

Taking the '*poo*' out of 'pool': Participatory systems modelling as a decision-support tool for even the messiest public environmental health problems

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A thesis submitted for the degree of Doctor of Philosophy at The University of Queensland in 2019

Faculty of Medicine

#### Abstract

It is widely recognised that environmental health services and interventions operate in dynamically complex systems. Environmental health researchers and practitioners work to solve complex problems yet continue to favour methods that eschew the concept of complexity. Conventional quantitative risk assessment methods used in environmental health, while valuable, are often based on studies that examine the effects of individual environmental health outcomes, such as disease outbreaks.

In this research, I explored the applicability of participatory system dynamic modelling as a means of assisting environmental health decision-makers in the management of dynamically complex infectious diseases. Using cryptosporidiosis in South East Queensland as a case study, I applied system dynamic modelling techniques to explore the population-level drivers of *Cryptosporidium* transmission in the study area, with a particular focus on the role of public aquatic facilities (i.e. public swimming pools).

This research was conducted in three stages. This first involved an extensive review of the literature on complexity and environmental health service delivery, decision-making for complex environmental health problems, and system dynamic modelling. This review highlighted current challenges environmental health decision-makers face when developing policies and interventions and identified ways in which system dynamic models can assist in overcoming some of these.

The second stage involved a participatory system modelling exercise, informed by a series of stakeholder consultation workshops and interviews. This process identified a series of interconnected drivers and barriers to *Cryptosporidium* transmission in South East Queensland and emphasised the often-overlooked role of the primary health care sector, as well as overseas travel, in local transmission dynamics. It also uncovered multiple interconnected feedback loops within the system that cross sectoral boundaries, highlighting the need for multisectoral collaboration to address *Cryptosporidium* outbreaks. These feedback loops were captured in a causal loop diagram.

Lastly, a quantitative system dynamic model was constructed to simulate the relationships identified in the participatory system modelling exercise, and empirically test a range of policy options to improve the management of cryptosporidiosis in the study area. The modelling process identified several policy-relevant insights, including (1) unclear guidelines for the management of disease risk associated with swimming pool water, (2) the non-cyclic nature of a seemingly cyclical pattern of cryptosporidiosis notifications, and, (3) the relative effectiveness of primary versus secondary outbreak prevention strategies in the overall management of cryptosporidiosis.

The implications and contributions of this study are two-fold. The insights mentioned above call into question many commonly held assumptions about community-level *Cryptosporidium* transmission dynamics. From a local perspective, the model provides a platform for stakeholders and decision-makers to tests these assumptions in a locally-relevant context. It also uncovered previously unexplored leverage points within the system, particularly those related to the primary healthcare sector that can be used to improve the management of cryptosporidiosis. More broadly, this research contributes to the body of evidence supporting the benefits of applying system dynamic modelling to understand and manage complex environmental health problems.

## **Declaration by author**

This thesis is composed of my original work, and contains no material previously published or written by another person except where due reference has been made in the text. I have clearly stated the contribution by others to jointly-authored works that I have included in my thesis.

I have clearly stated the contribution of others to my thesis as a whole, including statistical assistance, survey design, data analysis, significant technical procedures, professional editorial advice, financial support and any other original research work used or reported in my thesis. The content of my thesis is the result of work I have carried out since the commencement of my higher degree by research candidature and does not include a substantial part of work that has been submitted to qualify for the award of any other degree or diploma in any university or other tertiary institution. I have clearly stated which parts of my thesis, if any, have been submitted to qualify for another award.

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#### Publications during candidature

**Currie, DJ.,** Smith, C., Jagals, P. (2018) The application of system dynamics modelling to environmental health decision-making and policy - a scoping review., BMC Public Health

**Currie, DJ**. (2018) Aquatic Facilities and *Cryptosporidium*: Exploring the Bigger Picture using participatory systems modelling – Oral Presentation at Public Health Australia's Public Health Prevention Conference. Sydney, Australia

**Currie, DJ**. (2017) System dynamics modelling to support environmental health decision-making: the case of cryptosporidiosis in Queensland, Australia, - Plenary presentation at the 18th Annual PhD Colloquium of the Student Chapter of the System Dynamics Society. Boston, USA

**Currie DJ**, Smith C, Knibbs L, Reid S (2017) Participatory system dynamics as a tool for knowledge mobilization in environmental health, - Poster presentation at the 29th Annual Scientific Conference of the International Society of Environmental Epidemiology, Boston, USA

Hall, N., Barbosa, M. C., **Currie, D.,** et al. (2017). Water, sanitation and hygiene in remote Indigenous Australian communities: a scan of priorities. Global Change Institute discussion paper: water for equity and wellbeing series 2207-9602, Global Change Institute, The University of Queensland, Brisbane.

Hall, N., Acosta Jaramillo, C.M., Jagals, P., **Currie, D.,** et al. (2016) Strengthening community participation in meeting UN Sustainable Development Goal 6 for water, sanitation and hygiene, Global Change Institute Working Paper, The University of Queensland, Brisbane

## Publications included in this thesis

No publications included

Contributions by others to the thesis

No contributions by others

# Statement of parts of the thesis submitted to qualify for the award of another degree

None

# **Research Involving Human or Animal Subjects**

Ethical approval to conduct this research was obtained from Queensland Health's Royal Brisbane and Women's Hospital Human Research and Ethics Committee (approval number: HREC/16/QRBW/509) and the University of Queensland's Human Research and Ethics Committee (approval number: 2016001630). Copies of the approval letters can be found in Appendix C: Human Ethics Approval Letters.

#### Acknowledgements

The 4-year journey that has culminated in this thesis has been one of the most challenging, but also rewarding, experiences of my life. I wish to acknowledge the support and guidance of the many people who have made it possible. I also wish to acknowledge The University of Queensland and the Government of Australia for providing the funding that enabled me to pursue this PhD.

Firstly, I would like to thank my supervisory team, Dr Simon Reid, Dr Carl Smith and Dr Luke Knibbs. Your guidance, feedback, enthusiasm and unwavering support has been invaluable and very much appreciated. I truly could not imagine a better set of mentors. To Simon and Luke, a special thank you for taking a chance on me when I showed up at your doors mid-way through this journey. To Carl, thank you for not only opening my eyes to the world of system dynamics, but also for always believing I could do this even when I didn't quite believe it myself.

To Dr Paul Jagals, whose early support helped lay the foundation that became this project. Thank you for introducing me to the world of environmental health.

To Dr Greg Jackson, the entire Water Unit at Queensland Health, and all the other experts I worked with over the course of this project. Thank you for taking the time to share your knowledge and expertise with me. It is your expertise that sits at the centre of this work, and without you this research would never have been possible.

To all my fellow students who have passed through room 120. Thank you for you feedback, support, and of course, friendship. A special thank you to Dwan for your encouragement, humour, and most importantly, for putting up with endless discussions of 'accidental faecal releases'. I'm not sure I could have done this without you.

To my family, my deepest gratitude is due. To my parents David and Madelaine, my parents-in-law Rosemary O'Brien and Jennifer Oades and my brother Philippe. Your love, support, and persistent encouragement to never stop learning has spurred me on throughout this journey. Also, to my late father-in-law Brendan Reynolds, who knew just how to crack a poo-joke when things got stressful. This is for all of you.

And finally, to partner Sarah, who has had to live with me through all the highs and lows that took me to get here. Thank you for your love, patience, and tireless support. Thank you for joining me on this crazy journey. You are my world.

## **Financial support**

This research was supported by an Australian Government Research Training Program Scholarship.

## Keywords

environmental health; systems thinking, system dynamics, complex systems, decision support systems, simulation, infectious diseases, *Cryptosporidium* 

## Australian and New Zealand Standard Research Classifications (ANZSRC)

ANZSRC code: 111705, Environmental and Occupational Health and Safety 40% ANZSRC code: 080605 Decision Support and Group Support Systems, 40% ANZSRC code: 160508 Health Policy, 20%

## Fields of Research (FoR) Classification

FoR code: 1117 Public Health and Health Services, 60% FoR code: 0806 Information Systems, 40%

# Abbreviations and Acronyms

Term	Meaning	
AFR	Accidental Faecal Release	
AR	Action research	
CLD	Causal loop diagram	
EH	Environmental health	
NCOS	Notifiable Conditions System	
NHMRC	National Health of Medical Research Council	
NSW	New South Wales	
ODE	Ordinary differential equations	
PAF	Public Aquatic Facility	
PCR	Polymerase chain reaction	
PHU	Public Health Unit	
PMB	Participatory model building	
QH	Queensland Heath	
QLD	Queensland	
RWI	Recreational water illnesses	
SD	System dynamics	
SEQ	South East Queensland	
WHO	World Health Organisation	

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# **Chapter 1.** Introduction

#### 1.1. Background

The environment plays a key role in the health and wellbeing of people around the world, with an estimated one-quarter of the global burden of disease in adults, and one-third in children, attributed to modifiable environmental factors (Prüss-Ürsün and Corvalán, 2006). Managing the environmental drivers of health is challenging at the best of times. Environment-and-health relationships are complex, non-linear, unstable, and difficult to define. While, in the past, environmental health management has focused primarily on understanding, and subsequently managing risk associated with single cause-effect relationships, current knowledge points to the need to adopt a much more integrated approach. This approach must account for a number of interrelated social, behavioural, economic, technological and climatic factors, all of which can influence the way the environment impacts health and wellbeing (Institute of Medicine, 2009).

A gap remains between the conceptual understanding of environmental health problems as dynamically complex phenomena and the methods and tools needed to integrate that understanding into the design, development, implementation and evaluation of policies and interventions. Commonly used risk assessment frameworks do not capture the complexity of environmental health-related infectious disease problems, nor do they provide a clear way forward for practitioners and decision-makers to act upon their findings (Briggs et al., 2009). System dynamics, a computerbased modelling approach that focuses on seeking the root causes of problematic behaviour within complex systems, is a potentially promising approach to investigate systemic problems related to environmental health service delivery. Unlike many commonly used modelling techniques that rely on statistical correlation to provide decision makers with point-estimate prediction, system dynamics models instead help users identify and quantify causal structures and relationships within a system, with the goal of improving the users' overall understanding (i.e. mental model) of system behaviour. Doing so helps identify key leverage points within systems, which can be of great use when trying to improve the effectiveness of a system. While there has been limited application of system dynamics to investigate complex problems in the context of environmental health decisionmaking, it has shown to be a successful approach for improving our understanding of complex health problems.

Using cryptosporidiosis in South East Queensland (SEQ) as a case study, this project explores the use of participatory system dynamics to help decision makers gain a better understanding of the complex system behaviours that create and sustain complex environmental health problems. This

improved understanding can be used to design more effective strategies based on leverage points within the system, which in turn has the potential to lead to improved population health outcomes

#### **1.2.** Study Rationale

System dynamics (SD) modelling, as part of the broader systems thinking approach, is being advocated by organisations such as the World Health Organisation as a tool to support healthrelated decision-making processes by strengthening decision-makers' and stakeholders' understanding of the underlying causes of complex problems and identifying leverage points for the development of effective policies (Savigny, 2009). The complex and dynamic nature of environmental health issues make them ideal candidates for analysis using system dynamics modelling, yet there has currently been only limited application within the field of environmental health. Given the success of SD in a range of fields from ecology to economics (Sterman, 2001), this research intends to explore whether or not it can be successfully applied in the field of environmental health, using cryptosporidiosis in South East Queensland as a case study.

#### **1.3.** Case Study Introduction

Cryptosporidiosis, caused by the enteric protozoa *Cryptosporidium*, is one of the most common causes of diarrhoeal disease worldwide, with increasing prevalence in high income countries (Snel et al., 2009). It has been estimated that up to 20% of cases of childhood diarrhoea in high income countries is caused by infection with *Cryptosporidium* (Mosier and Oberst, 2000). The prediction, management and prevention of cryptosporidiosis outbreaks is extremely difficult and complex due to a number of factors including its multiple transmission pathways, its persistence in the environment, and its extended asymptomatic infectious period (Rossle and Latif, 2013).

While most attempts to model cryptosporidiosis transmission have focussed on contaminated drinking water (Casman et al., 2000, Brookhart et al., 2002, Okhmatovskaia et al., 2010, Perz et al., 1998), contaminated recreational water, and in particular swimming pools, is widely recognised as a major source of transmission (Lam et al., 2014). Swimming pools are thought to be a major source of *Cryptosporidium* associated with outbreaks due several factors. These include the exposure of large numbers of people who immerse themselves and swallow water (Schoefer et al., 2008), the resistance of *Cryptosporidium* oocytes to chemical disinfectants and the small size of the organism that allows it to bypass many filtration systems (Centers for Disease Control and Prevention, 2001). In addition, the poor hygiene practices of swimmers (Yoder et al., 2008), high levels of oocytes in a stool, a low infectious dose, and the continued excretion of oocytes in asymptomatic individuals and individuals whose diarrhoea has been resolved (Desai et al., 2012) also contribute.

In Australia, there have been a number of cryptosporidiosis outbreaks, many of which have been linked to swimming pools (Paterson and Goldthorpe, 2006, Black and McAnulty, 2006, Hellard et al., 2000a, Puech et al., 2001, Stafford et al., 2000, Lemmon et al., 1996, Mayne et al., 2011, Ng-Hublin et al., 2015). Sporadic cases of cryptosporidiosis have also been linked to the consumption of unpasteurized milk (Harper et al., 2002), contact with infected animals (Ng et al., 2008, Ashbolt et al., 2003) and attendance at childcare centres (Government of Australia, 2005).

In response to the increasing number of pool-related cryptosporidiosis outbreaks, the Government of Queensland released guidelines for the control of *Cryptosporidium* in swimming pools within its Swimming and Spa Pool Water Quality and Operational Guidelines (Queensland Health, 2004). Despite these guidelines, cryptosporidiosis remains a persistent problem in the state. In particular, the number of cases of cryptosporidiosis has increased significantly over the past 3 years, with the state reporting 668, 1314, and 2037 cases annually in 2014, 2015, and 2016 (year-to-date), respectively (Government of Australia, 2016). The persistence of cryptosporidiosis within Queensland, and Australia as a whole, points to the need for better management approaches.

There are a significant number of gaps in knowledge regarding the transmission and control of *Cryptosporidium* infections, which limit the robustness and applicability of many decision-support tools. These include inadequate human exposure data, unknown relative contribution of the various transmission pathways, a lack of data on pathogen loads in various environments, as well as bias in study design that largely focussed on the investigation of outbreaks and single transmission pathways such as contaminated drinking water scenarios. Additionally, there is a dearth of integrative research that approaches the issue of cryptosporidiosis from a holistic systems perspective, addressing the wider community-level drivers of the disease, as well as the multiple exposure pathways.

#### 1.4. Research Aims and Objectives

The central aim of this project is to develop a decision-support tool using system dynamics modelling to help decision-makers uncover the underlying environmental and social feedback mechanisms that contribute to the transmission of cryptosporidiosis in South East Queensland, Australia.

The aim of this project is guided by the following three specific research questions:

- Research Question 1: What are the population-level drivers of *Cryptosporidium* transmission in South East Queensland, and how do these drivers dynamically interact to create the trends in notified cases of cryptosporidiosis that have been observed in the region?
- Research Question 2: What policies or interventions could be used to more effectively reduce the incidence of cryptosporidiosis in South East Queensland?
- Research Question 3: Can system dynamics modelling add value as a decision-support tool for environmental public health decision-making processes, in particular in the management of cryptosporidiosis in South East Queensland?

## 1.5. Study Design

The construction of the system dynamics model will use group model building techniques, and will have 4 iterative methodological phases (as proposed by Maani and Cavana (2007)): (1) problem structuring, (2) causal loop modelling, (3) Dynamic modelling, (4) scenario planning and modelling (figure 2).



