purchased. Nicole (mother, aged 41) commented: "*Yeah, yep. And then Courtney* (daughter, aged 18) *might say, put in a request or something, or Josh* (son, aged 15) *might say…"* Joshua: *"Influence."* Nicole: *"Or let you know, for shopping."* Parents made the final food decisions because the boundaries of acceptable household foods were ultimately the parent's decision to make whilst out shopping for food.

Children helping and cooking in the kitchen. Some older children enjoyed cooking food at home, in the most part these consisted of sweet baked foods (similar to fathers' cooking preferences) rather than main meals that one would expect to have for an evening meal. One

teenager commented "*Yeah. Because I don't… I make sweets and cupcakes and cakes and cookies and slices…"* (Lily, daughter, aged 15). Some mothers expressed a desire for their children's help in meal preparation on a regular basis to ease the weekly cooking burden; conversely others found it was easier not to have their children help. Amelia (daughter, aged 13): "*I ask to help but mum doesn't like me helping*," Her two sisters agreed and Debra (mother, aged 43) conceded: "*So often it's just easier…*" Facilitator: "*Yeah."* Debra: "*It's borne out of an easy life… wanting one.*" The children in the present study preferred to cook for enjoyment and food pleasure rather than out of necessity to provide an evening meal for the family (e.g., as shown in the example with Lily previously who enjoyed baking). Adults were predominantly responsible for family food provision and household food availability; therefore, stronger influences from child to parent were evidenced by children regularly making their preferences known for breakfast or lunch. Most children in the present study also prepared their own breakfasts and chose snacks from within the boundaries of the available food options provided by parents. The following presents the reciprocal influences between grandparents and children that skip the parent generation altogether (see Figure 8).

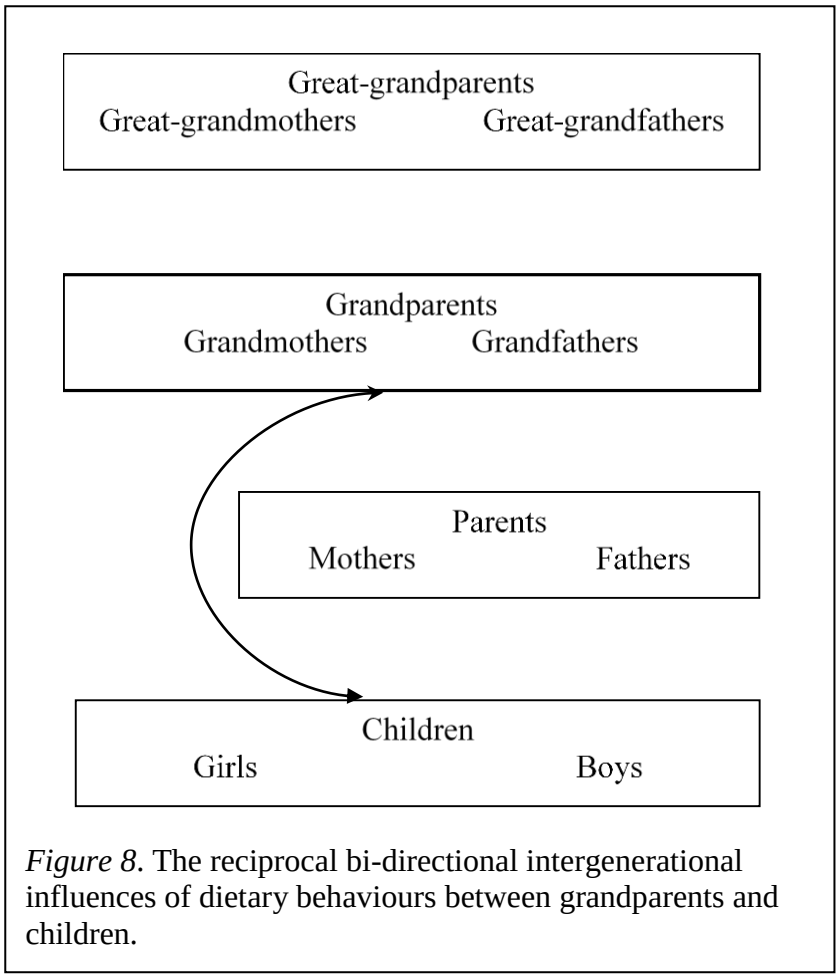

Figure 8. The reciprocal bi-directional intergenerational influences of dietary behaviours between grandparents and children.

The influence on children by grandparents. *Grandparents indulged grandchildren in unhealthy treats.* Although some grandparents seemed to be health-conscious, many family members also discussed how grandparents indulged grandchildren in unhealthy food and beverages that were not normally eaten in the parental home. Neil (son, aged 18): "*Oh we had supper. That was – we'd never heard of supper in our lives…*" Andrew (son, aged 17): "*Yeah.*" Facilitator: *(laughs)* Neil: "*…until grandpa came over and gave us finger buns and…*" It was common in the present study for grandparents to indulge grandchildren with treats that they did not even consume themselves. Peter (grandfather, aged 76) openly talked about his granddaughter (Isabella, aged 8): *"We won't tell you what she eats when she comes to our house!"* Sarah (mother, aged 41): *"I know it's shocking."* Jennifer (grandmother, age 73): *"She has a different diet sometimes! (laughs) or snacks, don't you? (laughs).*" Most grandparents purchased foods and beverages, that they would

not normally have had themselves, specifically for their grandchildren. Andrew (son, 17*)* and Neil (son, 18) remembered when they went to their grandfather's house to stay once a week as younger children. Neil said: "*We used to eat unhealthy whenever we went to grandpas.*" and Andrew said: "*Oh yeah, we sure did. Hmm. I don't think there was anything healthy there."* Their mother Sally (aged, 52) agreed at this point and the sons continued on to say, Neil: "*And special occasion he'd get us Tim Tams* [chocolate biscuits]*and ice… he may have – he bought us a milkshake maker so we made milkshakes. What did you used to have?*" Andrew: "*I don't know. He used to get me a big bag of chips; he used to get you a whole packet of Tim Tams."* Facilitator: "*Oh, separate things?"* Andrew: "*Yeah. And then – every week – but then one time when we came over he still bought a packet of chips, a packet of Tim Tams and then he bought fish and chips for tea* [evening meal] *and so it's like, there was no break from eating.*"

Then the anecdote ended with the sons talking about all of the chocolate they were given during the Christmas holiday period. The facilitator probed for clarification: *"So the grandparents didn't eat like that all the time though did they? So, that was just for you kids?*" Sally (mother, aged 52): "*Absolutely.*" Neil (son, aged 18): "*Grandpa never touched it.*" Facilitator: "*He didn't eat it himself?*" Neil: "*No.*" These findings highlight the potential implications on dietary behaviour for children in the regular care of grandparents, and the availability of unhealthy food options not typically offered by parents.

The influence of children on grandparents. *Old-fashioned food and new food.*

Considering over one third of Australian grandchildren are regularly in the part-time care of grandparents (ABS, 2014b), the potential for direct bidirectional influence between these generations is likely to be enhanced. In the present study, grandparents caring for children before and after school had requests made of them to prepare family meals according to the children's likes and dislikes. For example, before school Joshua (son, aged 15) requested his favourite meal when his grandmother Christine (aged 73) collected him in the morning, and she subsequently prepared it for the whole family's evening meal: "*Yeah, well I would come over here* [to the family home] *to*

drop him off at school or something and I would say [to Joshua]*, 'What, what shall we have tonight? What do you want?' yeah. It's usually sausages or something, yeah."* Grandmothers who regularly prepared the family's evening meal appeared to be influenced by children who did not particularly like the grandparent generation's typical diet. Judith (grandmother, aged 69) expressed her concern that the children did not like her cooking: "*And I said um, well, you know, the kids don't like all the things I cook, like I, we had a grill tonight with, and then we had um. Roast potato and…. What was the other?*" and Catherine (mother, aged 42) said: "*Yeah. My kids don't like a lot of the same sort of things that Mum will eat, because I don't cook a lot of meat and three veg, like hardly ever*." The discussion went on and in the end Judith conceded that she prepared alternative meals that the children liked: "*Or we will have tacos, or whatever they're called! Or things like that, that they'll eat.*" Since food preferences that develop in childhood persist into adulthood, well-meaning grandparents who regularly indulge children in unhealthy foods have the potential to unintentionally influence children's future food choices. Hence, shaping children's long-term food preferences could subsequently impact on their health adversely (Contento et al., 2006; Savage et al., 2007).

Another interesting conjoin across generations was that the children liked some of the grandparents' foods that were typical in the grandparent's diet years before. Stephanie (daughter, aged, 15) spoke with her grandmother Barbara (aged, 71): *"But I used to do these baked ham and chips. You loved my chips, didn't you?"* Stephanie: *"Yeah, she used to cook the chips. Yeah. Nanna's chips, yeah."* Although the parent generation often expressed disapproval of the grandparent's food choices, in some instances it seemed that these foods were eaten on such a regular basis when parents were children that they no longer cared to eat those foods anymore. Peter (grandfather, aged 76) began with: (cough) "*Well, <u>we're</u> not vegetarian.*" All: (*laughter*), Jennifer (grandmother, aged 73): "*We eat meat and fish and…*" Sarah: "*They are the reason <u>why</u> I am vegetarian! (laughs).* These results are now interpreted further in the discussion to follow together with any conclusions drawn in addition to showing directions for future research.

Discussion

The focus of Study 1 was to examine the intra- and inter-generational transmission of dietary behaviours and food choices within three generation Australian families. Taken together, the findings from the interviews bring new insights, having gone beyond the results of uni-directional (top-down) research by incorporating bi-directional influences across three generations, including influences between children and grandparents that bypassed the parent generation completely. In previous literature, mothers have been shown to predominantly influence food decisions in their households (e.g., Beydoun & Wang, 2009; Green et al., 2003; Green et al., 2009; Prelip et al., 2012; Wroten et al., 2012). Less was known about the influence that grandmothers have in their households, or whether intergenerational influences extend between grandparents and the parent-child households. In the present study, mothers and grandmothers in their respective households facilitated other family members' healthful food consumption. Mothers and grandmothers expressed the greatest health-conscious food attitudes in their respective households. However mothers, more so than grandmothers who placed fewer restrictions on food consumption, were shown to promote a positive attitude toward healthy food consumption within their own households and beyond it to include that of the grandparents as well. Mothers concern for the dietary health of others was noted to include their husbands, their children and the grandparents living in separate households. Not only did the mother show the greatest health-consciousness in dietary behaviours, the concern for others was also the greatest and extended the furthest. Thus, the first research aim of this study has been achieved by identifying the mother as the key individual within multigenerational families to disseminate health information more broadly within the family network. This information will enable future family-based disease prevention initiatives to successfully disseminate health messages by targeting the mother.

The second aim was to uncover the behaviours that contribute to mechanisms of influence within families and the direction of influence between family members. The mechanism of mother's influence on the child was promulgated by encouraging healthful eating, modeling healthy choices,

and creating rules about meals or snacks that restricted the consumption of unhealthy food. In contrast, fathers were more likely than mothers to show preferences for meals without vegetables, or to "indulge" children and themselves in unhealthy foods, whereas grandparents expressed being more lenient with 'treat' foods for their grandchildren, whilst not consuming these unhealthy foods themselves. The discussion now turns to the mechanisms of influence by each generation (oldest to youngest) starting with the grandparent generation.

Influences during the Grandparent Era

Family interviews in the present study aimed to garner information from three generations, however during the interviews, grandparents often reflected on their own childhoods and rich data was unexpectedly obtained regarding the child feeding practices of the great-grandparent generation also. From grandparents' childhood memories great-grandparents were reported to have had non-negotiable food rules where adult preferences were considered first and children had little choice but to eat what was provided for them. Great-grandmothers were said to have had the role of primary caregivers at home who made the food decisions. Grandparents recounted that as children they were exposed to different environmental factors such as food scarcity, availability of only plain foods, and limited or no exposure to multicultural foods (Banwell et al., 2012). This may explain pervading grandparent plain food preferences over time in this Anglo-Australian cohort. The plain meals that the grandparent generation grew up with were still enjoyed and preferred by them today. This suggests that present day food preferences were shaped by the grandparent's own parent's socialisation processes and family norms about food (Birch et al., 2007; Contento et al., 2006).

Grandparent preferences for plain and simple food. Although preferences were expressed for plain food, changes in some grandparents' diet occurred over time from healthier food preparation practices (such as no longer deep-frying chips), or through the introduction of 'new' foods by the parent and child generations. Interestingly, the parent generation tended to introduce new foods that were considered healthy (e.g., quinoa) and encouraged healthier food consumption (e.g., reducing salt intake), while grandchildren introduced grandparents to a wider variety of foods

that grandchildren typically preferred (e.g., tacos). In Australia, the literature informs us that Anglo-Australian children of the Baby Boomer generation (i.e., those born between the years 1946 – 1965 in Australia; ABS, 2003b) were typically provided with the same evening meals each week (e.g., fish every Friday, or roast every Sunday), and meals frequently consisted of meat and three vegetables (e.g., Banwell et al., 2012). Today's generational differences show changes over time that have been influenced by broader Australian environmental factors such as immigration and diverse multicultural food availability; advances in food preparation technology; a rise in health-conscious food choices, healthier food preparation methods and cooking habits, where there has also been a rise in and greater access to fast food (Block et al., 2004; Burns et al., 2015). The strength of family influence over environmental influences were shown by grandparents who accepted changes in their diet influenced by family members, however, had declined the consumption of fast food that became more available to them in their environment during their later adult years.

Gender-based distribution of responsibility for food and health. Grandmothers reported having the sole responsibility for food decision-making in their household, which was endorsed by other family members, and over time they had gained an implicit understanding of grandfathers' food preferences. Thus, grandmothers incorporated this knowledge into their regular household meal planning, shopping, and food preparation. Grandmothers expressed greater concern for healthy nutrition than grandfathers, which may have been due to grandmothers' primary responsibility for household food choice, a task that grandfathers had no need to concern themselves with. Therefore, since grandfathers were not as directly responsible for the nutritional value of their children's food, this may explain why males were less health-focused around food decisions. Grandparent's gender role differences in household food choice responsibility may account for gender differences in health-consciousness and subsequent food attitudes (i.e., grandmothers' healthier food preferences to grandfathers') that had developed over time.

Whilst grandmothers taught their children about the importance of health and nutrition,

including why it was important to eat fruit and vegetables, some grandfathers had vegetable gardens and fruit trees to provide fresh foods for the family to eat, or they kept chickens for meat and eggs. Grandfathers modeled similar "self-sufficiency" behaviours with home produce that were later adopted by some fathers. The grandparents' food preferences were introduced to the parent generation as they modeled their own food practices and eating behaviours. Grandfathers seemed to prefer sweet or unhealthy foods more so than grandmothers. Some grandmothers said they were able to eat a preferred and somewhat healthier diet after their husbands had passed away because they no longer catered to the grandfather's tastes. For a summary of grandmother's and grandfather's bi-directional mechanisms of influence see Figures 9 and 10.

Primary role of grandmothers	The people grandmother was influenced by (mechanism)
Food selection Food purchase Meal planning Food preparation Household diet	*Maternal great-grandmother* - her mother role-modeled being the dominant food decision-maker, taught cooking skills, enforced food rules around healthy eating, and controlled the family diet that consisted of plain food types. *The grandfather*'s preferences - her husband made his likes and dislikes known, her implicit understanding developed over time. *The mother*'s health concern - her daughter introduced and provided 'new healthy foods' discouraged unhealthy diet, broadened exposure to multicultural food. *The children*'s preferences - her grandchildren asked for specific foods; and refused food that they did not like. The people grandmother influenced (mechanism) *The grandfather* - her health-consciousness in her household affected his food provision. *The mother* - encouraged healthy food; role-modeled dominant food decision-maker, taught cooking skills, enforced food rules around healthy eating, provided plain foods. *The children*'s preferences - indulged with snacks and unhealthy treats. *The whole family* - part-time provision of family meals adjusted according to children's preferences.

Figure 9. The primary roles of grandmothers are shown alongside a summary of the intergenerational and intragenerational influence pathways between grandmothers and other family members.

Primary role of grandfathers	The people that grandfather was influenced by (mechanism) *Paternal great-grandfather* - his father role-modeled food self-sufficiency, thrift, discouraged food wastage. *The grandmother*'s preferences - plain food choice, her primary household food decision making, family health and nutrition focus and her food attitudes. *The children*'s preferences - asked grandmother to cook specific family meals; food refusal affecting grandmother's household food choices for all. The people grandfather influenced (mechanism) *The father* - he role-modeled food self-sufficiency, thrift and waste minimisation, preferred unhealthy food. *The children*'s preferences - indulgence with snacks and unhealthy treats.
Food economy	
Food self-sufficiency	
Food waste minimisation	

Figure 10. The primary roles of grandfathers are shown alongside a summary of the intergenerational and intragenerational influence pathways between grandfathers and other family members.

Health-consciousness and behaviour. Based on anecdotal evidence it was suspected that well-meaning grandparents enjoyed indulging their grandchildren in favored foods, however, empirical research on the influence of Australian grandparents on extended family food choice is limited. Some evidence from a Chinese cultural context supports the perception that grandparents indulge grandchildren (Jingxiong et al., 2007; Kicklighter et al., 2007). However, the reasons that grandparents provided differed (e.g., grandparents own childhood poverty, hopes that feeding grandchildren large serves would increase height), therefore the study may not be entirely applicable in the Anglo-Australian context. Although poverty and food scarcity may have been similar childhood influences on the current grandparent generation worldwide, influences on the Chinese grandparent generation are likely to differ from Anglo-Australians due to the availability of different food in China, and different cultural beliefs surrounding food. Nevertheless, the present study suggested that grandparents did indulge grandchildren in unhealthy foods in the form of snacks and treats. Paradoxically, grandmothers at the same time impressed upon the moderator the importance of healthy eating when raising their own children. In addition, grandfathers who endorsed indulgent treats for grandchildren also paradoxically provided fresh produce that

supplemented the household's healthy diet. Therefore, grandparents' priorities shifted over time from healthy food attitudes when raising their own children, to the enjoyment experienced when providing grandchildren with food treats in the present-day. Although grandmothers in this study appeared to be health-conscious to a certain degree, grandparents did not always act in accordance with the mothers' healthy food preferences when caring for grandchildren. It could be argued that grandmothers had healthy food attitudes, however to a lesser degree than mothers, or that mother's food attitudes become more relaxed with age due to different life-stage responsibilities.

Influences during the Parent Era

Since grandparents had provided plain food for their own children it was not until the parent generation had grown up and left home that they experimented with more adventurous and multicultural foods themselves (Banwell et al., 2012). This was one noteworthy generational difference between grandparents and parents evident in the present study. Mothers influenced the grandparent and child generations with new multicultural food preferences by introducing them to unfamiliar foods tailored to their individual taste tolerance.

Mothers the key healthy influencers in their household. Mothers in the parent generation, like their own mothers, described how they learnt to cook in class at school and helped their mothers at home with food preparation and cooking when they were children. Therefore, learning about meal preparation took place at school and this was typically reinforced at home (Maccoby, 1992; Medin, 1992). Female role-modeling at that time transmitted primary household food responsibility, whereas males of that era did not learn cooking at school and were less likely to have participated in cooking at home. As parents, mothers in the present study were aware of healthy food practices and provided examples of this. Such as, trimming the fat off meat, reducing salt in cooking, consuming less red meat, eating a wider variety of healthful foods, especially vegetables, mothers were more likely to be vegetarian, and cooked using healthier methods than their parents did (e.g., used monosaturated oils instead of saturated fats). Most of those healthy eating practices described by mothers were consistent with current NHMRC healthy dietary

recommendations (NHMRC, 2013a).

Overall, women in the present study had the principal influence on food choice, meal preparation and dietary-related communication. This is in line with previous literature that has demonstrated that mothers predominantly influence food decisions in their households (Beydoun & Wang, 2009; Green et al., 2003; Green et al., 2009; Prelip et al., 2012; Wroten et al., 2012). These findings are also consistent with observations from earlier studies in which women have been shown as more likely to follow healthy eating recommendations (Worsley & Scott, 2000), prefer the tastes of healthy foods (Turrell, 1997), and appear to make greater contributions to the quality of their family's diets (Schafer, Schafer, Dunbar, & Keith, 1999).

Mother's influential mechanisms on the dietary behaviours of others. With the increased availability of convenience foods in the parent generation, mothers restricted the consumption of unhealthy foods such as snack foods, sugar-sweetened beverages, fruit juice, and takeaway foods high in sugar, saturated fat, or salt. One of the mechanisms mothers used was to restrict less nutritious foods and beverages to one night of the week or month, by having "*chocolate night on Saturdays*" or "*takeaway night*." This was to teach children that unhealthy but favored foods were for occasional rather than for regular daily consumption. Some mothers said that avoiding unhealthy food altogether would not teach children how to eat in moderation and voiced concern that avoidance may cause unspecified dietary issues.

Australian dietary guidelines also recommend limited intake of a range of energy dense, nutrient poor foods and beverages (NHMRC, 2013c), showing that mothers health-consciousness was likely to be in the forefront of their minds when shaping the food consumption of offspring. Mothers in the present study acted in accordance with an authoritative parenting style (that combines authority and democracy, as shown in the examples above (Baumrind, 1973), which is said to foster healthy eating habits in children (Sleddens et al., 2011; Vereecken et al., 2004). Therefore, consistent with previous research in Europe, an authoritative parenting style links with healthier dietary habits in Australia also.

Healthy food attitude transmission across generations. Mothers, like the grandmothers before them, were shown to convey their own food attitudes by teaching children about the link between health and nutrition, and about illness prevention through diet and other healthy behaviours (e.g., the benefits of not smoking). These behaviours demonstrate food attitudes consistent with a belief that there is a link between diet and health (Rozin et al., 2003; Sharp et al., 2013). Beliefs in the links between diet and consequent disease can be understood with the Health Belief Model (HBM) and can act as motivating factors in fostering positive health behaviours. As was described in more detail in Chapter 1, the HBM suggests that in order to avoid chronic disease, one must engage in healthy behaviours such as adhering to a nutritious diet and avoid the consumption of unhealthy foods (e.g., Rosenstock, 1974).

Mothers in the present study facilitated healthy dietary beliefs by teaching children about the benefits of whole foods over processed foods, and restricted the consumption of nutrient-poor foods by keeping predominantly nutritious foods at home, or by encouraging fruit for snacks. Some mothers did not take children to the supermarket to restrict the child's influence on food purchases. One family did not allow their children to watch commercial television to avoid exposure to fast food advertising. Preventing access to so called "junk" food at home, and preventing children from influencing parents with their own requests whilst out shopping for food were some of the mechanisms parents indicated that they used to control healthy food consumption. These parental actions may show the strength of convictions in providing a healthy diet for their children, because preventing exposure to unhealthy food was emphasized indicating that it was of great importance to them. For a summary of mothers' mechanisms of influence see Figure 11.

Primary role of mothers	The people mother was influenced by (mechanism) *Maternal grandmother* - her mother role-modeled being the dominant family food decision maker. *Mother*'s own preferences – for meal variety, multicultural food, health-conscious food attitudes. *The father*'s preferences – he made his likes and dislikes known, her implicit understanding over time. *The children*'s preferences – asked for specific foods; food refusal; requested baking ingredients; prepared own foods with increasing age. The people mother influenced (mechanism) *Household* – encouraged healthy food; health conscious food attitudes, discontinued daily dessert habit. *The grandparents* - introduced new, more flavoursome and multicultural foods, encouraged healthy and discouraged unhealthy food consumption; delivered pre-prepared meals to household. *The father* – maintained her primary family food responsibility for meal provision, if father primary then she monitored, reminded, discouraged unhealthy, and encouraged healthy food preparation. *The children* – she modeled, reinforced, controlled, restricted, used conditional treats, gave healthy snack alternatives, provided for nutritional needs as perceived by her, implicit understanding of preferences over time, introduced multicultural food.
Food selection	
Food purchase	
Meal planning	
Food preparation	
Household diet	

Figure 11. The primary roles of mothers are shown alongside a summary of the intergenerational and intragenerational influence pathways between mothers and other family members.

Gender-based roles and food responsibility. Previous Food Life behaviour and attitude research across four western countries has demonstrated some gender differences. In relation to attitudes that diet affects health or disease, research shows women scored higher than men, however, associations between these food attitudes and various family roles have not been separately explored (Rozin et al., 2003; Sharp et al., 2013). In the present study, an intergenerational transmission of food attitudes that diet affects health or disease was apparent through sex role-modeling. Although previous research has not examined families and Food Life behaviour and attitudes, it could be that specific family roles provide possible underlying explanations for the gender differences found in previous Food Life behaviour and attitude research. Modeling is said to convey sex-role behaviours only when children have reached the age of gender identity attainment (i.e., over and above four to six years of age) (Bussey & Bandura, 1984). The

results of the present study suggest mothers sex role-modeled primary food responsibility and food attitudes to their daughters, and to an extent, grandmothers are likely to have similarly modeled healthful dietary practices and attitudes to the mother generation years beforehand (possibly the great-grandmothers to grandmothers as well). Although mothers model behaviours to children of both sexes, it may be that caring for others in the family is a female role stereotype that is more likely to be adopted by daughters than sons (Perry & Pauletti, 2011). Hence, evidence supporting sex role-modeling reinforces the notion that dietary behaviours are transmitted predominantly through the person acting in the mother role.

Fathers show lenience towards healthy food rules. Fathers in the present study, on the other hand, were more likely to be relaxed about family food rules and to indulge themselves and their children in unhealthy meals or snacks. Therefore fathers, evidently having less responsibility for family food decisions, unconsciously modeled male sex-roles to their offspring. Fathers dietary behaviours would therefore be expected to be adopted by sons more so than by daughters (Bradford Wilcox & Kovner Kline, 2013), as girls are more likely to adopt mothers' behaviours (Perry & Pauletti, 2011). Some fathers demonstrated their contribution to healthy eating by providing the household with home grown fresh produce, which could imply a stereotypical male role of father as 'provider' for the family. Nevertheless, fathers' more relaxed food attitudes, in addition to that of grandparents towards children, could have implications for the developing food attitudes of any children who are in their regular care.

Mothers shared with fathers some of the responsibility for food decision-making and cooking in their households, whereas grandfathers' food preferences over time were internalised by grandmothers who had incorporated this knowledge into their sole responsibility for household meal planning, shopping, and food preparation. Grandfathers' absence of responsibility for household food decision-making and cooking was a key difference from fathers in the parent generation who shared this responsibility with mothers. For a summary of fathers' mechanisms of influence see Figure 12.

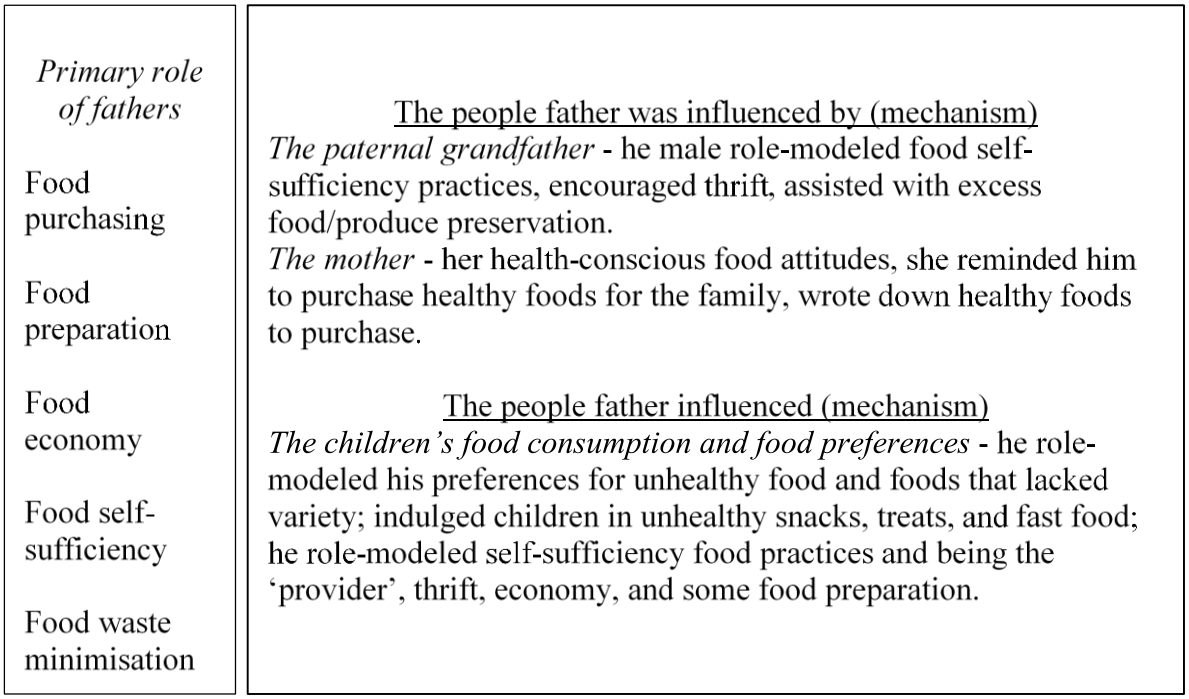

Figure 12. The primary roles of fathers are shown alongside a summary of the intergenerational and intragenerational influence pathways between fathers and other family members.

Children's Upward Influential Mechanisms towards Parents and Grandparents

Gender differences were noted to continue with the child generation, for example, daughters were shown to have a greater interest in healthful eating more so than sons. Sons were more likely to eat fast food and sometimes sons were the only ones in the family who indulged in fast food, even on "takeaway night." With a high proportion of both parents working in this study's generation of children (ABS, 2014b), children in the present study influenced the food choices of grandparents by requesting certain foods whilst in their care after school. This introduced new foods to the grandparent generation as they prepared foods for grandchildren that they had never previously considered. These requests favoured the child's own food preferences and were not always healthy. Children established that they also influenced food decisions in the parental home by asking for their preferred food types and having these considered then purchased for them. Some children in the present study influenced family food decisions by refusing to eat certain foods, by choosing to be vegetarian, or having other health concerns considered (e.g., sports diet, gluten free). For a summary of children's bi-directional mechanisms of influence see Figure 13.

Primary role of children	The people the child was influenced by (mechanism) *The grandparents* - indulged children in unhealthy food and beverages. *The grandmother* – her health-conscious family food choices; food rules, encouraged healthy food. *The mother* – her health-conscious family food choice; controlled home food availability; food rules, conditional treats, encouraged multicultural food acceptance; discouraged unhealthy food; encouraged healthy food; discontinued daily dessert habits typical of the grandparent generation (when mothers). *The father* - self-sufficiency food practices; preferences for unhealthy food; modeled relaxed food attitudes; indulged in treats.
Simple food preparation	
Assist parent's food preparation	
Independent food purchasing power (older children)	The people the child influenced (mechanism) *The grandmother* - food refusal; requested preferred family meals; introduced 'new' foods for family meals; requested old fashioned meals or desserts not usually provided by mother. *The mother* - purchasing requests e.g., snack foods, treats, quick meals to microwave, ingredients for baking; voiced preferences for school lunch box daily; voiced individual likes and dislikes; requested special dietary considerations.

Figure 13. The primary roles of children are shown alongside a summary of the intergenerational influence pathways between children and other family members.

Summary of Intergenerational Transmissions of Dietary Behaviours

Family discussions indicated that a number of influences on dietary behaviour were perpetuated across several generations. On the topic of healthy eating (e.g., fruit and vegetables), influence was perceived as transmitted from great-grandmother to grandmother, and through the females in each generation right down to some of the children (by role-modeling and operant conditioning). The reasons families consumed healthful food varied between generations; incorporating thrift, seasonal food availability, and rationing in the grandparent generation (Banwell et al., 2012), whereas health considerations and more global concerns in an obesogenic environment (Finucane et al., 2011) were reported among the parent and child generations. This shows generational differences in environmental contexts although the prevailing attitude to consume healthful foods remained constant when grandparents were parents of growing children, and in the present-day parent generation. For a summary of all intra- and intergenerational transmissions

showing direction and mode of influence see Table 1.

Table 1. *The identified dietary behaviours are shown alongside the intra- and intergenerational influence pathways between the various family member dyads.*

	Direction (and mode) of transmission	
Dietary behaviour	Intragenerational	Intergenerational
Food preparation		Great-grandmothers to grandmothers to mothers to children. Parents to children (role-modeling).
Establish food rules Control of family diet Food selection Meal planning		Great-grandmothers to grandmothers to mothers (role-modeling, operant conditioning, authoritative parenting).
Health-consciousness	Mothers to fathers.	Great-grandmothers to grandmothers to mothers to daughters. Mothers to grandparents (role-modeling, operant conditioning, food exposure, shaping).
Multicultural food acceptance		Mothers to grandparents; mothers to children (food exposure, shaping, operant conditioning).
Restrict fast food		Mothers to children (operant conditioning, authoritative parenting)
Relax food rules Relaxed food attitudes		Fathers to children (role-modeling).
Indulgent treats		Grandparents to children (food exposure, shaping).
Food self-sufficiency Food economy Food waste minimisation		Great-grandfathers to grandfathers to fathers (role-modeling).
Implicit food preferences Food purchasing	Grandfathers to grandmothers. Fathers to mothers.	Children to mothers (operant conditioning by food refusal, requests).
Preferred family meals		Children to grandmothers (operant conditioning by food refusal, requests).
Introduce new foods		Mothers to grandparents (food exposure). Children to grandparents (operant conditioning by food refusal, requests).

The previous literature emphasises that the socialization of growing children within families' plays an important role in developing long-term food acceptance and food preferences (Birch et al., 2007; Contento et al., 2006; Savage et al., 2007). In the present study, grandparents reported enjoying foods today reflecting preferences that were established in childhood decades ago. Even though these foods were infrequently consumed in the present time (e.g., brains, liver, or lard), and food availability has changed over time, food memories of earlier times were fondly recalled. In line with previous research showing food preferences established in childhood persisted into adulthood (e.g., Contento et al., 2006; Savage et al., 2007), the present study presumes that life-long food preferences continue well into old age. These results alert us to implications for today's parent generation as they age, because many have accepted fast-food into their diet whereas grandparents had not. Therefore, there is the potential for the emergence of the next older generation having a greater incidence of overweight and obesity from accepted fast food consumption compared to the present-day grandparent generation.

With the ever-increasing incidence of obesity and chronic disease in Australia, efforts that improve attitudes to the consumption of healthier food choices are critical. Overweight prevention initiatives that target the family network have so far shown moderate success in reducing the risk of obesity and chronic disease in the US (e.g., Claassen et al., 2010; Koehly et al., 2015; O'Neill et al., 2009; Ruffin et al., 2011). The effects of family socialization on food choice have also been demonstrated (Chadwick et al., 2013; Cullen et al., 2001). In addition to targeting the mother to disseminate health messages throughout the family network, attempts to introduce healthier foods and food preparation methods could usefully be targeted at the younger generations, in the hope that they will positively influence the grandparent generations of the future. Given that the older generations in the present study reported being influenced by the younger generations to try 'new' foods and develop 'new' food-related skills.

Consistent with previous research, mothers were dominant in the provision of family meals (Beydoun & Wang, 2009; Green et al., 2009; Prelip et al., 2012). Although there is less evidence in

the literature regarding grandmother influences on diet (Wroten et al., 2012), the present study supported the view that the female role of grandmother also plays the most dominant role in the grandparent household. In the present study, grandmothers had even tighter control over household food choice and meal preparation than mothers; however, this control was tempered by grandmothers respecting their husband's food preferences which were tacitly understood. The trend towards todays fathers' greater involvement in food preparation showed an intergenerational difference when compared to grandfathers, yet mothers still monitored the health status of the family diet and therefore remained involved.

Limitations

The findings of the present study should also be considered in light of some potential limitations. It was evident in the recruitment phase of the study that a large proportion of mothers were tertiary educated and expressed an interest in food or health when enquiring about initial participation. The implication of this is a constraint on generalisability, particularly to less health-conscious population groups. Future research should conduct intergenerational family interviews with families from wider educational and socio-economic backgrounds who have less interest in food. Another potential limitation of the study was that the higher number of female adults compared with male adults participated which may have led to a possible bias towards female perceptions within the families. However, as outlined previously, there is evidence to suggest that females still play a larger role in regards to meal preparations and food choices (e.g., Beydoun & Wang, 2009; Green et al., 2009), and this is consistent with these findings. Nevertheless, future research should endeavour to sample a broader population of males to ensure the generalisability of these findings. The final limitation identified is that the results reflect the views of Anglo-Australian families of English speaking backgrounds whereas Australia is regarded as one of the most ethnically diverse countries in the world (ABS, 2012; Department of Foreign Affairs and Trade (DFAT), 2012), therefore comparison with other ethnicities in similar proportions to population

distributions may reveal a more accurate picture of broader Australian viewpoints[11].

Conclusions and Applications

Aside from broad established factors that influence dietary behaviours, for example, modeling (Bandura, 1977b; Cullen et al., 2001), family socialization (Maccoby, 1992; Nicklas et al., 2001), feeding practices (Savage et al., 2007), and parenting style (Baumrind, 1973), the moderating psychological variables impacting on healthy and unhealthy food consumption within non-disordered eating individuals are yet to be clearly identified. One possible explanation that arose as a result of the present study was the extent that the health-conscious food attitudes of parents could potentially act as a protective mechanism on the subsequent dietary behaviours of children. Previous research has investigated a possible psychological variable moderating parental modeling on dietary intake between mothers and daughters (Prichard et al., 2012), and studies have associated diet-health food attitudes with healthy and unhealthy food consumption (Rozin et al., 2003; Sharp et al., 2013), however, the effect of parental food-attitudes on the healthy and unhealthy food consumption of offspring has not yet been examined. Intergenerational family socialization practices such as parents educating children about nutrition and links with health (or avoidance of disease) requires more research to identify how healthy food attitudes are shared with children and to determine the extent that children adopt them as their own (Nicklas et al., 2001). In addition, food attitudes that have the potential to influence healthy eating practices within families of three generations should be investigated further in order to inform family interventions that motivate dietary change.

Since beliefs are the link between socialisation and behaviour (Conner & Norman, 1995), beliefs that diet affects health arguably could be one psychological mechanism that also links family socialization with healthy or unhealthy dietary behaviours. It may be that parental attitudes impact

[11] This study was published in 2016 comparing Chinese-Australian and Italian-Australian ethnicities with this Anglo-Australian cohort (see Rhodes et al., 2016).

on older or adult children more so than modeling, and modeling impacts on younger children more so than parental attitudes. Possibly due to developmental differences and learning abilities at different childhood age stages (Berk, 2007). Parental influences conveyed by verbal messages and encouragement have been shown to have a stronger effect than modeling on the eating behaviours of adolescents and young adults. Highlighting the need for more research into parental attitudes and the eating behaviours of their children (Rodgers & Chabrol, 2009). Within the intergenerational family, the effect of mothers, fathers, and grandparent's food attitudes on the healthy and unhealthy food consumption of children could also be investigated.

To conclude, this study has identified a number of broad areas that have raised new insights into dietary behaviours that not only confirm the importance of the mother role, but have recognised individual influences on dietary behaviours from other family members and cohorts to include grandmothers, grandparents, fathers, parents and children. Each area of influence which is worthy of more detailed exploration in future research studies that examine the intergenerational transmission of dietary behaviours.

CHAPTER 3: STUDY 2

Title: Examining resemblances in food consumption within three generations of Australian families: Do food attitudes linking diet and health make a difference?

Dietary Choices: The Impact of Considerations about Health

People can make a difference to their modifiable risks for chronic disease by engaging in dietary behaviours that prevent overweight, obesity and consequent chronic disease. These include making "healthy" food choices that include diets high in fruits, vegetables, and fibre; and diets that avoid the overconsumption of sugar, saturated fats, and or salt (Blake et al., 2011; Lim et al., 2012; Lozano et al., 2012). Consistent with the previous literature, Study 1 observed the social norms surrounding food choice in the family context comprised a range of behavioural practices that were linked to the consumption of specific food types. For example, parent-child feeding style (Blake et al., 2011), modeling (Bandura, 1977a, 2001), food choice responsibility, development of child taste preferences (Chadwick et al., 2013) and specific family roles such as "mother" (Johnson et al., 2011; Johnson et al., 2010), which all influenced family food choice and consumption (Ball et al., 2010). Although family food choice can be complex and the responsibility for overall food choice may be shared by several people within the family context (Dunn et al., 2011), the mother was found to have the most influential family role, even when fathers had primary responsibility for family food preparation. Therefore, it was concluded that targeting the mother in sharing information about the link between diet and health throughout the intergenerational family network is important for future diet-related health interventions.

Even though mother's influence was shown to dominate in Study 1, families exist as a complex system and various behaviours that constituted mechanisms of influence on other family member's diet were found to be bidirectional. Study 1 revealed bi-directional influence pathways on dietary behaviours that extended beyond the mother-child dyad to include father-child, grandparent-child, and grandmother-mother. Most influence pathways were identified between intergenerational

dyads although several influences were also intragenerational (i.e., within the same age cohort such as between biological siblings, or marital relationships). Study 2 investigates whether family member and food consumption dyads can be confirmed using a correlational research design. Accordingly, comparisons will be made between dyads within three generation families (i.e., grandparents, parents, and children), and across gendered family roles (e.g., mothers, fathers) in order to confirm dyadic resemblances in food intake. Intergenerationally, the present study aims to investigate primarily the factors influencing children's food consumption. Therefore, Study 2 has referred to individuals within families according to their relationship to the nodal[12] child (e.g., parent, grandparent, siblings up to the age of 25) and when specifying gender, as grandmothers, grandfathers, mothers, or fathers. Children are referred to as sons and daughters rather than boys and girls to easily recognise child status that would also include adult children. Extended[13] family members (e.g., cousins, aunts, uncles) were excluded from the present study.

Study 1 also suggested that health consciousness was a prominent motivating influence on families' subsequent healthy dietary behaviours. As described in the introductory chapter, the present study uses the Health Belief Model (HBM; see Chapter 1) to suggest how Food Life behaviours and attitudes (i.e., Sharp et al., 2013) may act as a psychological mechanism motivating inter- and intragenerational influences on dietary behaviours transmitted between family dyads (e.g., mother-daughter, father-son; between siblings; and between parent and grandparent marital ties). Briefly, the HBM predicts unhealthy behaviours may be modified if an individual believes that susceptibility to chronic disease (e.g., cancer, heart disease, or diabetes) can be avoided by taking health action (e.g., engaging in healthy dietary behaviours), and that the benefits of doing so outweigh any perceived costs or barriers. Therefore, individuals who believe that food consumption impacts upon subsequent disease risk would be expected to engage in healthy dietary behaviours (e.g., fruit and vegetable consumption), and less likely to engage in unhealthy dietary behaviours

[12] All family members can be identified according to their relationship to the nodal child.
[13] Includes any others in the family beyond grandparents, parents, and children.

(e.g., snack and fast food consumption) (Becker et al., 1977).

As detailed previously (in Chapter 1), early research in food attitudes by Rozin and colleagues (1999) examined beliefs and behaviours linking diet, health, and food consumption. Subsequently, Sharp, Hutchinson, Prichard, & Wilson (2013) developed a shortened form of the Food Life Questionnaire (FLQ) originally developed by Rozin and colleagues (2003; 1999). Termed the 'Food Life Questionnaire – Short Form' (FLQ-SF), the two subscales of interest incorporated in the present study are the diet-health oriented behaviour (DHOB) and diet-health/disease linked attitudes (DHLA) measures[14] (Sharp et al., 2013). Individuals who modify their diet and make food choices that prioritise nutrition over taste (e.g., by minimising salt or sugar consumption) are said to exhibit *diet-health oriented behaviour*; and individuals who believe that diet influences subsequent disease (e.g., beliefs that diet can have an effect on obesity, cancer and heart disease) are said to have *diet-health/disease linked attitudes* (Rozin et al., 2003; Sharp et al., 2013). The difference between self-reported dietary behaviours (DHOB) and beliefs that diet can have an effect on subsequent health or disease (DHLA) is that the former represents behaviour aligned with healthy eating and the latter takes this one step further to represent beliefs that one's diet can contribute to obesity and disease causation (Rozin et al., 2003). Sharp and colleagues (2013) reported significant correlations between Food Life subscales and food consumption; higher scores on diet-health oriented behaviour (DHOB) were associated with higher levels of fruit and vegetable consumption; and lower levels of snack and fast food consumption; and higher scores on diet-health/disease linked attitudes (DHLA) were associated with lower levels of fast food consumption (Sharp et al., 2013). Figure 1 shows the expected healthy and unhealthy food consumption associations with low and high Food Life subscale scores.

[14] For ease of reference in this thesis subscale terms were updated. Originally termed Diet-Health Orientation (DHO) and Diet-Health Link (DHL) respectively (Sharp et al., 2013).

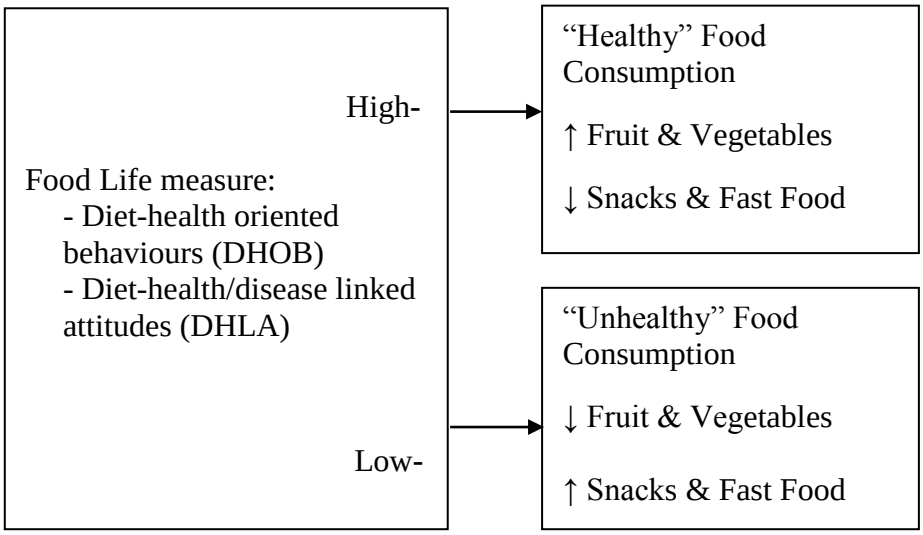

Figure 1. The predicted associations of healthy and unhealthy food consumption with high and low Food Life subscale scores.

Although emerging evidence in research by Sharp and collaborators (2013) suggests Food Life behaviours and attitudes may impact on food consumption, research to date remains scant. One of the limitations of previous research has been that the influence of family relationships on Food Life behaviours and attitudes has not been examined. The present study adds to the literature by examining differences and similarities in Food Life behaviours and attitudes between three generation family dyads and their food consumption, in addition to the Food Life behaviours and attitudes of parents and the food consumption of their offspring.

Summary

As was discussed in introductory Chapter 1, previous research has shown resemblances in food consumption between parents and children (Feunekes et al., 1997), and between mothers and daughters (Beydoun & Wang, 2009; Prichard et al., 2012), however, less is known about resemblances in food consumption between fathers and sons (Mallan et al., 2013), between children and grandparents, or between parents and grandparents (Wroten et al., 2012). The present study investigates the strength and direction of correlations between these family members' healthy and unhealthy food consumption.

The literature indicates various determinants of fruit and vegetable consumption,

notwithstanding the intake of parents (e.g., modeling; Bandura, 1977a, 2001), and access to food (Rasmussen et al., 2006). However, aside from disordered eating, there is less focus in the psychological literature on identifying Food Life behaviours and attitudes that may also play a role (Rasmussen et al., 2006; Shaikh et al., 2008). More research is needed to investigate both healthy and unhealthy food consumption and any relationship of these to attitudes that link diet with consequent disease.

The Present Study

A correlational research design is used in the present study to investigate any associations between *intra*generational and *inter*generational family food consumption attitudes within three generation family dyads. This chapter first investigates resemblances in fruit and vegetable (i.e., healthy food) consumption, and snack and fast food (i.e., unhealthy food) consumption between all dyads. Positive correlations between parent-child dyads are predicted, and female's higher healthy food and lower unhealthy food consumption than males are predicted as gender differences. Secondly, the present study investigates whether Food Life behaviours and attitudes act as motivating factors for families to consume healthy food and forgo unhealthy food by predicting higher associations of Food Life behaviours and attitudes with higher levels of healthy food consumption, and lower levels of unhealthy food consumption. Finally, any impact of parental Food Life behaviours and attitudes on the healthy and unhealthy food consumption of offspring will be explored by predicting higher parental Food Life behaviour and attitude associations with the -higher healthy, and -lower unhealthy food consumption of their biological offspring.

The independent variables are family position, gender, Food Life behaviours and attitudes (i.e., diet-health oriented behaviour, and diet-health/disease linked attitudes). The dependent variables are self-reported fruit, vegetable, snack, and fast food consumption. The relationships between variables are compared intra- and inter-generationally.

Hypotheses.

1. It is predicted that parents and children will demonstrate resemblances in healthy (fruit and

vegetables) and unhealthy (snack and fast food) consumption with positive correlations on each of the dependent variables.

2. It is predicted that females will demonstrate significantly higher scores of healthy- and lower scores of -unhealthy food consumption than males.
3. It is predicted that as Food Life behaviour and attitude scores increase:
    a. self-reported fruit and vegetable consumption will also increase, and
    b. self-reported snack and fast food consumption will decrease.
4. It is predicted that higher parental Food Life behaviour and attitude scores will correlate with their children's higher healthy- and lower -unhealthy food consumption.
5. It is predicted that healthy food consumption will be moderated by Food Life behaviour and attitude levels; higher scores will predict recommendations for fruit and vegetable consumption being met.

A procedural flow diagram is shown in Figure 2.

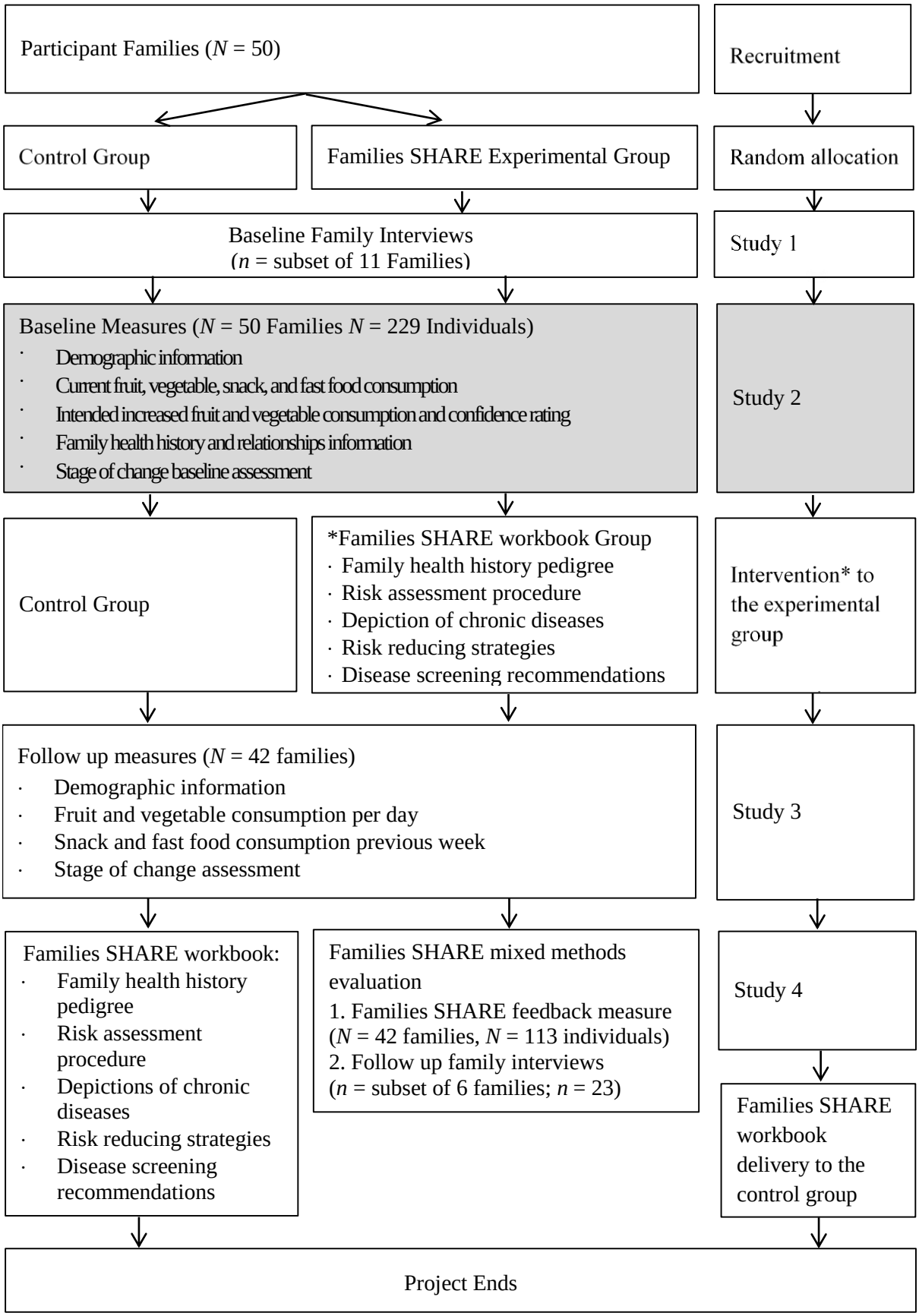

Figure 2. Procedural flow diagram showing Study 2 in relation to all studies.

Method

Participants

Participants were recruited from the community of metropolitan Adelaide (*n* =159), outer Adelaide (*n* = 35), country South Australia (*n* = 8), interstate (*n* = 21) and a location not specified (*n* = 6; *N* = 229 individuals, 50 families). Families that were recruited for the study comprised at least three, and up to seven, individuals across three generations. Each family had at least one child aged between six and 17 years, a parent, a grandparent, and additional family members could have included a second parent, sibling, a second grandparent, or a relative once removed (e.g., a great-aunt, uncle, cousin). There were 50 three generation families in total that were made up of between three and seven family members each (i.e., number of family members: three (*n* = 7 families); four (*n* = 11 families); five (*n* = 22 families); six (*n* = 7 families); seven (*n* = 1 family); except for two families that incorporated two generations). Families identified themselves as of Australian nationality and were of English speaking backgrounds. Families of ethnic background other than Australian and children under the age of six were excluded from the study. Of the 229 participants in the present study, 37% were male (*n* = 85); and 63% were female (*n* = 144). Individuals ranged in age from six to 88 years (*M* = 40.06, *SD* = 23.98). Relatives once removed who were considered extended family members (*n* = 13) were excluded from the present study. Participants who completed the survey were each provided with a supermarket voucher to the value of ten dollars as compensation for their time.

Materials

A questionnaire containing a number of measures was constructed and offered either as an online survey or as a paper survey that was posted to participants and returned in a reply-paid envelope. There were two versions, one for adults and an abridged age-appropriate version for children under the age of 18 years. The entire questionnaire took no more than forty minutes to complete. The following measures comprised the complete adult questionnaire used in the present

study. For the adult and child versions of questionnaire please see Appendices 3.A (adult) and 3.B (child).

Procedure

Sampling procedures. Families were recruited between the 7th May 2012 and the 6th March 2013 from the South Australian community. Recruitment activities included placing flyers on community noticeboards, distributing invitation letters to schools, placing newspaper advertisements, speaking on radio and at community centres, placing advertisements in the social media and via email distribution lists through organisations such as the Cancer Council of South Australia. Participants contacted the researchers directly and written consent was obtained for each individual family member. Written parental consent was obtained for children who had first agreed to participate. Ethical standards were met and approval for the study was granted through the Flinders University Social and Behavioural Research Ethics Committee.

Measures. The measures included demographic information, self-reported fruit, vegetable, snack and fast food consumption, and the diet-health oriented behaviour (DHOB) and diet-health/disease linked attitudes (DHLA) subscales of the 'Food Life Questionnaire – Short Form' (FLQ-SF) (Sharp et al., 2013).

Demographic information. Participants were asked to indicate their age, gender, height, weight, country of birth, marital status, number of children or siblings, number of people residing in their household and highest level of education attained. Self-reported height and weight were used to calculate body mass index (BMI: weight kg/height m^2).

Current food consumption. In order to calculate food consumption and compare the outcomes against NHMRC recommended guidelines, participants' food consumption was assessed using four food frequency questions following Sharp et al., (2013). Food frequency questionnaires used in early and contemporary research as multiple or single item questions measures have demonstrated validity (Glanz et al., 1998; Hu et al., 1999; Ruffin et al., 2011; Sharp et al., 2013).

In the current study, the four food types measured were fruit, vegetables, snacks, and fast

food. For example, participants were asked about their: fruit consumption, "*In the past week, how many serves of fruit did you eat each day?"* one serve of fruit was described as equivalent to one medium sized piece of fruit (e.g., apple, mango, banana), or two small pieces of fruit (e.g., apricot, kiwi fruit, plums), or eight strawberries, or half a cup of fruit juice, which closely approximates 150 grams; vegetable consumption, "*In the past week, how many serves of vegetables did you eat each day?"* one serve of vegetables was described as equivalent to one medium potato, or half a medium sweet potato, or half a cup of dark green leafy vegetables (e.g., spinach, broccoli) or one cup of other vegetables (e.g., salad, beans, lentils), which closely approximates 75 grams; snack consumption, "*In the past week, how many times did you eat snacks?*" (e.g., a chocolate bar, a piece of cake, a packet of chips/twisties/corn chips, ice cream, 3-4 sweet biscuits); and fast food consumption, "*In the past week, how many times did you eat meals that were bought from fast food outlets?"* (e.g., McDonalds, Hungry Jacks, Pizza Hut, KFC, Red Rooster, hamburger, pizza or fish and chip shops).

Participants indicated the number of serves of fruit and vegetables consumed per day, and the number of snacks or fast food meals consumed per week. Fruit and vegetable consumption was calculated as the number of serves per day as a continuous variable. Serves per day were then able to be dichotomized and interpreted as either: below, or equal to and above, the current Australian Dietary Guidelines for the recommended servings of fruit and vegetables per day (NHMRC, 2013a). To date, no guidelines exist for snack and fast food consumption. Fast food and snack consumption were calculated as the number of times consumed in the previous week, higher scores indicated higher consumption. For the complete questionnaire see Appendix 3.A (adult version) and Appendix 3.B (child version).

Food Life behaviours and attitudes. ***Diet-Health Oriented Behaviour (DHOB) and Diet-Health/disease Linked Attitudes (DHLA).*** Two subscales of the Food-Life Questionnaire, Short Form (FLQ-SF) (Sharp et al., 2013) were administered to measure participants' self-reported healthy dietary behaviours, and attitudes that diet can influence subsequent health or disease. The

FLQ-SF is a revised brief version of the Food-Life Questionnaire (FLQ) (Rozin et al., 2003) that contains five subscales in total. The two included subscales were: Diet-Health Oriented Behaviour (DHOB, containing 5 items, e.g., "*I am a healthy eater*"); and Diet-Health/disease Linked Attitudes (DHLA, containing 4 items, e.g., "*Diet can have a big effect on good health"*). Participants responded to each item on a seven-point Likert scale ranging from 1 ('*strongly disagree'*) to 7 ('*strongly agree'*). The three remaining subscales 'Weight Concern', 'Food Pleasure' and 'Natural' subscales were excluded from the present study because they were not relevant to the study's aims and objectives. Both DHOB and DHLA food attitude subscale scores were calculated by averaging participants' aggregate score on the number of items on each subscale, which represented at least 90% of the total items on each subscale. Higher scores indicated higher levels of each characteristic. Previous research has shown acceptable internal reliability for each subscale (with the present study's alpha coefficient values shown in parentheses[15]) DHOB Cronbach's alpha = .67 (.55); DHLA Cronbach's alpha = .75 (.77); and the for the total scales Cronbach's alpha = .75 (not applicable); Sharp et al., 2013). For the complete questionnaire please see Appendix 3.A (adult version) and Appendix 3.B (child version).

Results

Research Design and Statistical Analysis

The present study's research design used firstly, correlations to investigate resemblances in self-reported food consumption, self-reported Food Life healthy behaviours, and Food Life diet-health/disease linked attitudes, between different family members (i.e., grandparents, parents, and children) within three generation families. Secondly, Generalized Linear Mixed Models (GLiMMs) examined differences between generations and gender. Thirdly, Generalized Linear Models with Generalized Estimating Equations (GEE) adjustment examined the odds of fruit and vegetable

[15] Cronbach's alpha reliability tests were conducted on each subscale for each generation separately due to interdependencies of individuals nested within family groups. The separate alphas are presented later in the Results section. Readers interpreting total sample reliability alphas presented here should bear potential interdependency effects in mind, because Cronbach's alpha assumes an independent sample.

consumption meeting NHMRC recommendations based on generation, gender, and Food Life healthy dietary behaviours, and health/disease linked food attitudes. All correlations and modeling procedures controlled for family clustering (i.e., "nesting"). The alpha value was set at $p < .05$ and Bonferonni corrections were included in all statistical analysis to maintain an accurate Type 1 error rate. IBM SPSS 22 software was used for all analyses.

Data screening. Data were checked for errors, missing values, and assumptions of normality. Analyses examined the results between three generations of family members; specifically, the roles of children, parents, and grandparents. In order to examine biological family ties between parents and their offspring, children over the age of 18 years were retained as 'children' and did not exceed age 25. Extreme and impossible cases (e.g., weight 180 kg, 50 serves vegetables daily, BMI 49, height 0.0 m) were excluded from the data set. Missing values were evenly distributed and did not exceed eight percent. Univariate and multivariate outliers were checked prior to each analysis. For complex Generalized Linear Mixed Model (GLiMM) analyses, outliers for each of the four consumption variables were checked by visually inspecting the predicted-by-observed values on scatterplot then examining the Pearson residuals for the cases with the largest values. If the Pearson's residuals exceeded 4.00 (indicating substantially higher values than the rest of the sample), they were then excluded from analyses. Improved model fit was checked after each individual case deletion by observing the -2 pseudo log likelihood results each time (N = 216) (Coxe, West, & Aiken, 2009; Heck, Thomas, & Tabata, 2012). Fox (2008) describe the exact procedures for case deletions since no conventions exist for the interpretation of diagnostic statistics in GLiMM (as one would expect to find in "classical"[16] statistical regression procedures). Pearson's residuals are reported to work well when used in GLiMM for outlier diagnostic procedures and were used in the present study (Fox, 2008). A series of logistic regression analysis required the removal of an additional 5 outliers (N = 211). Healthy food outcome variables

[16] "Classical" statistics refers to Analysis of Variance and tests of multiple regression that rely on the ordinary least-squares (OLS) criterion (Atkins & Gallop, 2007).

were transformed to meet the requirements of logistic regression, that is, fruit and vegetable consumption was dichotomized according to age range and whether each individual met the NHMRC recommendations for the daily consumption of each food type.

Data analysis. The outcome variables fruit, vegetable, snack, and fast food consumption were positively skewed count data (i.e., all skew was greater than 1.9). The reasons for this were due to a large number of valid zero scores in the count data. For example, the grandparent generation rarely ate fast food each week, and amongst all of the consumption variables most individuals scored at the lower end of the range therefore more scores had accumulated below the mean (Elhai, Calhoun, & Ford, 2008; Heck, Thomas, & Tabata, 2012). According to Tabachnick and Fidell (2013), when data are so severely skewed, as occurred with the consumption variables in the present study, any improvements with transformations were likely to be barely negligible, therefore the data analysis strategy sought analyses that best fit the data.

It should be noted that all the quantitative data throughout this thesis were individuals nested within families and in order to control for the effect of similarities between family members (i.e., several individuals belonging to each family) the data could not treat all participants as independent individual participants. Tests of interclass correlations (ICCs) showed interdependencies "within-family" were greater in unhealthy food than healthy food outcome variables. That is, snack consumption three percent, and in fast food consumption eight percent of the total variance was explained by family interdependencies. Healthy food consumption ICCs were close to zero therefore individual responses could have been considered virtually independent of each other (for all ICC Tables see Appendix 3.C).

Correlations used non-parametric Spearman's Rho analyses due to the distributions of the dependent variables. The relative terms "small" (r = .10), "medium" (r = .30), and "large" (r = .50) were reported because they are currently the best bases for estimating correlation effect sizes in behavioural science (Cohen, 2013). More complex investigations were initially considered and first attempted using "classical" statistical approaches, however, given that the assumptions of normality

were violated (i.e., family nesting, skew and kurtosis beyond an acceptable range), tests were conducted using alternate approaches supported by the current statistical literature. The reasons and justifications for GLiMM and GEE statistical models chosen are outlined in more detail in Appendix 3.D because the models used in Study 2 have not been broadly adopted in psychological research and are more typically found in the bio-statistical, economic, or political literature (Atkins & Gallop, 2007; Lee, Wang, Scott, Yau, & McLachlan, 2006). At the time of writing there were no recommended statistical measures of effect size in GLiMM. Confidence intervals have been reported together with exponentiated coefficients (Exp(B)) in the current intergenerational family research literature (e.g., de la Haye, de Heer, Wilkinson, & Koehly, 2014a), therefore 95% confidence intervals have similarly been reported alongside exponentiated coefficients in the present study.

Characteristics of the Sample

There were 50 three generation Australian families that completed the study (N = 229). Individuals were grouped depending on their generational family membership and described according to their relationship to the nodal child[17]. They were: grandparents (25%, n = 57) who ranged in age from 59 to 88 years, parents (34%, n = 78) who ranged in age from 30 to 56 years, children and their siblings (35%, n = 81) who ranged in age from six to 25 years. The majority of families had two children (46%, n = 23), followed by families with one child (42%, n = 21), then families with three children (6%, n = 3), and lastly one family with four children (2%, n = 1). Excluding children, 42% of adults had a university qualification, 25% had a TAFE or technical qualification, and the remaining 33% had attended secondary school as their highest level of formal education.

Over two thirds of the sample, 65% of the males and 71% of the females, met the Australian

[17] The nodal child was the reference family member or "node" that enabled the identification of other family members according to their relationships with that node (e.g., sibling, parent, grandparent).

NHMRC guidelines for the recommended two serves of fruit per day[18] (*N* = 216, *Mdn* = 2, *Range* = 0 - 14) (NHMRC, 2013a, 2013b). Sixty eight percent of Study 2 participants met the recommended daily fruit consumption guidelines compared with 58% of the Australian population (ABS, 2014a). Daily fruit consumption was similar between grandparents (*Mdn* = 2.00, *Range* = 0 - 14), parents (*Mdn* = 2.00, *Range* = 0 - 10), and children (*Mdn* = 2.00, *Range* = 0 - 14). On the other hand, over three quarters of the sample (79%), 82% of the males and 77% of the females, did not meet the Australian NHMRC guidelines for the recommended serves of vegetables per day[19] (*N* = 216, *Mdn* = 3, *Range* = 0.5 – 14) (NHMRC, 2013a, 2013c). Twenty one percent of the sample in the present study consumed the recommended serves of vegetables per day, a higher percentage than the 6.8% of the Australian population who currently meet the recommendations (ABS, 2014a). Vegetable consumption was similar between grandparents (*Mdn* = 3.00, *Range* = 1 - 14), parents (*Mdn* = 3.00, *Range* = 0.5 - 6), and children (*Mdn* = 3.00, *Range* = 0.5 - 14) (see Table 1).

The NHMRC recommend that Australians consume less of the following foods: meat pies, fried hot chips, savoury snacks, processed meats, cakes, biscuits, confectionary, desserts, ice-cream, soft drinks, cordials, and other energy dense food and drinks (NHMRC, 2013a, 2013b). In Study 2, snacks (e.g., a chocolate bar, a piece of cake, a packet of chips/twisties/corn chips, ice cream, 3-4 sweet biscuits) were consumed by participants, on average, four times per week (*Mdn* = 4, *Range* = 0 - 25) and fast food (e.g., McDonalds, Hungry Jacks, Pizza Hut, KFC, Red Rooster, hamburger, pizza or fish and chips) less than once a week (*Mdn* = 0, *Range* = 0 - 12). Children consumed snacks and fast food more often (*Mdn* = 5.00, *Range* = 0 – 17; and *Mdn* = 1.00, *Range* = 0 – 10 respectively) than parents (*Mdn* = 4.00, *Range* = 0 – 25; and *Mdn* = 0.00, *Range* = 0 – 12 respectively), and grandparents (*Mdn* = 4.00, *Range* = 0 - 14; and *Mdn* = 0.00, *Range* = 0 – 4 respectively; see Table 1). Table 2 further presents the descriptive statistics on key variables within

[18] i.e., children 4 to 8 years: at least 1.5 serves, adults and children over 9 years: at least two serves, and one serve is 150 grams which is equivalent to one cup of chopped fruit (NHMRC, 2013c).

[19] i.e., children 4 to 8 years: at least 4.5 serves, adults and children over 9 years: at least five serves of vegetables per day) and one serve is 75 grams which is equivalent to one cup of salad or half a cup of cooked vegetables (NHMRC, 2013c).

the sample as a whole, and grouped by gender and generation.

Table 1.

Means and Standard Deviations, Medians and Range on Food Consumption Variables Among Three Generations.

	Children *n* = 80				Parents *n* = 77				Grandparents *n* = 54			
Variable	*M*	*SD*	*Mdn*	Range	*M*	*SD*	*Mdn*	Range	*M*	*SD*	*Mdn*	Range
Fruit	2.81	2.27	2.00	14.00	2.00	1.45	2.00	10.00	2.63	1.62	2.00	9.00
Vegetables	3.49	2.42	3.00	13.00	3.20	1.27	3.00	5.00	3.23	2.23	3.00	13.00
Snacks	5.76	3.44	5.00	17.00	3.76	2.50	4.00	14.00	3.88	2.91	4.00	14.00
Fast Food	1.09	1.53	1.00	10.00	0.58	0.73	0.00	3.00	0.13	0.34	0.00	1.00

Note. Extended family members excluded. Data were severely skewed and the median and range are shown, given that means and standard deviations would not be as meaningful.

Table 2

Means and Standard Deviations or Medians and Range on the Key Variables in each Group and in the Sample as a Whole.

	Children				Parents				Grandparents				Total			
	Males *n* = 34		Females *n* =47		Males *n* = 30		Females *n* = 48		Males *n* = 18		Females *n* = 39		*N* = 216			
Age range	6 – 18		7 - 25		34 - 56		30 - 55		62 - 88		59 - 84		6 - 88			
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*Mdn*	Range	*M*	*SD*
Age	11.63	3.27	13.04	3.79	46.23	6.02	43.50	6.01	73.61	7.36	69.79	6.93	42.50	6 - 84	40.06	23.98
Height (m)	1.54	0.20	1.56	0.16	1.79	0.07	1.64	0.06	1.75	0.10	1.58	0.06	1.64	1.18-1.98	1.63	0.15
Weight	46.55	18.37	49.28	16.32	87.48	14.60	68.14	12.17	85.59	15.42	65.96	11.32	63.20	24 - 130	64.87	20.58
BMI (kg/m^2)	18.57	2.85	19.60	3.65	27.48	4.00	25.48	4.20	27.91	2.97	26.23	4.44	23.65	13 – 40	23.72	5.16
	Mdn	Range	*Mdn*	Range	*Mdn*	Range	*Mdn*	Range	*Mdn*	Range	*Mdn*	Range	*Mdn*	Range	*M*	*SD*
Serves Fruit (/day)	2.50	0 - 10	2.00	0 - 14	2.00	0 – 4	2.00	1 - 10	2.00	0 – 4	2.00	0 - 09	2.00	0 - 14	2.53	1.96
Veg (/day)	3.00	1 - 14	3.00	0.5 10	3.00	1 - 6	3.00	0.5-6	2.00	1 – 6	3.00	1 - 14	3.00	0.5 - 14	3.39	2.18
Snacks (/week)	5.00	0 - 15	5.00	1 - 17	4.00	0 - 14	3.00	0 – 7	4.00	0 - 10	4.00	0 - 14	4.00	0 - 17	4.51	3.10
Fast food (/week)	1.00	0 – 7	1.00	0 - 10	1.00	0 – 3	0.00	0 – 2	0.00	0 – 1	0.00	0 – 1	<0.01	0 - 10	1.10	0.64

Note. Extended family members excluded. Where data were severely skewed the median and range are shown, given that means and standard deviations would not be meaningful.

Overall, the sample had high mean scores on Food Life behaviours and attitudes with means that ranged between five and six on a scale ranging one to seven (higher scores indicated more of the attribute); Self-reported healthy dietary behaviours (DHOB) mean was five (M = 4.99, SD = 0.84), and attitudes that diet influences subsequent health or disease (DHLA) mean was six (M = 6.01, SD = 0.91). The means and standard deviations on the two Food Life behaviours and attitude subscales can be compared across the three generations, and according to gender (see Table 3 and Table 4). The reliability statistics for the subscales for the total sample and by generations are presented in Table 5[20].

Table 3.

Means and Standard Deviations on Food Attitudes within Three Generationsand the Total Sample.

	Child	Parent	Grand	Total
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
DHOB	4.48 (0.96)	4.98 (0.80)	5.09 (0.83)	4.99 (0.84)
n	78	78	56	212
DHLA	5.53 (0.95)	6.10 (0.83)	5.87 (0.99)	6.01 (0.91)
n	78	78	56	212

Note. [a] Range of possible scores is 1 to 7.

Table 4.
Means and Standard Deviations on Food Attitude Subscales DHOB and DHLA within Generations and Gender.

	Children				Parents				Grandparents			
	Males *n* =34		Females *n* = 47		Males *n* = 30		Females *n* = 48		Males *n* = 18		Females *n* = 39	
Age range	6 – 18		7 – 25		34 – 56		30 – 55		62 – 88		59 – 84	
Variable [a]	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
DHOB	4.58	1.13	4.34	1.08	4.60	0.87	5.22	0.67	4.81	0.68	5.22	0.87
DHLA	5.56	1.00	5.79	1.08	5.83	0.84	6.31	0.74	5.56	1.16	6.02	0.88

Note. [a] Range of possible scores is 1 to 7.

[20] Note. Cronbach's alphas assume independence and these data are not independent.

Table 5.

Reliability Statistics for Food Attitude Subscales and Variability between Generations.

DHOB Reliability Statistics			DHLA Reliability Statistics		
Role in the family	Cronbach's Alpha	*N*	Role in the family	Cronbach's Alpha	*N*
Children	0.51	76	Children	0.69	77
Parents	0.58	56	Parents	0.78	54
Grandparents	0.43	57	Grandparents	0.85	57
Total	0.55	212	Total	0.77	212

Food Consumption Analysis

Due to the positive skew in the data, non-parametric Spearman's rho correlations were calculated. Correlations addressed specific hypotheses about bivariate relationships between family members' food consumption. These tests determined resemblances in the healthy and unhealthy food consumption between various intergenerational and intragenerational family relationship dyads (e.g., parent-child, and grandmother-grandfather respectively). For all correlational analysis, the original data were restructured and each of the data reflected the family unit ($N = 50$), with multiple variables as counts of consumption data for each relevant family member[21].

Food consumption resemblance within three generations: Inter- and intra-generational.

Correlations. Resemblances in food consumption between three generations were explored using Spearman's rho correlations (see Appendix 3E for Table E.1 Fruit, Table E.2 Vegetables, Table E.3 Snacks, and Table E.4 Fast Food). As predicted in the first hypothesis, parents and children[22] shared medium to strong resemblances in healthy food consumption, (fruit, $r = -.31$, $p < .05$, and vegetables, $r = .44$, $p < .01$). Parent-child resemblances in

[21] The reader should bear in mind when interpreting the results that family compositions varied within the sample. Most families had a total of five family members however; all families were not structured equally. Listwise deletions were considered, however, power would have been negligible and this was not a viable alternative to consider.

[22] All dyadic relationship results are reported within-family and therefore represent either biological or marital relationship ties (see Appendix 3.D for further details on statistical analysis that controlled for family nesting).

unhealthy food consumption were non-significant. An unexpected small to medium correlation in fast food consumption was apparent within the parent-grandparent dyad, (r = .27, p < .05). On further breakdown of the data into smaller family unit dyads, correlations in food consumption were as follows.

Healthy food correlations. Mothers and daughters shared moderate fruit consumption correlation (r =.45, p < .01) and grandparent dyads shared a strong correlation (r =.84, p < .01) in fruit consumption. Consistent with the current literature, mother-son and mother-daughter dyads shared moderate to strong resemblances in vegetable consumption (r =.53, p < .01, r = .49, p < .05, respectively). Consumption correlations for fruit and vegetables between all family members are presented in Appendix 3E Tables E.5 and E.6.

Unhealthy food correlations. Father-paternal grandmother and -paternal grandfather dyads shared a large positive correlation in snack consumption, although the sample was small (n < 6) therefore the generalizability of the results is questionable. Consistent with Study 1, grandparent dyads shared a correlation in snack consumption that neared significance, (r =-.47, p = .07; See Appendix 3E Table E.7). There was a very large, significant resemblance in fast food consumption between siblings (r = .89, p < .01), a medium significant resemblance between mother-father marital ties (r = .39, p < .05), and between mother-maternal grandfather dyads (r = .58, p < .05; see Appendix 3E Table E.8).

Healthy and Unhealthy Food: Analysis of Differences.

Hypothesis 2 predicted that females would demonstrate significantly higher healthy- and lower -unhealthy food consumption than males. The Generalized Linear Mixed Models (GLiMMs) family of analyses were used to test for effects of predictors on each food consumption outcome variable (fruit, vegetables, snacks, and fast food). Effects of gender, generation (child, parent, grandparent), and effect of the interaction between gender and generation were the predictors used to examine differences, while analyses also controlled for

the nesting effect of individuals within families[23] (Heck et al., 2012).

Healthy food Generalized Linear Mixed Models. Fruit consumption results of a Poisson GLiMM showed a significant interaction between gender and generation between parents, $t(1, 207) = 2.07$, $p = .04$, confirming that mothers' fruit consumption was greater than fathers (see Table 6, Figure 3). In addition, children's fruit consumption was greater than parents, $F(2, 209) = 5.24$, $p < .01$, pairwise contrasts indicated that the difference was within the parent-child dyad, $t(2, 209) = 3.20$, $p < .01$ (see Table 6 and Figure 3).

[23] Refer to Appendix 3.D for further explanation of analysis reasoning.

Table 6

Fruit consumption Poisson GLiMM Model 1.1. While controlling for the nesting effect of individuals within families on the dependent variable fruit consumption, the effect of gender, then the effect of generation, and effect of the interaction between gender and generation are presented in the table.

Model 1.1 Effect	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Corrected Model				(5, 209)			1.49	.19
Gender	Male-Female	0.32	1.34	(1, 207)	-0.78	0.15	1.79	.18
Generation				(2, 207)			5.24	**<.01**
Child-Parent		0.83	3.20		0.20	1.45		**<.01**
Child-Grand		0.33	1.08		-0.27	0.93		.28
Parent-Grand		0.50	1.69		-0.17	1.16		.18
Gender by Generation				(2, 207)			1.46	.23
Child x M-F		0.12	0.28	(1, 207)	-0.69	0.92	0.08	.78
Parent x M-F		0.68	2.07	(1, 207)	0.32	1.32	4.28	**.04**
Grand x M-F		0.24	0.50	(1, 207)	-0.69	1.16	0.25	.62

Note. Model 1.1: -2 log pseudo likelihood = 438.257. The effect of each variable has controlled for all other variables entered into the model.

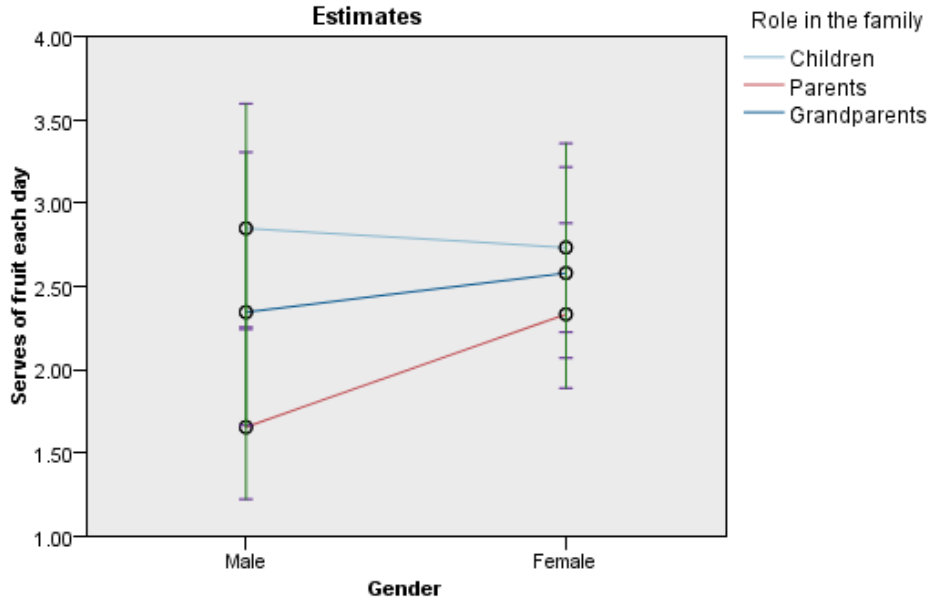

Figure 3. Fruit consumption, Poisson GLiMM Model 1.1. There was a non-significant effect of gender, a significant effect of generation between parent-child, and a significant interaction between gender and generation in the mother-father dyad. Error bars depict the 95% confidence intervals.

A negative binomial GLiMM analysis of vegetable consumption revealed significant differences in consumption between genders for the total sample, $F(1, 194) = 5.77$, $p = .02$, with female consumption greater than males (see Appendix 3.F Table F.1, Figure 4 is shown below).

Unhealthy food Generalized Linear Mixed Models. Predictions that females would demonstrate lower unhealthy food consumption than males were not supported, with non-significant effects for both snack and fast food revealed in the GLiMM negative binomial models[24]. Results included significant effects of generation on snack consumption $F(2, 205) = 5.79$, $p < .01$. The effects were evident between child-parent and child-grandparent dyads;

[24] Due to a high percentage of (valid) zero scores (i.e., > 55%) in fast food consumption, analysis recommendations suggest negative binomial use a 'zero-inflation' technique (Elhai et al., 2008). The zero-inflation option was not available in SPSS version 22 at the time of thesis writing and therefore results were obtained via standard negative binomial GLiMM. Zero inflated modeling is a new method and technique; the key paper was produced in 2008 and there are few examples of applied results in the published literature. The only software to date that can conduct this type of analysis is MPlus and due to time limitations this was not attempted therefore, a negative binomial GLiMM was the best alternative used with SPSS version 22 (Elhai et al., 2008).

child snack consumption exceeded both older generations (see Appendix 3.F Table F.2, Figure 5 is shown below).

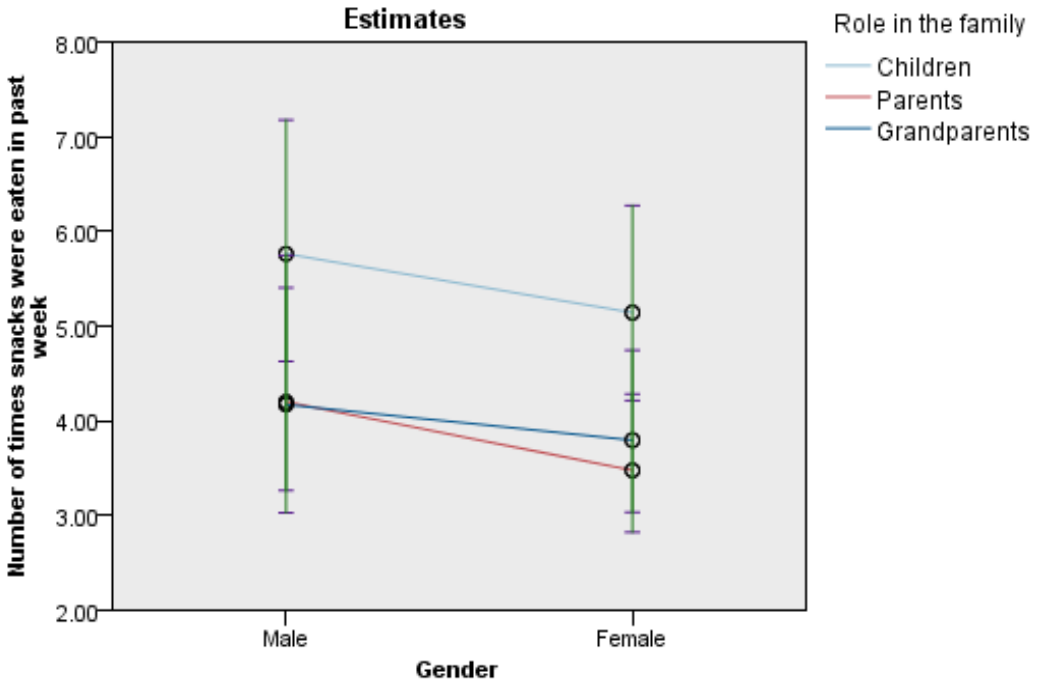

Figure 5. Snack consumption, negative binomial GLiMM Model 3.1. There was a non-significant effect of gender; a significant effect of generation between child-parent, and between child-grandparent; and a non-significant interaction between gender and generation. Error bars depict 95% confidence intervals.

There was a significant difference in fast food consumption between each generational dyad $F(2, 206) = 20.31$, $p < .001$; children's consumption was greater than that of parents and grandparents, and parental consumption was also greater than grandparents (i.e., child-parent dyad, $t(2, 206) = 2.39$, $p = .02$; child-grandparent dyad $t(2, 206) = 5.85$, $p < .001$; parent-grandparent dyad $t(2, 206) = 4.26$, $p < .001$; see Appendix 3.F Table F.3, Figure 6 is shown below).

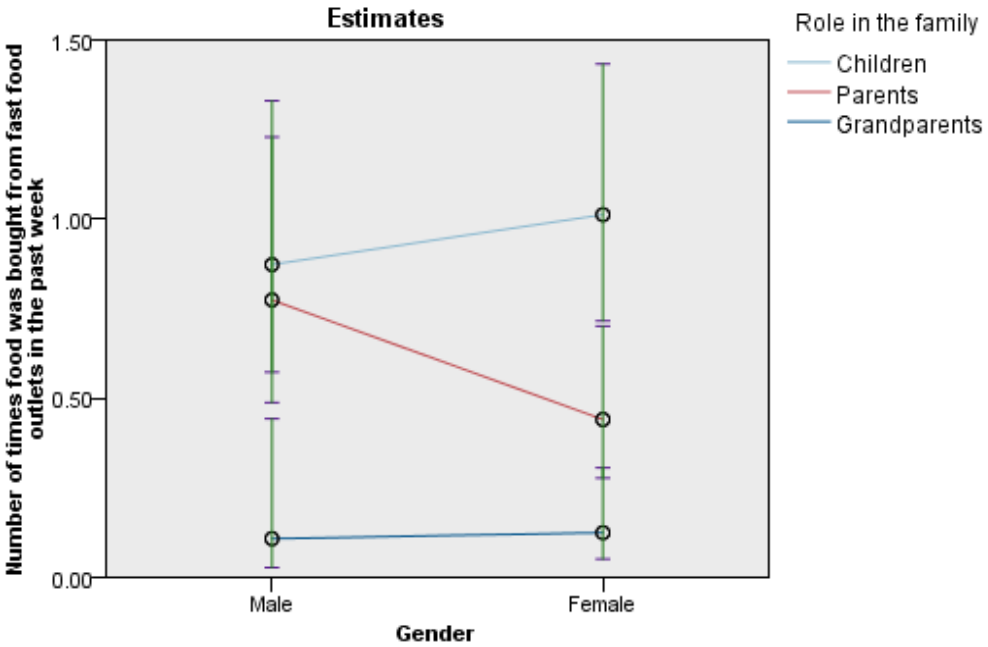

Figure 6. Model 4. 1 GLiMM fast food consumption. There was non-significant effect of gender, a significant effect of generation between each dyad, and a non-significant interaction between gender and generation. Error bars depict 95% confidence intervals.

Food Life Behaviours & Attitudes, Associations with Healthy and Unhealthy Food Consumption

Correlations within the full sample. Results based on the entire sample supported only partially hypothesis 3; as attitudes that diet influences health/disease (DHLA) increased: a) self-reported fruit and vegetable consumption was not impacted, and b) only self-reported snack consumption decreased (see Table 7). Self-reported healthy dietary behaviours (DHOB) showed stronger associations than did attitudes linking diet with subsequent health or disease (DHLA), which was not surprising given the behavioural self-report focus of the former measure.

