Newly emerging pathogens and disease outbreaks in areas previously undetected are an increasing concern as climate change causes host and vector distributions to shift. Fluctuating conditions require hosts to be able to tolerate changing pathogen diversity and abundance to survive. Understanding host-pathogen interactions in a common wild species may provide insight in how other species could respond to disease outbreaks. This thesis explores the host-pathogen interactions in an iconic Australian skink, *Tiliqua rugosa* that is widely distributed across the southern half of Australia and has close interactions with humans.

First, I explore immune genes of a *T. rugosa* population in Mount Mary, South Australia, across an ecological gradient. Using genome wide SNPs, I also investigated the *T. rugosa*'s population structure driven by two parasitic tick species over an ecological gradient. I found an association with certain Major Histocompatibility Complex class I (MHC I) alleles and tick infestation type — these alleles were also under selection. SNP data showed that the genetic differentiation shown by the MHC data was not simply reflective of a population differentiation that occurs across the whole genome. In the future, by understanding this differentiation in this system, and the association with tick vectors, studies may find evidence of the beginnings of a species divergent event without geographic barriers restricting gene flow.

Since MHC I is an antigen recognition molecule, the positive selection found could also be explained by varying viral abundances across the ecological gradient, and not necessarily via tick vectors. There are currently only two known viruses in *T. rugosa* (*Shingleback nidovirus 1* and Adenovirus), both of which are respiratory viruses. Therefore, I investigated the viral communities in the oral cavity of *T. rugosa* using flow cytometry and compared viral abundances across the ecological gradient of arid to humid environmental conditions. This presents a novel application of flow cytometry in a reptile system. I found two viral sub-populations, that are yet to be characterised, in greater than 95% of sampled lizards, and significant abundance variations across the gradient. These results support the positive selection found, as could be explained by pathogen mediated selection, and shows how changing environmental conditions influence viral abundances.

At the start of this PhD, there was one known virus in *T. rugosa* —the *Shingleback nidovirus 1* — that had been associated with the bobtail flu. The bobtail flu was detected in the

surrounding Perth Hills, Western Australia, and had a severe impact on the *T. rugosa*'s local wildlife population. However, there were also anecdotal reports of *T. rugosa* with bobtail flu symptoms in other states, including at Mount Mary, South Australia. I therefore aimed to determine whether the bobtail flu had come across from Perth and was in the Mount Mary study site. I sampled *T. rugosa* between Perth-Adelaide using reverse transcription real-time PCR (RT-qPCR) to screen for the *Shingleback nidovirus 1*' presence or absence in an individual. I found one positive sample (out of 91 samples) in a wild *T. rugosa* individual east of the Perth Hills. I did not find any positive samples in South Australia, or at the Mount Mary study site. This is the first attempt at identifying the *Shingleback nidovirus 1*'s distribution between Perth and Adelaide. My results also show that the viruses found using flow cytometry were not the *Shingleback nidovirus 1*, which then suggests there is at least one new virus for this species.

Finally, I explored how the bobtail flu effects *T. rugosa* by conducting a differential gene expression analysis using transcriptomics, between those diagnosed with the bobtail flu and those suffering from major trauma. Because *T. rugosa* does not currently have a reference genome, both long read (Iso-Seq) and short read (NovaSeq) sequencing technologies were used for the analysis. I found evidence that suggests the bobtail flu seems to suppress the host's innate immune system, although further investigation is required. The suppression of the host's immune system is particularly important should the bobtail flu be confirmed in South Australia in the future, as a closely related endangered species (*Tiliqua adelaidensis*) distributions overlaps with *Tiliqua rugosa* and could potentially be at risk.

This thesis uses novel and innovative techniques to understand how pathogens, parasites, and disease effect a common reptile species. As a non-model organism there is limited viral and genetic research *on T. rugosa* in response to pathogens, parasites, and disease. This thesis creates a framework for future research in this species to expand on while providing some insights into parasites and pathogens potentially affecting a species population structure; viruses shifting in abundance with changing environmental conditions on a fine scale; shows monitoring viruses and their distributions being accessible to non-model organisms; and how a respiratory disease effects a lizards immune system using transcriptomics.