Figure 2: model building process according to Maani and Cavana (2007)

Data and information used to develop the system dynamic model was collected primarily through a series of workshops and interviews with key stakeholders. A problem articulation workshop, which was attended by academic and government stakeholders and experts, was held at the beginning of the project. This workshop involved a series of divergent and convergent exercises aimed at identifying important variables and relationships within the system. The variables and feedback structures identified in the workshop, together with information and theories identified in the

literature, will form the basis of the dynamic hypothesis for the model. Following the consultation interviews, the causal loop diagram was translated into a system dynamics stock-and-flow model. The model was parameterised with available government data, and in the absence of data, assumptions were made based on expert opinion.

Once parameterised, the model was validated using several tests including comparing model behaviour with known behaviour, conservation of matter tests, extreme conditions tests and sensitivity tests. The model was then used to assess the potential impact of a variety of different policy-related scenarios on the dynamics of cryptosporidiosis in the region.

#### 1.6. Overall Contribution of the Research

This research intends to have two main contributions. The first is the creation of the first model of socio-environmental drivers of cryptosporidiosis transmission, as well as the first system dynamics model of infectious disease transmission related to public aquatic facilities. This model will improve understanding of the causal relationships between population-level drivers of cryptosporidiosis outbreaks in an Australian context. The goal of this model will be to improve the management of cryptosporidiosis in South East Queensland. The hope is that the insights that emerge from the model will translate into policies and interventions that reduce the burden of disease related to cryptosporidiosis. The second is to improve knowledge of the applicability of systems thinking and system dynamics to environmental health. The utility of system dynamics to support decision making has been established in a variety of fields yet remains largely untested in the context of environmental health infectious disease issues.

#### 1.7. Thesis Structure

This thesis contains 8 chapters as outlined in Table 1.1 that are divided into 5 sections, (i) Introduction, (ii) Background and Literature Review, (iii) Research Methodology and Case Study Context, (iv) Inquiry and Findings and (v) Conclusion.

The first section includes the introduction (Chapter 1) that outlines the aims, objectives and rationale of the study, provides a brief description of the case study and research methods and outlines the structure of the thesis.

The second section includes a literature review (Chapter 2) that examines and appraises the key concepts underpinning this thesis, including environmental health, systems theory and models of decision-making.

The third section also includes two chapters (Chapter 3 and 4). Chapter 3 also introduces the case study and gives background information on cryptosporidiosis and South East Queensland. Chapter 4 outlines and defends the study methodology and provides background information on the practical application of the system dynamics modelling methods.

The fourth section includes three chapters (Chapter 5, 6 and 7). Chapter 5 describes how participatory systems thinking was used to create qualitative systems maps of *Cryptosporidium* transmission dynamics in South East Queensland. Chapter 6 builds on the work of the previous chapter and outlines the transformation of the systems maps into a system dynamics simulation model. Using that system dynamics model from the previous chapter, Chapter 7 describes the policy analysis process and insights gained from using a system dynamics model to simulate *Cryptosporidium* transmission dynamics.

The fifth section (Chapter 8) brings a conclusion to the thesis and proposes questions and implications for further research.

Section	Chapters
SECTION I - Introduction	Chapter 1: Introduction
SECTION II - Literature Review	Chapter 2: Literature Review
SECTION III - Research Methodology and Case Study Context	Chapter 3: Case Study Background Chapter 4: Methodology and Methods
SECTION IV - Inquiry and Findings	Chapter 5: Conceptualizing the Problem Using Causal Loop Modelling Chapter 6: Design, testing and validation of a system dynamics model of cryptosporidiosis Chapter 7: Risk-management strategies for Cryptosporidium transmission in <i>South</i> East Queensland
SECTION V - Conclusion	Chapter 8: Conclusion

Table 1.1:	Outline	of thesis	structure
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# **Chapter 2.** Literature Review

#### 2.1. Complexity in Environmental Health

#### 2.1.1. Environmental Health Practice

The environments in which we live, work and play have a significant impact on our health and wellbeing. The field of environmental health (EH), which traditionally sits in the nexus between environmental management and public health, is the domain responsible for managing the complex relationships and interactions between health and the environment.

The World Health Organization has defined environmental health as follows:

"Environmental health addresses all the physical, chemical, and biological factors external to a person, and all the related factors impacting behaviours. It encompasses the assessment and control of those environmental factors that can potentially affect health. It is targeted towards preventing disease and creating health-supportive environments. This definition excludes behaviour not related to environment, as well as behaviour related to the social and cultural environment, and genetics" – World Health Organization (2015)

Although the WHO definition of Environmental Health is widely accepted, other definitions of environmental health do exist, often with varying acknowledgements of the impact of the "total environment" (i.e. physical, biological, social, cultural, etc. environments) on human health.

While environmental health research and practice has primarily focused on exposure to environmental hazards, it is increasingly being recognised that an exploration of distal determinants is needed to truly understand their root causes. Upon closer inspection, it often becomes clear that EH problems are not limited to a single agent, media, vector, exposure or health effect. Most environmental health problems simply represent the outcome of complex web of causality that causes the problematic situation to emerge over time (Briggs, 1999). Beyond these problematic events and situations lies a system of farther-reaching drivers and pressures, such as environmental degradation, population growth, poverty, climate change, and rapid urbanization which, while not necessarily set solely in the domain of environmental health, must be given significant consideration when investigating environmental health impacts and risk. While, in the past, environmental health management has focused primarily single cause-effect relationships, current knowledge points to the need to adopt a much more integrated approach. There is a growing need to stop examining exposure and effect relationships stripped from the broader political, social and environmental systems that created their existence in the first place(Krieger, 2008). This approach must account for a number of interrelated social, behavioural, economic, technological, political and climatic factors, all of which can influence the way the environment impacts health and wellbeing (Institute of Medicine, 2009).

#### 2.1.2. Infectious disease management within environmental health

The scope of environmental health practice is ever-widening. Historically, environmental health research and practice has focused predominantly on exposure to chemical and physical agents (Feingold et al., 2010). Recently, there has been growing recognition of the crucial role that the environment plays not only in the transmission of infectious diseases, but also in the emergence and re-emergence of infectious diseases. The World Health Organisation's environmental burden of disease study (Prüss-Üstün and Neira, 2016) estimated that adverse environmental conditions contribute between 15% and 30% of all infectious-disease related disability adjusted life-years, representing enormous impact on the health and wellbeing of populations around the world. These advances in our understanding of the linkages between the environment and infectious diseases means that environmental health practitioners are playing an increasing role in their management and control.

In a review of the environmental determinants of infectious disease, Eisenberg et al. (2007) present a framework (shown in Figure 2.1) outlining how both proximal and distal environmental factors/characteristics influence and shift the transmission cycle of infectious diseases, which ultimately affect the overall prevalence and severity of many infectious diseases within a region. This framework further highlights that, like other environmental health management problems, infectious disease problems from complex webs of causal factors.

To-date, much of the environmental health research and practice on infectious disease has centred on environmental and socio-environmental factors affecting individual exposure, with considerably less attention given to factors affecting transmission (Woolhouse, 2011). That which has focused on transmission has tended to focus on proximal environmental factors that directly influence disease transmission processes (Eisenberg et al., 2007). Despite growing recognition of the importance of more distal socio-environmental factors such as land use, transportation, water supply, hygiene, etc. relatively little attention has been given to investigating the impact of both proximal and distal environmental and socio-environmental factors on infectious disease transmission. While an understanding of the factors that influence an individual's risk of being exposed to an infectious disease is an important part of the policy puzzle, a comprehensive understanding of the factors which contribute to the transmission of the disease is equally important. The limited transmissionrelated evidence-base poses a challenge for environmental health decision-makers to design and implement effective interventions.



Figure 2.1: Framework of environmental drivers of infectious disease (source: Eisenberg et al. (2007))

From an environmental health perspective, addressing the drivers of infectious disease transmission often requires long-term investment in programs that may not appear directly related to health or disease transmission. For example, factors such as overcrowding, inappropriate disposal of solid waste, poor hygiene, and exposure to flooding conditions have all been linked to disease transmission and poor health (Australian Indigenous HealthInfoNet, 2008), and all of which require sustained and long-term investment to be corrected.

This integration of environmental health and infectious disease management presents several challenges for environmental health practitioners. Classic toxicological models, which were the traditional mainstay environmental health, most often approach toxicodymanics at the level of the individual, and don't typically address hazards whose quantity and virulence have the potential to change quickly and non-uniformly. They are also poorly-equipped to address the complexity of proximal and distal environmental, social, institutional and technological drivers of these disease, nor the feedback cycles which modulate the diseases (Eisenberg et al., 2007). Conceptual and analytical tools are needed which can help environmental health practitioners address the growing complexity of managing the environmental infectious disease problems.

#### 2.1.3. Complex systems

As discussed above, complex environmental-health-related infectious disease management problems should not be thought of as individual problems that arise and exist in isolations, but rather as the output of a much broader 'system' of causal factors and relationship. While many definitions of what a 'system' is exist within the literature, the early definition proposed by Hall and Fagen (1956) provides a good outline of the fundamental components of a system:

#### "A system is a set of objects together with relationships between the objects and between their attributes"

Hall and Fagen's definition focuses on the building blocks of a system, objects, attributes and relationships. Objects can be both tangible and intangible and represent the various components or variables within a system. Attributes are the properties and characteristics of the objects, which can vary within and amongst objects. Relationships are the connections and interactions between objects. While this definition provides some clarity on what a system is made up of, it does fall short in terms of capturing the essence of what a system is. The following definition by Ackhoff (1994) of what a system is fills this gap:

"A system is a whole consisting of two or more parts (1) each of which can affect the performance or properties of the whole, (2) none of which can have an independent effect on the whole, and (3) no subgroup of which can have an independent effect on the whole. In brief, then, a system is a whole that cannot be divided into independent parts or subgroups of parts"

Taken together, these two definitions explain what a system is and what it is made up of. A system is made up of objects, each with varying attributes, which are connected by relationships. While the components, attributes and relationships within a system are important, their whole is than just the sum of their parts. Systems can be mechanical, such as a car or a television, organismic, such the human heart or an ecosystem, or social, such as an organisation or a community (Ackoff, 1994).

Systems themselves can be simple, complicated or complex. The distinction between these is not based solely on the number of components within them, but also on the way that the components interact. On one end of the spectrum are simple systems, which are made up of a limited number of components that interact in well-defined and well-understood ways. In the middle of the spectrum are complicated systems, which are made up of many different components, whose interactions may not be easily understood. Though they vary in the number of components and interactions within the system, the behaviour of both simple and complicated systems is well predictable. Knowing the past behaviour of simple and complicated systems is a good predictor of future system behaviour.

At the other end of the spectrum are complex systems. Like complicated systems, complex systems are made up of many interacting components, but the behaviour of the system is not easily predictable, and the behaviour of the system changes and evolves over time. In addition to the dynamic nature of complex systems, Ladyman, Lamber and Wiesner (2013) outline 7 key characteristics that complex systems have in common:

- Nonlinearity: system outputs are not directly proportional to the inputs, demonstrating disproportional cause and effect
- Feedback: the output of a particular event within a system depends on the past and future state of that event
- **Spontaneous order**: system structure is not random, nor does it arise from intentional design or planning, but rather from the interactions of system components, which are governed by basic principles and rules.
- Robustness and lack of central control: system structure is organised in such a way that a shock in one area of the system do not automatically impact the stability of the whole system. This is possible because control of the system is not governed by a central entity, but rather by adaptive self-correcting feedback mechanisms within the system.
- **Emergence**: higher order system behaviour arises from the collective interactions of system components, but cannot be explained solely by examining the components alone
- **Hierarchical organisation**: systems are comprised of multiple structural levels (sub-systems), each interacting with the levels above and below it, and sharing common causal regularities.
- Numerosity: systems which are made up of a large number of parts which engage in a large number of interactions.
- 2.1.4. Environmental health problems as complex systems problems

Applying Ladyman, Lamber and Wiesner's characteristics of complex systems (as outlined in section 2.2), to the environmental health systems which produce many of the wicked environmental health problems we see today demonstrates how these systems can be considered complex systems. **Nonlinear** cause and effect relationships are commonly encountered when investigating the relationship between exposure to environmental health hazards and morbidity and mortality. An example of this is the health effect of parasitic infections. A non-linear relationship may be seen between parasite load within the infected individual and the health effect, due to factors such as nutritional status or past infections (Hochberg, 1991).

**Feedback** mechanisms are also commonplace within environmental health systems. Feedback mechanisms drive the growth of vector-borne disease epidemics such as malaria or yellow fever, with the number of infected mosquitos driving up the number of infected humans. This, in turn, drives up the number of mosquitos. Feedback mechanisms also drive the way people interact with the environment. For example, increases in large forest fires in the United States prompted fire suppression activities, which caused the progressive accumulation of flammable leaf litter in forests, which in turn lead to increases in large forest fires (Donovan and Brown, 2007).

The concepts of **spontaneous order, emergence, robustness** and **hierarchical organisation** can be used to describe many of the systems from which environmental health problems emerge. The robustness of environmental, biological, economic, health and social systems provides resilience against variability and shocks through both their ability to resist change, as well as their capacity to adapt to it. Fluctuations in the system, as well as the system being exposed to shocks of perturbations, facilitates self-organization and evolution of the system. Ecosystems, for example, exemplify these concepts as the structures and interactions which govern ecosystems emerge not from a central control, but rather from both salient and inconspicuous interactions between different ecosystem components; all of which happen according to general principles (such as supply and demand). Shocks to complex ecosystems, such as a forest fires or floods, expose the system to perturbations without necessarily destroying it, due to the systems' inherent robustness. The hierarchical organisation of ecosystems (as shown in Figure 2.2) can be elucidated by observing how the interactions between individual components within the ecosystem, such as individual rodents spreading the seeds of plants they have consumed, shape the dynamics at higher (population and community) level.



**Figure 2.2:** Example of the hierarchical organisation of ecosystems (adapted from (Reuter et al., 2010))

Much of the difficulty in managing complex environmental health problems, such EH-related infectious disease management problems, is because they emerge from complex systems. Addressing these complex problems requires environmental health professionals to identify and account for the web of interconnected environmental, social, institutional, economic and technological factors that are embedded in the dynamic fabric of the communities in which the work. Understanding the factors that make these systems 'complex' is a crucial component to uncovering the source of these complex problems.

#### 2.2. Decision-making for complex problems under uncertainty

Rooted within environmental health service delivery is the need for EH professionals to investigate environmental public health problems and make decisions regarding the management and control of environmental health hazards. The complex and multi-disciplinary nature of environmental health problem means that EH professionals are often encounter many unknowns making problem-related decisions.

Decision-making, whose overall premise is to either make a choice that permits something to happen, or prevents something from happening, occurs many times throughout planning, management and evaluation processes. More specifically, a decision-making situation exists when 1) a problem exists, 2) two or more possible actions are available, 3) there is some understanding of the objective of the decision and its connection to the problem, and 4) it is possible to estimate or predict the outcome of the decision (Skyttner, 1996). Fundamental to decision-making is the evaluation possible options, each having associated outcomes and consequences, and the selection of an alternative from the considered options.

The reality of environmental health management is that decisions often must be made despite uncertainty. Uncertainty, which Brown (2004) defines simply as "imperfect knowledge", generally fits within one of two categories, outcome uncertainty, and problem-based uncertainty.

Outcome uncertainty is related to the decision-maker's ability to predict the outcome of their decision. Decision situations typically exist within one of the following three categories of outcome-related conditions:

- **Decision making under certainty:** Decision-maker has a complete understanding of each actions (and their alternatives) and the outcomes of the actions on the problem.
- **Decision making under risk:** Decision-maker knows the possible outcomes and the probabilities that each will occur but does not know which outcome will actually occur.