Table 7

Spearman's Rho Correlations of Food Attitudes with Food Consumption of the Total Sample.

	Fruit	Veg	Snacks	Fast
DHOB	.166**	.283**	-.322**	-.315**
n	212	212	210	211
DHL	.076	.077	-.195**	-.089
n	209	208	207	208

Note. Extended family members excluded. *$p < 0.05$, **$p < 0.01$, (1-tailed).

Correlations: Parental Food Life Behaviours & Attitudes and the food consumption of offspring.

Hypothesis 4 predicted that parent's more positive attitudes to the association between food and health would be associated with the higher healthy and lower unhealthy food consumption among children. Higher scores on fathers' attitudes linking diet with subsequent health or disease (DHLA) were positively associated with son's fruit consumption, and with daughter's vegetable consumption. Unexpectedly, and contrary to the hypotheses, higher scores on mothers' self-reported healthy dietary behaviours (DHOB) had a negative association with daughter's fruit consumption and had a positive association with daughter's fast food consumption (see Table 8).

Table 8

Spearman's rho Correlations of Parent's Mean Diet-Health Food Attitudes with Children's Food Consumption

	Child Fruit		Child Vegetables		Child Snacks		Child Fast	
	M	F	M	F	M	F	M	F
Mother's DHOB	-.27	-.39*	-.27	-.06	-.15	-.26	-.05	.31*
n	26	33	26	33	26	32	26	32
Mother's DHL	-.21	.15	-.21	.21	-.15	-.06	-.16	-.06
n	26	33	26	33	26	32	26	32
Father's DHOB	.03	-.19	.34	.03	-.08	-.19	.11	.03
n	20	21	20	21	20	20	20	20
Father's DHL	.41*	.06	-.20	.53**	-.25	-.09	-.03	.10
n	20	21	30	20	20	20	20	20

*p < 0.05 one tailed, **p < 0.01, one tailed.

Supplementary Analyses

Resemblance in food life behaviours & attitudes and food consumption across three generations. Study 2 explored beyond the four hypotheses to examine which generation/s would reveal Food Life behaviours and attitudes that correlated with their own healthy and unhealthy food consumption. The following tables show generational Food Life behaviours and attitudes with each generation's healthy and unhealthy food consumption. (See Table 9 Fruit, Table 10 Vegetables, Table 11 Snacks, and Table 12 Fast Food consumption)

Table 9

Spearman's Rho Correlations Showing Resemblances in Food Attitudes and Fruit Consumption in Three Generations.

	Child	Parent	Grand
DHOB	.321*	-.057	.239
n	48	47	42
DHL	.207	-.034	.391**
n	46	47	41

Note. Extended family members excluded. *$p < 0.05$, **$p < 0.01$, (1-tailed).

Table 10

Spearman's Rho Correlations Showing Resemblances in Food Attitudes and Vegetable Consumption in Three Generations.

	Child	Parent	Grand
DHOB	.439**	-.005	.329*
n	47	47	43
DHL	.137	-.106	.181
n	45	47	42

Note. Extended family members excluded. *$p < 0.05$, **$p < 0.01$, (1-tailed).

Table 11

Spearman's Rho Correlations Showing Resemblances in Food Attitudes and Snack Consumption in Three Generations.

	Child	Parent	Grand
DHOB	-.370**	-.180	.001
n	47	47	43
DHL	-.203	.058	-.040
n	45	47	42

Note. Extended family members excluded. *$p < 0.05$, **$p < 0.01$, (1-tailed).

Table 12

Spearman's Rho Correlations Showing Resemblances in Food Attitudes and Fast Food Consumption in Three Generations.

	Child	Parent	Grand
DHOB	-.129	-.434**	-.100
n	47	46	42
DHL	-.128	.049	.063
n	45	46	41

Note. Extended family members excluded. *$p < 0.05$, **$p < 0.01$, (1-tailed).

Food Life behaviours & attitudes across three generations: Gender differences explored. Health-consciousness was more prominent in females than males in Study 1, and correlations showed gender variations in food consumption in the present study. Gender differences in Food Life behaviours and attitudes were investigated further in these supplementary analyses. Food Life behaviour and attitude variables met the assumptions for Poisson GLiMMs. Therefore, two tests were conducted for differences between generation, gender, and the interaction between generation and gender on the dependent variables diet-health oriented behaviour (DHOB), and on diet-health disease linked attitudes (DHLA) (SPSS outputs are included in Appendix 3.G, and correlation tables with negligible results).

Finally, the present study hoped to explore whether Food Life behaviours and attitudes have significant fixed effects on healthy and unhealthy food consumption, after gender and generation effects had been controlled for. Measures of Food Life behaviours and attitudes correlate with healthy and unhealthy food consumption in the current study, and show some, although very few, differences between generation and gender. However, which has the stronger effect, the effect of gender, generation, or Food Life behaviours and attitudes on food consumption? Classical statistics determine effect sizes that assist interpretation of how meaningful an independent variable's contribution is to the variance explained in the outcome variable. With these data, however, the alternative modeling strategies that follow

sought to answer further questions regarding the magnitude of effects.

Effects of Generations, Gender, and Food Life Behaviours & Attitudes on Food Consumption

The sets of models that follow were added as a second stage to those models conducted earlier in the results section (i.e., models 1.1 to 4.1). The dependent variables were fruit (Model 1.1[25]), vegetables (Model 2.1[26]), snacks (Model 3.1[27]), and fast food (Model 4.1[28]) consumption. The results of all first stage models were presented on pages 99 to 103 of this results section and are duplicated below for side-by-side comparison to second stage models added here. The predictors in the first stage models were gender, generation, and gender-by-generation entered all at once. In the second stage of modeling that follows, the Food Life behaviours and attitude variables diet-health oriented behaviour (DHOB) and diet-health/disease linked attitudes (DHLA) were then separately added to each of the first stage models (i.e., to Models 1.1 to 4.1).

Food Life behaviours & attitudes and healthy food. ***Vegetable consumption GLiMM.*** Model 2.2 (Figure 9 and Table 13) built upon the previous vegetable Model 2.1 (Table 15, and duplicated below) which showed female's vegetable consumption was greater than males. A negative binomial GLiMM tested for any independent effect of Food Life behaviours and attitudes controlling for the significant effect of gender evident in Model 2.1. In Model 2.2 DHOB was added to Model 2.1 and as a result, there was no longer a significant effect of gender. The chi square difference between Model 2.1 (-2 log pseudo likelihood = 299.582) and Model 2.2 (-2 log pseudo likelihood = 292.396) for 1 *df* was chi square X^2 = 7.19, which was greater than the critical value of 6.64. Therefore, self-reported healthy

[25] Food Life models DHOB & DHLA fruit consumption results were non-significant, see Appendix 3.H
[26] Food Life model DHLA vegetable consumption results were non-significant, see Appendix 3.H
[27] Food Life models DHOB & DHLA snack consumption results were both significant.
[28] Food Life model DHOB fast food consumption results were non-significant, see Appendix 3.H

dietary behaviours (DHOB) made a significant contribution to the vegetable consumption model at the $p < .01$ level and explained more than the differences that arose from gender (see Model 2.2, Figure 9, and Table 13).

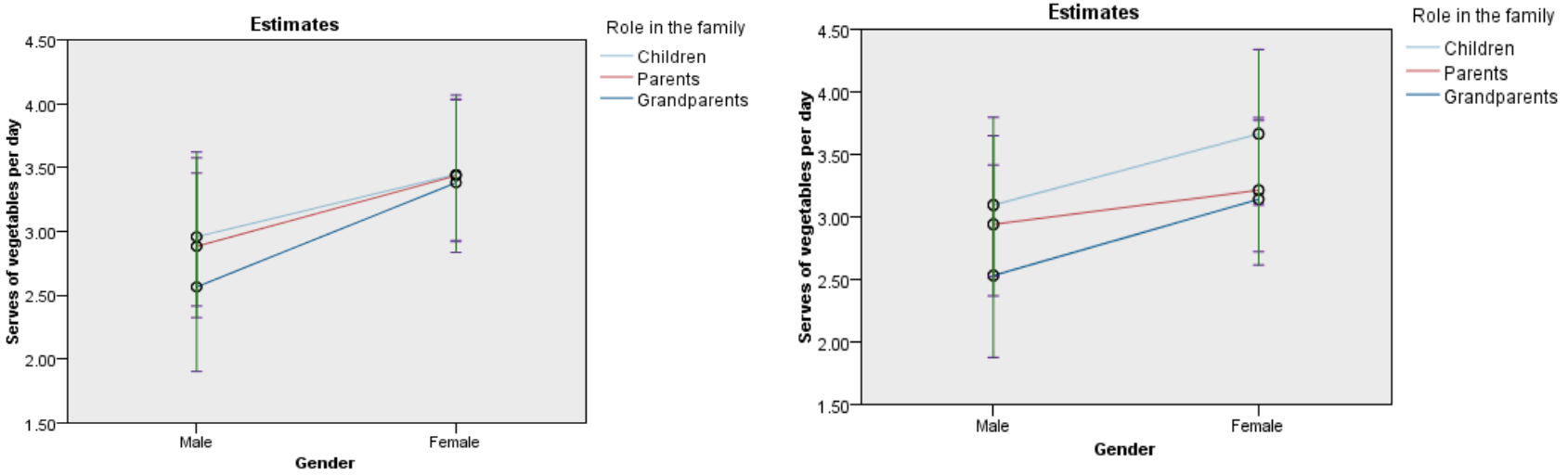

Figure 9. GLiMM vegetable consumption Model 2.1 (left) is compared alongside Model 2.2 (right). In the first Model there was a significant effect of gender, a non-significant effect of generation and no significant interaction between gender and generation. In Model 2.2 (right) DHOB was added to Model 2.1 and there was a non-significant effect of gender, a non-significant effect of generation, and no significant interaction between gender and generation. Each predictor was controlled for the effect of all other predictors within the model. Error bars depict 95% confidence intervals.

Table 13.

Vegetable consumption Model 2.1. While controlling for the nesting effect of individuals within families on the dependent variable vegetable consumption, the effect of gender, then the effect of generation (controlling for gender), and effect of the interaction between gender and generation are presented in the table.

Effect	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model				(5, 194)			1.11	.36
Gender	Male-Female	0.63	2.4	(1, 194)	0.11	1.14	5.77	
Generation				(2, 194)			0.3	.75
	Child-Parent	0.05	0.15	194	-0.6	0.66		1.0
	Child-Grand	0.25	0.74	194	-0.6	1.06		1.0
	Parent-Grand	0.2	0.33	194	-0.6	0.95		1.0
Gender by Generation				(2, 194)			0.17	.85
	Child x M-F	0.49	1.16	(1, 194)	-0.3	1.32	1.36	.25
	Parent x M-F	0.55	1.31	(1, 194)	-0	1.38	1.73	.19
	Grand x M-F	0.82	1.66	(1, 194)	-0.2	1.79	2.76	.10
[a]								

Note . Model 2.1 GLiMM negative binomial. -2 log pseudo likelihood = 299.582. Negative binomial. [a] Row intentionally left blank.

Vegetables Model 2.2: Results are shown with the addition of diet-health oriented behaviour (DHOB) to Model 2.1 (shown left for direct comparison).

	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model				(6, 192)			2.73	**.01**
Gender	Male-Female	0.47	1.85	(1, 192)	-1.01	0.03	3.42	.07
Generation				(2, 192)			1.27	.28
	Child-Parent	0.29	0.94		-0.41	1.00		.70
	Child-Grand	0.55	1.6		-0.28	1.38		.34
	Parent-Grand	0.25	0.78		-0.45	0.96		.70
Gender by Generation				(2, 192)			11.1	**.02**
	Child x M-F	0.57	1.27	(1, 192)	-1.45	0.31	1.62	.21
	Parent x M-F	0.27	0.65	(1, 192)	-1.12	0.56	0.42	.52
	Grand x M-F	0.61	1.27	(1, 192)	-0.34	1.56	1.61	.21
DHOB				(1, 192)			11.1	**.001**

Note . Model 2.2 GLiMM negative binomial. -2 log pseudo likelihood = **292.396**[**]. The X^2 difference between Model 2.1 and 2.2 for 1 *df* = 7.19 and is greater than the critical value of 6.64 therefore diet-health oriented behaviour made a significant contribution to the model at the $p < .01$ level, [phi-coefficient $\phi = .14$].

Food Life behaviours & attitudes and unhealthy food. ***Snack consumption GLiMMs.***

Model 3.1 (Figure 10 and Table 14) and Model 3.2 (Figure 10 and Table 14) were built using two negative binomial GLiMMs to test for any independent effects that Food Life behaviours and attitudes may have had on snack consumption, controlling for the effects of generation evident earlier in Model 3.1 (Appendix 3.F, and duplicated below) which showed child's snack consumption was significantly greater than that of parents and grandparents. In Model 3.2 self-reported healthy dietary behaviours (DHOB) was added to Model 3.1 and there were no significant effects of gender, generation, or interaction between gender and generation on snack consumption. The likelihood ratio test was significant at the $p < .001$ level. The -2 log pseudo likelihood of Model 3.1 (422.508) was less than Model 3.2 (407.607) and the chi square difference between Model 3.1 and 3.2 for 1 degree of freedom was $X^2 = 14.90$. The difference was greater than the critical value of 10.83 showing that self-reported healthy dietary behaviours (DHOB) made a significant contribution to Model 3.1, more so than did the effect of generation previously reported in Model 3.1 (see Models 3.1 and 3.2 in Figure 10 and Table 14).

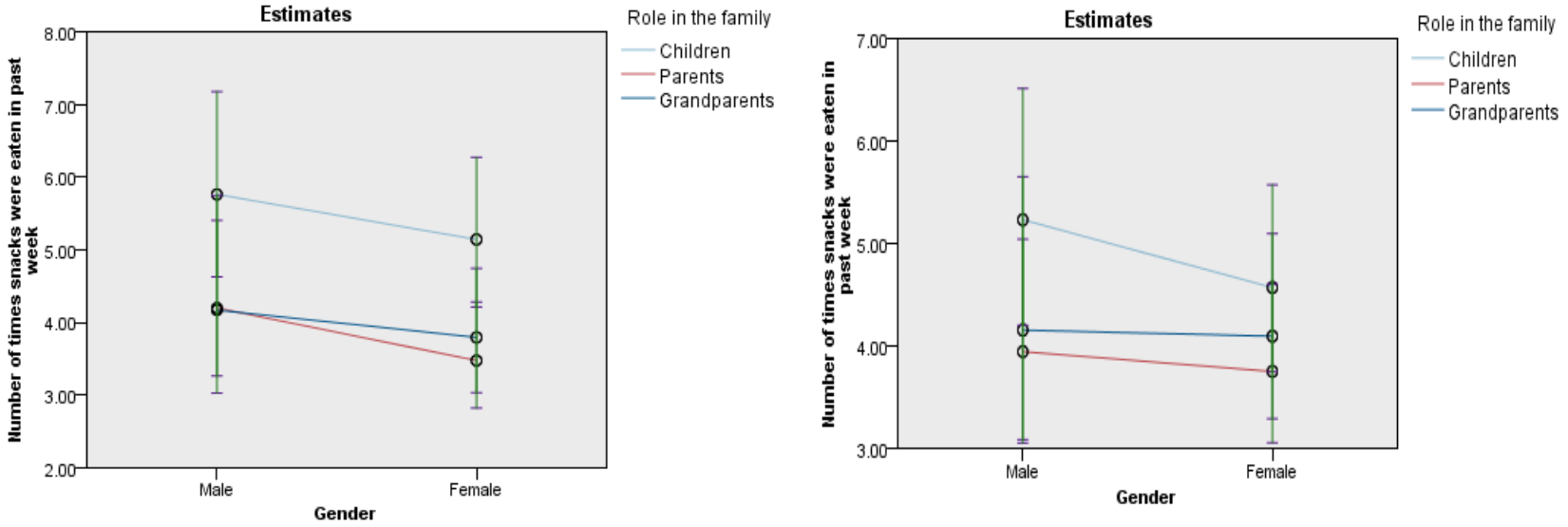

Figure 10. GLiMM snack consumption Model 3.1 (left) is compared alongside Model 3.2 (right). In the first model there was a non-significant effect of gender; significant effects of generation between child-parent, and between child-grandparent; and a non-significant interaction between gender and generation. In Model 3.2 (right) DHOB was added to Model 3.1 and there was a non-significant effect of gender or generation, and no significant interaction between gender and generation. Each predictor was controlled for the effect of all other predictors within the model. Error bars depict 95% confidence intervals

Table 14

Snacks Model 3.1. While controlling for the nesting effect of individuals within families on the dependent variable snack consumption, the effect of gender, then the effect of generation, and effect of the interaction between gender and generation are presented in the table.

	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower		*F*	*p*
Model 3.1.				(5, 205)			3.27	**.007**
Gender	Male-Female	0.58	1.32	(1, 205)	-0.3	1.4	1.74	.19
Generation				(2, 205)			5.79	**<.01**
	Child-Parent	1.62	3.25	205	0.42	2.8		**<.01**
	Child-Grand	1.47	2.67	205	0.23	2.7		**.02**
	Parent-Grand	0.16	0.32	205	-0.8	1.1		.75
Gender by Generation				(2, 205)			0.09	.92
	Child x M-F	0.62	0.77	(1, 205)	-1	2.2	0.59	.44
	Parent x M-F	0.72	1.14	(1, 205)	-0.5	2	1.3	.26
	Grand x M-F	0.38	0.48	(1, 205)	-1.2	1.9	0.23	.64
[a]								

Note . Model 3.1: GLiMM negative binomial. -2 log pseudo likelihood = 422.508. The effect of each variable has controlled for all other variables entered into the model. [a] Row has been intentionally left blank.

Snacks Model 3.2: Results are shown with the addition of diet-health oriented behaviour (DHOB) to Model 3.1 (shown left for direct comparison).

	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% C I Lower		*F*	*p*
Model 3.2				(6, 203)			5.8	**<.001**
Gender	Male-Female	0.28	0.69	(1, 203)	-0.53	1.1	0.47	.49
Generation				(2, 203)			2.51	**.08**
	Child-Parent	1.04	2.23		-0.09	2.2		.08
	Child-Grand	0.77	1.48		-0.44	2		.30
	Parent-Grand	0.28	0.57		-0.68	1.2		.57
Gender by Generation				(2, 203)			0.15	.86
	Child x M-F				-0.72	2.1	0.89	.35
	Parent x M-F	0.66	0.94	(1, 203)	-1.02	1.4	0.1	.75
	Grand x M-F	0.19	0.32	(1, 203)	-1.48	1.6	<.01	.94
DHOB				(1, 203)			18	**<.001**

Note . Model 3.2 GLiMM negative binomial. -2 log pseudo likelihood = 407.607***. The X^2 difference between Model 3.1 and 3.2 for 1 *df* = 14.90 and was greater than the critical value of 10.83 therefore diet-health orientation made a significant contribution to the model at the $p < .001$ level.

In Model 3.3 (Figure 11 and Table 15) self-reported healthy dietary behaviours (DHOB) was removed and diet-health/disease linked attitudes (DHLA) was added[29] to Model 3.1 (see Appendix 3.F and Table 15 below). The effect of generation was again significant between children and parents, and neared significance between children and grandparents. The -2-log pseudo likelihood was 416.244 compared with 422.508 in Model 3.1. The chi square difference between Model 3.1 and Model 3.3 was $X^2 = 6.26$ which is greater than the critical value of 3.84 for one degree of freedom at the $p < .05$ level. Therefore, attitudes that diet influences subsequent health/disease also made a significant contribution to snack consumption. Both generation and DHLA equally had significant effects on snack consumption (for Model 3.3 compared with Model 3.1 see Figure 11 and Table 15).

[29] Both DHOB and DHLA could not be entered into the model at once due to variable limitations on the analysis.

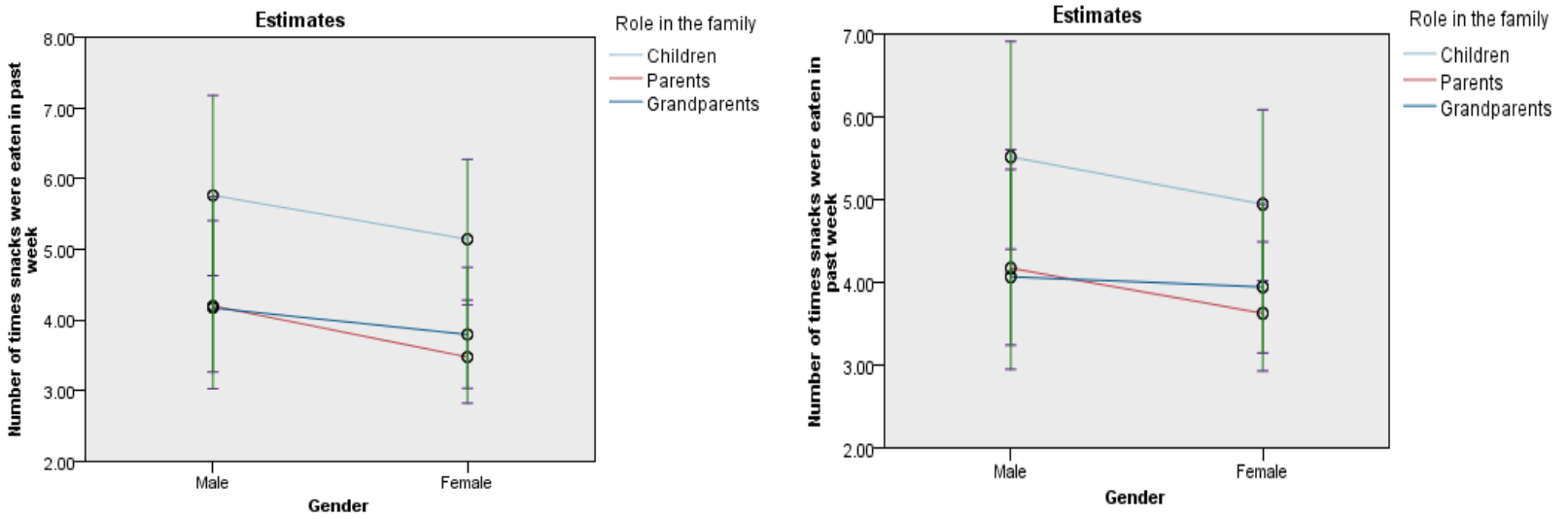

Figure 11. GLiMM snack consumption Model 3.1 (left) is compared alongside Model 3.3 (right). In the first model there was a non-significant effect of gender; significant effects of generation between child-parent, and between child-grandparent; and a non-significant interaction between gender and generation. In Model 3.3 (right) DHLA was added to Model 3.1 and there was no significant effect of gender; a significant effect of generation; and a non-significant interaction between gender and generation. Each predictor was controlled for the effect of all other predictors within the model. Error bars depict 95% confidence intervals.

Table 15

Snacks Model 3.1 consumption, negative binomial GLiMM Model 3.1. While controlling for the nesting effect of individuals within families on the dependent variable snack consumption, the effect of gender, then the effect of generation, and effect of the interaction between gender and generation are presented in the table.

	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower		*F*	*p*
Model 3.1.				(5, 205)			3.27	**.007**
Gender	Male-Female	0.58	1.32	(1, 205)	-0.29	1.4	1.74	.19
Generation				(2, 205)			5.79	**<.01**
	Child-Parent	1.62	3.25	205	0.42	2.8		**<.01**
	Child-Grand	1.47	2.67	205	0.23	2.7		**.02**
	Parent-Grand	0.16	0.32	205	-0.81	1.1		.75
Gender by Generation				(2, 205)			0.09	.92
	Child x M-F	0.62	0.77	(1, 205)	-0.97	2.2	0.59	.44
	Parent x M-F	0.72	1.14	(1, 205)	-0.53	2	1.3	.26
	Grand x M-F	0.38	0.48	(1, 205)	-1.18	1.9	0.23	.64
[a]								

Note . Model 3.1 GLiMM negative binomial. -2 log pseudo likelihood = 422.508. The effect of each variable has controlled for all other variables entered into the model.[a] Row intentionally left blank.

Snacks Model 3.3: Results are shown with the addition of diet-health/disease linked attitudes (DHLA) to Model 3.1 (shown left for direct comparison).

	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% C I Lower		*F*	*p*
Model 3.3.				(6, 200)			3.3	**.005**
Gender	Male-Female	0.4	0.92	(1, 200)	-0.46	1.3	0.9	.36
Generation				(2, 200)			3.8	**.02**
	Child-Parent	1.33	2.61		0.1	2.6		**.03**
	Child-Grand	1.22	2.22		-0.02	2.5		**.07**
	Parent-Grand	0.12	0.24		-0.86	1.1		.82
Gender by Generation				(2, 200)			0.10	.91
	Child x M-F	0.57	0.73	(1, 200)	-0.98	2.1	0.5	.47
	Parent x M-F	0.54	0.85	(1, 200)	-0.72	1.8	0.7	.40
	Grand x M-F	0.12	0.15	(1, 200)	-1.43	1.7	0	.88
DHLA				(1, 200)			4.2	**.04**

Note. Model 3.3 GLiMM negative binomial. -2 log pseudo likelihood = 416.244[*]. X^2 difference between Model 3.1 and Model 3.3 for 1 *df* = 6.26 which is greater than the critical value of 3.84 at the $p < .05$ level, therefore diet-health/disease linked attitudes made a significant contribution to Model 3.1.

Fast Food GLiMM. A negative binomial GLiMM was conducted to test for any independent effects of attitudes that diet influences subsequent health/disease (DHLA), controlling for the effects that generation and gender had on the previous fast food Model 4.1 (as seen in Appendix 3.F and duplicated below) which showed children's fast food consumption was significantly greater than parents and grandparents; and that parental fast food consumption was also greater than grandparents. In Model 4.3 DHLA was added to Model 4.1 (see Figure 12 and Table 16). The overall effect of generation was slightly less but remained significant for all family dyads. The likelihood ratio test revealed a significant effect of diet-health/disease linked attitudes on fast food consumption. The chi square difference between Model 4.1 and Model 4.3 for one degree of freedom was $X^2 = 15.03$ which is greater than the critical value of 10.83 at the $p < .001$ level. Therefore, attitudes linking diet with health and disease made a significant contribution to Model 4.1 in fast food consumption, as did the effect of generation in Model 4.3 (see Figure 12 and Table 16).

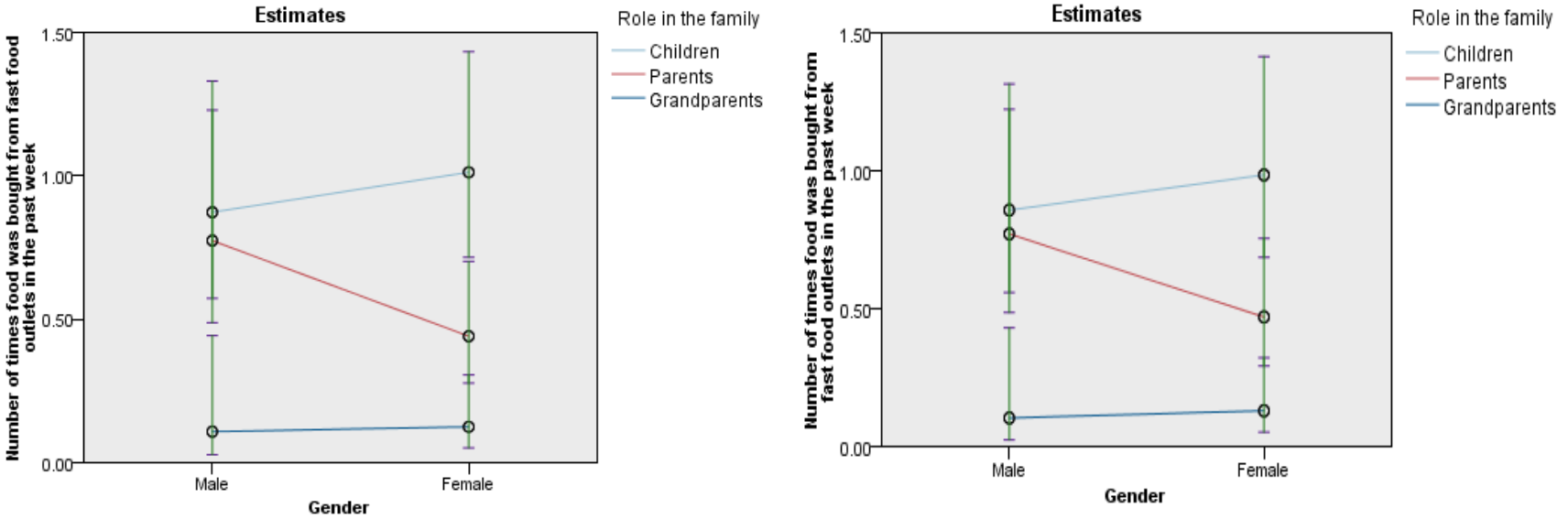

Figure 12. GLiMM fast food consumption Model 4.1 (left) is compared alongside Model 4.3 (right). In the first model, there was a non-significant effect of gender, a significant effect of generation between each dyad, and a non-significant interaction between gender and generation. In Model 4.3 (right) DHLA was added to Model 4.1 and there was a non-significant effect of gender, a significant effect of generation returned between all dyads; and there was a non-significant interaction between gender and generation. Each predictor was controlled for the effect of all other predictors within the model. Error bars depict 95% confidence intervals.

Table 16

Fast Food Model Model 4.1. While controlling for the nesting effect of individuals within families on the dependent variable fast food consumption, the effect of gender, then the effect of generation, and effect of the interaction between gender and generation are presented in the table.

Model 4.1	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower		*F*	*p*
				(5. 206)			6.7	**<.001**
Gender	Male-Female	0.04	0.28	(1, 206)	-0.22	0.29	0.1	.78
Generation				(2, 206)			20	
	Child-Parent	0.36	2.39	206	0.06	0.65		**.02**
	Child-Grand	0.83	5.85	206	0.48	1.17		
	Parent-Grand	0.47	4.26	206	0.22	0.72		
Gender by Generation				(2, 206)			1.6	.20
	Child x M-F	0.14	0.58	(1, 206)	-0.62	0.34	0.3	.57
	Parent x M-	0.33	1.69	(1, 206)	-0.06	0.72	2.9	.09
	Grand x M-F	0.02	0.18	(1, 206)	-0.17	0.2	0	.86

[a]

Note . Model 4.1 GLiMM negative binomial: -2 log pseudo likelihood = 767.282. The effect of each variable has controlled for all other variables entered into the model.

Fast Food Model 4.3: Results are shown with the addition of diet-health/disease linked attitudes (DHLA) to Model 4.1 (shown left for direct comparison).

Model 4.3	Pairwise Contrasts	Contrast Estimate	t	*df*	95% CI Lower	Upper	*F*	*p*
				(6, 201)			5.9	**<.001**
Gender	Male-Female	0.02	0.14	(1, 201)	-0.24	0.27	0	0.89
Generation				(2, 201)			20	**<.001**
	Child-Parent	0.32	2.01		<0.01	0.63		**0.05**
	Child-Grand	0.8	5.58		0.46	1.15		**<.001**
	Parent-Grand	0.49	4.28		0.23	0.74		**<.001**
Gender by Generation				(2, 201)			1.3	**0.28**
	Child x M-F	0.13	0.54	(1, 201)	-0.6	0.34	0.3	0.59
	Parent x M-	0.3	1.49	(1, 201)	-0.1	0.7	2.2	0.14
	Grand x M-F	0.03	0.28	(1, 201)	-0.16	0.21	0.1	0.78
DHL				(1, 201)			1.6	**<.001**

Note. Model 4.3 GLiMM negative binomial:. -2 log pseudo likelihood = 752.250[***]. The X^2 difference between Model 4.1 and Model 4.3 for 1 *df* was 15.03 which is greater than the critical value of 10.83 at the $p < .001$ level, therefore diet-health/disease linked attitudes made a significant contribution to Model 4.1 fast food consumption.

Were the Odds of Meeting Healthy Food Recommendations Moderated by Food Life Behaviours & Attitudes?

The aim of the next set of analyses was to determine whether NHMRC recommendations for fruit and vegetable consumption were "met" or "not met" and whether or not the odds ratio changed with the addition of gender, generation, Food Life behaviour or attitudes, and interaction predictors. It was predicted that the odds of an individual increasing healthy, and decreasing unhealthy, food consumption would be greater with higher (i.e., more positive) Food Life behaviour and attitude scores.

With the benefit of interpretation using odds ratios, a series of multiple logistic regression analyses was conducted using statistical procedures that managed family nested data[30]. Fruit and vegetable consumption were dichotomized according to NHMRC guidelines.

Was fruit consumption moderated by diet-health oriented behaviour (DHOB)?

The series of Model 5 (see Appendix 3.I) results confirmed that self-reported healthy dietary behaviours (DHOB) was a significant predictor of fruit consumption, when controlling for all other predictors entered into the model. The odds of meeting the recommended fruit consumption guidelines were between two (minimum Exp(B) = 2.04) and three (maximum Exp(B) = 3.22) times significantly greater when DHOB was added to each model (see Appendix 3.I, Models 5.1 through to 5.6 in Tables I.1 to I.6 for results of each stage of the model built).

Model 5.1 shows that of the 207 individuals within 50 families who either met the NHMRC daily fruit consumption recommendations (adjusted for age, e.g., > 2 serves per day for adults) or did not (e.g., < 2 serves per day), higher diet-health orientation (DHOB) scores

[30] Generalized Linear Modeling (GLiM) with a Generalized Estimating Equation (GEE) adjustment was the procedure used that accommodated multiple logistic regressions with the data conditions in the present study. Again, Bonferonni corrections were included in all modeling in order to maintain minimise Type 1 errors (Gardiner, Luo, & Roman, 2009; Heck, Thomas, & Tabata, 2014b; Hubbard et al., 2010).

were significant predictors of fruit consumption (DHOB coefficient = 0.71, SE = 0.23, p = .002, odds ratio = 2.04, 95% CI = 1.29 to 3.23) (Lang & Secic, 2006).

Was fruit consumption moderated by diet-health/disease linked attitudes (DHLA)? Model 6 results confirmed that attitudes linking diet with subsequent health/disease (DHLA) was a significant predictor of fruit consumption, when controlling for all other predictors entered into the model. The odds of meeting the recommended fruit consumption guidelines were one and a half times significantly greater with DHLA included in the model. When gender was added to the model, the odds of males meeting the fruit recommendations were significantly less than females. In the subsequent interaction between gender and DHLA, the odds of males with high DHLA scores meeting the recommended serves of fruit per day were shown to be significantly *greater* than females with high DHLA scores (see Appendix 3.J for Model tables 6.1 through to 6.6 in Tables J.1 to J.6 for results of each stage of the model built).

Model 6.3 was used when calculating the adjusted odds according to DHLA at the mean (6.01), as well as one standard deviation above (6.01 + 0.91 = 6.92) and below the mean (6.01 – 0.91 = 5.10). The resultant slopes showed the rate at which DHLA influenced males compared to females. The calculation used may be viewed in Appendix 3.J along with the full set of Model 6 tables for reference. Ultimately, the slope remained constant and small for females (0.05), whereas the slope was steeper for males (0.94). With a one standard deviation increase in DHLA, the odds of males meeting fruit consumption recommendations increased from 1.31 to 8.58 times more than females. Hence, DHLA was more important to males than females, and factors unknown (i.e., other than DHLA) influenced females who met fruit consumption recommendations (Coxe et al., 2009; Hubbard et al., 2010; Sandifer, 2007).

Was vegetable consumption moderated by diet-health oriented behaviour (DHOB)? Results showed that, with higher self-reported healthy dietary behaviour (DHOB) scores, the odds of meeting the recommended vegetable serves per day was one and a half times greater than with lower DHOB scores. With gender added to the model, the odds of males meeting the recommendations for vegetable consumption were lower than that of females. Lastly, when the gender-by-DHOB interaction was added to the model, the odds of males with higher DHOB scores meeting the recommended serves of vegetables per day were lower than that of females with higher DHOB scores. Refer to Appendix K for Models 7.1 to 7.6 (in Tables K.1 to K.6) that show all vegetable consumption and diet-health orientation modeling and slope calculation results.

In Model 7.4 the odds of males meeting the recommendations for vegetable consumption was lower than that of females (male coefficient = -4.03, SE = 1.89, p = .03, odds ratio = 0.02, 95% CI = 0.00 to 0.72). The gender-by-DHOB interaction showed that with higher DHOB scores, the odds of males meeting the recommendations for vegetable consumption was less than that of females and the result neared significance. The male-by-DHOB interaction coefficient = 0.72, SE = 0.37, p = **.052**, odds ratio = 2.06, 95% CI = 0.99 to 4.28 (Lang & Secic, 2006). However, both scores for males and females were negative which reveals that few participants of either gender were meeting the recommendations for vegetable consumption. This is not surprising given the 2014 ABS figures show that less than 7% of Australians and 21 % of the present study participants met the recommended vegetable consumption guidelines.

Was vegetable consumption moderated by diet-health link (DHLA)? Models 8.1 through to 8.6 with vegetable consumption and DHLA were conducted in the same manner as Models 6.1.to 6.6 with fruit consumption and DHLA. The results of Model 8 were non-significant and the tables.

Discussion

The most recent Australian Bureau of Statistics (ABS) Health Survey to date reported that in 2011-12 only 54% of Australians met the recommended serves of fruit per day and only 6.8% met the recommended serves of vegetables per day (ABS, 2014a). The previous Health Survey reported similar fruit consumption levels in 2011-12, however vegetable consumption was previously greater at 9% (ABS, 2006). Whilst fruit consumption is stable and remains adequate in half of the population, vegetable consumption is at very low levels and statistics are trending downward. Both fruit and vegetables are important dietary components that contribute significantly to disease prevention (Stewart & Wild, 2014). Hence the urgent need to increase fruit and especially vegetable consumption in the Australian diet.

Intergenerational Resemblances and Differences in Healthy Food Consumption

The literature has demonstrated the important role of "mother" in impacting the food consumption of offspring (Beydoun & Wang, 2009; Feunekes et al., 1997; Prichard et al., 2012; Wang, Beydoun, Li, Liu, & Moreno, 2011). The current study similarly confirmed mother-child resemblances in healthy food consumption and results support the mother's influence on children more so than fathers or grandparents.

Intergenerational correlations of fruit and vegetable consumption showed positive associations between the parent-child dyad. When broken down further, results indicated that the association was particularly robust in mother-daughter dyads. Similarly, with vegetable consumption, results showed larger positive correlations between parents and children, and in particular, between mother-daughter, and mother-son dyads.

Generalized Linear Mixed Model analysis examining the influence of gender indicated higher healthy food and lower unhealthy food consumption in females more so than males. Significant GLiMM analysis also showed mother's fruit consumption was greater than

that of fathers within the parent generation. Along with the absence of significant correlations between fathers and offspring, Study 2 supported the previous literature that shows the importance of the "mother" role on children's healthy food consumption (e.g., Johnson et al., 2011; Johnson et al., 2010). Mothers of young children have more control over what foods their children consume because younger children are less able to make their own food choices or prepare meals for themselves. Previous research has shown strong resemblances in food intake between parents and their older children, where mothers had stronger associations of food consumption with their adolescent children than fathers did (Feunekes, de Graaf, Meyboom, & van Staveren, 1998).

There were no significant grandmother-mother correlations evident with healthy food consumption. Interestingly, while these mother-child relationships were evident with mother-young child and mother-adolescent child dyads, the same relationships were not apparent with this biological family tie at a later life-stage (i.e., grandmother-parent was also a mother-child biological family-tie however at a later life-stage). One may assume separate households account for this different outcome; however, some grandparent-parent correlations were significant in unhealthy food consumption. An alternative explanation could be simply that generational changes occurs as parent-child relationships move across life-stages (Shapiro, 2004; Ward, Deane, & Spitze, 2014). In other words, mothers actively in the "mother" role might have a stronger focus on healthy eating when most responsible for the dietary intake of children, thus explaining the significant resemblances within mother-child dyads. Then, when mothers reach the grandmother life-stage, the mother-child family-tie changes, because grandmothers cease to act in the "mother" role with adult children's food consumption, thus explaining the non-significant resemblances within grandmother-parent dyads.

Intragenerational Resemblances and Differences in Healthy Food Consumption

Intragenerational correlations showed that child siblings shared a medium positive association in vegetable intake. Sibling resemblances in vegetable consumption were not surprising; children in the same family are likely to share the evening meal together and be exposed to who parents encourage consumption using the various methods described in Study 1 (e.g., conditional treats).

Grandparent marital ties shared strong resemblances in fruit, but not vegetable, consumption. Again, differences in maternal life-stage may account for these results. After relinquishing the mother role, along with responsibility for children's healthy food consumption, grandmothers' approach to vegetable consumption within their own separate households may become more relaxed. Although Study 2 was not a longitudinal study, shifts in life-stage over time may explain generational and even some of the gender differences in vegetable consumption. As suggested in Study 1, vegetable consumption was of great concern to mothers when parenting children and when modeling healthy eating practices. Study 1 also proposed grandmothers were more health-conscious than grandfathers.

Alternatively, differences in grandparents' vegetable consumption might be an indication of grandmothers' lack of control over what grandfathers eat or the fact that as they age men believe they no longer need to comply with healthy eating guidelines. Significant differences in gender were noted in the study's GLiMM analyses; female's vegetable consumption was greater than males. Future dietary interventions that take into account these inter- and intragenerational results should consider motivating consistent vegetable consumption as people age, especially in men, and promote greater fruit consumption in children. Future multigenerational dietary interventions that involve disseminating diet-health related information should consider increasing awareness through mothers and grandmothers within the multigenerational family network.

Inter- and Intra- Generational Resemblances and Differences in Unhealthy Food Consumption

GLiMM results showed significant intergenerational differences in snack consumption with children's consumption greater than that of parents and grandparents. The paternal grandparent-father dyad shared significant resemblances in snack consumption, however the correlation had only six pairs ($n = 6$). This contrasts with healthy food consumption; the same family-tie at an earlier life-stage showed a strong mother-son resemblance in vegetable consumption. This change from healthy to unhealthy food resemblance over time could imply that as life-stage progression occurs, parent's focus on healthy food consumption shifts in later life to more relaxed food habits when grandparents spend time with their adult sons. Grandparent's relaxed food attitudes over time could explain the large snack consumption correlation that neared significance, ($r = .47$, $p = .07$) between grandparent marital-ties.

The fast food consumption results were unsurprising at the intragenerational level. Parent marital-ties shared a moderate resemblance and child siblings shared a large resemblance in fast food consumption ($r = .89$, $p < .01$). This is likely to represent generational cohort attitudes towards fast food and the extent to which fast food occasions are shared family experiences. As suggested by the grandparent generation in Study 1, and corroborated in the present study, grandparent marital-ties generally did not consume, or only infrequently consumed, fast food and results indicated consumption decreased with each older generation. Results of GLiMMs on fast food consumption confirmed significant differences between all three generations. These results highlight how fast food appears to be more acceptable with each younger generation and could, to some extent, explain an emerging problem (Hebert, Allison, Archer, Lavie, & Blair, 2013; Popkin, Adair, & Ng, 2012). Grandparents' low levels of fast food consumption could reflect lack of exposure in

childhood[31], which may explain their disinclination in late adulthood. There are implications for parent and child generations exposed to fast food at a younger age; consumption habits laid down at a younger age may be perpetuated in life-long preferences that continue long into adulthood (Contento et al., 2006). Therefore, parent and child generations of the future who have socialized "acceptable" or "normal" fast food exposure and consumption habits, have the potential to negatively impact their later health with ageing. Alternatively, it is possible that food choices move away from blatantly unhealthy, regardless of birth cohort, and as people age health becomes an increasingly resonant concern, although snack consumption results were most strongly associated between grandparent marital ties in the present study. If life-stage progression does change healthy and unhealthy food consumption over time, future research should target each generational life-stage with age appropriate dietary health improvement strategies. Present day strategies that intervene at the parent and child generations may also consider efforts that reduce fast food consumption attitudes that normalise excessive consumption.

Parent's fast food consumption did not correlate with children's consumption. This may indicate that the parent and child generations preferred different types of fast food meals and parents consumed fast food independently of children. Parent marital-ties showed a smaller correlation than did child siblings, which supports Study 1 suggestion that parents sometimes did not consume fast food when their children did. Combined, Study 1 and 2 results suggest that parents purchase fast food for children that they do not consume themselves. One of the limitations of the present study was the way in which fast food was measured by self-report. Although the study measured virtually all possible types of fast food typically available in Australia, the study may have attracted health conscious participants

[31] Fast food only became widely available from the 1970s (Block et al., 2004).

interested in the topic of "Families, Food and Eating" and these participants' responses may have been distorted by influence from social desirability and impression management efforts. Consistent with this possibility, fruit and vegetables were reportedly consumed at higher recommended levels than the population also reflecting socially desirable responses or a particularly health conscious sample.

Food Life Behaviour & Attitudes and Food Consumption Explained by the Health Belief Model

The possibility that resemblances in food consumption among family members may reflect underlying similarities in attitudes to the link between food and health or consequent disease appears to have received limited attention. According to the Health Belief Model (HBM), the extent that an individual engages in healthy dietary choices may depend, at least in part, on whether risk of diet-related obesity or disease is perceived as something that will affect them; whether the threat of disease can be reduced by dietary means; and whether the cost of any dietary change is outweighed by the likely benefits (Janz & Becker, 1984; Linke, Robinson, & Pekmezi, 2013). It follows that individuals who have health attitudes that link diet with subsequent good health or disease (i.e., diet-health/disease linked attitudes; DHLA) would engage in healthy eating and report dietary behaviours (i.e., diet-health oriented behaviour; DHOB) consistent with their beliefs.

In the context of the current research, scores on a measure of Food Life attitudes that link dietary intake with health outcomes (e.g., the FLQ-SF; Sharp et al., 2013) were expected to correlate positively with fruit and vegetable (i.e., "healthy" food) consumption, and negatively with fast and snack (i.e., "unhealthy") food consumption. Results largely supported DHOB associations although effect sizes ranged from small to medium positive correlations for healthy food and medium negative correlations for unhealthy food. This shows DHOB correlated well with actual self-reported food consumption indicating that it

was validated by the self-reported food consumption data. However, the comparatively lower consistency in the correlation between the DHLA (i.e., a measure of attitude toward the link between diet and subsequent health or disease) and reported food consumption showed that healthy or unhealthy dietary consumption was not strongly linked with beliefs that diet can affect subsequent risk of obesity or chronic disease. Future research should therefore consider strategies to improve knowledge of the link between dietary choices and consequent disease prevention or acquisition. If research focuses on health information linking diet with disease that also reaches the multigenerational family unit, benefits could be achieved for each individual across the lifespan. Having identified the mother in Study 1 as the health gate-keeper within the three-generation family, targeting improved knowledge here may impact the entire extended family network.

The Relationship between Food Life Behaviours & Attitudes and Food Consumption

When Food Life behaviours and attitudes were added to the Generalized Linear Mixed Models (GLiMMs) that combined generation, gender and each consumption outcome variable, results indicated that higher diet-health orientated behaviour scores impacted vegetable and snack consumption, whereas a higher diet-health/disease linked attitudes predicted lower snack and fast food consumption. These results were similar to those reported by Sharp et al., (2013). The negative correlations between scores on DHOB and *unhealthy* food consumption were larger in both studies than significant positive correlations between DHOB and *healthy* food consumption. This suggests that perceptions about the impact of diet on disease focus on the compromising effects of specific unhealthy foods rather than the beneficial effects of healthy food consumption. Identifying the source of these differences requires further research. Recent public health campaigns that have focused on reducing overweight and obesity may have highlighted the importance of "unhealthy" food avoidance. Such campaigns tend to focus on the health impacts of being "fat" and can be very

stigmatizing (Puhl, Peterson, & Luedicke, 2013). A focus on this approach linking diet with obesity is contrary to the reported consumer preference for messages that encourage healthier eating (Lewis et al., 2010).

Future public health campaigns might benefit from a more gain rather than loss focused approach (Bannon & Schwartz, 2006; de Bruijn, Visscher, & Mollen, 2015). Such campaigns would highlight the health gains to be made by compliance with a healthy diet, with weight loss a potential, ancillary benefit to disease prevention. The aim would be to increase adequate fruit and vegetable consumption in Australia from the current low rates of 54% and 7% respectively (ABS, 2014a).

Food Life behaviours & attitudes and healthy food consumption. When the relationship between Food Life Behaviours and attitudes and healthy food consumption were compared between generations and genders, sons, daughters, fathers and grandmothers returned similar, significant positive correlations medium to large in size. Mothers were notably absent from the significant results, indicating healthy dietary behaviours in mothers were aligned with other factors. Previous cross-cultural research has attributed weight concern attitudes to females more so than males, which may explain this outcome (Rozin et al., 1999). Although weight concern was not a focus of this thesis, it could be a worthwhile area for future mother-daughter Food Life behaviour and attitude research.

Significant correlations between scores on attitudes that diet affects health and disease with healthy food were fewer and were apparent in grandmothers, grandfathers, and sons, suggesting that attitudes linking diet with disease were more salient in the older generation than most of the younger generations. With advancing age, it is more likely that an individual's risk for disease increases and some look towards dietary modifications to increase longevity.

Food Life behaviours & attitudes and unhealthy food consumption. When Food Life behaviours and attitudes and unhealthy food consumption correlations were examined between generations, the child and parent generations had significant negative correlations in self-reported healthy dietary behaviours (DHOB) with medium to large effect sizes. Consistent with the Health Belief Model higher Food Life behaviours and attitudes and lower unhealthy food consumption associations were expected. In the parent generation results are consistent with life-stage parenting responsibilities and the likelihood of discouraging children's unhealthy food consumption. Significant negative results with children suggest that parents have socialized their children to associate healthy food attitudes with limited unhealthy food exposure and consumption. The absence of a significant result within grandparents, together with a significant correlation between grandparent marital-ties snack consumption indicates food attitudes within the older generation were not associated with the consumption of fewer unhealthy foods. One of the limitations of this outcome was that over 50 percent of grandparent's fast food consumption were self-reported zero scores (i.e., never consume) therefore consumption was extremely low in this age group.

When comparing total sample food attitude correlations and unhealthy food consumption results between the present study and the FLQ-SF (Sharp et al., 2013), Study 2's results were similar in direction and effect size. Self-reported healthy dietary behaviours (DHOB) was negatively associated with snack consumption and both research studies had medium effect sizes (Sharp et al., 2013). Similarly with fast food consumption, both studies had negative correlations with medium to large effect sizes in DHOB. Diet-health/disease linked attitudes (DHLA) in both studies had small or non-significant negative correlations in snack and fast food consumption (Sharp et al., 2013).

Parents: The Importance of Fathers and Sons.

Mother-child resemblances in food intake are widely reported in the literature and the

present study also confirmed mother-child resemblances in healthy food consumption. Aside from known factors, mediating psychological mechanisms of influence within the mother-child's relationship with food consumption are being investigated in contemporary published research (Beydoun & Wang, 2009; Prichard et al., 2012). The present study had paradoxical results when reporting the relationship between mothers' Food Life behaviours and attitudes and the food consumption of offspring. More positive attitudes to healthy food among mothers were associated with lower fruit consumption among daughters and higher fast food consumption, without any significant mother-son associations noted. Mother's own Food Life behaviours and attitudes did not correlate with any food consumption outcomes, whereas father's own Food Life behaviours and attitudes had strong links with their own higher vegetable, and lower fast food consumption. Although the mother-child dyad did share significant resemblances in healthy food consumption, the mechanism underlying this strong association was evidently not Food Life behaviours and attitudes.

From the previous literature it has been shown that mothers and daughters have strong resemblances in food consumption, however less is known about the resemblances between fathers and sons, or fathers and daughters (Beydoun & Wang, 2009; Mallan et al., 2013; Prichard et al., 2012; Wroten et al., 2012). When examining the effect of food attitudes as a possible mechanism of influence on the family diet, father's higher scores on diet-health/disease linked attitudes (DHLA) correlated significantly with the healthy food consumption of children, and effect sizes were large. It was expected that mother's healthy food attitudes would have had a positive effect on the food consumption of children, or at least of daughters, however only father-son and father-daughter results were significant. Given the overall lower incidence of significant diet-health/disease linked attitude results when compared to diet-health orientated behaviour, the father-child outcomes were surprising. Since less is known about the father-child dyad's association with food

consumption (Mallan et al., 2013), this represents a novel intergenerational finding about family dietary behaviour that may be worth exploring further in future research. Generalized Linear Modeling (GLiM) using a Generalized Estimating Equation (GEE) analysis confirmed earlier father-son results, because the odds of healthy food consumption were twice as great in males with higher food attitude scores than females with similarly high food attitude scores. Significant results showed interactions in male's fruit consumption with diet-health/disease linked attitudes. With a one standard deviation increase in DHLA, the odds of males meeting fruit consumption recommendations increased from 1.31 to 8.58 times, whereas female odds remained fairly constant with changes in standard deviations above and below the mean. Vegetable consumption with diet-health oriented behaviour was lower in males than females, however remained similar and negative indicating both genders rarely met recommended guidelines for vegetable consumption. This is consistent with preceding results and previous findings that the declining rate of vegetable consumption in Australia is concerning (ABS, 2006, 2014a). Utilizing existing father-child influence on healthy food consumption by engaging the food attitudes of fathers could be of benefit to children in future family food related research.

If socialization is the link between attitude formation and subsequent behaviour (Baumrind, 1973; Maccoby, 1992), father's food attitudes may play an important role in influencing children's dietary behaviours. The father-child findings not only support HBM theory's directional hypotheses (i.e., that higher diet-health/disease linked food attitudes would be associated with higher healthy, and with lower unhealthy food consumption), they also raise the question whether father-child modeling has been a mediating factor (Bandura, 1977). Since father's diet-health/disease linked attitudes were associated with their own higher consumption of healthy food this increases the opportunities for father-child modeling to occur. If confirmed by future research, this outcome may provide new insight into one of

the mechanisms behind resemblances in healthy and unhealthy food consumption between fathers and children within families.

Limitations

Correlations in the present study need to be interpreted with consideration of the nature of the restructured data. Although data were analyzed at the family level, each family did not have the same composition; most families were comprised of five family members and ranged from two to seven family members per family. Future research could consider recruiting families with identical structure for more powerful analysis. Improvements could be made with greater numbers of participant families, where the age range of children could be broken down in order to examine differences or similarities between childhood developmental stages (e.g., early childhood, middle childhood, adolescence) and the influences of parents at each stage. To avoid the potential for social desirability bias, improvements to the food consumption measures could also be considered.

The GLiMMs used in the present study were relatively new statistical procedures and did not allow for the prediction of exact variance explained in the dependent variables as one might have expected using classical statistical procedures such as OLS multiple regression.

Finally, the present study recruited Australian participants from English speaking backgrounds. Future research could compare similarities and any differences in Food Life behaviours and attitudes with food consumption that may exist within other multicultural Australian ethnicities, or cross culturally, as has occurred in previous research (e.g., Rozin et al., 1999).

Conclusions

In three generation families both parents were shown to impact upon the healthy food consumption of children. The mother's importance was acknowledged by mother-daughter fruit consumption resemblances, and mother-child resemblances in vegetable consumption.

The importance of fathers' attitudes supporting dietary influences on subsequent health or disease and influences on children's healthy food consumption was shown by the correlation between fathers' food attitudes and fruit consumption in sons, and vegetable consumption in daughters. If we accept that fathers can have a positive impact on children's healthy dietary behaviours, then their food attitudes could be better utilized as a mechanism of influence within families receiving dietary guidance. Future research could also build on the limited psychological literature on father-son and father-child predictors of dietary behaviours by investigating Food Life behaviours and attitudes further.

Grandparent marital-ties shared a lower incidence of fast food consumption, however, shared strong resemblances in snack consumption. This contrast in generations could be considered as life-stage progression in two ways. Firstly, that the current child and parent generations have the potential to consume fast food into old age which could present greater disease rates in the older population over time. Secondly, that shared snack consumption associations within the grandparent age group may reveal possible relaxation of healthy food habits with age. In an already ageing population, Australian dietary research should consider these potential health concerns when developing future dietary interventions.

As a follow up study to the current study, Study 3 proposes to examine differences in healthy and unhealthy food consumption in response to tailored family health history feedback based on whether participants are at average or above-average risk of the four chronic diseases: colorectal cancer, breast cancer, heart disease and type 2 diabetes. Study 3 will examine the same outcome variables and the same family dyads within three generation Australian families.

CHAPTER 4: STUDY 3 - PART A

Title: The impact of family health history and disease risk information on motivation to change dietary behaviours within three generation Australian families.

Background

In Study 1, dietary behaviours were explored through interview with three generation Australian families. The aim was to identify the contributions from various family members to typical dietary intake. Consistent with past research, comments from family members emphasized the importance of the mother in disseminating health information within the nuclear family network and beyond to the grandparent generation, even where these family members lived in separate households.

Building on the major theme emerging from Study 1, Study 2 used a cross-sectional survey design to investigate the association between food attitudes and food consumption within families. Two measures of attitude to food and health were operationalized; diet-health oriented behaviour and diet-health/disease linked attitudes. The associations between these measures was compared across dyads. As predicted, within the sample as a whole, a higher level of positive and supportive attitudes that linked food to health were correlated with higher levels of healthy food, and lower levels of unhealthy food, consumption. Study 2 also confirmed the important role of mothers identified in Study 1 and, consistent with previous research, revealed moderate to strong healthy food consumption associations between mother-child dyads (e.g., Beydoun & Wang, 2009; Prichard et al., 2012; Wroten et al., 2012). Novel findings in Study 2 showed that diet-health/disease linked attitudes were strongest within father-child dyads in predicting healthy food consumption.

Chapter Introduction

This chapter presents Study 3 Part A[32]: an experimental study that utilizes the findings of Studies 1 and 2 to develop and test an intervention that targets concern about the link between diet and health risk to motivate intention to prepare and eat healthy food in the family setting. The primary aim is to target the principal gatekeeper of diet with the family, identified in Studies 1 and 2 as the mother, and to obtain information about familial risk for chronic disease that can be utilized to motivate behaviour change directly from the families themselves. The intervention consists of a program previously validated for the same purpose in the USA; The Families Sharing Health Assessment and Risk Evaluation (*Families SHARE*) workbook[33].

Study 3 aims to motivate healthy dietary behaviour change over time through exposure to the workbook and compare changes in food intake intentions and behaviour, before and after exposure, with changes in a no intervention control sample of families. This chapter first provides a brief overview of the health behaviour theory underpinning this thesis and how it relates to predicted changes in actual and intended health action in response to the Families SHARE workbook. The chapter will then explain the components of the Families SHARE workbook, which has been evaluated in the US (Koehly et al., 2015) and adapted in the research reported in this dissertation for use by Australians. As described above, the goal of the intervention is to improve intended and actual healthy food consumption and decrease unhealthy food consumption.

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[32]In order to focus solely on family health history and disease-risk factors in the present study, Study 3 Part B will separately present the 'Results' and 'Discussion' sections for generation and gender differences over time, along with any effects of the diet-health behaviour and diet-health-disease linked food attitude variables that were observed.

[33] *Families SHARE* in the present study is an Australian workbook based on the *Centre's for Disease Control's Family Healthware*™ (de la Haye, de Heer, Wilkinson, & Koehly, 2014b) which was subsequently evaluated in the US (see Koehly et al., 2015). For a copy of the workbook used in the present study see Appendix 4.A.

Dietary behaviour within families as risk factors for chronic disease. As outlined in Chapter 1, one way of reducing the modifiable risk factors that contribute to chronic disease is by motivating adherence to dietary behaviours consistent with the Australian Healthy Eating Guidelines (National Health and Medical Research Council, 2013). By engaging with families in disease prevention initiatives, rather than individuals, it may be possible to effect dietary behavioural change across a number of people and over several generations simultaneously. Research has shown that when there is commitment to shared intentions, individuals can act interdependently using collective free will to achieve healthy outcomes (Bandura, 2001). Interventions targeting dietary behaviour change that utilize the collective influence of family relationships are likely to have a wider impact than those that target individuals alone.

In a systematic review focused on fruit and vegetable consumption among children and adolescents, a need was identified for more theory based, international, longitudinal and multi-level studies that examine factors contributing to variation in healthy food consumption (Pearson, Biddle, & Gorely, 2009). Promoting the benefits of reducing the intake of unhealthy food to combat obesity and chronic disease remains a topic of much research in the obesity and disease prevention literature (e.g., NHMRC, 2009b; Nishida, Uauy, Kumanyika, & Shetty, 2004; NVDPA, 2012; The G. B. D. Obesity Collaboration et al., 2014). However, despite the government and health providers providing regular information to Australians about the importance of adequate daily intake of fruit and vegetables (e.g., Aune et al., 2016; NHMRC, 2013a), more than 90% of the Australian population currently consume fewer vegetables, and 42% consume fewer fruits than recommended (ABS, 2014a; NHMRC, 2013a). Increasing healthy food consumption is of paramount importance in this country if disease prevention aims are to be achieved.

Motivating families to change dietary behaviours utilizing family health history

and disease risk information. Risk for chronic disease plays an important role in disease risk assessment (Hovick, Wilkinson, Ashida, de Heer, & Koehly, 2014; Valdez et al., 2010), and provides a potentially useful strategy for triaging families and individuals at increased risk of disease. Identifying "at risk" individuals can be a motivating factor for some people to engage in preventative health behaviours even when asymptomatic (Chang et al., 2011). Research has demonstrated the provision of information on family health history and any associated risk can be an effective motivational intervention strategy for health behaviour change, including diet (Koehly et al., 2015; Ruffin et al., 2011; Vernon, 1999). Disease diagnosis in close family members has also been shown to motivate healthy dietary behaviour changes in other family members (Beagan & Chapman, 2004). For children, family health history and disease risk information may reduce the health burden if adults within families act early with appropriate dietary modifications that meet NHMRC guidelines and recommendations (Valdez et al., 2010). For families who are exposed to information about familial disease risk, responses may vary according to their readiness to change behaviour. Moreover, exposure to risk information may provide the necessary stimulus to move individuals within the family to a more serious consideration of lifestyle changes. Readiness to change (i.e., movement to the 'preparation' Stage of Change as described in the Transtheoretical Model; Prochaska et al., 1992) is a necessary precursor to any health behaviour change.

Brief Theoretical Background

The Transtheoretical Model (TTM): Stage of Change. Changes in the dependent variables, healthy and unhealthy food consumption, will be compared between the experimental and control groups across time in Study 3, and between various disease risk categories. Since responses at follow up are likely to depend upon participant's readiness to change dietary behaviour at baseline, participants will be categorized according to the

Transtheoretical Model at baseline (TTM; stages of change (SoC); Campbell et al., 1999; Godinho, Alvarez, & Lima, 2013; Prochaska et al., 1992; Sarkin et al., 2001) and then examined at follow up in relation to self-reported fruit and vegetable consumption.

The five stages of change, detailed further in Chapter 1, are pre-contemplation, contemplation, preparation, action, and maintenance. Study 3 will examine whether exposure to familial disease risk information will motivate individuals to a more serious consideration of dietary change when compared to individuals in the control condition (see the conceptual model in Figure 1). For the complete Australian version of the Families SHARE workbook in the present study see Appendix 4.A. The basis upon which this information may impact both cognition and behaviour can be found in a range of health psychology theories. These are detailed below.

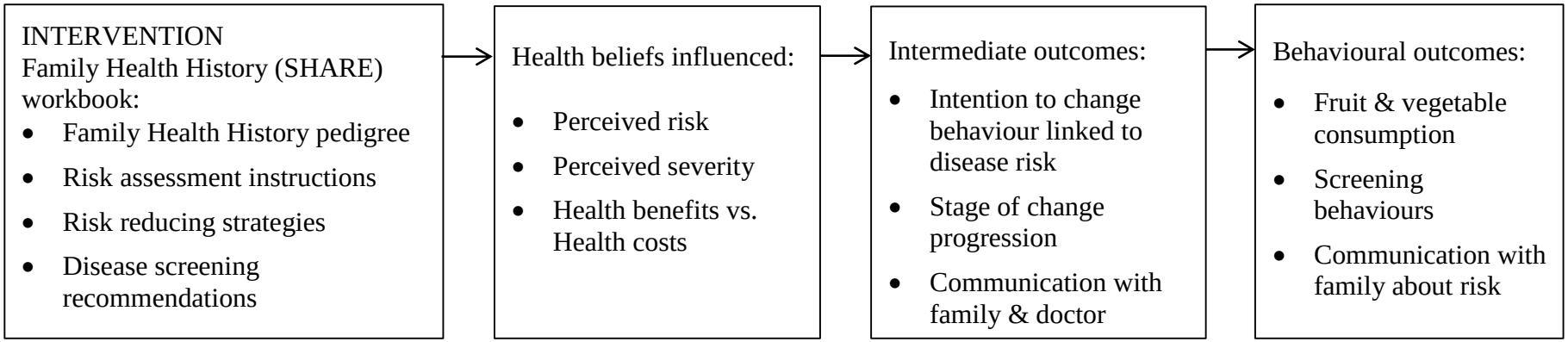


Figure 1. Conceptual model showing how tailored family health history, risk status and recommended risk reducing strategies would result in behavioural change.

Social Cognitive Theory (Bandura, 2001) suggests that by engaging with the family in disease prevention initiatives, rather than individuals, it may be possible to effect dietary behaviour change across a number of people and over several generations simultaneously. However, it is not clear whether interventions targeting dietary behaviour change utilizing the collective influence of family relationships are likely to have a wider impact than those that target individuals alone.

The Health Belief Model (HBM) has been found to be particularly useful as an explanatory model for short-term health behaviour change (i.e., up to six months post intervention) (Janz & Becker, 1984), and recent evidence has provided some evidence of validity for longer term healthy lifestyle behaviour change (Linke et al., 2013). Briefly restated, the HBM proposes that depending on the trade-off between perceived costs and benefits, individuals can be motivated to take health action when there is a perceived risk of developing serious disease (Conner & Norman, 1995; Rosenstock, 1974; Rosenstock, Strecher, & Becker, 1988). Evidence suggests that providing tailored disease risk feedback impacts risk perceptions for heart disease, diabetes and other chronic diseases (Hovick et al., 2014; Vernon, 1999). Recent research suggests that increasing risk perceptions for more than one disease is more effective that for one disease only (Hovick et al., 2014). In a systematic review of randomized control trials using personalized family health history to promote lifestyle behaviour change, those studies using visual risk feedback were described as the most effective (French, Cameron, Benton, Deaton, & Harvie, 2017). The limitations identified were studies that lacked theory, did not target self-efficacy or response-efficacy, and few behaviour change techniques were utilized. Study 3 examines the effectiveness of the five-part Families SHARE workbook comprising: tailored family health history indicating individual disease risk, disease information, recommended healthy lifestyle habits, and disease screening information. Using the workbook as a motivational tool, the present study

expects change in actual and/or intended dietary behaviours within three generation Australian families.

Summary

Previous research has shown the effectiveness of family health history information in conjunction with risk reducing strategies to motivate health-linked behaviour change in individuals and families (Hovick et al., 2014; Ruffin et al., 2011). However, dietary behaviours occur in the context of, and are very influenced by, the family (Contento et al., 2006; Feunekes et al., 1998; Patrick & Nicklas, 2005; Prichard et al., 2012). In addition, it is possible that influencing perceived disease risk in one family member, particularly the person who gate-keeps health, will impact the full family network (Bandura, 2001; Hendriks et al., 2012). The Health Belief Model (HBM) (Janz & Becker, 1984) will be applied to explain the mechanism of expected behaviour change in the present study. That is, when identifying families at average or above-average disease risk, it is anticipated that perceived familial risk and the severity of the four chronic diseases (i.e., breast cancer, colorectal cancer, heart disease, and type 2 diabetes) will motivate families to see the benefits of disease preventative health action.

The Present Study

Study 3 is theoretically based on a health behaviour model (the HBM) and uses an intervention (the Families SHARE workbook) as a motivational tool to measure any impact over time on actual and/or intended healthy and unhealthy dietary behaviours within three generation Australian families. Any variation in food consumption that may exist between disease risk groups (i.e., either at 'average risk', 'above-average risk' or 'diagnosed-with' disease) will be examined to determine behaviour change over time. The progress through participants' stage of readiness to change health behaviour as measured by the Trans-Theoretical Model (TTM) will be linked to intended behaviour change, whilst also

controlling for participants in the action and maintenance stages of change, who already meet current dietary recommendations.

Aims

Changes in food consumption intentions and behaviour over time will be compared between families in the experimental and control groups and on the basis of identified disease risk. The study aims to investigate whether changes in consumption occurs post-intervention, in order to reject the null hypothesis that 'no change in food consumption will occur over time'. Study 3 Part A, will investigate the following hypotheses.

Hypotheses

1. It is hypothesised experimental group families (i.e., whose mothers are provided with the Families SHARE workbook incorporating their unique familial risk for chronic disease) will
    a. increase intended and actual fruit and vegetable consumption, and
    b. decrease intended and actual fast food and snack consumption more than families in the control group who will not be exposed to the Families SHARE intervention (until the conclusion of the study).
2. After controlling for participants identified at the 'action' or 'maintenance' stage of healthy dietary behaviour at baseline, participants in the experimental condition identified at above-average disease risk are predicted to progress to a higher stage of readiness to change healthy and unhealthy food consumption at follow up, than participants at average disease risk.
3. Finally, it is predicted that participants within the experimental condition who are at identified at above-average risk will increase healthy food consumption and decrease unhealthy food consumption over time, when compared to participants identified at average risk. Participants within the experimental condition will be examined within three

groups (i.e., individual at risk, family member at risk, and mother at risk); and at three levels (i.e., above-average risk, average risk, and diagnosed with any of the four chronic diseases). The procedural flow diagram is presented in Figure 1.

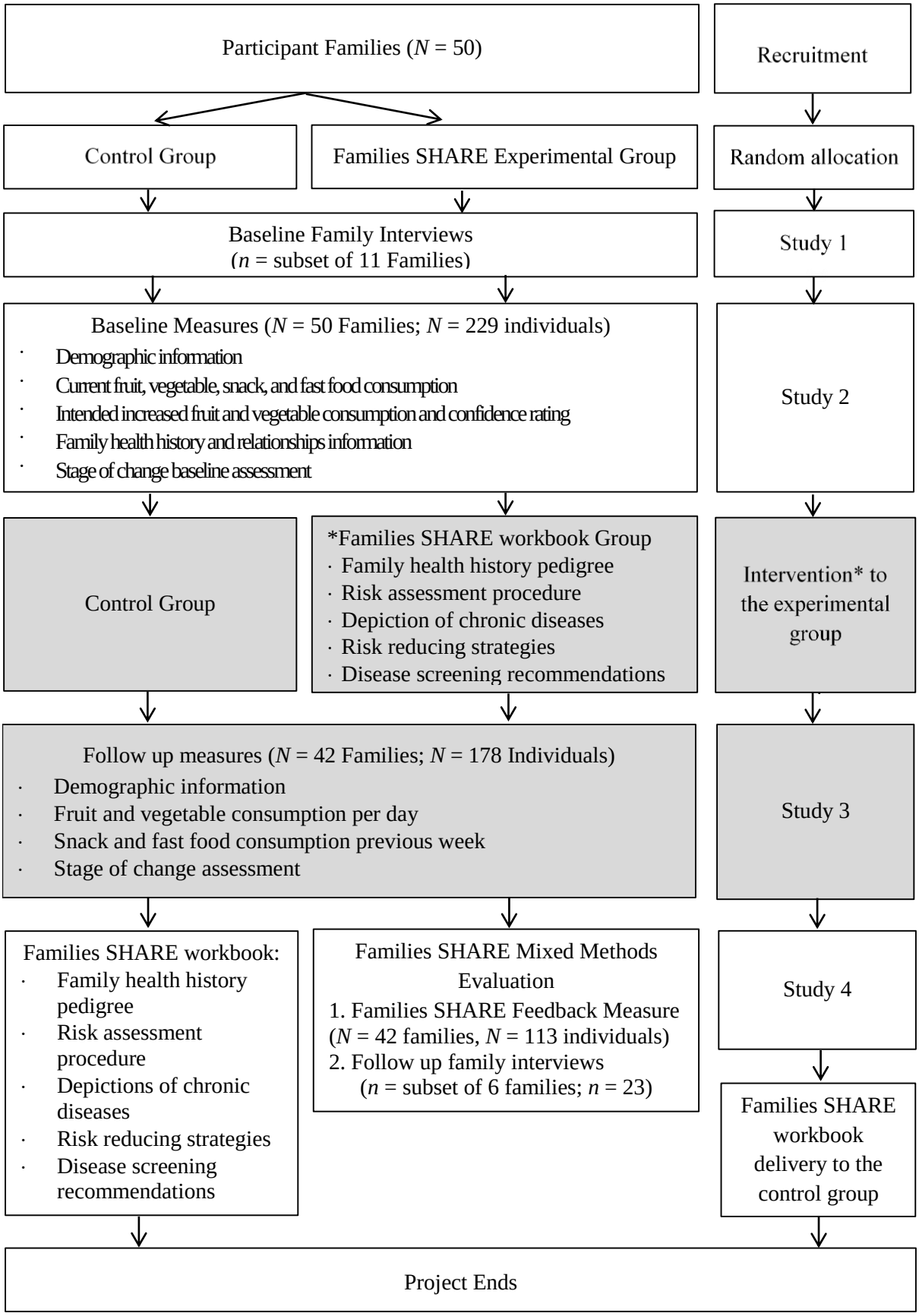


Figure 1. Procedural flow diagram showing Study 3 in relation to all studies.

Method

Participants

Study 3 participants comprised 178 individuals within 42 Anglo-Australian three generation families (retained from the 229 individuals within 50 families in Study 2). Of the 178 participants in the present study, 39% were male (*n* = 69); and 61% were female (*n* = 106); 37% were children (*n* = 66); 35% were parents (*n* = 62); and 28% were grandparents (*n* = 50). Individuals ranged in age from seven to 89 years (*M* = 40.09, *SD* = 24.29)[34].

The Model Design

Study 3 investigated individuals (*N* = 178) and whole families (*N* = 42) pre- and post-intervention (i.e., at baseline and six months later) on changes in healthy and unhealthy food consumption (i.e., fruit, vegetable, snack, and fast food), using a within-subjects factor with two levels (time 1 and time 2). To assess effects on the dependent consumption variables, the following independent variables were included into the model designs: condition: (intervention or control; between-subjects factor with two levels), individual disease risk (either 'average', 'above-average' or 'diagnosed' for each of the four diseases colorectal cancer, breast cancer, heart disease, and type 2 diabetes; between-subjects factor with three levels x 4); family disease risk (either average, above-average, or diagnosed for each of the four diseases; between-subjects factor with three levels x 4); mother's disease risk (either average, above-average, or diagnosed for each of the four diseases; between-subjects factor with three levels x 4) (Heck, Thomas, & Tabata, 2014a).

Materials

Study 3 was a cluster randomised control trial (RCT) with an intervention at three

[34] As occurred in Study 2, the author/candidate conducted primary data collection and analysis in the present study.

months, and surveys at two time points: one at baseline and another at follow up six months later (see the participant flow diagram in Figure 2). As in Study 2, a questionnaire containing a number of measures was constructed and offered either as an online survey or as a paper survey that was posted to participants and returned in a reply-paid envelope. There were two versions, one for adults and an abridged age-appropriate version for children aged 7 to 17 years inclusive. The entire questionnaire took no more than one hour to complete. The measures summarised in Figure 2 comprised the complete adult questionnaire used in the present study. For the adult and child versions of questionnaire please see Appendices 4.B (adult) and 4.C (child).

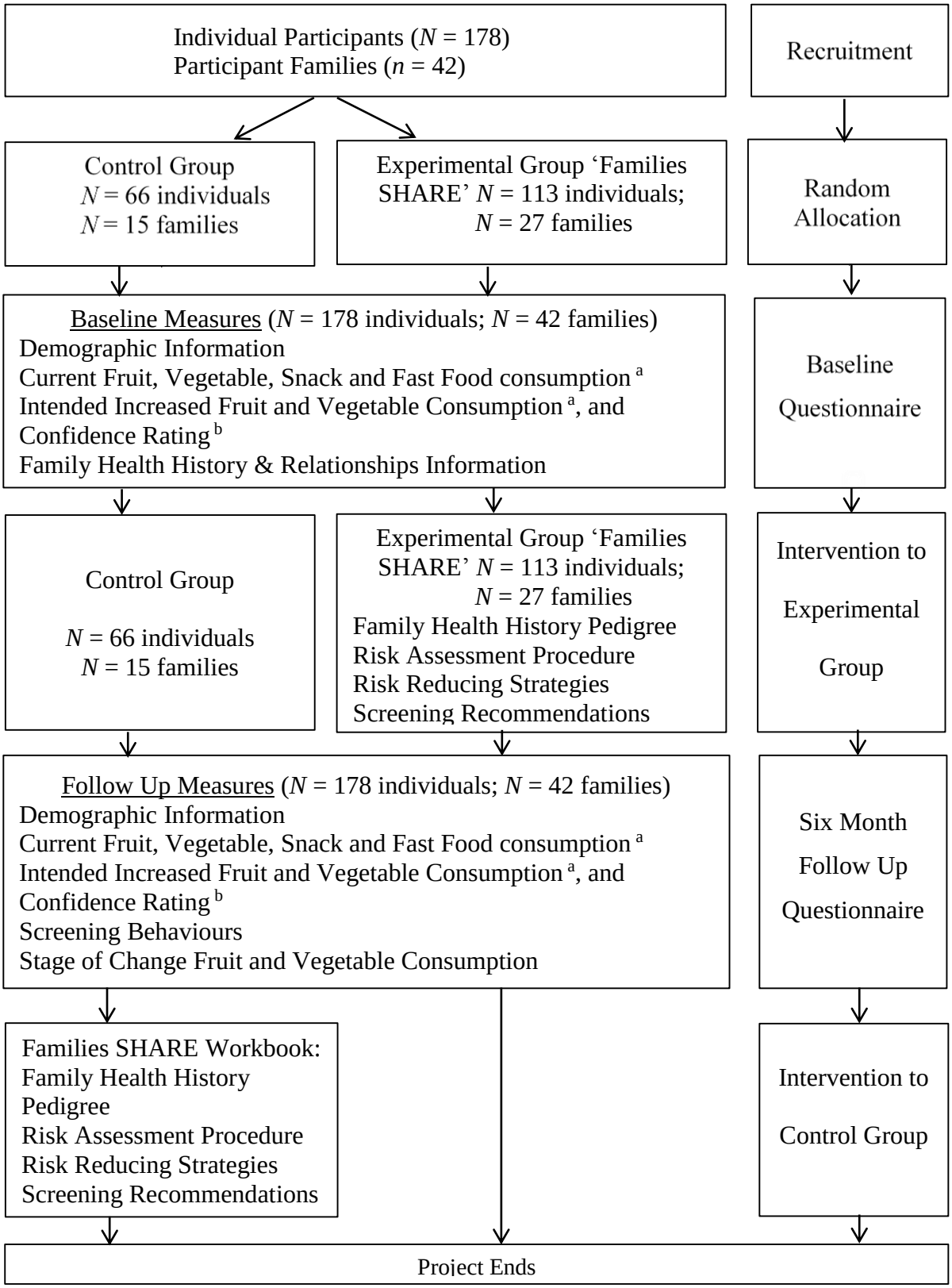


Figure 2. Study 3: Procedural flow diagram. [a] Current and intended food consumption together indicated Stage of Change. [b] Confidence rating indicated self-efficacy.

Procedure

At baseline, whole families were randomly allocated to either an experimental group receiving the Families SHARE workbook (consisting of tailored family health history information) at three months, or to the control group receiving the same workbook and information only at the conclusion of the study. Allocation was conducted by a Research Assistant, who was not involved in the later data analysis. Each individual's risk for each of the four chronic diseases, colorectal cancer, breast cancer, heart disease, and type 2 diabetes was assessed and categorized as at 'average risk', 'above-average risk', or 'diagnosed' on the basis of family health history information provided by participants at baseline. At three months after baseline measures, the mother in each family within the experimental group received a workbook titled "*Families SHARE*" comprising; descriptions/diagrams of, and risk factors for, each of the four diseases and information on preventative lifestyle behaviours for each, as recommended by the NHMRC, and as outlined below in the 'Materials' section (for an example see Figure 3).

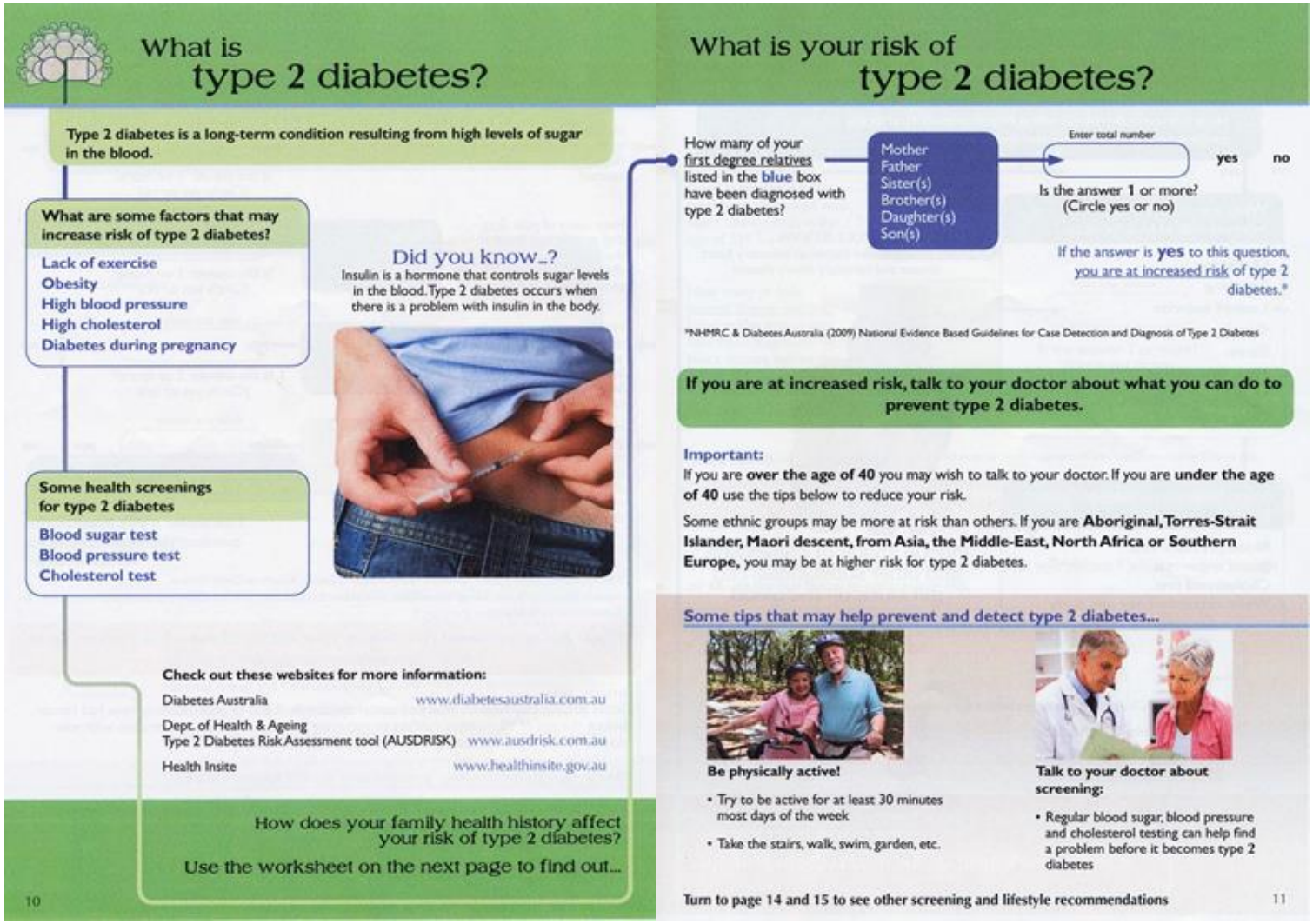


Figure 3. An example from the "*Families SHARE*" workbook showing type 2 diabetes. On page 10: disease description, risk factors, and health screening. On page 11: self-risk assessment, and information on preventative lifestyle and screening behaviours.

Using the workbook as a guide, individual family members were able to determine whether they were at either ‘average’ or ‘increased risk’ for the four diseases on the basis of their family health history pedigree, presented as a diagram (see Figure 4). Diagnosed individuals, as determined by information provided by the family, were indicated on the pedigree diagram.

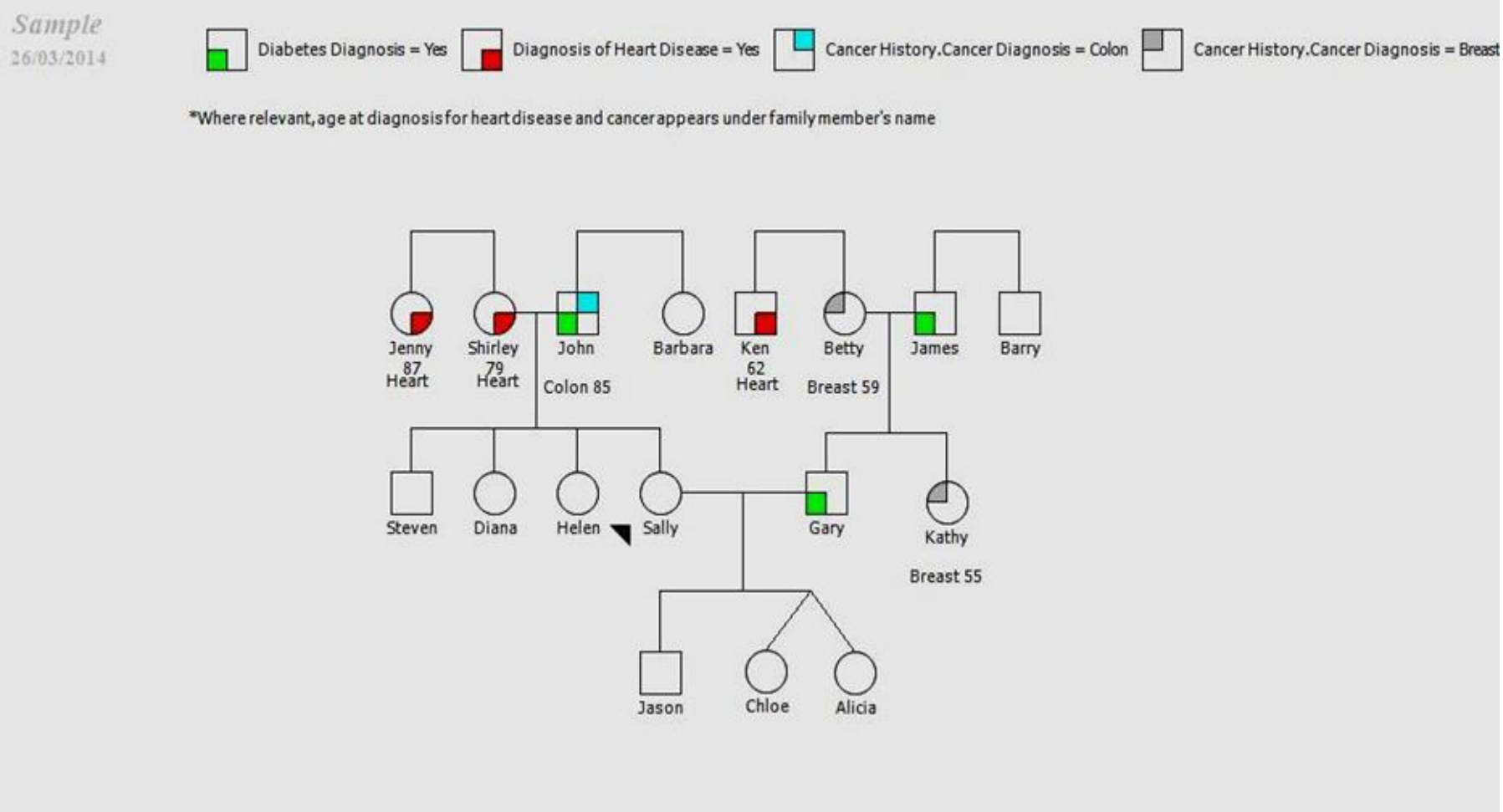


Figure 4. Sample pedigree diagram depicting family members diagnosed with type 2 diabetes, heart disease, colorectal cancer, breast cancer and age of diagnosis if known.