• **Decision making under uncertainty:** Decision-maker cannot predict the outcomes of the possible actions or the probability that the outcomes will occur.

With decisions under risk, the decision maker has sufficient information to understand the possible outcomes of a decision, and assign each outcome with a probability of occurring, but cannot ultimately know which outcome will occur. Under uncertainty, the decision maker also cannot confidently predict the possible outcome of a decision.

Problem uncertainty is related to the decision-makers' ability to understand the nature of the problem itself. This uncertainty can be broadly characterized as originating for one or more of the following three groups (Committee on Decision Making Under Uncertainty et al., 2013):

- Variability and heterogeneity: Also called aleatoric uncertainty, this type of uncertainty refers to natural variations in factors such as susceptibility and exposures that exist within environmental health system. These make it difficult to quantify or predict the effects of decisions on populations.
- **Systemic uncertainty:** Also known as epistemic uncertainty, this type of uncertainty is related to a lack of consensus or knowledge regarding the system of cause and effect relationships, or the parameters within a system.
- Deep (process) uncertainty: Deep uncertainty is a more fundamental lack of understanding of the underlying environmental or health process, or a lack of method to characterize the process, and/or disagreement on the desirability of various options.

Aleatoric uncertainty, which can also be thought of as analytical uncertainty, is the type of uncertainty that gets the greatest focus in EH-related studies, likely because it is the type of uncertainty that is easiest to detect (Briggs et al., 2009). Uncertainty due to variability within the population or environment can be managed to some degree by replicating studies, collecting and linking large datasets, performing sensitivity analysis on models, and using statistical analysis to account for error and variation.

Despite potentially having greater implications, systemic and process uncertainty, which can be thought of as conceptual uncertainty, receive far less discussion in EH-related studies. Conceptual uncertainty centres on whether the correct question is being asked. Briggs (2009) argues that widely-taken positivist paradigm – where 'facts' and 'values'/'perceptions' are fundamentally separate and the goal is the development of general laws and theories – may make it difficult for us to detect and acknowledge conceptual uncertainty. By failing to acknowledge that one's perspective

fundamentally shapes one's observation of 'facts', we are unable to see things from alternative perspectives, and thus may be unable to properly conceptualize the problem. This is particularly relevant in the context of complex problems, where non-linear causality and feedback cycles may make it difficult to adequately consider the important causal factors and relationships driving the problem.

# 2.2.1. Decision support tools in Environmental Health

Decision support tools provide decision-makers with an avenue to reduce uncertainty in the decision-making process, The idea of 'decision support' encompasses tools or approaches that can help guide decision-making by providing actionable information that can be used to examine the trade-offs between policy options (Walker et al., 2003).



Figure 2.3: Role of decision-support tools in the decision-making process

Figure 2.3 outlines where decision-support tool typically fit within the decision-making process. Their use is generally prompted by an upcoming policy- or intervention-related decision. Ideally, the tools are informed not only by the policy process, but also by the objectives of the stakeholders, the research evidence, and the broader context in which the decision is being made. Historically, risk assessment has been one of the key problem-focused decision-support tools used by environmental health decision makers<sup>1</sup>. Risk assessment provides decision-makers with an estimated probability and magnitude of effect from exposure to a particular environmental hazard given a certain level of exposure. In infectious disease management, models, and particularly regression-based statistical models, are one of the key decision support tools. The following section provided a brief overview of the use and applicability of these tools in the context of complex environment-related infectious disease management problems.

#### 2.2.1.1. Risk assessment

One of the most common decision-support tools used in environmental health management is risk assessment. Risk assessment is a process-based tool where information about a particular hazardous substance, process or event is collected and qualitatively or quantitatively analysed in relation to dose, exposure and susceptibility of various receptors (Willows et al., 2003). The outcome of a risk assessment is a measure of the predicted probability and magnitude of effect that will result in exposure to the hazard. In most cases, risk assessment considers one hazard at a time and assumes independence of outcomes, rarely considers cumulative exposure to many hazards, or the effect of human behaviour beyond that directly related to the exposure (Briggs et al., 2009).

Risk assessment process are generally built in such a way to account for variability and heterogeneity (problem uncertainty) and a degree of decision uncertainty using highly conservative estimates with large margins of error. By greatly over-estimating the magnitude and likelihood of effect, the risk assessment accounts for highly susceptible individuals. The usefulness of risk assessment is that it provided decision-makers with a future prediction of the likely outcome(s) related to a hazardous environmental health substance, process or event, given a set of circumstances. The remaining uncertainty therefore is primarily focused on whether or the circumstances that would produce that outcome will occur or not.

The UK Government's Guidelines for Environmental Risk Assessment and Management (Áine Gormley, 2011) outlines the following key principles of risk assessment in the context of EH problems:

<sup>&</sup>lt;sup>1</sup> While other decision support tools such as environmental impact assessment, health impact assessment, cost-benefit analysis and life cycle assessment are also commonly used in environmental health decision making, they are primarily project or solution focused, and are generally ill-suited for decision support in cases where the source of the problem is unclear.

- the importance of correctly defining the actual problem at hand;
- the need to screen and prioritise all risks before quantification;
- the need to consider all risks in the options appraisal stage; and
- the iterative nature of the process.

When the above-mentioned principles are followed, the process of decision-making under risk can be quite simple provided there is sufficient information about the likelihoods of events and consequences to make reasonable risk estimates, and that the risks related to all relevant hazards have been appropriately characterised. In reality, this is rarely the case with EH problems, which can leave decision-makers with significant ambiguity.

Furthermore, much of risk assessment relies on classic toxicological paradigm, which usually approach toxicodymanics at the level of the individual, and don't typically address hazards whose quantity and virulence have the potential to change quickly and non-uniformly. They are also poorly-equipped to address complexities such as disease transmission or the feedback cycles which modulate disease (Eisenberg et al., 2007).



Figure 2.4: The classic toxicological paradigm

An additional challenge to effectively using risk assessment as decision-support tool in the context of EH-related infectious disease problems relates to the first principle in the above-mentioned guidelines; the importance of correctly defining the actual problem at hand. In this context, a correctly defined problem requires that the problem itself has little-to-no systemic or process uncertainty. This is rarely the case with IDM problems. Because of this, the traditional risk-assessment-driven approach to EH decision-making is ill-suited to many of the complex EH problems practitioners and decision-makers face.

# 2.2.1.2. Infectious Disease Models

While rarely used in the field of environmental health, infectious disease models have been widely used within the public health and clinical medicine spheres as decision support tool. The term

'model' is used to describe a simplified representation of a reality that is built specifically to answer a question. While there are many different types of models used routinely in public health, Manheim et al. (2016) identified five main types of models commonly used to model infectious disease in the public health sector, divided into two main categories. The first category, statistical models, includes the following two types of models:

- **Regression-based models**: These models are often considered the 'standard' statistical approach to modelling infectious disease. They involve using statistics to identify best-fit between predictor and outcome variables and can be used even without an in-depth knowledge of the underlying causal structures. They are also the centre of the 'risk-factor' approach to epidemiology where individual 'risk factors' for disease are identified from historic case data.
- Machine-Learning models: These complex models compare input and output data across several parameters to 'learn' the structure of the relationships between variables, and subsequently predict future outcomes. While extremely useful in disease forecasting, these models can be highly complex and require large volumes of data.

The second category is theory-based models, and includes the following three types of models:

- **Compartmental population models**: These relatively-simple probabilistic or deterministic models divide the population into compartments based on disease status and are designed to capture disease transmission within a population. For example, the population may be divided into susceptible or infectious depending on disease status. System dynamics model are a more detailed and comprehensive form of compartmental population model.
- Event-based micro-simulation models: These individual-level models offer a more complex description of disease dynamics by represent the dynamics of discrete events that an individual may experience. For example, instead of simply separating the population based on infectious or not infectious, they may instead include individual level measures such as the infectiousness of a particular individual.
- Agent-based models: Like the micro-simulation models, agent-based models are individual-level, but instead of modelling events, they model the interaction between individual behaviour and disease using a set of pre-defined rules. For example, models of this type could model infectiousness as the number of contacts a sick individual meets in a given period of time.
Each type of model has its advantages and disadvantages, depending on the context in which is it used, and the intended used of the model's outputs. In general, statistical-type models excel at predicting future disease outbreaks, whereas theory-type models excel at helping understand the problem of interest and identify and analyse potential interventions to address the problem. Briggs et al.(2016) and Manheim et al. (2016) compared the advantages and disadvantages of each type of model in more detail, the results of which are summarised below in Table 2.1.

	Advantages	Disadvantages
Regression- based models	Work well when there is poor understanding of disease	Does not incorporate or communicate causality
	dynamics computationally fast	Requires significant data to be useful
		Cannot be easily used to compare interventions
	Work well when there is poor understanding of disease	Does not incorporate or communicate causality
Machine- Learning models	dynamics Can give accurate forecasts	Model structure can be difficult to interpret and communicate.
		Requires significant data to be useful
		Limited applicability in planning and evaluating intervention options
Compartmental Population	Useful for comparing interventions	Simplistic structure can lead to less accurate forecasts
Models /System Dynamics models	Computationally fast	Deterministic form can struggle to
	Structure and behaviour easy to communicate	accommodate variability or uncertainty
Event-based micro-simulation models	Useful for planning and comparing interventions	Model structure can be complex and difficult to communicate
	Flexible and precise	Computationally slow
	Can incorporate empirical data and expert opinion	Can be challenging to design
	Weill-suited for complex situations	

## Table 2.1: Advantages and disadvantages of model-based decision support systems

	Advantages	Disadvantages
Agent-based models	Useful for selecting optimal intervention(s) Well-suited for complex situations	Requires significant understanding of underlying theory driving individual behaviour Computationally slow and requires substantial computational power Model structure can be complex and difficult to communicate. Limited ability to forecast disease occurrence

Even in the presence of uncertainty and significant assumptions, quantitative models such have advantages over using our mental models for judgement-based decision-making. Quantitative models are transparent – allowing their underlying ideas and assumptions to be examined and critiqued. They also allow for potential interventions to be tested in a virtual world before being implemented in a real-world setting (Woolhouse, 2008).

## 2.3. System dynamics modelling for EH decision-making

System dynamics (SD) modelling, which has its roots in systems thinking, is an additional modelling-based decision support tool. System dynamics modelling uses similar nomenclature and structure as compartmental population models, but incorporates the more complex description of disease, community and environmental dynamics of micro-simulation models. The research associated with this literature proposed that system dynamics model, used in conjunction with other systems thinking techniques, may be another decision-support tool well suited for decision-makers confronted with a complex infectious disease problem. The following section explores systems thinking, system dynamics and their potential application to complex environmental health and infectious disease problems.

## 2.3.1. Theoretical basis of systems thinking

The concept of a 'system', as discussed in section 2.2.1.2, is rooted in "systems theory". The creation of "systems theory" has been credited to biologist Ludwig von Bertalanffy, who theorized in the late 1920's that investigating a single part of a living system cannot provide a complete understanding of that phenomena as it gives little-to-no understanding of the coordination of the processes within the system. He then theorized that it was therefore more important to determine the laws which govern biological systems than the individual components (Bertalanffy and Woodger, 1934). This contrasted with the prevailing theory at the time (Descarte's "scientific method"),

which viewed that systems were made up components which could be broken up into parts and investigated independently of one another, and systems could be described by adding all of the components of the system in a linear fashion. Descarte's "scientific method" neglected to address how the components of a system interact with one another, and the impacts that non-linear components could have on one another(Bertalanffy, 1972). Von Bertalanffy later solidified his theory by saying:

"There exists models, principles and laws that apply to generalized systems or their subclasses irrespective of their particular kind, the nature of the component elements, and the relations of "forces" between them. We postulate a new discipline called general systems theory (Bertalanffy, 1968)."

Von Bertalanffy's theory recognized that when investigating a living system, the system as a whole is greater than the sum of its parts.

2.3.2. Principles of systems thinking

Rather than being a single field, systems thinking is both a paradigm and a problem-solving approach. While definitions of systems thinking vary from extremely simple to highly complex depending on the field of study in which it is used, Arnold and Wade proposed the following multi-disciplinary definition of systems thinking:

"Systems thinking is a set of synergistic analytic skills used to improve the capability of identifying and understanding systems, predicting their behaviours, and devising modifications to them in order to produce desired effects. These skills work together as a system (Arnold and Wade, 2015)."

As a paradigm, systems thinking draws on several overarching principles that provide a framework for conceptualizing the world. While not an exhaustive list, the following foundational principles were identified as key components of the systems thinking paradigm:

**Holism**: The principle of holism is best summed up by Aristotle said "the whole is greater than the sum of its parts; the part is more than a fraction of the whole"(Hitchins, 2009). The principle of holism is essential in systems thinking as it requires systems thinkers to consider all parts of a systems as being connected, meaning that no one part of the system can be examined in isolation of the system as a whole.

**System as the cause**: This principle explains that problems that systems experience are most often created internally within a system, as opposed to being created by some external influence (Maani and Maharaj, 2001). When taking a systems thinking approach, one must look inwards for the

source of the problem, as opposed to blaming the problematic behaviour on something being driven by an external source that is outside your control.

**Closed-loop**: Linked to the principle of "system as the cause" the closed-loop principle maintains that causality does not run one way, but rather is reciprocal (Richmond, 1994). The outputs of a system will eventually come back around and become inputs of that same system (i.e. the ends can influence the means).

**Operational thinking**: This principle simply means that when examining causality, one must not focus solely on the fact that casualty or influence exists, but also on the mechanisms that brought about that causal relationship (Richmond, 1994). This principle aims to draw our attention toward the architecture and the interconnectedness of factors within a system.

Beyond providing an alternative point of view for approaching and understanding <u>problems</u>, 'systems thinking' is also a functional skill-set. Expanding on their definition of systems thinking (listed above), Arnold and Wade built on works previously done based primarily on the works of Sweeny and Sterman (2000), Hopper and Stave (2008), and Plate and Monroe (2014) and identified 8 key functions/elements of systems thinking:

- 1. Recognizing Interconnections
- 2. Identifying and Understanding Feedback
- 3. Understanding System Structure
- 4. Differentiating Types of Stocks, Flows, Variables
- 5. Identifying and Understanding Non-Linear Relationships
- 6. Understanding Dynamic Behaviour
- 7. Reducing Complexity by Modelling Systems Conceptually
- 8. Understanding Systems at Different Scales

When viewed together, the underlying principles and key functions of systems thinking provide practitioners and decision-makers with a set of tools for conceptualizing complex problems.

2.3.3. System dynamics approach

Many scientists and researchers have extended Von Bertalanffy's *General Systems* far beyond the realm of biological sciences into many other disciplines including economics, business, health and the social sciences. In particular, in the late 1950's, Jay W. Forrester's developed a theory, which

builds on Von Bertalanffy's theory, and pioneered the field of System Dynamics (then called Industrial Dynamics) (Forrester, 1958). System Dynamics is a policy-oriented computer-assisted modelling approach that is used to understand and manage complex systems that continuously change over time. It can be used to analyse any dynamic system that is characterized by interdependence, information feedback and mutual circular causality (System Dynamics Society, 2015). The system dynamics society<sup>2</sup> has defined the key features of the system dynamics approach as:

- "Defining problems dynamically, in terms of graphs over time.
- Striving for an endogenous, behavioural view of the significant dynamics of a system, a focus inward on the characteristics of a system that themselves generate or exacerbate the perceived problem.
- Thinking of all concepts in the real system as continuous quantities interconnected in loops of information feedback and circular causality.
- Identifying independent stocks or accumulations (levels) in the system and their inflows and outflows (rates).
  - Formulating a behavioural model capable of reproducing, by itself, the dynamic problem of concern. The model is usually a computer simulation model expressed in nonlinear equations but is occasionally left unquantified as a diagram capturing the stock-and-flow/causal feedback structure of the system.
  - Deriving understandings and applicable policy insights from the resulting model.
  - Implementing changes resulting from model-based understandings and insights" (System Dynamics Society, 2015).

## 2.3.4. Key concepts in system dynamics

## 2.3.4.1. Feedback

One of the driving concepts of system dynamics is feedback. Feedback, also known as circular causality, is a process that occurs when the output of a particular event depends on the past and future of that event (Sterman, 2000a). In other words, the outputs of a system will eventually come back around to their point of origin and become the inputs of the system. An example of a feedback loop is the human body's effort to maintain a constant temperature. As the body senses that its internal temperature is rising it begins to sweat, which causes the temperature to drop. Once the

<sup>&</sup>lt;sup>2</sup> The system dynamics society is the leading international organisation devoted to promoting the use and advancement of system dynamics modelling around the world.

optimal temperature has been reached, the body stops sweating until the body temperature rises again.

Two types of feedback exist within systems; positive and negative feedback (Figure 2.5). Positive feedback refers to loops where system behaviour causes the initial action to be reinforced. Despite the name "positive", these loops can be either vicious or virtuous cycles, as they enhance the action in whatever direction the change is imposed. Within a system, reinforcing loops cause accelerated growth or decline, which can have a destabilizing effect on the system. Oppositely, negative feedback refers to loops where system behaviour causes the initial action to self-correct or balance out. These loops seek to return the system to equilibrium.



Figure 2.5: Positive (reinforcing) and negative (balancing) feedback loops

As complex systems are made up of a network of interconnected feedback loops whose interactions generate and govern the overall behaviour of the system, understanding the structure, behaviours and interactions of the various feedback loops within a system is crucial to understanding the system as a whole.

## 2.3.4.2. Stock and flows

Stocks and flows are the essential building blocks of systems. In broad terms, stocks represent the current state of the system, and flows represent the rate of change within the system. Stocks are a visual representation of key variables within a system where accumulation (both positive and negative) and storage takes place and form the basis upon which decisions or actions about a system or its components are made. The current value of a stock is dependent on the previous values the stock has possessed, as stock are only able to change over time. A bank account is a simple example of a stock, as money can accumulate, be stored and be withdrawn at varying rates. The only way a stock can change is through flows, which control the rate at which things are added or withdrawn from a stock over time. In the absence of a flow, or when the value of a flow is 0, the value/contents of a stock are static. In the bank account example, the money deposited and the money withdrawn are both examples of flows associated with the bank account.

### 2.3.4.3. Delays

Delays are a fundamental component of dynamic behaviour in most systems. In real-world systems, the transfer of information and materials through a system takes time, making them an inherent component of most flows. While some delays are so short they can be considered negligible, other delays can be years. Examples of delays include time between ordering and receiving a product, between exposure to a hazard and the onset of symptoms, between recognising a hazard exists and initiating the behaviour to avoid the hazard, and between the emission of greenhouse gases and a perceivable change in the climate.

## 2.3.4.4. Endogeneity

Another key concept within system dynamics is the concept of endogeneity, meaning that system dynamics seek to find explanations for phenomena that lie within the system, as opposed to behaviours external to the system (Sterman, 2000a). Because of that, system dynamics model must be built in such a way that the behaviours and interactions which govern the system must fit within the defined system boundary (Richardson, 2011). Forrester described this principle as:

"In concept, a feedback system is a closed system. Its dynamic behaviour arises within its internal structure. Any action which is essential to the behaviour of the mode being investigated must be included inside the system boundary (Forrester, 1968)"

The overarching goal of system dynamics is to uncover and understand the behaviour of endogenous variables within the system. This way of thinking switches the perspective that the behaviour of a system is governed primarily by factors outside our purview and control, to a view that both the problems and solutions lie within the internal structure of the system (Richardson, 2011). By focusing on endogenous sources of system behaviour, system dynamics allows practitioners to explore system structure and patterns of interaction between variables, and ultimately develop and model policy changes that alter these structures and patterns in order to change system behaviour.

#### 2.3.5. System Dynamics for Environmental Health Decision-making

"Managers are not confronted with problems that are independent of each other, but with dynamic situations that consist of complex systems of changing problems that interact with each other... Managers do not solve problems, they manage messes" - Russell Ackoff (1979) The 'wicked' nature of environmental health management problems, which are often constantly changing and are almost always intertwined in a web of different political, cultural, social, economic and environmental structures (Kreuter et al., 2004), make them ideal candidates for analysis using a system dynamics approach. The integrated nature of the system dynamics approach allows for the inclusion of all the basic elements of the ecological approach when creating both qualitative and quantitative models, such as disease prevalence, health, risk and lifestyle behaviours, environmental conditions, and resources that provide health and social services or are involved in health-related social transformation (Homer and Hirsch, 2006).

As part of this research project (but not directly included in this thesis), a systematic scoping review of the application of system dynamics to complex environmental health problems (Currie et al., 2018) was conducted. The review identified only 15 published studies or report between 2000 and 2016 where system dynamics modelling had been used to inform environmental health policy or decision-making. The review found that a system dynamics approach has been used, in a limited but increasing capacity, to analyse a variety of different environmental health problems, such as designing sustainable strategies for managing contaminated land (McKnight and Finkel, 2013), investigating the health impacts of sea level rise (Diaz et al., 2012), understanding the impacts that that traffic congestion has on air pollution (Armah et al., 2010) and modelling the population health impacts of different forms of energy production (Diaz et al., 2011). While the majority of applications originated from within the health sector (n = 6), with the remaining came from other sectors including transport, public utilities, water, housing, food, agricultural, and urban and regional planning sectors. This highlighted the multi-sectoral nature of environmental health. No studies were identified that used system dynamics to inform policy related to water quality and infectious diseases.

Despite being well-suited for analysis using system dynamics models, there has been little application of system dynamics models to infectious disease problems within the field of environmental health. In the absence of a quantitative model, environmental health decision-makers must fall back on their mental model of disease transmission. While in some instances decisions based solely from a mental model may be sufficient, the complex non-linear dynamics of infection transmission means that in many cases this is likely to be insufficient. For example, previous experience and intuition might lead decision-makers to close a swimming pool that is suspected of being infected with *Cryptosporidium* to control an outbreak. While this initially appears logical, this action can have the unintended consequence of increasing the geographic spread of the outbreak. As their regular swimming facility is not available, swimmers from the infected pool may choose to

visit neighbouring pools, potentially infecting additional pools and exposing a larger segment of the population to the pathogen (McCann et al., 2014). The feedback mechanisms within socioenvironmental systems can cause them to behave in counterintuitive ways. System dynamics models provide a tool to help overcome our mental model's inability to simulate the complex dynamics that produce counterintuitive system behaviour.

#### 2.4. Summary

Environments play a key role in the health and wellbeing of populations around the world, with the health impact associated with modifiable environmental conditions representing a non-trivial portion of the global burden of disease. The field of environmental health involves the application of a diverse range of scientific and technical disciplines to understand, assess, and predict the relationships between environmental hazards and human health, and deliver effective environmental health services.

Despite remarkable strides being made in the field of environmental health, significant challenges remain in meeting its ultimate goals of reducing the environmental burden of disease and creating safe and healthy environments for all. It has been argued that many environmental and public health interventions fail to successfully meet those goals because they approach problems in isolation, rather than addressing them in a comprehensive manner that acknowledges that individual problematic events or environmental hazards rarely occur in isolation and are likely heavily influenced by behaviours within the wider system (Homer and Hirsch, 2006). Conventional analytic methods of investigating the causes and effects of environmental health interactions, and they often struggle to characterize the long delays between cause and effect that often accompany environmental health problems.

Quantitative models, and in particular system dynamics models, present a potentially valuable tool to help overcome many of these challenges posed by complex disease transmission problems. They provide an avenue to explore, anticipate and predict the feedback behaviour that makes up complex dynamic systems, even in situations where data is lacking or incomplete. Active participation in the system dynamics modelling process not only adds credibility and confidence in the model itself, but it also provides decision-makers with an opportunity to gain insight, generate testable hypotheses, predict the effect of interventions over short and long time periods and improve communication in their decision-making processes.

# Chapter 3. Case Study Background

The purpose of this chapter is to provide a general overview of information specifically-relevant to the case study of cryptosporidiosis in South East Queensland. The purpose of this chapter is not to provide an exhaustive review of the literature related to *Cryptosporidium*, but rather to serve as an evidence-based point of reference for factors that may contribute to the transmission of *Cryptosporidium* infections in the study area. It has been divided into two sections. The first provides a general overview of *Cryptosporidium* and cryptosporidiosis, including transmission, risk factors and the current literature on the management of cryptosporidiosis. The second provides background on the case study location, as well as an overview of the local context of known cryptosporidiosis risk factors.

## 3.1. Cryptosporidium spp. as infectious agents

*Cryptosporidium, a* protozoal parasite in the family Cryptosporidiae, was first identified in humans in 1976 (Meisel et al., 1976). *Cryptosporidium spp.* are now considered one of the most commonly identified intestinal parasites causing infection in humans throughout the world (Organization., 2002), with increasing incidence in developed countries (Snel et al., 2009). The prediction, management and prevention of cryptosporidiosis outbreaks is difficult and complex due to a number of factors including its multiple transmission pathways, its persistence in the environment, and its extended asymptomatic infectious period (Rossle and Latif, 2013).

#### 3.1.1. Taxonomy

Originally thought to be one species, there are now currently 19 known species of *Cryptosporidium* (Leitch and He, 2012), five of which (*C. hominis, C. parvum, C. meleagridis, C. felis, and C. canis*) are thought to be responsible for the majority of cases of cryptosporidiosis in humans (Xiao and Fayer, 2008). *Cryptosporidium hominis, C. parvum* are the two most frequently identified species in cryptosporidiosis cases in developed countries, representing over 90% of the cases (Bouzid et al., 2013). *Cryptosporidium hominis* is largely restricted to human hosts, and therefore has low zoonotic potential (Chalmers and Casemore, 2004). *Cryptosporidium parvum* has been detected in both animals (wild and livestock) and humans, and is a major pathogen in calves (Bouzid et al., 2013, Hashim et al., 2006). *Cryptosporidium felis*, and *C. canis* are most commonly associated with feline and canine hosts, respectively (Chalmers and Casemore, 2004). *Cryptosporidium meleagridis is associated with* avian hosts including turkeys, parrots and chickens but also has been associated with a small number (<1%) of human cases of cryptosporidiosis (Chappell et al., 2011).

The distribution of the various *Cryptosporidium spp.* in high-income countries varies considerably both geographically and seasonally. Generally speaking, urban areas experience a greater incidence of cryptosporidiosis caused by *C. hominis*, whereas rural areas often have greater incidence of *C. parvum* infections due to greater exposure to livestock (Leitch and He, 2012). Seasonal variations in the *Cryptosporidium spp.* responsible for outbreaks have been noted some countries. An example of this is New Zealand, which has seen an increase in zoonotic (C. *parvum*) cases in the spring and an increase in anthroponotic cases (C. *hominis*) in the autumn (Learmonth et al., 2004). The spring peak in incidence has been attributed to the spring lambing/calving season, and the autumn peak to increased use of aquatic facilities in the late summer.

## 3.1.2. Transmission

*Cryptosporidium* is transmitted through the faecal-oral route, and while it is primarily considered to be a waterborne pathogen, transmission can also occur through person-to-person contact, animal-to-person contact, and consumption of contaminated food (Figure 3.1). *Cryptosporidium* is highly infectious with a low median infective dose (35.7 oocytes using diarrheal illness as an indication of cryptosporidiosis) (Messner et al., 2001), making it easily transmittable. Following exposure to *Cryptosporidium* oocytes, the incubation time in humans is 1-12 days (mean 7.2 days) (DuPont et al., 1995).



Figure 3.1: Primary transmission pathways of Cryptosporidium

## 3.1.2.1. Waterborne Transmission – Drinking water

*Cryptosporidium* oocytes have been identified in most fresh water bodies, with higher concentrations found in water with heavy human use, nearby livestock and faecal pollution (DuPont et al., 1995). *Cryptosporidium* oocytes have also been identified in sewage-contaminated marine waters, demonstrating the ability to survive up to four days in saline conditions (Johnson et al., 1997). In general, *Cryptosporidium* oocytes are considered highly-persistent in the environment. Oocytes are known to survive for months in fresh surface water (Alum et al., 2014), though are viable for less than two hours on dry surfaces at room temperature (Robertson et al., 1992).

Contaminated drinking water sources have been associated with numerous cryptosporidiosis outbreak. A major outbreak in Milwaukee, USA in 1993, thought to be caused by sewage released from a sewage treatment plant upstream of a water treatment plant, resulted in over 400,000 individuals being infected over a 2 week period (Eisenberg et al., 2005). Outbreaks attributed to drinking water have been identified in a number of European countries, though the frequency varies considerably based on the quality of the public water supply and sewage treatment systems (Semenza and Nichols, 2007).

A study of waterborne outbreaks of gastrointestinal disease in Australia found that less than 20% of outbreaks were attributed to drinking water, and only 1 positively linked to *Cryptosporidium*. This single outbreak occurred in South Australia and was caused by the contamination of a private water supply (Dale et al., 2010) and not community supplied water.

## 3.1.2.2. Waterborne Transmission - Swimming pools and recreational waters

Public aquatic facilities are a frequently identified source of outbreaks, for several reasons. *Cryptosporidium* oocytes are both small enough to bypass many swimming pool filtration systems (Centers for Disease Control and Prevention, 2001) and highly resistant to chlorine disinfection (Suppes et al., 2016). This can result in their persistence in even well managed swimming pools. It is estimated that oocytes can survive 3.5-10 days in a swimming pool where free chlorine is maintained at the recommended level of 1–3 mg/L (Shields et al., 2008b).

A single accidental faecal release (AFR) by an infected swimmer can release 10<sup>9</sup> *Cryptosporidium* oocytes, which, coupled with an extremely low infectious dose of as little as 10 oocytes, can result in significant number of exposed swimmers becoming ill (Suppes et al., 2016). It should be noted that the spread of *Cryptosporidium* through an AFR is primarily limited to releases of loose stool, as studies have identified low prevalence of oocytes in formed stool (2001).

Additionally, public aquatic facilities are venues where a heterogeneous mixture of people not only bathe, but also inadvertently swallow water (Schoefer et al., 2008). A study by Dufour et al. (2006) determined that on average children and adults unintentionally swallow between 0 to 154 mL (mean 37 mL) and 0 to 53 mL (mean 16 mL) of pool water per swim, respectively.

While significant advancement has been made in the development of nappies which are capable of retaining solid stool during water-based activities, a study by Amburgey and Anderson (2011) determined that currently available "swim nappies" are not able to contain *Cryptosporidium*-sized particles of faecal-matter. Their study found that after 10 minutes of water-based play, 77-100% of oocytes-sized test particles deposited in the swim nappies had been released from the nappy into the water. This means that infected children who defecate in swim nappies while swimming are easily able to contaminate recreational water.

The 'shedding' of *Cryptosporidium* oocytes by bathers can also be a major source of contamination in recreational water (Ashbolt et al., 2010). Gerba (2000) determined that bathers, on average, shed 0.14 grams of faeces per swim. As infectious stools can have a concentration of  $10^9$  oocytes of *Cryptosporidium* per gram of stool (Rose et al., 2002), a single infected swimmer has the potential to shed 14,000,000 oocytes into a swimming pool without knowing it.