Families in the control group each received the same Families SHARE workbook and family health history pedigree diagram with their unique familial disease risk at study end. Participants completed a six month follow up questionnaire containing similar items to those at baseline, in addition to an evaluation of the family health history intervention (experimental group only).

Sampling procedures.

With the exception of eight families who left the Project prior to its completion, participants were the same as those recruited for Study 2 during the time period from the 7th May 2012 to the 6th March 2013.

Measures. The majority of Study 3's measures were identical to those provided in Study 2, however, Study 3 included some additional constructed items that enabled tailored family health history feedback, and stage of change categorization on the four food consumption variables. Follow up measures repeated those carried out at baseline, with certain items excluded because they were of no additional benefit if replicated (e.g., family health history and relationships information, education level). Therefore, any measures used in the present study that were described fully in Study 2 are only briefly restated in this chapter, while any additional measures not mentioned previously will be fully described in the paragraphs that follow.

Baseline measures. The baseline measures included the following: demographic information; self-reported fruit, vegetable, snack and fast food consumption; family health history information for colorectal cancer, breast cancer, heart disease, and type 2 diabetes; intentions to change dietary behaviour and confidence rating; and perceived disease risk.

Family health history information. To determine family pedigrees and individual risk assessment, adult participants were asked whether they had been told by a doctor that they had been diagnosed with colorectal cancer, breast cancer, heart disease or type 2 diabetes,

and if yes, at what age. To formulate a tailored family health history pedigree diagram for each family, and to determine risk assessments for each individual, all parents in the study were asked to complete a family health history questionnaire to address the maternal and paternal sides of each family. The first item asked whether any male family members had been diagnosed with breast cancer. The second item asked participants to indicate parents' first names, whether their parents had been diagnosed with any of the four diseases, and if yes, at what age were they first diagnosed. Each disease was listed with check boxes underneath to indicate the option, "yes," "no," "don't know," and "If yes, at what age? (in years)." In order to provide detailed family health history information to participants, the same family health history questions were repeated requesting information for: "children", "siblings", "aunts and uncles on the mother's side," "aunts and uncles on the father's side," and "additional people" if insufficient space was provided earlier, to ensure all family members were captured.

Components of the family health history and disease risk intervention: The Families SHARE workbook. The Australian Families SHARE workbook was an A4 sized, 16 page, colour printed booklet based on the Center's for Disease Control's Family Healthware™ (Koehly et al., 2015; O'Neill et al., 2009) titled "Families SHARE, Revised March 2013: Sharing Health Assessment and Risk Evaluation." For the complete workbook see Appendix 4.A. Attached within each workbook was an introductory letter from the project leader/researchers (see Appendix 4.D), a family health history pedigree diagram depicting unique familial disease risk for the four diseases (i.e., breast cancer, colorectal cancer, heart disease, and type 2 diabetes; see a sample pedigree in Appendix 4.E), and a copy of the "Patient and family fact sheet: Your family history" (contained within: Barlow-Stewart, Emery, & Metcalfe, 2007) (see Appendix 4.F).

The families SHARE booklet itself was comprised of the following sections.

1. Page 1: titled "*Sharing Health Assessment and Risk Evaluation*" contained a preamble to interpreting a family health history tree, and the disease risk worksheets for four diseases colorectal cancer, breast cancer, heart disease, and type 2 diabetes;
2. Page 2: contained an illustrated set of step-by-step instructions depicting, "*How to Read a Family Health History Tree*";
3. Page 3: provided an "Example Family Health History Tree - Part 1"; and instructions;
4. Page 4: "Your Family Health History Tree – Part 2"; provided information on reading, updating and learning from the pedigree diagram.
5. Page 5 was titled, "*Disease Risk Worksheets and Recommendations*" and provided an introduction to the four diseases that followed;
6. Page 6: was titled "*What is colorectal cancer?*" and provided an illustrated description of colorectal cancer, lifestyle factors that contribute to increased risk of colorectal cancer, health screening measures, and a list of Australian websites for further information;
7. Page 7 was titled "*What is your risk of colorectal cancer?*" and provided a worksheet for participants to use with their own family health history pedigree diagram to determine their own unique disease risk; lifestyle behaviours that may help prevent colorectal cancer, and health screening recommendations (NHMRC, 2005);
8. Pages 8 through to 13 presented information relating to breast cancer (National Breast and Ovarian Cancer Centre NBOCC, 2010; NHMRC, 1999), heart disease (National Vascular Disease Prevention Alliance, NVDPA, 2012),

and type 2 diabetes (NHMRC, 2009b), and in exactly the same format as described for colorectal cancer;

9. Page 14 was titled "*Healthy Recommendations*" and provided information on healthy lifestyle behaviours attributed to reducing one's disease risk (e.g., "*eat plenty of fruits and vegetables*") (NHMRC, 2006, 2009a, 2013a, 2013b);
10. Page 15 outlined the various screening recommendations for individuals identified at either "average-" or "above-average-risk" for each of the four diseases (NBOCC, 2010; NHMRC, 1999, 2005, 2009a; NVDPA, 2012); and finally,
11. Page 16 was titled: "*Sharing Your Family Health History*" which encouraged participants to share the workbook with family, friends and their doctors; and also provided contact details for individuals seeking further information regarding other diseases that were not included in the workbook.

Follow up measures. At follow up baseline measures were repeated as follows: demographic information (including height and weight for BMI), self-reported fruit, vegetable, snack and fast food consumption, intentions to change dietary behaviour and confidence rating, and perceived susceptibility to the four diseases. Additional measures at follow up included:

Stage of readiness to change dietary behaviours. Based on the Transtheoretical Model (stage of change) (TTM), a questionnaire was created to analyse participant's stage of readiness to change on each of the four consumption variables; fruit, vegetables, snacks, and fast food (Armitage, Sheeran, Conner, & Arden, 2004; Campbell et al., 1999; Prochaska et al., 1992; Sarkin et al., 2001).

Intended dietary changes. To assess intentions to modify dietary behaviours (i.e., 'contemplation' stage of change), questions were asked firstly about whether changes to

some aspects of lifestyle were being contemplated (e.g., "*Thinking about your lifestyle, are you currently contemplating changing some aspects?*"), if no, participants moved to the next section, if yes, participants were asked which aspects they were contemplating changing (i.e., "*What aspects of your lifestyle would like to change?"*). If yes, participants were asked to select what they were considering from the options[35] provided, (e.g., *"Yes, I would like to increase my fruit and vegetable consumption"*); if no, participants could respond by selecting "no" (e.g., "*No, I am happy with my current level of fruit and vegetable consumption*). Participants' confidence that their intentions would be carried out were measured in order to elicit self-efficacy, (e.g., "*If yes, how confident are you that you will increase your fruit and vegetable consumption?*"), by using a seven-point Likert scale ranging from 1 ('*Not at all confident'*) to 7 ('*Very confident*'). See the questions in Appendices 4.B and 4.C, 'Section 1 - Lifestyle', and 'Section 2 - Intended Lifestyle Changes').

Analysing stages of change (SoC) in food consumption over time. From the items in the questionnaires at time one (T1) and time two (T2) that assessed participant's current fruit and vegetable consumption, intended readiness to change dietary behaviours, and confidence in carrying out their intentions, movement in stage of change over time were evaluated. The data preparation process in creating SoC-by-consumption variables for healthy food is detailed in Appendix 4.G. Procedures were utilized as described in Heck et al., (2014a). In brief, the variables were created according to the SoC lifestyle questionnaire items in combination with NHMRC healthy food consumption guidelines that indicated whether fruit and vegetable consumption recommendations were either 'met' (met = 1), or 'not met' (not met = 2). The outcomes were coded numerically, (e.g., 1, 'Baseline fruit pre-contemplation' through to 5, 'Baseline fruit maintenance' SoC) for fruit and vegetable consumption

[35] Several options in the questionnaire were not included in this thesis. For example, alcohol, smoking.

separately, and at two separate time points (i.e., at baseline and follow up).

Family health history workbook (Families SHARE) evaluation. Participants in the treatment condition were asked to complete several questions about their experience of using the Families SHARE workbook. Questions asked whether individuals were able to assess their own degree of risk for the four diseases and to identify if they were at increased risk. The response options were “yes”, “no,” and “don't know.” Participants were then asked to indicate if the workbook had been shared with a GP, other health care provider, family member, friend, or other. They were also asked whether their family health history pedigree had been updated in any way after receiving it, and finally, whether any health screening for the four diseases had occurred in the six months preceding follow-up.

Randomization and reliability:

Study 3 was a cluster randomized control trial (RCT) where families were the unit of randomization and individuals were the unit of analysis. Fifty whole families, of which 42 completed follow up measures, were independently and randomly allocated to either the treatment or control condition upon entering the study pre-trial. A simple randomisation table was created by a Research Assistant using Microsoft Excel and each family that entered the study was subsequently allocated to the next available randomised condition provided within the table (Christie, 2004). Timing of the distribution of follow up measures were equidistant for each family, however, some variability occurred with the return of follow up measures by participant families. Therefore, statistical analyses that accommodated variable time differences were utilized. For the reasoning behind longitudinal modelling in nested data analysis see Appendix 4.H.

Results

Data and Statistical Analysis

The mixed model, repeated measures, multilevel design used longitudinal modeling

techniques as described by Heck et al. (2014a) to manage the clustered longitudinal data that controlled for the nesting effect of families. The rationale for using Generalized Linear Mixed Models in clustered data analysis was outlined in Study 2 (Chapter 3, see Appendix 3.D). Study 3 grew in complexity with the added effects of time, of condition (intervention versus control), of disease risk, and stages of change (SoC) variables to the study[36]. Each test was appropriately chosen for the outcome variable that was being analysed (using procedures described in Elhai et al., 2008). In short, multilevel modeling accurately analysed each individual separately, whilst simultaneously analysing all clustering (also termed 'nesting'), groups, and time contexts within the same model (Heck et al., 2014a). As in Study 2, the alpha value was set at $p < .05$, while modeling analyses adjusted for disproportionate sampling and cluster sampling to avoid over inflating the type 1 error rate (Heck et al., 2014a). IBM SPSS 22™ software (IBM Corporation, Released 2013) was used for all analyses.

Characteristics of the Sample

There were 42 three generation Australian families that completed the current study which represented an 84% retention rate between baseline (N = 216) and follow up (N = 178). Eight families dropped out of the follow up study for various reasons, including overseas travel, family crisis, or ageing family member's serious ill health. There were 71 males (40%) and 107 females (60%) in Study 3. As in Study 2, Study 3's families were grouped depending on their family relationship to the nodal child[37]. They were: grandparents (28%, n = 50) who ranged in age from 60 to 89 years; parents (35%, n = 62) who ranged in age from 34 to 57

[36] Refer to Appendix 4.G for stage of change data preparation and Appendix 4.H for longitudinal modelling reasoning.

[37] As in Study 2, individuals within families were identified according to their relationship to the nodal child (e.g., parent, grandparent, siblings up to the age of 25) and when specifying gender, as grandmothers, grandfathers, mothers, fathers, daughters, or sons.

years; and children and their siblings (37%, *n* = 66) who ranged in age from seven to 26 years. Family structure varied according to the number of children in each family. They comprised families with one child (48%, *n* = 20), two children (48%, *n* = 20), one family had three children (2%, *n* = 1), and lastly one family had four children (2%, *n* = 1). There were 27 families in the experimental condition (63%, *n* = 113), and 15 families in the control condition (37%, *n* = 66). As stated previously in Study 2, height, weight, and BMI for the total sample were similar at baseline and follow up; mean BMI was 23 at baseline and 24 at follow up. Although this indicates a healthy BMI, when excluding children aged under 18, the adult participants mean BMI was 26 which is situated in the overweight range. Excluding children under the age of 18, 42% of adults had a university qualification, 23% had a TAFE or technical qualification and the remaining 33% had attended secondary school as their highest level of formal education.

Individual participant's disease risk for the four diseases. The prevalence of above-average individual risk for the four chronic diseases was not high. *Average risk* categorization for colorectal cancer was at 98% (*n* = 174); for breast cancer 91% (*n* = 162); for heart disease 77% (*n* = 137); and for type 2 diabetes 82% (*n* = 145). For *above-average risk* the results showed a comparatively low proportion of participants: colorectal cancer was at 2% (*n* = 4); breast cancer 6% (*n* = 11); heart disease 20% (*n* = 36); and type 2 diabetes 14% (*n* = 25). Finally, individual participants previously diagnosed with at least one of the four diseases were few; colorectal cancer 0%; breast cancer 3% (*n* = 5); heart disease 3% (*n* = 5); and type 2 diabetes 5% (*n* = 8).

Family-at-risk and mother-at-risk. When examining prevalence of disease risk at the family level (i.e., risk identified on the basis of at least one being above-average), prevalence was as follows; 24% of families had average risk (*n* = 42), 71% had above-average risk (*n* = 127), and 5% had a family member diagnosed (*n* = 9). Similarly, when

examining mothers' risk status at the family level, 41% of families had a mother at average risk (*n* = 73), 46% of families had a mother at above-average risk (*n* = 83), and 12% of families had a mother that was diagnosed with at least one of the four diseases (*n* = 22).

Healthy food consumption within the total sample was similar at baseline and at follow up. At follow up, more than two thirds of the sample met the Australian NHMRC guidelines for the recommended serves of fruit per day[38] (NHMRC, 2013a). Sixty six percent of Study 3 participants met the recommended daily fruit consumption guidelines compared with 58% of the Australian population (ABS, 2014a). On the other hand, over three quarters of the sample (79%) did not meet the Australian NHMRC guidelines for the recommended daily serves of vegetables[39] (NHMRC, 2013a). Twenty one percent of the sample in the present study reported consuming the recommended serves of vegetables per day which is greater than the 6.8% of Australians who do so (ABS, 2014a). Table 1 presents healthy and unhealthy food consumption at baseline and follow up within the total sample, Table 2 presents healthy food by gender.

[38] Children 4 to 8 years: at least 1.5 serves, adults and children over 9 years: at least two serves, and one serve is 150 grams which is equivalent to one cup of chopped fruit; N = 178, Mdn = 2, Range = 0 - 12 (NHMRC, 2013a).

[39] Children 4 to 8 years: at least 4.5 serves, adults and children over 9 years: at least five serves) and one 75-gram serve is equivalent to one cup of salad or half a cup of cooked vegetables, (N = 178, Mdn = 3, Range = 1 – 14 (NHMRC, 2013a).

Table 1.

Total Sample's Healthy and Unhealthy Food Consumption at Time One and Time Two.

Variable	Time	*N*	*M*	*SD*	Median	Range	Min	Max
Fruit (serves/day) [a]	T1	177	2.60	2.07	2.00	14.00	0.00	10.00
Fruit (serves/day) [a]	T2	176	2.61	2.36	2.00	15.00	0.00	12.00
Veg (serves/day) [a]	T1	175	3.46	2.21	3.00	13.50	0.50	11.00
Veg (serves/day) [a]	T2	174	3.50	2.13	3.00	13.00	1.00	14.00
Snacks (serves/wk) [a]	T1	176	4.61	3.41	4.00	18.00	0.00	18.00
Snacks (serves/wk) [a]	T2	177	4.26	3.85	3.00	21.00	0.00	21.00
Fast (serves/wk) [a]	T1	177	0.66	1.40	0.00	12.00	0.00	12.00
Fast (serves/wk) [a]	T2	177	0.63	1.04	0.00	7.00	0.00	7.00

Note . [a] Skew positive, interpreting median and range will be most meaningful.

Table 2.

Males and Females Healthy Food Consumption at Time One and Time Two.

Variable	Gender	Time	*N*	*M*	*SD*	Median	Range	Min	Max
Fruit (serves/day) [a]	Male	T1	70	2.49	2.28	2.00	14.00	0.00	14.00
	Male	T2	70	2.14	1.50	2.00	10.00	0.00	10.00
	Female	T1	101	2.52	1.63	2.00	9.00	0.00	9.00
	Female	T2	101	2.66	2.20	2.00	12.00	0.00	12.00
Veg (serves/day) [a]	Male	T1	70	3.26	2.52	3.00	13.00	1.00	14.00
	Male	T2	70	3.02	1.83	3.00	12.00	1.00	13.00
	Female	T1	101	3.57	2.00	3.00	13.50	0.50	14.00
	Female	T2	101	3.77	2.23	3.00	13.00	1.00	14.00

Note. [a] Skew positive, interpreting median and range will be most meaningful.

Unhealthy food consumption. The NHMRC recommend that Australians limit their intake of a number of unhealthy foods[40] (NHMRC, 2013a). In Study 3, unhealthy food consumption at follow-up was slightly less than at baseline. Snacks[41] were consumed by participants on average, three times per week (*Mdn* = 3, *Range* = 0 - 21) and fast food[42] less than once a week (*Mdn* = 0, *Range* = 0 - 7). Table 3 presents unhealthy food consumption at baseline and follow up for males and females which decreased slightly over time and males consumed marginally more than females. Unhealthy food results for the total sample were shown previously together with healthy food in Table 1.

[40] That is, the following specific foods and beverages: meat pies, fried hot chips, savoury snacks, processed meats, cakes, biscuits, confectionary, desserts, ice-cream, soft drinks, cordials and other energy dense food and drinks (NHMRC, 2013a).

[41] Examples of snacks measured in the present study were defined as: a chocolate bar, a piece of cake, a packet of chips/Twisties/corn chips, ice cream, 3-4 sweet biscuits.

[42] Examples of fast food meals measured in the present study were defined as: McDonalds, Hungry Jacks, Pizza Hut, KFC, Red Rooster, hamburger, pizza or fish and chips.

Table 3.

Males and Females Unhealthy Food Consumption at Time One and Time Two.

Variable	Gender	Time	*N*	*M*	*SD*	Median	Range	Min	Max
Snacks (serves/week) [a]	Male	T1	70	5.04	3.48	5.00	15.00	0.00	15.00
	Male	T2	70	4.50	4.01	3.25	20.00	0.00	20.00
	Female	T1	102	4.35	3.39	4.00	18.00	0.00	18.00
	Female	T2	102	4.07	3.77	3.00	21.00	0.00	21.00
Fast (serves/day) [a]	Male	T1	70	0.81	1.72	0.00	12.00	0.00	12.00
	Male	T2	70	0.70	1.10	0.00	6.00	0.00	6.00
	Female	T1	102	0.57	1.16	0.00	10.00	0.00	10.00
	Female	T2	102	0.56	0.97	0.00	7.00	0.00	7.00

Note. [a] Skew positive, interpreting median and range will be most meaningful.

Intentions to Change Healthy Dietary Behaviour

Intentions to change healthy dietary behaviours were confirmed by measuring participant's self-reported intentions to increase fruit and vegetable consumption (measured together as a single item question). Of the total sample, 77% of participants indicated intentions to increase their fruit and vegetable consumption at follow up. Of the 77% who indicated intentions to change, participant's self-reported confidence to change (i.e., self-efficacy) was rated at 3 or above (from a range of 1, 'Not at all confident' to 7, 'Very confident').

Results of the Families SHARE Intervention: Changes in Food Consumption over Time

Conditions: Intervention and control.

Hypothesis 1 predicted that providing tailored family health history information to the participants in the experimental group would motivate individuals to improve dietary behaviours over time more so than control group individuals. Analyses were conducted using Generalized Linear Mixed Models (GLiMM; Poisson and negative binomial regression analyses)[43]. GLiMM analyses for the entire sample did not show any significant differences in food consumption between the intervention and control groups (i.e., the condition variable; for the interaction figures see Figures 5 to 8).

[43] For further details see Appendix 4.H.

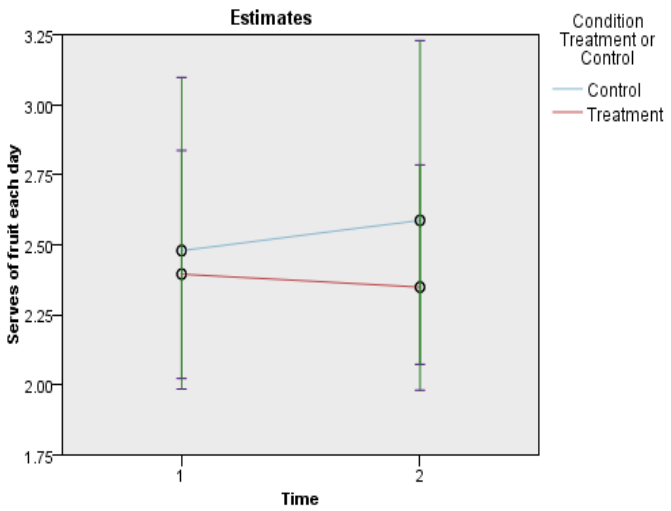


Figure 5. Intervention and control condition fruit consumption over time non-significant interaction results.

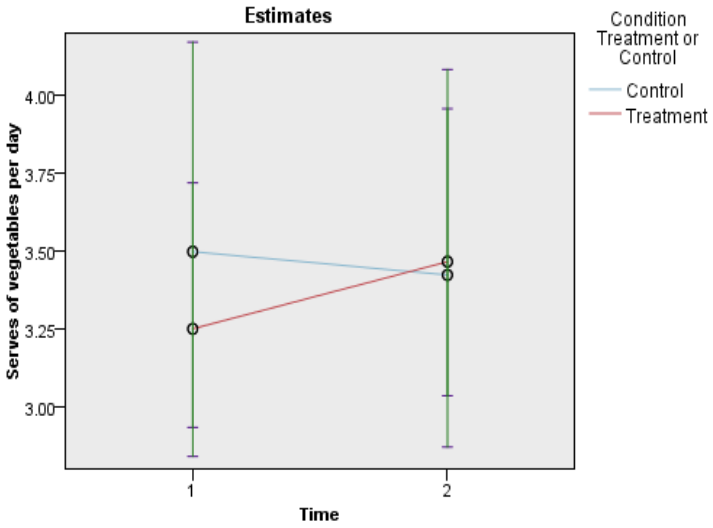


Figure 6. Intervention and control condition vegetable consumption over time non-significant interaction results.

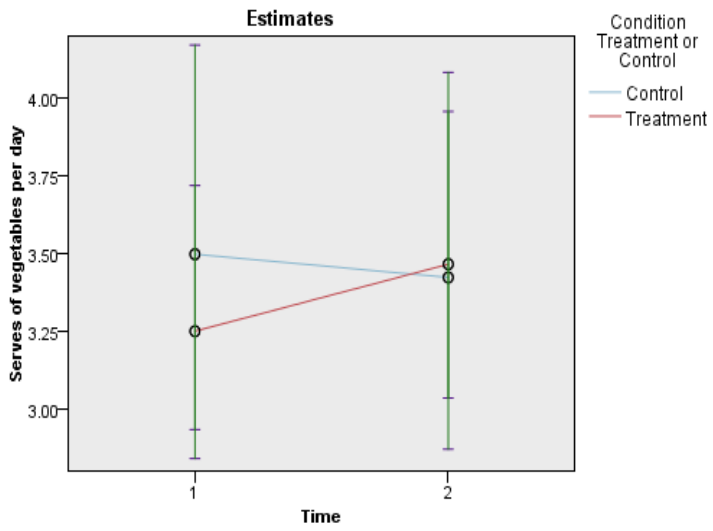


Figure 7. Intervention and control condition snack consumption over time non-significant interaction results.

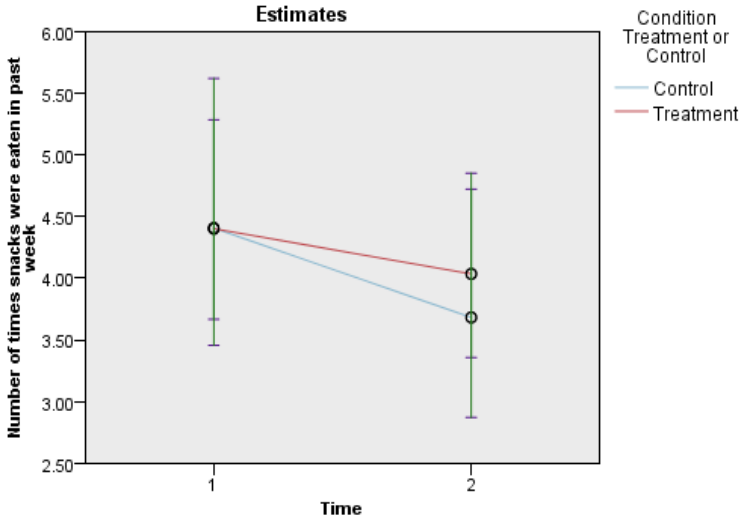


Figure 8. Intervention and control condition fast food consumption over time non-significant interaction results.

This may appear at first glance to be a failure of the intervention's effectiveness. However, given the unlikelihood of average disease-risk families in the experimental group being motivated to change dietary behaviours over time, additional analyses were then used to test hypothesis 2: families at *increased disease-risk (only)* will show more changed dietary behaviour than those not at increased risk.

Intervention and control group stage of change progression. Participant's stage of change status was measured at baseline and follow up to examine progression in stages over time; expecting progression from 'pre-contemplation' to 'contemplation', or from 'contemplation' to 'action' stages over time within participants in the experimental group at increased disease-risk, controlling for participants in the 'action' and 'maintenance' stage of change at baseline (TTM; stage of change; Conner & Norman, 1995; Prochaska et al., 1992; Sarkin et al., 2001). Hypothesis 2 predicted that experimental group participants would progress to a higher stage of readiness to change healthy and unhealthy food consumption (after controlling for individuals at the action or maintenance stage of change at baseline). Results showed a significant progression in stage of change over time in the experimental group for fruit consumption; with movement from preparation to the action stage (i.e., from stage 3 to 4); a GLiMM negative binomial was used (see Table 4 and Figure 9).

Table 4
Fruit Model 1.1: Stage of Change Fruit Consumption Over Time

Effects	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower Upper	*F*	*p*
Model 1.1				(3, 312)		2.94	**.030**
Time				(1, 312)		7.29	**.007**
	T1 – T2	-0.62	-2.72	(1, 312)	-1.07 -0.17		**.007**
Condition				(1, 312)		1.23	.270
	Treatment-Control	-0.26	-1.11	(1, 312)	-0.71 0.20		.270
Time*Condition				(1, 312)		0.03	.870
	Treatment			(1, 312)		4.44	**.040**
	Control			(1, 312)		3.27	.070
	Treatment T1 - T2	-0.56	-2.11	(1, 312)	-1.07 -0.04		**.040**
	Control T1 – T2	-0.68	-1.81	(1, 312)	-1.41 0.06		.070

Note . GLiMM Negative Binomial Vertical Multilevel Modeling. Model 1.1: -2 log pseudo likelihood = 322.884

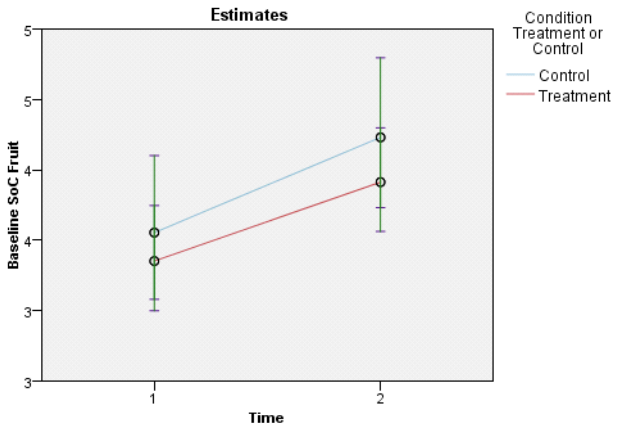


Figure 9. Stage of Change: Fruit consumption at baseline and follow up. Coloured lines depict the experimental and control conditions. Error bars depict the confidence intervals.

There was no significant progression in stage of change over time for individuals in the experimental group for vegetable consumption. Unhealthy food consumption analysis was not attempted since no recommendations about acceptable consumption levels exist at the time of writing and, consequently, unhealthy food could not be categorised according to recommendations being met or not met (for the SoC analysis rationale refer back to Appendix 4.G).

Level of risk. Hypothesis 3 predicted that participants in the experimental condition: either individuals-, individuals with a family member- or, individuals with a mother- at above-average disease risk or diagnosed with any of the four chronic diseases would increase healthy food consumption and decrease unhealthy food consumption over time. Results of all analyses are outlined below.

Individuals at increased risk of the four diseases. GLiMM analyses compared *individual risk* for each of the four diseases by condition, time, and three-way interaction for each of the four consumption outcome variables. None of the interactions were significant; therefore, the experimental group was not significantly different to controls on any of the consumption outcome variables over time depending on disease risk level. Interaction plots

showed non-significant directional trends that supported some of the hypotheses. Future research may provide more significant results given more power, other than that, just one significant result showed individuals diagnosed with heart disease consumed snacks less frequently than individuals in the average and above-average disease-risk groups (see Table 5 and Figure 10). This indicates an effect of diagnosed heart disease on reduced snack consumption over time, regardless of condition.

Table 5.

Snacks Model 4.1: Risk of Heart Disease Changes in Consumption Over Time

Effects	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 4.1				(2, 339)			5.63	**.004**
Time				(1, 339)			3.80	**.052**
	T1 – T2	1.12	1.95	(1, 339)	0.01	2.25		**.052**
Condition				(1, 339)			0.82	.37
	Treatment-Control	-0.69	-0.91	(1, 339)	-2.19	0.81		.37
Risk Heart				(2, 339)			5.63	**.004**
	Diagnosed - Average risk	-2.08	-3.35	(1, 339)	-3.57	-0.58		**.003**
	Diagnosed - Above average risk	-1.97	-2.99	(1, 339)	-3.44	-0.49		**.006**
	Above average risk - Average risk	-0.11	-0.32	(1, 339)	-1.78	0.56		.750

Note . GLiMM Poisson Vertical Multilevel Modeling. Model 3.2: -2 log pseudo likelihood = 858.648

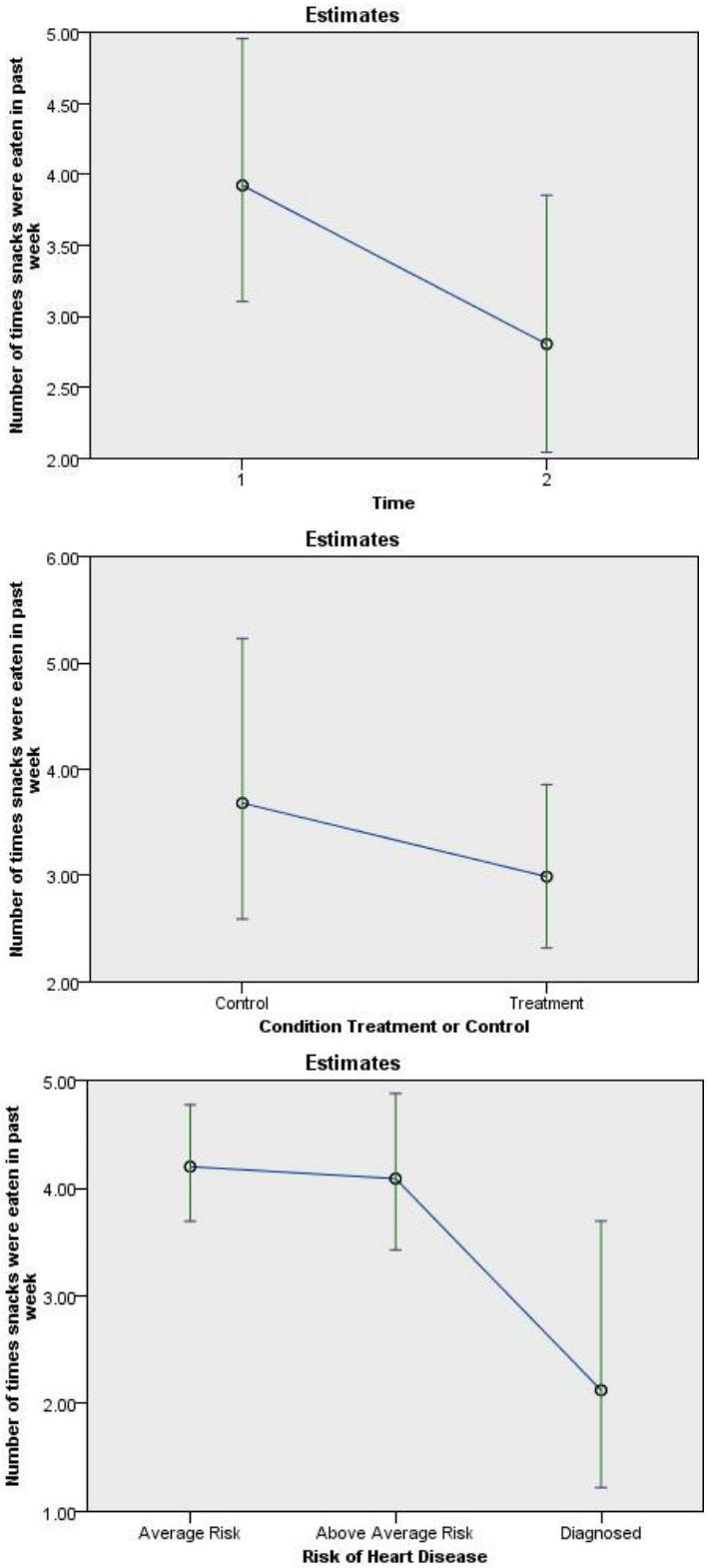


Figure 10. Snacks Model 4.1: Risk of heart disease changes in consumption over time. Error bars depict 95% confidence intervals.

Since there was no effect of the intervention on food consumption when examining individuals at increased disease-risk, the possibility that if a family member- or the mother- in

the experimental group was at above-average disease risk, or diagnosed, that other related family members could also change their behaviour over time was tested. Hypothesis 3 predicted that participants in the intervention condition would increase healthy food consumption and decrease unhealthy food consumption over time if: a family member- or a mother- was at increased risk or diagnosed with any one or more of the four chronic diseases.

A member of the family, or the mother at increased disease risk. GLiMM analyses initially examined condition (experimental and control) group differences over time comparing individuals who had at least one participant family member at increased risk or diagnosed with disease when compared to individuals who had families with participants of average disease risk (i.e., family-any-disease-risk*condition*time)[44]. Results on all four consumption variables were non-significant. If mothers in participant families were at increased risk or diagnosed, results showed average risk controls increased their fruit consumption over time (see Appendix 4.I for tables and figures of significant results).

Discussion

Characteristics of the Sample

Compared to the general Australian population the study sample's fruit and vegetable consumption was relatively high (ABS, 2014a). Fruit consumption was at 66% compared with 58% of Australians that regularly consumed the recommended two serves per day. Vegetable consumption that met the recommended guidelines (5 serves per day) were 21% of the sample, three times greater than the 7% of Australians that regularly consumed five serves per day. Clearly, compliance with recommended fruit and vegetable consumption could be improved, and vegetable intake was particularly concerning given its importance in

[44] All modeling analyses adjusted for disproportionate sampling and cluster sampling to avoid over inflating the type 1 error rate (Heck et al., 2014a).

disease prevention (Bradbury, Appleby, & Key, 2014; NHMRC, 2013a; Oyebode, Gordon-Dseagu, Walker, & Mindell, 2014).

The sample's snack consumption averaged three times per week. It is difficult to judge the health risk of consumption at this level given that there are no specific Australian recommendations other than to limit the intake of saturated fats, sugar, salt and alcohol (NHMRC, 2013a). Snack consumption showed a reduction at follow up when compared to baseline across both groups indicating an effect of time rather than an effect of the intervention. Conversely, fast food consumption did not change over time. This may be because consumption averaged less than once a week and consequent ceiling effects may have limited scope for improvement.

Effect of Condition and Time

Longitudinal modeling that examined changes over time and by condition showed that there were no significant differences in food consumption between the two conditions. Reasons for this include probable ceiling effects in the healthy sample in the study; individuals consuming healthy food at recommended levels at baseline cannot improve their outcome. Furthermore, many individuals in both groups were at average personal risk for chronic disease and this may have limited the motivational salience of the tailored family health history information.

Snack consumption did reduce over time in both groups. This result might indicate that the act of collecting family health history information at baseline had an influence on preparedness to consume less energy dense food. Previous research has shown similar findings in young adults aged 18 – 25 years (e.g., Prichard, Lee, Hutchinson, & Wilson, 2015). Simply talking with family members when completing baseline questionnaires about familial disease history may have increased participant's perceived disease susceptibility and acted as a "cue to action" (e.g., Stacy & Loyd, 1990), thereby reducing unhealthy food

consumption over time. Family health history questions were asked of all participants at baseline in order to construct family disease risk pedigree diagrams for both the experimental group (whose feedback was delivered at three months post-baseline), and the control group (whose feedback was delivered after the conclusion of the study). To overcome this potential confound it is suggested that future similar research change questionnaire delivery procedures by asking family health history questions of the experimental group at baseline, and of the control group at follow up. In this way, family health history would be discussed by participants in each respective condition at separate time points, and the subsequent impact of family health history on the experimental group may be better understood.

Stage of Change (SoC) and Healthy Food

Stages of change were examined for healthy food consumption because fruit and vegetable consumption were clearly assessable in terms of 'meeting' or 'not meeting' the NHMRC recommended serves per day. Food consumption variables combined with stage of change items enabled measurement of progression through the five stages of change between baseline and follow up (i.e., pre-contemplation, contemplation, preparation, action, and maintenance). The intervention was successful in motivating movement from the preparation to the action stage of change in fruit consumption for participants in the experimental condition. Promoting healthy food consumption within families was one of the aims of the present study and the resultant evidence confirms the findings of previous research showing individuals' uptake of healthy lifestyle behaviours in response to tailored family health history information (e.g., Claassen et al., 2010; Hovick et al., 2014; Pijl et al., 2009; Ruffin et al., 2011).

Effects of individual-, family- and mother's- disease-risk on food consumption. In terms of risk, results are summarised in Table 6 and are explored further in the discussion below.

Table 6. Summarizes the GLiMM analyses showing differences in risk group and condition.

Disease category/Risk	Average Risk	Above-Average Risk	Diagnosed
Family member at risk	ns	ns	ns
Mother at risk	Fruit consumption Controls T1 < Controls T2	ns	ns
Individual at risk Colorectal cancer	ns	ns	ns
Individual at risk Breast cancer	ns	ns	ns
Individual at risk Diabetes	ns	ns	ns
Individual at risk Heart disease	ns	ns	Snacks consumption Diagnosed < average and above average risk

Note. ns denotes non-significant results.

Family Risk and Mother Risk

In line with the Health Belief Model, above-average risk for disease was intended to be a motivating factor triggering health behaviour change (Rosenstock et al., 1988). Because 77% of individuals were at average risk for the four diseases, motivating change within this study sample could well have missed its mark by targeting largely unaffected individuals.

According to Bandura (2001), collective agency can have an impact on the behaviour of individuals within a coherent group who share a common goal; in this case, the family. Thus, analyses were broadened to examine this possibility. By grouping individuals in this way, 71% of participants had at least one family member at above-average risk of one of the four diseases, and 5% had a family member who was diagnosed with disease. Participants with a mother in the family at above-average risk for disease were 46%, and 12% of individuals had a mother diagnosed with at least one of the four diseases. When analysed by

condition, the control group with mothers of average risk increased fruit consumption significantly over time; which may have been an unintended effect of the questionnaire itself – prompting health behaviour change when participants realised their self-reported fruit consumption was inadequate. As stated previously, simply talking about family health history at baseline could have motivated behavioural change in the control group (Prichard et al., 2015), similarly self-reporting food consumption may have drawn participant's attention to it. Moreover, the study lacked enough power to provide a significant result in the experimental group when risk and healthy baseline consumption were also taken into account.

Specific Disease Risk

Individuals diagnosed with heart disease reduced snack consumption over time more so than average and above-average risk individuals. This result indicates that heart disease was perceived as the most controllable of the four diseases, a result consistent previously in studies with heart disease, diabetes and several cancers (Wang et al., 2009). In addition, heart disease may have been perceived as a significant enough health threat to trigger change in unhealthy food consumption. Doctors diagnosing and subsequently advising individuals about the link between diet and heart disease may have also had an impact, however, disease prevention by dietary behaviour is preferable to post-diagnostic dietary advice and subsequent change. This intervention may therefore prove to be particularly successful in targeted heart disease prevention, or as an adjunct to strategies that minimize the severity of existing heart disease in diagnosed individuals with potential flow on effects to their families. As has been established in previous research (e.g., Williams, Steptoe, & Wardle, 2013), it was not until diagnosis was made that any change in behaviour was noted, signifying that susceptibility needed to be experienced before any health action was taken (Janz & Becker, 1984).

Limitations

One of the limitations of the present study was the insufficient number of participants who were diagnosed, or at increased risk, for each of the four diseases individually. When analysed as 'any-family-member-at-risk' or the 'mother-at-risk' for one or more of the four diseases the analyses had more power. This study was quite complex and some interesting directional trends (confirming hypotheses) were noted in the non-significant data, which with greater participant numbers in future research may yield significance findings.

The sample had disproportionately low disease-risk coupled with healthier dietary behaviours than the Australian population. These factors limited the number of individuals in the 'above-average' or 'diagnosed' disease risk groups who simultaneously had the capacity to improve their dietary behaviours over time, whilst also being randomly allocated to the experimental condition. Results were therefore constrained to the proportion of participants in the experimental condition who met the criteria for potential dietary improvement for disease prevention reasons. Future research of this nature could consider more purposeful recruitment of participants who require dietary interventions and/or who are also at greater risk of the four diseases, and then randomly allocating to the experimental or control group conditions. In addition, with a wider geographical scope encompassing all Australian States and Territories, greater participant numbers could have potentially generated significant results that were apparent only as trends in the present study. Finally, recruitment of three-generation participant families with strict age and inclusion criteria prolonged recruitment time and resources (for details see Hughes, Hutchinson, Prichard, Chapman, & Wilson, 2015); subsequent data analysis was also more complex than anticipated, requiring local and international specialist statistical consultant advice on using procedures with family nested data correctly.

Summary

Fruit consumption showed the most promising result in response to the Families SHARE intervention as demonstrated by stage of change progression in the experimental group. Vegetable consumption, on the other hand, showed little change on any of the variables, and consumption was well below the NHMRC recommendations. Although participants in this thesis showed greater vegetable consumption than the Australian population, improvements in vegetable consumption were not evident. Hence, increasing Australians' recommended daily vegetable consumption (at less than 7% in the general population, and less than 25% of participants in the present study) poses a sizeable challenge for disease prevention initiatives.

Unhealthy food consumption, on the other hand, showed improvements, with decreases in snack consumption over time that may have been in response to family members' talking about family health history at baseline, whilst presumably some individuals also responded to the severity of heart disease as a cue to action. Although few changes in actual dietary behaviours were observed, intentions to change healthy dietary behaviours (i.e., increase fruit and vegetable consumption) were observed in more than three quarters of the sample. Confidence to enact these intentions (indicating self-efficacy), were rated at moderate or greater - and intentions are the pre-cursor to any behavioural change according to health behaviour theory (e.g., Conner & Norman, 1995; Prichard et al., 2015).

CHAPTER 5: STUDY 3 - PART B

Generational differences in healthy and unhealthy food consumption over time in response to tailored family health history information: Do food attitudes linking diet and health make a difference?

Background

As detailed in previous chapters, dietary behaviours occur in the context of, and are very influenced by, family relationships and socialization (Contento et al., 2006; Feunekes et al., 1998; Patrick & Nicklas, 2005; Prichard et al., 2012). Study 3 Part A focused on the importance of family health history and the effects on dietary behaviour change over time, however, age or generation might impact concerns about, and responsibility for, family health. Previous research has shown that the strength of intergenerational family ties play a role in providing health care to elderly relatives (e.g., Brody, 1981), and older adults have shown comparatively stronger health-promoting lifestyle behaviours (including diet) than young and middle-aged adults (e.g., Walker, Volkan, Sechrist, & Pender, 1988). However, fewer studies have shown children's capacity to act as health promotors within the family (e.g., Christensen, 2004; Montgomery-Anderson & Borup, 2012). Therefore, it is also critical to understand how each generation may respond differently to disease diagnosis or the realisation they have above-average familial disease risk. Influential family relationships may then be employed to motivate others in the family to making healthy dietary choices utilizing collective free will to make any necessary modifications together (Bandura, 2001; Hendriks et al., 2012). The underlying mechanisms that influence dietary behaviours, such as Food Life behaviours and attitudes (Sharp et al., 2013) that were identified in Study 2, could also moderate the link between provision of information about familial risk and diet related behaviour.

Aims

Study 3 Part B examines any changes over time between the various groups and interactions. That is, between-generation (i.e., child, parent, or grandparent) similarities and differences, and any gender by generation (e.g., grandmothers, grandfathers) similarities and differences in healthy and unhealthy food consumption, which may be impacted upon by disease diagnosis or above-average disease risk. The study also examines any moderating impact of Food Life behaviours and attitudes on these main and interaction effects.

The Present Study

The present study examines whether any differences exist in healthy and unhealthy food consumption when compared between generations in the experimental group in response to a disease diagnosis or self-rated above-average disease risk. Any similarities or differences in food consumption over time will be explored between the following groups: child, parent, or grandparent; male or female of average risk, above-average risk, or diagnosed with chronic disease at the level of 'individual-risk', 'family-risk' and 'mother-at-risk'. Subsequently, these models will be further tested by the addition of Food Life subscales diet-health oriented behaviour (DHOB) and diet-health/disease linked attitudes (DHLA) (Sharp et al., 2013).

Research Hypotheses

1. Information provided about familial disease risk will influence the food consumption of members from three generations differently. Specifically, it is predicted that the parent and grandparent generations will be positively impacted more so than the child generation.
2. The impact of family health history information on dietary behaviour will be greater in families with 'above-average' individual/group risk than in families with 'average' individual/group risk.

Method

The method for Study 3 Part B was identical to Study 3 Part A. Study 3 Part B also made use of additional Food Life behaviour and attitude data (as described in Study 2) collected at the same time points and from the same participants, to further examine the influence of intergenerational family relationships and changes in Food Life behaviour and attitudes over time that could have also impacted upon food consumption. The Food Life subscales (diet-health orientated behaviour (DHOB) and diet-health/disease linked attitudes (DHLA) (Sharp et al., 2013) outlined previously in Study 2 were administered at baseline and follow up at the same time points as Study 3 Part A.

Model Design

To assess effects on the dependent food consumption variables the following independent variables were included in the model testing: condition (intervention or control; between-subjects factor with two levels); individual disease risk (either 'average', 'above-average' or 'diagnosed' for each of the four diseases colorectal cancer, breast cancer, heart disease, and type 2 diabetes; between-subjects factor with three levels x 4); family disease risk (either 'average', 'above-average' or 'diagnosed' for each of the four diseases; between-subjects factor with three levels x 4); mother's disease risk (either 'average', 'above-average' or 'diagnosed' for each of the four diseases; between-subjects factor with three levels x 4); generation (child, parent, or grandparent; between-subjects factor with three levels), gender (between-subjects factor with two levels) and the Food Life variables diet-health oriented behaviour (DHOB) and diet-health/disease linked attitudes (DHLA); between-subjects factor, scaled variable with 7 levels x 2) (Heck et al., 2014b).

Measures. Study 3 Part B's measures were identical to those used in Study 2 (e.g., Food Life subscales) and Study 3 Part A (e.g., self-reported healthy and unhealthy food consumption) therefore details are not repeated in this chapter. The measures at baseline and

follow up are listed as follows.

Baseline measures. The baseline measures included demographic information, self-reported fruit, vegetable, snack, and fast food consumption; the Diet-Health Oriented Behaviour (DHOB), also termed *Diet-Health Orientation; DHO*), and Diet-Health/Disease Linked Attitudes (DHLA) also termed *Diet-Health Link (DHL)* subscales of the Food Life Questionnaire – Short Form (FLQ-SF) (Sharp et al., 2013); family health history information for colorectal cancer, breast cancer, heart disease, and type 2 diabetes; intentions to change dietary behaviour; and perceived disease risk.

Follow up measures. Follow up measures repeated baseline measures as follows: demographic information (for follow up BMI); self-reported fruit, vegetable, snack, and fast food consumption; the Diet-Health Oriented Behaviour (DHOB), and Diet-Health/Disease Linked Attitudes (DHLA) subscales of the Food Life Questionnaire – Short Form (FLQ-SF); intentions to change dietary behaviour and confidence rating. Stage of readiness to change on each of the four consumption variables: fruit, vegetables, snacks, and fast food (Armitage et al., 2004; Campbell et al., 1999; Prochaska et al., 1992; Sarkin et al., 2001).

Results

Data and Statistical Analysis

As with Study 3 Part A, Study 3 Part B used multilevel modeling techniques described by Heck et al. (2014a) to manage clustered longitudinal data that controlled for the nesting effect of families.[45] Each test was appropriately chosen for each outcome variable that was being analysed (Elhai et al., 2008). Study 3 Part B examined the data for effects of

[45] As detailed in Study 2 and Study 3 Part A, statistical analysis decisions were guided by Elhai, Calhoun, and Ford (2008). Results would normally have been achieved using 'classical statistics' i.e., a within-subjects repeated measures factorial ANOVA (Tabachnick & Fidell, 2013); however, these family data would have violated several assumptions if classical statistics were used. For the full rationalization refer to back to Chapter 3 Appendix D and Chapter 4 Appendix H. Modeling procedures and analyses were conducted using methods that manage the potential for over inflating the type 1 error rate (Heck et al., 2014a).

condition, generation, gender, generation-by-gender, generation-by-disease-risk, and the Food Life subscales: diet-health oriented behaviour and diet-health/disease linked attitudes. The same procedures, statistical analyses and software were used.[46] In short, multilevel modeling accurately analysed each individual separately, whilst simultaneously analysing all clustering (nesting) and groups within the same model, the alpha value was set at $p < .05$, and managing the Type 1 error rate to avoid over inflation (Heck et al., 2014b).[47] The results of Study 3 Part B are presented in the order of generations and healthy food consumption, then generations and unhealthy food consumption, disease risk, and lastly Food Life behaviours and attitudes. Characteristics of the (same) sample demographics were outlined earlier in Study 3 Part A.

Food Consumption

Intraclass correlations in food consumption reveal the extent that behaviours occur as whole families, or as independent individuals. Intraclass correlations (ICCs) were conducted to examine the within-family effects on healthy and unhealthy food consumption. The ICCs for fruit consumption had a total family variance of 1.6%; for vegetable consumption 1%; snack consumption 3%; and fast food 9.5%. These results show the least amount of family clustering in vegetable consumption and the greatest amount in fast food consumption indicating shared family dietary behaviour occurred most in fast food and least of all in vegetable consumption. One may have expected families to share meals together that include vegetables most of all, however the results of this sample suggest vegetable

[46] The only difference from Study 3 Part A statistical analysis was that the diet-related food attitudes (diet-health oriented behaviour and diet-health/disease linked attitudes) had variances that were less than the mean therefore met the assumptions for Poisson regression analysis instead of negative binomial when food attitudes were the dependent variable (Elhai et al., 2008).

[47] One of the limitations, due to finite computational capabilities within the available computer memory to date, was that a limited number of independent variables could be entered into each model at any one time, thus decisions were made as to how to best answer each of the research questions in turn, and some compromises had to be made.

consumption was an independent individual behaviour. [48] See Tables 1 to 4 for the ICC results.

[48] The analyses in this study controlled for family clustering in order to analyse behaviours as if they had occurred independently of each other, however, result interpretations should bear in mind where the greatest family interdependencies exist.

Table 1.

Fruit

Shown are the intraclass correlation (ICC) results. These indicate the ***variability in fruit consumption*** *associated with differences between families as a percentage. Wald Z shows Model 1 result significance (GLiMM negative binomial output).*

Random Effects Subject	Model	Within group variance (Residual Estimate)[a]	Between group variance (Intercept Estimate) [b]	Standard Error	95% CI		Wald Z	ICC [c]	Variance	*p*
					Lower	Upper			%	
FamilyID_A *N* = 42 families	Model 1.1	1	0.055	0.040	0.013	0.230	1.380	0.0164	1.60%	.169
	Model 1.2	1	0.052	0.390	0.012	0.231	1.311			.190
	Model 1.3	1	0.041	0.037	0.007	0.238	1.109	0.0165	1.65%	.268

Note. Calculation: ICC = b/(a + b) Tabachnick & Fidell (2012) p.826-7. However, when the residual variance is 1.00 the scale factor can be used to calculate an ICC $\pi^2/3 = 3.29$, therefore [a] = 3.29 and the equation used was ICC [c] = b/(a + b) Heck et al. (2012) p. 157. Model 1.1 Generation, Gender & Gen*Gend; Model 1.2 add DHOB; Model 1.3 remove DHOB and add DHLA.

Table 2

Vegetables

Shown are the intraclass correlation (ICC) results. These indicate the ***variability in vegetable consumption*** *associated with differences between families as a percentage. Wald Z shows Model 1 result significance (GLiMM negative binomial output).*

Random Effects Subject	Model	Within group variance (Residual Estimate)[a]	Between group variance (Intercept Estimate) [b]	Standard Error	95% CI		Wald Z	ICC [c]	Variance	*p*
					Lower	Upper			%	
FamilyID_A *N* = 42 families	Model 2.1	1	0.034	0.024	0.009	0.133	1.435	0.0102	1.02%	.151
	Model 2.2	1	0.030	0.024	0.007	0.14	1.278			.201
	Model 2.3	1	0.033	0.024	0.008	0.138	1.366	0.0099	0.99%	.172

Note. Calculation: ICC = b/(a + b) Tabachnick & Fidell (2012) p.826-7. However, when the residual variance is 1.00 the scale factor can be used to calculate an ICC $\pi^2/3 = 3.29$, therefore [a] = 3.29 and the equation used was ICC [c] = b/(a + b) Heck et al. (2012) p. 157. Model 1.1 Generation, Gender & Gen*Gend; Model 1.2 add DHOB; Model 1.3 remove DHOB and added DHLA.

Table 3

Snacks

Shown are the intraclass correlation (ICC) results. These indicate the ***variability in snack consumption*** *associated with differences between families as a percentage. Wald Z shows Model 1 result significance (GLiMM negative binomial output).*

Random Effects Subject	Model	Within group variance (Residual Estimate)[a]	Between group variance (Intercept Estimate) [b]	Standard Error	95% CI		Wald Z	ICC [c]	Variance	*p*
					Lower	Upper			%	
FamilyID_A *N* = 42 families	Model 3.1	1	0.100	0.053	0.036	0.282	1.899	0.0294	2.94%	.058
	Model 3.2	1	0.103	0.052	0.038	0.277	1.986			**.047**
	Model 3.3	1	0.087	0.050	0.028	0.268	1.749	0.0257	2.57%	.080

Note. Calculation: ICC = b/(a + b) Tabachnick & Fidell (2012) p.826-7. However, when the residual variance is 1.00 the scale factor can be used to calculate an ICC $\pi^2/3 = 3.29$, therefore [a] = 3.29 and the equation used was ICC [c] = b/(a + b) Heck et al. (2012) p. 157. Model 1.1 Generation, Gender & Gen*Gend; Model 1.2 add DHOB; Model 1.3 remove DHOB and add DHLA.

Table 4

Fast Food

Shown are the intraclass correlation (ICC) results. These indicate the ***variability in fast food consumption*** *associated with differences between families as a percentage. Wald Z shows Model 1 result significance (GLiMM negative binomial output).*

Random Effects Subject	Model	Within group variance (Residual Estimate)[a]	Between group variance (Intercept Estimate) [b]	Standard Error	95% CI		Wald Z	ICC [c]	Variance	*p*
					Lower	Upper			%	
FamilyID_A *N* = 42 families	Model 4.1	1	0.344	0.160	0.138	0.854	2.152	0.0946	9.46%	**.031**
	Model 4.2	1	0.308	0.158	0.112	0.082	1.946			.052
	Model 4.3	1	0.361	0.173	0.141	0.924	2.082	0.0988	9.88%	**.037**

Note. Calculation: ICC = b/(a + b) Tabachnick & Fidell (2012) p.826-7. However, when the residual variance is 1.00 the scale factor can be used to calculate an ICC $\pi^2/3 = 3.29$, therefore [a] = 3.29 and the equation used was ICC [c] = b/(a + b) Heck et al. (2012) p. 157. Model 1.1 Generation, Gender & Gen*Gend; Model 1.2 add DHOB: Model 1.3 remove DHOB and add DHLA.

A series of models determined differences in healthy and unhealthy food consumption between people distinguished by gender and generation. These results are summarized alongside any changes according to condition and/or time in Table 5.

Table 5.

Summary of the GLIMM Analyses comparing Food Consumption between Generations, Gender, Condition, Time, and the Interaction of Generation by Gender.

Variable	Generation	Gender	Generation*gender	Change over time
Fruit	Child > parents Above average risk grandparents > parents	ns	ns	Experimental group SoC for fruit consumption from preparation T1 to action T2
Vegetables	ns	Females > males	Sons > grandfathers	ns
Snacks	Child > parents Child > grandparents	ns	Daughters > mothers Daughters > grandmothers	Whole sample T1 > T2
Fast food	Child > parents Child > grandparents Parents > grandparents	ns	Sons > grandfathers Daughters > mothers Daughters > grandmothers	Parent and child T1 > T2

Note. ns denotes non-significant results. SoC denotes stage of change.

Healthy Food Consumption

Characteristics of the sample over time. Table 6 presents baseline and follow-up healthy food consumption across three generations. Daily fruit consumption was similar at both time points (shown in parentheses), but differed between grandparents, parents, and children. GLiMM analysis revealed fruit consumption was significantly greater for children than parents and was also greater for grandparents than parents. Refer to Appendix 5.A for

the significant GLiMM results showing differences in all food consumption variables when compared between generation, gender, time, and/or condition.

Table 6.

Healthy Food Consumption across generations at Time One and Time Two.

Variable	Generation	Time	*N*	*M*	*SD*	Median	Range	Min	Max
Fruit (serves/day)	Child	T1	62	2.74	2.05	2.00	10.00	0.00	10.00
	Child	T2	62	2.89	2.39	2.00	12.00	0.00	12.00
	Parent	T1	60	1.95	1.17	2.00	06.00	0.00	06.00
	Parent	T2	60	1.76	0.95	2.00	04.00	0.00	04.00
	Grand	T1	49	2.90	2.32	2.00	14.00	0.00	14.00
	Grand	T2	49	3.67	2.75	3.00	12.00	1.00	12.00
Veg (serves/day)	Child	T1	62	2.28	2.46	3.00	13.50	0.50	14.00
	Child	T2	62	3.70	2.61	3.00	13.00	1.00	14.00
	Parent	T1	60	3.30	1.33	3.00	05.50	0.50	06.00
	Parent	T2	60	3.33	1.27	3.00	05.00	1.00	06.00
	Grand	T1	49	3.67	2.75	3.00	13.00	1.00	14.00
	Grand	T2	49	3.33	2.21	3.00	09.00	1.00	10.00

Generational influence was further examined by discriminating between genders within generations (e.g., grandparents become grandmother and grandfather). Table B.1 in Appendix 5.B shows three generations by gender's fruit and vegetable consumption at time one and time two. Vegetable consumption was similar between grandparents, parents, and children. Although children's consumption increased over time, GLiMM analysis did not reveal any significant differences. The subsequent interaction between gender and generation revealed son's vegetable consumption was greater than grandfathers, and the results for gender showed female's vegetable consumption was greater than males overall (see Figure 1).

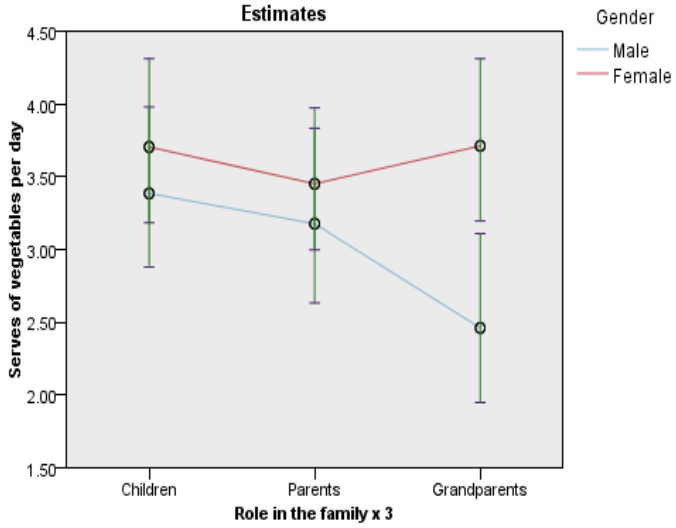


Figure 1. Vegetables serves per day, significant effect of generation by gender: females > males; and a significant effect of generation by gender: sons > grandfathers.

Healthy food consumption correlations: All correlations between generations, and between gender-by-generations on each of the food consumption variables are presented in Appendix 5.C. To summarize the healthy food results; parents and children shared 8% and 9% of the variance in fruit consumption at baseline and follow up respectively. Parents and children shared more similarity in vegetable consumption; 22% at baseline and 15% at follow

up[49].

Healthy food and family disease risk: Comparing generations. A series of longitudinal models determined differences healthy and unhealthy food consumption between disease risk groups, condition, and generations over time. These results are summarized in Table 7.

[49] Tests of significant differences between correlations over two time points, although a simple calculation, breached the independent samples assumption of normality in this clustered sample and were therefore not conducted (Pallant, 2011). Differences were therefore analysed using GLiMM analysis (Heck et al., 2014a).

Table 7. Summarizes the GLiMM analyses showing significant food consumption differences by risk group, generations and condition.

Risk / **Disease category**	**Average Risk**	**Above-Average Risk**	**Diagnosed**
Family member at risk of any disease	Fast: experimental group child > grand	Fruit: grand > parents Snacks: experimental group parents & grand > child Fast: experimental group child > grand	ns
Mother at risk of any disease Fruit: child & grand > parents	Fruit: controls T2 > controls T1	Fruit: experimental group grand > parents Fast: experimental group child > grand	ns
Individual at risk Colorectal cancer	ns	ns	ns
Individual at risk Breast cancer	ns	ns	ns
Individual at risk Diabetes	ns	ns	ns
Individual at risk Heart disease	ns	ns	Snacks: average and above average risk > diagnosed

Note. ns denotes non-significant results.

In the healthy food results, the next analysis examined family-risk-by-condition-by-generation, excluding the variable 'time'[50]. Grandparents in the experimental condition who had a family member at above-average disease risk showed greater fruit consumption than parents (in the experimental condition that also had a family member at above-average disease risk). Table 8 and Figure 2 show significant differences in fruit consumption between

[50] Due to computational and software limitations, all variables of interest could not be entered into the same model at once (i.e., family-disease-risk*condition*time*generation).

condition, generation, and family-member-at-risk.[51]

[51] Family member at risk of disease and vegetable consumption results were non-significant and were omitted.

Table 8

*Fruit Model 1.1: Condtion*Family Risk of any Disease*Generation, Differences in Consumption.*

Effects	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 1.1				(2, 335)			5.63	**.004**
Condition				(1, 335)			0.59	.440
	Treatment - control	-0.31	-0.91	(1, 335)	-1.10	0.48		.440
Generation				(2, 335)			3.42	**.030**
	Child - parent	0.82	2.47	(1, 335)	0.02	1.62		**.040**
	Child - grand	0.31	0.87	(1, 335)	-0.39	1.00		.390
	Parent - grand	-0.52	-1.71	(1, 335)	-1.19	0.16		.180
Family Disease Risk				(2, 335)			0.63	.540
	Diagnosed - average risk	-0.61	-0.97	(1, 335)	-2.07	-0.85		.800
	Diagnosed - above average risk	-0.62	-1.11	(1, 335)	-1.97	0.73		.800
	Above average risk - average risk	0.01	0.04	(1, 335)	-0.73	0.75		.970
Condition*Family risk* Generation [a]								
Treatment-Above average risk				(2, 335)			3.6	**.028**
	Treatment:Above average risk- Child - parent	0.52	1.62	(1, 335)	-0.20	1.23		.210
	Treatment: Above average risk- Child - grand	-0.54	-1.33	(1, 335)	-1.37	0.29		.210
	Treatment: Above average risk- Parent - grand	-1.06	-2.60	(1, 335)	-2.04	-0.08		**.029**

Note . GLiMM negative binomial vertical multilevel modeling. Model 1.1: -2 log pseudo likelihood = 736.854. [a] only significant interactions are shown.

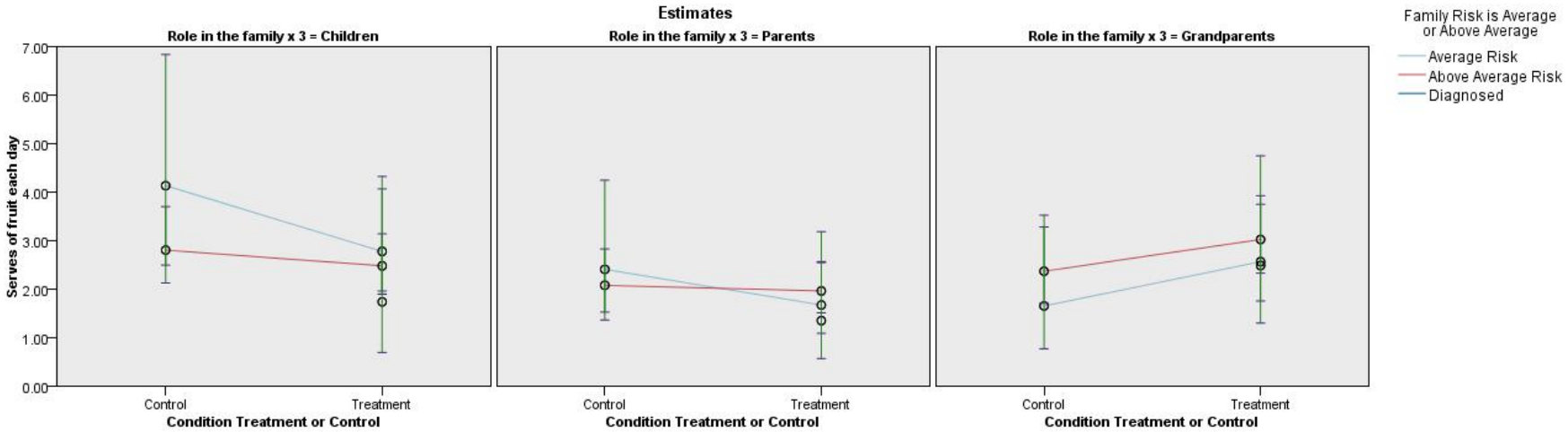


Figure 2. Fruit Model 1.1: Family risk of any disease, interaction condition*family-risk*generation showing above average risk grandparents greater fruit consumption than above average risk parents.

Healthy food and mother's disease-risk: Comparing generations. For the analysis examining generational differences when mothers in the family were at risk of chronic disease see the GLiMM tables and figures in Appendix 5.D. Briefly, with regard to healthy food consumption, in the experimental condition above-average risk group grandparents showed greater fruit consumption than parents (in the same at-risk group).

Unhealthy Food Consumption

Characteristics of the sample over time. Table 9 presents unhealthy food consumption descriptive results for the three generations, and Table E.1 in Appendix 5.E presents unhealthy food for three generations further divided by gender.

Table 9.

Three Generations of Unhealthy Food Consumption at Time One and Time Two.

Variable	Generation	Time	*N*	*M*	*SD*	Median	Range	Min	Max
Snacks (serves/wk) [a]	Child	T1	63	4.63	3.44	4.00	17.00	0.00	17.00
	Child	T2	63	5.48	4.95	4.00	21.00	0.00	21.00
	Parent	T1	60	3.97	3.09	3.50	18.00	0.00	18.00
	Parent	T2	60	3.51	2.97	3.00	14.00	0.00	14.00
	Grand	T1	49	3.90	2.98	4.00	14.00	0.00	14.00
	Grand	T2	49	3.56	2.74	3.00	14.00	0.00	14.00
Fast (serves/wk) [a]	Child	T1	63	1.05	1.61	1.00	10.00	0.00	10.00
	Child	T2	63	1.02	1.44	1.00	7.00	0.00	7.00
	Parent	T1	60	0.72	1.61	0.00	12.00	0.00	12.00
	Parent	T2	60	0.52	0.62	0.00	3.00	0.00	3.00
	Grand	T1	49	0.12	0.33	0.00	1.00	0.00	1.00
	Grand	T2	49	0.22	0.42	0.00	1.00	0.00	1.00

Note. [a] Skew positive, interpreting median and range will be most meaningful.

Snack consumption among the adult generations reduced over time, whereas children's snack consumption was the similar on both occasions with children consuming more than their parents. GLiMM analysis results for differences between generation by gender showed daughter's snack consumption was greater than that of mothers and grandmothers (for the interaction figures refer back to Appendix 5.A). In Appendix 5.F, the odds ratio results are explained using multiple logistic regression analysis[52]. The logit of snack consumption being above the sample average relative to being below the sample average was 0.71 times lower when all predictors were at zero (intercept B = -0.71, Exp(B) = 0.49, p = .04). The predictor 'time' was significant: the logit of snack consumption being above-average was 1.7 times greater at baseline relative to follow up (B = 0.52, Exp(B) = 1.67, p = .013).

Over time, fast food consumption reduced in parents and children, whereas grandparents' consumption remained the same. GLiMM analysis revealed generational differences in children's fast food consumption that was greater than parents and grandparents; and parent's consumption was greater than that of grandparents. Generation by gender analysis showed son's fast food consumption was greater than grandfathers, and daughter's consumption was greater than mothers and grandmothers (for the interaction figures see Appendix 5.A).

Unhealthy food consumption: Comparing generations. To summarize the unhealthy food results; snack consumption showed 8% shared variance between parents and grandparents at baseline, and 13% at follow up (refer back to Appendix 5.C for all food consumption correlation matrices). Fast food showed 10% shared variance between parents and grandparents at baseline (< 1% and non-significant at follow up), and 12% between

[52] Odd ratio analysis with healthy food was non-significant and were omitted.

parents and children at follow up (< 1% and non-significant at baseline). The previous ICC results also confirmed the highest within-family influence occurred in fast food consumption. Using odds ratios, results showing generational differences in fast food consumption were confirmed with the likelihood (logit) of fast food consumption being 5.4 times greater in children relative to grandparents (B = 1.69, Exp(B) = 5.43, $p < .001$), and 3.9 times greater in parents relative to grandparents (B = 1.36, Exp(B) = 3.89, $p < .001$), see Appendix 5.F for all logistic regression food consumption results.

Unhealthy food and family disease risk: Comparing generations. Results showed parents and grandparents snack food consumption was significantly less than that of children in the experimental condition who had a family member at above-average disease risk. In fast food consumption, the interaction revealed results for participants in the intervention condition: grandparents with a family member of average or above-average disease risk consumed significantly less fast food than grandchildren within the same at-risk families. Relevant result tables showing unhealthy food consumption associated with family-risk are presented in Appendix 5.G.

Unhealthy food and mother's- disease-risk: Comparing generations. Comparisons of unhealthy food consumption in the experimental condition between generations indicated that children showed greater fast food consumption than grandparents. For all results of generational differences when the mother in the family was at risk of chronic disease refer back to the GLiMM tables and figures in Appendix 5.D.

Food Life Behaviours and Attitudes

Characteristics of the sample over time. Similar scores were observed on the diet-health oriented behaviour (DHOB) and diet-health/disease linked attitudes (DHLA) subscales of the FLQ-SF (Sharp et al., 2013) over time. For example, within the total sample, mean scores on DHOB ($M = 4.86$, $SD = 0.91$) and DHLA ($M = 5.83$, $SD = 0.95$) were greater than

the median score of 3.50 (i.e., 3.50 was the median in a range of possible scores from one to seven, low to high).

Intraclass correlations in diet-related food attitudes reveal the extent that behaviours occur as whole families, or as independent individuals. Intraclass correlations (ICCs) were conducted to examine the within-family effects on both Food Life behaviour and attitude subscales. ICCs for DHOB and DHLA were all less than 0.2% indicating that individuals within-family behaviours and beliefs were all effectively independent of each other (see Table 10).

Table 10.

Food Attitudes

Shown are the interclass correlation (ICC) results. These indicate the variability in food attitudes associated with differences between families as a percentage. Wald Z shows Model 1 result significance (GLiMM negative binomial output).

Random Effects Subject	Model	Within group variance (Residual Estimate)[a]	Between group variance (Intercept Estimate) [b]	Standard Error	95% CI		Wald Z	ICC [c]	Variance %	*p*
					Lower	Upper				
DHOB T1	5.1	1	0.002	0.002	0.000	0.018	0.855	0.00061	0.06%	.392
DHOB T2	5.1	1	0.006	0.004	0.002	0.019	1.619	0.00182	0.18%	.106
DHLA T1	6.1	1	0.002	0.002	0.000	0.011	1.119	0.00061	0.06%	.263
DHLA T2	6.1	1	0.003	0.002	0.001	0.011	1.422	0.00091	0.09%	.155

Note. Calculation: ICC = b/(a + b) Tabachnick & Fidell (2012) p.826-7. However, when the residual variance is 1.00 the scale factor can be used to calculate an ICC $\pi^2 / 3 = 3.29$, therefore [a] = 3.29 and the equation used was ICC [c] = b/(a + b) Heck et al. (2012) p. 157.

Food Life behaviour and attitudes: Generation and gender differences. The diet-related food attitude statistics of the total sample and between generations are presented in Table 11, between gender comparisons are presented in Table 12 and, finally, table H.1 in Appendix H shows between gender and generation descriptive statistics.

Table 11.

Diet-health Food Attitude Mean Scores of the Total Sample and of Three Generations.

Food Attitudes	Generation/s	Time	*N*	*M*	*SD*	Median	Range	Min	Max
DHB	Total sample	T1	178	4.80	0.89	4.80	4.80	2.20	7.00
	Total sample	T2	176	4.81	0.93	4.80	4.00	3.00	7.00
DDB [a]	Total sample	T1	174	5.81	0.92	6.00	4.80	2.50	7.00
	Total sample	T2	174	5.90	0.94	6.00	5.00	2.00	7.00
DHB	Child	T1	62	4.36	0.88	4.40	4.40	2.20	6.60
	Child	T2	62	4.37	0.96	4.20	4.00	3.00	7.00
	Parent	T1	62	5.04	0.77	5.00	3.20	3.40	6.60
	Parent	T2	62	4.97	0.85	5.00	4.00	3.00	7.00
	Grand	T1	48	5.08	0.81	5.00	3.60	3.40	7.00
	Grand	T2	48	5.14	0.80	5.10	4.00	4.00	7.00
DDB [a]	Child	T1	62	5.47	0.90	5.50	4.50	2.50	7.00
	Child	T2	62	5.59	0.92	5.71	5.00	3.00	7.00
	Parent	T1	62	6.09	0.80	6.25	2.67	4.33	7.00
	Parent	T2	62	6.19	0.73	6.13	2.00	5.00	7.00
	Grand	T1	48	5.91	0.99	6.13	3.75	3.25	7.00
	Grand	T2	48	5.98	0.93	6.25	2.00	5.00	7.00

Note. [a] Skew negative, interpreting median and range will be most meaningful.

Table 12.

Males and Females Diet-health Food Attitude Mean Scores at Time One and Time Two.

Variable	Gender	Time	*N*	*M*	*SD*	Median	Range	Min	Max
DHB (scores 1 - 7)	Male	T1	69	4.61	0.86	4.60	4.40	2.20	6.60
	Male	T2	69	4.61	0.90	4.60	4.00	3.00	7.00
	Female	T1	103	4.93	0.89	5.00	4.20	2.80	7.00
	Female	T2	103	4.92	0.94	5.00	4.00	3.00	7.00
DDB (scores 1 - 7) [a]	Male	T1	69	5.61	0.97	5.76	4.50	2.50	7.00
	Male	T2	69	5.81	0.91	6.00	5.00	3.00	7.00
	Female	T1	103	5.95	0.87	6.00	3.25	3.75	7.00
	Female	T2	103	5.99	0.87	6.00	4.00	4.00	7.00

Note. [a] Skew negative, interpreting median and range will be most meaningful.

Food Life behaviours and attitudes: Resemblances between generation dyads.

Appendix 5.I shows Food Life behaviour and attitude correlation tables between three generations. The results showed 10% of the variance in diet-health oriented behaviours (DHOB) was shared between parents and grandparents at baseline. Then at follow up 12% of the variance was shared between parents and children, with medium correlations for both results. Diet-health/disease linked attitudes (DHLA) had a 10% shared variance between parents and grandparents at follow up. A summary of all significant Food Life behaviour and attitude correlation results is presented in Table 13, showing the percentage of the variance explained in each food consumption variable (in parentheses).

Table 13.
Summary of Significant Correlation Results - Variance in Food Attitudes (%).

Factor	Diet-health oriented behaviours DHOB (%)		Diet-health/disease linked attitudes DHLA (%)	
	Baseline	Follow up	Baseline	Follw up
Generations	Parent-grandparents (10)	Child-parent (12)	ns	Parent-grandparents (10)
Generation by gender	Siblings (16) Mothers-grandfathers (32)	Daughter-mother (13) Parent partners (28)	Parent partners (14)	Siblings (56)
Fruit	Children (13) Sons (29)	Children (14) Sons (24) Daughters (15)	Sons (14) Grandparents (14)	Grandparents (9) Grandfathers (18)
Vegetables	Children (20) Sons (34) Daughters (12) Grandmothers (14)	Grandparents (9)	ns	Sons (21) Grandmothers (17)
Snacks	Children (-12) Daughters (-19)	Children (-12) Daughters (-28) Grand (-12) Fathers (-20)	ns	Fathers (-26)
Fast	Parents (-8) Mothers (-10) Fathers (-20)	Children (-16) Daughters (-20) Mothers (-8) Parent (-15)	ns	ns

Note. Positive percentage indicates positive correlation, negative percentage indicates negative correlation; ns denotes non-significant.

Food Life behaviours and attitudes with healthy food consumption: Comparing generations. Appendix 5.J shows the correlation result tables of Food Life behaviours and attitudes with healthy and unhealthy food consumption between three generations and gender at baseline and follow up. All healthy food correlations with Food Life behaviour and attitudes were positive, confirming correlation results of Study 2 that predicted more positive Food Life behaviours and attitudes would be associated with greater consumption of healthy food. In sons, 29% of the variance in fruit consumption was linked to diet-health oriented behaviours (DHOB) at baseline and 24% at follow up; daughters had 15% shared variance at follow up and no significant correlation at baseline. Sons (at baseline) and grandfathers (at follow up) shared 14 and 18 percent respectively of the variance in fruit consumption with diet-health/disease linked attitudes (DHLA). Vegetable consumption among sons, daughters and grandmothers shared 34, 12, and 14 percent of the variance at baseline respectively with higher scores on diet-health oriented behaviour (DHOB), while at follow up, sons and grandmother's diet-health/disease linked attitudes (DHLA) accounted for 21 and 17 percent of the variance respectively (refer back to Table 13 for the results).

Food Life behaviour and attitudes of parents and the healthy food consumption of offspring. When correlating the Food Life behaviour and attitudes of parents with the food consumption of offspring, Study 2 mother-daughter results were confirmed in the present study with a stronger negative correlation in mother's DHOB and daughter's fruit consumption (see Table 14 and Appendix 5.K for all result tables). Although father's DHLA with son's fruit, and daughter's vegetable consumption (respectively) were not repeated at follow up, the present study revealed a new finding that positively correlated fathers DHOB with son's vegetable consumption.

Table 14.

Spearman's rho correlations of parent's mean diet-health orientated behaviour food attitude subscale scores with the healthy food consumption of offspring at time one and time two.

Healthy/DHOB	T1	T2	T1	T2	T1	T2	T1	T2
FLQ-SF Subscale	Child Fruit Consumption				Child Vegetable Consumption			
	Son	Son	Daughter	Daughter	Son	Son	Daughter	Daughter
Mother's DHOB	-.217	.196	**-.462****	.021	-.282	.322	-.203	-.327
n	23	23	26	26	23	23	26	25
Father's DHOB	.102	.085	-.090	.418	**.443***	.402	.262	.241
n	16	16	14	14	16	16	14	13

* $p < 0.05$ one tailed. ** $p < 0.01$ one tailed.

[53]

[53] Remaining non-significant parent food attitude and offspring's food consumption results may be viewed in Appendix I (all unhealthy food consumption results were non-significant).

Food Life behaviour and attitudes with unhealthy food consumption: Comparing generations. All unhealthy food correlations with Food Life behaviour and attitudes were negative; confirming Study 2 results that also showed a decrease in Food Life behaviour and attitudes correlating with reduced unhealthy food consumption. Daughter's diet-health oriented behaviour (DHOB) explained 19 and 28 percent of the variance in snack consumption at baseline and follow up; while fathers accounted for 20% at follow up only. Fathers diet-health/disease linked attitudes (DHLA) explained 26% of the variance in snack consumption at follow up. Mother's diet-health oriented behaviours (DHOB) accounted for 10 and 8 percent of the variance in fast food consumption at baseline and follow up; fathers 20% at baseline only; and daughters 20% at follow up only.

Food Life behaviour and attitudes and chronic disease risk. Appendix 5.L shows the pseudo-log-likelihood chronic disease risk results of Food Life behaviour and attitudes on each food consumption variable. Results indicated significant contributions of the Food Life behaviours and attitudes DHOB and DHLA to healthy and unhealthy food consumption at the $p < .001$ level[54] when a family member- or mother- was at increased risk.

Discussion

Healthy Food Consumption

As stated in Study 3 Part A; the present study sample's fruit and vegetable consumption was greater than that of the Australian population (ABS, 2014a). Generational differences were evident showing that children and grandparents consumed more fruit than parents. Study 1 indicated that some parents reported being time-poor and this perception

[54] Additional GLiMM analyses examined whether the food attitudes DHOB and DHLA improved upon the previous longitudinal 'family-risk' and 'mother-risk' GLiMM models for healthy and unhealthy food consumption. Pseudo log likelihood (-2LL) ratio tests (Sakamoto, Ishiguro, Kitagawa, Dordrecht, & Reidel, 1988) were used to determine differences between models, followed by chi square tests of significance which revealed food attitudes significantly added to each of the earlier models, see Appendix J for all results tables.

impacted upon their meal preparation. Previous research has suggested perceived time scarcity experienced by mothers can affect the quality of family food provision (Jabs et al., 2007). Conversely, research also indicates that working mother's confidence rather than time pressure is more likely to impact family meal quality (Beshara, Hutchinson, & Wilson, 2010). However, fruit does not constitute a meal and parents in the present study may have inadvertently neglected their own needs for healthy fruit in their own work day, while ensuring their children received adequate fruit for school lunches or as a bite to eat after school. This could feasibly occur when parents prepare school lunches for children and include fruit in their lunch boxes, whereas Study 1 results suggested parents own breaks at work were hurried, or missed. It is possible that the grandparent generation, many of whom have retired, have more time to prepare and consume healthy fruit without the added responsibility of dependent children or employment. Further intergenerational research could consider fruit consumption comparisons between cohorts and target parents increased fruit consumption if found to be inadequate.

An unexpected result evident in Study 2 and confirmed in the present study was a negative correlation between mother's diet-health oriented behaviour and daughters' fruit consumption, indicating that mother's self-reported healthy dietary behaviours had contrary associations with daughter's fruit consumption. In Study 2 it was noted that mother's diet-health oriented behaviour was not associated with their own food consumption, suggesting that a different motivating factor was playing a role. It is plausible that daughters may resist their mother's efforts to encourage fruit consumption if the underlying reason is incongruent with their own values (e.g., weight concern). Future research may consider examining differences in perceptions of the Food Life subscale 'weight concern' between mothers and daughters and any impact on food consumption.

Vegetable consumption was the only outcome variable that showed a gender difference with female's consumption greater than that of males. Study 1 and 2 suggested females' healthy food consumption was greater than males, and Study 3 confirms the important role of mothers chief decision-making regarding the family diet (Beydoun & Wang, 2009; Green et al., 2003; Green et al., 2009; Prelip et al., 2012; Wroten et al., 2012). In the present study, son's vegetable consumption was greater than grandfathers possibly because children's vegetable consumption was considered important to mothers and grandmothers in Study 1. The positive father-son association between attitudes that diet can affect health or disease and vegetable consumption shown in Study 2 at baseline was not repeated at follow up in the present study. However, father's own diet-health oriented behaviour and role-modeling was reflected in their son's fruit consumption in Study 3. Motivating Australians to increase mediocre fruit consumption and poor vegetable consumption rates (e.g., ABS, 2014a) requires more research to determine the underlying mechanisms that moderate healthy dietary behaviours, and to utilize any positive influences if consumption recommendations are to be reached.

Unhealthy Food Consumption

Conversely, the whole sample's snack consumption showed a reduction at follow up when compared to baseline. When examined more closely, parent and grandparent generations reduced snack consumption over time, by half- and one serve- per week respectively, whereas children's level of snack consumption remained constant. Consistent with Study 2, children's consumption was again greater than that of parents and grandparents. When broken down further, significant effects of generation by gender showed daughter's snack consumption was greater than mothers and grandmothers. This was not surprising given that grandparents were also shown to indulge children in snacks in Study 1, without consuming the same snacks or treats themselves.

At baseline in the present study grandmothers and grandfathers' snack consumption was strongly correlated, although the same correlations reduced to non-significance at follow-up. This result may reflect differential impacts of participation in the study so that grandmothers' consumption was improved while grandfathers did not change. Grandparents showed a moderate negative correlation between snack consumption and diet-health oriented behaviour (DHOB) at follow up that was non-significant at baseline. Alternatively, these results could be interpreted as a development in grandparents' healthier dietary behaviours over time. Together the absence of a snack consumption association and an emergent negative diet-health oriented behaviour correlation with snack consumption over time in the older generation suggests that a positive learning experience occurred in response to the Families SHARE workbook. Previous research has shown diet-health oriented behaviour is a strong negative predictor of unhealthy food consumption in a sample population aged between 17 and 88 years (Sharp et al., 2013), however, to date any effects between generations has not been compared.

Fast food, the other unhealthy food type, reduced at follow up compared to baseline in parents and children although remained constant for grandparents. This outcome was most likely because grandparent's fast food consumption averaged less than once a week at both time points, and floor effects prevented any further reduction over time. Consistent with the results of Study 2, children's fast food consumption was greater than parents and grandparents, and again parent's consumption was greater than grandparents. This suggests that with each younger generation fast food was more acceptable and was consumed more frequently. In this thesis so far, grandparents have been shown to consume little or no fast food (i.e., across Study 1, 2 and 3) indicating an apparent generational cohort effect. One plausible explanation is that grandparents have had the least childhood exposure to fast food of all the three generations, and having indicated a preference for 'plain food' in Study 1, it is

unlikely that grandparents ever developed a taste for fast food (since food preferences that develop in childhood can persist well into adulthood e.g., Contento et al., 2006; Krolner et al., 2011). Hence, there is the potential for negative health impacts for the current parent and child generation, who, across their lifetime have developed a taste for fast food. It is predicted that 25% of the Australian population will be aged over 65 years by 2050 (Michael et al., 2014), therefore there will be a proportional increase in older people consuming fast food as today's parents become older people. Furthermore, results of Study 2 and the present study confirmed that the largest family intraclass correlations occurred in fast food consumption; with almost 10% of the variance explained in this outcome variable on both occasions. Intraclass correlations showed within-family consumption correlations were quite strong[55] for fast food, having the disturbing potential for long-term within-family effects on the future generation of children also.

Overall, the present study showed reduced unhealthy food consumption over time more so than an increase in healthy food consumption. There were limited significant results in vegetable consumption apart from gender differences, and the noted absence of results confirms earlier suggestions that increasing healthy food consumption is of critical importance to Australians' future health status. Since 66% of Australians are currently either overweight or obese (NHMRC, 2013b), maintaining a healthy weight by *limiting the intake of unhealthy food* could be the focal issue for most people. Therefore, current perceptions of healthy eating may be viewed as predominantly *reducing* the intake of unhealthy foods, rather than *increasing* the intake of healthy food. Future research could investigate whether barriers to increasing healthy food consumption are based on perceptions of negative consequences such as weight gain, instead of having positive consequences for improved

[55] Inferences on effect size from intraclass correlations as discussed in Heck et al. (2012); McGraw and Wong (1996); and Srivastava (1984).

health outcomes and goals that prevent chronic disease.