While *Cryptosporidium* have been found in swimming pools during outbreaks, a variety of studies have also sought to determine the presence of oocytes in the water of public aquatic facilities in the absence of reported disease. A study of 5 public pools in the Netherlands identified *Cryptosporidium* oocytes in 4.6% of filter backwash samples over the period of one year (Schets et al., 2004). A study of 160 public pools in Atlanta, Georgia found the water in 3 pools was positive for *Cryptosporidium* spp. (Shields et al., 2008a). It is possible that this contamination is a result of undetected accidental faecal releases or to shedding by bathers.

Swimming pool operators and staff have themselves also been implicated in cryptosporidiosis outbreaks (Wheeler et al., 2007, Louie et al., 2004, Sorvillo et al., 1992). In all these cases at least one lifeguard swam while knowingly ill. An investigation of a large outbreak at a water park in California found that, while waterpark management was aware of illness among staff, ill staff were only excluded from the pool when too ill to work (Wheeler et al., 2007). It is likely that the potential employment and financial repercussions of excluding themselves from the swimming pool for the duration of the infectious period, coupled with a lack of knowledge of transmission dynamics, contributed to the lifeguards continuing to swim while ill. These outbreaks demonstrate that pool patrons are not the only contributors to outbreaks.

Only about 10% of recreational-water-related cases of cryptosporidiosis are associated with swimming in fresh water (Roy et al., 2004). This is likely because fresh water bathing areas tend to have a higher water volume and a lower bather density than swimming pools, especially in relation to incontinent bathers. It is possible that fresh water venues contribute more to sporadic cases of cryptosporidiosis than outbreaks.

#### 3.1.2.3. Person-to-person Transmission

Person-to-person transmission of *Cryptosporidium* is also thought to be a major transmission pathway associated with outbreaks. In particular, childcare centres have been implicated in multiple outbreaks of cryptosporidiosis (Cordell and Addiss, 1994). Much like swimming pools, childcare centres are venues where children without full bowel control are present in large numbers. It has been estimated that 50% of cases of infectious diarrhoea in children under 3 years old who attend day-care are acquired at the day-care facility (Morrow et al., 1991). It is also thought that most outbreaks of cryptosporidiosis in childcare settings occur because an infected child is brought into the childcare environment, and subsequently infects other children or staff members through direct contact as well as depositing *Cryptosporidium* oocytes on various surfaces in the environment (Cordell and Addiss, 1994). Staff members are less commonly implicated as the source of infection

Evidence of person-to-person transmission of Cryptosporidium has been documented in several outbreak investigations. An investigation of a 1993 outbreak in the United States related to the consumption of contaminated apple cider found that 33% of households with one primary case experienced one or more secondary cases of cryptosporidiosis (Millard et al., 1994). In this outbreak, 37% of primary cases aged 5 to 9 transmitted their infection to another member of their household, compared to 28% of primary cases aged 10 to 19 and 25% of primary cases over 20 years old. An investigation of a 1984 childcare centre outbreak in the United States has similar results with 38% of symptomatic children and 9% of asymptomatic children passing their infection to another member of their household (Heijbel et al., 1987). A somewhat lower transmission rate was observed in a Norwegian outbreak among schoolchildren (12-13 years old) visiting a holiday farm, where household person-to-person transmission accounted for 17% of cases (Johansen et al., 2015). In 75% of these cases, Cryptosporidium was transmitted from a child to a caregiver. A lower person-to-person transmission rate was reported in the large 1993 waterborne outbreak in Milwaukee, where person-to-person transmission rates involving a primary adult case were only 5% (MacKenzie et al., 1995b). A case-control study of sporadic cases of cryptosporidiosis in the United States also found that contact with a child 2 to 11 years old posed a significant risk, whereas no significant risk was associated with contact with an infected older child or adult (Roy et al., 2004).

This indicates that the age of the primary case likely has a considerable impact on the risk of secondary transmission.

## 3.1.2.4. Foodborne Transmission

While it is thought that cases of cryptosporidiosis related to contaminated food are less common than those related to water (Karanis et al., 2007), foodstuffs still remain a possible source of *Cryptosporidium* exposure. Foodborne-transmission of *Cryptosporidium* oocytes is associated with a variety of sources including food contaminated by surface water run-off, irrigation water and/or wash water (Chaidez et al., 2005, McKerr et al., 2015, Dixon et al., 2013, Amorós et al., 2010), flies (Graczyk et al., 2003), and through infected food handlers (Centers for Disease Control and Prevention, 1998, Quiroz et al., 2000). Foodborne outbreaks of cryptosporidiosis have also been attributed to the consumption of unpasteurized milk (poor udder hygiene) (Harper et al., 2002) and unpasteurized fresh-pressed apple cider (fallen apples) (Blackburn et al., 2006, Millard et al., 1994).

A review by Robertson and Chalmers (2013) identified 18 reported outbreaks of cryptosporidiosis globally that were attributed to foodborne transmission. Of these, 6 were attributed to uncooked vegetables/leafy greens, 6 to unpasteurized milk and milk products, 3 to unpasteurized apple cider, 1 to a meat product that was consumed raw, and 2 to infected food handlers. As with many other foodborne parasitic infections, identifying and investigating food-related outbreaks of cryptosporidiosis is extremely challenging as the long incubation time between infection and symptoms makes it difficult to identify the vehicle of infection (Robertson and Chalmers, 2013). This likely results in an under-reporting of foodborne cases of cryptosporidiosis.

## 3.1.2.5. Zoonotic Transmission

*Cryptosporidium* is highly prevalent in ruminants, such as cattle, and is excreted in large numbers in their faeces (Coffey et al., 2007). It has been estimated that 15 to 24% of dairy calves shed *Cryptosporidium* oocytes, and an estimated 67 – 96% of dairy herds have at least one shedding animal (Garro et al., 2016). A study conducted in the United Kingdom of farms linked to outbreaks of cryptosporidiosis found that farms with cattle, sheep and pigs had the highest prevalence of *Cryptosporidium* infection. Furthermore, faecal samples from young (pre-weaned) cattle were 11 times more likely to be positive for *Cryptosporidium* than samples from adult cows (Smith et al., 2010).

There have been numerous reported outbreaks of cryptosporidiosis related to livestock including in veterinary students working with young calves and lambs, and children visiting educational farms and petting zoos (Utsi et al., 2016, Sayers et al., 1996, Stefanogiannis et al., 2001, Preiser et al.,

2003, Ashbolt et al., 2003). Studies of people's hygiene-related behaviour in educational farms and petting zoos has found several common behaviours that would support the transmission of zoonotic pathogens such as *Cryptosporidium*. These behaviours include bringing items that come into contact with children's mouths (e.g. pacifiers and baby bottles) into the farm area, low hand-hygiene compliance, allowing animals to lick children's hands or face, and children and adults eating and/or drinking within animal contact areas (Erdozain et al., 2013, McMillian et al., 2007, Conrad et al., 2016, Weese et al., 2007).

The role that companion animals play in the transmission of cryptosporidiosis in high income countries is thought to be minimal. Studies investigating the prevalence of *Cryptosporidium* in domestic animals showed that the prevalence of infection ranged from 0.5% to 44.1% in dogs and 0% to 29.4% in cats (Lucio-Forster et al., 2010). Despite this relatively high prevalence, epidemiological studies have found that contact with cats and dogs has minimal (source) or no association with human cryptosporidiosis (Goh et al., 2004), and may even be protective (Robinson and Pugh, 2002).

## 3.2. Symptoms and management of cryptosporidiosis

Cryptosporidiosis is characterized by prolonged watery diarrhoea, abdominal cramping, and flu-like symptoms. Symptoms are normally self-limiting in immunocompetent people, but can cause life-threatening diarrhoea in immunocompromised people, particularly those with Acquired Immune Deficiency Syndrome (AIDS) (Cruickshank et al., 1988). There is currently no approved treatment for cryptosporidiosis in Australia. Supportive management and oral fluid replacement is the primary treatment approach.

Asymptomatic infection with oocyte shedding is possible, though little is known about the rate by which asymptomatic carriage occurs. Reported rates of asymptomatic cryptosporidiosis vary greatly, with a study of US children reporting rates of 6.4% in immunocompetent children and 22 % in immunocompromised children (Pettoello-Mantovani et al., 1995), to a study in Melbourne which reported an asymptotic carriage rate in the adult general public of 0.4% (Hellard et al., 2000b). A detailed investigation in an outbreak related to a childcare centre found that 24% of infected children were asymptomatic (Vandenberg et al., 2012). Infected individuals also continue to shed oocytes in their stool after symptoms have ceased. A study by Xiao et al. (2001) found that individuals infected with C. *hominis* have a longer shedding period than individuals have also been found to excrete larger quantities of oocytes in their stool than individuals infected with C. *parvum* 

(McLauchlin et al., 1999). Asymptomatic shedding of crypto oocysts is of great public health significance as individuals are unlikely to follow common advice such as not swimming and avoiding childcare for 2 weeks. The standard advice of avoiding swimming while you have symptoms such as diarrhoea is insufficient to stop cryptosporidiosis transmission.

Gastrointestinal symptoms associated with cryptosporidiosis frequently re-occur following recovery from the acute stage of cryptosporidiosis. A study by Hunter et al. (2004b) found that 40.9% of symptomatic patients had a relapse of gastrointestinal symptoms following their initial recovery. This is consistent with observations from a large cryptosporidiosis outbreak in Milwaukee in 1993 where 39% of patients experienced a reoccurrence of diarrhoea after 2 or more days of normal stool (median 3 days, range 2-10) (MacKenzie et al., 1995b). Of these individuals, only 6% had a reoccurrence after 45 or more days of normal stool (Osewe et al., 1996).

There is no conclusive evidence that infection with *Cryptosporidium* confers immunity to reinfection in humans. A study by Chappell et al. (1999), that involved infection of a small number of both uninfected and previously infected volunteers, showed that prior infection prevented reinfection at low doses (500 oocytes), but not at higher doses (10,000 or more oocysts). However, Okhuysen et al. (1998) found previous *Cryptosporidium* infections provided no significant protection one year following initial exposure, though they did find decreased severity (number of unformed stools) and intensity (likelihood of detecting oocytes in stool) of the disease in the previously exposed population. Interestingly, an analysis of data from a water-associated outbreak in Oregon found that long-term residents of the town were less likely to show symptoms of illness compared to short-term, or out-of-town visitors (Frost et al., 1998). This was interpreted as evidence of the protective effect of multiple exposures with regards a reduction in symptomatic illness (but not necessarily infection) in individuals.

#### 3.2.1. Healthcare-related behaviours

One of the factors that complicates the management of outbreaks of cryptosporidiosis is the relatively low rate of presentation for healthcare in patients with symptoms of cryptosporidiosis, and diarrhoea in general. Perz et al. (1998) estimated that only 5% of adults and 10% children with symptoms of diarrhoeal illness will seek medical care (increasing to 33% and 50% respectively when symptoms are moderate-severe). They further estimated that of the adults and children who present to a doctor with symptoms, only 25% of adults, and 50% of children will have their stool tested. Other studies have found that between 18%-23.5% (Scallan et al., 2011, Tam et al., 2012, Majowicz et al., 2005, Scallan et al., 2005) of individuals with diarrhoeal illness sought medical

attention, and of those only 14% - 26% (Scallan et al., 2005, Majowicz et al., 2005, Scallan et al., 2011) had their stool tested. The low stool testing rate may be due to a variety of factors such as physicians initially adopting a supportive approach, a lack of noteworthy circumstances to raise physician's suspicions, or concerns about the cost of testing. Figure 3.2 illustrates several additional factors at each stage of the surveillance pyramid that likely contribute to the under-reporting of *Cryptosporidium* infections in disease surveillance systems.



**Figure 3.2:** Surveillance pyramid for cryptosporidiosis, included factors influencing ascertainment. Physicians' knowledge of cryptosporidiosis also plays an important role in the management of both sporadic- and outbreak-associated cases of cryptosporidiosis. Studies of American physicians have found that more than 75% of gastroenterologists, general/family practitioners, and paediatricians rarely or never ordered diagnostic tests for patients with symptoms consistent with cryptosporidiosis (Morin et al., 1997). In addition, only 44.4% of obstetrician-gynaecologists could correctly identify that prolonged, intermittent diarrhoea could lead to a differential diagnosis of cryptosporidiosis (Domjahn et al., 2014). A similar study also found that 40% of physicians did not know the most appropriate tests for parasitic diseases such as cryptosporidiosis (Hennessy et al., 2004), which may result in requests for inappropriate diagnostic tests (Polage et al., 2011). Therefore, existing surveillance significantly underestimates the incidence of cryptosporidiosis because of the small proportion of symptomatic infectious cases that receive a clinical diagnosis and are subsequently reported to public health agencies.

## 3.2.2. Diagnostic Methods

Confirmation of *Cryptosporidium* infection is achieved by detection of oocysts or *Cryptosporidium* antigens or nucleic acid in faecal specimens from infected individuals.

Direct microscopy to visually detect oocysts involves the examination of infected stool under a microscope, typically using modified Kinyoun Acid-Fast stain or fluorescent monoclonal antibody (FA) staining reagents (Public Health Laboratory Network, 2017). Due to the difficulty visualizing *Cryptosporidium* oocytes, the examination of multiple stool specimens (up to 3) may be necessary to make a diagnosis. The sensitivity of direct microscopy varies considerably depending on the technician's experience and the staining technique used, but is thought to vary between 33% and 100% (mean 70.85%) (ten Hove et al., 2009, Van den Bossche et al., 2015).

Antigen detection involves the use of commercially available testing kits, which use either Enzyme immunoassays using microtitre plates or immunochromatographic immunoassays to detect *Cryptosporidium* oocytes in faeces (Public Health Laboratory Network, 2017). Antigen detection has often been the laboratories' preferred diagnostic method as detection can occur in a single step. The sensitivity of antigen detection varies considerably based on the kit used and the species of *Cryptosporidium*, but ranges from <35% to 100% %) (ten Hove et al., 2009, Van den Bossche et al., 2015). Unlike the other two methods, antigen detection can suffer from poor specificity if the stool sample contains blood.

Nucleic acid detection is the newest diagnostic method available for the detection of *Cryptosporidium* in stool. Real-time polymerase chain reaction (PCR) is a molecular diagnostic technique that targets specific genes within the *Cryptosporidium* oocyte. This technique has a high sensitivity, ranging from 80% to 100% (ten Hove et al., 2009, Stark et al., 2014, Liu et al., 2013), though this is often limited to the detection of *C. parvum* and *C. hominis*. Real-time PCR has largely replaced microscopy and antigen detection as the routine method used for diagnosis due to its speed and high sensitivity.

## 3.2.3. Cryptosporidiosis and international travel

Recent international travel to developing countries is considered a major risk factor for acquiring *Cryptosporidium* infections, particularly when the destination of travel is a low- or middle-income country (Roy et al., 2004). This is likely due to international traveller accidentally consuming food or beverages that contain inadequately treated water, as well as travellers visiting areas without improved sanitation and hygiene practices. However, *Cryptosporidium* is not considered one of the

primary pathogens responsible for traveller's diarrhoea. Jelinek et al. (1997) found that only 13 (2.8% of 469 German travellers with diarrhoea were infected with *Cryptosporidium*. This is similar to the results of recent studies that showed that 3% of US travellers to Guadalajara, Mexico (Bouckenooghe et al. (2002) and 1% of long-term Dutch travellers to the (sub)tropics (Soonawala et al. (2014)) were infected with *Cryptosporidium*.

A 5-year surveillance study (2004-2009) in the United States of travellers to low income countries found that travellers to Africa and Central America have higher than average risk of *Cryptosporidium* infections (9.6 cases per 100 000 travellers and 2.8 cases per 100 000 travellers respectively) (Kendall et al., 2012). It also found that overall, *Cryptosporidium* infections represent 3.8% of travel-associated enteric infections and 5.9% of non-travel-associated enteric infections in the American general population and estimated that travel-associated cases represent 8.9% of cryptosporidiosis in the United States.

Whilst the individual risk of infection with *Cryptosporidium* in travellers is low, the number of people travelling is high, which represents a significant potential source of infection for Australian communities. Indeed, Australia recorded 15 million international arrivals in 2012, 8.1 million of which were Australian residents returning from a short trip overseas (Australian Bureau of Statistics, 2012). While only a portion of these arrivals are likely from developing countries.

While not specific to cryptosporidiosis, existing research in diarrhoea in travellers may also provide some insight on cryptosporidiosis dynamics related to international travel. A review conducted by Diemert (2006) found that age plays a significant role in infectious diarrhoea in travellers, with young children and adults aged 21-29 years having the highest incidences of diarrhoea related to foreign travel. This study attributed the high rates of diarrhoea in young adults to a lack of vigilance in avoiding contaminated food and beverages and a more adventurous lifestyle that would bring them into contact with contaminated environments. It is possible that this contributes, at least to some degree, to the higher rates of cryptosporidiosis that have been identified in young children compared to other age groups.

## 3.2.4. Cryptosporidiosis and weather, climate, and seasonality

There has been limited study of the effect that weather and climate have on the transmission of cryptosporidiosis in high income countries. A study of weekly weather variability and cases of cryptosporidiosis in Brisbane, Australia identified a possible link to maximum temperature and relative humidity, with cryptosporidiosis cases increasing when temperature is above 31°C and relative humidity is below 63% (Hu et al., 2010). A similar study in Victoria, Australia showed that

a 1°C increase in monthly average minimum temperature was associated with a 22% increase in cryptosporidiosis notifications (Kent et al., 2015). Neither study identified any association between rainfall and cryptosporidiosis notifications. Furthermore, a study conducted in the United States found that increases in temperature, changes in river flow, and increased water pollution associated with climate change could increase the incidence of cryptosporidiosis. However, the authors argued that climate change-related factors are poor predictors of the risk of cryptosporidiosis in high-income countries as the overall impacts of public health investment and water treatment infrastructure overwhelm the effects of changes in climate (Casman et al., 2001).

The distribution and incidence of cryptosporidiosis have been found to differ according to seasons. In Canada and the United States, *Cryptosporidium* infections were found to peak in the late summer (Laupland and Church, 2005, Dietz and Roberts, 2000) whereas in New Zealand (Learmonth et al., 2001) and the United Kingdom (Naumova et al., 2005) observe peaks of incidence peaks in both the spring and autumn. It is thought that the spring peak coincides with the spring calving season. The United Kingdom also experiences a spring and autumn peak. In Australia, reported cases have been found to peak towards the end of the summer, with Queensland, Victoria and New South Wales also experiencing a smaller peak in the spring (Figure 3.3) (Lal et al., 2015).



**Figure 3.3:** Total weekly number of cryptosporidiosis notifications in Australia by state or territory, 2001-2012 (reprinted from Lal et al. (2015))

While many studies attribute seasonality to weather and climate conditions (Naumova et al., 2005, Naumova et al., 2007, Jagai et al., 2009), it is likely that routine seasonal occurrences such calving

and lambing seasons, and increased contact with recreational water in the summer, play a significant role in the seasonality of cryptosporidiosis. Evidence in some countries of increased C. *parvum* cases in the spring likely due to lambing and calving season, and spring runoff, and increased C. *hominis* in the late summer, likely due to increased recreational water activities and international travel, supports this hypothesis (Goh et al., 2004, Hunter et al., 2004a).

## 3.2.5. Cryptosporidiosis management

The primary methods currently used to prevent and manage the spread of cryptosporidiosis fit broadly into 3 categories: design and construction of facilities, operation and management, and public behaviour modification. This section will examine the existing literature on currently adopted cryptosporidiosis management approaches, with a focus on their impact on public aquatic facility. As the focus of this research project is not the evaluation of specific engineering elements of aquatic facility design and construction, that section will only provide a brief overview of cryptosporidiosis-related studies and interventions.

### Design and construction of facilities

As *Cryptosporidium* oocytes are not quickly inactivated at normal free chlorine levels, pool operators often rely on external filtration or treatment systems such as UV irradiation and ozone treatment. Because these treatments occur outside the pool, it is important that pool systems are designed with sufficiently regular turnover to ensure all water regularly passes through the treatment system, while also allowing for sufficient mixing of pool water to allow in-situ treatments to be effectively dispersed throughout the pool. A study conducted by Lewis et al. (2015) of computational fluid dynamics assessed the effects of operational practices on the risk to public health within large indoor swimming pools. They found that current pool design approaches sufficiently disperse disinfectants within the pool within a desired timeframe, but do not exchange water sufficiently, creating 'dead-zones' at either end of the pool which can harbour pathogens such as *Cryptosporidium*. This is of particular concern as the ends of the pool are the areas where people, particularly children, are likely to congregate.

## 3.2.5.1. Operation and management

One of the key methods used to control *Cryptosporidium* transmission in aquatic facilities is by establishing accidental faecal release (AFR) procedures. These procedures are used when a pool patron has a faecal accident in or around the pool. They generally consist of closing the aquatic venue, removing as much of the faecal matter as possible, disinfecting the pool water or draining

(typically through hyper-chlorination), and cleaning the venue. The water is then replaced, returning the chemical parameters in the water to a safe level, and then allowing patrols to return to the venue. The Centers for Disease Control and Prevention's *Model Aquatic Health Code* (Centers for Disease Control and Prevention, 2016a) recommends that following the detection of an diarrhoeal AFR, that free chlorine residual is raised to 20mg/mL and maintained for at least 12.75 hours or to circulate the pool water through a secondary disinfection system to theoretically reduce the concentration of *Cryptosporidium* oocytes to less than 1 oocyte/100 ml.

Unfortunately, cryptosporidiosis can be transmitted through faecal accidents that are undetected, or when contamination occurs though shedding. In these cases, pool operators may be unaware of the contamination and therefore do not implement the decontamination procedures until notified by a public health authority. This delay can result in a significant number of additional pool users being infected. In an attempt to overcome this delay, a county in Ohio tested a proactive approach where a pool, as well as two neighbouring pools, were ordered to hyper-chlorinate based on anecdotal evidence or pool use by infected individuals, and before the source of the outbreak was epidemiologically ascertained (Cope et al., 2015). Further investigations did find the pool significantly associated with the outbreak and that a community-wide outbreak had likely been avoided. While it is impossible to ascertain whether the proactive approach prevented a community-wide outbreak, the evidence does support this hypothesis.

Pool operators, who are responsible for the correct maintenance and operation of public aquatic facilities, play an integral role in the prevention and management of cryptosporidiosis outbreaks. To ensure that aquatic facility mismanagement isn't the source of disease outbreaks, there have been calls for mandatory pool operator training. While there have been no studies on the impact of pool operator training on the incidences of cryptosporidiosis in pool patrons, a study investigating the effectiveness of mandatory pool operator training on overall pool water quality was conducted in the United States. Buss et al. (2009) compared pool water quality violations in Nebraska counties with and without mandatory pool operator training, finding that violations were twice as likely in counties without mandatory training than those with mandatory training. While *Cryptosporidium* can be present even in well-maintained pools, their findings do support the idea that mandatory operator training and certification can reduce the health risk posed by public aquatic facilities.

## 3.2.5.2. Public behaviour modification through education and information

As current technical interventions cannot alone prevent the spread of cryptosporidiosis, interventions that aim to change the behaviour of people who can potentially catch and spread cryptosporidiosis are important.

There has been limited study of the behaviour of aquatic facility users in relation to the overall hygiene environment of public aquatic facilities. Of the studies conducted, most have identified a low level of compliance with basic hygiene-related behaviours such as pre-swim showering and not swimming while ill (Bonini et al., 2011, Wiant, 2011, Nett et al., 2010). As well, these studies have identified a poor understanding of the health-related rationale driving the need for compliance with these behaviours, and, with the exception of wart and mycosis, as a poor understanding of the existence and mechanisms of infectious diseases transmitted in aquatic facilities (Liguori et al., 2007, Galle et al., 2016, Amodio et al., 2014, McClain et al., 2005, Nett et al., 2010). Interestingly, a hygiene survey in the United States conducted by the Water Quality and Health Council found that despite admitting to non-compliance with hygiene-related behaviours themselves, the majority of Americans believe that their fellow swimmers are engaging in more unhygienic behaviours in the pool than they are (Wiant, 2011).

McClain et al. (2005) conducted a study of parents' perception of their children's risk for recreational water illnesses (RWI). Their results indicate that for parents with poor awareness of RWIs, that their perception of their child's vulnerability to RWIs and the perceived severity of RWIs had the biggest influence on their overall perception of risk. Once they acknowledge the potential risk associated with RWIs, the parent's perception of their ability implement/enforce behaviour modifications in their children (self-efficiency) had the bigger influence on their overall perception of risk.

One of the primary methods currently used to prevent bather contamination of public aquatic facilities is displaying pool rules and regulations. The WHO *Guidelines For Safe Recreational Water Environments* (World Health Organization, 2006) recommend that signage is used to convey personal hygiene measures such as pre-swim showers. In addition, the Center for Disease Control and Prevention's Model Aquatic Health Code requires conspicuous signage prohibiting polluting the water, swimming while ill with diarrhoea, swallowing pool water and requiring patrons to shower before entering the water (Centers for Disease Control and Prevention, 2016a). These recommendations are echoed within Australia including in the Government of Victoria's *Pool* 

*Operators Handbook* (Victorian Government, 2008), the Queensland Health *Swimming and Spa Pool Water Quality and Operational Guidelines* (Queensland Health, 2004) and the Government of New South Wales *Public swimming pool and spa pool advisory document* (Health Protection NSW, 2013). To date, there have been no published studies regarding the effectiveness of signage in relation to reducing or managing cryptosporidiosis, but a series of studies conducted in Italy of general health-related behaviours in swimming pool users found no association between knowledge of hygiene-related pool rules and regulations through viewed posted signage, and compliance with those rules (Galle et al., 2016). Similar results were found in a study of splash-park visitors in the United States, which found that educational signage did not appear to influence visitor's compliance with hygiene behaviours or attitudes (Nett et al., 2010). This indicates that knowledge of pool rules is insufficient on its own to ensure pool-users adopt the hygiene-related behaviours necessary to prevent pool-related cryptosporidiosis transmission.

Another method currently used by public health agencies that is aimed at reducing the spread of cryptosporidiosis by users of public aquatic facilities is through educational media campaigns. There has been little evaluation of cryptosporidiosis-specific education campaigns, with the exception of a 2008 campaign in Utah following a massive cryptosporidiosis outbreak the previous year (Centers for Disease Control and Prevention, 2012). This large campaign used online, television, radio and print media to convey messages such a "A Swimming Pool is like a community bathtub", as well as sent targeted messaging to high risk user groups such as water sports teams and childcare centres. Following the media campaign, the study found Utah residents had greater healthy swimming knowledge than residents of other states, particularly residents who had seen the television advertisements. While knowledge does not necessarily correspond with behaviour change, the authors did note that there had been no subsequent pool-related outbreaks in Utah following the media campaign.

## 3.3. Case Study – Cryptosporidiosis in South East Queensland

## 3.3.1. Location of Study

South East Queensland <sup>3</sup>(SEQ) is a geographical and administrative region of Queensland, located on the eastern coast of Australia, stretching from the New South Wales border in the south to the

<sup>&</sup>lt;sup>3</sup> The study region sits within a region commonly referred to as 'South East Queensland' (written as a proper noun), which specifically refers to region surrounding the metropolitan areas of Brisbane and the Gold Coast and does not encompass the entire southeast (geographical) portion of the state of Queensland.

city of Noosa in the north and the city of Toowoomba in the west. The two major urban areas with SEQ are the city of Brisbane and the city of Gold Coast. A variety of geographical boundaries for

South East Queensland exist, the boundary for the study area was defined using the Queensland Ministry of Health's boundaries for the Metro North, Metro South, and Gold Coast Health and Hospital Health Services (Figure 3.4).

## 3.3.2. Population

The population of the study area is estimated to be approximately 2.5 million persons, representing 54.9% of the total Queensland population (Queensland Government Statistician's Office,



**Figure 3.4** Study Area as defined by the Queensland Ministry of Health's Hospital and Health Services boundaries

2015). Population density in SEQ varies tremendously with areas of central Brisbane and Gold Coast having a population density exceeding 3500 persons/km<sup>2</sup>, to outer areas of the region having a population density below 500 persons/km<sup>2</sup> (Australian Bureau of Statistics, 2015c).



Figure 3.5: Estimated Resident Population by Age, South East Queensland, 2010 and 2015 (Australian Bureau of Statistics, 2015b)

Within the study area, the 2014 National Health survey determined that 55.9% of households had no children 0-14 years old, 14.6% had one child, 19.21% had 2 children, and 10.29% had 3 or more children (Australian Bureau of Statistics, 2015a). A total of 63.57% of adults in the study area have some form of post-secondary qualification, which is similar to the national average of 62.38% of adults, but somewhat higher than the Queensland average of 59.82% of adults (Australian Bureau of Statistics, 2015a).

#### 3.3.3. Cryptosporidiosis in South East Queensland

During the study period, cryptosporidiosis notifications showed an apparent cyclic trend, with increased periods of increased notifications being observed in 2009, 2012, 2015, 2016 and 2017 (Figure 3.6). All three regions of the study area follow a similar cyclic trend, except for the Gold Coast, which was less affected by the 2012 and 2015 peaks.



Figure 3.6: Weekly cryptosporidiosis notifications in the three PHU Regions of the study area (2007-2017)

Mean monthly cryptosporidiosis notifications are at their highest in the late summer / early fall across all three are groups and regions (Figure 3.7). Few cases occur from July-October in all three regions. That trend continues until December in the Gold Coast, whereas Metro North and Metro South Brisbane experience a notable increase as of November. Interestingly, the average monthly cases in individuals 5+ years old peaks one month before (February) those individuals 0-4 years old (March) in Metro South and Gold Coast.



Figure 3.7: Mean monthly cryptosporidiosis notifications in South East Queensland by age group and region (2007-2017)

## 3.3.3.1. Healthcare seeking behaviour

While there has been no study that specifically looked at healthcare seeking behaviour in individuals with cryptosporidiosis in SEQ, the Australian National Gastroenteritis Survey II conducted in 2010 (Kirk et al., 2014) found that 28% of survey respondents who reported gastroenteritis sought some kind of healthcare advice (just over half of which was advice from a pharmacist), with 15.5% seeking care from a doctor or health clinic, and 3.5% seeking care from an emergency/casualty department. Individuals with symptoms lasting two or more days were more likely to see a doctor. Vomiting was found to be significant predictors of going to see a doctor, whereas individuals with stomach cramps where less likely to seek medical attention. Of the cases who saw a doctor, approximately 24% submitted a stool sample for testing. A similar study in 2005 (Scallan et al., 2005) found that 19.5% of Australian respondents who had diarrhoea reported visiting a doctor, and of those who did visit a doctor, 18.4% were asked to submit a stool sample.

#### 3.3.4. Risk Factors

#### 3.3.4.1. Physical Activity and Swimming

The residents of the Metro North and Gold Coast areas of the study area are more active than the state and national average, whereas the activity level of residents of Metro South are less active and more closely match the state average (Australian Bureau of Statistics, 2015c). Overall, the residents of the study area have higher self-assessed health than the national or state average, with 58.4% of residents reporting they are in excellent or good heath, compared with 55.57% in Queensland, and 56.25% in Australia as a whole (Australian Bureau of Statistics, 2015c).

The proportion of individuals in each region who regularly swim, and the frequency of swimming, varies between the regions. Residents of the Gold Coast are more likely to regularly swim, though this is likely due to their close proximately to a number of public beaches.