Chronic Disease Risk

In the intervention group for individuals with a family member at increased risk for at least one of the four diseases some consumption behaviours varied between generations: grandparents had greater fruit consumption than parents; parents and grandparents consumed fewer snacks than children; and grandparents consumed less fast food than children. Results with adults in previous American family health history interventions have shown moderate increases in fruit and vegetable consumption (e.g., Hovick et al., 2014; Ruffin et al., 2011) and, as predicted in the present study, showed that adults were more likely than children to respond to having a family member at increased risk of disease and subsequently modify their diet by consuming more fruit and fewer snacks or fast food meals. The significance of family health history is less likely to be understood by children than it is by adults, nevertheless, the influence of adults through modeling healthier dietary behaviours to children would have additional benefits for the child generation over a longer period of time (Bandura, 1977b; Brown & Ogden, 2004; Bussey & Bandura, 1984; Ritchie, Welk, Styne, Gerstein, & Crawford, 2005). In the present study, Australians' vegetable consumption remained static, confirming the complex challenge faced by future disease prevention initiatives.

It has been suggested that disease risk in the family is more salient as a motivation for behaviour change among older adults (people are generally older when developing chronic disease, or have more advanced disease e.g., Oertelt-Prigione, Kendel, Lehmkuhl, Hetzer, & Regitz-Zagrosek, 2014). Although parents are aging, the threat of chronic disease may seem less immediate to them than it is to their own parents. This observation is consistent with research examining individual differences in measures of Consideration of Future Consequences and how distal disease outcomes impact current health behaviour (Joireman, Shaffer, Balliet, & Strathman, 2012; Strathman, Gleicher, Boninger, & Edwards, 1994). In

other words, where health messaging identifies above-average disease risk older generations may be more responsive to advice on how this risk might be mitigated.

For individuals in the intervention group with a mother in the family at increased risk for at least one of the four diseases, consumption behaviour varied across generations in the following ways: grandparents had greater fruit consumption than parents; and grandparents consumed fast food less frequently than children. One could conclude that these results were simply an effect of age, although if that were the case, there would have been no significant differences between intervention and control groups. Grandparents in the intervention condition had healthier dietary behaviours than children or parents did. Although this effect was not apparent when these grandparents themselves were at increased risk, it was only when a family member (or the mother) was at increased risk of disease that healthy dietary behaviour change was noted to occur. This suggests the intervention was a success in motivating healthy dietary behaviour change within the grandparent generation under specific circumstances. These results support the view that talking about family health history and sharing tailored family health history feedback between family members heightened awareness in grandparents who had a family member or mother at above-average risk for disease.

Food Life Behaviours and Attitudes

Food Life behaviours and attitudes did not vary significantly between conditions, generations, gender, and risk groups over time. In the total sample, mean scores on self-reported healthy dietary behaviours (DHOB) and attitudes that diet can affect subsequent health or disease (DHLA) measures were greater than the median score (i.e., 5 out of a possible 7, the median was 3.5) at baseline, therefore at follow up changes over time in response to the intervention were unlikely to be significant due to ceiling effects. As observed in Study 3 Part A, this is another indication of the sample being healthier than the Australian

population, not only in healthy food consumption (i.e., fruit and vegetable consumption was greater than the nation's average), but in diet-health oriented behaviour and diet-health/disease linked attitudes as well. One explanation for high mean scores on Food Life behaviours and attitudes, as stated previously, could be that the sample population was attracted to this particular study due its focus on food and health. Different recruitment approaches could be used in future research that broadens the scope of participants beyond those with a pre-existing interest in food and health.

Theoretical Conclusions

The Families SHARE workbook aligns with the four original constructs of the Health Belief Model: susceptibility, severity, benefits and barriers (Janz & Becker, 1984). The two additional constructs: 'cues to action' and 'self-efficacy' (Bylund, Galvin, Dunet, & Reyes, 2011) were also measured by the present study. For example, predicted changes in food consumption that occurred in above-average risk grandparents within the experimental group suggests the Families SHARE workbook motivated behaviour change, and acted as a cue to action. In addition, participant's self-reported confidence ratings in increasing fruit and vegetable consumption confirmed self-efficacy. Self-efficacy responses established that more than three quarters of participants (77%) showed moderate to high confidence in achieving their intentions to improve healthy dietary behaviours in the future.

The intervention increased participants knowledge about four chronic diseases, how to rate familial risk, how to meet NHMRC dietary recommendations, how to access free or affordable screening services, and all of this information was provided within the Families SHARE workbook. Future intergenerational research would benefit from measuring perceived future outcomes (Evans, Wilson, Buck, Torbett, & Williams, 2006; Strathman et al., 1994), and how this variable may impact on actual or intended behaviour change in response to family health history information.

Conclusion

With the exception of vegetable consumption, the grandparent generation showed greater dietary behaviour improvements than other generations. This was demonstrated by greater fruit consumption and less frequent snack and fast food consumption. The combination of ageing and discussing family health history between family members were the most likely contributing factors explaining this outcome. Realistically, floor effects on grandparent's fast food consumption meant that any reduction over time was not feasible within the older generation. However, fast food and snack consumption results, as were indicated by behavioural changes evident within this sample, show promise for the overall reduction in unhealthy food consumption in response to the study. The significant reduction in unhealthy food consumption over time within the whole sample (irrespective of group assignment) for snacks, and within the parent and child generations in fast food consumption, indicate a possible questionnaire measurement effect (e.g.,Godin, Sheeran, Conner, & Germain, 2008). As was noted in Study 3 Part A, and recent research with family health history (e.g., Prichard et al., 2015), asking family health history questions at baseline may have acted as a cue to action for some people.

Correlations in self-reported healthy dietary behaviours (DHOB) were shared between parents and grandparents at baseline. At follow up, correlations between parents and children achieved significance. The explanation for this finding is unclear although it is possible that discourse within the family about health and diet was focused more so within the nuclear family (parent-child) after the intervention. If this translates into parents talking about healthy eating and modeling these behaviours to children, this positive influence may be seen in children's own dietary behaviours at a future time. Future research could therefore track the link between parental attitudes and child behaviour over a more extended time period.

It is interesting to note patterns of associations when analysing attitudes that diet

affects subsequent health or disease. Parent-child resemblance in diet-health/disease linked attitudes (DHLA) observed at baseline differed at follow up with significant parent-grandparent correlations reported instead. This may suggest that the study motivated parents to talk with grandparents about the links between diet and disease and learn from this. Food Life behaviours and attitudes made significant contributions to food consumption when a family member or mother was at risk of chronic disease and it is suggested that future research examine the potential moderating influence of food attitudes on food consumption when there is an increased risk of disease within the family. Finally, the results supported the Health Belief Model constructs that underpin this thesis throughout, and it is suggested that adding 'consideration of future consequences' could enhance future intergenerational research when including family health history as a motivating factor.

CHAPTER 6: STUDY 4

Title: Evaluation of the Families Sharing Health Assessment and Risk Evaluation (Families SHARE) Workbook

Background

Study 3 Parts A and B investigated changes in dietary behaviours over time in response to the Families Sharing Health Assessment and Risk Evaluation (Families SHARE) workbook among three generation families. Differences were examined between identified chronic disease risk groups (average, above-average, or diagnosed); and according to which family member was at risk (the individual responding-, any family member-, or the mother-at-risk). The main findings showed that, participants in the experimental condition's fruit consumption progressed to the action stage of change; when participants had a family member at above-average disease risk; grandparents consumed more serves of fruit per day than parents; vegetable consumption remained unchanged over time; parents and grandparents consumed more snacks than children; and children consumed more fast food than grandparents. These results therefore provided only marginal support for the efficacy of family health history information provision as a tool to motivate lifestyle behaviour change within families.

When evaluating these results it is important to note that the Families (SHARE) workbook was designed by US researchers Koehly, Morris, Skapinsky, Goergen, & Ludden (2015), and was adapted for Australians based on current national health, nutrition and disease screening guidelines (refer to Chapter 4 Method section for a description of, and Appendix 4.A to see a copy of, the entire workbook). In order to substantiate findings from Study 3 Parts A and B, it is important to evaluate the acceptability and useability of the Families SHARE workbook quantitatively and qualitatively. Previous research has evaluated RCTs with qualitative studies conducted alongside, revealing few included theory, or

attempted to explain the RCT results. Consistent with the best practice advice of Lewin, Glenton, and Oxman (2009), Study 4 seeks to further explain the results of Study 3 Parts A and B, by incorporating the theory that underpins this thesis in the integration of the quantitative and qualitative data via a mixed methods evaluative study.

As detailed in Chapter 1, the Health Belief Model (HBM) may be used to understand health motivation and behavioural change in chronic disease prevention. According to the model, the benefits of engaging in health enhancing, or the disadvantages of engaging in health compromising behaviours depend upon an individual weighing up the perceived risk and severity of a potential disease with the likely benefits and barriers of taking any relevant health action (Conner & Norman, 1995; Harrison et al., 1992). In general, it is widely accepted that individual behaviour change can be predicted by using this model (Strecher & Rosenstock, 1997). The HBM was later extended (EHBM) to incorporate the constructs 'cue to action' and 'self-efficacy' (Bylund et al., 2011). By also accessing the benefits of family relationships and joint decision making, the cluster RCT in Study 3 predicted that collective agency would be channelled into motivating others in the family to engage in healthy dietary behaviours (Bandura, 2001; Hendriks et al., 2012).

The Present Study

Study 4 aims to explore whether providing information about family risk for four chronic diseases in a workbook depicting disease severity, healthy lifestyle behaviours and screening information, will be evaluated as effective in motivating three generation families to improve lifestyle and screening behaviours that contribute to disease prevention, in accordance with predictions arising from the HBM. Using a mixed method design, Study 4 will evaluate the usability and motivating nature of the Families SHARE workbook by firstly, examining the utility of the workbook as revealed in a survey of participants from the experimental condition; and secondly, by using semi-structured family interviews with a

subset of experimental group families who returned the evaluation questionnaire. The procedural flow diagram is provided in Figure 1.

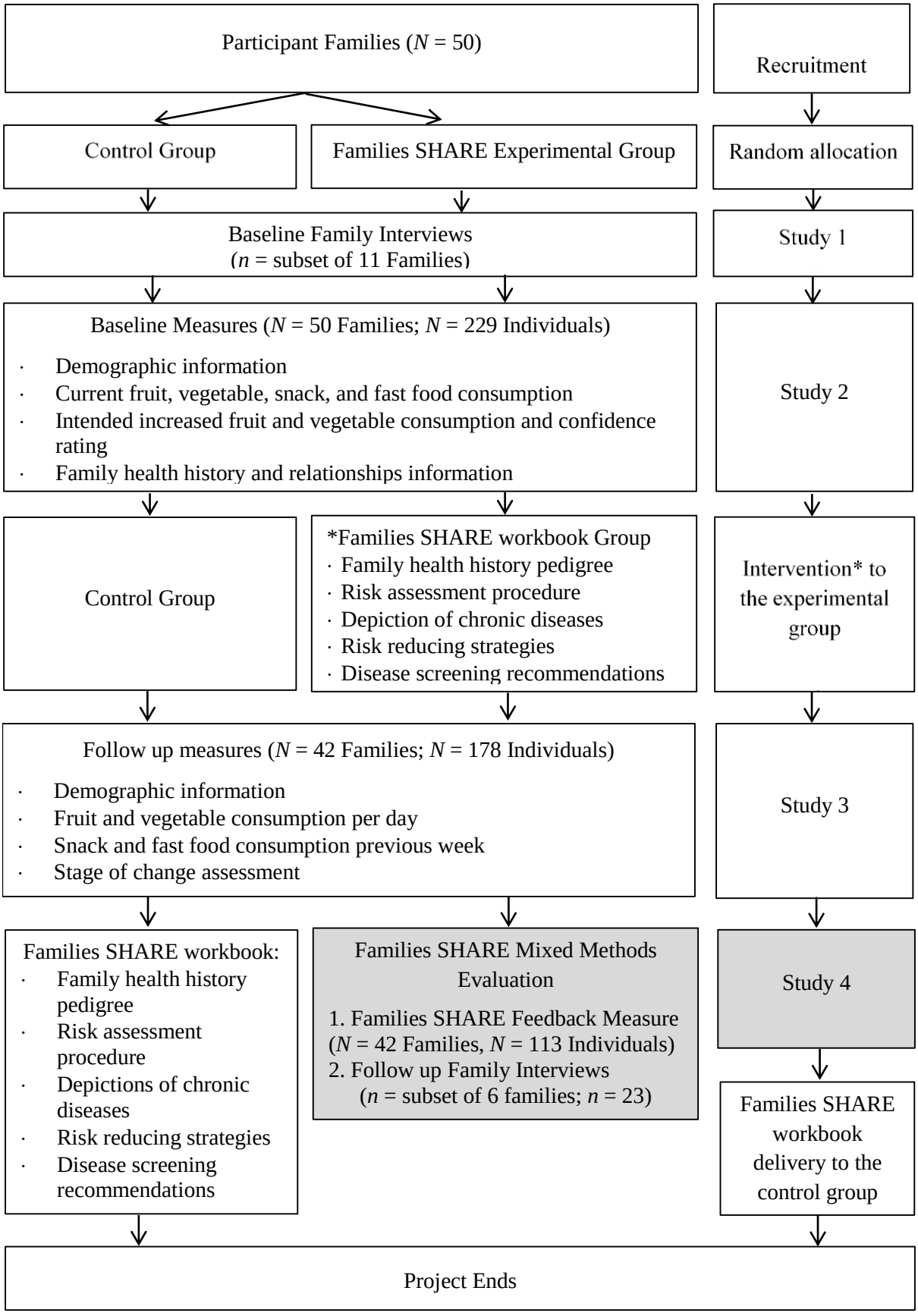


Figure 1. Procedural flow diagram showing Study 4 in relation to previous studies.

Method

Participant evaluation data from the experimental group (n = 113 individuals) were collected via a survey at follow-up (6 months after baseline assessment). Semi-structured family interviews with a subset of families (n = 6) from the experimental condition were subsequently conducted using the same method as Study 1 and involving a total sample of 23 participants.

Methodology

Study 4 is a mixed method evaluation of the Families SHARE workbook. Data from mixed methods have been shown to effectively investigate one research question utilising different forms of data. This process, known as 'triangulation', increases the validity of a study by the validating and extending information obtained each method (Greene, Caracelli, & Graham, 1989). Although qualitative and quantitative data differ markedly, if integrated well, broader insight can be gained than when using just one approach (Brannen, 2005). In Study 4, the quantitative, qualitative data collection, and results sections are outlined separately, followed by the integrated interpretation utilising both components together in the discussion section.

1. Survey Data Collection

Participants. The survey sample consisted of 27 three generation Australian families who were recruited using the same methods as Study 2 and 3. Eligibility for participation in this component of the study was age 18 or above. There were 189 individuals in the experimental condition, 113 of whom completed the Families SHARE evaluation questionnaire, which represented 60% of participants due to the exclusion of children (n = 113) from this particular survey.

Materials. *Family health history workbook (Families SHARE) evaluation survey.*

Participants in the treatment condition were asked to answer several questions about their experience of using the Families SHARE workbook. Questions asked whether individuals could assess their own degree of risk for the four diseases: colorectal cancer, breast cancer, heart disease and type 2 diabetes, and whether they were at above-average risk. The response options were "*yes*", "*no*," and "*don't know*." Participants were then asked to indicate if the workbook had been shared with a GP, other health care provider, family member, friend, or another person. They were also asked whether their family health history pedigree diagram had been updated in any way after receiving it, and finally, whether any health screening for the four diseases had occurred in the preceding six months (see Appendix 6.A for a copy of the survey).

Data analysis. Data were analysed using SPSS version 22 (IBM Corporation, Released 2013) to calculate frequencies, descriptives, correlations, and Kappa's Measure of Agreement. The alpha value was set at $p < .05$ and Bonferonni corrections were included in all statistical analysis to maintain an accurate Type 1 error rate.

2. Interview Data Collection

Participants. A subset of 23 individuals (6 families) from the survey sample participated in the Families SHARE family interviews as part of a more unstructured assessment of the workbook and its use. Children under the age of 11 were excluded due to the volume and complexity of the written material presented in evaluation interviews. Average family interview group size was four, with a range of between four and five family members across three generations. Families comprised: three boys, aged 15 to 18; five girls, aged 11 to 16; six mothers, aged 34 to 52; three fathers, aged 35 to 51; five grandmothers, aged 61 to 70; and one grandfather, aged 80.

Fieldwork approach and data collection. As in Study 1, semi-structured group interviews were chosen because this method of data collection uses prepared questions that

allow for some degree of researcher flexibility and greater participant elaboration rather than a straight question and answer format (Hennink et al., 2011). The topics were structured to support exploration of the HBM and extended HBM constructs. The family interview discussion guide (see Appendix 6.B) began with questions that asked family members about their unique family health history tree diagram (see the sample pedigree in Appendix 4.E) and continued through each section of the Families SHARE workbook from front to back cover (see Appendix 4.A). Enlarged copied sections of the questionnaire ('*Questions about your family health history*'- two pages), and two pages of the workbook ('*How to read a family health history tree*'-) were laminated for use as visual prompts (see Figures 6.1 and 6.2 in Appendix 6.C), thus facilitating simultaneous information sharing between family members and promoting interactive discussion (Krueger & Casey, 2009). Each family interview took between 30 and 45 minutes, was audio recorded and then transcribed verbatim. Upon conclusion of the interview each individual participant received a supermarket voucher to the value of ten dollars to compensate for their time.

Procedure

Follow-up family interviews and discussion guide. The same procedure was used as in Study 1 family interviews. In Study 4, participant families from the experimental condition were invited to participate in family interviews after follow-up questionnaires were completed. Interested families then contacted the researchers directly to schedule a suitable time. Interviews were conducted after hours in the family home when it was most convenient for participants.

Materials

Demographic information. Participants were asked to indicate their name, their age and each person's position in the family in relation to the 'nodal' child (e.g., mother, grandfather).

Follow-up family interview discussion guide and facilitation. The family interview discussion guide content was designed to evaluate the effectiveness of the Families SHARE workbook following its implementation. The interview facilitation procedure was modelled on guidance for rigorous preparation procedures as described by Krueger and Casey (2009) and Krueger (1998a). Interviews were conducted by the same moderator (a post-graduate psychology student who is also a registered nurse) and note-taker (a clinical psychologist) (Krueger, 1998b). For the complete list of semi-structured interview questions, including prompts, see Appendix 6.B.

Family interview discussion guide. At the start, several Families SHARE workbooks (refer to Appendix 4.A) were tabled, and a copy of each family's unique family tree diagram (see a sample pedigree in Appendix 4.E) was provided to refer to throughout the discussion. Laminated pages of the family health history measure and disease risk algorithm were used as prompts during the interview process (see Appendix 6.C). The topics of the discussion were: Familial disease risk and the family pedigree diagram; Collectors of family health history information at baseline; Sharers of disease risk information throughout the family network; Any barriers to sharing disease risk information; and health behaviour change in response to: i) healthy lifestyle recommendations, ii) family illness, iii) the Families SHARE workbook, iv) the questionnaire itself; Perceived importance and effectiveness of health recommendations; and Perceived effectiveness of family health history as a motivator of health behaviour change (for the complete discussion guide see Appendix 6.B).

Qualitative Data analysis

As occurred in Study 1, transcripts were progressively imported into QRS NVivo^{TM} computer software for coding and thematic analysis while data were being collected (for further analysis rationale and details refer to Chapter 2 Method section).

Results

Survey Results

Table 1 presents the descriptive statistics for the adult individuals who completed the survey (N = 113). Sixty-nine percent of respondents indicated that there were able to utilise the workbook to determine their own disease risk, whereas 31% reported they were not able to, suggesting that a significant number of people either experienced difficulty or did not use the risk assessment tool in the workbook.

Table 1.
Families SHARE Workbook Evaluation; Frequency Percentages

Item	Question	Response %		
		Yes	No	Don't know
1.	Using the Family Health Workbook, were you able to assess your own degree of risk for each disease?	69	31	-
	1a. Are you at increased risk for:			
	Colorectal cancer?	12	55	33
	Breast cancer?	11	60	29
	Heart disease?	32	39	29
	Diabetes?	20	50	30
2	Have you shared information from the Families SHARE workbook with any of the following people:			
	GP?	10	90	-
	Other health care provider?	5	95	-
	Family member?	61	39	-
	Friends?	4	96	-
	Other?	0	100	-
3	After receiving your Family Health History Tree diagram did you update it in any way?	3	97	-
4	In the last 6 months, have you participated in any screening behaviour for any of the following diseases:			
	Colorectal cancer?	13	87	-
	Breast cancer?	17	83	-
	Heart disease?	17	83	-
	Diabetes?	20	80	-

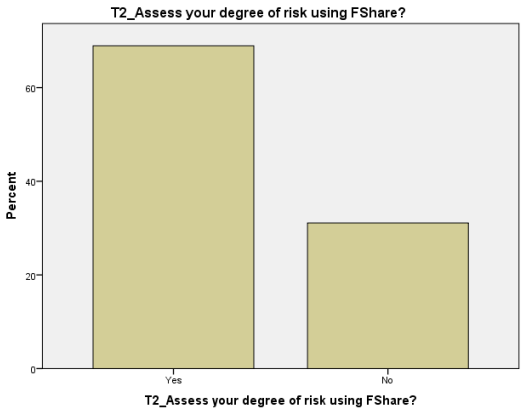


Figure 2. Percentage of participants in the intervention condition who assessed their level of chronic disease risk.

Risk assessment accuracy. To determine the accuracy of participant's self-reported risk assessments for the four chronic diseases, the Kappa measure of agreement analysis was calculated[56] (see Table 2).

Table 2
Kappa Measure of Agreement forActual Risk with Self Risk Assessmnet

Risk	Kappa	Agreement ES	Observed *n*	Self rated *n*	% Accurate	*p*
CRC average	0.17	Low	50.00	42.00	84.00	0.03
Above average			1.00	1.00	100.00	
Breast average	0.54	Moderate	45.00	43.00	96.00	< .001
Above average			3.00	1.00	33.00	
Heart average	0.25	Low	43.00	27.00	78.00	0.03
Above average			9.00	7.00	63.00	
Diabetes average	0.55	Moderate	44.00	36.00	82.00	< .001
Above average			7.00	7.00	100.00	

Note. Interventon condition only, diagnosed individuals were excluded.

Kappa agreement has been used previously to compare the accuracy of self-reported family history of cancer with a genealogy database indicating that respondents could recall family histories accurately (Kerber & Slattery, 1997). In the present study, the 'observed risk' of the

[56] The Kappa measure of agreement is a test typically used in the medical research literature to assess diagnosis agreement between two clinicians using the same measure; or to test the agreement in diagnosis of a single clinician using two different diagnostic measures (Peat, 2002, p. 228).

four diseases for individuals in the experimental condition was independently rated by the dissertation researcher. Ratings were determined according to each family health history pedigree diagram, and the relevant Australian familial risk for disease guidelines as outlined in the Families SHARE workbook. (NBOCC, 2010; NHMRC, 1999; NHMRC, 2005; NHMRC, 2009; NVDPA, 2012). Risk ratings were then compared with those self-reported by participants. Individuals who indicated that they were 'diagnosed' were excluded from the Kappa analyses. Thus, only individuals in the experimental condition who were of average or above-average disease risk were included. The low to moderate agreement in effect size suggests that many people experienced difficulty in calculating their own risk accurately.

Risk and disease screening behaviour. Individuals who identified themselves as above-average disease risk were encouraged within the Family SHARE workbook to participate in screening for the corresponding disease. Among the individuals that identified themselves at average risk for colorectal cancer (*n* = 38), 3 (8%) subsequently screened; of those who identified themselves at above-average risk (*n* = 6), 3 subsequently screened (50%) (See Table 3).

Table 3

Kappa Measure of Agreement for Self Risk Assessment with Subsequent Screening Behaviour (within Six Months)

			Participated in Screening			
Self Risk-assessment	Kappa Agreement	ES	Yes *n*	No *n*	% screened	*p*
CRC average	0.29	Low	3.00	35.00	8.57	**.04**
Above average			3.00	6.00	50.00	
Breast average	0.32	Low	6.00	36.00	16.66	**.02**
Above average			4.00	4.00	50.00	
Heart average	0.17	Low	5.00	24.00	20.83	ns
Above average			8.00	16.00	33.33	
Diabetes average	0.25	Low	5.00	30.00	16.66	.06
Above average			6.00	10.00	38.00	

Note. Interventon condition only. ns denotes non-significant

Similar results were reported for breast cancer; of the individuals that identified themselves at average risk for breast cancer (*n* = 42), 6 subsequently screened (14%) and of those who identified themselves at above-average risk (*n* = 6), 3 subsequently screened

(50%). Among individuals that identified themselves at average risk for heart disease (*n* = 29), 5 screened (17%); and of those who identified themselves at above-average risk (*n* = 24), 8 subsequently screened (33%). Lastly, individuals that identified themselves at average risk for type 2 diabetes (*n* = 35), 5 subsequently screened (14%) whereas among those who identified themselves at above-average risk (*n* = 16), 6 subsequently screened (38%). These results suggest that self-assessed risk for a specific disease impacted participation in screening behaviour in the manner predicted by the Health Belief Model because above-average risk participants screening behaviour outweighed those of average risk who screened. Correlation results indicated above-average risk participants in the experimental condition subsequently screened for colorectal cancer, breast cancer and diabetes with medium effect sizes shown (see Table 4).

Table 4.
Degree that Above-Average Risk Individuals Screened for Disease (*n*)

	Screening Behaviour			
	CRC	Breast	Heart	Diabetes
Above Average Risk				
CRC	.30* (48)	-	-	-
Breast	-	.33** (51)	-	-
Heart	-	-	ns	-
Diabetes	-	-	-	.27* (52)

Note. Spearman's Rho Correlations, 1-tailed

Family health history information dissemination. One of the main goals of the Family SHARE workbook was to stimulate family disease risk information sharing. Consequently, a key part of the evaluation required exploring the extent to which information was shared, and with whom in the family network. Of the 61 people who responded from a sample of 113, family health history information was described as being shared with husbands (10), fathers (2), wives (3), mothers (13), sons (6), daughters (9), children (gender not specified: 5), grandmothers (9), grandfathers (3), other "extended family" members (13), and four whole families (individual family member numbers were not specified) that were

related to the participants' family but did not participate in the study. Conversely, participants shared the intervention with only four friends (see Figure 3). These results therefore confirm that information was shared within families predominantly, however, the fact that 52 individuals did not respond to this question suggests that 46% did not share the information as anticipated.

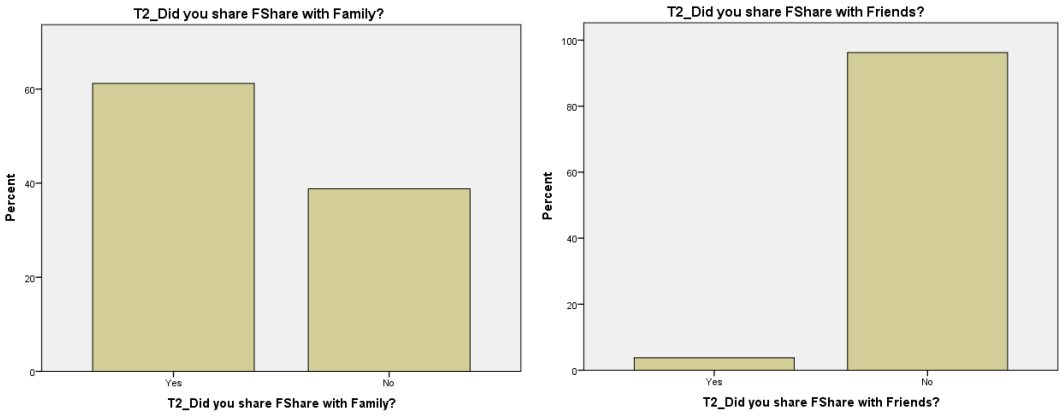


Figure 3. Percentage of participants in the intervention condition who shared the workbook with family (left) when compared with those who shared with friends (right).

Only 4 participant families reported updating their pedigree diagram after receiving it. A father who had died of colorectal cancer was added; an aunt and a cousin who both died of colorectal cancer were added; a mother with a recent diabetes diagnosis was added; and lastly, a grandmother and a cousin on the same side of the family, who both had diabetes, were also added.

Interview Results

Three main themes emerged from the data: 1) The perceived benefits of the Families SHARE workbook; 2) Health behaviours that link with theory and, 3) Barriers to the Families SHARE workbook's effectiveness. Each theme originated from a cluster of sub-topics (see Table 4).

Table 4.
Emergent Theme Topics from the Family Interview Data Comprising Several Sub-topics.

Item	Theme topic	Sub-topics
1.	Benefits of the Families SHARE workbook	· Sharing the workbook with family. · Ease of using the workbook. · Families evaluated the lifestyles of deceased relatives, learning about the links between diet and disease.
2.	Personalized health risk and links with theory.	· Tailoring health history to each family drew attention (*HBM cue to action*). · Parent's concerns for the future wellbeing of children (*HBM disease seriousness, severity*). · Families SHARE prompted contemplation about lifestyle behaviour change (*HBM benefits and barriers, TTM Stage of change*). · 'Contemplation' and 'action' stages of change were shown by intended or actual screening behaviours (*TTM Stage of change*).
3.	Limitations of the Families SHARE workbook's effectiveness.	· Difficulty obtaining family health history information. · Study ceiling effects. · The questionnaire itself prompted progression to the contemplation stage of change.

Note. Data was analysed using QRS NVivo™. Coded segments of text passages were grouped together to form nodes (e.g., topics such as 'Families SHARE benefits') and child nodes (e.g., sub-topics such as 'Ease of use').

Theme 1: The perceived benefits of the Families SHARE workbook. A number of respondents addressed the sub-topic 'sharing the workbook with family'. Comments suggested that active sharing occurred across three generations, usually via the mother in each family, as was the intended aim of the intervention. Sally, mother aged 52, suggested mothers were the contact point for family members about health issues: "I'd say they're normally the ones everyone comes to and says, "*I've got this wrong and that wrong" and, you know, "Can you sort out a doctor's appointment for me" and - I had that when I got home tonight, but we won't go into that.*"

Catherine (mother, aged 42) explained to the facilitator how the workbook was likely to be shared from parents to the child generation:

Mother: Yeah. If this [Families SHARE] is something that is for I reckon a certain age group. I don't, I wouldn't definitely say that teenagers and that would look at this. Parents might…
Facilitator: Hmm.
Mother: …and share that with them…

In some instances, the workbook information prompted sharing with other related families who were also noted to be at increased familial disease risk. One father, John aged 51 commented, "*I was a bit worried about the kids, that sort of thing, and also my other brothers, because we don't have any contact with my father for 40 years, so* [I contacted my brothers interstate] *just to let them know* [about their increased risk potential]."

The following field note written by the facilitator after one family interview highlights the sub-topic 'ease of using the workbook'.

> "*The family comprising grandmother Karen (aged 65), mother Emma (aged 43), children Thomas (aged 15) and Mia (aged 14) were advised by the researchers* [beforehand] *not to prepare for the Families SHARE evaluation, because it was not a test… This family had little recollection of the Families SHARE workbook* [when the interview began]*; therefore, it was good to have included in the discussion guide "how to read a family tree diagram" or pedigree. Since the family was not prepared, it was good to see that with simple reminders that they grasped the concepts quite easily as the interview went along*" [extract from Family 705 facilitator field notes].

A number of families highlighted how their discussions about familial disease risk led to discussions about causes of death among deceased relatives (sub-topic 'Families evaluated the lifestyles of deceased relatives'). Importantly, the discussions revealed that families explored health histories to identify lifestyle risk behaviours exhibited by earlier generation family members. For example, family members Liz (grandmother, aged 61) and Rebecca (mother, aged 34) commented,

Grandmother: And we talked about Mary and her lifestyle, and Bill [great aunt and uncle].
Mother: Yeah, her lifestyle was probably fairly - that was pretty much why she passed away so early, actually died at 55.
Facilitator: Really?

Mother: Yeah, she pretty much had the heart condition and died from it.
Facilitator: Gee.
Mother: But she pretty much avoided diagnosis and she wasn't particularly healthy. She wasn't very careful with her diet, she smoked, she didn't really exercise; she was quite overweight.

Theme 2: Personalized health risk and links with theory. The sub-topic 'tailoring health history to each family' highlighted how personal information was perceived as salient, confirming the importance of the perceived 'susceptibility' construct from the Health Belief Model as a motivation for behaviour change (Janz & Becker, 1984). After receiving the workbook, Catherine (mother, aged 42) emphasised to Judith (grandmother, aged 69) how important it was to discover more about her own risk for colorectal cancer.

Mother: And I didn't know that my grandma had died of bowel cancer. I didn't know that…
Grandmother: I wonder why you didn't?
Facilitator: Is this recently by doing this [study], or…?
Grandmother: No, no, no.
Mother: I did not.
Grandmother: I knew.
Mother: I found out.
Facilitator: When did you find out?
Mother: Yeah, I found out through this.
Facilitator: In the study?
Mother: Like talking about this.
Facilitator: Oh okay.
Mother: So, I didn't know that. No, I did not know that she had bowel cancer until we started talking…
Grandmother: Really?

The following father's comments indicate how the Families SHARE workbook could act as a '*cue to action*' to initiate behaviour change, just as disease diagnosis may do so. Cues to action are described as part of the extended Health Belief Model (Bylund et al., 2011). John (aged 51) illustrates this point as follows:

Father: I guess even if there's a small change, even if there's a small change, usually it's something's happened that shocks people into changing, like, they've had cancer or someone close to them has had something like that, and that often makes the change.
Facilitator: Like you said with cancer, that made you check.
Father: Yeah, so, I mean, this may highlight to somebody, look, you're really a risk because of, you know, once you fill in the boxes and if you were

all blue, or whatever the one indicates, I'd think, oh wow, I'd better do something.

The sub-topic 'parent's concerns for the future wellbeing of children' was shown by a focus among respondents on family health history increasing *susceptibility* to disease in offspring. Therefore, the parent generation reported most appreciating the workbook. The mother (Rebecca, aged 34) in the following family revealed:

Mother: …[the] impact that would have on Melissa [the child].
Facilitator: Yep.
Mother: Yeah, and I think we were kind of looking at some of the things that would impact on Melissa, like sort of just going through like, and we were talking about diabetes a bit as well.

The information made available in the wider workbook addressed how risk could be managed proactively. This was reflected in comments suggesting that the Families SHARE workbook prompted 'contemplation' (i.e., TTM Stage of change) about lifestyle behaviour change. Frequently, workbook advice appeared to move people from 'pre-contemplation' of the lifestyle behaviours that increase risks for chronic disease to 'contemplation', in a manner consistent with the Trans-Theoretical Model (TTM) Stage of change theory (e.g., Armitage et al., 2004). Laura (mother, aged 38) said, "*I guess for myself I would like to increase my physical activity. Facilitator: Mm hmm. Laura, Yeah also, yeah keep doing that for a long-term rather than just for a week, or two weeks (laughs)*."

Similarly, contemplation of lifestyle risk behaviours included alcohol consumption. Rebecca (mother, aged 34) said:

Facilitator: Okay, so did the workbook make you think about changing anything?
Mother: Oh yeah, a little quick thought.
Facilitator: Which things?
Mother: Oh, my biggest one's this one [pointing to the healthy recommendations for alcohol consumption on page 14 of the workbook].
Facilitator: Okay.
Mother: I didn't think about, I just sort of went, mm, I should.
Facilitator: You actually acknowledged it?
Mother: Yes, exactly.
Facilitator: So, that was "Limiting or avoiding…" [reading aloud the healthy recommendations for alcohol consumption on page 14 of the

workbook].

Mother: Yeah, but I'm, I enjoy going out and having a drink with my friends too much to sort of go, going to cut it out completely. I can't even do "*October Sober*" or whatever it's called, I just, or "*Dry July*" and, you know, there's a month - every month's got something.

The workbook section titled 'Current Screening Recommendations' was successful in raising awareness of the actions that one could take to screen for the four chronic diseases. In an example of motivating movement from the 'pre-contemplation' to the 'action' stage of change, Rebecca (mother, aged 34) disclosed:

Mother: Yeah, which I did used to do [screening], but I just don't think I've done it for a long time, yeah.
Facilitator: So, did this make you think about it or…
Mother: That did.
Facilitator: ...it just made you think about it now?
Mother: No, that - seeing it in the workbook did make me think. I remember being very aware of… and that I haven't done that.
Facilitator: Oh good, so it's triggered a memory.
Mother: And I have, since I saw that, actually checked myself, but yep, that was probably the one that I noticed the most.

The workbook's screening recommendations prompted some individuals to contemplate screening behaviour as this mother's (Emma, aged 43) emphatic response shows:

Facilitator: Okay. And did that, workbook at all, make you think as well?
Mother: Yeah. Yeah.
Facilitator: It did?
Mother: Definitely, especially knowing how to deal with breast cancer...
Facilitator: Yeah.
Mother: …and aware of that.
Facilitator: You could see what you could do [screening]?
Mother: Yeah.

Theme 3: Limitations of the Families SHARE workbook's effectiveness. One limitation some families experienced was incorporated into the sub-topic 'difficulty obtaining family health history information'. This difficulty arose because the older generations were deceased or family health history was generally not discussed. For example, Catherine (mother. aged 42) stated, "*Well this is what… yeah… well yeah, because dad was already*

dead. We didn't really talk with dad about what people died of, but he did always know didn't he, because of his research (laughs)."

'Study ceiling effects' was a sub-topic also noted in Study 2 and 3 as a potential limitation, for example, the sample's fruit and vegetable consumption was healthier than the Australian population. Some families confirmed a commitment to healthy lifestyle and screening behaviours that were evident prior to baseline questionnaires. Karen (grandmother, aged 65) and Emma (mother, aged 43) commented:

Grandmother: She does all [of the healthy recommendations] I think…
Mother: I did, like we walk, we do exercise, that's all I eat is fruit and veggies.
Facilitator: Yep.
Mother: Alcohol I don't have it.
Grandmother: Mammograms.
Mother: Yep. I've had all that done. I try to do it once a year that one.

Also, Sally (mother, aged 52) and John (father, aged 51) mentioned their family's healthy lifestyle behaviours:

Mother: We eat well, we don't drink alcohol, we don't smoke, we're reasonably active.
Father: We do at least 30 minutes a day.
Mother: Yeah, definitely.

Participant discussions suggested the final sub-topic that 'the questionnaire itself prompted behaviour change'. In the current study, Sally (mother, aged 52) moved from the 'pre-contemplation' stage in physical activity to the 'contemplation' stage due to her realisations during baseline questionnaire completion.

Mother: Yeah, so John got the Fitbit because he's really a gadget man. So I hadn't heard of the Fitbit, but from the questionnaire I was a bit disturbed about how much sitting around people do, including myself, and I don't think I actually answered it honestly because it was crazy, when you try and think about, like at work, you sit all day, sit in a car, sit here, sit there, so it actually prompted me to realise that we've got to do a lot more walking around, which is what the Fitbit does, 'cause it makes you do however many steps, 10,000 steps a day or whatever else, so before I had the Fitbit I was a bit more conscious about, you know, getting up and walking around the office and stuff like that, and

doing all those things, and you know.

Facilitator: Yep, because I heard you took John's off him and he had to get another one.

Mother: Yeah, well - I mean, and it did obviously prompt that because I think, I mean obviously, that's out there in the media now about people not walking around enough and about the incidental exercise and things like that, but when you actually have to answer that question yourself, you think...

Facilitator: It made you think.

Mother: ...wow.

Discussion

As detailed in the literature review and in the introduction to Study 3 Part A, the aim of providing families with the Families SHARE workbook was to influence health beliefs and stimulate behaviour change designed to address chronic disease risk. This was achieved by highlighting 'susceptibility' to each individual's chronic disease using tailored family health history information, by clearly depicting the 'severity' of four chronic diseases, and by providing healthy lifestyle and screening recommendations to moderate one's risk. The aim was to then prompt individuals to weigh up the 'benefits' of health promoting behaviour against any perceived 'barriers' (Janz & Becker, 1984). Moreover, consistent with the extended Health Belief Model incorporating 'cues to action' (e.g., Bylund et al., 2011), drawing participants attention to their own family health history and chronic disease risk was intended to act as a salient stimulus to motivate participants progression through the health behaviour stages of change (Prochaska et al., 1992).

The cluster RCT detailed in Study 3 Parts A and B yielded few significant results, and several limitations have been detailed in chapters four and five. Significant generational differences were evident in food consumption variables, with the exception of vegetables, within the experimental group for above-average risk participants (for a summary of results see chapter 5, Table 8). The present mixed methods study shows above-average risk participants in the experimental group were also motivated to engage in screening behaviour more so than average risk participants. The extent that the workbook successfully engaged

with families, the accuracy of participant's disease risk assessments, capacity to change lifestyle and screening behaviours, all impacted the Families SHARE workbook's efficacy.

Tailoring Family Health History Information

Data obtained from the survey confirmed that many shared their family disease risk information with other family members, as intended. Comments suggested that the relevance of tailored health history information was a key factor in engaging with families. Comments exchanged between family members highlighted the perceived importance of disease risk and personal significance. Some families shared their workbooks beyond those involved in the present study to extended family members and other related whole families. These results suggest the possible broader impact of Families SHARE. Ten percent shared the workbook with their general practitioner, with implications for further screening or health education contributing to ongoing disease prevention.

Families discussed their great-grandparents' disease history, evaluated previous deceased generations' lifestyle behaviours that contributed positively to longevity, or negatively to early mortality. In considering this, in combination with the educational nature of the workbook itself, participants demonstrated learning about the links between lifestyle behaviours and chronic disease, from salient examples in their own family history. Moreover, individuals clearly identified with the unique meaning that their own family health history had for each family member in the present time, who then considered the implications for their own future health outcomes or for those of their children.

Perceived Susceptibility, Disease Risk, and Screening

Secondly, Kappa's measure of agreement[57] in the present study indicated participants who calculated their risk for the four chronic diseases resulted in a concordance rate of

[57] Refer to Table 2.

between 78 and 96 percent when compared to the risk calculations of the researcher. Previous research with Families SHARE indicated that 100 percent of participants were able to use the algorithm to calculate their own risk (Koehly et al., 2015). Qualitative comments in Study 4 indicated participants easily understood the workbook instructions, therefore highlighting both the reliability and face validity of the workbook. However quantitative data suggests almost one third of adults did not assess their risk at all. This may have occurred because one person in the family, possibly the mother, calculated risks for all family members in the study, including children.

The present study showed that, among individuals in the experimental group approximately 20 percent screened for each of the four diseases[58]. The survey also revealed that participants who were of above-average risk for colorectal cancer, breast cancer, and type 2 diabetes, were more likely to screen than average-risk participants. This finding was consistent with comments suggesting movement from 'pre-contemplation' to 'contemplation', to the 'action' stage of change in relation to screening behaviour. These results suggest that the Families SHARE workbook was effective in triggering a 'cue to action' (e.g., Bylund et al., 2011).

Consistent with previous research, results suggest that identifying "at risk" individuals can be a motivating factor for some people to engage in preventative health behaviours even when asymptomatic (Chang et al., 2011). Heart disease results showed 33 percent of above-average risk, and 21 percent of average risk participants screened. As stated in chapter 5, heart disease may have been considered the most severe of the four diseases motivating the highest uptake of screening in the average risk group. Nevertheless, it is important to acknowledge that the intervention also motivated some average risk individuals to screen for

[58] Refer to Table 3.

all four of the chronic diseases. Within the qualitative data, some families indicated that they had contemplated, or changed, lifestyle behaviours including increased physical activity, made healthier dietary choices, and decreased alcohol consumption.

Limitations

In evaluating the effectiveness of the cluster RCT in Study 3, the previous literature cites one limitation of tailoring family health history; that it is time consuming and difficult to implement within a large population (e.g., Ruffin et al., 2011). However, it has been reported that tailored health messages are more effective in motivating health behaviour change than generalized health messages (Claassen et al., 2010). Ruffin et al., (2011) suggested that using automated family health history tools could reduce the time in collecting and interpreting data (e.g., O'Neill et al., 2009). Yoon et al. (2002) suggest the use of family health history and pedigree analysis is rare in preventative medicine at the primary healthcare level, leaving many people at above-average risk undetected. The present study encountered its own limitations, specifically, difficulties in obtaining family health history information from older relatives. Previous research has indicated that family health history information obtained from older people is less accurate than younger people, although accuracy rates were similar to results in the present study (e.g., Bensen et al., 1999).

The interview component of Study 4 corroborated findings from Study 3 that the sample's lifestyle behaviours were healthier when compared to the population statistics (ABS, 2015), therefore increasing the difficulty of achieving significant behaviour change in response to the intervention. Three ceiling effects were noted; a number of families in the experimental condition were engaged in many of the recommended healthy lifestyle behaviours at baseline; some participants already knew about their familial disease risks (or were diagnosed) and were undertaking regular screening at baseline; and a great proportion of participants were of average disease risk, limiting the potential for motivating health

behaviour change. The family interviews in the present study have provided valuable insight into the effectiveness of the Families SHARE workbook when the RCT faced these limitations. Future research could consider targeting demographic regions with higher specific disease profiles; or recruit more families to increase the chances of obtaining greater numbers of above-average risk individuals in the study.

As shown in previous research (e.g., Prichard et al., 2015), the effect of the questionnaire itself was shown to progress some individuals from the pre-contemplation to the contemplation and even to the action stage of change in lifestyle behaviours that prevent chronic disease. This issue was raised as a possible limitation in Study 3, and Study 4 substantiates the need for future research to address ways to minimise this potential occurrence. There is some research (e.g., Morwitz & Fitzsimons, 2004) that suggests the nature of questions can influence intentions to change behaviour by accessing cognitions about options of the specific topic category (i.e., "mere measurement effect") and future research could consider whether careful wording might moderate this difficulty (see Williams, Fitzsimons, & Block, 2004).

Conclusions

Notwithstanding the limitations, the present study suggests that the Families SHARE workbook is an effective family health history intervention tool that could be used to engage families and promote screening behaviours. Some success was shown in movement of lifestyle behaviours from pre-contemplation, to contemplation, and to the action stages of change (Prochaska et al., 1992). The dissemination of information beyond the nuclear family showed promise for intervening at the intergenerational family level in motivating health behaviour change, rather than aiming strategies at individuals or nuclear families. In utilizing collective free will to change behaviours (Bandura, 2001), and engaging family support to improve family lifestyle and screening behaviours that prevent chronic disease (e.g., Rolland,

2005), strategies such as the one trialled here could be utilised in future intervention initiatives.

CHAPTER 7: GENERAL DISCUSSION

Background – Setting the scene

Obesity and chronic disease rates continue to rise worldwide (AIHW, 2012; Stewart & Wild, 2014; Swinburn et al., 2011), despite the fact that maintaining a healthy weight and consuming the recommended serves of fruit and vegetables daily are recognized as protective against chronic diseases including colorectal cancer, breast cancer, heart disease, and type 2 diabetes (Amine et al., 2002; Aune et al., 2016). Examination of dietary intake in Australia highlights the fact that a large proportion of the Australian population's fruit and vegetable consumption has remained below the recommended serves per day for decades (ABS, 2006, 2014a). The percentage of Australians who meet the recommendations for vegetable consumption is extremely low at less than 7% (ABS, 2015; NHMRC, 2013a). Effective interventions are required to mitigate the problem of low fruit and vegetable consumption to assist in reducing the incidence of chronic diseases in future generations.

The theories underpinning the present series of studies were the Health Belief Model (HBM) (Rosenstock, 1974) and the Transtheoretical (Stage of Change) Model (TTM) (Prochaska et al., 1992) (see Figure 1).

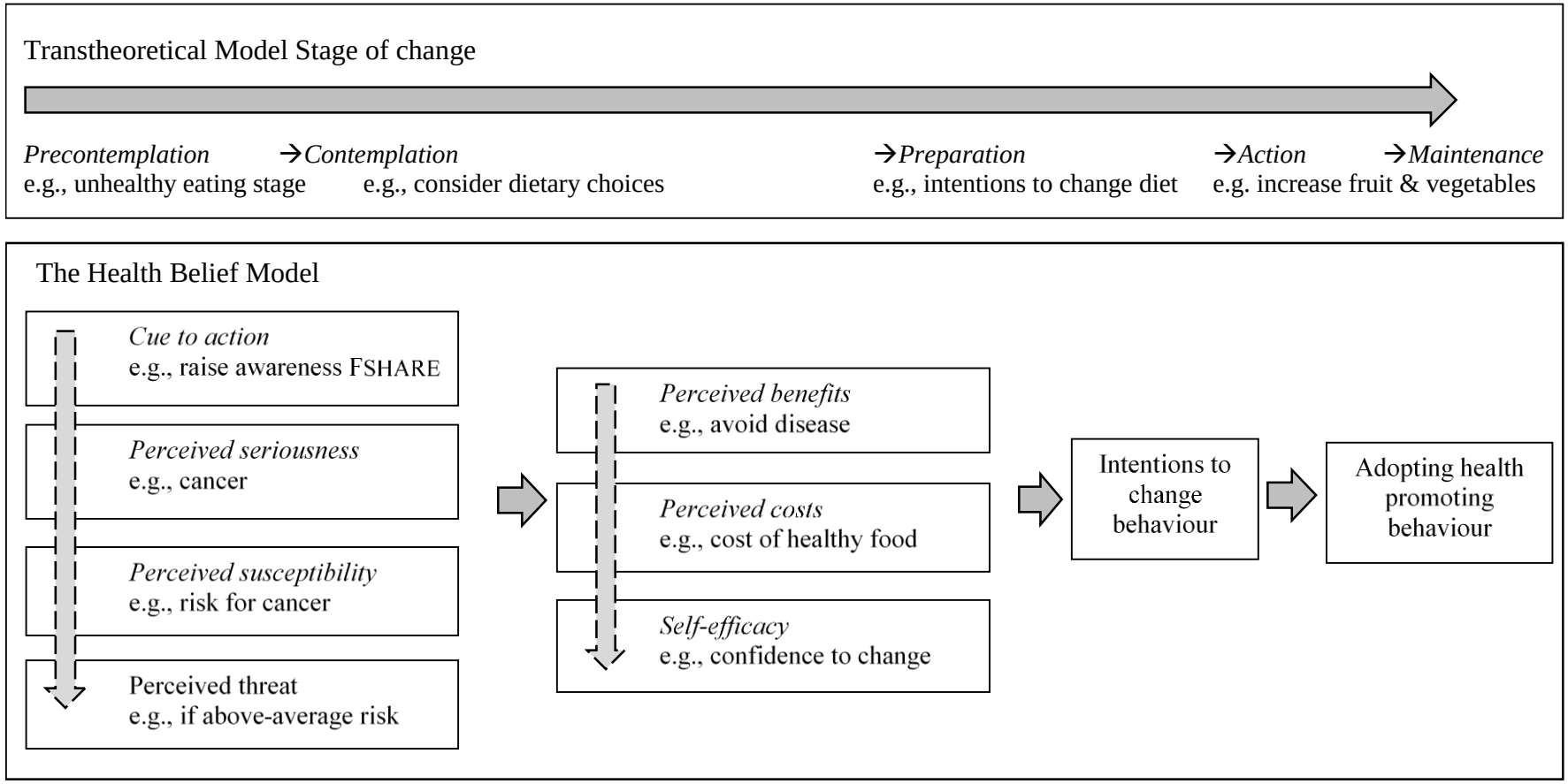


Figure 1. The Transtheoretical Model Stage of Change is shown as a simultaneous progression alongside the Health Belief Model.

As detailed in the introductory chapter, the HBM is a framework that can be applied to understand health motivation using disease risk as an impetus to increase healthy lifestyle behaviours. For interventions to be effective, one needs to believe that disease *susceptibility* (i.e., one's risk) is *serious* enough (i.e., the disease is severe e.g., cancer) to elicit fear arousal, then in weighing up the *costs and benefits*, decide that health *behaviour change* will be worthwhile to prevent the onset of disease. The extended HBM incorporates the added concepts: '*cues to action*' which, in the present research, was a function of the Families SHARE workbook in its entirety; and '*self-efficacy*' which was measured as confidence in one's intentions to change future behaviour (Linke et al., 2013).

To date, the main focus of the literature in addressing poor fruit and vegetable consumption has been in changing the behaviour of individuals. This ignores the potential to alter the social environment to change behaviour (c.f. Bandura, 2001). Few studies have examined intergenerational similarities and differences in dietary behaviours or how relationships with families influence dietary choices, although there has been some focus on the parent-child relationship and food choice (e.g., Beydoun & Wang, 2009; Jingxiong et al., 2007; Shapiro, 2004). Using a tailored family health history approach demonstrating ones unique familial risk for chronic disease (e.g., Koehly et al., 2015) may prove an effective obesity and disease prevention strategy. Utilizing a family health history approach, chronic disease risk is expected to be ameliorated by motivating health behaviour change that instils healthy lifestyle choices within the family context and home food environment.

Furthermore, few if any, studies have examined the associations between parental Food Life behaviours and attitudes (Sharp et al., 2013) and the dietary intake of offspring. As a potential moderating variable on healthy and unhealthy food consumption within families, Food Life behaviours and attitudes were also examined in the current studies (Rozin et al., 1999). Two subscales from the Food-Life Questionnaire: Short Form (FLQ:SF; Sharp et al.,

2013), were chosen because they closely relate to the HBM framework, and they are likely to impact upon the main outcome variables: healthy and unhealthy food consumption. The two FLQ:SF subscales were, firstly, diet-health oriented behaviour (DHOB) which aligns with self-reported dietary behaviours that are health protective, for example, 'trimming fat from meat'. Secondly, diet-health/disease linked attitudes (DHLA) was chosen to measure attitudes that one's dietary choices can make a difference to subsequent health protection or disease causation.

By incorporating the Transtheoretical (Stage of Change) Model (TTM) only those participants who were at *pre-contemplation*, *contemplation*, or *preparation* stages of change at baseline were focused on as having capacity to make healthy behaviour change at follow up. Participants in the *action* or *maintenance* stage of change at baseline who were meeting NHMRC guidelines (NHMRC, 2013a) for fruit and vegetable consumption, therefore had no capacity to progress their stage of change any further. By using two health behaviour models, the HBM and TTM together in this way, predicted outcomes that healthy and unhealthy food consumption could be examined. Previous research has cited the more successful familial risk interventions as ones that utilize health behaviour theory to predict outcomes, with greatest results noted within dietary and smoking cessation health behaviour change (French et al., 2017).

This final chapter integrates the findings from the four studies comprising this dissertation, while relating findings to the Health Belief Model and the Transtheoretical (Stage of Change) Model. The implications for future family oriented health interventions are considered.

The thesis aims

This dissertation aimed to improve understanding of healthy and unhealthy food consumption. It attempted this by examining the influence of the family network on dietary choices. It also a tested whether, consistent with predictions based on the HBM, the provision of tailored information about familial risk for chronic disease could improve dietary and other health decisions that impact disease prevalence, among those in the earlier stages of readiness to change diet, consistent with the TTM. In the course of addressing these aims a number of questions were posed: who are the key influencers on decisions that relate to diet and health in the family; do different people within the family, representing different roles (e.g., mother, father) and generations, serve a different purpose in this respect; how do food attitudes in one family member relate to food consumption in another; and can the provision of a tool that identifies familial risk for chronic disease improve diet and disease screening intention and behaviour within families at above-average disease risk.

Review of findings

Study 1

The first study involved semi-structured family interviews that explored food purchasing, preparation and consumption, and examined bi-directional influences on food-related behaviours within the extended nuclear family (i.e., across three adjacent generations). Results were consistent with previous research indicating that mothers were the dominant decision-makers in family food choices (Beydoun & Wang, 2009; Green et al., 2009). Study 1 further suggested that grandmothers and mothers dominated family food choice decisions even in those households where fathers shouldered more responsibility for the preparation of family meals. The women in each generation influenced fruit and vegetable consumption by control of purchasing, insisting on consumption, monitoring and reminding, implementing food rules, using conditional treats, and restricting others food choices. Men,

more so than women, preferred "indulgence" (i.e., unhealthy) foods and were prepared to "relax" family food rules. Grandparents and children directly influenced each other's consumption independent of (and even in opposition to) the parent generation.

Comments from family members highlighted how mother's primary influence on the family diet extended beyond the nuclear family to include the encouragement of healthy eating, and the discouragement of unhealthy eating practices, among the oldest (grandparent) generation as well as among their partners and children. The mother appeared to fulfil the main gatekeeper role, disseminating information about food and health to both the older and younger generations within the full family network whereas other family members' influence was restricted to dyadic interactions across two generations. This was an important finding in determining which family member was best suited to direct health information to, so that it would be shared across three generations. Results suggested that targeting the mother was likely to maximize health information dissemination, which could cue the combined agency of family members (Bandura, 2001; Lyons, Mickelson, Sullivan, & Coyne, 1998) to engage in behaviour change. This approach is consistent with previous research highlighting the role of women and parents in sharing health information among family members (Ashida & Schafer, 2015; Koehly et al., 2009; Koehly et al., 2003), and more recently, the finding that mothers may be effective in the role of genomics health educators within the extended family network (Koehly et al., 2015).

Study 2

The second study used a correlational, cross-sectional design, utilizing baseline data collection, to examine associations between healthy and unhealthy food consumption among family members within three generation families, with a focus on bivariate associations between family dyads. Using two subscales of the Food-Life Questionnaire Short Form (Sharp et al., 2013), analyses tested whether food-related behaviours and attitudes,

specifically, diet-health oriented behaviour (DHOB; self-reported healthy dietary behaviours e.g., trimming fat from meat) and diet-health/disease linked attitudes (DHLA; beliefs that diet has an effect of subsequent health or disease) correlated with healthy and unhealthy food consumption, with a focus on whether food attitudes of parents correlated with the food consumption of children. Food Life behaviours and attitudes selected for the present research were aligned with the two main components of the Health Belief Model (Janz & Becker, 1984), which assumes, firstly, that individuals aim to avoid illness, and secondly, can avoid succumbing to illness by engaging in healthy behaviours. Beliefs in the link between diet and health or disease, and engaging in healthy dietary behaviours are critical to the model successfully predicting behavioural intention and behaviour.

Food-Life Questionnaire results supported hypotheses that as participants scores on both subscales increased, healthy food consumption measured through food frequency, also increased, and unhealthy food consumption decreased. These results confirmed that self-reported healthy dietary behaviours, specifically, higher fruit and vegetable consumption and lower snack and fast food consumption, correlated with broader measures of behaviour and attitude to food. The latter supported Health Belief Model theory whereby attitudes associating diet with subsequent health or disease can have a positive impact upon self-reported healthy and unhealthy food consumption. Moreover, results indicated that diet-health oriented behaviours and diet-health/disease linked attitudes of mothers and fathers impacted differently upon the food consumption of children. The mother's importance was highlighted by correlations between mother and daughter fruit consumption and mother and daughter/son correlations with vegetable consumption.

Gender differences in parental food attitudes: Mothers. The results reported in the baseline, cross-sectional survey were consistent with previous research that has shown similar mother-child resemblances in healthy and unhealthy food consumption (Feunekes et al.,

1998), yet the underlying psychological mechanisms mediating and moderating these dyadic influences are not yet fully known. Research by Prichard, Hodder, Hutchinson and Wilson (2012) examined whether closeness in the mother and young-adult daughter relationship was a moderating variable on food consumption, however, resemblances in food consumption were reported regardless of how close the women were. The current study tested whether food attitudes as measured by diet-health oriented behaviours and diet-health/disease linked attitudes moderated or mediated this same association using Generalized Linear Mixed Modeling. Results showed significant effects of food attitudes contributing to the variance in vegetable and snack consumption only. Mixed findings suggest that simple modeling of behaviour, separate from any deliberative decision making, may be causally implicated as the main influencing factor. Future research is needed to identify further drivers of the correlation between food intakes across generations. If simple modeling is implicated then gender differences in mother and father parental modeling needs closer examination to explore the influences of each on children's dietary behaviours, both healthy and unhealthy.

Baseline survey data suggested that one FLQ:SF subscale measure of mother's self-reported healthy food intake, was associated with daughter's food consumption in the opposite direction to that predicted. Specifically, higher scores by mothers on a self-report measure of their healthy dietary behaviours (DHOB) were associated with lower fruit consumption by daughters, and higher levels of fast food consumption. Consistent with this, the hypothesis that mother's belief that diet can impact health generally, and chronic disease incidence specifically (i.e., beliefs that diet can have an effect of subsequent health or disease; DHLA), would predict intake of healthy and unhealthy food in their offspring was not supported.

These findings require close consideration. Comparison with Sharp et al (2013) suggests that these results are at least partially consistent with prior research. Sharp et al.'s

results indicated that self-reported Diet-Health Orientation correlated significantly and in the hypothesized direction, although mostly only marginally (.12 to -.51) with consumption of fruit, vegetables, fast foods and snacks in the previous week. Only the latter two produced correlations of any size (i.e., -.51 and -.27 respectively). Moreover, the belief that diet was related to chronic disease shared a very small amount of variance with fast food consumption in the anticipated direction (-.12) and did not correlate with anything else.

These results are likely to reflect, at least in parts, problems with social desirability in responding. The self-reported DHOB measure asks respondents about eating healthily, a behaviour that most mothers would find difficult to indicate poor compliance with. Additionally, samples in studies like Sharp et al. and the current study are likely to be subject to ceiling effects in both attitudes to healthy diet and in reported behaviour given the nature of self-selection bias.

An explanation for inconsistency in correlations may rest with influences on an association not tested here. One possible explanation for a result that is opposite to that hypothesized is that daughters may have disagreed with, or rebelled against, their mother's attitude to "healthy eating". Alternatively, previous research has suggested that attitudes and consumption are driven by variables not examined in the present studies; for example 'weight concern' (e.g., Rozin et al., 2003). Future mother-daughter research may consider exploring this possibility.

Previous research has indicated gender differences in food attitudes as measured by FLQ:SF subscales; diet-health oriented behaviours and diet-health/disease linked attitudes, with more positive behaviour and attitudes among women than men. Similarly, gender differences have been shown in the food attitude subscales 'weight concern' and 'disordered eating'; women score higher than men on both. However, in the last remaining subscale, 'negative reaction to food', men have shown greater food pleasure than women (Rozin et al.,

2003). The latter gender difference in food associated pleasure is consistent with observations from Study 1 that fathers were more lenient with the family food rules than mothers. Food attitudes have not been examined previously in association with parental influences on children's diet. Moreover, food attitude research has not separately examined three generation effects on food consumption, therefore, comparisons cannot be made with these novel findings.

Although mothers model behaviours to children of both sexes, results here are consistent with an interpretation that "caring" for others in the family is a female role stereotype that is more likely to be adopted by daughters than sons (Perry & Pauletti, 2011). Hence, evidence supporting sex-role modeling reinforces the notion that dietary behaviours are transmitted predominantly through the person acting in the mother role. Nevertheless, gender differences observed in the present series of studies suggest fathers' food attitudes could have implications for the developing food attitudes and food preferences of any children who are in their regular care. Consequently, research into the father-child relationship and its influences on dietary behaviours is likely to reveal some important dynamics.

Gender differences in parental food attitudes: Fathers. Baseline, cross-sectional survey results in Study 2 revealed the importance of fathers' food attitudes and their influence on children's healthy food consumption. This is highlighted in the positive correlations between father's beliefs that diet can have an effect of subsequent health or disease (DHLA) and fruit consumption in sons, and vegetable consumption in daughters. Correlation effect sizes were moderate to strong. Given that in Study 1 participant comments suggested that fathers were more lenient with the family food rules, and men are more inclined to view food as pleasure than women (e.g., Rozin et al., 2003), this outcome was surprising. Father-child self-reported healthy dietary behaviour (DHOB) results were non-significant. Therefore, the

influence of mother's and father's food attitudes appear to differ in the nature of the associations with the food consumption of offspring. This finding provides additional support for gender-differences in parental modeling being implicated as a causal factor in children's food consumption.

The hypothesis confirming father's beliefs that diet affects future health or disease correlate with children's food consumption, suggests that gender differences within several co-existent psychological variables have contributed to this outcome. As outlined in Chapter 1, according to social learning theory modeling by parents and significant others influences development in children (Bandura, 1977b). Furthermore, sex-role modeling is a major mode of gender identity transmission in child development (Bussey & Bandura, 1984). In the family social environment, parents socialize girls and boys differently and, by modeling gendered behaviours, shape the development of gender differences that persist in to adulthood (Tenenbaum & May, 2014). One could hypothesize that parental sex-role modeling also impacts children's developing dietary behaviours. The mechanism of influence may differ between boys and girls, depending on parental modeling and behaviour shaping, with this proceeding according to mothers' and fathers' own differing food attitudes. Since studies have shown food attitude differences between men and women in previous research (e.g., Rozin et al., 2003), any differences that exist between mothers and fathers have implications for child development and warrants further investigation.

Gender differences in parenting styles. Chapter 1 introduced parenting styles that have been shown to impact upon children's overweight and obesity. Using a cross-sectional design and data from the Longitudinal Study of Australian Children (Australian Institute of Family Studies, 2016), Wake and colleagues (2007) conducted an Australian National Population Study with almost 5,000 pre-school children. It was the first to examine parenting styles of both parents in relation to the BMI of children separately, while also controlling for

the BMI of parents (a widely documented correlate of child BMI). Younger children were selected for analysis within this population survey because of the primacy of the family as the context for the development of early childhood behaviours, and because overweight or obesity at school entry generally persists throughout the primary school years. As discussed in the introductory chapter, Baumrind's four parenting styles (i.e., authoritative, authoritarian, permissive, and rejecting/neglecting also termed 'disengaged' or 'uninvolved' in some texts; Baumrind, 1966, 1991) are each associated with different child developmental outcomes. 'Authoritative' parenting style suggests the most positive social, cognitive, and behavioural outcomes for children, and is characterized by high parental 'warmth' and 'control'. In Wake's (2007) research, further evidence that parental sex-role differences affect children's dietary behaviours was supported by differences between maternal and paternal parenting styles and associated BMI. For example, the odds of a child being in a higher weight (BMI) category was not associated with mothers' parenting style. Conversely, when compared to authoritative parenting as the reference category, fathers with a permissive parenting style increased the odds of a child being in a heavier BMI category by 59%, and fathers with a disengaged parenting style increased the odds by 35%. This suggests that authoritative parenting in fathers influence a child's healthier weight status whereas mothers' parenting style does not. This is important because it suggests that the mother-child dominated literature on dietary behaviour should include a focus on father-child influences on dietary behaviours, and specifically the mechanisms by which influence occurs.

Research has examined co-existing psychological parenting variables that positively influence children's healthy food consumption. For example, research by Blissett (2011), suggests that authoritative parenting and feeding styles, and modeling consumption, encourages greater fruit and vegetable consumption in children (see Figure 2).

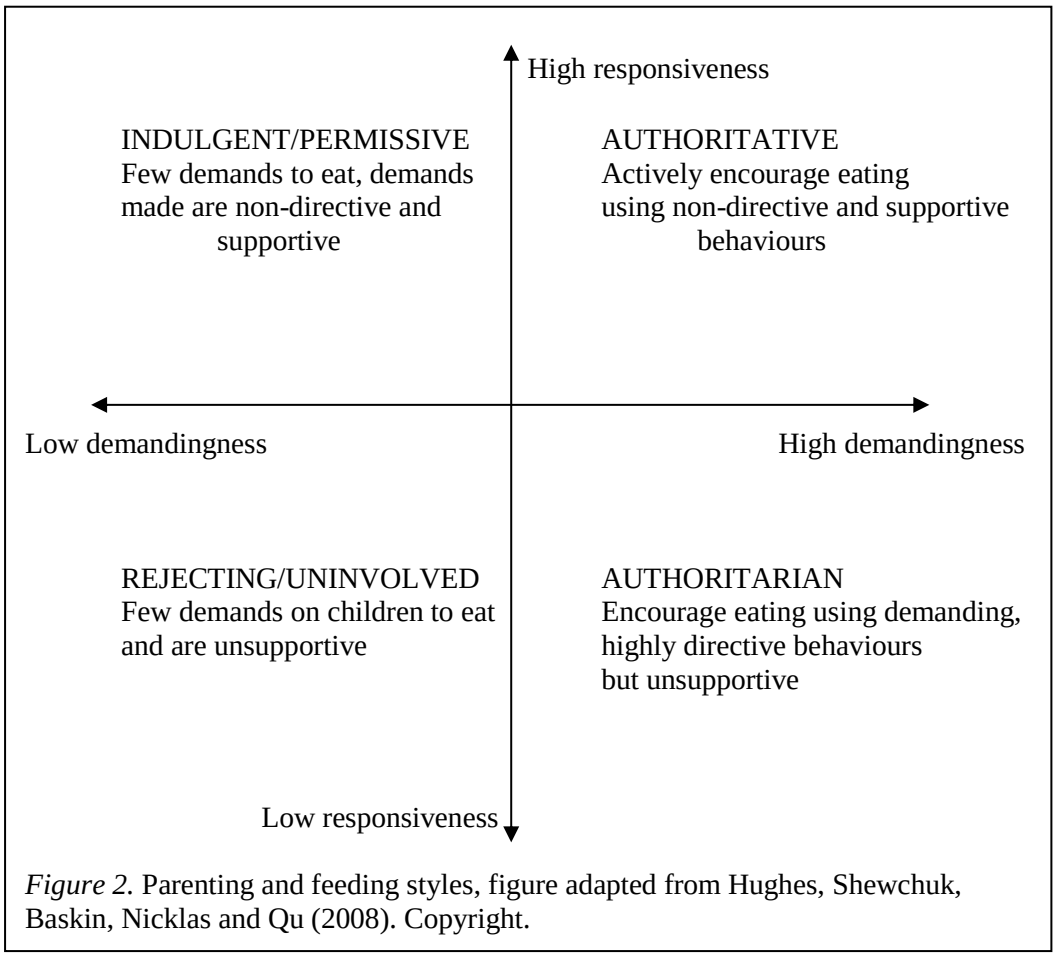


Figure 2. Parenting and feeding styles, figure adapted from Hughes, Shewchuk, Baskin, Nicklas and Qu (2008). Copyright.

Although findings are mixed and parents are usually measured together, overall an authoritative parenting style is most frequently associated with healthy childhood BMI, and fruit and vegetable consumption, across a wide variety of child age groups studied (e.g., Sokol, Qin, & Poti, 2017). In Vollmer and Mobley's (2013) review of the parenting and feeding style literature, predictors of fruit and vegetable consumption differed between mothers and fathers. For example, fathers' permissive parenting style predicted daughters' fruit and vegetable consumption, and mothers' authoritative parenting style and fathers' non-authoritative parenting styles were associated with greater fruit and vegetable consumption in children. In reviews of the literature, parenting and feeding styles dedicated solely to fathers was found to be limited (e.g., Khandpur, Blaine, Fisher, & Davison, 2014) and results inconsistent (Fraser et al., 2011). With mixed findings to date, father-child focused dietary

behaviour research investigating the underlying mechanisms of influence necessitates greater attention.

Alsharairi and Somerset (2015), used the same longitudinal Australian data (AIFS, 2016) as Wake et al. (2007) cited above, with 5,000 primary school children. Results highlight how maternal and paternal parenting style impacts the fruit and vegetable consumption of offspring in different age groups beyond the very young. Children aged four to five years with authoritative and permissive parenting fathers were more likely to consume fruit and vegetables when six and eight years of age. Girls aged four to five years with authoritative-parenting mothers were more likely to consume fruit and vegetables at six and eight years of age. Boys aged six to nine years with authoritative-parenting mothers were less likely to consume fruit and vegetables. These results reflect not only the differences in paternal and maternal parenting styles on the healthy food consumption of offspring, they also show different outcomes according to a parent's gender or to a child's gender and age. Considering the literature to date, it is possible to hypothesize that higher scores on fathers' beliefs linking diet with subsequent health and disease, and a co-occurring authoritative parenting style, will increase the likelihood of a child consuming fruit and vegetables and being of a healthy weight (see Figure 3). Further father-child research may support this hypothesis.

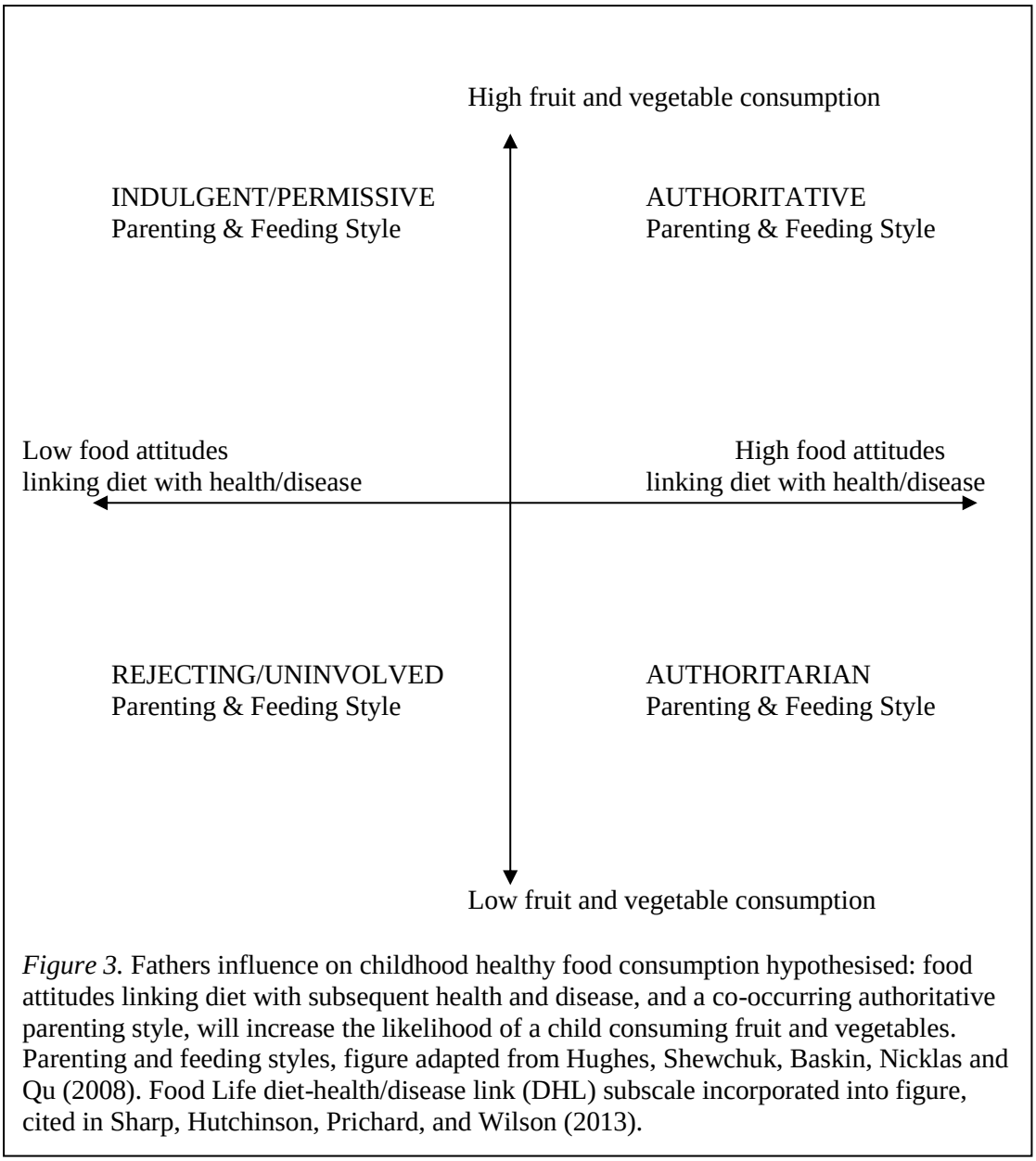


Figure 3. Fathers influence on childhood healthy food consumption hypothesised: food attitudes linking diet with subsequent health and disease, and a co-occurring authoritative parenting style, will increase the likelihood of a child consuming fruit and vegetables. Parenting and feeding styles, figure adapted from Hughes, Shewchuk, Baskin, Nicklas and Qu (2008). Food Life diet-health/disease link (DHL) subscale incorporated into figure, cited in Sharp, Hutchinson, Prichard, and Wilson (2013).

The distinction between mothers and fathers is critical because the underlying psychological mechanisms driving childhood fruit and vegetable consumption needs to be identified if family-focused dietary interventions for disease prevention are to be more targeted and effective. Further research is needed to explore how mothers and fathers impact children's healthy and unhealthy food consumption differently, whether by parenting style, feeding style, parental food attitudes, sex-role modeling or, if optimal psychological food-related strategies will together impact positively on children's healthier food consumption. In identifying mechanisms of healthy influence, the development of interventions increasing

fruit and vegetable consumption may be viable. Recommended levels of fruit and, particularly, vegetable consumption, are disturbingly low in Australia (ABS, 2015). When compared to other Western Nations (Micha et al., 2015), 'high income North America' also has one of the lowest vegetable consumption rates in the world. If the underlying positive psychological factors influencing children's fruit and vegetable consumption were identified parental dietary interventions could be better directed and utilized.

In addition to increasing fruit and vegetable consumption, it is also important to minimize the intake of unhealthy foods. A recent review of the literature examining fathers' parenting and feeding styles (Khandpur et al., 2014), revealed maternal and paternal differences consistent with Study 1. Specifically, results that showed fathers were less likely to monitor children's food consumption or restrict food access when compared to mothers. Fathers appear to be assuming more responsibility for parenting than previous generations and it is important to examine paternal influences on unhealthy food consumption in order to preserve the health of future generations. Identified strategies of paternal influence could be incorporated into parenting skills training for families with overweight children (e.g., Golley, Magarey, Baur, Steinbeck, & Daniels, 2007), to ultimately promote healthy eating habits and discourage unhealthy eating habits in children.

Future research should examine parental sex-role differences and healthy food consumption, but it is also necessary to examine how a relaxed feeding style and permissive parenting could negatively impact unhealthy dietary habits in children and BMI. Specifically, it is critical to identify the combination of independent variables (e.g., feeding, parenting, sex-role, and modeling) that positively impact diet and, conversely, which combination of the same variables will negatively impact, healthy and unhealthy food consumption. In addition, examining mother-child and father-child differences might reveal findings that identify mechanisms of dietary influence that are to be encouraged, and those mechanisms to be

discouraged, in dietary interventions that target parenting.

Generational differences in fast food consumption and their implications. Grandparents linked by marital-ties in Study 2 shared a lower prevalence of fast food consumption (frequency of meals consumed per week) compared to younger generations. The differences in fast food consumptions between generational cohorts is an important issue to consider given the apparently higher levels of consumption among the younger generations in the current study (children and parents) compared to the oldest generation (grandparents). This finding reveals the potential to impact obesity prevalence, and associated disease risk in the future (Rosenheck, 2008).

This result could be interpreted as suggesting that old age is "protective" against fast food consumption, notwithstanding the grandchild-grandparent correlation, but the result is more likely to reflect a cohort rather than age-based influence. It is noteworthy that Pizza Hut only opened in Australia in 1970 and the first McDonalds was opened in 1971 (O'Connell, 2015); most grandparents in the current sample would have been adults at this time and therefore were not exposed to fast foods during their childhoods. As described in the introductory chapter, food preferences acquired in childhood are likely to persist into adulthood (e.g., Contento et al., 2006; Savage et al., 2007). Results showed that the grandparent cohort in the current study, who were probably not exposed to fast food in childhood, consumed fast food infrequently and preferred 'plain food', as was suggested in Study 1. Conversely, given current intakes of younger generations, prevalence of fast food consumption among older Australians has the potential to increase over time. The implication is a greater disease burden in the younger cohorts as the parent and child generations grow old. Although reviews in the literature suggest fast food is not the cause of obesity, there is enough evidence to suggest that fast food consumption should be limited in overall diet (e.g., Rosenheck, 2008). Future research needs to consider generational differences in targeting

health messages appropriately for each generation, taking into consideration cohort differences in food preferences acquired at critical periods in lifespan development.

Study 3

Dietary behaviour changes over time and consistency with health belief theories.

The third study reported the results from a cluster randomized control trial that investigated whether tailored feedback about disease risk based on self-assessed family health history (i.e., the Families SHARE workbook revised for Australian use; (Koehly et al., 2015) motivated dietary behaviour change over time. The attitude and behaviour of intervention participants was compared to a control group, who participated in survey completion only[59]. It was further hypothesized that the impact would be moderated by disease risk so that diet-related health attitudes and intentions would be improved more in those at above-average risk for chronic disease versus those at average risk. As outlined in the introductory chapter, the Health Belief Model (HBM) (Janz & Becker, 1984) was the main theoretical framework underpinning this dissertation, and hypotheses in Study 3 aligned with theoretical predictions. The four health beliefs *perceived susceptibility* to illness, perceived *severity* of illness, and weighing up the perceived *benefits* and *barriers* of any preventative health actions, make up the original model constructs (Janz & Becker, 1984). The extended HBM includes *cue to action* and *self-efficacy* (e.g., Bylund et al., 2011). Study 3, using an experimental design, incorporated the extended HBM constructs as outlined in Figure 4. The Transtheoretical Model (TTM: Stage of change) (Prochaska et al., 1992) was incorporated to further refine the results in order to show behaviour change occurring in those participants who had the capacity to do so, that is to say, only participants who were *not* consuming the recommended serves of fruit and vegetables per day, in the experimental condition at baseline, were

[59] Each family in the control group were provided with the FHHA at the conclusion of Study 3.

included in subsequent analyses.

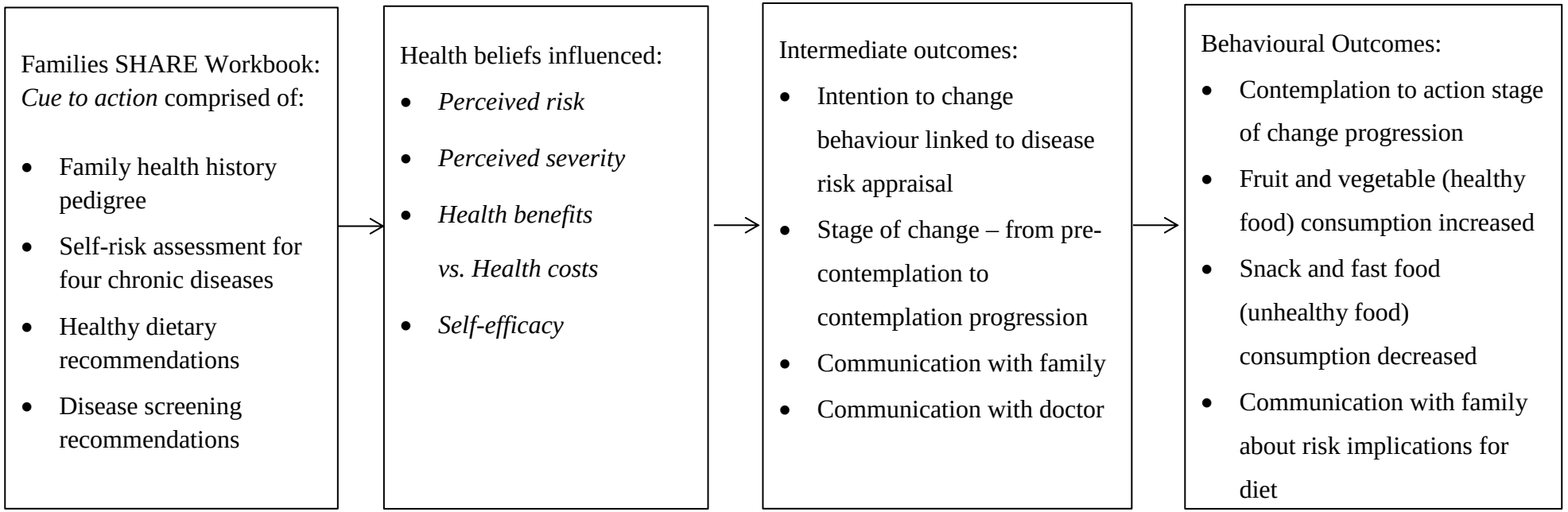


Figure 4. Conceptual model showing how tailored family health history, risk status and recommended risk reducing strategies would result in behavioural change. The extended Health Belief Model beliefs are italicized.

Results of the RCT were few. The main finding using Generalized Linear Mixed Models supported the hypothesis that experimental condition fruit consumption would increase more over time in response to provision of the Families SHARE workbook than the control group. Analysis of stage of readiness to consume fruit indicated significant improvements for the experimental group, who progressed from preparation to the action stage of change, whereas vegetable consumption did not alter. When examining for a greater improvement in the above-average risk participants in the experimental group, results were non-significant. Generational differences in the experimental group when a family member, or when the mother, was at above-average risk for at least one of the four diseases, revealed grandparents had greater fruit consumption than parents; parents and grandparents consumed fewer snacks than children; and grandparents consumed less fast food than children.

Risk information in the contemporary literature

The modest results from Study 3 could reflect some uncertainties reported in the recent literature on the efficacy of personalized genetic risk information and subsequent behaviour change, a hypothesis supported by health behaviour theory (e.g. Hollands et al., 2016). Godino, van Sluijs, Marteau, Sutton, and Griffin (2016) questioned the effectiveness of the provision of personalized genetic risk information for lifestyle change. The researchers conducted a RCT in the UK with healthy individuals who had a mean age of 45 years. Participants were provided with lifestyle advice alone, or in addition to personalized genetic risk assessments for type 2 diabetes (T2D). The outcome measures were physical activity, diet, weight, worry and anxiety. Although perceptions of risk were considered accurate, there were no significant effects of the intervention on any of the outcome measures, including risk sub-groups or behavioural intentions. It was noted that no psychological harm was borne by participants. It was, however, unclear to the reader what proportion of participants were at above-average-risk for type 2 diabetes, although there was enough power to conduct the

analyses. The authors questioned the quality of comparable studies, and cited their non-significant results as consistent with systematic review studies. It was acknowledged that targeting individuals in an environment with restricted opportunity to improve physical activity and diet was a limitation. Nevertheless, the cohort was of similar ethnicity and education level as some parent and grandparent participants in this dissertation, therefore is comparable.

Examining Godino et al. (2016) further, in theory one would expect personalized genetic risk information to be a sufficient 'cue to action' for behaviour change, given that T2D is modifiable by appropriate participation in health promoting lifestyles. However, medication is also available to treat TD2, and since physical activity was the primary outcome measure, participants in the 45-year-old age cohort were required to expend substantial effort for the study to achieve results. The researchers conceded that risk of T2D may not have caused sufficient concern for individuals compared to concern for diseases such as cancer, and they cited evidence suggesting risk information may influence decisions regarding the use of medication for heart disease (e.g., Usher-Smith, Silarova, Schuit, Moons, & Griffin, 2015). On balance, middle-aged persons opting to take medication to manage disease risk, rather than make diet and physical activity modifications, may reflect preference for the "easier choice" with perceived faster results. Future studies may consider evaluating whether participants decided to take up the option of medication for heart disease or diabetes during the time period between intervention and follow up survey (if these two diseases are included).

The strengths of the RCT in this dissertation was the use of a personalized risk-information tool (Families SHARE), that contained risk for *four* chronic diseases, including two cancers, to highlight disease severity: it required engagement by the users to calculate their own risk, and facilitated discussion across three generations of family members in order

for participants to learn about the links between diet and disease. This approach to motivating health behaviour change is theoretically sound, using all of the HBM constructs, even those of the extended HBM because the workbook in its entirety acted as a 'cue to action'. In addition, Study 3 incorporated a measure of 'confidence' as an indication of 'self-efficacy' to change.

The failure of the approach to achieve the desired change should be considered in light of other similar work. In a recent "systematic review of systematic reviews" conducted by French, Cameron, Benton, Deaton, and Harvie (2017), the researchers concluded that providing personalized risk-information alone, even when studies were of good quality, is not effective in motivating strong health behaviour change. Nonetheless, they did report that the most promising effects were found in studies that included dietary behaviour outcomes. Criticisms of the literature were that few studies incorporated theory (since those underpinned in theory have been shown to be more effective e.g.: Webb, Joseph, Yardley, & Michie, 2010), few targeted self-efficacy or response efficacy, and few studies focused on demonstrably efficacious Behaviour Change Techniques (i.e., modeled behaviour, fear arousal, provide information on consequences for individuals, prompting by use of imagery; Abraham & Michie, 2008).

There is potential advantage to the method of providing personalized risk information utilized in the current study. When personalized genetic risk information is provided by computer algorithm and given to individuals with minimal recipient input the opportunity to calculate one's own risk is missed. In the current study providing families with the Families SHARE workbook ensured that participants interacted with the material, engaged with it, and came to understand how a family pedigree works. This learning opportunity may be an important step in theory. HBM requires understanding of one's own risk *susceptibility* and belief in the *severity* of the risk. Once this minimum realization is achieved health decision-

making requires weighing up perceived *costs* and *benefits*. The strength of the Families SHARE workbook is that it targets families, not individuals; that discussion between family members occurred to acknowledge diseases were *severe* enough (as evidenced by the uptake of screening behaviours). Moreover, families learnt from the past health behaviours of deceased relatives, and on this basis, were exposed to information about dietary and screening participation that can assist in the management of disease risk. Consequently, not only did learning occur about the link between diet and disease, but family engagement in analysis and discussion of risk had the potential to improve salience, personal relevance and provide added meaning to the terms *susceptibility*. The strategy of engaging and sharing with family members adds an emotional element to the risk information which should not be compared alongside personalized genetic risk information provided by a computer algorithm.