All three regions have similar densities of public council swimming pools, with 8-12 aquatic centres per region (Brisbane City Council, n.d., City of Gold Coast, n.d.). These facilities generally contain a combination of different swimming pools and aquatic features, including shallow learn-to-swim pools, splash parks, indoor and outdoor pools, lap pools, diving pools and hydrotherapy pools. These pools vary in size ranging from small learn-to-swim pools (70,000 to 200,000 litres) to Olympic-sized lap pools (2,500,000 litres). Except for the Gold Coast Aquatics Centre, council-owned pools are operated and maintained under long-term contract by private leisure management companies. Approximately 1/3 of the pools are closed to public from late fall to mid-spring.

**Table 3.1:** Adults (18 years+) who reported swimming in the last week, 2012 (source: (Australian Bureau of Statistics, 2012a))

	Brisbane South	Brisbane North	Gold Coast	Total
Adults who reported swimming in the last week	49,300	31,500	33,900	116,000
Total adult population (18 years+)	816,100	640,800	454,800	1,904,100
Percentage of adults who reported swimming in the last week	6.0%	4.9%	7.5%	6.1%

The region is also home to numerous privately-owned aquatic facilities including pools in elementary and secondary school. As there is no central registry of publicly-accessible aquatic facilities in Queensland, the study will be limited to council-owned aquatic facilities.

There is currently no legislation in Queensland that legislate the design and operation of swimming pools in relation to water quality, except for the *Public Act, 2005*, which states that a swimming pool must not pose a public health risk. In 2004, the Queensland Department of Health released the *Swimming and Spa Pool Water Quality and Operational Guidelines*. These voluntary guidelines provide swimming pool operators with guidance and advice on how to maintain the quality of aquatic facilities they operate.

There are also currently no guidelines in Australia that outline or recommend an acceptable (tolerable) risk of infection related to contact with swimming pool water. Therefore, swimming pool water can only be compared to the threshold for tolerable risk for either natural water bodies or drinking water. The state of Queensland has adopted the federal *Guidelines for Managing Risks in* 

*Recreational Water* produced by Australia's National Health of Medical Research Council (NHMRC), though these guidelines only apply to natural waterbodies. The NHMRC guidelines rank water quality on a scale of A (very good) to D (poor) with estimated risks of gastrointestinal illness from enterococci per swimming event of <1%, 1-5%, 5-10% and >10% for categories A, B, C and D respectively (NHMRC, 2008). While the risk posed to humans from enterococci differs somewhat from that posed by *Cryptosporidium*, enterococci are a commonly used indicator of microbial water quality in recreational water bodies. Additionally, while these categories are not direct indications of acceptable risk thresholds, bodies of water within category A, which have very low or low susceptibility of contamination (as would be the case with swimming pools) likely meets that threshold (NHMRC, 2008). Bodies of water in category B are also considered to be 'very good'. Alternatively, a much more stringent threshold of 1 infection per 10,000 people per year is widely used as a reference point for tolerable risk related to drinking water (Hunter and Fewtrell, 2001).

## 3.3.4.2. Childcare

In terms of adults providing care for children, approximately 23% of adults in South East Queensland report caring for either their own, or someone else's child at least once in the last 2 weeks (Table 3.2). While only a portion of these people likely cared for a young child, it does represent a rough estimate of the proportion of the adult population within the study site that has regular contact with a known high-risk group for cryptosporidiosis (children).

	Number of adults (15 years +)	Percent of adult (15 years +) population
Total number of people 15 years+ who cared for one or more children in the last two weeks	655,622	22.98%
Cared for own child/children	469,283	16.45%
Cared for other child/children	162,873	5.71%
Cared for own child/children and other child/children	23,466	0.82%
Total number of people 15 years+ who did not care for any children in the last two weeks	2,197,815	77.02%
Did not provide child care	1,460,120	51.17%
Not stated	173,548	6.08%
Not applicable	564,147	19.77%
Total population	2,853,437	

 Table 3.2: Number of adults in South East Queensland\* who report caring for (unpaid) one or more children in the last two weeks, 2011 (source: (Australian Bureau of Statistics, 2011))

\*The geographical boundaries used in this survey (QLD Major Urban) does not precisely match the study area as it includes both the study area as well as small portions of Ipswich and the Sunshine Coast

## 3.3.4.3. International travel

South East Queensland is home to two large international airports, as well as a cruise ship termal. On average, Brisbane International Airport and Gold Coast International Airport together receive over one million arrivals per month, peaking in July and October, and decreasing in February (Figure 3.8) (Australian Department of Infrastructure Regional Development and Cities, 2018). While the majority of these passangers are international vistors and/or people transitting through to onward destinations, a portion of them are local SEQ residents who are returning home from shortterm visits overseas. These residents can serve as potential vectors, introducing overseas-aquired *Cryptosporidium* into the local community.



Figure 3.8: Monthly passenger arrivals at Brisbane International Airport and Gold Coast International Airport, 2009-2017(Australian Department of Infrastructure Regional Development and Cities, 2018)

Of those Queensland residents returning from short-term stays abroud, slightly more than half of them are arriving from desitination with a lower risk of aquiring cryptopsoridiosis infections such as New Zealand, western Europe or North America (Figure 3.9) (Australian Bureau of Statistics,



**Figure 3.9:** Percent of Queensland residents returning from short-term overseas travel, by age and destination-related *Cryptosporidium* transmission risk, July 2007 – 2017 (Australian Bureau of Statistics, 2017)

Of those residents returning from high-risk destinations, the majority came from South and South East Asia, Oceania (not including New Zealand), and East Asia (Figure 3.10). Children 0-4 years old represent a small proportion of all returning residents, irrespective of travel destination.



**Figure 3.10:** Queensland residents returning from short-term overseas travel to high-risk destinations, by sub-region of travel, July 2007 – 2017 (Australian Bureau of Statistics, 2017)

## 3.3.4.4. Source of drinking water

Most residents in the study area obtain their drinking water from community water supply (Table 3.3). Catchment management, bulk water supply and drinking water treatment in South East Queensland is the responsibility of Seqwater, a statutory authority of the Government of Queensland. Under the Australian Drinking Water Guidelines, Seqwater is required to remove pathogens including *Cryptosporidium*. This is accomplished through a multi-barrier approach of source water protection, microfiltration during water treatment process, and UV disinfection at treatment plants with high levels of turbidity that may reduce the effectiveness of the filtration stage (Seqwater, 2013).

Drinking water source	Brisbane North	Brisbane South	Gold Coast
Community water supply	93.2%	84.7%	91.7%
Tank	2.3%	9.1%	4.9%
Other	2.3%	2.3%	0.0%
Spring	2.7%	0.4%	0.0%
Never drink tap water	1.2%	4.8%	2.1%

**Table 3.3:** Primary source of drinking water by location of primary dwelling, 2012 (source:(Australian Bureau of Statistics, 2012a))

While there is no routine testing to detect *Cryptosporidium* in drinking water systems, several samples have been taken from catchments supplying the study area. Of those tested, all had low levels of *Cryptosporidium* (<4 oocytes/ 10 litres) (Seqwater, 2012). Additionally, no reported cryptosporidiosis outbreaks within the study have been attributed to the community water supply. For that reason, drinking water is not considered to be a main source of *Cryptosporidium* exposure in the study area.

## 3.3.4.5. Contact with livestock

While contact with livestock is considered a risk-factor for acquiring *Cryptosporidium* infections, it is not considered to be a major source of infection in the study area. Residents of the area have, in general, little-to-no contact with livestock due to its urban and peri-urban nature. Additionally, few people in the study area are employed in the agricultural industry, with 709,826, and 314 people living in Metro North, Metro South and the Gold Coast respectively reporting in the 2016 census that they work in livestock-related agricultural jobs. This represents less than 1% of the employed persons in those regions (Australian Bureau of Statistics, 2016).

The one exception is the Royal Queensland Show (commonly referred to as 'Ekka'), which is an annual 10-day agricultural show held in the centre of Brisbane in mid-August. During this show, which is attended by over 400,000 visitors and over 10,000 animals annually, the general public is given the opportunity to interact with livestock on display and in petting zoos (RNA, 2016). In the past, the EKKA has been implicated in at least one large zoonotic disease outbreak. In 2013, a petting zoo at the Ekka was associated with a confirmed outbreak of Shiga toxin producing E. coli O157 (STEC) infection involving 57 cases (Government of Australia, 2013). Never the less, there have been no reported outbreaks of *Cryptosporidium* infections associated with the Ekka, nor any major outbreaks that coincide with the timing of the show.

# 3.4. Cryptosporidiosis in South East Queensland as a complex environmental health problem

The trend of cryptosporidiosis notifications over time (as shown in Figure 3.6) demonstrates how effective management solutions for the cryptosporidiosis problem in South East Queensland have thus far eluded local and state decision makers. The nature of this problem shares many of the characteristics of complex problems as defined in section 2.3.1. For example, this problem is characterised by nonlinearity, system outputs (e.g. number of new cases in the community) are not directly proportional to the inputs (e.g. infectious cases in the community), demonstrating disproportional cause and effect. Feedback loops also drives this problem, such as the circular

relationship between the number of new cases in the community and the number of infectious cases that are currently in the community. This problem also exhibits emergence, where higher order system behaviour arises from the collective interactions of system components (e.g. public aquatics facilities, the public health system response, international travel, etc.), but cannot be explained solely by examining the components alone. Additionally, the irregular frequency of outbreaks makes it difficult to identify what is causing the outbreaks, and therefore makes it difficult to prevent outbreaks from occurring.

Beyond the complex nature of the problem itself, decision-making related to cryptosporidiosis in SEQ is also hindered by high levels of all three types of problem uncertainty (as described in section 2.2). There is significant natural variability and heterogeneity (aleatoric uncertainty) in factors such as susceptibility, infectivity, exposures, and human behaviour that exist within the systems, which make it difficult to quantify or predict the effects of decisions on the local population. Additionally, there is broader systemic uncertainty (epistemic uncertainty) related to a lack of information and consensus regarding the system of cause and effect relationships, as well as the parameters within a system specific to SEQ. For example, there is very little SEQ-specific information about Cryptosporidium exposure events, person-to-person and environmental contact behaviours of people within the community, or treatment behaviours of local healthcare providers. Additionally, the interconnections between person-to-person exposure, waterborne exposure and zoonosis is unclear in the region. Finally, despite there being a strong level of certainty related to the individual-level biological mechanisms of transmission, there is a great deal of deep uncertainty related to the local community-level social, environmental, technological and health process driving this problem at the population-level. For example, it is unclear whether some unknown factor is driving the seemingly cyclic 2-3 year pattern of outbreaks, or if this is just a random effect.

The problem of cryptosporidiosis in South East Queensland is a clear example of a complex problem that environmental health practitioners must tackle on a regular basis. Despite *Cryptosporidium* being a relatively well researched issue, local decision makers are not equipped with the tools that can capture the degree of complexity presented by this problem and translate the existing evidence into actionable information. It is for this reason that the problem of cryptosporidiosis in South East Queensland has been chosen as a case study for this project to explore the value of system dynamics modelling as a decision-support tool for environmental public health decision-making processes.

## 3.5. Summary

The intention of this chapter was to provide a broad overview of the current state of the literature regarding *Cryptosporidium* and cryptosporidiosis, as well as background on the case study location and the local context of known cryptosporidiosis risk factors. Current research indicates that *Cryptosporidium* has numerous modes of transmission, many of which are temporarily or geographically-specific. Recreational water, and particularly swimming pools, is a major source of transmission, and presents numerous management challenges. The chlorine-resistant nature of this pathogen allows it to persist in swimming pools for an extended period of time, allowing for prolonged periods of exposure to high-risk populations (i.e. small children). Though limited, the research also indicates cryptosporidiosis remains a persistent problem in SEQ, particularly during the summer months when swimming pool attendance is at its peak. While common sources of exposure such as drinking water and contact with livestock are reported to play an important role in SEQ. The information presented in this chapter forms much of the evidence-based foundation that was used to support the qualitative systems map and quantitative system dynamics model presented in chapters 5 and 6 respectively.
# **Chapter 4.** Methodology and Methods

This chapter is broken into two sections, an overview of the general methodology used in this thesis, and a description of the methods used in the case study. This chapter will build on the discussion of system dynamics in chapter 2, describing system dynamics from an applied perspective. It will also introduce action research as a complementary methodology for studying the applicability of systems thinking and system dynamics to environmental health decision-making.

#### 4.1. Theoretical approach and justification

#### 'there is nothing so practical as a good theory' – Kurt Lewin (1951)

From a broader perspective, this research follows a constructivist paradigm, the premise of which is that the truth is dependent on, and relative to, one's perspective. The ontology of the constructivist paradigm is relativist, meaning that multiple realities of the same situation exist, and that reality is socially constructed (Labonte and Robertson, 1996). The constructivist paradigm, as adopted in this thesis views that the prior ideas and experiences of the research participants shape their individual and collective experience of reality. Therefore, this project seeks to bring together multiple realities and perspectives to develop a consensus construct of a problem.

This research in this thesis combines two different methodologies that are often thought to sit on different ends of the continuum of research paradigms: action research and system dynamics. Action research (AR), which predominantly sits on the qualitative side of the research paradigm continuum, involves the 'researcher' and the research 'subjects' jointly defining a problem and then undertaking a sequence of iterative cycles of action and reflection, where each cycle seeks to prove or disprove the findings of the previous cycle (Scholl, 2004).

System dynamics (SD), on the other hand, traditionally sits on the quantitative side of the continuum of research paradigms. Traditionally, system dynamics modelling takes a top down approach, with the modeller (or team of modellers) identifying a problem within a problematic situation, defining variables and interactions, and then simulating the behaviour of system they are trying to model.

While AR and SD may differ in the way they approach problem solving, they share a number of similarities that make them well suited for integration into a combined methodology. The following section describes each methodology, as well as the commonalities and differences between them. It

concludes with a description of how this research project has joined AR and SD into a combined method using participatory model building.

## 4.1.1. System Dynamics Modelling

As discussed in Chapter 2, system dynamics modelling approaches problems based on the premise that the structure of a complex system (i.e. the causal relationships among components) is the source of the system's dynamic behaviour. Therefore, to effectively develop policy interventions for complex problems, one must understand the structure of the system.

From an applied perspective, system dynamics modelling is a process where a mathematical simulation model is constructed through a series of iterations of increasing complexity as the model is built and continuously refined. New information that is gathered during each portion of the process is used to future refine past and future portions of the process (Sterman, 2000a). Once produced, the model can be used to predict if changes in policy variables, structures or mental models can lead to improved performance (Ford, 2009).

The SD modelling process is both flexible and intuitive, which means that each project will provide a unique output. However, the process to develop a system dynamics model can be generalized into the following four iterative methodological phases (as proposed by Maani and Cavana (2007)): (1) problem structuring, (2) causal loop modelling, (3) dynamic modelling, and (4) scenario planning and modelling (Figure 4.1).



Figure 4.1: Model building process according to Maani and Cavana (2007)

As with all dynamic system modelling methodologies, the steps in the process are not expressly linear, and information gained in each step is used to refine prior steps. There is also no specific endpoint in the modelling process as the model continuously evolves as the system which is being modelled evolves in the real world (Sterman, 2000a). It is important to note that the learning contained within the conceptual and simulation models sit within the broader context of the continuous learning cycles occurring within the organization, society and the environment. Continuous changes within those systems provides feedback, in response to which the model can be progressively modified.