Limitations of the RCT in Study 3. It is possible that the following limitations impacted upon the results. In addition to HBM theory, TTM Stage of change theory was also incorporated. In doing so, participant exclusions were completed based on those who were meeting the recommended serves of fruit and vegetables per day (depending on length of time, determined either at the 'action' or 'maintenance' stage of change). One cannot expect improvement in outcome measures to take place based on above-average risk alone if ceiling effects are also taken into account. By adding Stage of Change theory, the quality of the RCT was improved, however, the study lost power. This is because for dietary behaviour change to be observed over time, participants needed to be: in the experimental condition, of above-average disease risk, and in the precontemplation, contemplation, or preparation stage of change at baseline.

Prevalence of intention to change behaviour. Notwithstanding the limited findings within Study 3, 77% of participants indicated that they had *intentions* to improve upon 'fruit and vegetable consumption' (measured as a single item). Associated *self-efficacy*, measured

as confidence to increase fruit and vegetable consumption, had a mean rating greater than three out of seven (range 1, 'not at all confident' to 7, 'very confident'). Actual vegetable consumption may not have improved in response to the intervention, however, intentions and self-efficacy to improve upon current levels of fruit and vegetable consumption reflects a potential for future behaviour change.

Webb and Sheeran (2006) examined the assumption that intentions *cause* subsequent behaviour change in a meta-analysis of experimental health psychology studies. Results showed that a medium to large intention to change led to a medium to small change in subsequent behaviour. Since intentions are the pre-cursor to any behavioural change according to health behaviour theory, future research could consider incorporating a final long-term follow-up measure twelve months post-intervention to capture whether participants' intentions to improve healthy food consumption and confidence in doing so predicted variance in behaviour at a later time point. A meta-analysis of interventions utilizing risk perceptions to change intentions and behaviour (Sheeran, Harris, & Epton, 2014) showed similar results to those in the present research. As occurred in Study 3, intentions to change behaviour were greater than actual behaviour change, and influenced risk perceptions (e.g., increased susceptibility heightened risk perception).

The influence of Food Life attitudes and behaviour on dietary outcomes. Generalized Linear Mixed Modelling with -2 log pseudo likelihood tests and chi square tests of significance (Heck et al., 2014a) were used to determine whether the two food subscales of interest (self-reported healthy dietary behaviours, and beliefs that diet can have an effect on subsequent health or disease), measured by the Food-Life Questionnaire Short Form (Sharp et al., 2013), contributed any variance to healthy and unhealthy food consumption of the total sample (cf. Coxe, West, & Aiken, 2009). Behaviours contributing to a healthy diet (the diet-health oriented behaviour subscale) made a significant contribution to the shared variance in

vegetable consumption that was greater than the effect of gender in the same model[60]. A significant contribution to the variance in snack consumption was also evident. Similar recent research has noted an association of the diet-health orientation measure with vegetable consumption and with maintaining healthy nutrition even when food choices are restricted (Pilla, Loblay, Director, Soutter, & Swain, 2016).

The subscale of the Food Life Questionnaire that measures individual differences in beliefs that diet affects subsequent health or disease (DHLA) shared variance with snack and fast food consumption in Study 3. This suggested that understanding how poor food choices impact health may be more important predictors of food consumption, particularly "discretionary" energy dense foods.

Study 3 results are consistent with an interpretation of differential effectiveness for health promotion campaigns; messages describing energy dense-foods as "unhealthy" are currently widely promulgated, with advocacy groups including medical colleges, attempting to motivate government to tax "sugar" in sweetened beverages in the way it currently taxes tobacco and alcohol (Scott, 2016). Similar calls have been made for the imposition of a "fat" tax (Broadstock, 2015). Recent modelling of the impact of the impact of a tax on fat, salt, sugar and sugar-sweetened beverages and a subsidy on fruit and vegetables suggested it would avert approximately 470,000 Disability Adjusted Life Years (Cobiac, Tam, Veerman, & Blakely, 2017). By contrast, the "Eat Healthy" campaigns and "5 and 2 a day" have been around for a long time and may not have the same salience, particularly in a sample of already "healthy" eaters.

To date, few studies have since incorporated the Food-Life Questionnaire (Sharp et al., 2013) in research (i.e., Pilla et al., 2016; Wilson et al., 2016; Wolinsky, 2016) and not all

[60] Controlling for the effect of all other predictors in the model: generation, gender, and generation*gender.

studies used the complete set of subscales, making comparison with the present research difficult. In sum, if results of the present research were generalizable to the Australian population, it would suggest that more needs to be done to promote beliefs linking fruit and vegetable consumption with disease prevention. Future research exploring whether similar results occur with a culturally diverse Australian population may confirm the present findings with implications for the direction of future interventions.

The impact of the provision of the Families SHARE workbook on non-dietary health behaviour.

Study 4 evaluated the Families SHARE workbook, revealing the extent that screening behaviour was influenced amongst adults in the experimental group. The fact that access to the booklet primed intention to screen, as advocated in the material, suggests that non-habitual behaviours could also be primed by family health history information, with provision of the booklet acting as a 'cue to action'. Data on screening behaviour collected in Study 3 indicated that approximately 20 percent of individuals screened for each of the four diseases. The original Families SHARE workbook was designed with the inclusion of prominent mortality causing diseases in the US that also had familial and modifiable behavioural risk factors, with the intention of promoting health behaviour change that would impact disease prevention (Koehly et al., 2015). The same four diseases were incorporated into the revised Australian workbook. Families who identified themselves at above-average disease risk also took notice of the need to screen earlier than those of average disease risk. Increased disease risk indicates either earlier, and or, more frequent screening recommendations (NHMRC, 1999, 2005, 2009b; NVDPA, 2012). Heart disease results showed the greatest percentage of average risk participants that screened (21%) and it is important to acknowledge that the intervention also motivated a proportion of average risk individuals in the experimental group to screen for disease.

Strategies for improving the effectiveness of provision of the Families SHARE workbook require further examination. In the current version, both maternal and paternal sides of tailored family health history pedigrees are collected which enabled parents to assess their own individual risk, then the combined familial risks that applied to offspring. Koehly et al. (2015), suggest that focusing parents on children's risk may be a successful strategy to prompt action. Other research confirms the primacy of the mother again, even in the context of the general management of health (Case & Paxson, 2001).

Results of the present research suggested that active learning took place, as was one intention of the original Families SHARE workbook (Koehly et al., 2015). The importance of direct communication about the material covered and the extent to which this occurred cannot be judged from the results of the present study although Study 4 supports the suggestion that active discussion did follow intervention exposure in the experimental group, at least for some families. Future research utilizing digital family pedigree exercises completed or provided in isolation may find them less effective as an educational tool, and more difficult to share with family members for discussion, comment, or for making additional notes.

Limitations

In addition to the limitations discussed throughout this chapter, some final limitations are outlined in the following section. Firstly, the difficulty in measuring unhealthy food consumption as accurately as healthy food consumption is a common difficulty likely to impact the reliability of the former (Livingstone & Robson, 2007; Shim, Oh, & Kim, 2014). Healthy food consumption in the current series of studies was quantified by serves per food group (e.g., "*1 serve of vegetables is equivalent to 1 medium potato, or ½ a medium sweet potato, or ½ cup of dark green leafy vegetables (e.g., cabbage, spinach, broccoli, or brussel sprouts), or 1 cup of other vegetables (e.g., lettuce, beans, lentils, peas, zucchini, cucumber, mushrooms*)". In having self-reported serves per day, researchers can create surveys that

estimate healthy food consumption, that are also easily relatable for participants to recall. Furthermore, consumption may then be analysed as either meeting- or not meeting- the recommended serves per day (e.g., NHMRC, 2013a). Therefore, healthy food analysis and interpretation of results are not only valid and reliable, but comparable across studies by different researchers. By contrast, unhealthy foods have numerous forms, contain varying amounts of sugar, sodium, and fats, making measurement consistency across studies more difficult. The present research quantified unhealthy food according to an approach used in a previous Australian study (e.g., Sharp et al., 2013). The problem with assessing unhealthy versus healthy food consumption is exacerbated by the ambiguity of guidelines with regard to the former. The lack of a clear benchmark (c.f. "2 and 5 a day") also prevents comparison across studies and impedes goal directed dietary compliance among consumers.

The achievement of significant dietary change in the intervention study was also hampered by ceiling effects. Families recruited to the study indicated compliance with a "healthy" diet and consequently any room for improvement was limited. Additionally, risk for the targeted chronic diseases was also generally assessed as "average", thereby impacting the number of families potentially benefiting from the intervention. Future research should attempt to recruit families at "above-average" risk of the four chronic diseases, as well as those with "average" risk. The participant pool could then be randomly allocated to experimental and control conditions with a greater chance of people with above-average disease risk being exposed to the intervention. This participant population may be available through primary healthcare providers or in outpatient departments in major hospitals. Furthermore, recruitment for a similar future study could consider utilizing a city with a larger population, or by recruiting at the national Australian level (population 24.22 million in 2016), rather than limiting recruitment to the Statewide population (population 1.71

million in 2016; ABS, 2017)[61].

The generalizability of findings is likely to be limited because the sample was restricted to English speaking Australian participants. Australia is one of the most diverse multicultural nations in the world (ABS, 2012) and this alone justifies a broader, more inclusive approach. Study 1 has since been extended to include Chinese- and Italian-Australians (Rhodes et al., 2016) and future research will provide more diversity (i.e., Wilson et al., 2016). Consequently, future research may require that the Families SHARE workbook is translated into different languages, particularly for the grandparent generations in Australia.

A final limitation was that 30% of participants did not assess their individual risk using the Families SHARE workbook. A possible explanation for this is that one family member may have assessed other individuals risk within the family – conceivably the mother - because this was the person targeted to receive communication from the researchers. Up to 50% of above-average risk participants in the experimental group engaged in screening behaviour; 29% more than in average risk families. A further longitudinal follow up questionnaire may have revealed greater uptake of screening behaviours at a later time point when intentions to screen may have been acted upon.

Conclusion

Australian fruit and vegetable consumption rates continue to wane as obesity and chronic disease rates rise worldwide (ABS, 2006, 2015; Lim et al., 2012). Vegetable consumption in Australia has declined over recent decades to levels where less than 7% of the population meet the recommended five serves per day (ABS, 2015). Although parental psychological variables moderating healthy and unhealthy food consumption in children

[61] More detail on recommendations for recruitment of participants for intergenerational and cross-cultural research pertaining to this Project has since been published (see Hughes et al., 2015).

require further investigation, the current research suggests that food attitudes, parenting style, feeding style, and sex role-modeling may all play a part in unison.

Motivating modifiable lifestyle behaviours that have an impact upon chronic disease prevalence by using the Families SHARE workbook showed tentative success in improving fruit consumption, and initiating screening behaviour, not only in participants at above-average risk, but also in participants at average disease risk. Conversations within families showed the workbook cued behaviour change and was shared as an educational tool that informed food attitudes linking diet with health and subsequent disease potential. Greater understanding of this important link, along with the provision of tailored family health history feedback, is likely to have motivated these changes.

It is recommended that public health messages focus more on *increasing* healthy dietary behaviours by *increasing* the consumption of fruit and vegetables. Public perceptions may be currently more attuned to *reducing* overweight by *reducing* the consumption of unhealthy foods, or *reducing* portion sizes of all food types. Greater emphasis on consuming *more* healthy fruits and vegetables, including the reasons for doing so by highlighting the impact on disease prevention, is required. Being more informative about the links between diet and good health or disease may yield greater success in achieving healthier dietary outcomes and weight loss (or avoiding weight gain). Conceivably a "less is more" approach could be adopted: *less* weight gain and *less* disease incidence can be achieved via *more* fruit, and *more* vegetable consumption.

REFERENCES

Abood, D. A., Black, D. R., & Feral, D. (2003). Nutrition education worksite intervention for university staff: Application of the health belief model. *Journal of Nutrition Education and Behavior*, *35*(5), 260-267.

Abraham, C., & Michie, S. (2008). A taxonomy of behavior change techniques used in interventions. *Health Psychology*, *27*(3), 379.

ABS. (1988). *Child Care Arrangements Survey (1984)*. (Catalogue number: 4402.0.55.001). Canberra (ACT): Commonwealth of Australia.

ABS. (1997). *Child Care 1996*. (Catalogue number: 1301.0). Canberra (ACT): Commonwealth of Australia.

ABS. (2000). *Child Care 1999*. (Catalogue number: 1301.0). Canberra (ACT): Australian Bureau of Statistics.

ABS. (2002). *1944-45 Year Book Australia*. (Catalogue number: 1301.0). Canberra (ACT): Commonwealth of Australia.

ABS. (2003a). *Child Care 2002*. (Catalogue number: 1301.0). Canberra (ACT): Commonwealth of Australia.

ABS. (2003b). *Newsletter: Age Matters*. (Catalogue number: 4914.0.55.001). Canberra (ACT): Commonwealth of Australia.

ABS. (2005). *Childhood Education and Care Australia*. (Catalogue number: 4402.0). Canberra (ACT): Commonwealth of Australia.

ABS. (2006). *National Health Survey 2004 - 2005*. (Catalogue number: 4364.0). Canberra (ACT): Commonwealth of Australia.

ABS. (2012). *Cultural diversity in Australia, reflecting a nation: Stories from the 2011 census*. Canberra (ACT): Commonwealth of Australia Retrieved from http://www.abs.gov.au/ausstats/abs@.nsf/Latestproducts/BCDF2C64DD5B539CCA2571B90011998C?opendocument on the 15th August, 2013.

ABS. (2014a). *Australian health survey: Nutrition first results - Food and nutrients, 2011 - 12*. (Catalogue number: 4364.0.55.007). Canberra (ACT): Commonwealth of Australia.

ABS. (2014b). *Childhood Education and Care Australia*. (Catalogue number: 4402.0). Canberra (ACT): Commonwealth of Australia.

ABS. (2015). *Australian Health Survey: First Results, 2011-12* (Catalogue number: 4364.0.55.001). Canberra (ACT): Commonwealth of Australia Retrieved from http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/66544AD757B73867CA257AA30014B671?opendocument.

ABS. (2017). *Australian Demographic Statistics: September Quarter 2016* (Catalogue number: 3101.0). Canberra (ACT): Commonwealth of Australia Retrieved from http://www.ausstats.abs.gov.au/Ausstats/subscriber.nsf/0/140A826A4F3DF1A6CA2580EB00133610/$File/final%2031010_sep%202016.pdf.

AIHW. (2012). *Cancer incidence projections: Australia, 2011 to 2020*. Retrieved from Canberra:

Ajzen, I., & Fishbein, M. (1980). *Understanding attitudes and predicting social behavior*. Englewood Cliffs: Prentice-Hall.

Aldridge, V., Dovey, T. M., & Halford, J. C. (2009). The role of familiarity in dietary development. *Developmental Review, 29*(1), 32-44.

Alm, S., Olsen, S. O., & Honkanen, P. (2015). The role of family communication and parents' feeding practices in children's food preferences. *Appetite, 89*, 112-121. doi:http://dx.doi.org/10.1016/j.appet.2015.02.002

Alsharairi, N. A., & Somerset, S. M. (2015). Associations between Parenting Styles and Children's Fruit and Vegetable Intake. *Ecology of Food and Nutrition, 54*(1), 93-113. doi:10.1080/03670244.2014.953248

Amine, E., Baba, N., Belhadj, M., Deurenbery-Yap, M., Djazayery, A., Forrester, T., . . . MBuyamba, J. (2002). *Diet, nutrition and the prevention of chronic diseases: report of a Joint WHO/FAO Expert Consultation*: World Health Organization.

Anderson, P. M., & Butcher, K. F. (2006). Childhood obesity: trends and potential causes. *The Future of Children, 16*(1), 19-45.

Anderson, S. E., Must, A., Curtin, C., & Bandini, L. G. (2012). Meals in our household: Reliability and initial validation of a questionnaire to assess child mealtime behaviors and family mealtime environments. *Journal of the Academy of Nutrition and Dietetics, 112*(2), 276-284. doi:10.1016/j.jada.2011.08.035

Armitage, C. J., Sheeran, P., Conner, M., & Arden, M. A. (2004). Stages of change or changes of stage? Predicting transitions in transtheoretical model stages in relation to healthy food choice *Journal of Consulting and Clinical Psychology, 73*(3), 491 - 499.

Ashida, S., & Schafer, E. J. (2015). Family health information sharing among older adults: reaching more family members. *J Community Genet, 6*.

Ashida, S., Wilkinson, A. V., & Koehly, L. M. (2012). Social influence and motivation to change health behaviors among Mexican-origin adults: implications for diet and physical activity. *Am J Health Promot, 26*.

Atkins, D. C., & Gallop, R. J. (2007). Rethinking how family researchers model infrequent outcomes: A tutorial on count regression and zero-inflated models. *Journal of Family Psychology, 21*(4), 726-735. doi:10.1037/0893-3200.21.4.726

Aune, D., Giovannucci, E., Boffetta, P., Fadnes, L. T., Keum, N., Norat, T., . . . Tonstad, S. (2016). Fruit and vegetable intake and the risk of cardiovascular disease, total cancer

and all-cause mortality: A systematic review and dose-response meta-analysis of prospective studies. *International Journal of Epidemiology*. doi:10.1093/ije/dyw319

Australian Institute of Family Studies. (2016). *The Longitudinal Study of Australian Children Annual Statistical Report 2015*. Retrieved from Melbourne:

Baker, C. W., Whisman, M. A., & Brownell, K. D. (2000). Studying intergenerational transmission of eating attitudes and behaviors: Methodological and conceptual questions. *Health Psychology, 19*(4), 376-381. doi:10.1037//0278-6133.19.4.376

Ball, K., Jeffery, R., Abbott, G., McNaughton, S., & Crawford, D. (2010). Is healthy behavior contagious: associations of social norms with physical activity and healthy eating. *International Journal of Behavioral Nutrition and Physical Activity, 7*(1), 86.

Bandura, A. (1977a). Self-efficacy: toward a unifying theory of behavioral change. *Psychological Review, 84*(2), 191-215.

Bandura, A. (1977b). *Social Learning Theory* Englewood Cliffs, N.J. : Prentice Hall.

Bandura, A. (2001). Social cognitive theory: An agentic perspective. *Annual Review of Psychology, 52* 1-26

Bannon, K., & Schwartz, M. B. (2006). Impact of nutrition messages on children's food choice: Pilot study. *Appetite, 46*(2), 124-129. doi:http://dx.doi.org/10.1016/j.appet.2005.10.009

Banwell, C., Broom, D., Davies, A., & Dixon, J. (2012). *Weight of modernity: An intergenerational study of the rise in obesity*. Dordrecht, The Netherlands: Springer Science and Business Media

Barlow-Stewart, K., Emery, J., & Metcalfe, S. (2007). Genetics in family medicine: the Australian handbook for general practitioners. *Biotechnology Australia, Commonwealth of Australia*.

Barrett-Connor, E., & Khaw, K. (1984). Family history of heart attack as an independent predictor of death due to cardiovascular disease. *Circulation, 69*(6), 1065-1069. doi:10.1161/01.cir.69.6.1065

Baumrind, D. (1966). Effects of authoritative parental control on child behavior. *Child development*, 887-907.

Baumrind, D. (1973). The development of instrumental competence through socialization. *In Minnesota symposium on child psychology, 7*, 3 - 46.

Baumrind, D. (1991). Parenting styles and adolescent development. *The encyclopedia of adolescence, 2*, 746-758.

Beagan, B. L., & Chapman, G. E. (2004). Family influences on food choice: Context of surviving breast cancer. *Journal of Nutrition Education and Behavior, 36*(6), 320-326.

Becker, M. H., Maiman, L. A., Kirscht, J. P., Haefner, D. P., & Drachman, R. H. (1977). The Health Belief Model and prediction of dietary compliance: A field experiment. *Journal of Health and Social Behavior*, 348-366.

Beech, B., Rice, R., Myers, L., Johnson, C., & Nicklas, T. (1999). Knowledge, attitudes, and practices related to fruit and vegetable consumption of high school students. *J Adolesc Health, 24*, 244 - 250.

Begg, C., Cho, M., Eastwood, S., Horton, R., Moher, D., Olkin, I., . . . Simel, D. (1996). Improving the quality of reporting of randomized controlled trials: the CONSORT statement. *JAMA, 276*(8), 637-639.

Bell, A., & Swinburn, B. (2004). What are the key food groups to target for preventing obesity and improving nutrition in schools? *Eur J Clin Nutr, 58*, 258 - 263.

Bensen, J. T., Liese, A. D., Rushing, J. T., Province, M., Folsom, A. R., Rich, S. S., & Higgins, M. (1999). Accuracy of proband reported family history: The NHLBI Family Heart Study (FHS). *Genetic Epidemiology, 17*

(2). doi:10.1002/(SICI)1098-2272(1999)17:2

Berge, J. M., Arikian, A., Doherty, W. J., & Neumark-Sztainer, D. (2012). Healthful eating and physical activity in the home environment: Results from multifamily focus groups. *Journal of Nutrition Education and Behavior, 44*(2), 123-131. doi:10.1016/j.jneb.2011.06.011

Berk, L. E. (2007). *Development through the lifespan* (4th ed.). Boston: Allyn and Bacon.

Beshara, M., Hutchinson, A., & Wilson, C. (2010). Preparing meals under time stress. The experience of working mothers. *Appetite, 55*(3), 695-700. doi:http://dx.doi.org/10.1016/j.appet.2010.10.003

Beydoun, M. A., & Wang, Y. (2009). Parent–child dietary intake resemblance in the United States: Evidence from a large representative survey. *Social Science and Medicine, 68*(12), 2137-2144. doi:http://dx.doi.org/10.1016/j.socscimed.2009.03.029

Binkley, J. K. (1997). The relation between dietary change and rising U.S. obesity. *Journal of Obesity, 24*(8), 1032-1039.

Birch, L. L., Savage, J. S., & Ventura, A. (2007). Influences on the development of children's eating behaviours: From infancy to adolescence. *Canadian Journal of Dietetic Practice and Research, 68*(1), s1-s56.

Blake, C. E., Wethington, E., Farrell, T. J., Bisogni, C. A., & Devine, C. M. (2011). Behavioural contexts, food-choice coping strategies, and dietary quality of a multi-ethnic sample of employed parents. *Journal of the American Diet Association, 113*(3), 401-407. doi:10.1016/j.jada.2010.11.012

Blissett, J. (2011). Relationships between parenting style, feeding style and feeding practices and fruit and vegetable consumption in early childhood. *Appetite, 57*(3), 826-831. doi:http://doi.org/10.1016/j.appet.2011.05.318

Block, J. P., Scribner, R. A., & DeSalvo, K. B. (2004). Fast food, race/ethnicity, and income a geographic analysis *American Journal of Preventive Medicine, 27*(3), 211-217.

Bouhlal, S., McBride, C. M., Ward, D. S., & Persky, S. (2015). Drivers of overweight mothers' food choice behaviors depend on child gender. *Appetite, 84*, 154-160. doi:http://dx.doi.org/10.1016/j.appet.2014.09.024

Bradbury, K. E., Appleby, P. N., & Key, T. J. (2014). Fruit, vegetable, and fiber intake in relation to cancer risk: findings from the European Prospective Investigation into Cancer and Nutrition (EPIC). *American Journal of Clinical Nutrition*. doi:10.3945/ajcn.113.071357

Bradford Wilcox, W., & Kovner Kline, K. (2013). *Gender and parenthood: Biological and scientific perspectives* (W. Bradford Wilcox & K. Kovner Kline Eds.). New York: Columbia University Press.

Brannen, J. (2005). Mixing methods: The entry of qualitative and quantitative approaches into the research process. *International Journal of Social Research Methodology, 8*(3), 173-184. doi:10.1080/13645570500154642

Braun, V., & Clarke, V. (2008). Using thematic analysis in psychology. *Qualitative Research in Psychology, 3*(2), 77-101. doi:10.1191/1478088706qp063oa

Bray, G. A. (2006). Obesity: the disease. *Journal of Medical Chemistry, 49*(14), 4001-4007.

Broadstock, A. (2015). Health groups want fat, sugar tax introduced in Australia to lower obesity. *The Advertiser*.

Brody, E. M. (1981). "Women in the middle" and family help to older people. *The Gerontologist, 21*(5), 471-480.

Broom, A., & Willis, E. (2007). Competing paradigms and health research. *Researching health: qualitative, quantitative and mixed methods*, 16-30.

Brown, R., & Ogden, J. (2004). Children's eating attitudes and behaviour: a study of the modelling and control theories of parental influence. *Health Education Research, 19*(3), 261-271. doi:10.1093/her/cyg040

Burns, C., Bentley, R., Thornton, L., & Kavanagh, A. (2015). Associations between the purchase of healthy and fast foods and restrictions to food access: a cross-sectional study in Melbourne, Australia. *Public Health Nutrition, 18*(01), 143-150. doi:doi:10.1017/S1368980013002796

Bussey, K., & Bandura, A. (1984). Influence of gender constancy and social power on sex-linked modeling. *Journal of Personality and Social Psychology, 47*(6), 1292 - 1302.

Butterly, L. F., Goodrich, M., Onega, T., Greene, M. A., Srivastava, A., Burt, R., & Dietrich, A. (2010). Improving the quality of colorectal cancer screening: assessment of familial risk. *Dig Dis Sci, 55*.

Bylund, C. L., Galvin, K. M., Dunet, D. O., & Reyes, M. (2011). Using the Extended Health Belief Model to understand siblings' perceptions of risk for hereditary

hemochromatosis. *Patient Education and Counseling, 82*(1), 36-41. doi:http://dx.doi.org/10.1016/j.pec.2010.03.009

Calle, E. E., Rodriguez, C., Walker-Thurmond, K., & Thun, M. J. (2003). Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. *The New England Journal of Medicine, 348*(17), 1625-1638.

Campbell, M., Fitzpatrick, R., Haines, A., Kinmonth, A. L., Sandercock, P., & Spiegelhalter, D. (2000). Framework for design and evaluation of complex interventions to improve health. *British Medical Journal, 321*, 694-696.

Campbell, M., Reynolds, K., Havas, S., Curry, S., Bishop, D., Nicklas, T., . . . Heimendinger, J. (1999). Stages of change for increasing fruit and vegetable consumption among adults and young adults participating in the National 5-a-Day for Better Health community studies. *Health Educ Behav, 26*(4), 513 - 534.

Case, A., & Paxson, C. (2001). Mothers and others: Who invests in children's health? *Journal of Health Economics, 20*(3), 301-328. doi:http://dx.doi.org/10.1016/S0167-6296(00)00088-6

Chadwick, P. M., Crawford, C., & Ly, L. (2013). Human food choice and nutritional interventions. *Nutrition Bulletin, 38*(1), 36-42. doi:10.1111/nbu.12005

Chang, M.-H., Valdez, R., Ned, R. M., Liu, T., Yang, Q., Yesupriya, A., . . . Khoury, M. J. (2011). Influence of familial risk on diabetes risk-reducing behaviours among U.S. adults without diabetes. *Diabetes Care, 34*(11), 2393-2399. doi:10.2337/dc11-0876/-/DCI

Chavda, H., Haley, M., & Dunn, C. (2005). Adolescents' influence on family decision making. *Young Consumers, Quarter 2*, 68-79.

Christensen, P. (2004). The health-promoting family: a conceptual framework for future research. *Social Science & Medicine, 59*(2), 377-387. doi:http://dx.doi.org/10.1016/j.socscimed.2003.10.021

Christie, D. (2004). Resampling with Excel. *Teaching Statistics, 26*(1), 9-14. doi:10.1111/j.1467-9639.2004.00136.x

Claassen, L., Henneman, L., Janssens, A., Cecile, J. W., Wijdenes-Pijl, M., Qureshi, N., . . . Timmermans, D. R. M. (2010). Using family history information to promote healthy lifestyles and prevent diseases; a discussion of the evidence. *BMC Public Health, 10*(1), 248-248.

Cobiac, L. J., Tam, K., Veerman, L., & Blakely, T. (2017). Taxes and subsidies for improving diet and population health in Australia: A cost-effectiveness modelling study. *PLOS Medicine, 14*(2), e1002232. doi:10.1371/journal.pmed.1002232

Cohen, J. (2013). *Statistical Power Analysis for the Behavioral Sciences* Retrieved from http://flinders.eblib.com/patron/FullRecord.aspx?p=1192162

Cohen, J., & Cohen, P. (1983). *Applied multiple regression/correlation analysis for the behavioral sciences*. Hillsdale, NJ: Erlbaum.

Conner, M., & Norman, P. (Eds.). (1995). *Predicting health behaviour: Research and practice with social cognition models*. Buckingham, UK: Open University Press.

Contento, I. R., Williams, S. S., Michela, J. L., & Franklin, A. B. (2006). Understanding the food choice process of adolescents in the context of family and friends. *Journal of Adolescent Health, 38*(5), 575-582. doi:http://dx.doi.org/10.1016/j.jadohealth.2005.05.025

Cousins, J. H., Rubovits, D. S., Dunn, J. K., Reeves, R. S., Ramirez, A. G., & Foreyt, J. P. (1992). Family versus individually oriented intervention for weight loss in Mexican American women. *Public Health Reports, 107*(5), 549-555.

Coxe, S., West, S. G., & Aiken, L. S. (2009). The analysis of count data: A gentle introduction to poisson regression and its alternatives. *Journal of Personality Assessmment, 91*(2), 121-136. doi:10.1080/00223890802634175

Creswell, J. W. (2003). *Research design*. Thousand Oaks, California: Sage Publications.

Cullen, K. W., Baranowski, T., Rittenberry, L., Cosart, C., Hebert, D., & de Moor, C. (2001). Child-reported family and peer influences on fruit, juice and vegetable consumption: reliability and validity of measures. *Health Education and Research, 16*, 187 - 200.

Cullen, K. W., Baranowski, T., Rittenberry, L., & Olvera, N. (2000). Social-environmental influences on children's diets: results from focus groups with African-, Euro- and Mexican-American children and their parents. *Health Educ Res, 15*, 581 - 590.

Daly, J., Willis, K., Small, R., Green, J., Welch, N., Kealy, M., & Hughes, E. (2007). A hierarchy of evidence for assessing qualitative health research. *Journal of clinical epidemiology, 60*(1), 43-49.

De Bourdeaudhuij, I. (1997). Perceived family members' influence on introducing healthy food into the family. *Health Education Research, Theory & Practice, 12*(1), 77-90.

de Bruijn, G.-J., Visscher, I., & Mollen, S. (2015). Effects of previous fruit intake, descriptive majority norms, and message framing on fruit intake intentions and behaviors in Dutch adults Across a 1-week period. *Journal of Nutrition Education and Behavior, 47*(3), 234-241.e231. doi:http://dx.doi.org/10.1016/j.jneb.2015.02.001

de la Haye, K., de Heer, H. D., Wilkinson, A. V., & Koehly, L. M. (2014a). Predictors of parent-child relationships that support physical activity in Mexican-American families. *Journal of Behavioural Medicine, 37*(2), 234-244. doi:10.1007/s10865-012-9471-8

de la Haye, K., de Heer, H. D., Wilkinson, A. V., & Koehly, L. M. (2014b). Predictors of parent-child relationships that support physical activity in Mexican-American families. *Journal of Behavioural Medicine, 37*, 234 - 244. doi:DOI 10.1007/s10865-012-9471-8

Deblinger, L. (2001). Nutrition and cancer prevention. *Patient Care, 23*(16), 21-30.

Department of Foreign Affairs and Trade (DFAT). (2012). *People, culture and lifestyle: About Australia* Retrieved from https://www.dfat.gov.au/facts/people_culture.html (Accessed 11th June 2014).

Drewnowski, A. (2003). Fat and sugar: an economic analysis. *American Society for Nutritional Sciences, 66*(3), 838-840.

Drewnowski, A. (2004). Obesity and the food environment: Dietary energy density and diet costs. *American Journal of Preventive Medicine, 27*(3, Supplement), 154-162. doi:http://dx.doi.org/10.1016/j.amepre.2004.06.011

Drewnowski, A., & Darmon, N. (2005). The economics of obesity: Dietary energy density and energy cost. *The American Journal of Clinical Nutrition, 82*(1), 265S-273S.

Drewnowski, A., Monsivais, P., Maillot, M., & Darmon, N. (2007). Low-Energy-Density Diets Are Associated with Higher Diet Quality and Higher Diet Costs in French Adults. *Journal of the American Dietetic Association, 107*(6), 1028-1032. doi:http://dx.doi.org/10.1016/j.jada.2007.03.013

Duffey, K. J., & Popkin, B. M. (2011). Energy density, portion size, and eating occasions: Contributions to increased enerrgy intake in the United States, 1977-2006. *PLOS Medicine, 8*(6), e1001050. doi:10.1371/journal.pmed.1001050

Dunn, K. I., Mohr, P., Wilson, C. J., & Wittert, G. A. (2011). Determinants of fast-food consumption. An application of the Theory of Planned Behaviour. *Appetite, 57*(2), 349-357. doi:http://dx.doi.org/10.1016/j.appet.2011.06.004

Ehrenberg, A. S. C. (1968). The elements of lawlike relationships. *Journal of the Royal Statistical Society. Series A (General)*, 280-302.

Elhai, J. D., Calhoun, P. S., & Ford, J. D. (2008). Statistical procedures for analyzing mental health services data. *Psychiatry Research, 160*, 129 - 136. doi:10.1016/j.psychres.2007.07.003

Eli, K., Howell, K., Fisher, P. A., & Nowicka, P. (2016). A question of balance: Explaining differences between parental and grandparental perspectives on preschoolers' feeding and physical activity. *Social Science & Medicine, 154*, 28-35. doi:http://dx.doi.org/10.1016/j.socscimed.2016.02.030

Evans, A., Chow, S., Jennings, R., Dave, J., Scoblick, K., Sterba, K. R., & Loyo, J. (2011). Traditional foods and practices of Spanish-speaking Latina mothers influence the home food environment: Implications for future interventions. *Journal of the American Dietetic Association, 111*(7), 1031-1038. doi:10.1016/jada.2011.04.007

Evans, A. E., Wilson, D. K., Buck, J., Torbett, H., & Williams, J. (2006). Outcome expectations, barriers, and strategies for healthful eating - A perspective from adolescents from low-income families. *Family Community Health, 29*, 17 - 27.

Fagherazzi, G., Vilier, A., Sartorelli, D. S., Lajous, M., Balkau, B., & Clavel-Chapelon, F. (2013). Consumption of artificially and sugar-sweetened beverages and incident type 2 diabetes in the Etude Epidé miologique auprè s des femmes de la Mutuelle Gé né rale de l'Education Nationale-European Prospective Investigation into Cancer and

Nutrition cohort1-4. *American Journal of Clinical Nutrition, 97*(3), 517-523. doi:10.3945/ajcn.112.050997

Feunekes, G. I. J., de Graaf, C., Meyboom, S., & van Staveren, W. A. (1998). Food choice and fat intake of adolescents and adults: Associations of intakes within social networks. *Preventative Medicine, 27*, 645-656.

Feunekes, G. I. J., Stafleu, A., de Graaf, C., & van Staveren, W. A. (1997). Family resemblance in fat intake in the Netherlands. *European Jouranl of Clinical Nutrition, 51*, 793-799.

Finer, N. (2010). Medical consequences of obesity. *Medicine, 39*(1), 18-23.

Finucane, M. M., Stevens, G. A., Cowan, M. J., Danaei, G., Lin, J. K., Paciorek, C. J., . . . Ezzati, M. (2011). National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9·1 million participants. *The Lancet, 377*(9765), 557-567. doi:http://dx.doi.org/10.1016/S0140-6736(10)62037-5

Fishbein, M., & Yzer, M. C. (2003). Using theory to design effective health behavior interventions. *Communication theory, 13*(2), 164-183.

Forero, O., & Smith, G. (2010). The reproduction of 'cultural taste' amongst the Ukrainian Diaspora in Bradford, England. *The Sociological Review, 58*, 78-96. doi:10.1111/j.1467-954X.2011.01963.x

Fox, J. (2008). *Applied regression analysis, linear models, and related methods* (2nd ed.). Los Angeles SAGE.

Francis, J. J., Johnston, M., Robertson, C., Glidewell, L., Entwistle, V., Eccles, M. P., & Grimshaw, J. M. (2010). What is an adequate sample size? Operationalising data saturation for theory-based interview studies. *Psychology and Health, 25*(10), 1229 - 1245.

Fraser, J., Skouteris, H., McCabe, M. P., Ricciardelli, L. A., Milgrom, J., & Baur, L. A. (2011). Paternal influences on children's weight gain: A systematic review. *Fathering: A Journal of Theory, Research, and Practice about Men as Fathers, 9*(3), 252-267.

French, D. P., Cameron, E., Benton, J. S., Deaton, C., & Harvie, M. (2017). Can Communicating Personalised Disease Risk Promote Healthy Behaviour Change? A Systematic Review of Systematic Reviews. *Annals of Behavioral Medicine*, 1-12. doi:10.1007/s12160-017-9895-z

Fuchs, C. S., Giovannucci, E. L., Colditz, G. A., Hunter, D. J., Speizer, F. E., & Willett, W. C. (1994). A prospective study of family history and the risk of colorectal cancer. *New England Journal of Medicine, 331*(25), 1669-1674.

Gardiner, J. C., Luo, Z., & Roman, L. (2009). Fixed effects, random effects and GEE: What are the differences? *Statistics in Medicine, 28*(2), 221-239. doi:10.1002/sim.3478

Gardner, W., Mulvey, E. P., & Shaw, E. C. (1995). Regression analyses of counts: Poisson, overdispersed poisson and negative binomial models. *Psychological Bulletin, 118*(3), 392 - 404.

Given, L. M. (2008). *The Sage encyclopedia of qualitative research methods*: Sage Publications.

Glanz, K., Basil, M., Maibach, E., Goldberg, J., & Snyder, D. A. N. (1998). Why Americans eat what they do: Taste, nutrition, cost, convenience, and weight control concerns as influences on food consumption. *Journal of the American Dietetic Association, 98*(10), 1118-1126. doi:http://dx.doi.org/10.1016/S0002-8223(98)00260-0

Glanz, K., & Bishop, D. B. (2010). The role of behavioral science theory in development and implementation of public health interventions. *Annual Review of Public Health, 31*, 399-418.

Glaser, B. G., & Strauss, A. L. (1967). *The discovery of grounded theory, strategies for qualitative research*. Piscataway, New Jersey: Transaction Publishers, Rutgers.

Gluckman, P. D., Hanson, M., Zimmet, P., & Forrester, T. (2011). Losing the war against obesity: the need for a developmental perspective. *Science Translational Medicine, 3*(93), 93cm19.

Godin, G., Sheeran, P., Conner, M., & Germain, M. (2008). Asking questions changes behavior: Mere measurement effects on frequency of blood donation. *Health Psychology, 27*(2), 179. doi:10.1037/0278-6133.27.2.179

Godinho, C. A., Alvarez, M.-J., & Lima, M. L. (2013). Formative research on HAPA model determinants for fruit and vegetable intake: Target beliefs for audiences at different stages of change. *Health Education Research*. doi:10.1093/her/cyt076

Godino, J. G., van Sluijs, E. M. F., Marteau, T. M., Sutton, S., & Griffin, S. J. (2016). Lifestyle advice combined with personalized estimates of genetic or phenotypic risk of type 2 diabetes, and objectively measured physical activity: A randomized controlled trial. *PLOS Medicine, 13*(11). doi:https://doi.org/10.1371/journal.pmed.1002185

Golley, R. K., Magarey, A. M., Baur, L. A., Steinbeck, K. S., & Daniels, L. A. (2007). Twelve-month effectiveness of a parent-led, family-focused weight-management program for prepubertal children: A randomized, controlled trial. *Pediatrics, 119*(3), 517-525.

Green, J., E., W., Haikerwal, A., O'Neill, C., Raman, S., Booth, M. L., & Gibbons, K. (2003). Social, cultural and environmental influences on child activity and eating in Australian migrant communities. *Child: Care, Health & Development, 29*(6), 441-448.

Green, T., Owen, J., Curtis, P., Smith, G., Ward, P., & Fisher, P. (2009). Making healthy families? In P. Jackson (Ed.), *Changing families, changing food*. Basingstoke, U.K.: Palgrave Macmillan.

Greene, J. C., Caracelli, V. J., & Graham, W. F. (1989). Toward a conceptual framework for mixed-method evaluation designs. *Educational Evaluation and Policy Analysis, 11*(3), 255-274. doi:10.3102/01623737011003255

Hall, K. D., Sacks, G., Chandramohan, D., Chow, C. C., Wang, Y. C., Gortmaker, S. L., & Swinburn, B. A. (2011). Quantification of the effect of energy imbalance on bodyweight. *The Lancet, 378*(9793), 826-837.

Harrison, J. A., Mullen, P. D., & Green, L. W. (1992). A meta-analysis of studies of the Health Belief Model with adults. *Health Education Research, 7*(1), 107 - 116.

Haslam, S. A., Jetten, J., Postmes, T., & Haslam, C. (2009). Social identity, health and well-being: an emerging agenda for applied psychology. *Applied Psychology, 58*(1), 1-23.

Hearn, M. D., Baranowski, T., Baranowski, J., Doyle, C., Smith, M., Lin, L. S., & Resnicow, K. (1998). Environmental influences on dietary behavior among children: availability and accessibility of fruits and vegetables enable consumption. *Journal of Health Education, 29*(1), 26-32.

Heck, R. H., Thomas, S. C., & Tabata, L. N. (2012). *Multilevel modeling of categorical outcomes using IBM SPSS* (1st ed.). New York: Routledge.

Heck, R. H., Thomas, S. C., & Tabata, L. N. (2014a). *Multilevel and longitudinal modeling with IBM SPSS* (2nd ed.). New York: Routledge.

Heck, R. H., Thomas, S. C., & Tabata, L. N. (2014b). *Multilevel modeling of categorical outcomes using IBM SPSS* (2nd ed.). New York: Routledge.

Hendriks, A. M., Gubbels, J. S., Jansen, M. W. J., & Kremers, S. P. J. (2012). Health Beliefs regarding dietary behavior and physical activity of Surinamese immigrants of Indian descent in The Netherlands: A qualitative study. *ISRN Obesity, 2012*, 1-8. doi:10.5402/2012/903868

Hennink, M., Hutter, I., & Bailey, A. (2011). *Qualitative reseach methods*. London: SAGE.

Hollands, G. J., French, D. P., Griffin, S. J., Prevost, A. T., Sutton, S., King, S., & Marteau, T. M. (2016). The impact of communicating genetic risks of disease on risk-reducing health behaviour: systematic review with meta-analysis. *BMJ, 352*. doi:10.1136/bmj.i1102

Hovick, S. R., Wilkinson, A. V., Ashida, S., de Heer, H. D., & Koehly, L. M. (2014). The impact of personalized risk feedback on Mexican Americans' perceived risk for heart disease and diabetes. *Health Education Research, 29*(2), 222-234. doi:10.1093/her/cyt151

Hsueh, W., & Deng, T. (2016). Obesity, Inflammation, and Cancer. *Annual Review of Pathology: Mechanisms of Disease, 11*(1), null. doi:doi:10.1146/annurev-pathol-012615-044359

Hu, F. B., Rimm, E., Smith-Warner, S. A., Feskanich, D., Stampfer, M. J., Ascherio, A., . . . Willett, W. C. (1999). Reproducibility and validity of dietary patterns assessed with a

food-frequency questionnaire. *The American Journal of Clinical Nutrition, 69*(2), 243-249.

Hubbard, A. E., Ahern, J., Fleischer, N. L., Van der Laan, M., Lippman, S. A., Jewell, N., . . . Satariano, W. A. (2010). To GEE or not to GEE: comparing population average and mixed models for estimating the associations between neighborhood risk factors and health. *Epidemiology, 21*(4), 467-474.

Hughes, D., Hutchinson, A., Prichard, I., Chapman, J., & Wilson, C. (2015). Challenges associated with recruiting multigenerational, multicultural families into a randomised controlled trial: Balancing feasibility with validity. *Contemporary Clinical Trials, 43*, 185-193. doi:http://dx.doi.org/10.1016/j.cct.2015.06.004

Hughes, S. O., Shewchuk, R. M., Baskin, M. L., Nicklas, T. A., & Qu, H. (2008). Indulgent Feeding Style and Children's Weight Status in Preschool. *Journal of developmental and behavioral pediatrics : JDBP, 29*(5), 403-410. doi:10.1097/DBP.0b013e318182a976

IBM Corporation. (2013). *IBM SPSS statistics 22 command syntax reference*. Chicago, Illinois: IBM Software Group,.

Institute of Medicine (US) Committee on Health and Behavior: Research, P., and Policy (2001). *Health and Behavior: The Interplay of Biological, Behavioral, and Societal Influences Chapter 5 Individuals and Families: Models and Interventions* Retrieved from http://www.ncbi.nlm.nih.gov/books/NBK43749/

Jabs, J., Devine, C. M., Bisogni, C. A., Farrell, T. J., Jastran, M., & Wethington, E. (2007). Trying to find the quickest way: Employed mothers' constructions of time for food. *Journal of Nutrition Education and Behavior, 39*(1), 18-25. doi:http://dx.doi.org/10.1016/j.jneb.2006.08.011

Jain, A., Sherman, S., Chamberlin, D., Carter, Y., Powers, S., & Whitaker, R. (2001). Why don't low-income mothers worry about their preschoolers being overweight? *Pediatrics, 107*(5), 1138 - 1146.

Janz, N. K., & Becker, M. H. (1984). The health belief model: A decade later. *Health Education Behaviour, 11*(1), 1-47.

Jeffery, R. W., Baxter, J., McGuire, M., & Linde, J. (2006). Are fast food restaurants an environmental risk factor for obesity? . *International Journal of Behavioral Nutrition and Physical Activity, 3*(2), 1-6.

Jingxiong, J., Rosenqvist, U., Huishan, W., Greiner, T., Guangli, L., & ASarkadi, A. (2007). Influence of grandparents on eating behaviours of young children in Chinese three-generation families. *Appetite, 48*, 377 - 383. doi:10.1016/j.appet.2006.10.004

Johnson, C. M., Sharkey, J. R., Dean, W. R., McIntosh, A. W., & Kubena, K. S. (2011). It's who I am and what we eat. Mothers' food-related identities in family food choice. *Appetite, 57*(1), 220-228. doi:10.1016/j.appet.2011.04.025

Johnson, C. M., Sharkey, J. R., McIntosh, A. W., & Dean, W. R. (2010). "I'm the Momma": Using photo-elicitation to understand matrilineal influence on family food choice. *BioMed Central Women's Health, 10*(21), 1-14.

Joireman, J., Shaffer, M. J., Balliet, D., & Strathman, A. (2012). Promotion Orientation Explains Why Future-Oriented People Exercise and Eat Healthy. *Personality and Social Psychology Bulletin, 38*(10), 1272-1287. doi:doi:10.1177/0146167212449362

Kahi, C. J., & Lieberman, D. (2016). Family History of Colorectal Adenomas: Taking the Methodological Bull by the Horns. *Gastroenterology, 150*(3), 550-552. doi:http://dx.doi.org/10.1053/j.gastro.2016.01.015

Katz, D. (2014). Perspective: Obesity is not a disease. *Nature, 508*(7496), S57-S57.

Kerber, R. A., & Slattery, M. L. (1997). Comparison of self-reported and database-linked family history of cancer data in a case-control study. *American Journal of Epidemiology, 146*(3), 244-248. doi:10.1093/oxfordjournals.aje.a009259

Khandpur, N., Blaine, R. E., Fisher, J. O., & Davison, K. K. (2014). Fathers' child feeding practices: A review of the evidence. *Appetite, 78*, 110-121. doi:https://doi.org/10.1016/j.appet.2014.03.015

Kicklighter, J. R., Whitley, D. M., Kelley, S. J., Shipskie, S. M., Taube, J. L., & Berry, R. C. (2007). Grandparents raising grandchildren: A response to a nutrition and physical activity intervention. *Journal of the American Dietetic Association, 107*(7), 1210 - 1213. doi:http://dx.doi.org/10.1016/j.jada.2007.04.006

Kling, S. M. R., Roe, L. S., Keller, K. L., & Rolls, B. J. (2016). Double trouble: Portion size and energy density combine to increase preschool children's lunch intake. *Physiology & Behavior, 162*, 18-26. doi:http://dx.doi.org/10.1016/j.physbeh.2016.02.019

Koehly, L. M., Morris, B. A., Skapinsky, K., Goergen, A., & Ludden, A. (2015). Evaluation of the Families SHARE workbook: an educational tool outlining disease risk and healthy guidelines to reduce risk of heart disease, diabetes, breast cancer and colorectal cancer. *BMC Public Health, 15*(1), 1-15. doi:10.1186/s12889-015-2483-x

Koehly, L. M., Peters, J. A., Kenen, R., Hoskins, L. M., Ersig, A. L., Kuhn, N. R., . . . Greene, M. H. (2009). Characteristics of health information gatherers, disseminators, and blockers within families at risk of hereditary cancer: Implications for family health communication interventions. *American Journal of Public Health, 99*(12), 2203-2209.

Koehly, L. M., Peterson, S. K., Watts, B. G., Kempf, K. K., Vernon, S. W., & Gritz, E. R. (2003). A social network analysis of communication about hereditary nonpolyposis colorectal cancer genetic testing and family functioning. *Cancer Epidemiol Biomarkers Prev, 12*.

Kratt, P., Reynolds, K., & Shewchuk, R. (2000). The role of availability as a moderator of family fruit and vegetable consumption. *Health Educion and Behaviour, 27*(4), 471 - 482.

Krolner, R., Rasmussen, M., Brug, J., Klepp, K.-I., Wind, M., & Due, P. (2011). Determinants of fruit and vegetable consumption among children and adolescents: A review of the literature. Part II: Qualitative studies. *International Journal of Behavioral Nutrition and Physical Activity, 8*(1), 112.

Krueger, R. A. (1998a). *The focus group kit: Developing questions for focus groups* (Vol. 3). Thousand Oaks, California: SAGE Publications Inc.

Krueger, R. A. (1998b). *The focus group kit: Moderating focus groups* (Vol. 4). Thousand Oaks, California: SAGE Publications Inc.

Krueger, R. A., & Casey, M. A. (2009). *Focus groups: A practical guide for applied research* (4th ed.). Thousand Oaks, California: SAGE Publications, Inc.

Lachat, C., Nago, E., Verstraeten, R., Roberfroid, D., Van Camp, J., & Kolsteren, P. (2012). Eating out of home and its association with dietary intake: A systematic review of the evidence. *Obesity Reviews, 13*(4), 329-346.

Lawrence, V. J., & Kopelman, P. G. (2004). Medical consequences of obesity. *Clinics in Dermatology, 22*, 296-302. doi:*10.1016/j.clindermatol.2004.01.012*

Lee, A. H., Wang, K., Scott, J. A., Yau, K. K. W., & McLachlan, G. J. (2006). Multi-level zero-inflated Poisson regression modelling of correlated count data with excess zeros. *Statistical Methods in Medical Research, 15*(1), 47-61. doi:10.1191/0962280206sm429oa

Leung, D. Y., Wong, E. M., & Chan, C. W. (2016). Determinants of participation in colorectal cancer screening among community-dwelling Chinese older people: Testing a comprehensive model using a descriptive correlational study. *European Journal of Oncology Nursing, 21*, 17-23.

Levine, M. E., Suarez, J. A., Brandhorst, S., Balasubramanian, P., Cheng, C.-W., Madia, F., . . . Longo, V. D. (2014). Low protein intake is associated with a major reduction in IGF-1, cancer, and overall mortality in the 65 and younger but not older population. *Cell metabolism, 19*(3), 407-417. doi:10.1016/j.cmet.2014.02.006

Lewin, S., Glenton, C., & Oxman, A. D. (2009). *Use of qualitative methods alongside randomised controlled trials of complex healthcare interventions: Methodological study* (Vol. 339).

Lewis, S., Thomas, S. L., Hyde, J., Castle, D., Blood, R. A., & Komesaroff, P. A. (2010). "I don't eat a hamburger and large chips every day!" A qualitative study of the impact of public health messages about obesity on obese adults. *BMC Public Health, 10*, (1), 1. doi:10.1186/1471-2458-10-309

Liamputtong, P. (2013). *Research methods in health: Foundations for evidence based practice* (P. Liamputtong Ed. 2nd ed.). Melbourne: Oxford University Press.

Lim, S. S., Vos, T., Flaxman, A. D., Danaei, G., Shibuya, K., Adair-Rohani, H., . . . Ezzati, M. (2012). A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic

analysis for the Global Burden of Disease Study 2010. *The Lancet, 380*(9859), 2224-2260. doi:http://dx.doi.org/10.1016/S0140-6736(12)61766-8

Linke, S. E., Robinson, C. J., & Pekmezi, D. (2013). Applying psychological theories to promote healthy lifestyles. *American Journal of Lifestyle Medicine, 10*(10), 1 - 11. doi:10.1177/1559827613487496

Livingstone, M. B. E., & Robson, P. J. (2007). Measurement of dietary intake in children. *Proceedings of the Nutrition Society, 59*(2), 279-293. doi:10.1017/S0029665100000318

Longbottom, P., Wrieden, W., & Pine, C. (2002). Is there a relationship between the food intakes of Scottish 5 1/2-81/2-year-olds and those of their mothers? *J Hum Nutr Diet, 15*, 271 - 279.

Lozano, R., Naghavi, M., Foreman, K., Lim, S. S., Shibuya, K., Aboyans, V., . . . Hoen, B. (2012). Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *The Lancet, 380*(9859), 2095-2128. doi:10.1016/S0140-6736(12)61728-0

Lykins, E. L. B., Graue, L. O., Brechting, E. H., Roach, A. R., Gochett, C. G., & Andrykowski, M. A. (2008). Beliefs about cancer causation and prevention as a function of personal and family history of cancer: a national, population-based study. *Psycho-Oncology, 17*(10), 967-974. doi:10.1002/pon.1306

Lyons, R. F., Mickelson, K. D., Sullivan, M. J., & Coyne, J. C. (1998). Coping as a communal process. *Journal of Social and Personal Relationships, 15*(5), 579 - 605.

Maccoby, E. E. (1992). The role of parents in the socialization of children: An historical overview. *Developmental Psychology, 28*(6), 1006 - 1017.

MacFarlane, A., Abbott, G., Crawford, D., & Ball, K. (2010). Personal, social and environmental correlates of healthy weight status amongst mothers from socioeconomically disadvantaged neighborhoods: findings from the READI study. *International Journal of Behavior Nutrition and Physical Activity, 7*, 23.

Macpherson, A., Bonita, R., Beaglehole, R., & Kjellström, T. (2008). *Basic Epidemiology*. Geneva: World Health Organisation.

Mallan, K. M., Daniels, L. A., Nothard, M., Nicholson, J. M., Wilson, A., Cameron, C. M., . . . Thorpe, K. (2013). Dads at the dinner table: A cross-sectional study of Australian fathers' child feeding perceptions and practices. *Appetite*.

McBride, C. M., Koehly, L. M., Sanderson, S. C., & Kaphingst, K. A. (2010). The behavioral response to personalized genetic information: will genetic risk profiles motivate individuals and families to choose more healthful behaviors? *Annu Rev Public Health, 31*.

McBride, C. M., Persky, S., Wagner, L. K., Faith, M. S., & Ward, D. S. (2013). Effects of providing personalized feedback of child's obesity risk on mothers' food choices using a virtual reality buffet. *Int J Obes (Lond), 37*.

McGraw, K. O., & Wong, S. P. (1996). Forming inferences about some intraclass correlation coefficients. *Psychological methods, 1*(1), 30.

McWhirter, J. E., & Hoffman-Goetz, L. (2016). Application of the Health Belief Model to US Magazine Text and Image Coverage of Skin Cancer and Recreational Tanning (2000–2012). *Journal of health communication*, 1-15.

Medin, D. L. (1992). *The psychology of learning and motivation* (Vol. 28). San Diego: Academic Press Incorporated.

Melvin, J. C., Wulaningsih, W., Hana, Z., Purushotham, A. D., Pinder, S. E., Fentiman, I., . . . Van Hemelrijck, M. (2016). Family history of breast cancer and its association with disease severity and mortality. *Cancer Medicine*, n/a-n/a. doi:10.1002/cam4.648

Micha, R., Khatibzadeh, S., Shi, P., Andrews, K. G., Engell, R. E., & Mozaffarian, D. (2015). Global, regional and national consumption of major food groups in 1990 and 2010: a systematic analysis including 266 country-specific nutrition surveys worldwide. *BMJ open, 5*(9). doi:10.1136/bmjopen-2015-008705

Michael, H., Jennie, H., Matthew, K., Mark, D., Bill, M., & Alan, J. (2014). Asthma, COPD, Respiratory Symptoms And Comorbidities In "Baby Boomers": The Busselton Healthy Ageing Study *Asthma and COPD: Etiology, morbidity, and mortality* (pp. A4608-A4608). USA: American Thoracic Society.

Montgomery-Anderson, R. A., & Borup, I. (2012). Family support and the child as health promoting agent in the Arctic: "the Inuit way". *Rural and Remote Health, 12*, 1-9.

Morgan, D. L. (1997). *Focus groups as qualitiative research* (Vol. 16). Thousand Oaks, California: Sage Publications.

Morwitz, V. G., & Fitzsimons, G. J. (2004). The mere-measurement effect: Why does measuring intentions change actual behavior? *Journal of Consumer Psychology, 14*(1), 64-74. doi:http://dx.doi.org/10.1207/s15327663jcp1401&2_8

Muntaner, C., & Gómez, M. B. (2003). Qualitative and quantitative research in social epidemiology: Is complementarity the only issue? *Gaceta Sanitaria, 17*, 53-57.

Musher-Eizenman, D. R., de Lauzon-Guillain, B., Holub, S. C., Leporc, E., & Charles, M. A. (2009). Child and parent characteristics related to parental feeding practices. A cross-cultural examination in the US and France. *Appetite, 52*(1), 89-95.

National Health and Medical Research Council. (2013). *Eat for health: Australian dietary guidelines, Summary*. Retrieved from Canberra:

NBOCC. (2010). *Advice about familial aspects of breast cancer and epithelial ovarian cancer: A guide for health professionals*. Retrieved from NSW, Australia:

Nestle, M., Wing, R., Birch, L., DiSogra, L., Drewnowski, A., Middleton, S., . . . Economos, C. (1998). Behavioral and Social Influences on Food Choice. *Nutrition Reviews, 56*(5), 50-64. doi:10.1111/j.1753-4887.1998.tb01732.x

NHMRC. (1999). *Familial aspects of cancer: A guide to clinical practice*. Canberra, : National Health and Medical Research Council.

NHMRC. (2005). *Clinical practice guidelines for the prevention, early detection and management of colorectal cancer*. Canberra,: National Health and Medical Research Council.

NHMRC. (2006). *Nutrient reference values for Australia and New Zealand*. Canberra,: National Health and Medical Research Council.

NHMRC. (2009a). *Australian guidelines to reduce risks from drinking alcohol*. Canberra,: National Health and Medical Research Council.

NHMRC. (2009b). *National evidence based guideline for the primary prevention of type 2 diabetes* Canberra,: National Health and Medical Research Council.

NHMRC. (2013a). *Australian Dietary Guidelines*. Canberra,: National Health and Medical Research Council.

NHMRC. (2013b). *Clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children in Australia*. Canberra,: National Health and Medical Research Council.

NHMRC. (2013c). *Eat for health: Australian dietary guidelines, Summary*. Canberra,: National Health and Medical Research Council.

Nicklas, T. A., Baranowski, T., Baranowski, J. C., Cullen, K. W., Rittenberry, L., & Olvera, N. (2001). Family and Child-care Provider Influences on Preschool Children's Fruit, Juice, and Vegetable Consumption. *Nutrition Reviews, 59*(7), 224-235. doi:10.1111/j.1753-4887.2001.tb07014.x

Nishida, C., Uauy, R., Kumanyika, S., & Shetty, P. (2004). The Joint WHO/FAO Expert Consultation on diet, nutrition and the prevention of chronic diseases: Process, product and policy implications. *Public Health Nutrition, 7*(1a), 245-250. doi:10.1079/PHN2003592

NVDPA. (2012). *Guidelines for the management of absolute cardiovascular disease risk*. Canberra,: National Heart Foundation of Australia.

O'Connell, J. (2015). *Me and My Big Mouth*. United States: Blurb Incorporated.

O'Neill, S. M., Rubinstein, W. S., Wang, C., Yoon, P. W., Acheson, L. S., Rothrock, N., . . . Ruffin, M. T. (2009). Familial risk for common diseases in primary care: The Family Healthware Impact Trial. *American Journal of Preventative Medicine, 36*(6), 506-514. doi:10.1016/j.amepre.2009.03.002

O'Neill, S. M., Rubinstein, W. S., Wang, C., Yoon, P. W., Acheson, L. S., Rothrock, N., . . . Ruffin, M. T. (2009). Familial risk for common diseases in primary care: the Family Healthware™ Impact Trial. *Am J Prev Med, 36*.

Oertelt-Prigione, S., Kendel, F., Lehmkuhl, E., Hetzer, R., & Regitz-Zagrosek, V. (2014). Impact of gender and age on risk factor distribution and health perception: evaluation

in a prospective population with heart disease. *Journal of Public Health, 22*(3), 219-226. doi:10.1007/s10389-013-0609-0

Oyebode, O., Gordon-Dseagu, V., Walker, A., & Mindell, J. S. (2014). Fruit and vegetable consumption and all-cause, cancer and CVD mortality: Analysis of Health Survey for England data. *Journal of Epidemiology and Community Health, 68*(9), 856-862. doi:10.1136/jech-2013-203500

Pallant, J. (2011). *SPSS survival manual: A step by step guide to data analysis using SPSS* (Fourth ed.). Crows Nest, NSW: Allen & Unwin.

Patrick, H., & Nicklas, T. A. (2005). A review of family and social determinants of children's eating patterns and diet quality. *Journal of the American College of Nutrition, 24*(2), 83-92.

Pearson, N., Biddle, S., & Gorely, T. (2009). Family correlates of fruit and vegetable consumption in children and adolescents: a systematic review. *Public Health and Nutrition, 12*, 267 - 283.

Peat, J. (2002). *Health science research: A handbook of quantitative methods*. Sydney: Allen and Unwin.

Perry, D. G., & Pauletti, R. E. (2011). Gender and adolescent development. *Journal of Research on Adolescence, 21*(1), 61-74. doi:10.1111/j.1532-7795.2010.00715.x

Pijl, M., Timmermans, D. R. M., Claassen, L., Janssens, A. C. J. W., Nijpels, G. J., Dekker, J. M., . . . Henneman, L. (2009). Impact of communicating familial risk of diabetes on illness perceptions and self-reported behavioural outcomes: A randomized controlled trial. *Diabetes Care, 4*, 597-599.

Pilla, R., Loblay, R. H., Director, R., Soutter, V., & Swain, A. R. (2016). *Psychology-Diet Interactions in Patients Following the Royal Prince Alfred Hospital Elimination Diet and Challenge Protocol*. Royal Prince Alfred Hospital Sydney.

Pollard, C., Miller, M., Woodman, R. J., Meng, R., & Binns, C. (2009). Changes in knowledge, beliefs, and behaviors related to fruit and vegetable consumption among Western Australian adults from 1995 to 2004. *American Journal of Public Health, 99*(2), 355-361. doi:10.2105/ajph.2007.131367

Popkin, B. M., Adair, L. S., & Ng, S. W. (2012). Global nutrition transition and the pandemic of obesity in developing countries. *Nutrition Reviews, 70*(1), 3-21. doi:10.1111/j.1753-4887.2011.00456.x

Prelip, M., Kinsler, J., Thai, C. L., Erausquin, J. T., & Slusser, W. (2012). Evaluation of a School-based Multicomponent Nutrition Education Program to Improve Young Children's Fruit and Vegetable Consumption. *Journal of Nutrition Education and Behavior, 44*(4), 310-318. doi:http://dx.doi.org/10.1016/j.jneb.2011.10.005

Prichard, I., Hodder, K., Hutchinson, A., & Wilson, C. (2012). Predictors of mother-daughter resemblance in dietary intake. The role of eating styles, mothers' consumption and closeness. *Appetite, 58*, 271-276. doi:10.1016/j.appet.2011.10.012

Prichard, I., Lee, A., Hutchinson, A. D., & Wilson, C. (2015). Familial risk for lifestyle-related chronic diseases: Can family health history be used as a motivational tool to promote health behaviour in young adults? *Health Promotion Journal of Australia*, Online early. doi:http://dx.doi.org/10.1071/HE14104

Prochaska, J. O., DiClemente, C. C., & Norcross, J. C. (1992). In search of how people change: Applications to addictive behaviors. *American Psychologist, 47*(9), 1102-1114.

Puhl, R., Peterson, J. L., & Luedicke, J. (2013). Fighting obesity or obese persons? Public perceptions of obesity-related health messages. *International Journal of Obesity*(6), 774-782. doi:10.1038/ijo.2012.156

Rasmussen, M., Krolner, R., Klepp, K.-I., Lytle, L., Brug, J., Bere, E., & Due, P. (2006). Determinants of fruit and vegetable consumption among children and adolescents: A review of the literature. Part 1: Quantitative studies. *International Journal of Behavioral Nutrition and Physical Activity, 3*(22). doi:10.1186/1479-5868-3-22

Raubenheimer, D., Machovsky-Capuska, G. E., Gosby, A. K., & Simpson, S. (2015). Nutritional ecology of obesity: from humans to companion animals. *British Journal of Nutrition, 113*(SupplementS1), S26-S39. doi:doi:10.1017/S0007114514002323

Reifsnyder, P. C., & Leiter, E. H. (2000). Genetic and environmental links to obesity and diabetes. *Journal of the Royal Society for the Promotion of Health, 120*(4), 209-210.

Resnicow, K., Davis-Hearn, M., Smith, M., Baranowski, T., Lin, L., Baranowski, J., . . . Wang, D. (1997). Social-cognitive predictors of fruit and vegetable intake in children. *Health Psychol, 16*, 272 - 276.

Rhodes, K., Chan, F., Prichard, I., Coveney, J., Ward, P., & Wilson, C. (2016). Intergenerational transmission of dietary behaviours: A qualitative study of Anglo-Australian, Chinese-Australian and Italian-Australian three-generation families. *Appetite, 103*, 309-317.

Rhodes, K., Wilson, C., Prichard, I., Hutchinson, A., Coveney, J., & P., W. (2014). Dietary choices within multigenerational Australian families: Does the mother still play the most important role? *International Journal of Behavioral Medicine, 21*(Supplement 1), S28. doi:10.1007/s12529-014-9418-2

Ritchie, L. D., Welk, G., Styne, D., Gerstein, D. E., & Crawford, P. B. (2005). Family Environment and Pediatric Overweight: What Is a Parent to Do? *Journal of the American Dietetic Association, 105*(5, Supplement), 70-79. doi:http://dx.doi.org/10.1016/j.jada.2005.02.017

Rodgers, R., & Chabrol, H. (2009). Parental attitudes, body image disturbance and disordered eating amongst adolescents and young adults: A review. *European Eating Disorders Review, 17*(2), 137-151. doi:10.1002/erv.907

Rolland, J. S. (1987). Chronic illness and the life cycle: A conceptual framework. *Family process, 26*(2), 203-221.

Rolland, J. S. (2005). Cancer and the family: an integrative model. *Cancer, 104*(S11), 2584-2595.

Romaguera, D., Vergnaud, A.-C., Peeters, P. H., van Gils, C. H., Chan, D. S. M., Ferrari, P., . . . Norat, T. (2012). Is concordance with World Cancer Research Fund/American Institute for Cancer Research guidelines for cancer prevention related to subsequent risk of cancer? Results from the EPIC study. *The American Journal of Clinical Nutrition, 96*(1), 150-163. doi:10.3945/ajcn.111.031674

Rosenheck, R. (2008). Fast food consumption and increased caloric intake: A systematic review of a trajectory towards weight gain and obesity risk. *Obesity Reviews, 9*(6), 535-547.

Rosenkranz, R. R., & Dzewaltowski, D. A. (2008). Model of the home food environment pertaining to childhood obesity. *Nutrition Reviews, 66*(3), 123-140.

Rosenstock, I. M. (1974). Historical origins of the Health Belief Model *Health Education Monographs, 2*(4), 175 - 183.

Rosenstock, I. M., Strecher, V. J., & Becker, M. H. (1988). Social Learning Theory and the Health Belief Model. *Health Education Quarterly, 15*(2), 175 - 183.

Rowen, L., Milner, J. A., & Ross, S. (2010). Obesity, cancer and epigenetics. *Bariatric Nursing and Surgical Patient Care, 5*(4). doi:10.1089/bar.2010.9987

Rozin, P., Bauer, R., & Catanese, D. (2003). Food and life, pleasure and worry, among American college students: Gender differences and regional similarities. *Journal of Personality and Social Psychology, 85*, 132-141. doi:10.1037/0022-3514.85.1.132

Rozin, P., Fischler, C., Imada, S., Sarubin, A., & Wrzesniewski, A. (1999). Attitudes to food and the role of food in life in the USA, Japan, Flemish Belgium and France: Possible implications for the diet-health debate. *Appetite, 33*, 163 - 180.

Ruffin, M. T., Nease, D. E., Sen, A., Pace, W. D., Wang, C., Acheson, L. S., . . . Gramling, R. (2011). Effect of preventive messages tailored to family history on health behaviors: The Family Healthware impact trial. *The Annals of Family Medicine, 9*(1), 3-11. doi:10.1370/afm.1197

Sakamoto, Y., Ishiguro, M., Kitagawa, G., Dordrecht, D., & Reidel, G. (1988). A Bayesian approach to nonparametric test problems. *Annual Institute of Statistical Mathematics, 40*(3), 587 - 602.

Saks, M., & Allsop, J. (2012). *Researching health: Qualitative, quantitative and mixed methods*. London: Sage.

Salari, R., & Filus, A. (2016). Using the Health Belief Model to explain mothers' and fathers' intention to participate in universal parenting programs. *Prevention Science*, 1-12.

Saldana, J. (2012). *The coding manual for qualitative researchers*. London: SAGE Publications Limited.

Sarkin, J. A., Johnson, S. S., Prochaska, J. O., & Prochaska, J. M. (2001). Applying the Transtheoretical Model to Regular Moderate Exercise in an Overweight Population: Validation of a Stages of Change Measure. *Preventive Medicine, 33*(5), 462-469. doi:10.1006/pmed.2001.0916

Savage, J. S., Orlet Fisher, J., & Birch, L. L. (2007). Parental influence on eating behavior: Conception to adolescence. *Journal of Law and Medical Ethics, 35*(1), 22 - 34. doi:10.1111/j.1748-720X.2007.00111.x.

Scarborough, P., Nnoaham, K. E., Clarke, D., Capewell, S., & Rayner, M. (2012). Modelling the impact of a healthy diet on cardiovascular disease and cancer mortality. *Journal of Epidemiology and Community Health, 66*(5), 420-426. doi:10.1136/jech.2010.114520

Scarinci, I. C., Bandura, L., Hidalgo, B., & Cherrington, A. (2012). Development of a Theory-Based (PEN-3 and Health Belief Model), Culturally Relevant Intervention on Cervical Cancer Prevention Among Latina Immigrants Using Intervention Mapping. *Health Promotion Practice, 13*(1), 29-40. doi:10.1177/1524839910366416

Scott, S. (2016). Sugar tax: Doctors call for sweet drink levy to tackle obesity in Australia, News *ABC News*. Retrieved from http://www.abc.net.au/news/2016-11-11/doctors-groups-call-for-sugar-tax-to-tackle-obesity/8012626

Seaborn, C., Suther, S., Lee, T., Kiros, G. E., Becker, A., Campbell, E., & Collins-Robinson, J. (2016). Utilizing Genomics through Family Health History with the Theory of Planned Behavior: Prediction of Type 2 Diabetes Risk Factors and Preventive Behavior in an African American Population in Florida. *Public Health Genomics, 19*(2), 69-80.

Shaikh, A. R., Yaroch, A. L., Nebeling, L., Yeh, M.-C., & Resnicow, K. (2008). Psychosocial predictors of fruit and vegetable consumption in adults: A review of the literature. *American Journal of Preventive Medicine, 34*(6), 535-543.e511. doi:http://dx.doi.org/10.1016/j.amepre.2007.12.028

Shapiro, A. (2004). Revisiting the generation gap: Exploring the relationships of parent/adult-child dyads. *The International Journal of Aging and Human Development, 58*(2), 127-146. doi:10.2190/evfk-7f2x-kqnv-dh58

Sharp, G., Hutchinson, A. D., Prichard, I., & Wilson, C. (2013). Validity and reliability of the Food-Life Questionnaire. Short form. *Appetite, 70*, 112-118. doi:10.1016/j.appet.2013.07.001

Sheeran, P., & Abraham, C. (1996). The health belief model. *Predicting health behaviour, 2*, 29-80.

Sheeran, P., Harris, P. R., & Epton, T. (2014). Does heightening risk appraisals change people's intentions and behavior? A meta-analysis of experimental studies. *Psychological Bulletin, 140*(2), 511-543. doi:http://dx.doi.org/10.1037/a0033065

Shim, J.-S., Oh, K., & Kim, H. C. (2014). Dietary assessment methods in epidemiologic studies. *Epidemiology and Health, 36*, e2014009. doi:10.4178/epih/e2014009

Skov, T., Deddens, J., Petersen, M. R., & Endahl, L. (1998). Prevalence proportion ratios: Estimation and hypothesis testing. *International Journal of Epidemiology, 27*(1), 91-95.

Sleddens, E. F. C., Gerards, S. M. P. L., Thijs, C., de Vries, N. K., & Kremers, S. P. J. (2011). General parenting, childhood overweight and obesity-inducing behaviors: A review. *International Journal of Pediatric Obesity, 6*(2Part2), e12-e27. doi:10.3109/17477166.2011.566339

Sobal, J., Bisogni, C., Devine, C. M., & Jastran, M. (2006). A conceptual model of the food choice process over the life course In R. Shepherd, M. Raats, & Nutrition Society (Great Britain) (Eds.), The psychology of food choice (pp. 1-7). Wallingford, Oxford: CAB International Retrieved from https://books.google.com.au/books?hl=en&lr=&id=_t0IoTcVxIIC&oi=fnd&pg=PA1&dq=Development+of+habitual+food+choices&ots=1miQwIjdef&sig=pjzqHOCWGXjWhxqjO9eHoTlwQmI#v=onepage&q&f=false.

Sokol, R. L., Qin, B., & Poti, J. M. (2017). Parenting styles and body mass index: a systematic review of prospective studies among children. *Obesity Reviews, 18*(3), 281-292. doi:10.1111/obr.12497

Srivastava, M. S. (1984). Estimation of interclass correlations in familial data. *Biometrika, 71*(1), 177-185. doi:10.1093/biomet/71.1.177

Stacy, R. D., & Loyd, B. H. (1990). An investigation of beliefs about smoking among diabetes patients: Information for improving cessation efforts. *Patient Education and Counselling, 15*, 181-189.

Stewart, B., & Wild, C. P. (Eds.). (2014). *World Cancer Report*. Lyon: International Agency for Research on Cancer & WHO Press.

Story, M., Kaphingst, K. M., Robinson-O'Brien, R., & Glanz, K. (2008). Creating healthy food and eating environments: Policy and environmental approaches. *Annual Reviews Public Health, 29*, 253 - 272. doi:10.1146/annurev.publhealth.29.020907.090926

Strathman, A., Gleicher, F., Boninger, D. S., & Edwards, C. S. (1994). The consideration of future consequences: Weighing immediate and distant outcomes of behaviour. *Journal of Personality and Social Psychology, 66*(4), 742-752. doi:10.1037/0022-3514.66.4.742

Strecher, V. J., & Rosenstock, I. M. (1997). The health belief model *Cambridge handbook of psychology, health and medicine* (pp. 113-117). Cambridge, UK: Cambridge University Press.

Swinburn, B. A., Sacks, G., Hall, K. D., McPherson, K., Finegood, D. T., Moodie, M. L., & Gortmaker, S. L. (2011). The global obesity pandemic: shaped by global drivers and local environments. *The Lancet, 378*(9793), 804-814.

Tabachnick, B. G., & Fidell, L. S. (2013). *Using multivariate statistics* (6th ed.). New Jersey: Pearson Education Incorporated.

Tenenbaum, H. R., & May, D. (2014). Gender in parent–child relationships. *Gender and development*, 1-19.

The G. B. D. Obesity Collaboration, Ng, M., Fleming, T., Robinson, M., Thomson, B., Graetz, N., . . . Gakidou, E. (2014). Global, regional and national prevalence of overweight and obesity in children and adults 1980-2013: A systematic analysis. *Lancet (London, England), 384*(9945), 766-781. doi:10.1016/S0140-6736(14)60460-8

The Lancet Diabetes & Endocrinology Review. (2016). Sugar: pushing forward on cutting back. *The Lancet Diabetes & Endocrinology, 4*(2), 85. doi:http://dx.doi.org/10.1016/S2213-8587(16)00009-7

The Royal Australian College of General Practitioners (RACGP), Australian Government Department of Health and Ageing (AGDHA), The Cancer Council Australia (CCA), & Australian Cancer Network (ACN). (2008). Familial aspects of bowel cancer: A guide for health professionals (pp. 1 - 4). Canberra: Commonwealth of Australia.

Usher-Smith, J. A., Silarova, B., Schuit, E., Moons, K. G., & Griffin, S. J. (2015). Impact of provision of cardiovascular disease risk estimates to healthcare professionals and patients: a systematic review. *BMJ open, 5*(10), e008717.

Valdez, R., Yoon, P. W., Qureshi, N., Green, R. F., & Khoury, M. J. (2010). Family history in public health practice: A genomic tool for disease prevention and health promotion. *Annual Review of Public Health, 31*, 69-87. doi:10.1146/annurev.publhealth.012809.103621

Vereecken, C. A., Keukelier, E., & Maes, L. (2004). Influence of mother's educational level on food parenting practices and food habits of young children. *Appetite, 43*(1), 93-103. doi:http://dx.doi.org/10.1016/j.appet.2004.04.002

Vernarelli, J. A., Mitchell, D. C., Rolls, B. J., & Hartman, T. J. (2014). Dietary energy density is associated with obesity and other biomarkers of chronic disease in US adults. *European Journal of Nutrition, 54*(1), 59-65. doi:10.1007/s00394-014-0685-0

Vernon, S. W. (1999). Risk perception and risk communication for cancer screening behaviors: a review. *J Natl Cancer Inst Monogr, 25*, 101-119.

Vollmer, R. L., & Mobley, A. R. (2013). Parenting styles, feeding styles, and their influence on child obesogenic behaviors and body weight. A review. *Appetite, 71*, 232-241.

Wake, M., Nicholson, J. M., Hardy, P., & Smith, K. (2007). Preschooler Obesity and Parenting Styles of Mothers and Fathers: Australian National Population Study. *Pediatrics, 120*(6), e1520-e1527. doi:10.1542/peds.2006-3707

Walker, S. N., Volkan, K., Sechrist, K. R., & Pender, N. J. (1988). Health-promoting life styles of older adults: comparisons with young and middle-aged adults, correlates and patterns. *Advances in Nursing Science, 11*(1), 76-90.

Wang, C., O'Neill, S. M., Rothrock, N., Gramling, R., Sen, A., Acheson, L. S., . . . Ruffin Iv, M. T. (2009). Comparison of risk perceptions and beliefs across common chronic diseases. *Preventive Medicine, 48*(2), 197-202. doi:http://dx.doi.org/10.1016/j.ypmed.2008.11.008

Wardle, J. (1993). Food choices and health evaluation. *Psychology and Health, 8*(1), 65-75.

Wardle, J., & Cooke, L. (2008). Genetic and environmental determinants of children's food preferences. *British Journal of Nutrition 99*(Supplement S1), S15 - S21. doi:10.1017/S000711450889246X

Wasser, H. M., Thompson, A. L., Siega-Riz, A. M., Adair, L. S., & Hodges, E. A. (2013). Who's feeding baby? Non-maternal involvement in feeding and its association with dietary intakes among infants and toddlers. *Appetite, 71*, 7-15. doi:10.1016/j.appet.2013.06.096

Webb, T. L., Joseph, J., Yardley, L., & Michie, S. (2010). Using the Internet to Promote Health Behavior Change: A Systematic Review and Meta-analysis of the Impact of Theoretical Basis, Use of Behavior Change Techniques, and Mode of Delivery on Efficacy. *Journal of Medical Internet Research, 12*(1), e4. doi:10.2196/jmir.1376

Webb, T. L., & Sheeran, P. (2006). Does changing behavioral intentions engender behavior change? A meta-analysis of the experimental evidence. *Psychological Bulletin, 132*(2), 249.

White, M. (2007). Food access and obesity *Obesity Reviews, 8*(1), 99-107.

Whitney, E., & Rolfes, S. R. (2007). *Understanding nutrition*. Boston: Cengage Learning.

WHO, & FAOUN. (2003). *Diet, nutrition and the prevention of chronic diseases: Report of a joint World Health Organisation/Food and Agricultural Organization of the United Nations expert consultation* (Joint Technical Report 916). Retrieved from Geneva: http://www.fao.org/docrep/005/ac911e/ac911e00.HTM

Williams, K., Steptoe, A., & Wardle, J. (2013). Is a cancer diagnosis a trigger for health behaviour change? Findings from a prospective, population-based study. . *British Journal of Cancer*. doi:doi: 10.1038/bjc.2013.254

Williams, P., Fitzsimons, G. J., & Block, L. G. (2004). When Consumers Do Not Recognize "Benign" Intention Questions as Persuasion Attempts. *Journal of Consumer Research, 31*(3), 540-550. doi:10.1086/425088

Wilson, A., Renzaho, A. M. N., McCabe, M., & Swinburn, B. (2010). Towards understanding the new food environment for refugees from the Horn of Africa in Australia. *Health & place, 16*(5), 969-976.

Wilson, C. J., de la Haye, K., Coveney, J., Hughes, D. L., Hutchinson, A., Miller, C., . . . Koehly, L. M. (2016). Protocol for a randomized controlled trial testing the impact of feedback on familial risk of chronic diseases on family-level intentions to participate in preventive lifestyle behaviors. *BMC Public Health, 16*(1), 965.

Wing, R. R., Venditti, E., Jakicic, J. M., Polley, B. A., & Lang, W. (1998). Lifestyle Intervention in Overweight Individuals With a Family History of Diabetes. *Diabetes Care, 21*(3), 350-359. doi:10.2337/diacare.21.3.350

Wolinsky, A. S. (2016). *Food attitudes across adolescence.* Rutgers University-Camden Graduate School.

Wroten, K. C., O'Neil, C. E., Stuff, J. E., Liu, Y., & Nicklas, T. A. (2012). Resemblance of dietary intakes of snacks, sweets, fruit, and vegetables among mother–child dyads from low income families. *Appetite, 59*(2), 316-323. doi:http://dx.doi.org/10.1016/j.appet.2012.05.014

Yoon, P. W., Scheuner, M. T., Paterson-Oehlke, K. L., Gwinn, M., Faucett, A., & Khoury, M. J. (2002). Can family history be used as a tool for public health and preventative medicine? *Genetics in Medicine, 4*(4), 304-310.

APPENDICES

CHAPTER 2 APPENDICES

Appendix 2.A: Family Interview Discussion Guide (Brief Version).

Family Interview Discussion Guide (Brief Version).

Let's begin by going around the room one by one to get to know each other and find out how you all relate to each other. Could we start by stating our first names, age of the children and your position in the family?

1. [Original] First, let's talk through a typical *weekday* and the occasions that involve eating food. I'd be interested to hear about your typical eating occasions, such as meals, snacks, coffee breaks etc.?
    1. [Revised] First, let's talk through your usual *weekday*, and the times of day that involve eating food. For example, when would you usually have meals, snacks or coffee breaks?
    2. I wondered how this might be different on *weekends*.
    3. I wondered *when* your family members might talk about food.
    4. I'm really interested to hear which family members talk most with you about food? (e.g., describe some of the family influences on food decisions).
    5. I'd like to go on now, to discuss where you get information from about healthy eating or food preparation?
    6. Of these, which do you think influence your family most?
    7. Next, we would like to talk about meal planning. I'd be really interested to hear how your family *plans* meals.
    8. I wondered *who* is typically involved in preparing the food in your family.
    9. Next I'd like to discuss how the way you prepare food may have *changed over time.* Half-way point acknowledgement "We are about half way through the discussion

right now and you have raised some very important points. I think the discussion is going really well – how is everyone else going with it?"

10. Next I'd like to discuss shopping for food…I'm really interested in how your family *decides* what food to purchase?
11. Can you tell us *who* typically purchases food for the whole family?
12. Also, I wondered if you had any thoughts about how the *types* of foods you eat may have changed over time (e.g., more multi-cultural/fast/healthier foods).
13. …I'm really interested in what you think has influenced these changes?
14. I'm really interested to hear about the *type* of foods that you eat most often (e.g., multi-cultural, recipes handed down generations)
15. I wondered if you had any thoughts about how *new* foods are introduced to your family's diet and where ideas for new foods may come from.
16. [Original] Now I'd like to go on to discuss the *family food rules* in your family, I'm interested to hear when certain people in the family might control what others eat.
16. [Revised] Now I'd like to go on to discuss the *family food rules* in your family.
17. So far, we have talked about… [reiterate the relevant influences discussed so far], I wondered if you have any views about what might be the *main influence* on the family in making food choices.
18. Overall, I'd be interested to hear what you think we could do to help people make healthier food choices?

To finish off now, is there anything we have overlooked in our discussion?

Closing: Thank you so much for your time - that was all very helpful.

Appendix 2.B: Family Interview Discussion Guide (Full Version)

Family Interview Discussion Guide (Full Version)

INTRODUCTION

The researchers:

Introduce themselves by name and position, show ID,

State the research aims through the Letter of Introduction,

Explain the routine and expected time frame,

State importance of each participant's contribution,

Explain rights, confidentiality, anonymity of data, audio recording permissions,

Consent forms are signed, any concerns?

Families settle into a comfortable room (seated in a circle if possible) and are pre-prepared to hold the discussion minimising interruptions or distractions.

Background questions

Let's begin by going around the room one by one to get to know each other and find out how you all relate to each other. Could we start by stating our first names, age of the children and your position in the family (*e.g., mother, grandfather, or child*)?

TOPIC 1 – EATING OCCASIONS

Opening questions

1. [Original] First, let's talk through a typical **weekday** and the occasions that involve eating food. I'd be interested to hear about your typical eating occasions, such as meals, snacks, coffee breaks etc.?
    - Times of day, locations, together, apart?
    - [Revised] First, let's talk through your usual weekday, and the times of day that involve eating food. For example, when would you usually have meals, snacks or coffee breaks?
    - Times of day, locations, together, apart?

1. I wondered how this might be different on **weekends**. I'd like to hear about the occasions that involve eating food on the **weekend**.
    - Times of day, locations, together, apart, at movies or at sports?

TOPIC 2 – DISCUSSING FOOD

Key Questions

2. I wondered when your family members might talk about food?
    - Around the dinner table?
    - When preparing or planning meals for the week?
    - At the supermarket with children?
3. I'm really interested to hear **which** family members **talk most** with you about food? (e.g., describe some of the family influences on food decisions).
    - Children to parents?
    - Grandparents to parents?
4. I'd like to go on now to discuss where you get information from about healthy eating or food preparation?
    - Family members?
    - Friends?
    - Community members?
    - Health practitioners?
    - Newspapers?
    - Other media? TV, radio?
5. Of these, which do you think influence your family most?

TOPIC 3 – MEAL PLANNING AND PREPARATION

Key questions

6. Next, we would like to talk about meal planning.

   I'd be really interested to hear how your family plans meals

   - Through discussion?
   - One person takes responsibility?
   - Weekly menu?
   - Weekend baking in advance?

7. I wondered **who** is typically involved in **preparing** the food in your family?

   - Are there differences on weekdays? On weekends?
   - Do children help?
   - Are older people involved?
   - Mothers or fathers?

8. Next I'd like to discuss how the way you prepare food may have changed over time.

   - How do children help prepare food?
   - How might older people/elders be involved?

Half-way point acknowledgement: We are about half way through the discussion right now and you have raised some very important points. I think the discussion is going really well – how is everyone else going with it?

TOPIC 4 – FOOD PURCHASING

Key Questions

9. Next I'd like to discuss shopping for food… I'm really interested in how your family **decides** what food to purchase?

   - Who influences food purchasing decisions?
   - Do children ask for certain foods?

- Do parents decide what is best?
- Do grandparents have a say?
- Who decides for main meals at home?
- Who talks to the decision maker about food choices?
- Who decides for school lunches?
- Who decides for snack foods?
- Who decides for dinners out?
- What else? Location of shops? Cost of food and budget?

10. Can you tell us **who** typically purchases food for the whole family?
    - Who does the main food shopping, how often?
    - Are there differences on weekdays? On weekends? When out for dinner?

TOPIC 5 – FOOD TYPES

Key Questions

11. Also, I wondered if you had any thoughts about how the **types** of foods you eat may have **changed over time**.
    - As children grow older?
    - More fast foods?
    - Healthier foods?
    - Multi-cultural foods?
12. …I'm really interested in what you think has influenced these changes?
    - Children's tastes have developed?
    - Children purchase their own foods?

TOPIC 6 – MULTI-CULTURAL FOOD INFLUENCES

Key Questions

13. I'm really interested to hear about the **type** of foods that you eat most often.

- What multi-cultural food types?
- How frequently are these food types eaten?
- Who cooks these?
- Who enjoys these most?
- What foods are traditional to your family (e.g., recipes handed down the generations)?

14. I wondered if you had any thoughts about how new foods are introduced to your family's diet and where ideas for new foods may come from.
    - Grandparents to parents?
    - Children to parents?
    - Parents to children?
    - Advertising or other sources outside the family?

TOPIC 7 – FAMILY FOOD RULES

Transition Questions - Time 1 and Time 2

15. Now I'd like to go on to discuss the **family food rules** in your family.

I'm interested to hear when certain people in the family might control what others eat.

- Are sweet foods limited to certain times for children?
- Are there restrictions for religious or cultural practice reasons?
- Is anyone on a special diet for health or preference reasons (e.g., diabetes, vegan)

TOPIC 8 - OVERVIEW

Closing questions - Time 1 and Time 2

16. So far, we have talked about… [reiterate the relevant influences discussed so far], I wondered if you have any views about what might be the **main** influence on the family in making food choices…

    - Of those discussed so far, what is the most **important** in your family?
    - Certain family members? Position in the family?
    - Traditions?
    - Health concerns?
    - External constraints (e.g., cost)

17. **Overall**, I'd be interested to hear what you think we could do to help people make healthier food choices?

    - Better understanding of health risks?
    - Targeted advertising?
    - Culturally applicable?
    - Age appropriate?
    - Health promotion activities?

To finish off now, is there anything we have overlooked in our discussion?

When leaving - close with:

Thank you so much for your time – that was very helpful.

Appendix 2.C: Information Sheet

Carlene Wilson
CCSA Chair Cancer Prevention
Flinders Prevention, Promotion, and Primary Health Care, Public Health

Flinders University

GPO Box 2100
Adelaide SA 5001
Tel: 08 7221 8473
Fax: 08 8291 4268
E:carlene.wilson@flinders.edu.au

CRICOS Provider No. 00114A

INFORMATION SHEET

A Family Interview Study Investigating the Cross-Cultural and Intergenerational Transmission of Eating Behaviour in Families

You are invited to take part in a 2-part family interview study which is supplementary to a larger research project on food choices and eating behaviours in families from different cultural backgrounds. By holding a discussion with individual families, this project aims to improve our understanding of the role of the family, across several generations, on food choices and eating behaviour. In addition, it will identify the family members that are involved in decision-making about the family's diet. This would allow health messages about food to be more effective by targeting family members with the most influence on food choices.

You have received this Information Sheet because we thought your family may be able to help us further with our research interests. However, you do not have to participate in this additional study. If your family chooses to participate you will be asked to take part in a discussion about food choices and eating habits within your family, which will be conducted in the family home. Participation will take approximately one hour on two separate occasions, 6 months apart.

If you do not wish to participate in this supplementary study, you do not need to do anything further and your participation in the larger study will not be compromised in any way. If you would like to take part, please confirm your involvement by contacting the researchers Kate Rhodes (phone: 7221 8447, or email: kate.rhodes@flinders.edu.au), or Donna Hughes (phone: 7221 8436 or email: donna.hughes@flinders.edu.au). Please indicate if an interpreter will be required. Be assured that any information provided will be treated in the strictest confidence and participants will not be individually identifiable in the resulting reports or other publications. You are free to withdraw from the project at any time or to decline to answer particular questions.

The research is funded by the Australian Research Council and family members will be reimbursed for their time with supermarket vouchers to the value $50 per family - on both occasions. The results of this study will be used in further grant applications and may be published. If you have any questions about this project please contact the researchers supervisor: Carlene Wilson (phone: 8291 4345, or email carlene.wilson@flinders.edu.au) or the researchers Kate Rhodes and Donna Hughes as above.

This research project has been approved by the Flinders University Social and Behavioural Research Ethics Committee (Project Number 5572). For more information regarding ethical approval of the project the Executive Officer of the Committee can be contacted by telephone on 8201 3116, by fax on 8201 2035 or by email human.researchethics@flinders.edu.au

Appendix 2.D: Letter of Introduction

Carlene Wilson
CCSA Chair Cancer Prevention
Flinders Prevention, Promotion, and Primary Health Care, Public Health

Flinders University

GPO Box 2100
Adelaide SA 5001
Tel: 08 7221 8473
Fax: 08 8291 4268
E: carlene.wilson@flinders.edu.au

CRICOS Provider No. 00114A

LETTER OF INTRODUCTION

Dear Sir/ Madam,

This letter is to introduce Ms Kate Rhodes who is a PhD student in the School of Medicine at Flinders University. She is undertaking research leading to a thesis and publications on the subject of food choices and eating behaviours in families from various cultural backgrounds. She will produce her student card, which carries a photograph, as proof of identity.

Kate would be most grateful if your family would volunteer to assist in this two-part project, by granting two interviews which cover certain aspects of this topic. No more than one hour on two occasions (6 months apart) would be required.

Be assured that any information provided will be treated in the strictest confidence and none of the participants will be individually identifiable in the resulting reports or other publications. Anyone in your family is, of course, free to discontinue participation at any time or to decline to answer particular questions.

Since she intends to make a tape recording of the interview, she will seek your consent, on the attached form, to record the interview. De-identified data from this may be used in the preparation of the thesis or other publications; this ensures that your name or identity is not revealed. It may be necessary to make the recording available to secretarial assistants for transcription, in which case you may be assured that such persons will be advised of the requirement that your name or identity not be revealed and that the confidentiality of the material is respected and maintained.

If you have any questions about this project please contact the researcher's supervisor: Carlene Wilson (phone: 8291 4345, or email carlene.wilson@flinders.edu.au) or the researcher Kate Rhodes (phone: 7221 8447, or email kate.rhodes@flinders.edu.au).

Thank you for your attention and assistance.

Yours sincerely,

Prof Carlene Wilson
CCSA Chair in Cancer Prevention (Behavioural Research)
School of Medicine

This research project has been approved by the Flinders University Social and Behavioural Research Ethics Committee (Project Number 5572). For more information regarding ethical approval of the project the Executive Officer of the Committee can be contacted by telephone on 8201 3116, by fax on 8201 2035 or by email human.researchethics@flinders.edu.au..

Appendix 2.E: Consent Form

CONSENT FORM FOR PARTICIPATION IN RESEARCH

(by interview)

Families and Eating Behaviour: Interview Study

I .....

being over the age of 18 years hereby consent to participate as requested in the Letter of Introduction and Information Sheet for the research project on "A Cross-Cultural Investigation of the Intergenerational Transmission of Eating Behaviour: A Family Interview Study"

1. I have read the information provided.
2. Details of procedures and any risks have been explained to my satisfaction.
3. I agree to audio recording of my information and participation.
4. I am aware that I should retain a copy of the Information Sheet and Consent Form for future reference.
5. I understand that:
   - I may not directly benefit from taking part in this research.
   - I am free to withdraw from the project at any time and am free to decline to answer particular questions.
   - While the information gained in this study will be published as explained, I will not be identified, and individual information will remain confidential.
   - I may ask that the recording/observation be stopped at any time, and that I may withdraw at any time from the session or the research without disadvantage.
6. I have had the opportunity to discuss taking part in this research with a family member or friend.

Participant's signature.....Date.....

I certify that I have explained the study to the volunteer and consider that she/he understands what is involved and freely consents to participation.

Researcher's name.....

Researcher's signature.....Date.....

NB: Two signed copies should be obtained.

PARENTAL CONSENT FORM FOR CHILD PARTICIPATION IN RESEARCH

CONSENT FORM FOR PARTICIPATION IN RESEARCH

(by interview)

Families and Eating Behaviour: Interview Study

I .....

being over the age of 18 years hereby consent to my child .....

participating, as requested, in the in the Letter of Introduction and Information Sheet for the research project on "A Cross-Cultural Investigation of the Intergenerational Transmission of Eating Behaviour: A Family Interview Study".

1. I have read the information provided.
2. Details of procedures and any risks have been explained to my satisfaction.
3. I agree to audio recording of my child's information and participation.
4. I am aware that I should retain a copy of the Information Sheet and Consent Form for future reference.
5. I understand that:
   - My child may not directly benefit from taking part in this research.
   - My child is free to withdraw from the project at any time and is free to decline to answer particular questions.
   - While the information gained in this study will be published as explained, my child will not be identified, and individual information will remain confidential.
   - My child may ask that the recording/observation be stopped at any time, and he/she may withdraw at any time from the session or the research without disadvantage.

Participant's signature.....Date.....

I certify that I have explained the study to the volunteer and consider that she/he understands what is involved and freely consents to participation.

Researcher's name.....

Researcher's signature.....Date.....

NB: Two signed copies should be obtained.

CHAPTER 3 APPENDICES

Appendix 3.A: Adult Questionnaire

‘Families, Food & Eating’ Research Study

Adult Questionnaire

For _____ to complete

This questionnaire will ask you questions about yourself, your family, and your exercise and eating habits. It also asks a few questions on your perceived risk of disease. Please answer the questions as honestly as possible **within the next two weeks**. If you have any queries, please do not hesitate to contact one of us:

Donna Hughes (ph: 7221 8436)
Kate Rhodes (ph: 7221 8447)

When you have finished the questionnaire, please detach this front page and post the rest of the questionnaire back to us in the reply-paid envelope provided.

Flinders UNIVERSITY

Cancer Council SA

ID:

Section 1
Demographics

First of all, we are going to ask you some questions about yourself and your cultural background.

1. In which country were you born?

☐$_1$ Australia (Go to Question 3)
☐$_2$ Vietnam
☐$_3$ Italy
☐$_4$ Other country (please specify)_____

2. How long have you lived in Australia? *(if you were born outside Australia)* _____ years

3. What suburb do you live in?_____ Post code _____

4. In which country was your mother born?

☐$_1$ Australia
☐$_2$ Vietnam
☐$_3$ Italy
☐$_4$ Other country (please specify)
_____

5. In which country was your father born?

☐$_1$ Australia
☐$_2$ Vietnam
☐$_3$ Italy
☐$_4$ Other country (please specify)
_____

ID:

6. What is the main language that you speak at home?

☐$_1$ English
☐$_2$ Vietnamese
☐$_3$ Italian
☐$_4$ Other (please specify)_____

7. Do you identify your family as

☐$_1$ Australian
☐$_2$ Vietnamese-Australian
☐$_3$ Italian-Australian
☐$_4$ Other (please specify)_____

Section 2
Lifestyle

Now, in this section, we will ask you some questions about your lifestyle.

Many of these questions ask you to think about the things that you did and ate during the last 7 days. Try to respond as accurately as possible, even if this week was not a normal or typical week for you.

1. Do you smoke tobacco, including cigarettes, a pipe or cigars?

☐$_1$ Yes, I currently smoke cigarettes, a pipe, or cigars
☐$_2$ No, I used to smoke
☐$_3$ No, I have never smoked (or I have smoked less than 100 cigarettes, pipes or cigars in my lifetime)

2. During the last 7 days, on how many days did you do vigorous physical activities, such as heavy lifting, digging in the garden, climbing upstairs, fast bicycling, aerobics, or running? Think about only those physical activities that you did for at least 10 minutes at a time.

☐$_0$ No vigorous physical activity (go to question 3)
☐$_1$ _____days per week (go to question 2a)

2a. On average, how much time on one of those days did you usually spend doing vigorous physical activities? _____ hours per day _____ minutes per day

3. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate physical activities such as carrying light loads, sweeping, washing windows, raking in the garden, or bicycling at a regular pace? Please do not include walking.

ID:

☐$_0$ No moderate physical activity (go to question 4)

☐$_1$ _____ days per week (go to question 3a)

3a. On average, how much time on one of those days did you usually spend doing moderate physical activities? _____ hours per day _____ minutes per day

4. During the last 7 days, on how many days did you walk for at least 10 minutes at a time from place to place?

☐$_0$ No periods of walking for 10 minutes at a time (go to question 5)

☐$_1$ _____ days per week (go to question 4a)**4a. On average, how much time on one of those days did you usually spend walking?**

_____ hours per day _____ minutes per day

5. During the last week, on a weekday (Mon-Fri), how much time per day did you usually spend sitting?

_____ hours _____ minutes per day

6. During the last week, on a weekend day (Sat-Sun), how much time per day did you usually spend sitting?

_____ hours _____ minutes per day

7. Please indicate how often you participated in physical activity as a family during the last 7 days (e.g., jogging, walking, team sports)?

_____ times

5a. On average, how long did you spend on each activity?

_____ hours _____ minutes

8. In the past week, how many serves of fruit did you eat each day?

1 serve of fruit is equivalent to 1 medium sized piece of fruit (e.g., apple, orange, mango, mandarin, banana, pear, peach), 2 small pieces of fruit (e.g., apricots, kiwifruit, plums, figs), 8 strawberries, or ½ cup of fruit juice.

_____ serves of fruit per day

9. In the past week, how many serves of vegetables did you eat each day?

1 serve of vegetables is equivalent to 1 medium potato, or ½ a medium sweet potato, or ½ cup of dark green leafy vegetables (e.g., cabbage, spinach, broccoli, or brussel sprouts), or 1 cup of other

vegetables (e.g., lettuce, beans, lentils, peas, zucchini, cucumber, mushrooms).

_____ serves of vegetables per day

10. **In the past week, how many times did you eat meals that were bought from fast food outlets** such as McDonalds, Hungry Jacks, Pizza Hut, KFC, Red Rooster, hamburger, pizza or fish and chip shops?
_____ times last week

11. **In the past week, how many times did you eat snacks** such as a chocolate bar, a piece of cake, a packet of chips/twisties/corn chips, ice cream, 3-4 sweet biscuits?
_____ times last week

12. **On average, in the past week, on how many days did you drink alcohol?**

_____ days

13. **On average, how many standard drinks did you have on days that you drank alcohol?**

(please refer to standard drinks guide below)

_____ standard drinks per day that I drank alcohol

SPARKLING WINE	WINE	LIGHT BEER	REGULAR BEER	FORTIFIED WINE	SPIRITS
100 mL	100 mL	425 mL	285 mL	60 mL	30 mL
13% alc/vol	13% alc/vol	2.7% alc/vol	4.9% alc/vol	20% alc/vol	40% alc/vol

(Information from Drug & Alcohol Services SA)

ID:

Section 3
Food Attitudes

Food is a more important aspect of life for some people than it is for others. In this section, we are interested in your attitudes towards food. Please read each statement below and circle the number that best represents how much you agree or disagree with it. Remember, there are no right or wrong answers, just your opinion.

Here is an example:

	Strongly Disagree		Disagree		Agree		Strongly Agree
I think that food gives us energy	1	2	3	4	5	6	⑦

In this example, you can see that I have circled number 7. This means that, for me, I strongly agree with the statement.

Now, complete the questionnaire below by indicating your opinions.

		Strongly Disagree		Disagree		Agree		Strongly Agree
1	I have fond memories of family food occasions	1	2	3	4	5	6	7
2	I rarely think about the long-term effects of my diet on health	1	2	3	4	5	6	7
3	I am concerned about being overweight	1	2	3	4	5	6	7
4	I feel guilty when I overeat	1	2	3	4	5	6	7
5	Diet can have a big effect on heart disease	1	2	3	4	5	6	7
6	I eat low-fat food on a regular basis	1	2	3	4	5	6	7
7	Diet can have a big effect on obesity	1	2	3	4	5	6	7
8	Taste is more important to me than nutrition	1	2	3	4	5	6	7
9	I am a healthy eater	1	2	3	4	5	6	7
10	I am currently on a diet	1	2	3	4	5	6	7
11	I eat fast food on a regular basis	1	2	3	4	5	6	7
12	I consciously hold back at meal time, so as not to gain weight	1	2	3	4	5	6	7
		Strongly Disagree		Disagree		Agree		Strongly Agree

ID:

13	Diet can have a big effect on good health	1	2	3	4	5	6	7
14	Money spent on food is well spent	1	2	3	4	5	6	7
15	Diet can have a big effect on cancer	1	2	3	4	5	6	7
16	Enjoying food is one of the most important things in my life	1	2	3	4	5	6	7
17	I think about food in a positive way	1	2	3	4	5	6	7
18	I think natural, organic foods are better for you than commercially grown/processed foods	1	2	3	4	5	6	7
19	In my opinion, my thighs are too fat	1	2	3	4	5	6	7
20	I control my caloric intake	1	2	3	4	5	6	7
21	I think natural, organic foods taste better than commercially grown/processed foods	1	2	3	4	5	6	7

Section 4

Disease Risk

We are all becoming more aware that some chronic diseases (such as heart disease, diabetes and cancer) can have a link to aspects of our lifestyle, as well as other factors. We are interested in your views about your own risk of developing one of these diseases. For some people, this is a topic that they think about a lot, but for others, it may not seem as important to them at this time. Please indicate your opinions by circling the best response on the scale next to each statement below.

		Not at all likely			Moderately likely			Extremely likely
1	In the next 6 months, how likely are you to talk to your <u>doctor</u> about your health risk for lifestyle-related chronic diseases (e.g., colorectal cancer (also known as bowel or colon cancer), breast cancer, heart disease OR diabetes)?	1	2	3	4	5	6	7
2	In the next 6 months, how likely are you to talk to your <u>family</u> about your health risk for lifestyle-related chronic diseases (e.g., colorectal cancer (also known as bowel or colon cancer), breast cancer, heart disease OR diabetes)?	1	2	3	4	5	6	7

ID:

Please now think about the diseases listed below. How likely do you think it is that you could develop one or more of these diseases in your lifetime? Indicate your answer by circling the best response. If you don't know or have no opinion about this, circle DK.

		Not at all likely			Moderately likely			Extremely likely	I don't know	Not Applicable
3	Colorectal cancer	1	2	3	4	5	6	7	DK	NA
4	Breast cancer	1	2	3	4	5	6	7	DK	NA
5	Heart disease	1	2	3	4	5	6	7	DK	NA
6	Diabetes	1	2	3	4	5	6	7	DK	NA

Note. If you have already received a diagnosis for one of these diseases, please select NA

Now, we'd like you to think about lifestyle factors and how they might contribute to the risk of disease. Indicate your opinions below by circling the response that is best for you.

		Not at all		A little		A fair bit		A great deal
7	How much do you think <u>eating habits</u> contribute to the risk of lifestyle-related diseases such as heart disease, diabetes, breast cancer, and colorectal cancer?	1	2	3	4	5	6	7
8	How much do you think <u>alcohol consumption</u> contributes to the risk of lifestyle-related diseases such as heart disease, diabetes, breast cancer, and colorectal cancer?	1	2	3	4	5	6	7
9	How much do you think <u>physical inactivity</u> contributes to the risk of lifestyle-related diseases such as heart disease, diabetes, breast cancer, and colorectal cancer?	1	2	3	4	5	6	7
10	How much do you think <u>genetic factors</u> contribute to the risk of lifestyle-related diseases such as heart disease, diabetes, breast cancer, and colorectal cancer?	1	2	3	4	5	6	7

ID:

Section 5

Demographics

In our research, to make it more comprehensive, we need to be able to describe the people who have participated. Please take a moment to consider the questions in this section. There are no right or wrong answers. If the question has a blank, please fill in the blank. If the question has a list of choices, please mark the box with the response that best reflects your answer.

1. Age: _____ (years)

2. Gender: ☐1 *Male* ☐2 *Female* (tick one)

3. Height:_____ (cm)

4. Weight:_____ (kg) (Pregnant women should indicate weight prior to pregnancy)

5. How many people live with you at your current residence? _____

6. How many children do you have? _____

7. Your current marital status is:

☐1 Single
☐2 Married
☐3 Living as married
☐4 Separated
☐5 Divorced
☐6 Widowed

8. What was the **annual income** received by everyone in your household **BEFORE TAX** in the last financial year?

☐1 0 - $20,000
☐2 $20,001 - $35,000
☐3 $35,001 - $50,000
☐4 $50,001 - $75,000
☐5 $75,001 - $100,000
☐6 $100,001 - $125,000
☐7 $125,001 - $150,000
☐8 $150,001 - $200,000
☐9 $200,001 or more

9. What is the **highest level of formal education** that you have completed? (Tick one box)

☐1 University qualification
☐2 TAFE or technical qualification
☐3 Secondary School
☐4 Primary School

Thank you very much for your time!

Please detach the front page now and return the completed questionnaire to us in the reply-paid envelope provided.

Appendix 3.B: Child Questionnaire

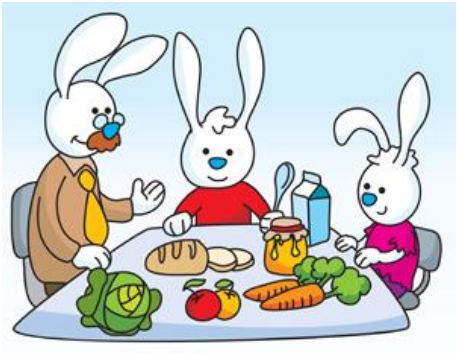
‘Families, Food & Eating’ Research Study

Child Questionnaire

For participants aged 8 – 17 years

For _____ to complete

This questionnaire will ask you questions about yourself, your family, and your exercise and eating habits. Please answer the questions as honestly as you can. If you need any help, please ask an adult nearby.



ID:

Date:

Section 1
Demographics

First of all, we are going to ask you some questions about yourself and your cultural background.

8. In which country were you born?

☐$_1$ Australia (Go to Question 3)
☐$_2$ Vietnam
☐$_3$ Italy
☐$_4$ Other country (please specify)_____

9. How long have you lived in Australia? *(if you were born outside Australia)* _____ years

10. What suburb do you live in?_____ Post code _____

11. In which country was your mother born?

☐$_1$ Australia
☐$_2$ Vietnam
☐$_3$ Italy
☐$_4$ Other country (please specify)
_____

12. In which country was your father born?

☐$_1$ Australia
☐$_2$ Vietnam
☐$_3$ Italy
☐$_4$ Other country (please specify)
_____

13. What is the main language that you speak at home?

☐$_1$ English
☐$_2$ Vietnamese
☐$_3$ Italian
☐$_4$ Other (please specify)_____

14. Do you identify your family as

☐$_1$ Australian
☐$_2$ Vietnamese-Australian
☐$_3$ Italian-Australian
☐$_4$ Other (please specify)_____

ID:

Section 2
Lifestyle

Now, in this section, we will ask you some questions about your lifestyle.

Many of these questions ask you to think about the things that you did and ate during the last 7 days. Try to respond as accurately as possible, even if this week was not a normal or typical week for you.

Physical Activity

14. During the last 7 days, on how many days did you do vigorous physical activities, such as heavy lifting, digging in the garden, climbing upstairs, fast bicycling, aerobics, or running? Think about only those physical activities that you did for at least 10 minutes at a time.

☐$_0$ No vigorous physical activity (go to question 2)

☐$_1$ _____days per week (go to question 1a)

1a. On average, how much time on one of those days did you usually spend doing vigorous physical activities? _____ hours per day _____ minutes per day

15. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate physical activities such as carrying light loads, sweeping, washing windows, raking in the garden, or bicycling at a regular pace? Please do not include walking.

☐$_0$ No moderate physical activity (go to question 3)

☐$_1$ _____ days per week (go to question 2a)

2a. On average, how much time on one of those days did you usually spend doing moderate physical activities? _____ hours per day _____ minutes per day

16. During the last 7 days, on how many days did you walk for at least 10 minutes at a time from place to place?

☐$_0$ No periods of walking for 10 minutes at a time (go to question 4)

☐$_1$ _____ days per week (go to question 3a)

3a. On average, how much time on one of those days did you usually spend walking?
_____ hours per day _____ minutes per day

ID:

17. Please indicate how often you participated in physical activity with your family during the last 7 days (e.g., jogging, walking, team sports).

_____ times

4a. On average, how long did you spend on each activity? _____ hours _____ mins

Seated Activity

18. During the last week, on a weekday (Mon-Fri), how much time per day did you usually spend sitting? _____ hours _____ minutes per day

19. During the last week, on a weekend day (Sat-Sun), how much time per day did you usually spend sitting? _____ hours _____ minutes per day

Below are some questions on ***sedentary or seated behaviour*** such as computer/video game use, watching television, sitting listening to music, sitting and talking on the phone, homework/studying, reading for recreation etc.

20. On the average day, about how many hours do you do the following when you are not at school? (if less than 1 hour, please estimate how many minutes you spend)

	Mon-Fri	**Sat-Sun**
Homework	_____hours	_____hours
Watch TV/videos/DVDs	_____hours	_____hours
Use the internet / play computer games *(do not include computer use for homework)*	_____hours	_____hours
Use Facebook, Twitter, or Myspace,	_____hours	_____hours
Use other social networking sites (please specify.....)	_____hours	_____hours
Sit and listen to music	_____hours	_____hours
Sit and read for recreation	_____hours	_____hours
Sit and talk on the phone	_____hours	_____hours
Sit and do another activity (please specify.....)	_____hours	_____hours

Eating

ID:

We'd now like to ask you some questions about food that you might eat in a typical week. Thinking over the last week, try to answer as accurately as you can, even if this week was not a normal or typical week for you.

21. In the past week, how many serves of fruit did you eat each day?

1 serve of fruit is equivalent to 1 medium sized piece of fruit (e.g., apple, orange, mango, mandarin, banana, pear, peach), 2 small pieces of fruit (e.g., apricots, kiwifruit, plums, figs), 8 strawberries, or ½ cup of fruit juice.

_____ serves of fruit per day

22. In the past week, how many serves of vegetables did you eat each day?

1 serve of vegetables is equivalent to 1 medium potato, or ½ a medium sweet potato, or ½ cup of dark green leafy vegetables (e.g., cabbage, spinach, broccoli, or brussel sprouts), or 1 cup of other vegetables (e.g., lettuce, beans, lentils, peas, zucchini, cucumber, mushrooms).

_____ serves of vegetables per day

23. In the past week, how many times did you eat meals that were bought from fast food outlets such as McDonalds, Hungry Jacks, Pizza Hut, KFC, Red Rooster, hamburger, pizza or fish and chip shops?
_____ times last week

24. In the past week, how many times did you eat snacks such as a chocolate bar, a piece of cake, a packet of chips/twisties/corn chips, ice cream, 3-4 sweet biscuits?

Section 4

Food Attitudes

Food is a more important aspect of life for some people than it is for others. In this section, we are interested in your attitudes towards food. Please read each statement below and circle the number that best represents how much you agree or disagree with it. Remember, there are no right or wrong answers, just your opinion.

Here is an example:

	Strongly Disagree	Disagree	Agree	Strongly Agree

ID:

	I think that food gives us energy	1	2	3	4	5	6	⑦

In this example, you can see that I have circled number 7. This means that, for me, I strongly agree with the statement.

Now, complete the questionnaire below by indicating your opinions.

		Strongly Disagree		**Disagree**		**Agree**		**Strongly Agree**
1	I have fond memories of family food occasions	1	2	3	4	5	6	7
2	I rarely think about the long-term effects of my diet on health	1	2	3	4	5	6	7
3	Diet can have a big effect on heart disease	1	2	3	4	5	6	7
4	I eat low-fat food on a regular basis	1	2	3	4	5	6	7
5	Diet can have a big effect on obesity	1	2	3	4	5	6	7
6	Taste is more important to me than nutrition	1	2	3	4	5	6	7
7	I am a healthy eater	1	2	3	4	5	6	7
8	I eat fast food on a regular basis	1	2	3	4	5	6	7
9	Diet can have a big effect on good health	1	2	3	4	5	6	7
10	Money spent on food is well spent	1	2	3	4	5	6	7
11	Diet can have a big effect on cancer	1	2	3	4	5	6	7
12	Enjoying food is one of the most important things in my life	1	2	3	4	5	6	7
		Strongly Disagree		**Disagree**		**Agree**		**Strongly Agree**
13	I think about food in a positive way	1	2	3	4	5	6	7
14	I think natural, organic foods are better for you than commercially grown/processed foods	1	2	3	4	5	6	7
15	I control my caloric intake	1	2	3	4	5	6	7
16	I think natural, organic foods taste better than commercially grown/processed foods	1	2	3	4	5	6	7

ID:

Section 5
Demographics

In our research, to make it more comprehensive, we need to be able to describe the people who have participated. If the question has a blank, please fill in the blank. If the question has a list of choices, please mark the box with the response that best reflects your answer.

10. Age: _____ (years)

11. Gender: ☐$_1$ *Male* ☐$_2$ *Female* (tick one)

12. Height: _____ (cm)

13. Weight: _____ (kg)

14. How many people live with you at your house? _____

15. How many brothers or sisters do you have? _____

16. Which television stations do you watch most often?

☐$_1$ ABC/SBS
☐$_2$ Commercial stations (e.g., Channels 7, 9 & 10)
☐$_3$ Subscription television (e.g., Foxtel, Austar)
☐$_4$ I do not watch television

Thank you very much for your time!

Please detach the front page now and return the completed questionnaire to us in the reply-paid envelope provided.

Appendix 3.C: Interclass Correlations for Healthy and Unhealthy Food Consumption Outcome Variables

Table C.1.

Fruit Consumption. Shown are the interclass correlation (ICC) results. These indicate the variability in fruit consumption associated with differences between families as a percentage. Wald Z shows results are non-significant (GLiMM Poisson output).

Random Effects Subject	Model	Within group variance (Residual Estimate)[a]	Between group variance (Intercept Estimate)[b]	Standard Error	95% CI Lower	95% CI Upper	Wald Z	ICC [c]	Variance %	*p*
N = 50 families	Model 1	1.00	0.122	0.04	0.06	0.24	2.92	0.036	3.6	.16
	Model 2	1.00	0.111	0.04	0.06	0.22	2.78	0.033	3.3	.01
	Model 3	1.00	0.116	0.04	0.06	0.23	2.83	0.034	3.4	.04

Note. Calculation: ICC = b/(a + b) Tabachnick & Fidell (2012) p.826-7. However, when the residual variance is 1.00 the scale factor can be used to calculate an ICC to describe the proportion of variance between units (families) relative to the total variance. The logistic distribution with a scale factor 1.00 is π^2 /3 = 3.29, therefore [a] = 3.29 and the equation used was ICC [c] = b/(a + b) Heck et al. (2012) p. 157.

Table C.2

Vegetable consumption. Shown are the interclass correlation (ICC) results. These indicate the variability in vegetable consumption associated with differences between families as a percentage (GLiMM Negative binomial output). The Wald Z shows the results are non-significant.

Random Effects Subject	Model	Within group variance (Residual Estimate)[a]	Between group variance (Intercept Estimate) [b]	Standard Error	95% CI Lower Upper		Wald Z	ICC [c]	Variance %	*p*
N = 50 families	Model 1	1.00	0.000							
	Model 2	1.00	0.000							
	Model 3	1.00	0.009	0.018	0.000	0.41	.521	0.003	0.3	.60

Note. ICC calculation is ICC = b/(a + b) Tabachnick & Fidell (2012) p.826-7]. However, when the residual variance is 1.00 the scale factor can be used to calculate an ICC to describe the proportion of variance between units (families) relative to the total variance. The logistic distribution with a scale factor 1.00 is π^2 /3 = 3.29, therefore [a] = 3.29 and the equation is ICC [c] = b/(a + b) Heck et al. (2012) p. 157.

Table C.3

Snack Consumption. Shown are the interclass correlation (ICC) results. These indicate the variability in snack consumption associated with differences between families as a percentage. The ICCs were non-significant (GLiMM Negative binomial).

Random Effects Subject	Model	Within group variance (Residual Estimate)[a]	Between group variance (Intercept Estimate) [b]	Standard Error	95% CI Lower	95% CI Upper	Wald Z	ICC [c]	Variance %	*p*
N = 50 families	Model 1	1.00	0.037	0.03	0.01	0.15	1.43	0.011	1.1	.16
	Model 2	1.00	0.040	0.03	0.01	0.14	1.52	0.012	1.2	.13
	Model 3	1.00	0.044	0.03	0.01	0.15	1.55	0.013	4.2	.12

Note. [c] ICC calculation is ICC = b/(a + b) Tabachnick & Fidell (2012) p.826-7. However, when the residual variance is 1.00 the scale factor can be used to calculate an ICC to describe the proportion of variance between units (families) relative to the total variance. The logistic distribution with a scale factor 1.00 is π^2 /3 = 3.29, therefore [a] = 3.29 and the equation is ICC [c] = b/(a + b) Heck et al. (2012) p. 157.

Table C.4

Fast Food Consumption. Shown are the interclass correlation (ICC) results for fast food consumption (GLiMM Negative binomial). The Wald Z show the results are non-significant.

Random Effects Subject	Model	Within group variance (Residual Estimate)[a]	Between group variance (Intercept Estimate) [b]	Standard Error	95% CI Lower	95% CI Upper	Wald Z	ICC [c]	Variance %	*p*
N = 50 families	Model 1	1.00	0.313	0.14	0.13	0.74	2.28	0.087	8.7	.02
	Model 2	1.00	0.297	0.14	0.12	0.73	2.20	0.083	8.3	.03
	Model 3	1.00	0.296	0.13	0.12	0.72	2.20	0.083	8.3	.03

Note. [c] ICC calculation is ICC = b/(a + b) Tabachnick & Fidell (2012) p.826-7 The ICC indicates the variability in fast food consumption associated with differences between families (i.e., variance as a percentage). However, when the residual variance is 1.00 the scale factor can be used to calculate an ICC to describe the proportion of variance between units (families) relative to the total variance. The logistic distribution with a scale factor 1.00 is π^2 /3 = 3.29, therefore [a] = 3.29 and the equation is ICC [c] = b/(a + b) Heck et al. (2012) p. 157.

Appendix 3.D: Procedures Used for the Data Analysis of Individuals Nested Within Families

Steps Addressing the Analysis of Individuals Nested Within Family Data

The quantitative data throughout this thesis were individuals nested within families. In order to control for the effect of similarities between family members one could not treat all participants as independent individual participants. Although Appendix C showed tests of interclass correlations (ICCs) within-family interdependencies were greater in unhealthy food than healthy food outcome variables, all analyses were nevertheless conducted consistently controlling for family nesting. The following explains the procedures utilized throughout the present study.

Data analysis procedures. Data were aggregated to nest the 216 family members within 50 relevant families to test non-parametric correlations. In addition, to effectively predict variation in consumption based on our psychological variables of interest, various Generalized Linear Mixed Models (GLiMM) analyses were selected according to a statistical decision flowchart outlined in Elhai, Calhoun and Ford (2008) (see Figure D.1).

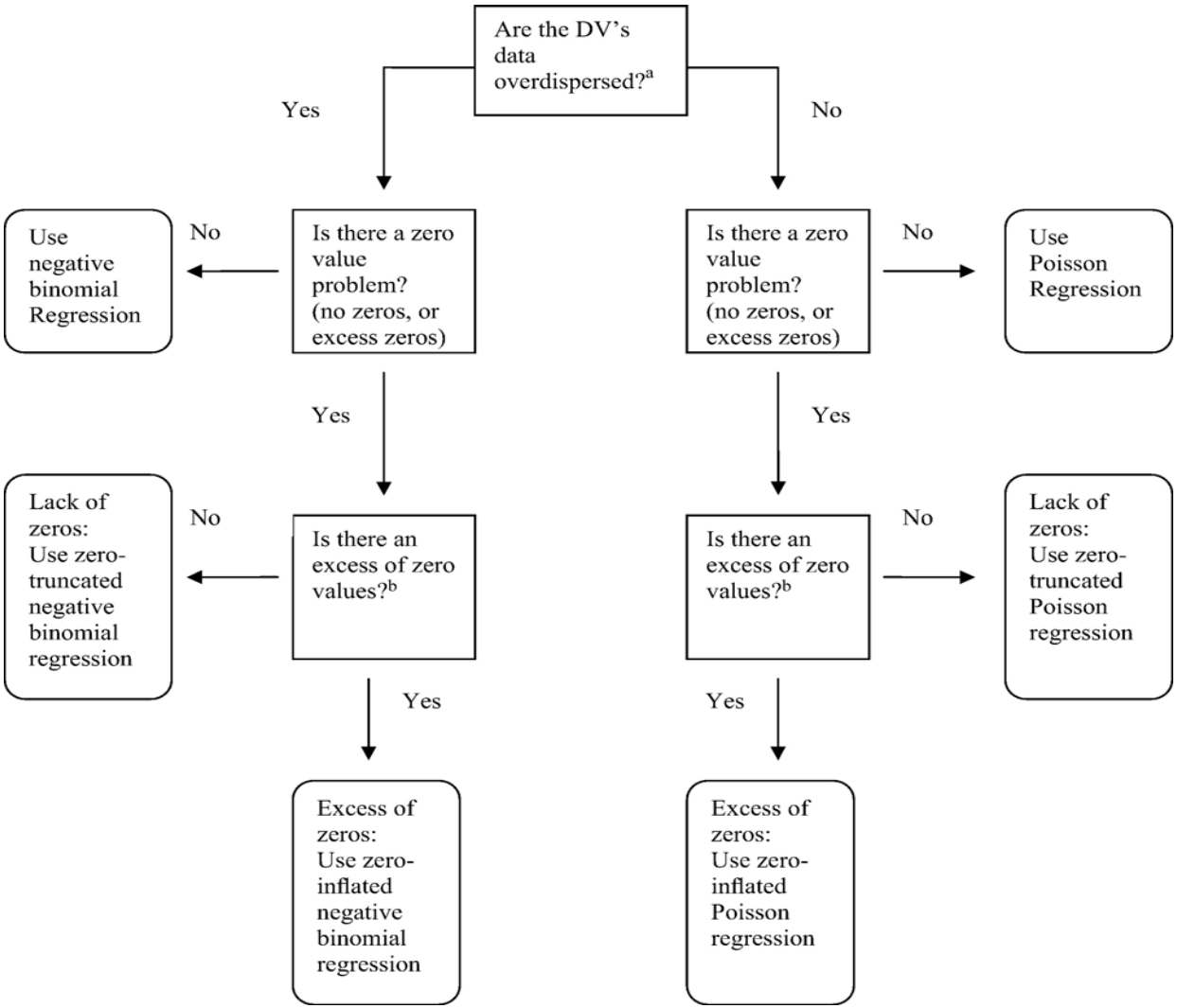


Figure D.1. Generalized Linear Mixed Models 'Decision Flow Chart' for selecting a count regression model. Assuming a) a non-negative integer, count dependent variable, and b) predictor variables that are continuously scaled, binary coded, or a mixture. Note. DV = dependent variable. a = Testable with a likelihood ratio test. b = Testable with a Vuong test for non-nested data. Flowchart adapted from (Elhai et al., 2008), Statistical procedures for analyzing mental health services data. Psychiatry Research, 160, 129 - 136. doi: 10.1016/j.psychres.2007.07.003.

Generalized Linear Models (GLiM) with a Generalized Estimating Equation (GEE) adjustment were used for tests of logistic regression; the GEE adjustment controlled for family nesting on dichotomized outcome variables (Hubbard et al., 2010). Both GLiMM and GLiM with GEE techniques controlled for the nesting effect of families; GLiMM's also successfully analysed skewed data, required no variable transformations, and enabled the results to retain their original value (e.g., counts of fruit in serves per day). Retaining the original data values facilitates the more meaningful interpretation of results and preserves measurement of any incremental changes over time for later comparison in any follow up studies (Elhai et al., 2008; Skov, Deddens, Petersen, & Endahl, 1998). Moreover, other problems were avoided, for example, violating assumptions of normality by the use of "classical" statistics, producing impossible negative integers in the results, or reducing continuous variables to unsubstantiated categories which had the potential to vary results depending on choice of cut-off points. Finally, Bonferonni corrections were included in all GLiMM and GEE modeling in order to maintain an accurate Type 1 error rate (Atkins & Gallop, 2007; Gardner, Mulvey, & Shaw, 1995; Heck et al., 2012).

GLiM with GEE was used for logistic regression in the present study to determine whether or not NHMRC guidelines were met for fruit and vegetable ("healthy food") consumption. This procedure process results as odds ratios that were able to be interpreted as increasing or decreasing with the addition of predictors and interactions. GLiMMs were the best choice for analysis that examined differences between generations and gender, or interactions between various predictors, without dichotomization (Rationale for the GLIMM procedure followed is provided in the footnote) To examine the differences in fruit, vegetable, snack and fast food consumption in this thesis, three different GLiMM procedures were used because they have been reported to provide the soundest analyses for asymmetrically distributed low count outcome data, that also produce the greatest statistical

power while preserving an accurate Type 1 error rate (Coxe et al., 2009). The most appropriate count regression model within the family of GLiMM analyses was selected according to how each count outcome variable met the required assumptions of normality (Elhai et al., 2008) (see statistical decision flowchart in Figure D.1).

Firstly, a Poisson regression model was used for tests on fruit consumption because it was the only outcome variable that the assumptions for Poisson were met. Poisson requires that the following assumptions are met: no negative scores can be present in the data; scores must be entered as whole integers, therefore SPSS rounded up any fractions of scores equal to or higher than 0.5, or rounded down any scores equal to or below 0.49; dependent variables must be recorded within a set time frame; and the time frame must be consistent for all participants (Elhai et al., 2008). The final Poisson assumption was also met; that the variance did not exceed the mean (if violated overdispersion is indicated). Secondly, for vegetables and snacks consumption a negative binomial regression was used as it accommodated overdispersed data. Thirdly, for fast food consumption, the assumptions for a negative binomial model were not met because there were 56% (valid percent) scores of zero. To adjust for this it is recommended these data utilize a test of zero-inflated negative binomial regression, however, to date, SPSS 22 does not offer this function and a negative binomial was used as the best alternative available at the time of writing (Coxe et al., 2009; Elhai et al., 2008; Heck et al., 2012). The food attitude subscales DHOB and DHLA were normally distributed; however, all data were individuals nested within families which violated the assumptions for classical statistics. Therefore, food attitude subscales were tested using GLiMM or GLiM with GEE techniques that met the assumptions for each consumption outcome variable that was included in the model (i.e., GLiMM Poisson, negative binomial, zero inflated, or GLiM with GEE as applicable). If consumption outcome variables were omitted from the analysis, a GLiMM Poisson analysis was used on normally distributed food

attitude variables to account for nesting assumption violations that would have otherwise occurred if "classical"[62] statistics were used.

[62] "Classical" statistics refers to Analysis of Variance and tests of multiple regression that rely on the ordinary least-squares (OLS) criterion (Atkins & Gallop, 2007)

Appendix 3.E: Food consumption resemblance within three generations: Inter- and intra-generational correlations.

Table E.1

Spearman's Rho Correlations Showing Resemblances in Fruit Consumption Among Generational Family Roles.

	Child	Parent	Grand
Child	-	.307*	.085
n	48	45	42
Parent		-	.021
n		47	40
Grandparent			-
n			42

Note. Extended family members excluded.
*$p < 0.05$, **$p < 0.01$, (1-tailed)

Table E.2

Spearman's Rho Correlations Showing Resemblances in Vegetable Consumption Among Generational Family Roles.

	Child	Parent	Grand
Child	-	.436**	.103
n	47	44	42
Parent		-	.017
n		47	41
Grandparent			-
n			43

Note. Extended family members excluded.
*$p < 0.05$, **$p < 0.01$, (1-tailed)

Table E.3

Spearman's Rho Correlations Showing Resemblances in Snack Consumption Among Generational Family Roles.

	Child	Parent	Grand
Child	-	.100	-.043
n	47	44	42
Parent		-	.206
n		47	41
Grandparent			-
n			43

Note. Extended family members excluded.
*$p < 0.05$, **$p < 0.01$, (1-tailed).

Table E.4

Spearman's Rho Correlations Showing Resemblances in Fast Food Consumption Among Generational Family Roles.

	Child	Parent	Grand
Child	-	.117	.036
n	47	43	41
Parent		-	.273*
n		46	39
Grandparent			-
n			42

Note. Extended family members excluded.
*$p < 0.05$, **$p < 0.01$, (1-tailed).

Table E.5

Spearman's Rho Correlations Showing Resemblances in Fruit Consumption Among Family Members.

	Son	Daughter	Mother	Father	Grandmother	Grandfather
Son	-	.382	.28	.314	.419	.346
n	27	14	26	20	12	19
Daughter		-	.453**	-.159	-.115	-.487
n		35	33	20	28	9
Mother			-	.158	-.001a	-.071b
n			47	29	29	13
Father				-	.565[c]	.500[d]
n				29	7	4
Grandmother					-	.837**
n					38	11
Grandfather						-
n						17

Note . *p < 0.05, **p < 0.01, (1-tailed). N = 216. [a] Mother-maternal grandmother, [b] mother-maternal grandfather, [c] father-paternal grandmother, [d] father-paternal grandfather.

Table E.6.

Spearman's Rho Correlations Showing Resemblances in Vegetable Consumption Among Family Members.

	Son	Daughter	Mother	Father	Grandmother	Grandfather
Son	-	.487*	.531**	.076	-.013	.186
n	27	14	26	20	19	13
Daughter		-	.389*	.037	-.033	.034
n		35	32	20	28	9
Mother			-	.001	.075[a]	-.101[b]
n			46	28	28	14
Father				-	-.407[c]	-.056[d]
n				29	7	4
Grandmother					-	.435
n					38	11
Grandfather						-
n						18

Note. *p < 0.05, **p < 0.01 (1-tailed). N = 216. [a] Mother-maternal grandmother, [b] mother-maternal grandfather, [c] father-paternal grandmother, [d] father-paternal grandfather.

Table E.7.
Spearman's Rho Correlations Showing Resemblances in Snack Consumption Among Family Members.

	Son	Daughter	Mother	Father	Grandmother	Grandfather
Son	-	.321	.254	-.091	-.203	.017
n	27	14	25	20	19	13
Daughter		-	.219	-.141	.030	-.460
n		34	32	19	27	9
Mother			-	.215	.208	-.100
n			46	28	28	13
Father				-	.773*	.949*
n				30	6	4
Grandmother					-	.471[a]
n					38	11
Grandfather						-
n						18

* $p < 0.05$ (1-tailed). ** $p < 0.01$ (1-tailed). [a] $p = 0.07$ (1-tailed). $N = 216$. Mother-maternal grandmother, mother-maternal grandfather, father-paternal grandmother, father-paternal grandfather.

Table E.8
Spearman's Rho Correlations Showing Resemblances in Fast Food Consumption Among Family Members

	Son	Daughter	Mother	Father	Grandmother	Grandfather
Son	-	.892**	.116	.222	.171	.085
n	27	14	25	19	19	13
Daughter		-	.057	.123	.127	.216
n		34	32	19	27	9
Mother			-	.390*	.203[d]	.577* [e]
n			46	29	27	14
Father				-	[b f]	[c g]
n				29	7	3
Grandmother					-	[a]
n					38	11
Grandfather						-
n						18

Note. * $p < 0.05$ (1-tailed). ** $p < 0.01$, (1-tailed). [a] Grandparents mean fast food scores were < 1.0. [b] Paternal grandmothers and [c] paternal grandfathers scores were zeroes. [d] Mother-maternal grandmother tie, [e] mother-maternal grandfather tie, [f] father-paternal grandmother tie, [g] father-paternal grandfather tie.

Appendix 3.F: Healthy and Unhealthy Food: Analysis of Differences.

Table F.1

Vegetable consumption negative binomial GLiMM Model 2.1. While controlling for the nesting effect of individuals within families on the dependent variable vegetable consumption, the effect of gender, then the effect of generation, and effect of the interaction between gender and generation are presented in the table.

Model 2.1 Effect	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Corrected Model				(5, 194)			1.11	.36
Gender	Male-Female	.63	2.40	(1, 194)	0.11	1.14	5.77	**.02***
Generation				(2, 194)			0.30	.75
	Child-Parent	0.05	0.15	194	-0.57	0.66		1.0
	Child-Grand	0.25	0.74	194	-0.56	1.06		1.0
	Parent-Grand	0.20	0.33	194	-0.55	0.95		1.0
Gender by Generation				(2, 194)			0.17	.85
	Child x M-F	0.49	1.16	(1, 194)	-0.34	1.32	1.36	.25
	Parent x M-F	0.55	1.31	(1, 194)	-0.03	1.38	1.73	.19
	Grand x M-F	0.82	1.66	(1, 194)	-0.15	1.79	2.76	.10

Note. Model 2.1: -2 log pseudo likelihood = 299.582. The effect of each variable has controlled for all other variables entered into the model.

Table F.2

Snack consumption, negative binomial GLiMM Model 3.1. While controlling for the nesting effect of individuals within families on the dependent variable snack consumption, the effect of gender, then the effect of generation, and effect of the interaction between gender and generation are presented in the table.

Model 3.1. Effect	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Corrected Model				(5, 205)			3.27	**.007**
Gender	Male-Female	.58	1.32	(1, 205)	-0.29	1.44	1.74	.19
Generation				(2, 205)			5.79	**<.01**
	Child-Parent	1.62	3.25	205	0.42	2.83		**<.01**
	Child-Grand	1.47	2.67	205	0.23	2.71		**.02**
	Parent-Grand	0.16	0.32	205	-0.81	1.13		.75
Gender by Generation								
	Child x M-F	0.62	0.77	(1, 205)	-0.97	2.21	0.59	.44
	Parent x M-F	0.72	1.14	(1, 205)	-0.53	1.97	1.30	.26
	Grand x M-F	0.38	0.48	(1, 205)	-1.18	1.94	0.23	.64

Note. Model 3.1: -2 log pseudo likelihood = 422.508. The effect of each variable has controlled for all other variables entered into the model.

Table F.3.

Fast food consumption, negative binomial Model 4.1. While controlling for the nesting effect of individuals within families on the dependent variable fast food consumption, the effect of gender, then the effect of generation, and effect of the interaction between gender and generation are presented in the table.

Model 4.1 Effect	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Corrected Model				(5. 206)			6.71	**<.001**
Gender	Male-Female	.04	0.28	(1, 206)	-0.22	0.29	0.08	.78
Generation				(2, 206)			20.31	**<.001**
	Child-Parent	0.36	2.39	206	0.06	0.65		**.02**
	Child-Grand	0.83	5.85	206	0.48	1.17		**<.001**
	Parent-Grand	0.47	4.26	206	0.22	0.72		**<.001**
Gender by Generation	Child x M-F	0.14	0.58	(1, 206)	-0.62	0.34	0.33	.57
	Parent x M-F	0.33	1.69	(1, 206)	-0.06	0.72	2.85	.09
	Grand x M-F	0.02	0.18	(1, 206)	-0.17	0.20	0.03	.86

Note. Model 4.1: -2 log pseudo likelihood = 767.282. The effect of each variable has controlled for all other variables entered into the model.

Appendix 3.G: Non-significant Gender Difference Trends in Diet-Health Oriented Behaviour (DHOB) and Diet-Health/disease Linked Attitudes (DHLA) SPSS 22 Output Tables

Non-significant Gender Difference Trends in Diet-Health Oriented Behaviour (DHOB)

SPSS 22 Output Tables

With a non-significant result, a Poisson GLiMM tested for differences between generation, gender, and the interaction between generation and gender on the dependent variables diet health orientation (DHOB) and diet health/disease linked attitudes (DHLA). Parents showed the greatest difference between genders, mother's scores were higher than fathers, while children and grandparents shared gender similarities in levels of DHOB F = (2, 45), 0.19, p = .83. Diet-health/disease linked attitude was tested with the same analysis which was also non-significant. The largest gender difference was again between parents; mother's scores were greater than fathers. The next largest difference was between grandparents; grandmothers scored slightly higher than grandfathers, while children shared gender similarities in DHLA levels DHLA levels F = (2, 68), 0.07, p = .93. The SPSS Output tables and figures are shown below.

Model Term	Coefficient ▼	Std.Error	t	Sig.	Exp(Coefficient)	95% Confidence Interval for Exp (Coefficient)	
						Lower	Upper
Intercept	1.610	0.141	11.382	.000	5.001	3.761	6.648
FamilyRolex3=1	-0.051	0.269	-0.191	.850	0.950	0.552	1.635
FamilyRolex3=2	0.060	0.186	0.321	.750	1.061	0.730	1.543
FamilyRolex3=3	0[a]						
Gender=1	-0.034	0.233	-0.146	.885	0.967	0.604	1.547
Gender=2	0[a]						
[FamilyRolex3=1]*[Gender=1]	0.066	0.357	0.184	.855	1.068	0.520	2.192
[FamilyRolex3=1]*[Gender=2]	0[a]						
[FamilyRolex3=2]*[Gender=1]	-0.131	0.311	-0.421	.676	0.877	0.469	1.642
[FamilyRolex3=2]*[Gender=2]	0[a]						
[FamilyRolex3=3]*[Gender=1]	0[a]						
[FamilyRolex3=3]*[Gender=2]	0[a]						

Probability distribution:Poisson
Link function:Log

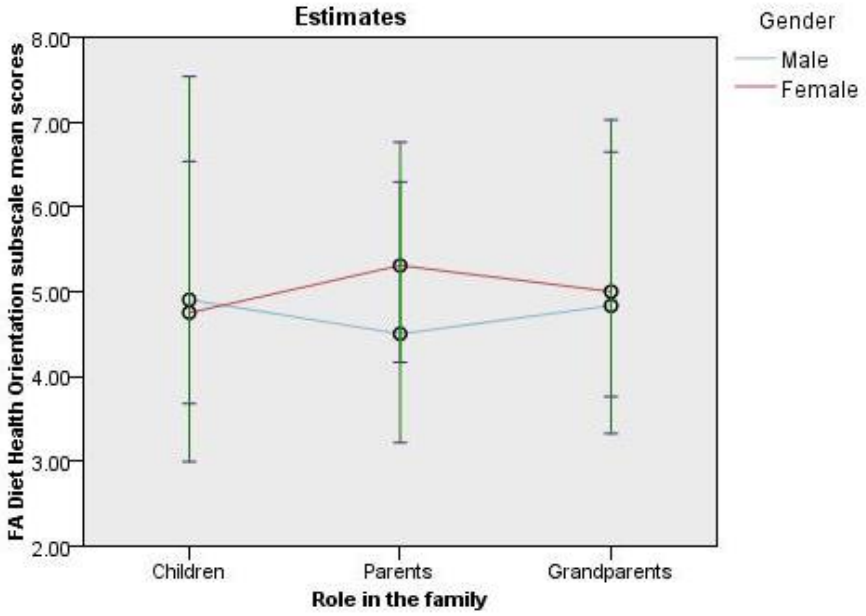
[a]This coefficient is set to zero because it is redundant.

Source	F	df1	df2	Sig.
Corrected Model ▼	0.144	5	45	.981
FamilyRolex3	0.006	2	45	.994
Gender	0.164	1	45	.687
Gender*FamilyRolex3	0.189	2	45	.829

Probability distribution:Negative binomial
Link function:Log

Overall Test Results

Gender	F	df1	df2	Sig.
Male	0.083	2	45	0.920
Female	0.114	2	45	0.892



Non-significant Gender Difference Trends in Diet-Health/disease Linked Attitudes (DHLA) SPSS 22 Output Tables

Model Term	Coefficient ▼	Std.Error	t	Sig.	Exp(Coefficient)	95% Confidence Interval for Exp (Coefficient)	
						Lower	Upper
Intercept	1.854	0.110	16.890	.000	6.385	5.129	7.948
FamilyRolex3=1	-0.077	0.166	-0.467	.642	0.926	0.665	1.288
FamilyRolex3=2	0.041	0.136	0.301	.764	1.042	0.794	1.368
FamilyRolex3=3	0[a]						
Gender=1	-0.062	0.200	-0.311	.757	0.940	0.631	1.400
Gender=2	0[a]						
[FamilyRolex3=1]*[Gender=1]	0.049	0.264	0.186	.853	1.050	0.621	1.777
[FamilyRolex3=1]*[Gender=2]	0[a]						
[FamilyRolex3=2]*[Gender=1]	-0.041	0.255	-0.162	.872	0.960	0.577	1.595
[FamilyRolex3=2]*[Gender=2]	0[a]						
[FamilyRolex3=3]*[Gender=1]	0[a]						
[FamilyRolex3=3]*[Gender=2]	0[a]						

Probability distribution:Poisson
Link function:Log

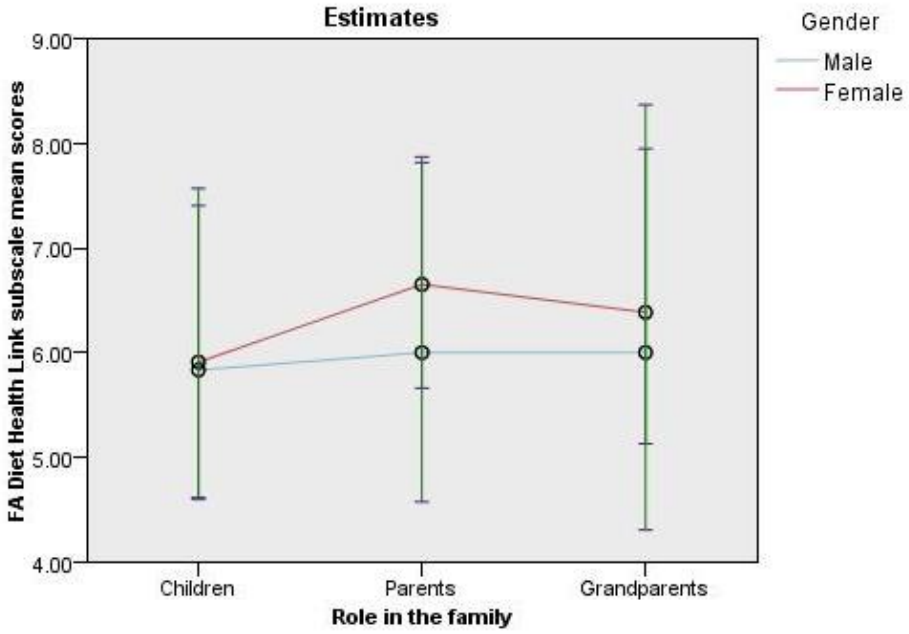
[a]This coefficient is set to zero because it is redundant.

Source	F	df1	df2	Sig.
Corrected Model ▼	0.263	5	68	.932
FamilyRolex3	0.202	2	68	.817
Gender	0.335	1	68	.564
FamilyRolex3*Gender	0.074	2	68	.928

Probability distribution:Poisson
Link function:Log

Overall Test Results

F	df1	df2	Sig.
0.205	2	68	0.815



Food attitudes and healthy food consumption across three generations: Gender explored. ***Correlations.*** Since gender difference trends were noted in food attitudes above, associations between diet-health food attitudes (DHOB and DHLA)[63] and food consumption with generations were investigated further with correlations by gender. Grandmothers had a positive correlation between diet-health/disease food attitude (DHLA) and healthy food consumption, and grandfathers had a positive DHLA with fruit consumption correlation. Positive correlations existed between son's food attitudes and healthy food consumption, as did a positive correlation in fathers' DHOB with vegetable consumption (see Table G.1)

[63] Note that DHOB relates to healthy dietary behaviour and DHLA relates to diet-health/disease linked attitudes.

Table G.1

Spearman's rho Correlations of Food Attitude Subscales with Fruit and Vegetable Consumption by Generation and Gender.

	Fruit Consumption						Vegetable Consumption					
	Children		Parents		Grandparents		Children		Parents		Grandparents	
	M	F	M	F	M	F	M	F	M	F	M	F
FLQ-SF Subscale												
DHOB	.551**	.351*	.178	-.035	.121	.195	.670**	.273	.546**	-.164	.110	.404**
n	26	35	29	47	17	38	26	35	29	46	18	38
DHLA	.336*	-.040	.21	-.049	.437*	.388**	.243	.054	.170	-.100	-.200	.234
n	26	33	29	47	17	37	26	33	29	46	18	37

Note. * $p < 0.05$ one tailed. ** $p < 0.01$ one tailed. Diet-health oriented behaviour (DHOB); Diet-health/disease linked attitudes (DHLA).

Correlations in food attitudes and unhealthy food consumption across three generations:

Correlations examining the relationship between food attitudes[64] and unhealthy food consumption were extended also to include gender. Table E.2 shows the associations between measures of food attitudes and snack and fast food consumption. Results showed daughters negative correlation with snack consumption, and fathers a large negative correlation with fast food consumption (see Table G.2).

[64] Note that DHOB relates to healthy dietary behaviour and DHLA relates to diet-health/disease linked attitudes.

Table G.2.
Spearman's rho Correlations of Food Attitude Subscales with Snack and Fast Food Consumption by Generation and Gender.

	Snack Consumption						Fast Food Consumption					
	Child		Parent		Grand		Child		Parent		Grand	
	M	F	M	F	M	F	M	F	M	F	M	F
DHOB	-.302	-.476**	-.194	-.238	.097	-.156	.063	-.040	-.548**	-.228	-.051	-.113
n	26	34	29	46	18	38	26	34	29	46	18	37
DHLA	-.188	-.110	-.194	-.068	.125	-.199	.011	-.232	-.136	.133	.137	-.009
n	26	32	29	46	18	37	26	32	29	46	18	36

Note. * $p < 0.05$ one tailed. ** $p < 0.01$ one tailed. Diet-health oriented behaviour (DHOB); Diet-health/disease linked attitudes (DHLA).

Appendix 3.H: Non-significant Independent Effects of Diet-Health Oriented Behaviour (DHOB) or Diet-Health/disease Linked Attitudes (DHLA) on Food Consumption

Food Attitudes and Healthy Food

Fruit consumption GLiMMs. Two second stage Poisson GLiMMs examined for any independent effect that diet-health oriented behaviour (DHOB) or diet-health/disease linked attitudes (DHLA) might have had beyond the effects of generations and gender found in fruit consumption earlier in Model 1.1 (as seen in Table 14 on p. 26) which is duplicated below for direct comparison). Second stage models added DHOB to Model 1.1 (Figure H.1, Table H.1) and Model 1.3 added DHLA to Model 1.1 (Figure H.2, Table H.2). When comparing each second stage model separately to the first stage model, results of subsequent tests of significance informed whether the food attitudes DHOB and DHLA had any independent effect on the outcome variable. Independent effects can only be determined by likelihood ratio test and chi square test of significance[65] each time a new predictor is added to an existing model.

In Model 1.2, DHOB was added to Model 1.1. A likelihood ratio test showed the difference in -2 log pseudo likelihood was greater, indicating a non-significant effect of DHOB. In Model 1.3 when DHOB was removed and DHLA was added[66] to Model 1.1, the -2 log pseudo likelihood result was smaller; therefore the model was improved upon, however the chi-square statistic on the difference was not significant. Therefore, the second stage food attitudes model did not significantly add to the first stage model for fruit consumption (see Figure H.1 and Table H.1, Figure H.2 and Table H.2 for Models 1.2 and 1.3 compared with Model 1.1). The same likelihood ratio test was used when comparing the addition of food

[65] Likelihood ratio tests calculate the difference between two models and a chi square statistic tests for significance. If the stage two model -2 log pseudo likelihood result is greater than the stage one model, then the difference effect is non-significant. If the stage two model -2 log pseudo likelihood result is less than the stage one model, then the model is improved with the addition of a new predictor, however, a chi square test of significance reveals whether the difference between the two model's -2 log pseudo likelihood results are significant or not. Effect size was not available at time of writing; however, phi coefficient was experimented with as an indication of effect size.

[66] Both DHOB and DHLA could not be entered into the model at once due to variable limitations on the analysis.

attitudes to the remaining food consumption variables: vegetables, snacks and fast food models that follow.

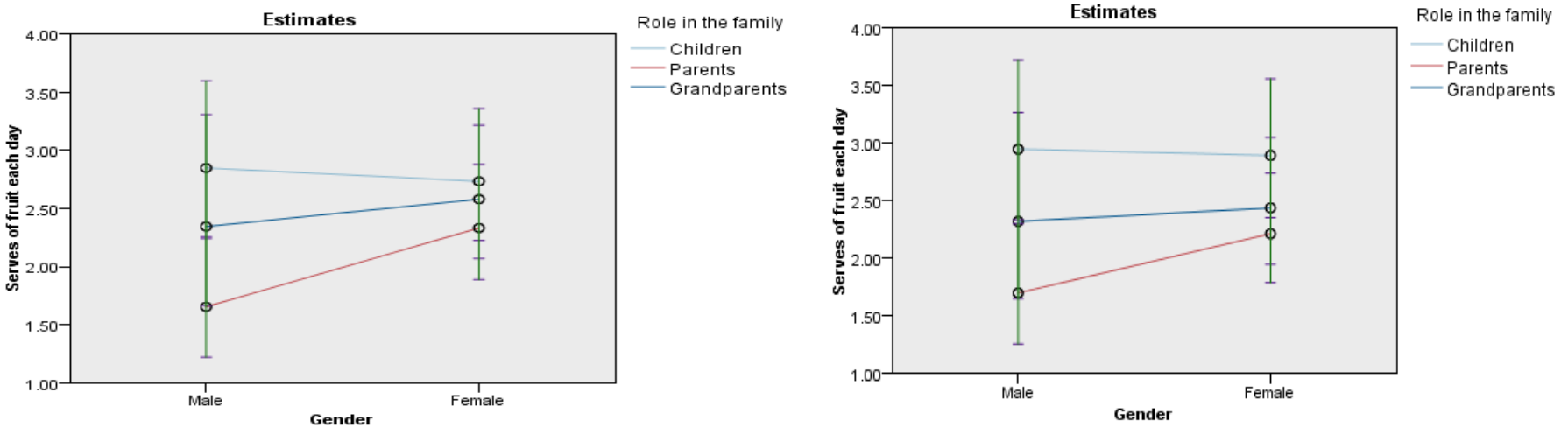


Figure H.1. GLiMM Fruit consumption Model 1.1 (left) is compared alongside Model 1.2 (right). In the first model there was a non-significant effect of gender, a significant effect of generation between parent-child, and a significant interaction between gender and generation between mothers and fathers. In Model 1.2 (right) DHOB was added to Model 1.1 where it can be seen that there was a non-significant effect of gender, a significant effect of generation between parent-child, and a non-significant interaction between gender and generation. Each predictor was controlled for the effect of all other predictors within the model. Error bars depict 95% confidence intervals.

Table H.1.

Fruit Poisson Model 1.1. While controlling for the nesting effect of individuals within families on the dependent variable fruit consumption, the effect of gender, then the effect of generation (controlling for gender), and

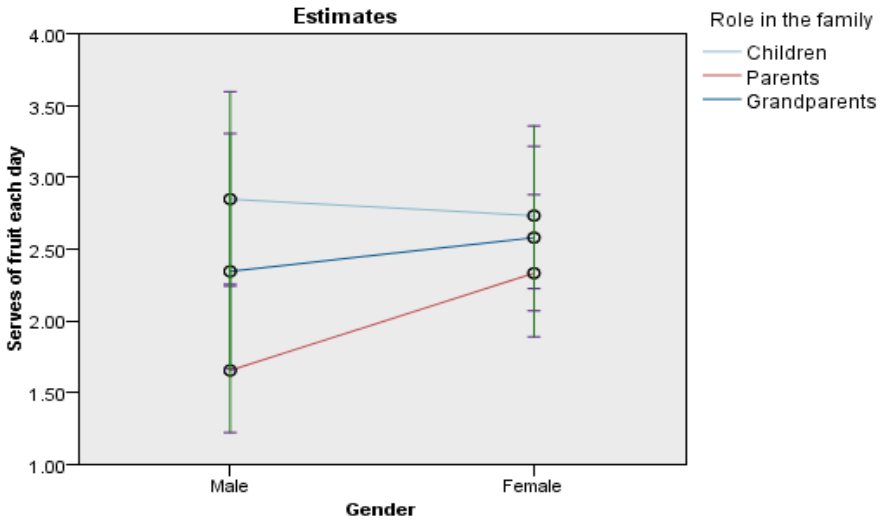
	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 1.1				(5, 209)			1.49	.19
Gender	Male-Female	0.32	1.34	(1, 207)	-0.8	0.2	1.79	.18
Generation				(2, 207)			5.24	**<.01**
	Child-Parent	0.83	3.2		0.2	1.5		**<.01**
	Child-Grand	0.33	1.08		-0.3	0.9		.28
	Parent-Grand	0.5	1.69		-0.2	1.2		.18
Gender by Generation				(2, 207)			1.46	.23
	Child x M-F	0.12	0.28	(1, 207)	-0.7	0.9	0.08	.78
	Parent x M-F	0.68	2.07	(1, 207)	0.32	1.3	4.28	**.04**
	Grand x M-F	0.24	0.5	(1, 207)	-0.7	1.2	0.25	.62
[a]								

Note . Model 1.1 GLiMM Poisson. -2 log pseudo likelihood = 438.257. [a] Row intentionally left blank.

Fruit Model 1.2: Results are shown with the addition of diet-health orientated behaviour (DHOB) to Fruit Model 1.1 (shown left for direct comparison).

	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 1.2				(6, 205)			3.14	**.006**
Gender	Male-Female	0.23	0.98	(1, 205)	-0.70	0.24	0.97	.33
Generation				(2, 205)			6.76	**.001**
	Child-Parent	0.98	3.65		0.33	1.63		**.001**
	Child-Grand	0.54	1.73		-0.17	1.25		.17
	Parent-Grand	0.44	1.54		-0.16	1.04		.17
Gender by Generation				(2, 205)			0.79	.46
	Child x M-F	0.05	0.13	(1, 205)	-0.79	0.90	0.02	.89
	Parent x M-F	0.51	1.56	(1, 205)	-0.14	1.17	2.43	.12
	Grand x M-F	0.12	0.25	(1, 205)	-0.79	1.03	0.07	.80
DHOB				(1, 205)			6.79	**.01**

Note . Model 1.2 GLiMM Poisson. -2 log pseudo likelihood = 439.493 and is greater than Model 1.1 therefore diet-health oriented behaviour (DHOB) does not make a significant contribution to Model 1.1.



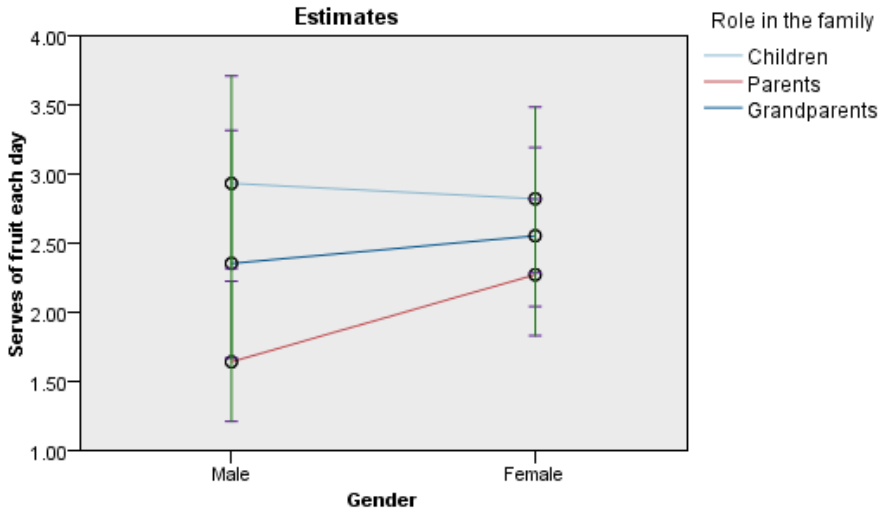


Figure H.2. GLiMM fruit consumption Model 1.1 (left) is compared alongside Model 1.3 (right). In the first model there was a non-significant effect of gender, a significant effect of generation between parent-child, and a significant interaction between gender and generation between mothers and fathers. In Model 1.3 DHLA was added to Model 1.1 and there was a non-significant effect of gender, a significant effect of generation between parent-child and an interaction between gender and generation that neared significance between parents. Each predictor was controlled for the effect of all other predictors within the model. Error bars depict 95% confidence intervals.

Table H.2

Fruit Poisson Model 1.1. While controlling for the nesting effect of individuals within families on the dependent variable fruit consumption, the effect of gender, then the effect of generation (controlling for gender), and effect of the interaction between gender and generation are presented in the table.

	Pairwise Contrasts	Contrast	*t*	*df*	95% CI Lower		*F*	*p*
Model 1.1				(5, 209)			1.49	.19
Gender	Male-Female	0.32	1.3	(1, 207)	-0.8	0.15	1.79	.18
Generation				(2, 207)			5.24	**<.01**
	Child-Parent	0.83	3.2		0.2	1.45		**<.01**
	Child-Grand	0.33	1.1		-0.3	0.93		.28
	Parent-Grand	0.5	1.7		-0.2	1.16		.18
Gender by Generation				(2, 207)			1.46	.23
	Child x M-F	0.12	0.3	(1, 207)	-0.7	0.92	0.08	.78
	Parent x M-	0.68	2.1	(1, 207)	0.32	1.32	4.28	**.04**
	Grand x M-F	0.24	0.5	(1, 207)	-0.7	1.16	0.25	.62
[a]								

Note . Model 1.1 GLiMM Poisson. -2 log pseudo likelihood = 438.257. [a] Row intentionally left blank.

Fruit Model 1.3. Results are shown with DHOB removed and diet-health/disease linked attitudes (DHLA) added to Model 1.1 (shown left for direct comparison).

		Contrast	*t*	*df*	95% CI Lower		*F*	*p*
Model 1.3				(6, 202)			2.22	**.04**
Gender	Male-Female	0.29	1.23	(1, 202)	-0.2	0.8	1.51	.22
Generation				(2, 202)			7.1	**.003**
	Child-Parent	0.95	3.45		0.28	1.6		**.002**
	Child-Grand	0.43	1.35		-0.2	1.1		.18
	Parent-Grand	0.52	1.78		-0.1	1.2		.15
Gender by Generation				(2, 202)			1.29	.28
	Child x M-F	0.11	0.26	(1, 202)	-0.7	1	0.07	.79
	Parent x M-F	0.63	1.94	(1, 202)	-0	1.3	3.74	**.054**
	Grand x M-F	0.2	0.42	(1, 202)	-0.7	1.1	0.18	.67
DHLA				(1, 202)			1.26	.26

Note. Model 1.3 GLiMM Poisson. -2 log pseudo likelihood = 435.228. The X^2 difference between Model 1.1 and 1.3 for 1 *df* is 3.03 which is less than the critical value of 3.84 at the $p < .05$ level, therefore diet-health/disease linked attitudes (DHLA) did not make a significant contribution to Model 1.1.

Vegetable consumption GLiMM. Model 2.3 (Figure H.3 and Table H.3) built upon the previous vegetable Model 2.1 (as seen in Table 15 on p. 28) which is duplicated below for direct comparison). A negative binomial GLiMM tested for any added effect of food attitudes over and above the significant effect of gender evident in Model 2.1 (i.e., female's vegetable consumption was greater than males). In Model 2.3 when DHLA was added to Model 2.1, it can be seen that the significant effect of gender was retained. The likelihood ratio test between Model 2.1 and Model 2.3 showed no significant improvement therefore DHLA did not make a significant contribution to vegetable consumption (see Figure H.3 and Table H.3).

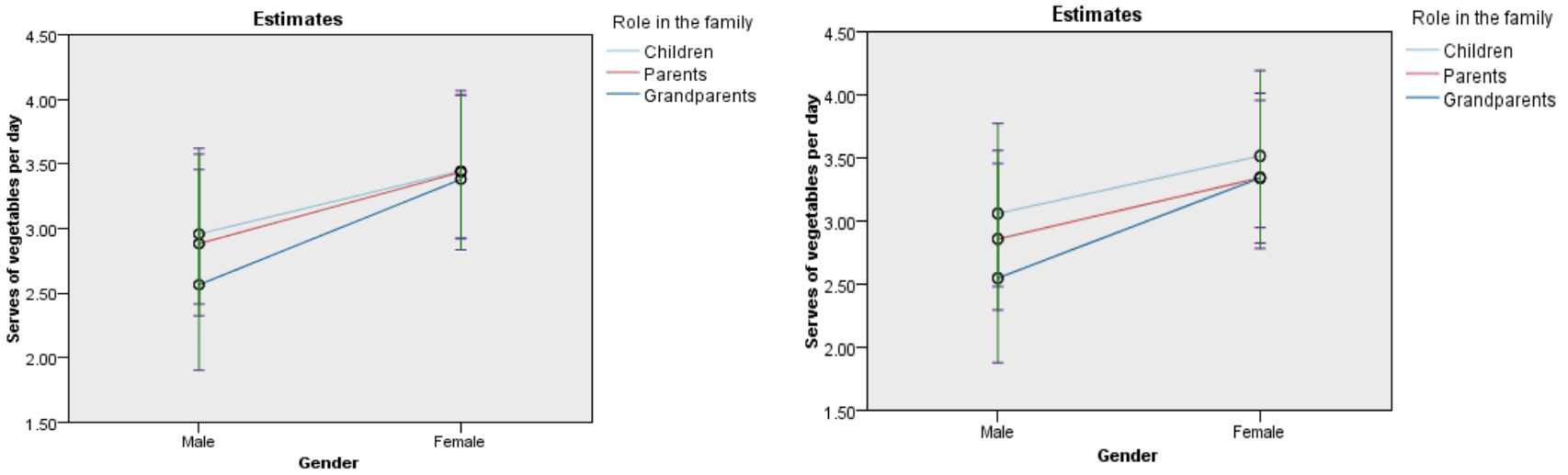


Figure H.3. GLiMM vegetable consumption Model 2.1 (left) is compared alongside Model 2.3 (right). In the first model there was a significant effect of gender, a non-significant effect of generation and no significant interaction between gender and generation. In Model 2.3 (right) DHLA was added to Model 2.1 and there was a significant effect of gender, a non-significant effect of generation, and no significant interaction between gender and generation. Each predictor was controlled for the effect of all other predictors within the model. Error bars depict 95% confidence intervals.

Table H.3

Vegetable consumption Model 2.1. While controlling for the nesting effect of individuals within families on the dependent variable vegetable consumption, the effect of gender, then the effect of generation (controlling for gender), and effect of the interaction between gender and generation are presented in the table.

	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	Upper	*F*	*p*
Model				(5, 194)			1.11	.36
Gender	Male-Female	0.63	2.4	(1, 194)	0.11	1.14	5.77	**.02***
Generation				(2, 194)			0.3	.75
	Child-Parent	0.05	0.15	194	-0.57	0.66		1.0
	Child-Grand	0.25	0.74	194	-0.56	1.06		1.0
	Parent-Grand	0.2	0.33	194	-0.55	0.95		1.0
Gender by Generation				(2, 194)			0.17	.85
	Child x M-F	0.49	1.16	(1, 194)	-0.34	1.32	1.36	.25
	Parent x M-F	0.55	1.31	(1, 194)	-0.03	1.38	1.73	.19
	Grand x M-F	0.82	1.66	(1, 194)	-0.15	1.79	2.76	.10
[a]								

Note . Model 2.1 GLiMM negative binomial. -2 log pseudo likelihood = 299.582. Negative binomial. [a] Row intentionally left blank.

Vegetables Model 2.3: Results are shown with the addition of diet-health/disease linked attitudes (DHLA) to Model 2.1 (shown left for direct comparison).

	Pairwise Contrasts	Contrast Estimate	t	*df*	95% CI Lower	Upper	*F*	*p*
Model				(6, 189)			1.27	.27
Gender	Male-Female	0.56	2.17	(1, 189)	0.05	1.17	4.73	**.03**
Generation				(2, 189)			0.55	.58
	Child-Parent	0.19	0.6		-0.53	0.9		1.0
	Child-Grand	0.36	1.05		-0.47	1.2		.89
	Parent-Grand	0.17	0.51		-0.56	0.91		1.0
Gender by Generation				(2, 189)			0.19	.83
	Child x M-F	0.46	1.02	(1, 189)	-1.33	0.42	1.05	.31
	Parent x M-F	0.49	1.15	(1, 189)	-1.31	0.35	1.32	.25
	Grand x M-F	0.79	1.59	(1, 189)	-1.78	0.19	2.52	.11
DHL				(1, 189)			1.91	.17

Note. Model 2.3 GLiMM negative binomial. -2 log pseudo likelihood = 297.362. X^2 difference between Model 2.1 and Model 2.3 for 1 *df* = 2.22 which is less than the critical value of 3.84 at the $p < .05$ level, therefore diet-health/disease linked attitudes (DHLA) did not make a significant improvement on Model 2.1.

Food Attitudes and Unhealthy Food

Fast Food GLIMM. A negative binomial GLiMM was conducted to test for any independent effects of diet-health oriented behaviour, controlling for the effects that generation and gender had on the previous fast food Model 4.1 which showed children's fast food consumption was significantly greater than parents and grandparents; and that parental fast food consumption was also greater than grandparents. (as seen in Table 17 on p. 32). Model 4.2 built on Model 4.1 (see Figure F.4 and Table F.4). In Model 4.2 when DHOB was added to Model 4.1 the model changed however the effect of DHOB was not strong enough to be significant. The likelihood ratio test between Model 4.1 and Model 4.2 did not exceed the critical value of 3.84 for 1 degree of freedom (the X^2 difference was 3.14). Therefore, DHOB did not add significantly to Model 4.1 for fast food consumption (see Figure F.4 and Table F.4).

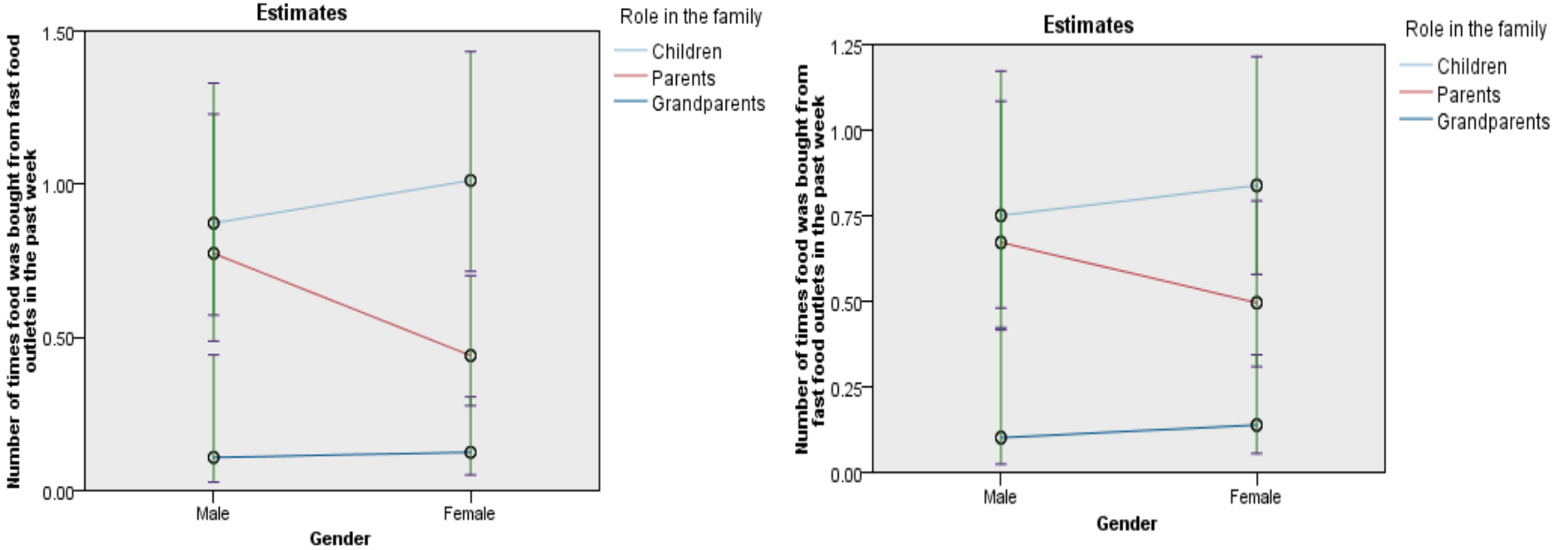


Figure H.4. GLiMM fast food consumption Model 4.1 (left) is compared alongside Model 4.2 (right). In the first model there was a non-significant effect of gender, a significant effect of generation between each dyad, and a non-significant interaction between gender and generation. In Model 4.2 (right) DHOB was added to Model 4.1 and there was a non-significant effect of gender, a significant effect of generation (although no longer significant between child-parent dyad), and a non-significant interaction between gender and generation. Each predictor was controlled for the effect of all other predictors within the model. Error bars depict 95% confidence intervals

Table H.4.

Fast Food Model 4.1. While controlling for the nesting effect of individuals within families on the dependent variable fast food consumption, the effect of gender, then the effect of generation, and effect of the interaction between gender and generation are presented in the table.

	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 4.1				(5. 206)			6.7	**<.001**
Gender	Male-Female	0.04	0.3	(1, 206)	-0.2	0.29	0.1	.78
Generation				(2, 206)			20	**<.001**
	Child-Parent	0.36	2.4	206	0.06	0.65		**.02**
	Child-Grand	0.83	5.9	206	0.48	1.17		**<.001**
	Parent-Grand	0.47	4.3	206	0.22	0.72		**<.001**
Gender by Generation				(2, 206)			1.6	.20
	Child x M-F	0.14	0.6	(1, 206)	-0.6	0.34	0.3	.57
	Parent x M-F	0.33	1.7	(1, 206)	-0.1	0.72	2.9	.09
	Grand x M-F	0.02	0.2	(1, 206)	-0.2	0.2	0	.86
[a]								

Note . Model 4.1: GLiMM negative binomial. -2 log pseudo likelihood = 767.282. The effect of each variable has controlled for all other variables entered into the model. [a] Row intentionally left blank.

Fast Food Model 4.2: *Results are shown with the addition of diet-health oriented behaviour (DHOB) to Model 4.1 (shown left for direct comparison).*

	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 4.2				(6, 204)			7.3	**<.001**
Gender	Male-Female	0.28	0.1	(1, 204)	-0.2	0.25	0	.90
Generation				(2, 204)			16	**<.001**
	Child-Parent	0.22	1.5		-0.1	0.5		.13
	Child-Grand	0.68	5		0.35	1		**<.001**
	Parent-Grand	0.46	4.1		0.21	0.71		**<.001**
Gender by Generation				(2, 204)			0.6	.56
	Child x M-F	0.09	0.4	(1, 204)	-0.5	0.32	0.2	.67
	Parent x M-F	0.18	0.9	(1, 204)	-1.2	0.56	0.8	.36
	Grand x M-F	0.04	0.4	(1, 204)	-0.2	0.23	0.2	.70
DHOB				(1, 204)			11	**.001**

Note . Model 4.2: GLiMM negative binomial. -2 log pseudo likelihood = 764.139. The X^2 difference between Model 4.1 and 4.2 for 1 *df* = 3.14 and did not exceed the critical value of 3.84 therefore diet-health oriented behaviour did not make a significant contribution to the model at the $p < .05$ level.

Appendix 3.I: Odds of Fruit Consumption Moderation with Diet-Health Oriented Behaviour (DHOB)

Model 5 Fruit Consumption Moderation with DHOB

Model 5.1 (Table I.1) results show that of the 207 individuals within 50 families who either met the NHMRC daily fruit consumption recommendations (adjusted for age, e.g., > 2 serves per day for adults) or did not (e.g., < 2 serves per day), higher diet-health oriented behaviour (DHOB) food attitude scores were significant predictors of fruit consumption (DHOB coefficient = 0.71, SE = 0.23, p = .002, odds ratio = 2.04, 95% CI = 1.29 to 3.23). Generation was not a significant predictor of fruit consumption (see Table I.1) (Lang & Secic, 2006).

Table I.1

Model 5.1

Fruit Multiple Logistic Regression all predictors entered together.

		Fruit recommendations met? [d] (serves per day) N = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	p	
Intercept		-2.46	0.09	0.008	0.93	**0.043**	
Generation							
	Child	0.31	1.37	0.53	3.52	0.52	
	Parent	-0.59	0.55	0.23	1.31	0.176	
	Grand	0.00[c]	1				
Food Attitudes							
	DHOB[e]	0.71	2.04	1.29	3.23	**0.002**	SE = 0.23

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

In Model 5.2 (Table I.2) gender was added to the model and no significant contribution was made to the model. Diet-health oriented behaviour (DHOB) remained a significant predictor of fruit consumption, (DHOB coefficient = 0.70, SE = 0.24, p = .004, odds ratio = 2.02, 95% CI = 1.25 to 3.24; see Table I.2) (Lang & Secic, 2006).

Table I.2
Model 5.2
Fruit Multiple Logistic Regression all predictors entered together.

		Fruit recommendations met? [d] (serves per day) *N* = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	*p*	
Intercept		-2.35	0.10	0.008	1.15	.06	
Generation							
	Child	0.31	1.37	0.53	3.51	.52	
	Parent	-0.59	0.55	0.23	1.32	.18	
	Grand	0.00[c]	1				
Gender							
	Male	-0.14	0.87	0.46	1.64	.67	
	Female	0.00[c]					
Food Attitudes							
	DHOB[e]	0.70	2.02	1.25	3.24	**.004**	SE = 0.24

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

In Model 5.3 (Table I.3) the interaction gender-by-DHOB was added to the previous model and no significant contribution was made. Diet-health oriented behaviour (DHOB) food attitudes remained a significant predictor of fruit consumption, (DHOB coefficient = 0.76, SE = 0.24, p = .004, odds ratio = 2.13, 95% CI = 1.33 to 3.43; see Table I.3) (Lang & Secic, 2006).

Table I.3
Model 5.3
Fruit Multiple Logistic Regression all predictors entered together.

		Fruit recommendations met? [d] (serves per day) *N* = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	*p*	
Intercept		-2.61	0.07	0.006	0.91	**.04**	
Generation							
	Child	0.33	1.39	0.53	3.62	.50	
	Parent	-0.60	0.55	0.23	1.32	.18	
	Grand	0.00[c]	1.00				
Gender							
	Male	0.44	1.56	0.10	29.40	.76	
	Female	0.00[c]	1.00				
Food Attitudes							
	DHOB[e]	0.76	2.13	1.33	3.43	**.004**	SE = 0.24
Interaction	Gender*DHOB[e]						
	Male*DHOB[e]	-0.13	0.88	0.47	1.66	0.70	
	Female*DHOB[e]	0.00[c]					

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

In Model 5.4 (Table I.4) the interaction generation-by-DHOB was added to the previous model and no significant contribution was made. Diet-health oriented behaviour (DHOB) food attitudes remained a significant predictor of fruit consumption, (DHOB coefficient = 0.97, SE = 0.37, p = .01, odds ratio = 2.62, 95% CI = 1.26 to 5.47; see Table I.4) (Lang & Secic, 2006).

Table I.4

Model 5.4

Fruit Multiple Logistic Regression all predictors entered together.

		Fruit recommendations met? [d] (serves per day) N = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	p	
Intercept		-3.60	0.03	0.001	1.23	.06	
Generation							
	Child	0.29	1.33	0.009	>100.00	.91	
	Parent	2.19	8.96	0.05	>100.00	.40	
	Grand	0.00[c]	1.00				
Gender							
	Male	0.40	1.49	0.09	25.10	.78	
	Female	0.00[c]					
Food Attitudes							
	DHOB[e]	0.97	2.62	1.26	5.47	**.01**	SE = 0.37
Interaction	Gender*DHOB						
	Male*DHOB[e]	-0.13	0.88	0.46	1.67	.69	
	Female*DHOB[e]	0.00[c]	1.00				
Interaction	Generation*DHOB						
	Child*DHOB	0.05	1.05	0.38	2.92	0.93	
	Parent*DHOB	-0.57	0.51	0.21	1.55	0.27	
	Grand*DHOB	0.00[c]	1				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

In Model 5.5 (Table I.5) the interaction generation-by-gender was added to the previous model and no significant contribution was made. Diet-health oriented behaviour (DHOB) food attitudes remained a significant predictor of fruit consumption, (DHOB coefficient = 0.92, SE = 0.39, p = .02, odds ratio = 2.51, 95% CI = 1.18 to 5.34; see Table I.5) (Lang & Secic, 2006).

Table I.5
Model 5.5
Fruit Multiple Logistic Regression all predictors entered together.

		Fruit recommendations met? [d] (serves per day) *N* = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	*p*	
Intercept		-3.20	0.04	0.001	2.28	.12	
Generation							
	Child	-0.05	0.95	0.006	>100.00	.98	
	Parent	1.71	5.52	0.02	>100.00	.56	
	Grand	0.00[c]	1.00				
Gender							
	Male	-0.27	0.77	0.03	21.31	.88	
	Female	0.00[c]					
Food Attitudes							
	DHOB[e]	0.92	2.51	1.18	5.34	**.02**	SE = 0.39
Interaction	Gender*DHOB						
	Male*DHOB[e]	-0.09	0.91	0.46	1.84	.80	
	Female*DHOB[e]	0.00[c]	1.00				
Interaction	Generation*DHOB						
	Child*DHOB	0.64	1.07	0.37	3.1	.91	
	Parent*DHOB	-0.52	0.60	0.2	1.79	.36	
	Grand*DHOB	0.00[c]	1.00				
Interaction	Generation*Gender						
	Sons	0.61	1.85	0.40	8.60	.44	
	Daughters	0.00[c]	1.00				
	Fathers	0.59	1.80	0.39	8.40	.45	
	Mothers	0.00[c]	1.00				
	Grandfathers	0.00[c]	1.00				
	Grandmothers	0.00[c]	1.00				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

Finally, in Model 5.6 (Table I.6) a three-way interaction was added to the previous model and this was also non-significant. Again, diet-health oriented behaviour (DHOB) food attitudes remained a significant predictor of fruit consumption, (DHOB coefficient = 1.17, SE = 0.51, p = .02, odds ratio = 3.22, 95% CI = 1.19 to 8.69). Generation, gender and interactions were not significant predictors of fruit consumption (see Table I.6) (Lang & Secic, 2006).

Table I.6

Model 5.6

Fruit Multiple Logistic Regression all predictors entered together.

Predictor		Fruit recommendations met? [d] (serves per day) *N* = 207 B[a]	Exp(B)[b] OR	95% CI Lower	95% CI Upper	*p*	
Intercept		-4.41	0.01	0.00	1.78	.08	
Generation							
	Child	2.43	11.4	0.02	>100.00	.44	
	Parent	2.20	9.01	0.004	>100.00	.58	
	Grand	0.00[c]	1.00				
Gender							
	Male	3.10	22.28	0.009	>100.00	.44	
	Female	0.00[c]					
Food Attitudes							
	DHOB[e]	1.17	3.22	1.19	8.69	**.02**	SE = 0.51
Interaction	Gender*DHOB						
	Male*DHOB[e]	-0.79	0.45	0.09	2.31	.34	
	Female*DHOB[e]	0.00[c]	1.00				
Interaction	Generation*DHOB						
	Child*DHOB	-0.49	0.61	0.16	2.28	.46	
	Parent*DHOB	-0.62	0.54	0.12	2.49	.43	
	Grand*DHOB	0.00[c]	1.00	0.00			
Interaction	Generation*Gender						
	Sons	-5.44	0.004	0.00	20.79	.21	
	Daughters	0.00[c]	1.00				
	Fathers	-1.44	0.24	0.00	>100.00	.80	
	Mothers	0.00[c]	1.00				
	Grandfathers	0.00[c]	1.00				
	Grandmothers	0.00[c]	1.00				
Interaction	Generation*Gender*DHOB						
	Sons*DHOB	1.35	3.85	0.66	22.51	.14	
	Daughters*DHOB	0.00[c]	1.00				
	Fathers*DHOB	0.43	1.53	0.17	14.23	.71	
	Mothers*DHOB	0.00[c]	1.00				
	Grandfathers*DHOB	0.00[c]	1.00				
	Grandmothers*DHOB	0.00[c]	1.00				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group or redundant; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

Appendix 3.J: Odds of Fruit Consumption Moderation by Diet-Health/disease Linked Attitudes (DHLA)

Model 6 Fruit Consumption with DHLA

Will fruit consumption vary with increasing diet-health/disease linked (DHLA) food attitudes? Model 6 results confirmed that diet-health/disease linked attitudes (DHLA) was a significant predictor of fruit consumption, when controlling for all other predictors entered into the model. The odds of meeting the recommended fruit consumption guidelines were one and a half times significantly greater with DHLA food attitudes included. When gender was added to the model, the odds of males meeting the vegetable recommendations were significantly less than females. In the subsequent interaction between gender and DHLA, the odds of males with high DHLA scores were shown to remain significantly less than females with high DHLA scores (see Tables J.1 to J.6 for results of each stage of the model built). Model 6.1 (Table J.1) shows of the 207 individuals within 50 families who either met the NHMRC daily fruit consumption recommendations (e.g., > 2 serves per day) or did not (e.g., < 2 serves per day), higher diet-health/disease linked attitudes (DHLA) food attitude scores were significant predictors of fruit consumption (DHLA coefficient = 0.41, SE = 0.20, p = .04, odds ratio = 1.51, 95% CI = 1.02 to 2.25). Generation was not a significant predictor of fruit consumption (Lang & Secic, 2006).

Table J.1
Model 6.1
Fruit Multiple Logistic Regression all predictors entered together.

		Fruit recommendations met? [d] (serves per day) *N* = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	*p*	
Intercept		-1.36	0.26	0.02	3.25	.29	
Generation							
	Child	0.002	1.00	0.39	2.56	.99	
	Parent	-0.68	0.51	0.22	1.17	.11	
	Grand	0.00[c]	1.00				
Food Attitudes							
	DHLA[e]	0.41	1.51	1.02	2.25	**.04**	SE = 0.20

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

In Model 6.2 (Table J.2) with the addition of gender there was no significant improvement made to the model. DHLA remained a significant predictor of fruit consumption, (DHLA coefficient = 0.40, SE = 0.20, p = .04, odds ratio = 1.49, 95% CI = 1.01 to 2.20) (Lang & Secic, 2006).

Table J.2
Model 6.2
Fruit Multiple Logistic Regression all predictors entered together.

		Fruit recommendations met? [d] (serves per day) *N* = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	*p*	
Intercept		-1.23	0.29	0.03	3.43	.33	
Generation							
	Child	0.02	1.02	0.40	2.61	.97	
	Parent	-0.67	0.51	-1.52	0.18	.97	
	Grand	0.00[c]	1.00				
Gender							
	Male	-0.20	0.82	0.46	1.47	.51	
	Female	0.00[c]					
Food Attitudes							
	DHLA[e]	0.40	1.49	1.01	2.20	**.04**	SE = 0.20

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

In Model 6.3 (Table J.3) there was a significant interaction in gender, when the gender-by-DHLA interaction was added to the model. The odds of males meeting the recommendations for fruit consumption was significantly lower than that of females (Male coefficient = -5.30, SE = 1.71, p = .002, odds ratio = 0.005, 95% CI = 0.00 to 0.14). The interaction coefficient = 0.89, SE = 0.30, p = .003, odds ratio = 2.43, 95% CI = 1.35 to 4.38. Generation and DHLA were not significant predictors of fruit consumption (Lang & Secic, 2006)[67].

[67] Refer to Model 6.3. When calculating the adjusted odds according to DHLA at the mean (6.01), as well as one standard deviation above (6.01 + 0.91 = 6.92) and below the mean (6.01 – 0.91 = 5.10), the resultant slope shows the rate at which DHLA influences males compared to females by using the following calculation:
1.Using the Mean DHLA=6.01, SD=0.91, and exp = 2.71828 [bracketed number is to the power of n]
Female Adjusted Odds: exp[0.87 + (0.05 x 6.01)] = exp[1.17]=3.22 therefore = 2.718281.17 = 3.22
Male Adjusted Odds: exp[(0.87-5.30) + (0.05+0.89)(6.01)]=exp[1.21]=3.35 = 2.718281.21 = 3.35
2. If we use DHLA= 5.10 (1 SD below the mean), Female A_Odds: exp[1.12]=3.06 therefore Male A_Odds: exp[0.27]=1.31.
3. If we use DHLA=6.92 (1 SD above the mean) Female A_Odds: exp[1.22]=3.39; Male A_Odds: exp[2.15]=8.58; (Coxe et al., 2009; Hubbard et al., 2010; Sandifer, 2007). Hence the slope remains constant and small for females (0.05), whereas the slope is steeper for males (0.94) and with a one standard deviation increase in DHLA the odds increased from 1.31 to 8.58 times more likely to meet fruit consumption recommendations than females. This can be interpreted as DHLA being more important to males than females. Factors other than DHLA would therefore influence females meeting the fruit consumption recommendations.

Table J.3

Model 6.3

Fruit Multiple Logistic Regression all predictors entered together.

Predictor		Fruit recommendations met? [d] (serves per day) *N* = 207					
		B[a]	Exp(B) [b] OR	95% CI Lower	95% CI Upper	*p*	
Intercept		0.87	2.40	0.13	43.15	.55	
Generation							
	Child	-0.07	0.93	0.36	2.44	.89	
	Parent	-0.70	0.50	0.22	1.14	.10	
	Grand	0.00[c]	1.00				
Gender							
	Male	-5.30	0.005	0.00	0.14	**.002**	SE = 1.71
	Female	0.00[c]	1.00				
Food Attitudes							
	DHL[e]	0.05	1.06	0.67	1.66	.82	
Interaction	Gender*DHLA[e]						
	Male*DHLA[e]	0.89	2.43	1.35	4.38	**.003**	SE = 0.30
	Female*DHLA[e]	0.00[c]					

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

In Model 6.4 (Table J.4) there was a significant gender-by-DHLA result when the interaction generation-by-DHLA was added to the model. Again, overall the odds of males meeting the recommendations for fruit consumption was significantly lower than that of females (male coefficient = -5.28, SE = 1.74, p = .002, odds ratio = 0.005, 95% CI = 0.00 to 0.15). In the interaction with DHLA, with higher DHLA scores, the odds of males meeting the recommendations for fruit consumption was significantly two and a half times greater than that of females, (the interaction coefficient = 0.89, SE = 0.31, p = .004, the odds ratio = 2.44, 95% CI 1.32 to 4.50) (Lang & Secic, 2006).

Table J.4
Model 6.4
Fruit Multiple Logistic Regression all predictors entered together.

		Fruit recommendations met? [d] (serves per day) *N* = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	*p*	
Intercept		-0.87	0.42	0.002	88.42	.75	
Generation							
	Child	2.30	9.93	0.02	>100.00	.48	
	Parent	1.35	3.85	0.006	>100.00	.68	
	Grand	0.00[c]	1.00				
Gender							
	Male	-5.28	0.005	0.00	0.15	**.002**	SE = 1.74
	Female	0.00[c]					
Food Attitudes							
	DHLA[e]	0.35	1.42	0.59	3.43	.44	
Interaction	Gender*DHLA[e]						
	Male*DHLA[e]	0.89	2.44	1.32	4.50	**.004**	SE = 0.31
	Female*DHLA[e]	0.00[c]	1.00				
Interaction	Generation*DHLA[e]						
	Child*DHLA[e]	-0.41	0.66	0.22	1.98	.46	
	Parent*DHLA[e]	-0.35	0.71	0.25	2.03	.46	
	Grand*DHLA[e]	0.00[c]	1.00				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

Scatter plots confirmed the direction of interaction results interpretation (see Figures J.1 and J.2). The two figures aid in the interpretation of the results and show firstly, the effect of

gender on the relationship between DHLA and fruit consumption of the whole sample (N = 216); and secondly the relationship between DHLA and fruit consumption of the whole sample when controlling for family nesting (N = 50) but without gender.

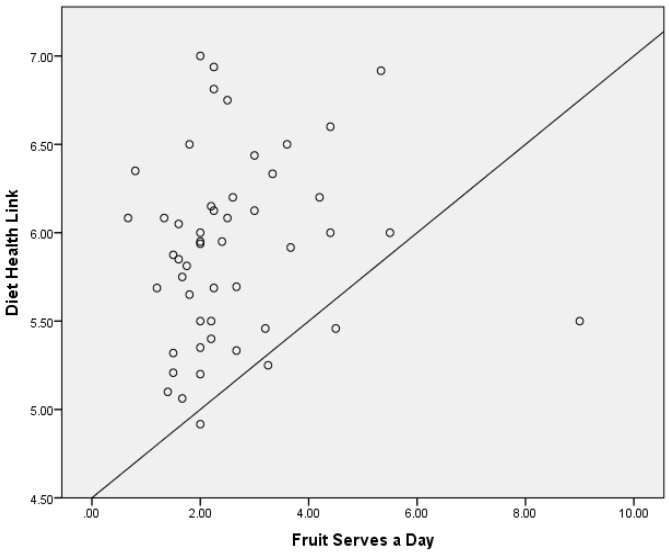


Figure J.1. The scatter plot represents the family nested sample, *N* = 50 and shows the direction of the relationship between food attitudes and fruit consumption for appropriate results interpretation.

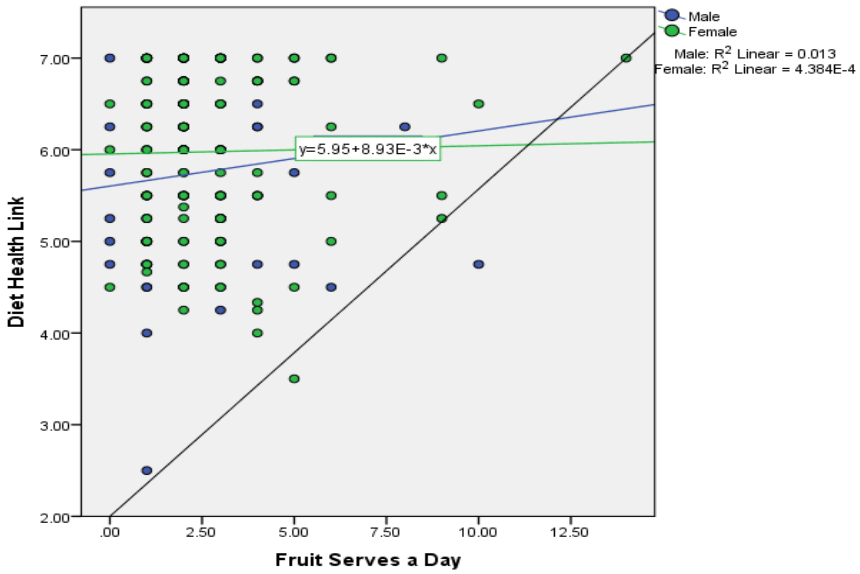


Figure J.2. The scatter plot is of the total sample N = 207 and shows the direction of the interaction between gender and food attitudes for appropriate results interpretation.

Model 6.5 (Table J.5) results are similar to those in Model 6.4 (Table J.4) although the odd ratio is larger in the significant gender-by-DHLA interaction.

Table J.5
Model 6.5
Fruit Multiple Logistic Regression all predictors entered together.

Predictor		Fruit recommendations met? [d] (serves per day) N = 207 B[a]	Exp(B)[b] OR	95% CI Lower	95% CI Upper	p	
Intercept		0.26	1.30	0.003	>100.00	.93	
Generation							
	Child	1.03	2.80	0.003	>100.00	.77	
	Parent	0.91	2.48	0.001	>100.00	.81	
	Grand	0.00[c]	1.00				
Gender							
	Male	-6.72	.001	0.00	0.05	**.000**	SE = 1.90
	Female	0.00[c]					
Food Attitudes							
	DHL[e]	0.19	1.21	0.45	3.25	.71	
Interaction	Gender*DHLA[e]						
	Male*DHLA[e]	1.04	2.83	1.48	5.42	**.002**	SE = 0.33
	Female*DHLA[e]	0.00[c]	1.00				
Interaction	Generation*DHLA[e]						
	Child*DHLA[e]	-0.27	0.76	0.24	2.40	.64	
	Parent*DHLA[e]	-0.28	0.64	0.76	0.23	.64	
	Grand*DHLA[e]	0.00[c]	1.00				
Interaction	Generation*Gender						
	Sons	1.24	3.47	3.45	0.64	.15	
	Daughters	0.00[c]	1.00				
	Fathers	0.23	1.25	0.2	7.90	.81	
	Mothers	0.00[c]	1.00				
	Grandfathers	0.00[c]	1.00				
	Grandmothers	0.00[c]	1.00				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

Table J.6

Model 6.6

Fruit Multiple Logistic Regression all predictors entered together.

Predictor		Fruit recommendations met? [d] (serves per day) N = 207 B[a]	Exp(B)[b] OR	95% CI Lower	Upper	p
Intercept		-1.69	0.19	0.00	141.48	.62
Generation						
	Child	3.19	24.36	0.01	>100.00	.40
	Parent	3.59	36.24	0.01	>100.00	.41
	Grand	0.00[c]	1.00			
Gender						
	Male	-2.40	0.09	0.00	>100.00	.59
	Female	0.00[c]	1.00			
Food Attitudes						
	DHLA[e]	0.51	1.67	0.55	5.06	.36
Interaction	Gender*DHLA[e]					
	Male*DHLA[e]	0.29	1.34	0.29	6.26	.71
	Female*DHLA[e]	0.00[c]	1.00			
Interaction	Generation*DHLA[e]					
	Child*DHLA[e]	-0.64	0.53	0.15	1.91	.33
	Parent*DHLA[e]	-0.72	0.49	0.12	1.97	.31
	Grand*DHLA[e]	0.00[c]	1.00			
Interaction	Generation*Gender					
	Sons	-3.68	0.03	0.00	>100.00	.50
	Daughters	0.00[c]	1.00			
	Fathers	-6.31	0.002	0.00	>100.00	.32
	Mothers	0.00[c]	1.00			
	Grandfathers	0.00[c]	1.00			
	Grandmothers	0.00[c]	1.00			
Interaction	Generation*Gender*DHLA[e]					
	Sons*DHLA[e]	0.86	2.37	0.35	16.06	.38
	Daughters*DHLA[e]	0.00[c]	1.00			
	Fathers*DHLA[e]	1.12	3.06	0.38	24.71	.30
	Mothers*DHLA[e]	0.00[c]	1.00			
	Grandfathers*DHLA[e]	0.00[c]	1.00			
	Grandmothers*DHLA[e]	0.00[c]	1.00			

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group or redundant; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

Model 6.6 was non-significant when the three-way interaction was added.

Appendix 3.K: Odds of Vegetable Consumption Moderation with DHOB

Vegetable consumption moderation by diet-health oriented behaviour (DHOB) food attitudes. GLiM with GEE analysis was used with fruit consumption in the previous model and the vegetable consumption analysis that follows was conducted in exactly the same way. Multiple logistic regression results showed that firstly, with higher DHOB scores the odds of meeting the recommended vegetable serves per day was a significant one and a half times greater than with lower DHOB scores. Secondly, with gender added to the model, the odds of males meeting the recommendations for vegetable consumption were significantly lower than that of females. Lastly, when the gender-by-DHOB interaction was added to the model, the odds of males with higher DHOB scores meeting the recommended serves of vegetables per day were significantly greater than that of females with higher DHOB scores.

Results of Model 7.1 (Table K.1) shows that of 206 individuals within 50 families who either met the NHMRC daily vegetable consumption recommendations (adjusted for age, e.g., > 5 serves per day for adults) or did not (e.g., < 5 serves per day), higher diet-health oriented behaviour (DHOB) food attitude scores were significant predictors of vegetable consumption (DHOB coefficient = 0.51, SE = 0.16, p = .002, odds ratio = 1.67, 95% CI = 1.21 to 2.29). Generation was not a significant predictor of vegetable consumption (Lang & Secic, 2006).

Table K.1

Model 7.1

Vegetables Multiple Logistic Regression all predictors entered together.

Predictor		Veg recommendations met? [d] (serves per day) *N* = 207 B[a]	Exp(B)[b] OR	95% CI Lower	95% CI Upper	*p*	
Intercept		-3.91	0.02	0.003	0.13	**<.001**	
Generation							
	Child	0.32	1.38	0.53	3.42	.49	
	Parent	-0.36	0.70	0.27	1.81	.46	
	Grand	0.00[c]	1				
Food Attitudes							
	DHOB[e]	0.51	1.67	1.21	2.29	**.002**	SE = 0.16

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

In Model 7.2 (Table K.2) gender was added to the model and there was no significant effect. Higher diet-health oriented behaviour (DHOB) food attitude scores were significant predictors of vegetable consumption (DHOB coefficient = 0.50, SE = 0.17, p = .002, odds ratio = 1.65, 95% CI = 1.20 to 2.29). Generation and gender were not significant predictors of vegetable consumption (Lang & Secic, 2006).

Table K.2

Model 7.2

Vegetables Multiple Logistic Regression all predictors entered together.

		Veg recommendations met? [d] (serves per day) N = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	p	
Intercept		-3.79	0.02	0.003	0.16	**<.001**	
Generation							
	Child	0.36	1.43	0.56	3.62	.45	
	Parent	-0.35	0.70	0.27	1.84	.47	
	Grand	0.00[c]	1				
Gender							
	Male	-0.28	0.75	0.37	1.54	.43	
	Female	0.00[c]					
Food Attitudes							
	DHOB[e]	0.50	1.65	1.20	2.29	**.002**	SE = 0.17

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

In Model 7.3 (Table K.3) when the interaction gender-by-DHOB was added to the model, the odds of males meeting the recommendations for vegetable consumption was significantly lower than that of females (male coefficient = -3.65, SE = 1.81, p = .04, odds ratio = 0.03, 95% CI = 0.001 to 0.90). Generation, DHOB and two-way interactions were not significant predictors of vegetable consumption, although gender-by-DHOB approached significance, both male and female were negative (Lang & Secic, 2006).

Table K.3
Model 7.3
Vegetables Multiple Logistic Regression all predictors entered together.

		Veg recommendations met? [d] (serves per day) *N* = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	*p*	
Intercept		-2.41	0.90	-0.008	0.97	**.05**	
Generation							
	Child	0.19	1.21	0.47	3.08	.69	
	Parent	-0.37	0.69	0.27	1.77	.44	
	Grand	0.00[c]	1.00				
Gender							
	Male	-3.65	0.03	0.001	0.90	**.04**	SE = 1.81
	Female	0.00[c]	1.00				
Food Attitudes							
	DHO[e]	0.24	1.27	0.82	1.98	.28	
Interaction	Gender*DHOB[e]						
	Male*DHOB[e]	0.67	1.96	0.96	4.00	**.06**	
	Female*DHOB[e]	0.00[c]					

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

In Model 7.4 [68] (Table K.4) generation-by-DHOB was added to the model. The odds of males meeting the recommendations for vegetable consumption remained significantly lower than that of females (male coefficient = -4.03, SE = 1.89, p = .03, odds ratio = 0.02, 95% CI = 0.00 to 0.72). The gender-by-DHOB interaction showed that with higher DHOB scores, the odds of males meeting the recommendations for vegetable consumption was less than that of females. The interaction generation-by-DHOB approached significance, the interaction coefficient = 0.72, SE = 0.37, *p* = .052, odds ratio = 2.06, 95% CI = 0.99 to 4.28. (Lang & Secic, 2006).

[68] The calculations used to determine Model 7.4 adjusted odds and slopes that confirmed differences between males and females were as follows:
1. Using the Mean DHOB = 4.99, SD=0.84
And exp = 2.71828 [bracketed number is to the power of n]
Female Adjusted Odds: exp[-2.98 + (0.72 x 4.99)] = exp[0.61]= 2.718280.61 = 1.84
Male Adjusted Odds: exp[((-2.98) +(-4.03)) + ((0.36+0.72)x(4.99))]=exp[-1.62]= 2.71828-1.62 = 1.10
2. Using DHOB = 4.15 (1 SD below the mean):
Female Adjusted Odds exp[-2.98 + (0.72 x 4.15)] = exp[0.008]= 2.718280.008 = 1.008
Male Adjusted Odds: exp[((-2.98) +(-4.03)) + ((0.36+0.72)x(4.15))]=exp[-2.52]= 2.71828-2.52 = 0.08
3. If we use DHOB = 4.83 (1 SD above the mean):
Female Adjusted Odds exp[-2.98 + (0.72 x 4.83)] = exp[0.4976]= 2.718280.4976 = 1.645
Male Adjusted Odds: exp[((-2.98) +(-4.03)) + ((0.36+0.72)x(4.83))]=exp[-4.55]= 2.71828-4.55 = 0.01 (Coxe et al., 2009; Hubbard et al., 2010; Sandifer, 2007).

Table K.4

Model 7.4

Vegetables Multiple Logistic Regression all predictors entered together.

Predictor		Veg recommendations met? [d] (serves per day) *N* = 207					
		B[a]	Exp(B)[b] OR	95% CI Lower	95% CI Upper	*p*	
Intercept		-2.98	0.05	0.00	5.73	.22	
Generation							
	Child	-0.67	0.51	0.001	>100.00	.83	
	Parent	4.00	54.57	0.08	>100.00	.23	
	Grand	0.00[c]	1.00				
Gender							
	Male	-4.03	0.02	0.00	0.72	**.03**	SE = 1.89
	Female	0.00[c]					
Food Attitudes							
	DHO[e]	0.36	1.43	0.60	3.40	.42	
Interaction	Gender*DHOB						
	Male*DHOB[e]	0.72	2.06	0.99	4.28	**.052**	SE = 0.37
	Female*DHOB[e]	0.00[c]	1.00				
Interaction	Generation*DHOB						
	Child*DHOB	0.2	1.22	0.37	4.01	.74	
	Parent*DHOB	-0.85	0.43	0.13	1.44	.17	
	Grand*DHOB	0.00[c]	1				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

With the addition of the interaction generation-by-gender in Model 7.5 (Table K.5), the results remained similar to the preceding model. The odds of males meeting the recommendations for vegetable consumption was significantly lower than that of females (male coefficient = -4.81, SE = 2.22, p = .04, odds ratio = 0.008, 95% CI = 0.00 to 0.63). In the gender-by-DHOB interaction the odds of males meeting the recommendations for vegetable consumption was significantly less than that of females. The interaction coefficient = 0.79, SE = 0.38, p = .04, odds ratio = 2.19, 95% CI = 1.04 to 4.63 (Lang & Secic, 2006).

Table K.5
Model 7.5
Vegetables Multiple Logistic Regression all predictors entered together.

		Veg recommendations met? [d] (serves per day) N = 207					
Predictor		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	p	
Intercept		-2.66	0.07	0.001	6.22	.25	
Generation							
	Child	-0.61	0.55	0.001	>100.00	.85	
	Parent	4.06	58.01	0.07	>100.00	.23	
	Grand	0.00[c]	1.00				
Gender							
	Male	-4.81	0.008	0.00	0.63	**.03**	SE = 2.22
	Female	0.00[c]					
Food Attitudes							
	DHO[e]	0.32	1.37	0.59	3.16	3.16	
Interaction	Gender*DHOB						
	Male*DHOB[e]	0.79	2.19	1.04	4.63	**.04**	SE = 0.38
	Female*DHOB[e]	0.00[c]	1.00				
Interaction	Generation*DHOB						
	Child*DHOB	0.12	1.13	0.33	3.85	.85	
	Parent*DHOB	-0.87	0.42	0.12	1.47	.18	
	Grand*DHOB	0.00[c]	1.00				
Interaction	Generation*Gender						
	Sons	1.00	2.72	0.32	23.27	.36	
	Daughters	0.00[c]	1.00				
	Fathers	0.19	1.21	0.13	11.40	.87	
	Mothers	0.00[c]	1.00				
	Grandfathers	0.00[c]	1.00				
	Grandmothers	0.00[c]	1.00				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

With the addition of the final three-way interaction, Model 7.6 in Table K.6 was non-significant.

Table K.6

Model 7.6

Vegetables Multiple Logistic Regression all predictors entered together.

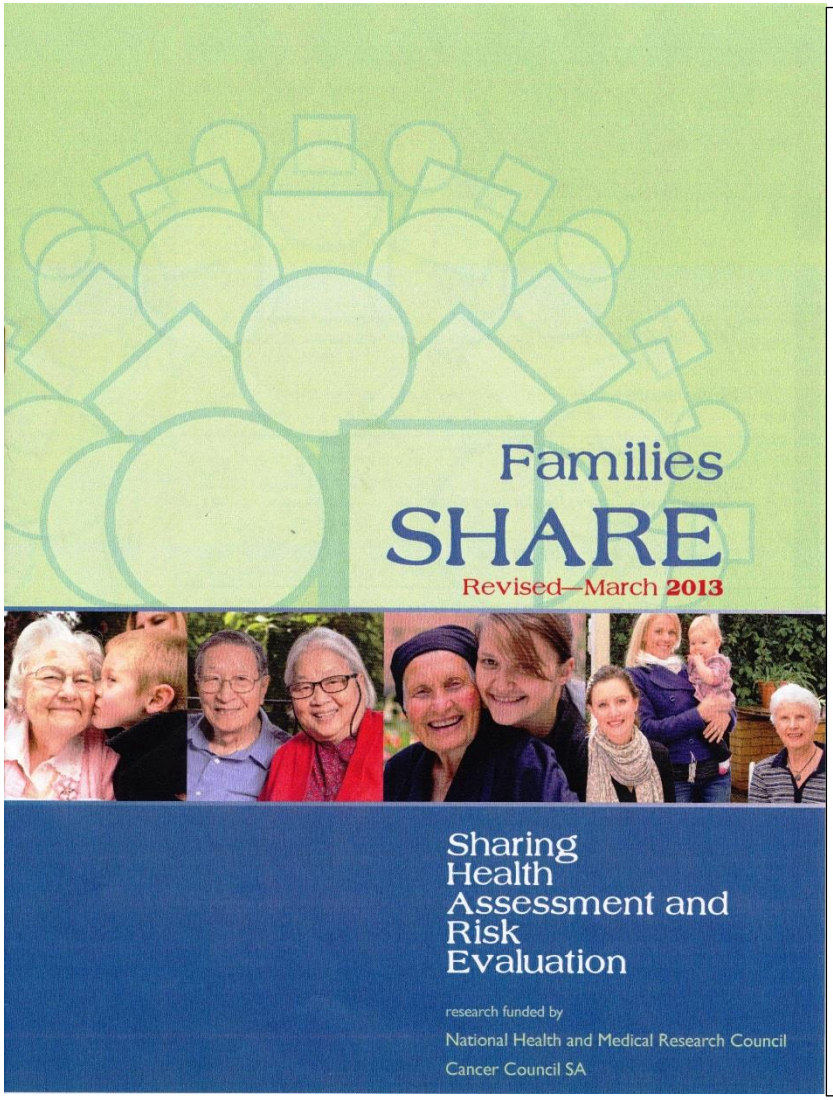
Predictor		Veg recommendations met? [d] (serves per day) *N* = 207				
		B[a]	Exp(B)[b] OR	95% CI Lower	Upper	*p*
Intercept		-3.71	0.02	0.00	2.72	.12
Generation						
	Child	0.42	1.53	0.003	>100.00	.89
	Parent	6.79	888.66	0.35	> 1K	.09
	Grand	0.00[c]	1.00			
Gender						
	Male	2.73	15.34	0.00	> 1K	.66
	Female	0.00[c]				
Food Attitudes						
	DHOB[e]	0.51	1.66	0.71	3.93	.25
Interaction	Gender*DHOB					
	Male*DHOB[e]	-0.73	0.48	0.04	5.94	.57
	Female*DHOB[e]	0.00[c]	1.00			
Interaction	Generation*DHOB					
	Child*DHOB	-0.07	0.93	0.27	3.14	.91
	Parent*DHOB	-1.4	0.25	0.06	1.09	.07
	Grand*DHOB	0.00[c]	1.00			
Interaction	Generation*Gender					
	Sons	-6.50	0.002	0.00	> 1K	.35
	Daughters	0.00[c]	1.00			
	Fathers	-11.29	0.00[c]	0.00	12.33	.11
	Mothers	0.00[c]	1.00			
	Grandfathers	0.00[c]	1.00			
	Grandmothers	0.00[c]	1.00			
Interaction	Generation*Gender*DHOB					
	Sons*DHOB	1.50	4.49	0.26	77.76	.30
	Daughters*DHOB	0.00[c]	1.00			
	Fathers*DHOB	2.30	10.02	0.62	163.09	.11
	Mothers*DHOB	0.00[c]	1.00			
	Grandfathers*DHOB	0.00[c]	1.00			
	Grandmothers*DHOB	0.00[c]	1.00			

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group or redundant; [d] Reference category = No; [e] scale scores 1 – 7, low to high.

CHAPTER 4 APPENDICES

Appendix 4.A: Families SHARE workbook

Reduced in size to 85%



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Families SHARE

Table of contents

Families SHARE

Sharing Health Assessment and Risk Evaluation

What is Families SHARE?

Families SHARE helps families learn how their family health history affects their risk for diseases. It is funded by the National Health and Medical Research Council and Cancer Council SA.

Your family health history plays a part in your risk for many different diseases. In this workbook, we will focus on four diseases: colorectal cancer, breast cancer, type 2 diabetes and heart disease.

How do you get started with your Families SHARE workbook?

We have created a Family Health History Tree for you. This workbook shows you how to read the tree, and learn what it means for you and your family. The workbook has three parts:

Part 1: Example Family Health History Tree

Learn about a Family Health History Tree by looking at an example.

Part 2: Your Family Health History Tree

Look over your own Family Health History Tree (found in the back of this workbook). Update information that has changed and add in any new information.

Part 3: Disease Risk Worksheets and Recommendations

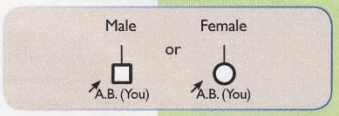
Work through the questions to find your risk for each disease. Learn about tips to lower your risk and protect your family.

How To Read A Family Health History Tree

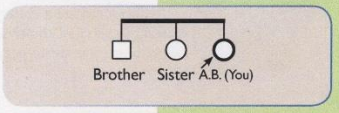
In Part 1 of this booklet, we will introduce you to reading a sample family health history tree. Then in Part 2, you will get a chance to read your own family health history tree.

A family health history tree is a diagram that provides information about you and your first degree and second degree relatives.* Also it shows the people in your family who have, or have had, colorectal cancer, breast cancer, diabetes or heart disease, according to the information that you gave to us. For your privacy, we are only providing the first name of you and your family members. Follow the instructions below to help you read the diagram and what each of the symbols mean.

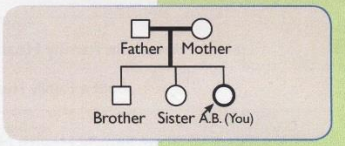
Look for your name (in this example the name is A.B.). If you are male, you will be a square. If you are female, you will be a circle.



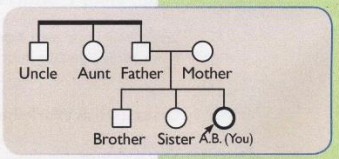
The circles and squares connected to you by a horizontal line above your symbol represent your brothers and/or sisters if you have any. Their names will be below their symbols.



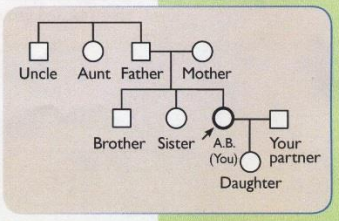
The line going straight up from your line connects to a single line across and forms a T shape that leads to your parents. Their names will be below their symbols.



If your parents have brothers and/or sisters, they will be connected by a horizontal line above them, just like you and your siblings.



Finally, if you have a spouse or partner, that person is connected to you by a horizontal line directly between your symbols. If you have any children with that person, they will be connected to both of you by a vertical line going straight down to their symbol.



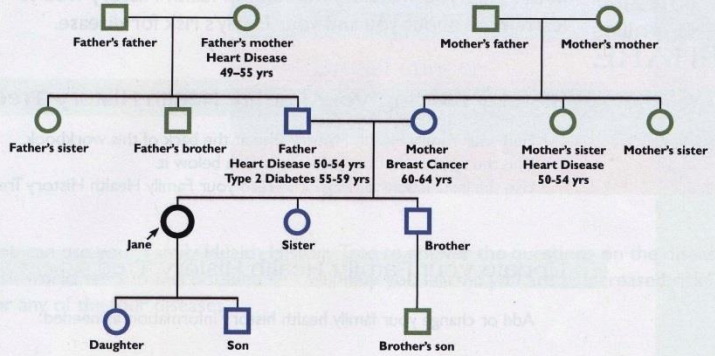
You can follow this same process for another family member by looking for their name and starting there!

*First degree relatives (FDR) are parents, siblings and children. Second degree relatives (SDR) are grandparents, aunts, uncles and grandchildren.

Example Family Health History Tree Part 1

The Family Health History Tree below is about a woman named Jane and her family. All of the information is fictional. Follow Jane's steps to see how she finds out her risk of heart disease.

- The people in **blue** are Jane's first-degree relatives.
- The people in **green** are Jane's second-degree relatives.



Having **heart disease** in her family can affect Jane's risk of heart disease. Let's see how she would use her Family Health History Tree to find her risk.

How many of Jane's first degree relatives listed in the **blue** box have been diagnosed with heart disease before age 55?

Mother
Father
Sister(s)
Brother(s)
Daughter(s)
Son(s)

Enter total number: 1

Is the answer 1 or more? (Circle yes or no) — yes / no

How many of Jane's second degree relatives listed in the **green** box have been diagnosed with heart disease before age 55?

Grandmother(s)
Grandfather(s)
Aunt(s)
Uncles(s)
Nieces(s)
Nephew(s)

Enter total number: 2

Is the answer 1 or more? (Circle yes or no) — yes / no

Because the answer is **yes** to either of these questions, Jane learns that she is at increased risk of heart disease.

Part 2

Your Family Health History Tree

Families SHARE

In this part, you will use your Family Health History Tree to learn more about you and your family's risk for disease.

Start by reading your Family Health History Tree

- Find your Family Health History Tree at the back of this workbook.
- Find the symbol that has your first name below it.
- Use the instructions on Page 2 to read your Family Health History Tree.

Update your Family Health History Tree

Add or change your family health history information, as needed.

Learn about your risk of disease

Use your Family Health History Tree to complete the disease risk worksheets for colorectal cancer, breast cancer, type 2 diabetes and heart disease in Part 3.

Part 3

Disease Risk Worksheets and Recommendations

Next you will learn about:

- Colorectal cancer
- Breast cancer
- Type 2 diabetes
- Heart disease

You can use your Family Health History Tree to answer the questions on the disease risk worksheets in this booklet. This will help you learn if you are at increased risk for any of the four diseases.

Leading a healthy lifestyle and having regular screening for disease are a couple of ways to stay healthy. You will find other tips in this workbook that may help you prevent disease.

Be sure to talk to your doctor about your family health history and your risk of disease!

What is colorectal cancer?

Colorectal cancer – tumours in the large intestine (colon) or the rectum (end of the colon). Most colorectal cancers begin as polyps, which are growths along the colon or rectum.

What are some factors that may increase the risk of colorectal cancer?

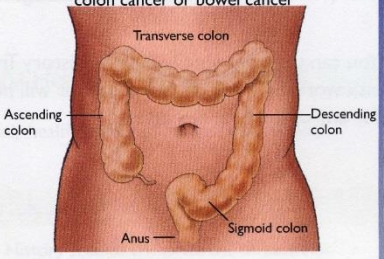
- Eating a diet high in fat and/or processed meats
- Eating a diet low in fibre
- Lack of exercise
- Obesity
- Smoking tobacco
- Heavy alcohol use

Some health screenings for colorectal cancer

- Faecal occult blood test
- Colonoscopy

Did you know...?

Colorectal cancer is also known as colon cancer or bowel cancer



Colon cancer can often be treated if it is found early

Check out these websites for more information:

Bowel Cancer Australia	http://bowelcanceraustralia.org
National Cancer Screening Programs	www.cancerscreening.gov.au
Bowel Screen Australia	http://bowelscreenaustralia.org
Cancer Council SA	www.cancersa.org.au

How does your family health history affect your risk of colorectal cancer?

Use the worksheet on the next page to find out...

What is your risk of colorectal cancer?

How many of your first degree relatives listed in the **blue** box have been diagnosed with colorectal cancer before the age of 55 years?

Mother, Father, Sister(s), Brother(s), Daughter(s), Son(s)

Enter total number ____ yes no

Is the answer **1** or more? (Circle yes or no)

How many of your first degree relatives on the same side of the family listed in the **blue** box have been diagnosed with colorectal cancer at any age?

Mother, Father, Sister(s), Brother(s), Daughter(s), Son(s)

Enter total number here and below ____ yes no

Is the answer **2** or more? (Circle yes or no)

How many of your second degree relatives on the same side of the family listed in the **green** box have been diagnosed with colorectal cancer?

Grandmother(s), Grandfather(s), Aunt(s), Uncles(s), Nieces(s), Nephew(s)

Enter total number here and above ____ **AND** ____ yes no

Is the answer **1** or more **in both**? (Circle yes or no)

If the answer is **yes** to any of these questions, you are at increased risk of colorectal cancer.*

*NHMRC (2005) Clinical Practice Guidelines for the Prevention, Early Detection and Managment of Colorectal Cancer.

If you are at increased risk, talk to your doctor about what you can do to prevent colorectal cancer.

Important:
Some families may have an inherited cancer syndrome. If you or your relatives have had cancer before the age of 50, multiple cancers or recurring cancers, share this information with your doctor to see if genetic testing is recommended for your family.

Some tips that may help prevent and detect colorectal cancer...

Eat fruits and vegetables for vitamins, minerals, fibre and antioxidants:

Adults should eat
- 5 serves of vegetables per day
- 2 serves of fruit per day
- 25-30 grams of fibre

Talk to your doctor about screening:

- Polyps can be removed if found early, before they become cancer

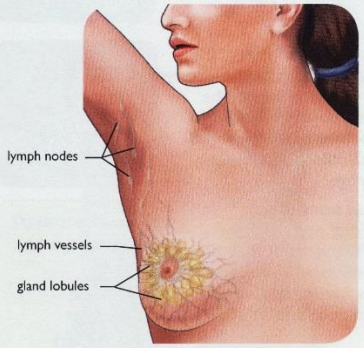
Turn to page 14 and 15 to see other screening and lifestyle recommendations

What is breast cancer?

Breast cancer – tumours that form in the cells of the breasts.

What are some factors that may increase risk of breast cancer?

Heavy alcohol use
Obesity



Some health screenings for breast cancer

Self breast exam
Clinical breast exam
Mammogram

Screening each year can help find breast cancer early. Treatment may be more successful if cancer is found early.

Check out these websites for more information:

National Breast and Ovarian Cancer Centre	www.nbocc.org.au
Breast Cancer Network Australia	www.bcna.org.au
National Cancer Screening Programs	www.cancerscreening.gov.au
BreastScreen SA (ph 13 20 50)	www.breastscreensa.sa.gov.au
Cancer Council SA	www.cancersa.org.au

How does your family health history affect your risk of breast cancer?

Use the worksheet on the next page to find out...

What is your risk of breast cancer?

		Enter total number		
How many relatives listed in the **red** box have been diagnosed with breast cancer?	Any male relatives	Is the answer **1** or more? (Circle yes or no)	yes	no
How many of your first degree relatives listed in the **blue** box have been diagnosed with breast cancer before age 50 years?	Mother Sister(s) Daughter(s)	Is the answer **1** or more? (Circle yes or no)	yes	no
How many of your first degree relatives on the same side of the family listed in the **blue** box have been diagnosed with breast cancer?	Mother Sister(s) Daughter(s)	Is the answer **2** or more? (Circle yes or no)	yes	no
How many of your second degree relatives on the same side of the family listed in the **green** box have been diagnosed with breast cancer, with one diagnosed before the age of 50 years?	Grandmother(s) Aunt(s) Niece(s)	Is the answer **2** or more? (Circle yes or no)	yes	no

If the answer is **yes** to any of these questions, you are at increased risk of breast cancer.*

*NHMRC (1999) Clinical Practice Guidelines Familial Aspects of Cancer: A Guide to Clinical Practice.
National Breast and Ovarian Cancer Centre (2010) Advice about Familial Aspects of Breast Cancer and Epithelial Ovarian Cancer: A Guide for Health Professionals.

If you are at increased risk, talk to your doctor about what you can do to prevent breast cancer.

Important:
Some families may have an inherited cancer syndrome. If you or your relatives have had cancer before the age of 50, multiple cancers or recurring cancers, share this information with your doctor to see if genetic testing is recommended for your family.

Some tips that may help prevent and detect breast cancer...

If you don't drink alcohol, don't start:

- Limit alcohol to no more than two standard drinks per day

Talk to your doctor about screening:

- Lumps can be removed if found early
- Cancer found early can be treated with more success

Turn to page 14 and 15 to see other screening and lifestyle recommendations

What is type 2 diabetes?

Type 2 diabetes is a long-term condition resulting from high levels of sugar in the blood.

What are some factors that may increase risk of type 2 diabetes?

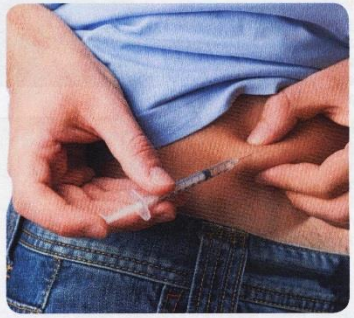
- Lack of exercise
- Obesity
- High blood pressure
- High cholesterol
- Diabetes during pregnancy

Some health screenings for type 2 diabetes

- Blood sugar test
- Blood pressure test
- Cholesterol test

Did you know...?

Insulin is a hormone that controls sugar levels in the blood. Type 2 diabetes occurs when there is a problem with insulin in the body.



Check out these websites for more information:

Diabetes Australia	www.diabetesaustralia.com.au
Dept. of Health & Ageing Type 2 Diabetes Risk Assessment tool (AUSDRISK)	www.ausdrisk.com.au
Health Insite	www.healthinsite.gov.au

How does your family health history affect your risk of type 2 diabetes?

Use the worksheet on the next page to find out...

What is your risk of type 2 diabetes?

How many of your first degree relatives listed in the **blue** box have been diagnosed with type 2 diabetes?

- Mother
- Father
- Sister(s)
- Brother(s)
- Daughter(s)
- Son(s)

Enter total number

Is the answer **1** or more? (Circle yes or no)

yes **no**

If the answer is **yes** to this question, you are at increased risk of type 2 diabetes.*

*NHMRC & Diabetes Australia (2009) National Evidence Based Guidelines for Case Detection and Diagnosis of Type 2 Diabetes

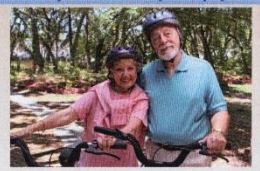
If you are at increased risk, talk to your doctor about what you can do to prevent type 2 diabetes.

Important:

If you are **over the age of 40** you may wish to talk to your doctor. If you are **under the age of 40** use the tips below to reduce your risk.

Some ethnic groups may be more at risk than others. If you are **Aboriginal, Torres-Strait Islander, Maori descent, from Asia, the Middle-East, North Africa or Southern Europe,** you may be at higher risk for type 2 diabetes.

Some tips that may help prevent and detect type 2 diabetes...



Be physically active!

- Try to be active for at least 30 minutes most days of the week
- Take the stairs, walk, swim, garden, etc.

Talk to your doctor about screening:

- Regular blood sugar, blood pressure and cholesterol testing can help find a problem before it becomes type 2 diabetes

Turn to page 14 and 15 to see other screening and lifestyle recommendations

What is heart disease?

Heart disease develops when blood vessels that bring blood and oxygen to the heart become more narrow.

What are some factors that may increase risk of heart disease?

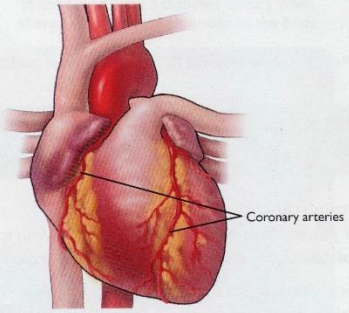
- Diabetes
- Smoking
- Lack of exercise
- Obesity
- Stress
- High cholesterol
- High blood pressure
- Drug abuse

Some health screenings for heart disease

- Blood pressure test
- Blood sugar test
- Cholesterol test
- Waist circumference and body mass index

Did you know...?

Heart disease is also known as coronary heart disease and coronary artery disease



Plaque (made of cholesterol and other substances) can get stuck on the blood vessel walls and reduce blood flow.

Check out these websites for more information:

Heart Foundation www.heartfoundation.org.au

How does your family health history affect your risk of heart disease?

Use the worksheet on the next page to find out...

What is your risk of heart disease?

How many of your first degree relatives listed in the **blue** box have been diagnosed with heart disease before the age of 55?

Mother
Father
Sister(s)
Brother(s)
Daughter(s)
Son(s)

Enter total number

Is the answer 1 or more? (Circle yes or no)

yes **no**

How many of your second degree relatives listed in the **green** box have been diagnosed with heart disease before the age of 55?

Grandmother(s)
Grandfather(s)
Aunt(s)
Uncles(s)
Nieces(s)
Nephew(s)

Enter total number

Is the answer 1 or more? (Circle yes or no)

yes **no**

If the answer is **yes**, you are at increased risk of heart disease.*

* National Vascular Disease Prevention Alliance (2012). Guidelines for the Management of Absolute Cardiovascular Disease Risk.

If you are at increased risk, talk to your doctor about what you can do to prevent heart disease.

Important:

If you are **over the age of 45** you may wish to talk to your doctor. If you are **under the age of 45** use the tips below to reduce your risk.

Some tips that may help prevent and detect heart disease...

Quit smoking or don't start:

- One year after quitting, your added risk of heart disease drops down to half of a smoker's risk

Talk to your doctor about screening:

- Regular blood sugar, blood pressure and cholesterol testing can help find a problem before it becomes heart disease

Turn to page 14 and 15 to see other screening and lifestyle recommendations

Healthy Recommendations

Now that you have a better understanding of your family's health history, there are a few things that you can do to reduce your disease risk. This page offers tips for adding healthy habits to your daily life.

Be sure to share what you have learned with your family!

LIMIT or AVOID ALCOHOL

Experts recommend no more than 2 drinks per day for men and women.

If you don't drink alcohol, don't start.

- Women who are planning pregnancy, are pregnanat or breast feeding, not drinking is the safest option because alcohol can harm a developing baby.
- Anyone who has a problem controlling how much alcohol they drink should not drink at all.

EAT FRUITS & VEGETABLES

Eat plenty of fruits and vegetables.

Adults should try to eat at least 5 serves of vegetables and 2 serves of fruit every day.

- See http://www.gofor2and5.com.au/ to learn more.
- Eat different coloured fruits and vegetables each day, especially dark green, orange and yellow foods.
- There are many ways to add fruits and vegetables to your meals. For example, you can put sliced bananas on top of cereal, add slices of green and red capsicum to cheese pizza or put slices of tomato on cheese sandwiches.

EAT FIBRE

Make sure you get enough dietary fibre: eat a variety of plant foods and whole grain foods.

About 25-30 grams of fibre should be consumed daily.

- Fibre can be found in plant foods, such as cereals, vegetables, fruit, dried peas, beans, lentils and nuts.
- To increase your fibre, try replacing white breads and cereal with more bran and whole grain varieties, and eat raw fruit and vegetables whenever possible.
- Also drink plenty of fluids because this helps in gaining the benefits from fibre.

DON'T SMOKE

Quit smoking or continue to avoid smoking.

If you smoke, medication and counselling can help you quit.

Make a plan and set a quit date. Tell your family, friends, and co-workers you are quitting and ask for their support.

If you are pregnant and smoke, quitting now will help prevent health problems for you and your baby.

BE PHYSICALLY ACTIVE

Be active every day in as many ways as you can. Even reducing or breaking up the time you spent sitting down has important health benefits.

Try to get 30 minutes of exercise that makes you puff or an hour of something gentler on most days of the week. Be as active as you can!

- Exercise helps build and maintain healthy bones, muscles and joints and makes you feel better.
- Your 30 minutes of exercise each day can be done all at once or broken up throughout the day.
- Breaking up or reducing the time that you sit every day can make you healthier.

Current Screening Recommendations

Colorectal Cancer

Cancer Council SA

www.cancersa.org.au

National Cancer Screening Program
Information Line 1800 118 868
www.cancerscreening.gov.au

Average risk

- Faecal occult blood testing (FOBT) every second year from age 50 years.
- Your doctor may consider sigmoidoscopy every 5 years from age 50 years.

Increased risk

- Your doctor may consider colonoscopy at 5 year intervals from age 50 years or 10 years younger than the earliest diagnosis in the family.
- If your doctor suspects an inherited cancer syndrome, referral to a familial cancer service for further risk assessment, genetic counselling, possible genetic testing and guidance in cancer screening may be considered.

Breast Cancer

Cancer Council SA

www.cancersa.org.au

BreastScreenSA: 13 20 50
www.breastscreensa.sa.gov.au

- Be 'Breast Aware' by familiarising yourself with the normal look and feel of your breasts.
- Visit your doctor promptly if you notice any breast changes.
- Women aged 50–69 years without breast symptoms are eligible for free breast screening through BreastScreen SA.
- If at increased risk your doctor may recommend more frequent screening and assessment. Women aged 40–49 and over 70 are still eligible for free breast screening.

Type 2 Diabetes

Diabetes AUSTRALIA

www.diabetesaustralia.com.au

- Visit www.ausdrisk.com.au to complete the free online risk assessment tool and discuss your results with your doctor.
- Risk assessment should begin at age 40 (and age 18 in Aboriginal and Torres Strait Islanders).
- If at average risk reassess every 3 years. If at increased risk reassess every year or as often as recommended by your doctor.

Heart Disease

Heart Foundation™

www.heartfoundation.org.au

Average risk

- After initial assessment by your doctor, risk for heart disease should be reviewed every 2 years from age 45 (and age 35 in Aboriginal and Torres Strait Islanders).

Increased risk

- After initial assessment by your doctor, risk for heart disease should be reviewed every 6–12 months (or as often as recommended by your doctor) from age 45 (and age 35 in Aboriginal and Torres Strait Islanders).

Please discuss your family health history with your doctor

Sharing Your Family Health History

Hopefully, after going through this workbook you will feel more comfortable reading your family health history tree. This booklet is for you to keep. Please feel free to share what you learned here today with your family and friends. Remember that your family health history changes over time, so be sure to add to your Family Health History Tree as you learn more about your family's health.

We hope that you will talk to your doctor about any questions you have. Your doctor is the best person to look over your family health history and discuss how to improve your health and reduce your risk of disease.

* Please note: Recommendations and risk criteria may change over time or differ between Australian States, Territories and their organisations. This booklet is current as at March 2013 and is based on National Health and Medical Research Council publications. The NHMRC is the peak body for developing health advice for the Australian community http://www.nhmrc.gov.au

Where do you go from here?

This workbook is just a start. Here are some important next steps:

- Share this information with your doctor.
- Share this information with your family. Add new family health history information to the tree.

Do you have other diseases in your family that are not in this workbook?

- For information on other types of cancer please visit the Cancer Council SA website *www.cancersa.org.au* or phone Cancer Council Helpline 13 11 20.
- Visit HealthInsite *http://www.healthinsite.gov.au* a government website providing access to a wide range of up to date and quality assessed information on a variety of disease, health and lifestyle topics.

Families
SHARE

Appendix 4.B: Questionnaire Adult Version

ID:

'Families, Food & Eating' Research Study

Families SHARE Questionnaire[69]

For _____ to complete

This questionnaire will ask you questions about yourself, your family, and your exercise and eating habits. It also asks a few questions on your perceived risk of disease. Please answer the questions as honestly as possible **within the next two weeks**. If you have any queries, please do not hesitate to contact one of us:

Donna Hughes (ph: 7221 8436)
Kate Rhodes (ph: 7221 8447)

[69] This thesis was part of a larger Project that measured a number of lifestyle behaviors as outcome variable, however, only the dietary variables are reported in this thesis.

ID:

When you have finished the questionnaire, please detach this front page and post the rest of the questionnaire back to us in the reply-paid envelope provided.

Flinders UNIVERSITY

Cancer Council SA

Date: _____

Section 1
Lifestyle

First of all, in this section, we will ask you some questions about your lifestyle, including your current level of physical activity, the food that you eat and any alcohol that you may drink.

Many of these questions ask you to think about the things that you did and ate during the last 7 days. Try to respond as accurately as possible, even if this week was not a normal or typical week for you.

25. Do you smoke tobacco, including cigarettes, a pipe or cigars?

☐ 1 Yes, I currently smoke cigarettes, a pipe, or cigars
☐ 2 Yes, but I smoke less than I did 6 months ago
☐ 3 No, I used to smoke
☐ 4 No, I have never smoked (or I have smoked less than 100 cigarettes, pipes or cigars in my lifetime)

26. During the last 7 days, on how many days did you do vigorous physical activities, such as heavy lifting, digging in the garden, climbing upstairs, fast bicycling, aerobics, or running? Think about only those physical activities that you did for at least 10 minutes at a time.

☐ No vigorous physical activity
(go to question 3)

☐ _____days per week
(go to question 2a)

2a. On average, how much time on one of those days did you usually spend doing vigorous physical activities? _____ hours per day _____ minutes per day

27. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate physical activities such as carrying light loads, sweeping, washing windows, raking in the garden, or bicycling at a regular pace? Please do not include walking.

☐ No moderate physical activity (go to question 4) ☐ _____ days per week (go to question 3a)

3a. On average, how much time on one of those days did you usually spend doing moderate physical activities? _____ hours per day _____ minutes per day

28. During the last 7 days, on how many days did you walk for at least 10 minutes at a time from place to place?

☐ No periods of walking for 10 minutes at a time (go to question 5) ☐ _____ days per week (go to question 4a)

4a. On average, how much time on one of those days did you usually spend walking?

_____ hours per day _____ minutes per day

29. During the last week, on a weekday (Mon-Fri), how much time per day did you usually spend sitting?

_____ hours _____ minutes per day

30. During the last week, on a weekend day (Sat-Sun), how much time per day did you usually spend sitting?

_____ hours _____ minutes per day

31. Please indicate how often you participated in physical activity as a family during the last 7 days (e.g., jogging, walking, team sports)?

_____ times

5a. On average, how long did you spend on each activity?

_____ hours _____ minutes

32. In the past week, how many serves of fruit did you eat each day?

1 serve of fruit is equivalent to 1 medium sized piece of fruit (e.g., apple, orange, mango, mandarin, banana, pear, peach), 2 small pieces of fruit (e.g., apricots, kiwifruit, plums, figs), 8 strawberries, or ½ cup of fruit juice.

_____ serves of fruit per day

33. In the past week, how many serves of vegetables did you eat each day?

1 serve of vegetables is equivalent to 1 medium potato, or ½ a medium sweet potato, or ½ cup of dark green leafy vegetables (e.g., cabbage, spinach, broccoli, or brussel sprouts), or 1 cup of other

vegetables (e.g., lettuce, beans, lentils, peas, zucchini, cucumber, mushrooms).

_____ serves of vegetables per day

34. In the past week, how many times did you eat meals that were bought from fast food outlets such as McDonalds, Hungry Jacks, Pizza Hut, KFC, Red Rooster, hamburger, pizza or fish and chip shops?
_____ times last week

35. In the past week, how many times did you eat snacks such as a chocolate bar, a piece of cake, a packet of chips/Twisties/corn chips, ice cream, 3-4 sweet biscuits?
_____ times last week

36. On average, in the past week, on how many days did you drink alcohol?

_____ days

37. On average, how many standard drinks did you have on days that you drank alcohol?

(please refer to standard drinks guide below)

_____ standard drinks per day that I drank alcohol

SPARKLING WINE	WINE	LIGHT BEER	REGULAR BEER	FORTIFIED WINE	SPIRITS
100 mL	100 mL	425 mL	285 mL	60 mL	30 mL
13% alc/vol	13% alc/vol	2.7% alc/vol	4.9% alc/vol	20% alc/vol	40% alc/vol

(Information from Drug & Alcohol Services SA)

ID:

Section 2
Intended Lifestyle Changes

All of us are living lifestyles that aren't perfect. We often think about ways to be more healthy and active. You may or may not have made changes to aspects of your lifestyle over the last 6 months. Even if you have made some changes, you may still be considering further changes.

At the beginning of 2012, were you thinking about changing any aspects of your lifestyle?

☐ Yes, I was already thinking about changing some aspects of my lifestyle.

If yes, please indicate which aspects:

☐ Increasing my fruit and vegetable consumption ☐ Increasing my fibre consumption

☐ Increasing my level of physical activity ☐ Decreasing my alcohol consumption

☐ Reducing or stopping smoking

☐ No, I had not even thought about changing any aspects of my lifestyle.

Thinking about your lifestyle right now, are you currently contemplating changing any aspects?

Tick the response below that best applies (1 only)

☐ Yes, I am thinking about changing some aspects of my lifestyle

If yes, please indicate the time period you are intending to make these changes

☐ In the next month ☐ In the next 3 months (or more)

☐ No, I am happy with things the way they are

☐ I have already made changes to some aspects of my lifestyle in the last 6 months

If so, please indicate which aspects of your lifestyle you have changed in the last 6 months:

☐ Increased my fruit and vegetable consumption ☐ Increased my fibre consumption

☐ Increased my level of physical activity ☐ Decreased my alcohol consumption

☐ Reduced or stopped smoking

ID:

What aspects of your lifestyle would like to change (if any)? Please respond to each item.

1. Fruit and vegetables

☐$_1$ Yes, I would like to <u>increase</u> my fruit and vegetable consumption

☐$_2$ No, I am happy with my current fruit and vegetable consumption

If yes, how confident are you that you will increase your fruit and vegetable consumption within the next year? (circle the number that best represents your answer)

1 2	**3**	**4**	**5**	**6**	**7**
Not at all confident			Moderately confident		Very confident

2. Fibre

☐$_1$ Yes, I would like to <u>increase</u> my fibre consumption

☐$_2$ No, I am happy with my current fibre consumption

If yes, how confident are you that you will increase your fibre consumption within the next year? (circle the number that best represents your answer)

1 2	**3**	**4**	**5**	**6**	**7**
Not at all confident			Moderately confident		Very confident

3. Physical Activity

☐$_1$ Yes, I would like to <u>increase</u> my level of physical activity

☐$_2$ No, I am happy with my current level of physical activity

If yes, how confident are you that you will increase your level of physical activity within the next year? (circle the number that best represents your answer)

1 2	**3**	**4**	**5**	**6**	**7**
Not at all confident			Moderately confident		Very confident

ID:

4. Alcohol

☐$_1$ Yes, I would like to <u>decrease</u> my alcohol consumption

☐$_2$ No, I am happy with my current level of alcohol consumption

☐$_3$ Not applicable – I don't drink alcohol

If yes, how confident are you that you will decrease your alcohol consumption within the next year? (circle the number that best represents your answer)

1 2	**3**	**4**	**5**	**6**	**7**	
Not at all confident			Moderately confident			Very confident

5. Smoking

☐$_1$ Yes, I would like to <u>reduce or stop</u> smoking

☐$_2$ No, I am happy with my current level of smoking

☐$_3$ Not applicable – I don't smoke

If yes, how confident are you that you will reduce or stop smoking within the next year? (circle the number that best represents your answer)

1 2	**3**	**4**	**5**	**6**	**7**	
Not at all confident			Moderately confident			Very confident

Section 3

Food Attitudes

In this section, we are interested in your attitudes towards food. Food is a more important aspect of life for some people than it is for others. Please read each statement below and circle the number that best represents how much you agree or disagree with it. Remember, there are no right or wrong answers, just your opinion.

Here is an example:

	Strongly Disagree		Disagree		Agree		Strongly Agree
I think that food gives us energy	1	2	3	4	5	6	7

In this example, you can see that I have circled number 7. This means that, for me, I strongly agree with the statement.

Now, complete the questions below by indicating your opinions.

		Strongly Disagree		Disagree		Agree		Strongly Agree
1	I have fond memories of family food occasions	1	2	3	4	5	6	7
2	I rarely think about the long-term effects of my diet on health	1	2	3	4	5	6	7
3	I am concerned about being overweight	1	2	3	4	5	6	7
4	I feel guilty when I overeat	1	2	3	4	5	6	7
5	Diet can have a big effect on heart disease	1	2	3	4	5	6	7
6	I eat low-fat food on a regular basis	1	2	3	4	5	6	7
7	Diet can have a big effect on obesity	1	2	3	4	5	6	7
8	Taste is more important to me than nutrition	1	2	3	4	5	6	7
9	I am a healthy eater	1	2	3	4	5	6	7
10	I am currently on a diet	1	2	3	4	5	6	7
11	I eat fast food on a regular basis	1	2	3	4	5	6	7
12	I consciously hold back at meal time, so as not to gain weight	1	2	3	4	5	6	7

		Strongly Disagree		Disagree		Agree		Strongly Agree
13	Diet can have a big effect on good health	1	2	3	4	5	6	7
14	Money spent on food is well spent	1	2	3	4	5	6	7
15	Diet can have a big effect on cancer	1	2	3	4	5	6	7
16	Enjoying food is one of the most important things in my life	1	2	3	4	5	6	7
17	I think about food in a positive way	1	2	3	4	5	6	7
18	I think natural, organic foods are better for you than commercially grown/processed foods	1	2	3	4	5	6	7
19	In my opinion, my thighs are too fat	1	2	3	4	5	6	7
20	I control my caloric intake	1	2	3	4	5	6	7
21	I think natural, organic foods taste better than commercially grown/processed foods	1	2	3	4	5	6	7

Section 5

Disease Risk

We are all becoming more aware that some chronic diseases (such as heart disease, diabetes and cancer) can have a link to aspects of our lifestyle, as well as other factors.

We are interested in your views about your own risk of developing one of these diseases. For some people, this is a topic that they think about a lot, but for others, it may not seem as important to them at this time.

1. Have you talked about your health risk for lifestyle-related chronic diseases (e.g., colorectal/bowel cancer, breast cancer, heart disease OR diabetes) to any of the following people in the past 6 months (tick all that apply):

   ☐ GP

   ☐ Family member (please list any family members you may have talked to about your health risk)

   _____

   ☐ Friends (please list any friends you may have talked to about your health risk)

   _____

   ☐ Other (please specify)_____

Now, please indicate your opinions by circling the best response on the scale next to

each statement below.

		Not at all likely			Moderately likely			Extremely likely
2	In the next 6 months, how likely are you to talk to your doctor about your health risk for lifestyle-related chronic diseases (e.g., colorectal cancer (also known as bowel or colon cancer), breast cancer, heart disease OR diabetes)?	1	2	3	4	5	6	7
3	In the next 6 months, how likely are you to talk to your family about your health risk for lifestyle-related chronic diseases (e.g., colorectal cancer (also known as bowel or colon cancer), breast cancer, heart disease OR diabetes)?	1	2	3	4	5	6	7

Please now think about the diseases listed below. How likely do you think it is that <u>you</u> could develop one or more of these diseases in your lifetime? Indicate your answer by circling the best response. If you don't know or have no opinion about this, circle DK.

		Not at all likely			Moderately likely			Extremely likely	I don't know	Not Applicable
4	Colorectal cancer	1	2	3	4	5	6	7	DK	NA
5	Breast cancer	1	2	3	4	5	6	7	DK	NA
6	Heart disease	1	2	3	4	5	6	7	DK	NA
7	Diabetes	1	2	3	4	5	6	7	DK	NA

Note. If you have already received a diagnosis for one of these diseases, please select NA

Now please think about how likely it is that <u>your child/children</u> could develop one or more of these diseases in their lifetime? Indicate your answer by circling the

best response.

		Not at all likely			Moderately likely			Extremely likely	I don't know	Not Applicable
8	Colorectal cancer	1	2	3	4	5	6	7	DK	NA
9	Breast cancer	1	2	3	4	5	6	7	DK	NA
10	Heart disease	1	2	3	4	5	6	7	DK	NA
11	Diabetes	1	2	3	4	5	6	7	DK	NA

Now, we'd like you to think about lifestyle factors and how they might contribute to the risk of each disease listed. Please indicate your opinions below by circling the response that is best for you.

		Not at all		A little		A fair bit		A great deal
12	How much do you think the risk of developing **heart disease** is contributed to by the following:							
	eating habits	1	2	3	4	5	6	7
	alcohol consumption	1	2	3	4	5	6	7
	physical inactivity	1	2	3	4	5	6	7
	smoking	1	2	3	4	5	6	7
	genetic factors	1	2	3	4	5	6	7
13	How much do you think the risk of developing **diabetes** is contributed to by the following:							
	eating habits	1	2	3	4	5	6	7
	alcohol consumption	1	2	3	4	5	6	7
	physical inactivity	1	2	3	4	5	6	7
	smoking	1	2	3	4	5	6	7
	genetic factors	1	2	3	4	5	6	7
14	How much do you think the risk of developing **breast cancer** is contributed to by the following:	1	2	3	4	5	6	7
	eating habits							
	alcohol consumption	1	2	3	4	5	6	7
	physical inactivity	1	2	3	4	5	6	7
	smoking	1	2	3	4	5	6	7
	genetic factors	1	2	3	4	5	6	7
15	How much do you think the risk of developing **colorectal cancer** is contributed to by the following:	1	2	3	4	5	6	7
	eating habits							
	alcohol consumption	1	2	3	4	5	6	7
	physical inactivity	1	2	3	4	5	6	7
	smoking	1	2	3	4	5	6	7
	genetic factors	1	2	3	4	5	6	7

Section 7

Family Health History Evaluation

[70]

As part of your participation in this study, you received the Families SHARE package including a Family Health History Tree detailing your risk for developing heart disease, diabetes, breast cancer and colorectal cancer. This section asks you some questions about your thoughts on the Families SHARE package.

1. **Using the Family Health Package, were you able to assess your own degree of risk for each disease?**
   ☐ Yes ☐ No

   1a. Are you at increased risk for:

Colorectal cancer	☐ Yes	☐ No	☐ Don't know
Breast cancer	☐ Yes	☐ No	☐ Don't know
Heart disease	☐ Yes	☐ No	☐ Don't know
Diabetes	☐ Yes	☐ No	☐ Don't know

2. **Have you shared information from the Families SHARE package with any of the following people (tick all that apply):**
   ☐ GP

   ☐ Other health care provider (e.g., nurse, specialist, pharmacist). If so, please indicate who:

   _____

   ☐ Family member (Please list the names of any family members you have shared your family health history with):

   _____

   _____

   _____

   _____

   ☐ Friends (Please list the names of any friends you have shared your family health history with):

   _____

   _____

   _____

   ☐ Other (please specify)_____

   _____

[70] *Note.* Only the experimental condition received Section 7 - Family Health History Evaluation

_____

_____

3. After receiving your Family Health History Tree did you update it in any way?

☐ Yes ☐ No

If yes, please list any changes you made:

_____

_____

_____

Were any changes based on the advice of someone else, and if so, who?

_____

4. In the last 6 months, have you participated in any screening behaviour for any of the following diseases?

Colorectal cancer	☐ Yes	☐ No
Breast cancer	☐ Yes	☐ No
Heart disease	☐ Yes	☐ No
Diabetes	☐ Yes	☐ No

Section 8

Demographics

In our research, to make it more comprehensive, we need to be able to describe the people who have participated. If the question has a blank, please fill in the blank. If the question has a list of choices, please mark the box with the response that best reflects your answer.

1. **Age:** _____ (years)

2. **Gender:** ☐$_1$ *Male* ☐$_2$ *Female* (tick one)

3. **Height:** _____ (cm)

4. **Weight:** _____ (kg) (Pregnant women should indicate weight prior to pregnancy)

5. **How many people live with you at your current residence?** _____

6. **How many children do you have?** _____

7. **Your current marital status is:**
   ☐$_1$ Single ☐$_4$ Separated
   ☐$_2$ Married ☐$_5$ Divorced
   ☐$_3$ Living as married ☐$_6$ Widowed

8. **Do you currently have health insurance?** ☐$_1$ Yes ☐$_2$ No

Thank you very much for your time!

Please detach the front page now and return the completed questionnaire to us in the reply-paid envelope provided.

Appendix 4.C: Questionnaire Child Version

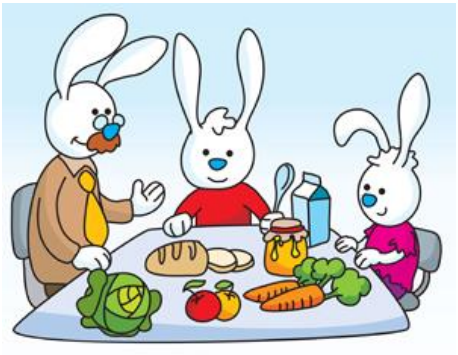
‘Families, Food & Eating’ Research Study

Child Questionnaire

For participants aged 7 – 17 years

For _____ to complete

This questionnaire will ask you questions about yourself, your family, and your exercise and eating habits. Please answer the questions as honestly as you can. If you need any help, please ask an adult nearby.



Date: _____

Section 1
Lifestyle

First, in this section, we will ask you some questions about your lifestyle.

Many of these questions ask you to think about the things that you did and ate during the last 7 days. Try to respond as accurately as possible, even if this week was not a normal or typical week for you.

Physical Activity

1. **During the last 7 days, on how many days did you do vigorous physical activities, such as heavy lifting, digging in the garden, climbing up stairs, fast bicycling, aerobics, or running? Think about only those physical activities that you did for at least 10 minutes at a time.**

   ☐0 No vigorous physical activity (go to question 2)

   ☐1 _____days per week (go to question 1a)

   1a. On average, how much time on one of those days did you usually spend doing vigorous physical activities? _____ hours per day _____ minutes per day

2. **Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate physical activities such as carrying light loads, sweeping, washing windows, raking in the garden, or bicycling at a regular pace? Please do not include walking.**

   ☐0 No moderate physical activity (go to question 2)

   ☐1 _____ days per week (go to question 2a)

   2a. On average, how much time on one of those days did you usually spend doing moderate physical activities? _____ hours per day _____ minutes per day

3. **During the last 7 days, on how many days did you walk for at least 10 minutes at a time from place to place?**

   ☐0 No periods of walking for 10 minutes at a time (go to question 17)

   ☐1 _____ days per week (go to question 3a)

   3a. On average, how much time on one of those days did you usually spend

walking? _____ hours per day _____ minutes per day

4. **Please indicate how often you participated in physical activity with your family during the last 7 days (e.g., jogging, walking, team sports).**

   _____ times

   4a. **On average, how long did you spend on each activity?** _____ hours _____ mins

Seated Activity

5. **During the last week, on a weekday (Mon-Fri), how much time per day did you usually spend sitting?** _____ hours _____ minutes per day

6. **During the last week, on a weekend day (Sat-Sun), how much time per day did you usually spend sitting?** _____ hours _____ minutes per day

Below are some questions on ***sedentary or seated behaviour*** such as computer/video game use, watching television, sitting listening to music, sitting and talking on the phone, homework/studying, reading for recreation etc.

7. **On the average day, about how many hours per day do you do the following when you are not at school? (if less than 1 hour, please estimate how many minutes you spend per day)**

	Weekdays (e.g., Mon-Fri)	**Sat or Sun**
Homework	_____hours/mins	_____hours/mins
Watch TV/videos/DVDs	_____hours/mins	_____hours/mins
Use the internet / play computer games *(do not include computer use for homework)*	_____hours/mins	_____hours/mins
Use Facebook, Twitter, or Myspace,	_____hours/mins	_____hours/mins
Use other social networking sites (please specify.....)	_____hours/mins	_____hours/mins
Sit and listen to music	_____hours/mins	_____hours/mins
Sit and read for recreation	_____hours/mins	_____hours/mins
Sit and talk on the phone	_____hours/mins	_____hours/mins
Sit and do another activity (please specify.....)	_____hours/mins	_____hours/mins

Eating

We'd now like to ask you some questions about food that you might eat in a typical week. Thinking over the last week, try to answer as accurately as you can, even if this week was not a normal or typical week for you.

8. In the past week, how many serves of fruit did you eat each day?

1 serve of fruit is equivalent to 1 medium sized piece of fruit (e.g., apple, orange, mango, mandarin, banana, pear, peach), 2 small pieces of fruit (e.g., apricots, kiwifruit, plums, figs), 8 strawberries, or ½ cup of fruit juice.

_____ serves of fruit per day

9. In the past week, how many serves of vegetables did you eat each day?

1 serve of vegetables is equivalent to 1 medium potato, or ½ a medium sweet potato, or ½ cup of dark green leafy vegetables (e.g., cabbage, spinach, broccoli, or brussel sprouts), or 1 cup of other vegetables (e.g., lettuce, beans, lentils, peas, zucchini, cucumber, mushrooms).

_____ serves of vegetables per day

10. In the past week, how many times did you eat meals that were bought from fast food outlets such as McDonalds, Hungry Jacks, Pizza Hut, KFC, Red Rooster, hamburger, pizza or fish and chip shops?
_____ times last week

11. In the past week, how many times did you eat snacks such as a chocolate bar, a piece of cake, a packet of chips/Twisties/corn chips, ice cream, 3-4 sweet biscuits?
_____ times last week

Section 2
Intended Lifestyle Changes

People of all ages often think about ways to be more healthy and active. You may or may not have made changes to aspects of your lifestyle over the last 6 months. Even if you have made some changes, you may still be considering further changes.

At the beginning of 2012, were you thinking about changing any aspects of your lifestyle?

☐ Yes, I was already thinking about changing some aspects of my lifestyle.

If yes, please indicate which aspects:

☐ Increasing my fruit and vegetable consumption

☐ Increasing my level of physical activity

☐ No, I had not even thought about changing any aspects of my lifestyle.

☐ I don't know

Thinking about your lifestyle right now, are you currently contemplating changing any aspects?

Tick the response below that best applies (1 only)

☐ Yes, I am thinking about changing some aspects of my lifestyle

If yes, please indicate the time period you are intending to make these changes

☐ In the next month ☐ In the next 3 months (or more)

☐ No, I am happy with things the way they are

☐ I have already made changes to some aspects of my lifestyle in the last 6 months

If yes, please indicate which aspects of your lifestyle you have changed in the last 6 months:

☐ Increased my fruit and vegetable consumption

☐ Increased my level of physical activity

What aspects of your lifestyle would like to change (if any)? Please respond to each item.

1. Fruit and vegetables

☐$_1$ Yes, I would like to increase my fruit and vegetable consumption

☐$_2$ No, I am happy with my current fruit and vegetable consumption

If yes, how confident are you that you will increase your fruit and vegetable consumption within the next year? (circle the number that best represents your answer)

1	**2**	**3**	**4**	**5**	**6**	**7**
Not at all confident			Moderately confident			Very confident

2. Physical Activity

☐$_1$ Yes, I would like to increase my level of physical activity

☐$_2$ No, I am happy with my current level of physical activity

If yes, how confident are you that you will increase your level of physical activity within the next year? (circle the number that best represents your answer)

1	**2**	**3**	**4**	**5**	**6**	**7**
Not at all confident			Moderately confident			Very confident

Section 3

Food Attitudes

Food is a more important aspect of life for some people than it is for others. In this section, we are interested in your attitudes towards food. Please read each statement below and circle the number that best represents how much you agree or disagree with it. Remember, there are no right or wrong answers, just your opinion.

Here is an example:

	Strongly		Disagree		Agree		Strongly Agree
I think that food gives us energy	1	2	3	4	5	6	7

In this example, you can see that I have circled number 7. This means that, for me, I strongly agree with the statement. Now, complete the questionnaire below by indicating your opinions.

		Strongly Disagree		Disagree		Agree		Strongly Agree
1	I have fond memories of family food occasions	1	2	3	4	5	6	7
2	I rarely think about the long-term effects of my diet on health	1	2	3	4	5	6	7
3	Diet can have a big effect on heart disease	1	2	3	4	5	6	7
4	I eat low-fat food on a regular basis	1	2	3	4	5	6	7
5	Diet can have a big effect on obesity	1	2	3	4	5	6	7
6	Taste is more important to me than nutrition	1	2	3	4	5	6	7
7	I am a healthy eater	1	2	3	4	5	6	7
8	I eat fast food on a regular basis	1	2	3	4	5	6	7
9	Diet can have a big effect on good health	1	2	3	4	5	6	7
10	Money spent on food is well spent	1	2	3	4	5	6	7
11	Diet can have a big effect on cancer	1	2	3	4	5	6	7
12	Enjoying food is one of the most important things in my life	1	2	3	4	5	6	7
13	I think about food in a positive way	1	2	3	4	5	6	7
14	I think natural, organic foods are better for you than commercially grown/processed foods	1	2	3	4	5	6	7
15	I control my caloric intake	1	2	3	4	5	6	7
16	I think natural, organic foods taste better than commercially grown/processed foods	1	2	3	4	5	6	7

Section 5

Demographics

In our research, to make it more comprehensive, we need to be able to describe the people who have participated. If the question has a blank, please fill in the blank. If the question has a list of choices, please mark the box with the response that best reflects your answer.

17. **Age:** _____ (years)

18. **Gender:** ☐$_1$ *Male* ☐$_2$ *Female* (tick one)

19. **Height:** _____ (cm)

20. **Weight:** _____ (kg)

21. **How many people live with you at your house?** _____

22. **How many brothers or sisters do you have?** _____

Thank you very much for your time!

Please detach the front page now and return the completed questionnaire to us in the reply-paid envelope provided.

Appendix 4.D: Letter of Introduction

Professor Carlene Wilson
CCSA Chair in Cancer Prevention
Flinders Prevention, Promotion and Primary Health Care, Public Health
School of Medicine
Flinders University
GPO Box 2100
Adelaide SA 5001

Tel: 08 72218473
Carlene.Wilson@flinders.edu.au

CRICOS Provider No. 00114A

LETTER OF INTRODUCTION

Dear Sir/Madam,

I hold the position of CCSA Chair in Cancer Prevention in the School of Medicine at Flinders University. I am undertaking research leading to the production of publications on the subject of Eating Behaviour in Families across three generations. This research involves a whole family approach across three generations. As such, families incorporating at least one child aged 8-16 years, and at least one grandparent who would be willing to participate, are invited to take part. Altogether, 5 members from your family are needed for the project (e.g., 2 parents, 1 child, & 2 grandparents; or 1 parent, 2 children, 1 aunt, 1 grandparent).

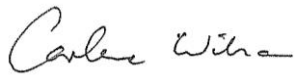
I would be most grateful if you and your family would volunteer to assist in this project. Participation would involve each family member completing a questionnaire on eating and health on two occasions (now and in six months time). The questionnaire will take no more than 30-45 minutes on each occasion. Lastly, as part of the project, all families may be given a Family Health History booklet to read. Altogether, no more than two hours of your time would be required over a 6-month period.

Be assured that any information provided will be treated in the strictest confidence and none of the participants will be individually identifiable in any resulting publications. You, and your family are, of course, free to discontinue participation at any time or to decline to answer particular questions. If you complete the questionnaires online, your data will be sent to a secure, password protected server. Reply-paid envelopes have been provided if you wish to complete the paper-version of the questionnaires.

Please also be advised that the topics of colorectal cancer, breast cancer, cardiovascular disease and diabetes make up part of the questionnaire, so please consider this before you choose to participate. If the questionnaire raises any issues that you would like to discuss, please contact our research team who will refer you on to a free service, or consult your medical practitioner. Any enquiries you may have concerning this project should be directed to me at the address given above or by telephone on 87221 8473, or by email (carlene.wilson@flinders.edu.au).

Thank you for your attention and assistance.

Yours sincerely,

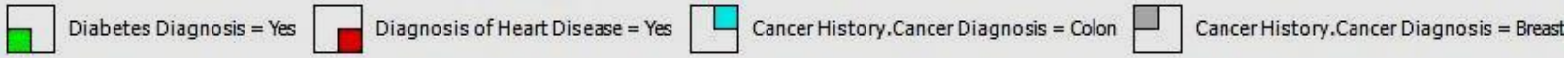


Prof Carlene Wilson
CCSA Chair in Cancer Prevention
School of Medicine

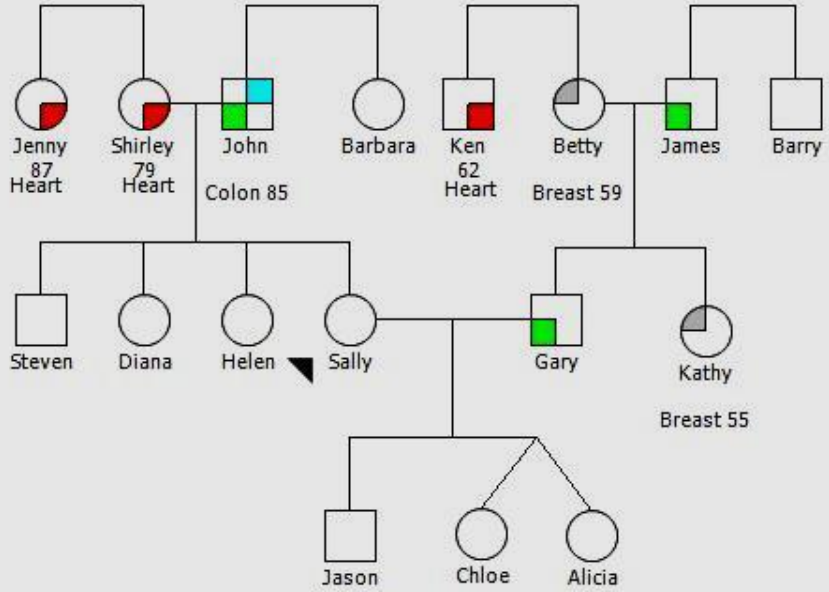
This research project has been approved by the Flinders University Social and Behavioural Research Ethics Committee (Project Number 5514). For more information regarding ethical approval of the project the Executive Officer of the Committee can be contacted by telephone on 8201 3116, by fax on 8201 2035 or by email human.researchethics@flinders.edu.au.

Appendix 4.E: Sample Pedigree

Sample
26/03/2014



*Where relevant, age at diagnosis for heart disease and cancer appears under family member's name



Appendix 4.F: Your Family Health History: Patient and Family Fact Sheet

Reduced in size to 85%

71

Patient and family fact sheet

Your family history

Genes are passed from parent to child, so genetic conditions can run in families.

This is not always the case. It is possible to have a genetic condition that no-one else in the family has.

It is also possible for several members of a family to have a particular condition without the condition being genetic. For example, if everybody in a family smokes, that family can have lots of heart disease and cancer.

Having said that, genes and families go together quite strongly.

So if you're thinking about genes, or about diseases running in the family, one important thing you can do is learn about your family's health history.

When looking closely at genetic conditions that may run in your family, your doctor will want to draw a family tree. He or she will want to know about the person you're talking about (whether that's you or someone in your family), as well as all the parents, grandparents, uncles, aunts, cousins and children, especially those who are blood relatives.

Your doctor will ask a lot of questions in order to examine your family history. Your doctor will want to know:

- All the relationships in your family
- The ages of everyone involved
- Who is alive?
- How old were family members when they died and what did they die of?
- Do they or did they have any medical conditions?
- The names of doctors or hospitals that have cared for affected family members
- What's the ancestry of your family? What country did grandparents and great-grandparents come from?
- Did anybody in the family have any miscarriages, stillborn children or children born with abnormalities?
- Has anybody in the family lived for long periods in a psychiatric institution?
- Has anybody in the family been adopted?
- Has anybody in the family had children with a relative?
- Has anybody in the family been told they have or are a carrier for a genetic condition?
- Has anybody in the family ever been tested for a genetic condition? What was the result?

If you can get answers to some or all of these questions, you and your doctor will be able to make a lot more sense of your family history. If you visit the webpage at **http://www.genetics.edu.au/publications/fhtcons.htm** you can see a sample family history.

[71] Fact Sheet contained within Barlow-Stewart et al. (2007) that was inserted into the Families SHARE workbook.

Contacts and further information

- Your local genetic service, which you can contact through your nearest community health centre, public hospital or health department.
- Australasian Genetic Alliance at **http://www.australasiangeneticalliance.org.au**
- The Centre for Genetics Education at **http://www.genetics.edu.au**
- For other related fact sheets, you can contact the Gene Technology Information Service on **free call Australia-wide 1800 631 276** or email **gtis-australia@unimelb.edu.au** or visit Biotechnology Australia's website at **http://www.biotechnology.gov.au**

Appendix 4.G: Stage of Change Data Preparation

Stage of Change (SoC) Data Preparation

All possible SoC combinations over time were calculated in an Excel table. Prior to running the analyses, each potential SoC outcome was entered into the spreadsheet to make sure all of the stages of change were calculated correctly.

Data preparation. Using the SoC Excel spreadsheet and using the combined T1 and T2 single level data set, four new variables for SoC in healthy dietary behaviours were created. These were baseline and follow up stage of change for fruit and vegetables. Outcomes were coded 1 – 5, pre-contemplation to maintenance SoC for fruit and vegetables. Next, using vertical multilevel longitudinal modeling procedures as used previously, and described in Heck et al., (2014a), the single level data file was restructured to create a vertical data set that included the SoC consumption variables (the two levels appeared as N = 178 x 2 = 356, however 2 horizontal lines were apparent for each individual representing T1 and T2). The newly created vertical multilevel data set included all of the disease risk variables as before, and enabled analysing changes over time that also controlled for family nesting.

Appendix 4.H: Longitudinal Modelling Reasoning in Data Analysis

Longitudinal Modelling Reasoning in Data Analysis

As occurred in Study 2, statistical analysis decisions were guided by Elhai, Calhoun, and Ford (2008). When making multiple comparisons, all modeling analyses adjusted for disproportionate sampling and cluster sampling to avoid over inflating the type 1 error rate (Heck et al., 2014a). Results would normally have been achieved using a within-subjects repeated measures factorial ANOVA (Tabachnick & Fidell, 2013); however, these family data would have violated several assumptions if "classical" statistics were used. Firstly, partially missing data meant the loss of data per measure for each case in ANOVA. Secondly, the nesting effect of families meant that individuals were not independent of each other thus breaching the assumption of sphericity, and thirdly the time elapsed between measurements needed to be constant. As stated in Chapter 3 "classical" statistics refers to Analysis of Variance and tests of multiple regression that rely on the ordinary least-squares criterion (Atkins & Gallop, 2007).

Study 2 used a single level modeling approach that had a horizontal data matrix analysing variables that were aligned side-by-side (in the 'data view' of SPSS version 22). Study 3 analysed data from the same individuals measured at baseline and at follow up. Study 3 data were collected and cleaned using the same procedures as described in Study 2. Time one (T1) data were merged with time two (T2). The combined data file was then restructured to create a single data file with two-levels (N = 178 individuals, 42 families), using procedures as described by Heck, Thomas and Tabata (2014a). Variables were arranged horizontally and vertically to enable family nested data analysis over time. In other words, during data restructuring a "time" index variable was created and each outcome variable (i.e., fruit, vegetables, snacks and fast food) were arranged vertically to represent a score at each measurement time period for each individual. The unique family identifier variable that was used in Study 2 was retained to control for the same family nesting (i.e., within-family

associations between family members of each family). As in Study 2, missing data on a single item within scales did not mean losing the remaining data for that case, therefore maximizing the use of available data. Multilevel modeling incorporated probability distributions that differ from the normal bell shaped curve therefore used multinomial, binomial and Poisson techniques (Heck et al., 2014b). This modeling technique was chosen according to Elhai et al. (2008). A test of negative binomial is indicated when the variance exceeds the mean as occurred with the dependent variables in the single level data. However in the multilevel data, dependent variable distributions were different and were therefore analysed with Poisson and negative binomial analyses according to the guidelines.

SoC variables were computed for healthy food consumption by using the COMPUTE command in SPSS as described in Heck et al. (2014a). Thus, the data file contained four new SoC variables: one each for fruit and vegetable consumption at baseline and follow up with outcomes measured as 1 through to 5 for each stage of change. For more information on how this was computed refer back to Appendix G.

One of the limitations, due to finite computational capabilities within the available computer memory to date, was that only a limited number of independent variables could be entered into each model at any one time, thus decisions were made as to how to best answer each of the research questions in turn, and some compromises had to be made.

Appendix 4.I: Mother risk*Time*Condition Result Tables and Figures

Mother risk*Time*Condition Result Tables and Figures

Table I.1

*Fruit Model 1.2: Mother Risk of any Disease*Time*Condtion, Changes in Consumption Over Time*

Effects	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 1.2								
Condition		ns						
	Treatment-Control	ns						
Time		ns						
	Time 1 - 2	ns						
Mother Disease Risk		ns						
	Diagnosed - Average risk	ns						
	Diagnosed - Above average risk	ns						
	Above average risk - Average risk	ns						
Condition*Mother risk* Time [a]								
Control-Average risk				1, 338			4.11	**.04**
	Control-Average risk- T1 - T2	-1.28	-2.03	1, 338	-2.52	-0.04		**.04**
	Control-Above average risk- T1 - T2	0.38	0.92	1, 338	-0.43	1.19		.36
	Control-Diagnosed- T1 - T2	0.57	0.63	1, 338	-1.23	2.37		**.53**

Note . GLiMM negative binomial vertical multilevel modeling. Model 1.2: -2 log pseudo likelihood = 752.918. [a]Only significant interactions are shown.

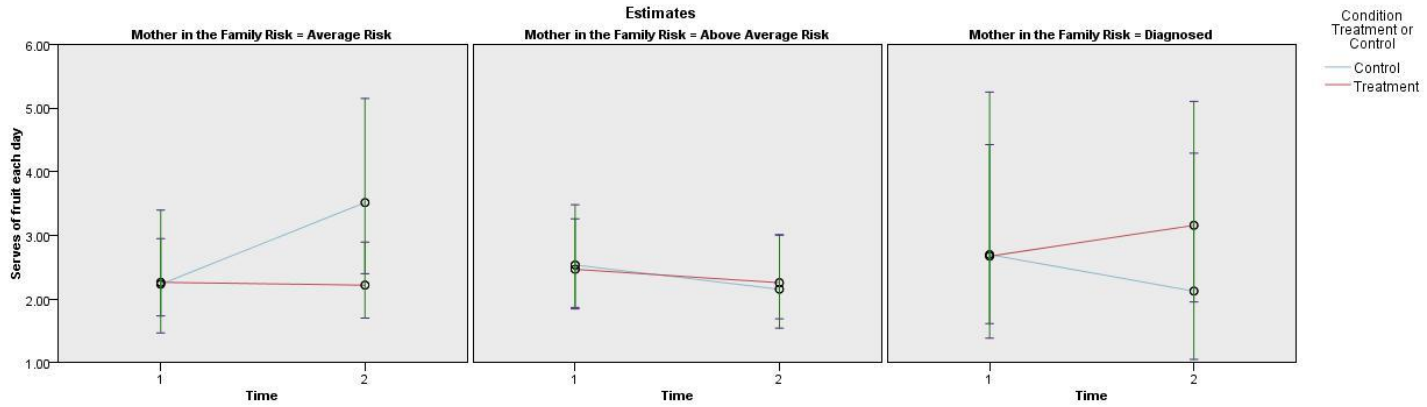


Figure I.1. Significant changes over time if mother is at risk in the family: Average risk controls were significant for fruit consumption changes over time.

Table I.2

*Snacks Model 3.2: Mother Risk of any Disease*Condtion*Time, Changes in Consumption Over Time*

Effects	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 3.2								
Condition		ns						
	Treatment-Control	ns						
Time							4.04	**.045**
	Time 1 - 2	0.84	2.01	1, 339	0.02	1.67		**.045**
Mother Disease Risk		ns						
Condition*Mother risk* Time [a]		ns						

Note . GLiMM negative binomial vertical multilevel modeling. Model 3.2: -2 log pseudo likelihood = 752.918.

[a]Only significant interactions are shown.

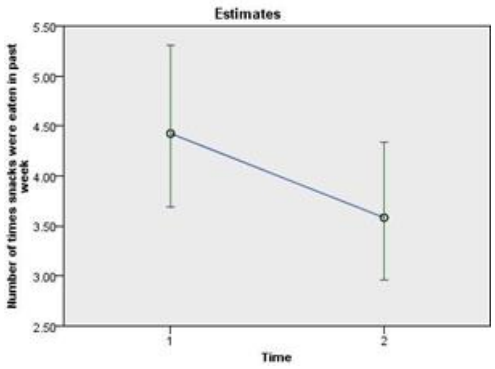


Figure I.2. Significant effect of time for snack consumption if mother is at risk in the family.

CHAPTER 5 APPENDICES

Appendix 5.A: Longitudinal Modeling Healthy and Unhealthy Food Consumption Over Time Between Gender and Generation

Longitudinal models determined differences in healthy and unhealthy food consumption between people distinguished by gender and generation

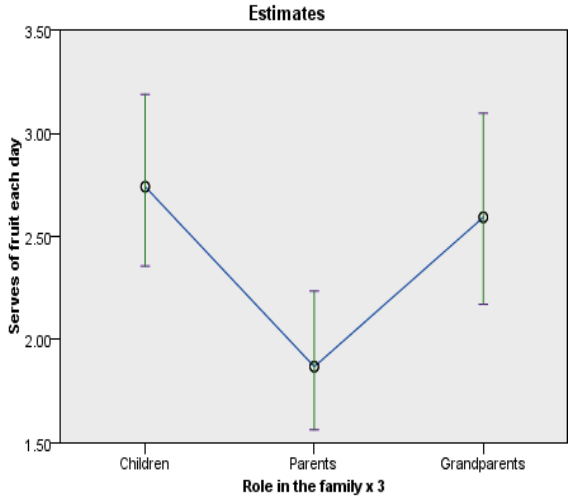


Figure A.1. Fruit serves per day, the figure shows a significant effect of generation.

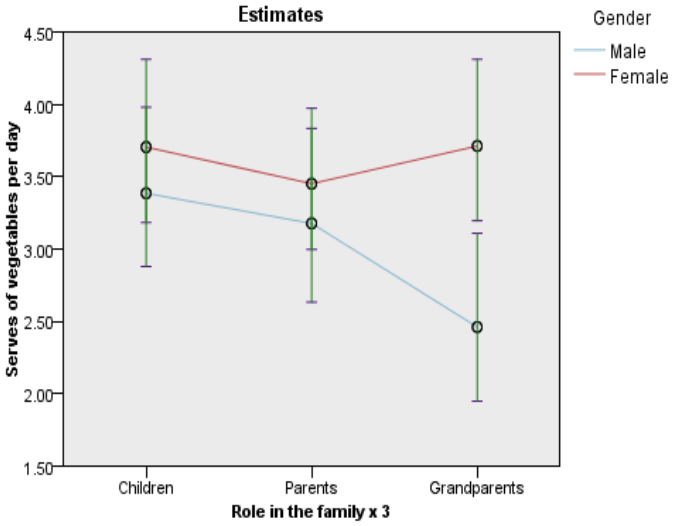


Figure A.2. Vegetables serves per day, significant effect of generation by gender: Females > Males; significant effect of generation by gender sons > grandfathers.

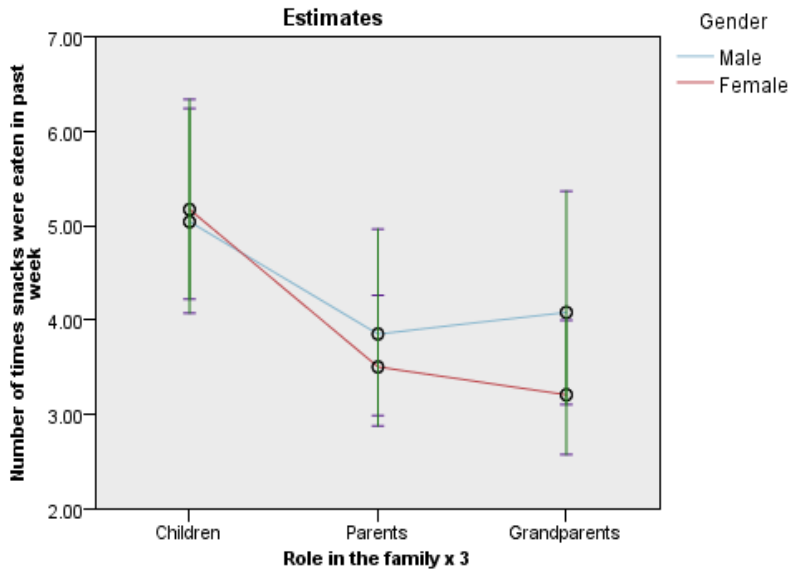


Figure A.3. Snack consumption, significant effect of generation: child > parents, child > grandparents; significant effect of gender*generations: daughters > mothers, daughters > grandmothers.

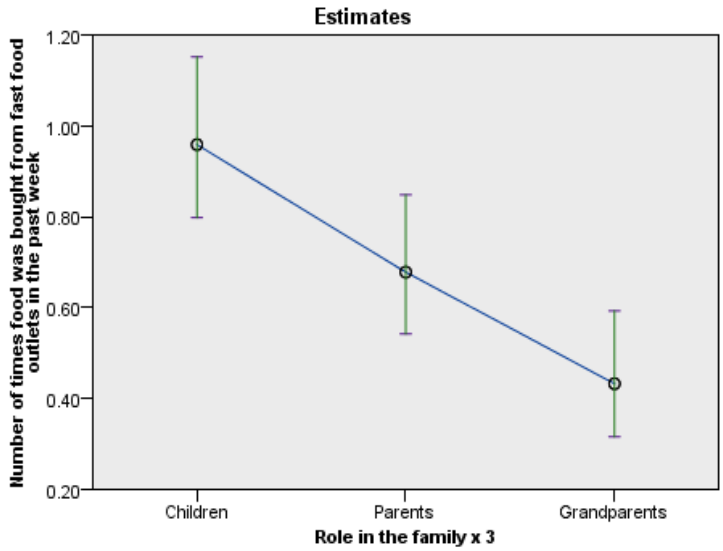


Figure A.4. Fast food consumption. Significant effect of generation: children greater than parents and grandparents.

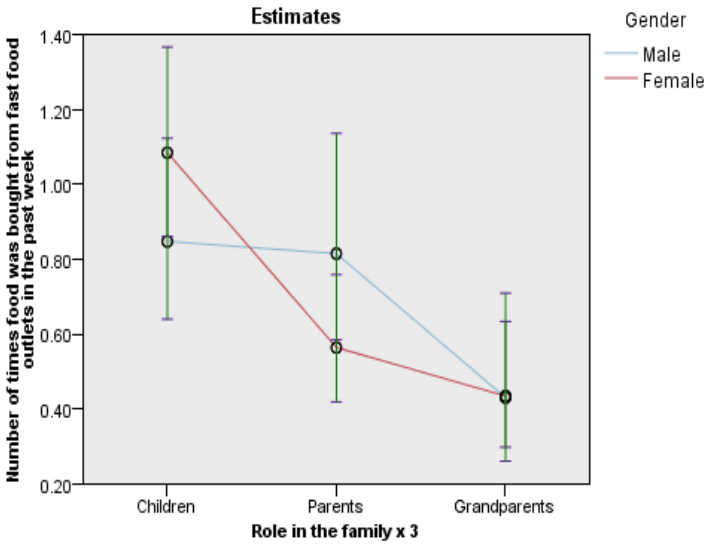


Figure A.5. Fast food consumption. Significant effect of generation by gender sons greater than grandfathers, daughters greater than mothers and grandmothers.

Appendix 5.B: Three Generations by Gender Showing Descriptive Statistics of Healthy Food Consumption at Time One and Time Two.

Table B.1.

Three Generations by Gender Showing Healthy Food Consumption at Time One and Time Two.

Variable	Family Role	Time	*N*	*M*	*SD*	Median	Range	Min
Fruit (serves/day)	Son	T1	30	2.83	2.29	2.00	10.00	0.00
	Son	T2	30	2.50	1.90	2.00	10.00	0.00
	Daughter	T1	32	2.66	1.82	2.00	08.00	0.00
	Daughter	T2	32	3.25	2.75	2.50	12.00	0.00
	Mother	T1	38	2.13	1.19	2.00	05.00	1.00
	Mother	T2	38	1.86	0.95	2.00	04.00	0.00
	Father	T1	22	1.64	1.09	1.50	04.00	0.00
	Father	T2	22	1.59	0.96	1.50	04.00	0.00
	Grandmother	T1	31	2.87	1.84	2.00	09.00	0.00
	Grandmother	T2	31	3.05	2.43	2.00	11.00	1.00
	Grandfather	T1	18	2.94	3.04	2.50	14.00	0.00
	Grandfather	T2	18	2.19	1.14	2.00	05.00	1.00
Veg (serves/day)	Son	T1	30	3.37	2.81	3.00	13.00	1.00
	Son	T2	30	3.45	2.28	3.00	12.00	1.00
	Daughter	T1	32	3.44	2.13	3.00	09.50	0.50
	Daughter	T2	32	3.94	2.91	3.00	13.00	1.00
	Mother	T1	38	3.37	1.23	3.00	05.50	0.50
	Mother	T2	38	3.47	1.24	3.25	05.00	1.00
	Father	T1	22	3.18	1.47	3.00	05.00	1.00
	Father	T2	22	3.09	1.31	3.00	05.00	1.00
	Grandmother	T1	31	3.97	2.54	3.00	13.00	1.00
	Grandmother	T2	31	3.97	2.41	3.00	09.00	1.00
	Grandfather	T1	18	3.17	3.09	2.50	13.00	1.00
	Grandfather	T2	18	2.22	1.25	2.00	04.00	1.00

Appendix 5.C: Correlations between Generations, and between Gender-by-Generations on each Food Consumption Variable

Correlations

Fruit Consumption by Generations

Table C.1.
Spearman's rho Three Generations Fruit Consumption Correlations at Time One and Time Two

Generation	T1 Child	T1 Parent	T1 Grand	T2 Child	T2 Parent	T2 Grand
Child	.	**.283***	.112	.	**.301***	.258
n	41	39	38	41	39	37
Parent		.	-.063		.	.164
n		40	37		40	37
Grand			.			.
n			39			38

Note. * $p < 0.05$, ** $p < .01$ (1-tailed). T1 8% shared varience; T2 9% shared variance.

Fruit Consumption by Generation and Gender

Table C2.

Spearman's rho Three Generations by Gender Showing Fruit Consumption Correlations at Time One and Time Two.

Fruit			Time	One					Time	Two		
Family Role	Son	Daughter	Mother	Father	Grandmother	Grandfather	Son	Daughter	Mother	Father	Grandmother	Grandfather
Son	.	**.655***	.269	.194	**.433***	.377	.	-.152	.239	-.249	**.488***	0.05
n	24	10	23	16	16	13	24	10	22	16	16	13
Daughter		.	**.362***	-.031	-.069	-.460		.	.245	-.172	.295	-.317
n		27	26	14	21	8		27	25	14	20	8
Mother			.	.306	-.057 a	.036 [a]			.	.356	-.301 a	.170 [a]
n			40	22	23	14			39	21	23	13
Father				.	.333 [b]	.000 [b]				.	.000 [b]	.866 [b]
n				22	4	3				22	4	3
Grandmother					.	**.776****					.	.475
n					31	10					30	10
Grandfather						.						.
n						18						18

Note. * $p < 0.05$, ** $p < .01$ (1-tailed), [a] maternal grandparents, [b] paternal grandparents.

Vegetable Consumption by Generations

Table C.3.

Spearman's rho Three Generations Vegetable Consumption Correlations at Time One and Time Two

Family Role	T1 Child	T1 Parent	T1 Grand	T2 Child	T2 Parent	T2 Grand
Child	.	**468****	.032	.	**.385****	.230
n	41	39	38	40	38	37
Parent		.	.026		.	.118
n		40	37		40	37
Grand			.			.
n			39			39

Note. * $p < 0.05$, ** $p < .01$ (1-tailed). T1 22% shared variance; T2 15% shared variance.

Vegetable Consumption by Generation and Gender

Table C.4.

Spearman's rho Three Generations by Gender Showing Vegetable Consumption Correlations at Time One and Time Two.

Vegetables			Time	One					Time	Two		
Family Role	Son	Daughter	Mother	Father	Grandmother	Grandfather	Son	Daughter	Mother	Father	Grandmother	Grandfather
Son	.	**.725****	.599	.338	.042	.186	.	.451	**.513****	.411	.166	0.237
n	24	10	23	16	16	13	24	10	23	16	16	13
Daughter		.	**.595****	-.018	-.185	-.049		.	**.479****	.050	.007	.102
n		27	25	14	21	8		26	24	13	20	8
Mother			.	.021	-.006 [a]	-.101 [a]			.	-.075	-.057 [a]	.265 [a]
n			39	21	22	14			39	21	22	14
Father				.	-.833 [b]	.866 [b]				.	-.316 [b]	.866 [b]
n				22	4	3				22	4	3
Grandmother					.	.337					.	.033
n					31	10					31	10
Grandfather						.						.
n						18						18

Note. * $p < 0.05$, ** $p < .01$ (1-tailed), [a] maternal grandparents' [b] paternal grandparents.

Snack Consumption by Generations

Table C.5.

Spearman's rho Three Generations Snack Consumption Correlations at Time One and Time Two

Generation	T1 Child	T1 Parent	T1 Grand	T2 Child	T2 Parent	T2 Grand
Child	.	.106	-.025	.	.088	.270
n	41	39	38	41	39	37
Parent		.	**.277***		.	**-.356***
n		40	37			37
Grand			.			.
n			39			38

Note. * $p < 0.05$, ** $p < .01$ (1-tailed). T1 8% shared variance; 13% shared variance where childrens goes up grandparents go down.

Snack Consumption by Generation and Gender

Table C.6.

Spearman's rho Three Generations by Gender Showing Snack Consumption Correlations at Time One and Time Two

Snacks		Time One					Time Two					
Relationship	Son	Daughter	Mother	Father	Grandmother	Grandfather	Son	Daughter	Mother	Father	Grandmother	Grandfather
Son	.	.438	.215	-.035	-.174	.017	.	**.593***	.292	-.220	**.533***	0.16
n	24	10	23	16	16	13	24	10	23	15	13	16
Daughter		.	.180	-.063	-.039	-.601		.	.194	-.061	.257	-.325
n		26	25	13	20	8		27	26	14	21	8
Mother			.	.294	**.377*** [a]	-.067 [a]			.	.158	-.270 [a]	.225 [a]
n			40	22	29	14			40	21	23	14
Father				.	.500 [b]	.866 [b]				.	-.738 [b]	.866 [b]
n				22	4	3				21	4	3
Grandmother					.	**.734****					.	-.049
n					31	10					31	10
Grandfather						.						.
n						18						18

Note. * $p < 0.05$, ** $p < .01$ (1-tailed), [a] maternal grandparents' [b] paternal grandparents. T1 14% of shared variance existed between grandmother and mother, and 54% was shared between grandparent partners. T2 35% between siblings, 28% between grandmother-son realtionships.

Fast Food Consumption by Generations

Table C.7.
Spearman's rho Three Generations Fast Food Consumption Correlations at Time One and Time Two

Generation	T1 Child	T1 Parent	T1 Grand	T2 Child	T2 Parent	T2 Grand
Child	.	.163	.034	.	**.352***	.210
n	40	37	37	41	39	37
Parent		.	**.322***		.	.061
n		40	36			36
Grand			.			.
n			39			38

Note. * $p < 0.05$, ** $p < .01$ (1-tailed). T1 10% shared variance between parents and grandparents; T2 12% shared variance between parents and children.

Fast Food Consumption by Generation and Gender

Table C.8

Spearman's rho Three Generations by Gender Showing Fast Food Consumption Correlations at Time One and Time Two.

Fast Food			Time	One					Time	Two			
Relationship	Son	Daughter	Mother	Father	Grandmother	Grandfather	Son	Daughter	Mother	Father	Grandmother	Grandfather
Son	.	**.879****	.183	.231	.227	.085	.	**.799****	**.442***	.212	.052	.337
n	24	10	22	16	16	13	24	10	23	16	15	13
Daughter		.	.095	.059	.085	.283		.	**.376***	.177	.136	**.722***
n		26	25	13	20	8		27	26	14	20	8
Mother			.	.289	.341 [a]	.577 [a]			.	**.432***	.041 [a]	**.519*** [a]
n			39	22	22	14			40	22	22	14
Father				.	. [bc]	. [bc]				.	.0 [b]	. [bc]
n				22	4	3				22	4	3
Grandmother					.	. [c]					.	-.167
n					31	10					30	10
Grandfather						.						.
n						18						18

Note. * $p < 0.05$, ** $p < .01$ (1-tailed), [a] maternal grandparents' [b] paternal grandparents, [c] insufficient data or too many zero scores. T1 77% of the variance was shared between siblings. T2 64% between siblings, 20% between mother-son, 14 % between mother-daughter, 19% between parent partners, 52% between grandfather-granddaughter, 27% between grandfather-mother relationships.

Appendix 5.D: Mother's Disease Risk Results

Results – Mother's Disease Risk, Condition, Generation, and Food Consumption[72]

Table D.1.

*Fruit Model 1.3: Mother Risk of any Disease*Condtion*Generation, Changes in Consumption Over Time*

Effects	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 1.3				(17, 332)			1.75	**.033**
Condition		ns						
	Treatment-Control	ns						
Generation				(2, 332)			7.56	**.001**
	Child - Parent	1.02	3.65	(1, 332)	0.35	1.69		**.001**
	Child - Grand	0.14	0.44	(1, 332)	-0.48	0.76		.660
	Parent - Grand	-0.88	-2.82	(1, 332)	-1.58	-0.18		**.010**
Mother's Disease Risk		ns						
	Diagnosed - Average risk	ns						
	Diagnosed - Above average risk	ns						
	Above average risk - Average risk	ns						
Condition*Mother risk* Generation [a]								
Treatment-Above average risk				(2, 332)			3.98	**.020**
	Treatment-Above average risk- Child - Parent	0.65	1.66	(1, 332)	-0.24	1.54		.200
	Treatment-Above average risk- Child - Grand	-0.77	-1.49	(1, 332)	-1.87	0.33		.200
	Treatment-Above average risk- Parent - Grand	-1.43	-2.74	(1, 332)	-2.67	-0.18		**.019**

Note . GLiMM negative binomial vertical multilevel modeling. Model 1.3: -2 log pseudo likelihood = 736.854. [a] only significant interactions are shown.

[72] Vegetable and snack consumption results were non-significant and are not shown.

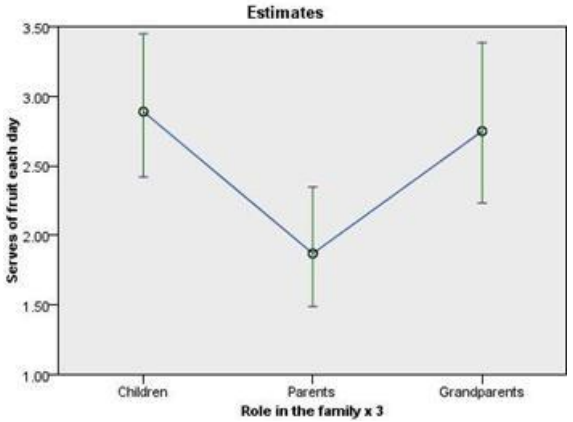


Figure D.1. Fruit consumption with differences between generations shown.

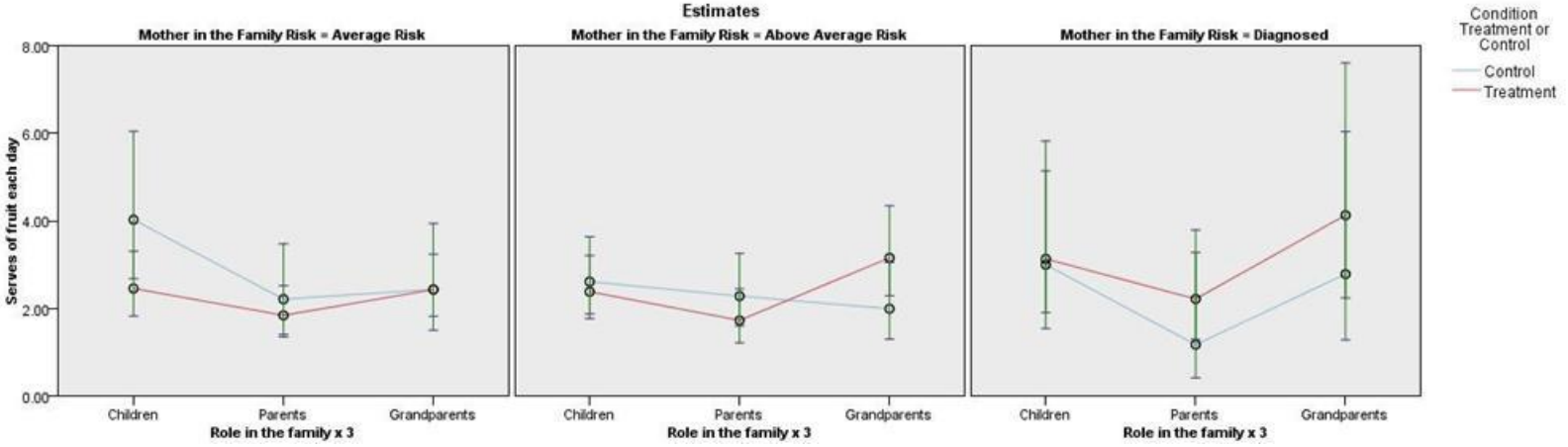


Figure D.2. Fruit consumption with interaction differences shown when a mother in the family is at risk or diagnosed with chronic disease, treatment and control groups are also shown.

Table D.2.

*Fast Food Model 4.3: Mother Risk of any Disease*Condition*Generation, Changes in Consumption Over Time*

Effects	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 4.3				(17, 329)			2.03	**.010**
Condition								ns
	Treatment-Control							ns
Generation				(2, 329)			8.13	**<.001**
	Child - Parent	0.35	2.66	(1, 329)	0.60	0.71		**.016**
	Child - Grand	0.57	4.02	(1, 329)	0.23	0.90		**.016**
	Parent - Grand	0.22	-1.39	(1, 329)	-0.07	0.43		.170
Mother's Disease Risk				(2, 329)			0.45	.640
	Diagnosed - Average risk							ns
	Diagnosed - Above average risk							ns
	Above average risk - Average risk							ns
Interaction Condition*Family risk* Generation [a]								
Treatment-Average risk				(2, 329)			5.07	**.007**
	Treatment-Average risk - Child - Parent	0.12	0.45	(1, 329)	-0.36	0.57		.660
	Treatment-Average risk - Child - Grand	0.56	2.7	(1, 329)	0.06	1.06		**.022**
	Treatment-Average risk - Parent - Grand	0.45	2.41	(1, 329)	0.03	0.88		**.033**
Treatment-Above average risk								ns
Treatment-Diagnosed				(2, 329)			3.10	**.046**
	Treatment-Diagnosed- Child - Parent	0.9	2.03	(1, 329)	0.02	1.91		.090
	Treatment-Diagnosed- Child - Grand	1.14	2.45	(1, 329)	0.02	2.25		**.045**
	Treatment-Diagnosed- Parent - Grand	0.23	0.65	(1, 329)	-0.47	0.94		.520

Note . GLiMM negative binomial vertical multilevel modeling. Model 4.3: -2 log pseudo likelihood = 978.741 [a] only significant interactions are shown.

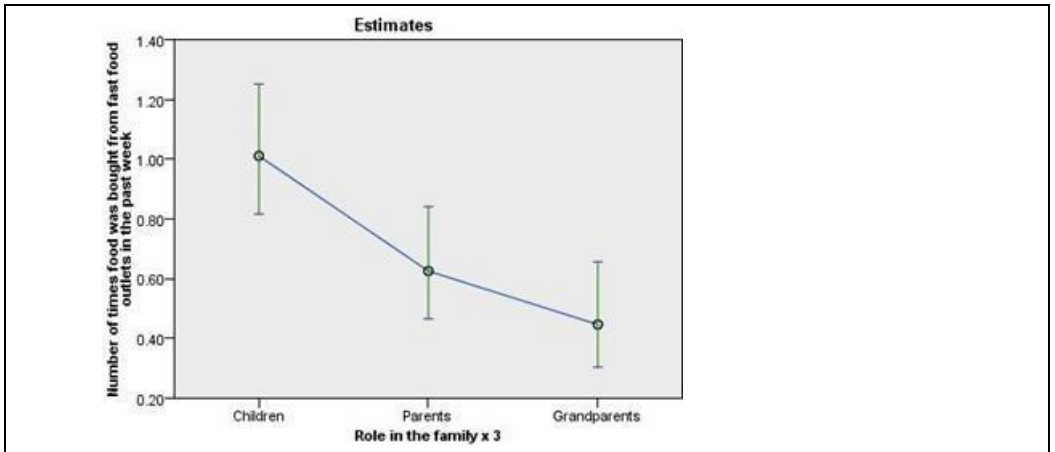


Figure D.3. Fast food consumption with differences between generations shown.

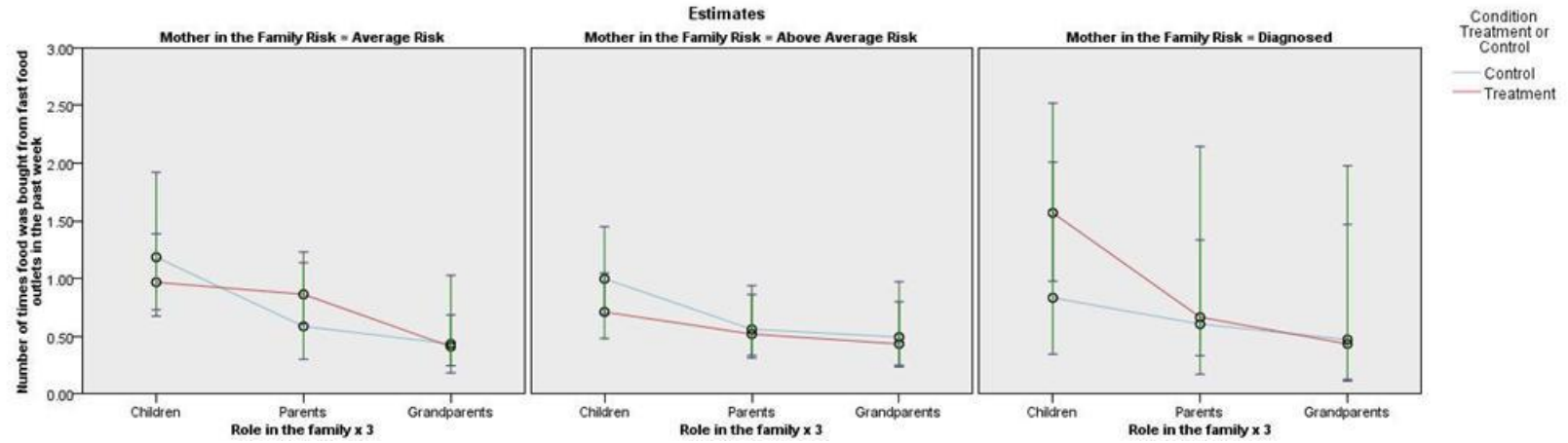


Figure D.4. Fast food consumption with interaction differences shown when a mother in the family is at risk or diagnosed with chronic disease, treatment and control groups are also shown.

Appendix 5.E: Three Generations by Gender Showing Descriptive Statistics of Unhealthy Food Consumption at Time One and Time Two.

Table E.1.

Three Generations by Gender Showing Unhealthy Food Consumption at Time One and Time Two.

Variable	Family Role	Time	*N*	*M*	*SD*	Median	Range	Min	Max
Snacks (serves/wk) [a]	Son	T1	31	6.23	3.65	5.00	15.00	0.00	15.00
	Son	T2	31	5.31	4.88	4.00	20.00	0.00	20.00
	Daughter	T1	32	5.47	3.91	5.00	17.00	0.00	17.00
	Daughter	T2	32	5.47	3.91	5.00	17.00	0.00	17.00
	Mother	T1	39	3.95	3.02	4.00	18.00	0.00	18.00
	Mother	T2	39	3.95	3.02	4.00	18.00	0.00	18.00
	Father	T1	21	4.00	3.30	3.00	14.00	0.00	14.00
	Father	T2	21	3.67	2.75	3.00	11.00	1.00	12.00
	Grandmother	T1	31	3.71	3.07	3.00	14.00	0.00	14.00
	Grandmother	T2	31	3.26	2.21	3.00	7.00	0.00	7.00
	Grandfather	T1	18	4.22	2.88	4.50	10.00	0.00	10.00
	Grandfather	T2	18	4.08	3.48	3.25	14.00	0.00	14.00
Fast (serves/wk) [a]	Son	T1	31	0.97	1.38	1.00	7.00	0.00	7.00
	Son	T2	31	0.90	1.45	0.00	6.00	0.00	6.00
	Daughter	T1	32	1.13	1.83	1.00	10.00	0.00	10.00
	Daughter	T2	32	1.13	1.45	1.00	7.00	0.00	7.00
	Mother	T1	39	0.46	0.55	0.00	2.00	0.00	2.00
	Mother	T2	39	0.36	0.49	0.00	1.00	0.00	1.00
	Father	T1	21	1.19	2.58	1.00	12.00	0.00	12.00
	Father	T2	21	0.81	0.75	1.00	3.00	0.00	3.00
	Grandmother	T1	31	0.13	0.34	0.00	1.00	0.00	1.00
	Grandmother	T2	31	0.23	0.43	0.00	1.00	0.00	1.00
	Grandfather	T1	18	0.11	0.32	0.00	1.00	0.00	1.00
	Grandfather	T2	18	0.22	0.43	0.00	1.00	0.00	1.00

Note. [a] Skew positive, interpreting median and range will be most meaningful.

Appendix 5.F: Odds Ratio Analyses of Food Consumption and Diet-Related Food Attitudes Using Multiple Logistic Regression

Odds Ratio Results Explained

Each of the four food consumption variables was dichotomized in order to conduct multiple logistic regression analyses. Fruit and vegetable consumption were each dichotomized on the basis of whether or not NHMRC recommended guidelines had been met (i.e., yes = 1, met; no = 0, not met). For unhealthy food, snacks were dichotomized on the mean (i.e., 4.4 serves per week was the cut point) and termed below the sample average (0), or above the sample average (1); fast food contained more than 50% valid zero scores, therefore was dichotomized according to whether fast food had been consumed or not (i.e., was any fast food consumed? 1 = yes, and 0 = no)[73]. Predictors entered simultaneously into each model were: time, condition, and generation.

Results of GLiM with GEE parameter estimates (see Tables F.1 and F.2 for healthy food consumption). For fruit consumption recommendations being 'met' relative to being 'not met' showed consumption was 0.92 (intercept) times higher when all model predictors were at zero (even though zero was not a meaningful score for some variables); recommendations 'not met' was the reference category; intercept B = 0.92, Exp(B) = 2.50, p = .006). The predictors time, condition and generation were non-significant. Vegetable consumption had a negative result, indicating that consumption was 1.3 times lower when all model predictors were at zero (intercept B = -1.29, Exp(B) = 0.28, p = .001). Again, the IVs time, condition, and generation were non-significant.

[73]To control for family nesting, Generalized Linear Modeling (GLiM) was used with a Generalized Estimating Equation (GEE) that corrects for any interdependencies within the data set, as occurred in Study 2. Fast food was the only dependent variable with high interclass correlations (ICCs; refer to all ICC tables in the Results section), nevertheless GLiM with GEE were used to run all logistic regression analyses with a binomial distribution and logit link function. The working correlation matrix structure (CORRTYPE) selected was 'independent' for fruit, vegetable and snack consumption because each ICC result was close to zero. For fast food consumption, however, the ICC result was closer to 1; therefore an 'EXCHANGEABLE' working correlation matrix structure was selected (IBM Corporation, 2013).

Table F.1.

Fruit Multiple Logistic Regression all predictors entered together.

Predictor		Fruit recommendations met? [d] (serves per day) *N* = 178					
		B[a]	Exp(B)[b] OR	95% CI Lower	95% CI Upper	*p*	*SE*
Intercept		-0.71	2.50	1.30	4.81	**.006**	0.33
Time	T1	0.52	1.02	0.73	1.41	.91	
	T2	0.00[c]	1				
Condition	Experimental	-0.13	1.33	0.70	2.51	.38	
	Control	0.00[c]	1				
Generation	Child	-0.12	0.89	0.41	1.92	.76	
	Parent	-0.62	0.54	0.25	1.17	.12	
	Grand	0.00[c]	1				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No.

Table F.2.

Vegetables Multiple Logistic Regression all predictors entered together.

Predictor		Vegetable recommendations met? [d] (serves per day) *N* = 178					
		B[a]	Exp(B)[b] OR	95% CI Lower	95% CI Upper	*p*	*SE*
Intercept		-1.29	0.28	0.13	0.57	**.001**	0.37
Time	T1	0.002	1.00	0.63	1.60	.99	
	T2	0.00[c]	1				
Condition	Experimental	0.29	1.35	0.75	2.42	.32	
	Control	0.00[c]	1				
Generation	Child	-0.03	0.97	0.43	2.18	.94	
	Parent	-0.45	0.64	0.27	1.50	.30	
	Grand	0.00[c]	1				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = No.

In unhealthy food, the logit of snack consumption being above the sample average relative to being below the sample average was 0.71 times lower when all predictors were at zero (intercept B = -0.71, Exp(B) = 0.49, $p = .04$). The predictor time was significant: the logit of snack consumption being above average was 1.7 times greater at baseline relative to follow up (B = 0.52, Exp(B) = 1.67, $p = .013$). Finally, in fast food consumption, the logit of fast food being consumed relative to none being consumed was 1.5 times lower when all predictors were at zero (intercept B = -1.504, Exp(B) = 0.22, $p < .001$). The predictor generation was significant: the logit of fast food being consumed was 5.4 times greater in children relative to grandparents (B = 1.69, Exp(B) = 5.43, $p < .001$), and 3.9 times greater in parents relative to grandparents (B = 1.36, Exp(B) = 3.89, $p < .001$).

Table F.3.

Snacks Multiple Logistic Regression all predictors entered together.

Predictor		Snacks either above or below sample mean score [d] (serves per week) *N* = 178 B[a]	Exp(B)[b] OR	95% CI Lower	95% CI Upper	*p*	*SE*
Intercept		-0.71	0.49	0.25	0.96	**.037**	0.34
Time	T1	0.52	**1.68**	1.11	2.52	**.013**	0.21
	T2	0.00[c]	1				
Condition	Experimental	-0.13	0.88	0.47	1.64	.69	
	Control	0.00[c]	1				
Generation	Child	0.57	1.77	0.92	3.40	.09	
	Parent	-0.40	0.67	0.33	1.39	.28	
	Grand	0.00[c]	1				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] denotes the referent group; [d] Reference category = Below sample mean score.

Table F.4.

Fast Food Multiple Logistic Regression all predictors entered together.

Predictor		Fast Food any consumed? Yes/No [d] (serves per week) *N* = 178 B[a]	Exp(B)[b] OR	95% CI Lower	95% CI Upper	*p*	*SE*
Intercept		-1.50	0.22	0.12	0.43	**<.001**	0.33
Time	T1	-0.03	0.97	0.63	1.49	.90	
	T2	0.00[c]	1				
Condition	Experimental	0.04	1.04	0.48	2.27	.92	
	Control	0.00[c]	1				
Generation	Child	1.69	**5.43**	2.96	9.98	**<.001**	0.31
	Parent	1.36	**3.89**	1.99	7.61	**<.001**	0.34
	Grand	0.00[c]	1				

Note. [a] B denotes the coefficient; [b] Exp(B) denotes the exponiated coefficient which is equivalent to the odds ratio; [c] Denotes the referent group; [d] Reference category = No.

Note. When the food attitude subscales diet-health oriented behaviour (DHOB) and diet-health/disease linked attitudes (DHLA), gender and the interaction gender*generation were subsequently added to the healthy and unhealthy food models, there was likely collinearity occurring that over-inflated the results and therefore these results were omitted. As stated earlier, decisions had to be made that best answered the research questions whilst also maintaining the integrity of the data analysis and subsequent results.[74]

[74] Collinearity statistical advice was obtained in supervision.

Appendix 5.G: Family Risk – Family Disease Risk, Condition, Generation and Healthy Food Consumption

Table G.1.
*Fruit Model 1.1: Condtion*Family Risk of any Disease*Generation, Differences in Consumption.*

Effects	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 1.1				(2, 335)			5.63	**.004**
Condition				(1, 335)			0.59	.440
	Treatment - control	-0.31	-0.91	(1, 335)	-1.10	0.48		.440
Generation				(2, 335)			3.42	**.030**
	Child - parent	0.82	2.47	(1, 335)	0.02	1.62		**.040**
	Child - grand	0.31	0.87	(1, 335)	-0.39	1.00		.390
	Parent - grand	-0.52	-1.71	(1, 335)	-1.19	0.16		.180
Family Disease Risk				(2, 335)			0.63	.540
	Diagnosed - average risk	-0.61	-0.97	(1, 335)	-2.07	-0.85		.800
	Diagnosed - above average risk	-0.62	-1.11	(1, 335)	-1.97	0.73		.800
	Above average risk - average risk	0.01	0.04	(1, 335)	-0.73	0.75		.970
Condition*Family risk* Generation [a]								
Treatment-Above average risk				(2, 335)			3.6	**.028**
	Treatment:Above average risk- Child - parent	0.52	1.62	(1, 335)	-0.20	1.23		.210
	Treatment: Above average risk- Child - grand	-0.54	-1.33	(1, 335)	-1.37	0.29		.210
	Treatment: Above average risk- Parent - grand	-1.06	-2.60	(1, 335)	-2.04	-0.08		**.029**

Note . GLiMM negative binomial vertical multilevel modeling. Model 1.1: -2 log pseudo likelihood = 736.854. [a] only significant interactions are shown.

Note. Family Risk & Vegetable Consumption – results were non-significant and were omitted.

Family Risk & Snack Consumption

Table G.2.

*Snacks Model 3.1: Family Risk of any Disease*Condition*Generation, Changes in Consumption Over Time*

Effects	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*	
Model 3.1				(2, 336)			4.87	**.008**	
Condition				(1, 336)			0.59	.610	
	Treatment-Control	0.34	0.51	(1, 336)	-0.98	1.65		.610	
Generation				(2, 335)			4.87	**.008**	
	Child - Parent	1.53	2.60	(1, 336)	0.20	2.86		**.020**	
	Child - Grand	1.80	3.04	(1, 336)	0.38	3.23		**.008**	
	Parent - Grand	0.27	0.58	(1, 336)	-0.65	1.19		.560	
Family Disease Risk				(2, 336)			1.63	.200	
	Diagnosed - Average risk	2.39	1.5	(1, 336)	-1.44	6.22		.400	
	Diagnosed - Above average risk	1.65	1.06	(1, 336)	-1.62	4.92		.400	
	Above average risk - Average risk	0.74	1.34	(1, 336)	-0.53	2.01		.400	
Interaction Condition*Family risk* Generation [a]									
Treatment-Above average risk				(2, 336)			9.02	**<.001**	
	Treatment-Above average risk- Child - Parent	2.88	4.2	(1, 336)	1.23	4.54		**<.001**	
	Treatment-Above average risk- Child - Grand	2.42	3.28	(1, 336)	0.76	4.08		**.002**	SE 0.74
	Treatment-Above average risk- Parent - Grand	-0.47	-0.79	(1, 336)	-1.62	0.69		.430	

Note . GLiMM negative binomial vertical multilevel modeling. Model 3.1: -2 log pseudo likelihood = 759.284. [a] only significant interactions are shown.

Family Risk & Fast Food Consumption

Table G.3.
*Fast Food Model 4.1: Family Risk of any Disease*Condition*Generation, Changes in Consumption Over Time*

Effects	Pairwise Contrasts	Contrast Estimate	*t*	*df*	95% CI Lower	95% CI Upper	*F*	*p*
Model 4.1				(2, 332)			6.28	**.002**
Condition		ns						
	Treatment-Control	ns						
Generation				(2, 332)			6.28	**.002**
	Child - Parent	0.35	2.04	(1, 332)	-0.04	0.74		.080
	Child - Grand	0.57	1.69	(1, 332)	0.18	0.97		**.002**
	Parent - Grand	0.22	1.69	(1, 332)	-0.04	0.48		.090
Family Disease Risk				(2, 332)			0.07	.930
	Diagnosed - Average risk	-0.07	-0.32	(1, 332)	-0.59	0.46		1.00
	Diagnosed - Above average risk	-0.32	-0.16	(1, 332)	-0.43	0.37		1.00
	Above average risk - Average risk	-0.39	-0.32	(1, 332)	-0.30	0.22		1.00
Interaction Condition*Family risk* Generation [a]								
Treatment-Average risk				(2, 332)			3.91	**.020**
	Treatment-Average risk - Child - Parent	0.36	1.12	(1, 332)	-0.27	0.99		.260
	Treatment-Average risk - Child - Grand	0.76	2.63	(1, 332)	0.06	1.45		**.030**
	Treatment-Average risk - Parent - Grand	0.4	1.65	(1, 332)	-0.15	0.94		.200
Treatment-Above average risk				(2, 332)			3.95	**.020**
	Treatment-Above average risk- Child - Parent	0.22	1.27	(1, 332)	-0.13	0.57		.240
	Treatment-Above average risk- Child - Grand	0.47	2.79	(1, 332)	0.06	0.87		**.017**
	Treatment-Above average risk- Parent - Grand	0.25	1.56	(1, 332)	-0.11	0.60		.240

Appendix 5.H: Three Generations by Gender Showing Descriptive Statistics of Diet-health Food Attitudes

Table H.1.

Three Generations by Gender Showing Diet-health Food Attitude Mean Scores at Time One and Time Two.

Variable	Family Role	Time	*N*	*M*	*SD*	Median	Range	Min	Max
DHB (scores 1 - 7)	Son	T1	29	4.39	0.98	4.40	4.40	2.20	6.60
	Son	T2	29	4.50	1.12	4.40	4.00	3.00	7.00
	Daughter	T1	33	4.33	0.80	4.20	3.40	2.80	6.20
	Daughter	T2	33	4.25	0.79	4.20	3.00	3.00	6.00
	Mother	T1	40	5.21	0.80	4.20	3.40	2.80	6.20
	Mother	T2	40	5.20	0.82	5.20	3.00	4.00	7.00
	Father	T1	22	4.74	0.80	4.70	3.20	3.40	6.60
	Father	T2	22	4.54	0.74	4.60	3.00	3.00	6.00
	Grandmother	T1	30	5.24	0.86	5.40	3.40	3.60	7.00
	Grandmother	T2	30	5.29	0.86	5.20	3.00	4.00	7.00
	Grandfather	T1	18	4.81	0.68	4.90	2.60	3.40	6.00
	Grandfather	T2	18	5.56	1.16	5.63	3.75	3.25	7.00
DDB (scores 1 - 7)	Son	T1	29	5.47	0.98	5.50	4.50	2.50	7.00
	Son	T2	29	5.71	0.94	5.75	5.00	3.00	7.00
	Daughter	T1	33	5.47	0.85	5.50	2.75	4.25	7.00
	Daughter	T2	33	5.49	0.90	5.50	4.00	4.00	7.00
	Mother	T1	40	6.22	0.79	6.38	2.67	4.33	7.00
	Mother	T2	40	6.33	0.66	6.38	2.00	5.00	7.00
	Father	T1	22	5.85	0.77	6.00	2.50	4.50	7.00
	Father	T2	22	5.95	0.80	6.00	2.00	5.00	7.00
	Grandmother	T1	30	6.13	0.82	6.50	3.25	3.75	7.00
	Grandmother	T2	30	6.09	0.87	6.25	3.00	4.00	7.00
	Grandfather	T1	18	5.56	1.16	5.63	3.75	3.25	7.00
	Grandfather	T2	18	5.79	1.01	5.88	3.00	4.00	7.00

Note. [a] Skew negative, interpreting median and range will be most meaningful.

Appendix 5.I: Correlations – Food Life Behaviours and Attitudes

Diet-health oriented behaviours by Generations

Table I.1.
Spearman's rho Three Generations DHOB Correlations at Time One and Time Two

Generation	T1 Child	T1 Parent	T1 Grand	T2 Child	T2 Parent	T2 Grand
Child	.	.099	-.249	.	**.350***	.005
n	41	39	38	41	39	38
Parent		.	**.309***		.	-.059
n		40	37		40	37
Grand			.			.
n			39			39

Note. * $p < 0.05$, ** $p < .01$ (1-tailed). T1 10% shared variance; T2 12% shared variance.

Diet-health oriented behaviours – Generations by Gender

Table I.2.

Spearman's rho Three Generations by Gender Showing DHOB Food Attitude Correlations at Time One and Time Two

DHOB			Time	One					Time	Two		
Family relationship	Son	Daughter	Mother	Father	Grandmother	Grandfather	Son	Daughter	Mother	Father	Grandmother	Grandfather
Son	.	**.887****	-.032	.414	-.237	.134	.	.127	0.248	.146	.072	-0.407
n	23	9	22	15	15	12	24	9	22	15	15	12
Daughter		.	-.254	.113	**-.396***	-.442		.	**.356***	.306	.236	-.024
n		27	26	14	21	8		27	26	14	20	8
Mother			.	.062	.334 [a]	**.562*** [a]			.	**.528****	.199 [a]	-.282 [a]
n			39	22	23	14			40	22	22	14
Father				.	-.500 [b]	.500 [b]				.	-.632 [b]	-.500 [b]
n				22	4	3				22	4	3
Grandmother					.	.336					.	.487
n					31	10					30	9
Grandfather						.						.
n						18						18

Note. * $p < 0.05$, ** $p < .01$ (1-tailed), [a] maternal grandparents' [b] paternal grandparents. T1 79% shared variance between siblings, 16% grandmother-granddaughter negative correlation, 32% grandfather-mother. T2 13% mother-daughter, and 28% parent partners.

Diet-health/disease linked attitudes by Generations

Table I.3.
Spearman's rho Three Generations DHLA Correlations at Time One and Time Two.

Generation	T1 Child	T1 Parent	T1 Grand	T2 Child	T2 Parent	T2 Grand
Child	.	-.133	.038	.	.081	.193
n	40	38	36	40	38	37
Parent		.	.276		.	**.320***
n		40	36		40	37
Grand			.			.
n			38			39

Note. * $p < 0.05$, ** $p < .01$ (1-tailed). T2 only 10% shared variance between grandparents and parents.

Diet-health/disease linked attitudes – Generations by Gender

Table I.4.

Spearman's rho Three Generations by Gender Showing DHL Food Attitude Correlations at Time One and Time Two

DHLA			Time One						Time Two				
Family Relationship	Son	Daughter	Mother	Father	Grandmother	Grandfather	Son	Daughter	Mother	Father	Grandmother	Grandfather
Son	.	.797	-.152	.388	.276	-.313	.	**.751****	.193	-.238	.114	.168
n	23	9	22	15	15	12	22	9	21	15	14	12
Daughter		.	-.284	.316	.143	-.383		.	.151	-237	-.040	.189
n		27	25	13	19	8		27	26	14	20	8
Mother			.	**.378***	.210 [a]	.445 [a]			.	.352	.095 [a]	.242 [a]
n			39	22	22	14			40	22	22	14
Father				.	-.316 [b]	.000 [b]				.	-.400 [b]	**1.000**** [b]
n				22	4	3				22	4	3
Grandmother					.	.509					.	.096
n					31	10					30	9
Grandfather						.						.
n						18						18

Note. * $p < 0.05$, ** $p < .01$ (1-tailed), [a] maternal grandparents' [b] paternal grandparents. T1 showed 14% of the variance between parent partners. T2 showed 56% of the variance between siblings, and 100% between 3 granparent partners.

This appears to be an intergenerational transmission of diet-disease link knowledge shifting from the parent generation to the children, and also to males with grandfather-father. Although numbers are very small, therefore infer conservatively.

Appendix 5.J: Correlations - Food Life Behaviour and Attitudes with Food Consumption at Baseline and Follow up

Fruit Consumption Correlations with Food Life Behaviour and Attitudes by Generation at Time 1 and Time 2

Table J.1.

Fruit Consumption and Food Attitudes . Spearman's rho Three Generations of Food Consumption with Food Attitude Correlations at Time One and Time Two.

	T1	T2	T1	T2	T1	T2
	Child	Child	Parent	Parent	Grand	Grand
DHOB [a]	**.362***	**.376****	.078	.087	.140	-.209
n	41	41	40	40	39	38
DHLA [a]	.224	.100	-.073	-.075	**.296***	**.323***
n	40	40	40	40	38	38

N+A409:I417ote. 13% of the diet-health oriented behaviour variance occurred in children at T1, and 14% at T2. 9% of the diet-health/disease linked attitudes occurred in grandparents at T1, and 10% at T2.

Fruit Consumption Correlations with Food Life Behaviour and Attitudes by Generation and Gender at Time 1 and Time 2

Table J.2.

Fruit consumption. Spearman's rho Correlations of Gender and Three Generations Healthy and Unhealthy Food Consumption with Food Attitudes at Time One and Time Two.

Fruit	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
	Son	Son	Daughter	Daughter	Mother	Mother	Father	Father	GrandMo	GrandMo	GrandFa	GrandFa
DHOB (%)	**.543**** (29)	**.488**** (24)	.285	**.338*** (15)	-.001	.120	.116	-.132	.133	-.212	-.005	.178
n	23	23	27	27	40	39	22	22	31	29	18	18
DHLA (%)	**.368*** (14)	.315	.106	.118	-.075	-.105	.143	-.138	.304	.170	.225	**.425*** (18)
n	23	22	26	27	40	39	22	22	30	29	18	18

Note. * $p < 0.05$, ** $p < .01$ (1-tailed).

Vegetable Consumption Correlations with Food Life Behaviour and Attitudes by Generation at Time 1 and Time 2

Table J.3. *Vegetable Consumption and Food Attitudes*

	T1	T2	T1	T2	T1	T2
	Child	Child	Parent	Parent	Grand	Grand
DHOB [a]	**.450****	.129	.193	.071	**.304***	-.035
n	41	40	39	40	39	39
DHLA [a]	.111	.193	-.008	-.023	.003	.223
n	40	39	40	40	38	39

Note. T1 showed 20% of the diet-health oriented behaviour variance occurred in children and 9% in grandparents.

Vegetable Consumption Correlations with Food Life Behaviour and Attitudes by Generation and Gender at Time 1 and Time 2

Table J.4. *Vegetable Consumption and Food Attitudes. Spearman's rho Correlations of Gender and Three Generations Healthy Food Consumption with Food Attitudes at Time One and Time Two.*

Vegetables	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
	Son	Son	Daughter	Daughter	Mother	Mother	Father	Father	GrandMo	GrandMo	GrandFa	GrandFa
DHOB (%)	**.583**** (34)	.279	**.351*** (12)	.155	-.111	.025	.458	.032	**.371*** (14)	-.014	.113	-.148
n	23	23	27	26	39	39	22	22	31	30	18	10
DHLA (%)	.065	**.454*** (21)	.088	.100	-.090	-.098	.278	.064	.093	**.415*** (17)	-.222	-.330
n	23	22	26	26	39	39	22	22	30	30	18	10

Note. * $p < 0.05$, ** $p < .01$ (1-tailed).

Snack Consumption Correlations with Food Life Behaviour and Attitudes by Generation at Time 1 and Time 2

Table J.5. *Snack Consumption and Food Attitudes*

	T1	T2	T1	T2	T1	T2
	Child	Child	Parent	Parent	Grand	Grand
DHOB [a]	**-.346***	**-.353***	-.008	-.177	.105	**-.343***
n	40	41	40	40	39	39
DHLA [a]	-.116	-.207	-.045	.022	.031	-.103
n	39	40	40	40	38	39

Note. Negative correlations between diet-healthoriented behaviour and snack consumption in children showed the variance was 12% at T1 and T2. With grandparents it was 12% but only at T2.

Snack Consumption Correlations with Food Life Behaviour and Attitudes by Generation and Gender at Time 1 and Time 2

Table J.6. *Snack Consumption and Food Attitudes. Spearman's rho Correlations of Gender and Three Generations Unhealthy Food Consumption with Food Attitudes at Time One and Time Two.*

Snacks	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
	Son	Son	Daughter	Daughter	Mother	Mother	Father	Father	GrandMo	GrandMo	GrandFa	GrandFa
DHOB (%)	-.251	-.318	**-.440* (19)**	**-.531**(28)**	-.218	-.159	.096	**-.445* (20)**	-.042	-.241	.097	-.144
n	23	23	26	27	40	40	22	21	31	30	18	18
DHLA (%)	-.130	-.257	-.186	-.181	-.124	-.157	-.156	**-.512**(26)**	-.090	.053	.125	.100
n	23	22	25	27	40	40	22	21	30	30	18	18

Note. * $p < 0.05$, ** $p < .01$ (1-tailed).

Fast Food Consumption Correlations with Food Life Behaviour and Attitudes by Generation at Time 1 and Time 2

Table J.7. *Fast Food Consumption and Food Attitudes*

	T1	T2	T1	T2	T1	T2
	Child	Child	Parent	Parent	Grand	Grand
DHOB [a]	-.133	**-.395****	**-.285***	**-.389****	-.076	-.185
n	40	41	39	40	39	38
DHLA [a]	-.081	-.092	.189	-.221	.180	,037
n	39	40	39	40	38	37

Note. * $p < 0.05$, ** $p < .01$ (1-tailed). [a] Food attitudes at mean child, parent and grandparent levels were correlated with each family member's own food consumption. Negative correlations in children showed 16% of the variance in diet-health oriented behaviour at T2 only, and parents had 8% at T1 and 15% of the variance at T2.

Fast Food Consumption Correlations with Food Life Behaviour and Attitudes by Generation and Gender at Time 1 and Time 2

Table J.8. *Fast Food Consumption and Food Attitudes. Spearman's rho Correlations of Gender and Three Generations Unhealthy Food Consumption with Food Attitudes at Time One and Time Two.*

Fast Food	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
	Son	Son	Daughter	Daughter	Mother	Mother	Father	Father	GrandMo	GrandMo	GrandFa	GrandFa
DHOB (%)	-.069	-.234	-.103	**-.451** (20)**	**-.322* (10)**	**-.283* (8)**	**-.443* (20)**	-.356	-.113	-.245	-.051	.117
n	23	23	26	27	39	40	22	22	31	29	18	18
DHLA (%)	-.011	-.041	-.224	-.258	.088	-.141	.023	-.188	.053	.073	.137	-.365
n	23	22	25	27	39	40	22	22	30	29	18	18

Note. * $p < 0.05$, ** $p < .01$ (1-tailed).

Appendix 5.K: Correlations of Parent's Food Attitudes with Children's Healthy and Unhealthy Food Consumption at Baseline and Follow up

Children's Healthy Food Consumption and Parent's Diet-health Oriented Behaviour at Time 1 and Time 2

Table K.1.

Spearman's rho Correlations of Parent's Mean Diet-Health Orientated Behaviour (Food Attitude Subscale Scores) with the Healthy Food Consumption of Offspring at Time One and Time Two.

Healthy/DHOB	T1	T2	T1	T2	T1	T2	T1	T2
FLQ-SF Subscale	Child Fruit Consumption				Child Vegetable Consumption			
	Son	Son	Daughter	Daughter	Son	Son	Daughter	Daughter
Mother's DHOB	-.217	.196	**-.462****	.021	-.282	.322	-.203	-.327
n	23	23	26	26	23	23	26	25
Father's DHOB	.102	.085	-.090	.418	**.443***	.402	.262	.241
n	16	16	14	14	16	16	14	13

* $p < 0.05$ one tailed. ** $p < 0.01$ one tailed.

Children's Healthy Food Consumption and Parent's Diet-health/disease Linked Attitudes at Time 1 and Time 2

Table K.2.

Spearman's rho Correlations of Parent's Mean Diet-Health/disease Linked Attitudes (Food Attitude Subscale Scores) with the Healthy Food Consumption of Offspring at Time One and Time Two.

Healthy/DHLA	T1	T2	T1	T2	T1	T2	T1	T2
FLQ-SF Subscale	Child Fruit Consumption				Child Vegetable Consumption			
	Son	Son	Daughter	Daughter	Son	Son	Daughter	Daughter
Mother's DHLA	-.294	-.185	.208	-.157	-.287	.009	.228	-.089
n	23	23	26	26	23	23	26	25
Father's DHLA	.300	.168	.405	.330	.322	.331	.231	.288
n	16	16	14	14	16	16	14	13

* $p < 0.05$ one tailed. ** $p < 0.01$ one tailed.

Children's Unhealthy Food Consumption and Parent's Diet-health Oriented Behaviour at Time 1 and Time 2

Table K.3.

Spearman's rho Correlations of Parent's Mean Diet-Health Orientated Behaviour (Food Attitude Subscale Scores) with the Unhealthy Food Consumption of Offspring at Time One and Time Two.

Unhealthy/DHOB	T1	T2	T1	T2	T1	T2	T1	T2
FLQ-SF Subscale	Child Snack Consumption				Child Fast Food Consumption			
	Son	Son	Daughter	Daughter	Son	Son	Daughter	Daughter
Mother's DHOB	-.311	.065	.288	-.142	-101	.039	.246	-.183
n	23	23	25	26	23	23	25	26
Father's DHOB	-.337	.322	-.158	-.062	.135	.372	.160	.381
n	16	16	13	14	16	16	13	14

* $p < 0.05$ one tailed. ** $p < 0.01$ one tailed.

Children's Unhealthy Food Consumption and Parent's Diet-health/disease Linked Attitudes at Time 1 and Time 2

Table K.4.

Spearman's rho Correlations of Parent's Mean Diet-Health/disease Linked Attitudes (Food Attitude Subscale Scores) with the Unhealthy Food Consumption of Offspring at Time One and Time Two.

Unhealthy/DHLA	T1	T2	T1	T2	T1	T2	T1	T2
FLQ-SF Subscale	Child Snack Consumption				Child Fast Food Consumption			
	Son	Son	Daughter	Daughter	Son	Son	Daughter	Daughter
Mother's DHLA	-.040	-.011	.096	.056	-.048	-.265	-.082	-.109
n	23	23	25	26	23	23	25	26
Father's DHLA	-.193	.262	-.028	-.078	.056	.072	.455	.130
n	16	16	13	14	16	16	13	14

* $p < 0.05$ one tailed. ** $p < 0.01$ one tailed.

Appendix 5.L: Pseudo Log Likelihood Results Chronic Disease Risk Results of Food Life Behaviour and Attitudes on each Food Consumption Variable

Food Attitudes and Food Consumption when a Family Member or Mother is at increased Risk of Chronic Disease

Additional GLiMM analyses examined whether the food attitudes DHOB and DHLA improved upon the previous longitudinal 'family-risk' and 'mother-risk' GLiMM models for healthy and unhealthy food consumption. Pseudo log likelihood (-2LL) ratio tests (Sakamoto et al., 1988) were used to determine differences between models, followed by chi square tests of significance which revealed food attitudes significantly added to each of the earlier models, Appendix L shows the -2 LL results tables. One of the limitations of this modeling technique is that to date, effect size is not able to be determined from pseudo log likelihood (-2 LL) ratio testing. The chi-square significance test, determines the significance of -2 LL ratio tests, however it is not clear whether a phi coefficient applied to the chi-square statistic may act as an effect size for the result. Since this has not been attempted previously, a phi coefficient statistic could be considered in future research. .

Table L.1

Fruit Consumption with Food Life Behaviours and Attitudes.

Dependent Variable	Independent Variable	Model 1 -2 pseudo log likelihood	Model 2 (Added variable) -2 pseudo log likelihood	Difference	Chi-square statistic 3 IVs and 1 df the critical value is 10.83 at the $p < .001$ level
Fruit	Family disease risk	756.602	(DHOB) 725.095	31.507	> critical value of 10.83 $p < .001$
Fruit	Family disease risk	756.602	(DHLA) 735.530	21.072	> critical value of 10.83 $p < .001$
Fruit	Mother disease risk	753.004	(DHOB) 725.475	27.529	> critical value of 10.83 $p < .001$
Fruit	Family disease risk	753.004	(DHLA) 732.282	20.722	> critical value of 10.83 $p < .001$

Table L.2

Vegetable Consumption with Food Life Behaviours and Attitudes.

Dependent Variable	Independent Variable	Model 1 -2 pseudo log likelihood	Model 2 (Added variable) -2 pseudo log likelihood	Difference	Chi-square statistic 3 IVs and 1 df the critical value is 10.83 at the p < .001 level
Vegetables	Family disease risk	560.695	(DHOB) 548.721	11.974	> critical value of 10.83 *p* < .001
Vegetables	Family disease risk	560.695	(DHLA) 545.559	15.136	> critical value of 10.83 *p* < .001
Vegetables	Mother disease risk	561.566	(DHOB) 548.756	11.939	> critical value of 10.83 *p* < .001
Vegetables	Family disease risk	561.566	(DHLA) 546.886	13.809	> critical value of 10.83 *p* < .001

Table L.3

Snack Consumption with Food Life Behaviours and Attitudes.

Dependent Variable	Independent Variable	Model 1 -2 pseudo log likelihood	Model 2 (Added variable) -2 pseudo log likelihood	Difference	Chi-square statistic 3 IVs and 1 df the critical value is 10.83 at the p < .001 level
Snacks	Family disease risk	775.233	(DHOB) 700.777	74.456	> critical value of 10.83 *p* < .001
Snacks	Family disease risk	775.233	(DHLA) 723.584	51.649	> critical value of 10.83 *p* < .001
Snacks	Mother disease risk	785.810	(DHOB) 704.409	54.548	> critical value of 10.83 *p* < .001
Snacks	Family disease risk	785.810	(DHLA) 731.262	13.809	> critical value of 10.83 *p* < .001

Table L.4

Fast Food Consumption with Food Life Behaviours and Attitudes.

Dependent Variable	Independent Variable	Model 1 -2 pseudo log likelihood	Model 2 (Added variable) -2 pseudo log likelihood	Difference	Chi-square statistic for 3 IVs and 1 *df* the critical value is 10.83 at the $p <$ .001 level
Fast Food	Family disease risk	1002.975	(DHOB) 700.777	65.010	> critical value of 10.83 $p < .001$
Fast Food	Family disease risk	1002.975	(DHLA) 723.584	62.044	> critical value of 10.83 $p < .001$
Fast Food	Mother disease risk	997.508	(DHOB) 942.204	55.304	> critical value of 10.83 $p < .001$
Fast Food	Family disease risk	997.508	(DHLA) 939.221	58.287	> critical value of 10.83 $p < .001$

CHAPTER 6 APPENDICES

Appendix 6.A: Family Health History Evaluation Questionnaire

Reduced in size to 85%

Family Health History Evaluation Questionnaire

As part of your participation in this study, you received the Families SHARE workbook including a Family Health History Tree detailing your risk for developing heart disease, diabetes, breast cancer and colorectal cancer. This section asks you some questions about your thoughts on the Families SHARE workbook.

1. Using the Family Health Workbook, were you able to assess your own degree of risk for each disease?

   ☐ Yes ☐ No

   1a. Are you at increased risk for:

   Colorectal cancer ..... ☐ Yes ☐ No ☐ Don't know

   Breast cancer ..... ☐ Yes ☐ No ☐ Don't know

   Heart disease ..... ☐ Yes ☐ No ☐ Don't know

   Diabetes..... ☐ Yes ☐ No ☐ Don't know

2. Have you shared information from the Families SHARE workbook with any of the following people (tick all that apply):

   ☐ GP

   ☐ Other health care provider (e.g., nurse, specialist, pharmacist). If so, please indicate who:

   _____

   ☐ Family member (please list the names of any family members you have shared your family health history with):

   _____

   ☐ Friends (please list the names of any friends you have shared your family health history with):

   _____

   ☐ Other (please specify)_____

3. After receiving your Family Health History Tree diagram did you update it in any way?

   ☐ Yes ☐ No

   If yes, please list any changes you made:

   _____

   If yes, were any changes based on the advice of someone else, and if so, who?

   _____

4. In the last 6 months, have you participated in any screening behaviour for any of the following diseases?

   Colorectal cancer ☐ Yes ☐ No

   Breast cancer ☐ Yes ☐ No

   Heart disease ☐ Yes ☐ No

   Diabetes ☐ Yes ☐ No

Thank You for your Participation!

Appendix 6B: Families SHARE Evaluation: Family Interview Discussion Guide

Families SHARE Evaluation: Family Interview Discussion Guide

OPENING QUESTION - First, I'd like to start by going over the example Family Health History Tree on pages 2 – 3 to refresh everyone's memory. For example, if Jane is the mother, who can point out her uncle?

1. Next, I have brought along a copy of your family tree diagram. I wondered how your family worked out if they were at increased disease risk, see pages 7 - 13 [*laminated pages (4) depicting disease risk instructions*].
    - [prompt] …and who was it that worked out the risks for the whole family, or did each person work out their own risk? (e.g., sibships will differ from other generations).

2. One of the reasons we chose mothers to be the main point of contact for this study was because we believe that mothers may be the most effective communicators in the family and the "keepers" of family health history information.
    - [prompt] …if you can think back to the first questionnaire [*show laminated page of family health history questionnaire*], how did your family collect the required family health history information?

3. After your family received the workbook, how did your family share and talk about your family health history?
    - [prompt] …and how was disease risk shared and discussed?
    - Did you find there were any barriers to (e.g., distance), or other facilitators of, communication (e.g., a grandparent taking the lead)?

4. I'd like to go on now to the "Healthy Recommendations" that reduce disease risk on page 14 [*show laminated page*]. But first, I wonder if you could explain the importance of health to your family?
    - [prompt] …I'd really like to hear whether any of your diet and lifestyle choices have changed due to the experience of illness in your family?
    - [prompt] …or whether any of your diet and lifestyle choices were affected by the workbook?
    - [prompt] …or whether any of your diet and lifestyle choices were affected due to the questionnaire itself (i.e., before receiving the workbook)?
    - [prompt] …okay, now I'd be interested to hear which of the recommendations were most important to your family? Explain…

5. So far, we've talked about things in the workbook like disease risk and health recommendations. But I'm curious as to whether you believe these recommendations are actually effective. How much control do you think you have over disease risk? For example, do you think you can avoid different diseases such as cancer or heart disease and diabetes - by making changes to your diet or other lifestyle activities (such as exercise)?

6. For families who aren't healthy, do you think that disease risk information is a good way to motivate them to make healthier lifestyle choices?
    - [prompt] …I wondered if you could tell us what factors have motivated your family to increase any healthy lifestyle behaviours or reduce unhealthy lifestyle behaviours in the past 6-12 months.
    - [prompt] (If applicable ask)…and what do you think has motivated your family to maintain healthier lifestyles over time?

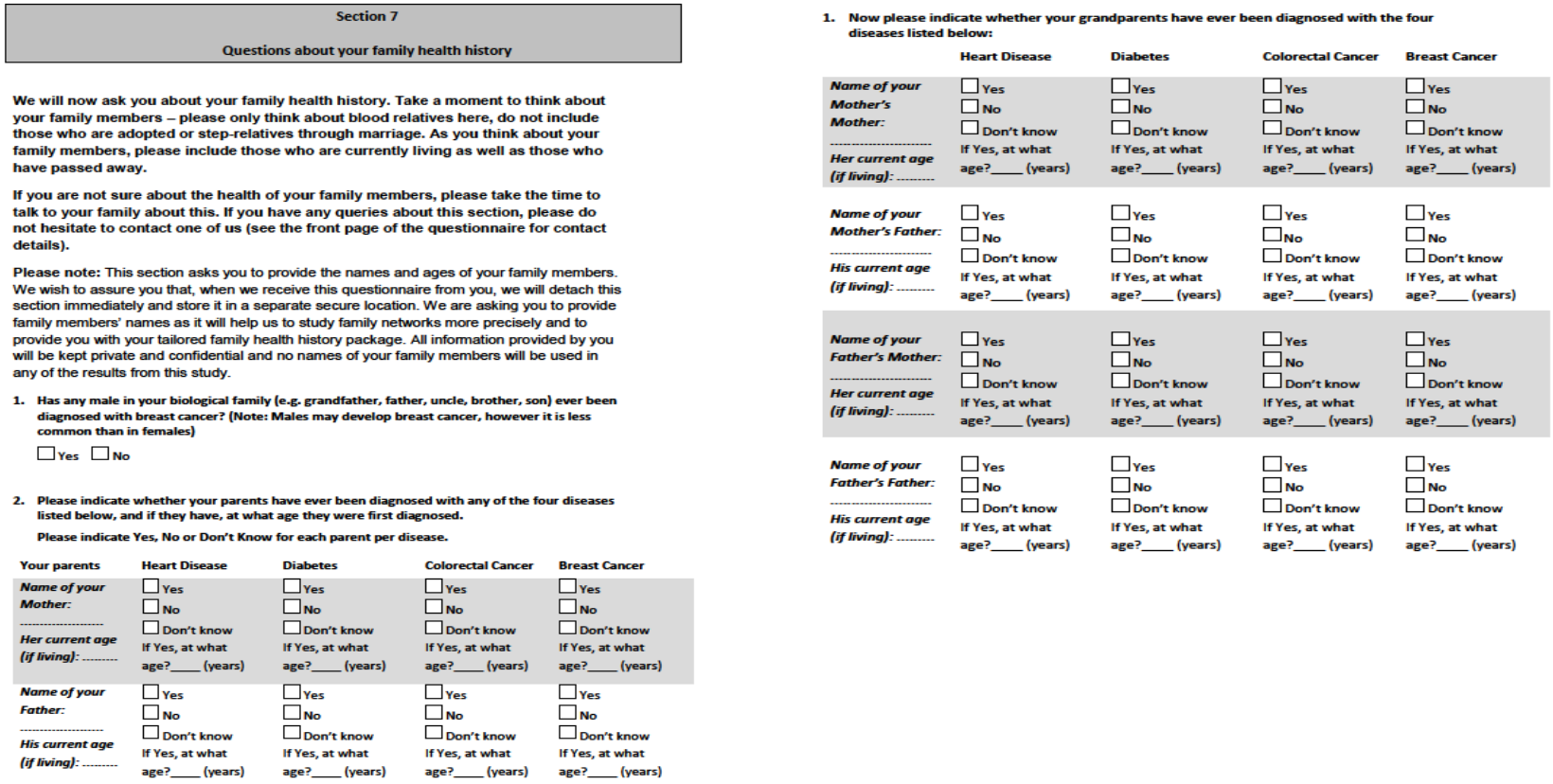
7. Lastly, I would be interested to hear whether your family thought about any of the screening recommendations on page 15? [*show laminate page*]

8. CLOSING QUESTION – the purpose of this study is to provide families with disease risk information. We wanted to figure out the best ways to provide this information. We've talked about a number of issues including [summarise main points raised], did the workbook affect your family in any other ways?
    - Is there something that we missed?
    - Is there any reason why the workbook wasn't found useful to you?
    - Or is there anything you were hoping to discuss but didn't get a chance to?

Thank You for your Participation!

Appendix 6.C: Family Interview Visual Prompts

Reduced in size to 25%



Section 7

Questions about your family health history

We will now ask you about your family health history. Take a moment to think about your family members – please only think about blood relatives here, do not include those who are adopted or step-relatives through marriage. As you think about your family members, please include those who are currently living as well as those who have passed away.

If you are not sure about the health of your family members, please take the time to talk to your family about this. If you have any queries about this section, please do not hesitate to contact one of us (see the front page of the questionnaire for contact details).

Please note: This section asks you to provide the names and ages of your family members. We wish to assure you that, when we receive this questionnaire from you, we will detach this section immediately and store it in a separate secure location. We are asking you to provide family members' names as it will help us to study family networks more precisely and to provide you with your tailored family health history package. All information provided by you will be kept private and confidential and no names of your family members will be used in any of the results from this study.

1. **Has any male in your biological family (e.g. grandfather, father, uncle, brother, son) ever been diagnosed with breast cancer? (Note: Males may develop breast cancer, however it is less common than in females)**

   ☐ Yes ☐ No

2. **Please indicate whether your parents have ever been diagnosed with any of the four diseases listed below, and if they have, at what age they were first diagnosed.**

   Please indicate Yes, No or Don't Know for each parent per disease.

Your parents	Heart Disease	Diabetes	Colorectal Cancer	Breast Cancer
Name of your Mother: ---------- ***Her current age (if living):*** ----------	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)
Name of your Father: ---------- ***His current age (if living):*** ----------	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)

Grandparents

1. **Now please indicate whether your grandparents have ever been diagnosed with the four diseases listed below:**

	Heart Disease	Diabetes	Colorectal Cancer	Breast Cancer
Name of your Mother's Mother: ---------- ***Her current age (if living):*** ----------	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)
Name of your Mother's Father: ---------- ***His current age (if living):*** ----------	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)
Name of your Father's Mother: ---------- ***Her current age (if living):*** ----------	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)
Name of your Father's Father: ---------- ***His current age (if living):*** ----------	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)	☐ Yes ☐ No ☐ Don't know If Yes, at what age?____ (years)

Figure C.1. Visual prompt: Questions about family health history from the questionnaire (enlarged and laminated as one A3 size page).

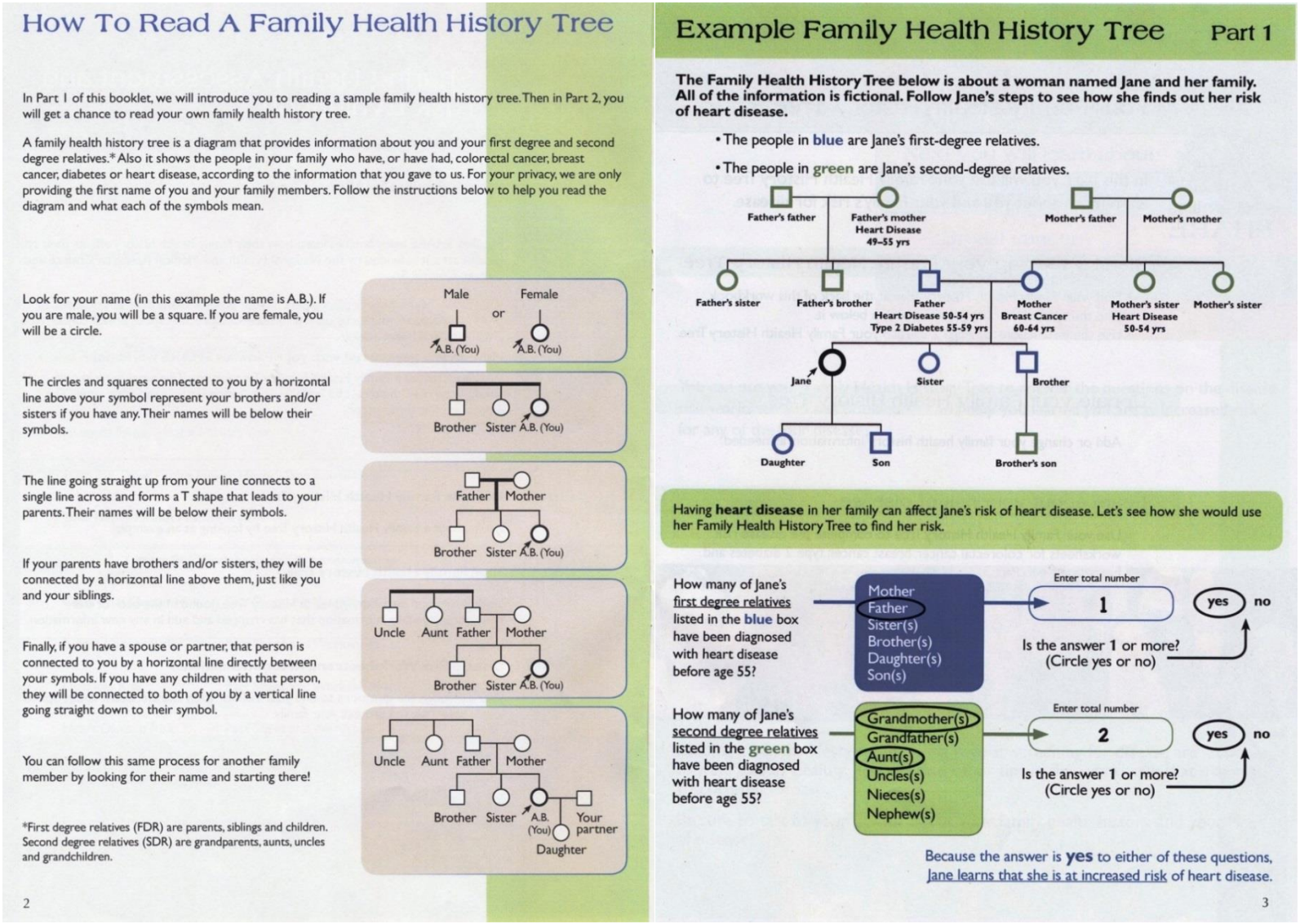


Figure C.2. Visual prompt: How to read a family health history tree from the families share workbook (enlarged and laminated as one A3 size page).

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