### 4.1.1.1. Problem structuring

The first step of SD modelling process, the problem structuring stage, involves identifying the root problem to be modelled. It is arguably the most important stage in model design, as it ensures that the correct problem (or element of the problem) is modelled, and that the model has a clear purpose (Sterman, 2000a). According to Sterman (2000a), key questions to be answered at the stage include:

- What is the problem that is being addressed?
- Who are the main stakeholders involved?
- Who are the model's intended users?
- What are the conceptual, temporal and geographic boundaries of the inquiry?
- What are the key variables and relationship that make up the system in which the problem sits?
- Is SD modelling the correct methodological approach for the problem?

One common characteristic of complex problems is that the root problem that is causing the symptoms is not always apparent at first inspection, nor is the solution obvious once the problem has been defined (Kreuter et al., 2004). It is important at this stage to differentiate between the true problem and symptoms of the problem. It is also the stage where the scope and boundaries of the problem are defined, ensuring that only relevant information is included in the model.

#### 4.1.1.2. Causal Loop Modelling (dynamic hypothesis)

A dynamic hypothesis is a working theory of what factors and system behaviours caused the problem (Sterman, 2000a). Conceptually mapping the problem helps identify causal structures and feedback relationships within the model. The dynamic hypothesis forms the basis of future simulation models.

From a practical perspective, dynamic hypotheses are typically expressed in the form of causal loop diagrams (CLD). Causal loop diagrams are a type of diagram in which causal relationships between factors are linked together with arrows describing causality. Each arrow within the CLD is marked

with an 's' or 'o', polarity depending on the nature of the relationship between two variables. An 's' polarity is used to indicate that two variables move in the same direction. An 'o' polarity is used to indicate that two variables move in opposite directions.

A feature that differentiates CLDs from other causal diagrams is the identification of circular feedback relationships between variables. Reinforcing and balancing feedback loops within the system (as described in detail in section 2.3.4.1) are depicted using the (R) and (B) symbols respectively.

### 4.1.1.3. Dynamic Modelling

While qualitative causal loop diagrams are a useful tool for describing the structure of relationships between system variables and identify potential future intervention points, they alone cannot predict how dynamic changes in system structure and/or behaviour will impact future system outcomes. Quantitative system dynamics models have the advantage of being able to capture the dynamics of relationships between system variables. This provides users with the opportunity to gain insight on why a system behaves the way it does as a function of its structure.

It is important to note that unlike some other forms of mathematical modelling, the purpose of system dynamics modelling is not to produce numerical forecasts at a given point in time in the future, but rather to generate predictions that allow the user to compare how the system may behave under a variety of different policy scenarios. This allows users to better understand why and how certain outcomes can be achieved.

As described by Maani and Cavana (2007), system dynamics models are built from four key elements called stocks, flows, converters and connectors, each represented by a specific symbol (Figure 4.2). The symbols shown in figure 4.2 represent the standard symbols used in the Stella Architect program, which may differ slightly from those used in other system dynamics software.



Figure 4.2: Symbols used as part of the system dynamics modelling process

**Stocks** represent accumulations within the system. They are most easily thought of as a container in which something can either accumulate or be withdrawn from. The value of a stock depends on the net flow through the stock (inflows minus outflows). Mathematically, the value of a stock (S) as it changes over a given period of time (t), can be expressed by the following equation:

$$S_t = \int_{t_0}^t [inflow(t) - outflow(t)] dt + S(t_0)$$

Two types of stocks were used in the dynamic modelling stage of this research project, reservoirtype stocks, and conveyor-type stocks. Reservoir-type stocks, represented using a simple rectangle, simply accumulate their net flow. The contents of the stock are uniformly mixed together. Conveyor-type stocks, represented using a rectangle filled with vertical lines, operate like a conveyor-belt, where each element within the stock enters the stock, rides the conveyor for a given period, and then exits.

**Flows** control the rate at which materials is transferred to and from stocks. Flow that transfer material into a stock are called *inflows*, whereas flows that transfer material out of a stock are called *outflows*. A third type of flow, called a *biflow* (bi-directional flow), can change dynamically from an inflow to an outflow (or vice versa) depending on the system's behaviour. The cloud-shaped icons attached to flows represents the boundary of the system.

**Converters** are the auxiliary system elements that either represent the boundary of the system (i.e. elements whose value is not determined by the behaviour of the system itself) or are part of the

system itself (i.e. elements whose value is derived from system behaviour). Converters can be constants, dynamically updating equations (behavioural relationships) or graphical relationships. They are particularly useful in that they break-up complex equations into manageable components. The inclusion of converters into the system dynamics models also makes them easier to understand.

**Connectors** are used to depict the relationship between variables/converter and stocks, flows, or other variables/converters. Unlike flows, which represent actual transfer of material within the system, connectors simply represent the effect or influence that variables and/or converters have on other system elements. Two different types of connectors, distinguished by their appearance, are used in this model; *action connectors*, and *information connectors*. Action connectors, shown using a solid direct line, transmit actions (the result of a decision) between variables. Information connector, shown using a direct dashed line, transmit information needed to make decisions.

The dynamic modelling phase also involves testing the simulation models to determine whether they adequately represent past system behaviours (where appropriate), as well as testing the strength, robustness and sensitivity of the model when different parameters within the model are modified. It is at this point where the model can be used to identify leverage points, points where a small shift in one part of the system can have a large effect in other parts of the system. Identifying points of leverage allows users to direct future interventions to parts of the system where they are likely to have the greatest effect.

#### 4.1.1.4. Scenario planning and modelling

One of the greatest strengths of system dynamics models is their ability to be used for scenario planning. In this stage, 'policies', which refer to pre-defined intentional changes to one or more system variables, are postulated and tested (Maani and Cavana, 2007). The simulation model allows these policies to be designed, refined, and tested under various external conditions. It is through this analysis that some of the greatest insights from SD models arise.

#### 4.1.1.5. Implementation and organisational learning

The over-arching goal of system dynamics modelling is organisational learning and decisionsupport. Organisational learning can occur in several ways. The most tangible way is through using the policies and insights that emerge from the scenario planning and modelling in a real-world decisional context. An additional way that organisational learning can occur is through management flight simulators. These simulators typically have user-friendly interactive dashboard that users can explore and adjust (Maani and Cavana, 2007). Individual and organisation learning occurs as users experiment with different scenarios and expand their understanding of the possible system behaviours produced when structures internal and external to the system are changed.

4.1.1.6. Strengths of System Dynamics Modelling in the Context of Complex EH Problems In addition to being well-suited to capture the complexity of environmental health problems, system dynamic models have several strengths when used in this context. Proposed interventions where the effect on human health is unknown can be severely limited for moral and ethical reasons. Because of this, EH decision-makers have little opportunity to experiment with the outcome of potential actions before making decisions. SD models provide decision-makers with an opportunity to test the outcome of various potential interventions in an 'artificial world', before taking action in the real world. SD modelling also can accommodate the long time-delays that often exist within complex EH problems and therefore give decision-makers the ability to anticipate the long-term consequences of their actions. A scoping review of previous applications of SD modelling to environmental health decision making (Currie et al., 2018) (as discussed in Chapter 2) identified a number of additional strengths of using SD in the context of environmental health decisions, including the integration of information from separate sectors and planning processed into a shared understanding of the problem, the use of SD modelling as a platform for stakeholder engagement, and ultimately using the model's outputs as an advocacy tool for alternative policy options.

#### 4.1.2. Action Research

# "theory is really only useful insofar as it is put in the service of a practice focused on achieving positive social change" (Brydon-Miller et al., 2003b)

Action research is reflective and problem-focused research approach designed to pursue both research and action while diagnosing problems and developing practical solutions. Reason and Bradbury (2001) define action research as:

"a participatory, democratic process concerned with developing practical knowing in the pursuit of worthwhile human purposes, grounded in a participatory worldview which we believe is emerging at this historical moment. It seeks to bring together action and reflection, theory and practice, in participation with others, in the pursuit of practical solutions to issues of pressing concern to people, and more generally the flourishing of individual persons and their communities"

While a variety of action research methodologies exist, all have at least three features in common. The first is that both researchers and the practitioners/decision-makers are actively involved in the project. The collaboration between the researcher and the subjects, where they are joint actors in the inquiry, is one of the fundamental components of action research. The second is that the two groups jointly identify and define the problem and one or more potential interventions to decrease the problem or its effect. The third is the dual purpose of action (improving the subjects/ organization) and research (generating new knowledge) (Scholl, 2004).

Action research differs from more fundamental types of research in that it does not subscribe to the idea of value-free and objective knowledge-generation, but rather views knowledge-generation as explicitly socially-engaged and democratic (Brydon-Miller et al., 2003a). In the simplest terms, action research can be thought of as an investigation with people, rather than an investigation on or about people.

The process of action research has been described as repeated cycles of diagnosing, planning, acting, evaluating and reflecting (Kolb, 1984) (Figure 4.3). The process can be entered at any stage, but once started follows the cycle of steps.



Figure 4.3: The cyclical process of action research (adapted from Susman and Evered (1978))

This process also differs from traditional research approaches, which test a hypothesis by seeking evidence. The action research cycle instead builds confidence in its findings by attempting to refute the previous cycle's findings. An acceptable 'solution' to the problem has been found if and when the researchers and practitioners fail to find counter-evidence to refute the validity of the previous action (Scholl, 2004). The iterative process of reflecting on the previous action while planning the next action gives the user additional clarity on the relationship between the process and the problem being addressed.

# 4.1.2.1. Strengths and limitations of system dynamics modelling and action research in the context of complex EH problems

System dynamics modelling and action research are well-suited to a combined approach due to each methodology's ability to counteract some of the other's key limitations when used in the context of complex environmental health problems.

For example, implementing interventions where the impact on human health is unknown in the real world can be severely limited for moral and ethical reasons. Because of this, EH decision-makers have little opportunity to experiment with decisions. SD models provide decision-makers with an opportunity to test the outcome of various potential interventions in an 'artificial world', before acting in the real world. SD modelling also can accommodate the long time-delays that often exist within complex EH problems.

Conversely, as the outcome of many environmental decisions can have non-negligible impacts on individual and community health, action-related decisions must be strongly weighed against the possible health outcomes of the decision. The can severely limit the breadth of potential actions at the researcher's disposal when conducting traditional action research. Additionally, the long delays between action and noticeable change within the system that are often embedded within complex environmental health systems can pose a challenge to action research. Changes to the environment, such as the construction of a solid waste landfill, may have no noticeable health impacts for many years. This long delay can make it difficult to successfully reflect on the impact of an action before the next action-reflection cycle. The extended time scale of environmental health problems can hinder the usefulness of traditional action research in these contexts.

One of the key limitations of traditional system dynamics modelling is that it tends to be expertdriven, where the participation of stakeholders or practitioners is severely limited if they are not competent modellers themselves. This can both limit the amount of local knowledge drawn upon to design the model, but also limits the potential 'new knowledge' the stakeholders gain to insights gained from the final working model.

Oppositely, one of action-research's key strengths is that practitioners/ decision-makers and researchers work in unison on a project to improve practice generate new knowledge. The new knowledge is generated not only through the research findings, but also though the research process

itself. In the context of this project, a strength of an action research approach is that there are three forms of "new knowledge" that have the potential to be generated. The first is knowledge related to the community-level dynamics of cryptosporidiosis in South East Queensland that is gained from the model and the results of the policy analysis. The second is knowledge and insight about cryptosporidiosis dynamics that are gained from reflecting on the modelling process. The third form of knowledge is a higher-level conceptual form of knowledge related to how complex problems are investigated and approached by the local decision-makers.

#### 4.2. Research approach – Participatory model building

#### 4.2.1. Method overview

The approach taken for this research projects (shown in Figure 4.4) combined the modelling underpinning of a traditional system dynamics modelling process, with the collaborative and actiondriven nature of action research. For this project, the multi-methodology approach taken is referred to as participatory model building (PMB). Drawing from the definition of Action Research, a PMB approach was selected in recognition that those embedded within practical and decision-making process hold significant untapped knowledge and ability to understand and action problems that they are immediately confronted with. The PMB (also referred to as group model building) has been widely used by system dynamics practitioners (Siokou et al., 2014) and centres itself on incorporate the active participation of stakeholders and decision makers in both the scoping of the problem itself, and the exploration of potential interventions(Vennix, 1999).



Figure 4.4: Stages of the participatory model-building approach used in this research project (adapted from (Maani and Cavana, 2007))

Much like the diagnosis stage of action research, the first stage of the PMB process involved engaging with stakeholders to identify a problem of current relevance to them, for which potential upcoming opportunities for action exist. Through the PMB process, a problem that was likely to encounter opportunities for action was actively sought, to increase the relevance of both the model and the model-building process to stakeholders.

The following 3 stages of the PMB process drew heavily on the SD problem scoping and causal modelling process, except they take the form of a stakeholder-driven systematic inquiry. During this stage, the stakeholders and the researcher jointly developed a theory about the nature of the problem and the boundaries in which it sits. In order to capture the different mental models of the problem that exist within the stakeholder group, stakeholders were first given the opportunity map their understanding of the problem independently before the joint theory (causal loop diagram) of the problem was developed. The systematic inquiry process also sought to engage stakeholder in the process of identifying and bounding areas within the system where interventions may be possible. This was done to ensure that proposed interventions carried forward in the model-building process were realistic and potentially actionable. The final two stages of the PMB process closely followed the dynamic modelling and scenario planning stages of the SD modelling process.

The final stage of the SD process, implementation and organisational learning, which closely resembles the action and reflection stage of action research, fell outside the scope of this research project. The intention of this research is the provide stakeholders with insight on the dynamic system behaviours that contributed to the daily trend of cryptosporidiosis notification in South East Queensland communities over the past 10 years, in support of upcoming actions.

#### 4.2.2. Data collection

At the commencement of the project, a problem identification and scoping meeting was held with key member of the environmental health unit of the Queensland Department of Health. The purpose of this meeting was to identify one or more complex environmental health problems that are currently of interest to the Department, and for which there is a need for future decision support. The result of this meeting was that cryptosporidiosis, and particularly its assumed connection with public aquatic facilities in the region, was chosen as the problem to be investigated. Cryptosporidiosis was chosen as it is a particularly perplexing problem to Queensland Health because previous management interventions were not able to control outbreaks and the frequency of outbreaks was not regular, making it difficult to identify what the cause of outbreaks was and therefore difficult to control. Data to support the problem definition and causal modelling as part of this project were collected predominantly through consultation with stakeholder and key-informants and supplemented by a detailed review of the academic and grey literature. Stakeholders were identified in consultation with Queensland Health during the problem scoping meeting, and included individuals outlined in table 4.1. Consultation with stakeholder and key-informants occurred during of two participatory workshops, as well as several semi-structured interviews with stakeholders. All stakeholders were invited to attend the workshops. Stakeholders who were unable to attend a workshop were individually interviewed.

Organisation/ Industry	Roles	Number of participants
Queensland	Management (Health Protection)	6
Department of	Senior Environmental Health Officer	
Health	Senior Scientist (Microbiology)	
Public Health	Environmental Health Officer	8
Units	Public Health Physician	
Local City	Manager	27
Council	Environmental Health Officer	
Aquatic Industry	Swimming Pool Operator	3
	Swimming Pool Inspector	
Academic	Health Promotion	7
Researchers	Infectious Disease Management (OneHealth)	
	Microbiology	

Table 4.1: Stakeholders (by industry) who participated in the workshops and interviews

Two problem structuring workshops were held, the first attended by stakeholders from the Department of Health, local public health units, pathology laboratories, and public health and infectious disease researchers, and the second by stakeholder from the local government. Attendees collectively had expertise in various components of public and environmental health such as water management, epidemiology, outbreak investigations, policy, etc. Initially, workshop participants were given a short lesson on the concepts of system dynamics and qualitative causal mapping. Participants were then asked to independently, and collectively, brainstorm and group social/behavioural, environmental, technical, institutional, and economic factors related to *Cryptosporidium* transmission in the region. Following the brainstorming session, participants were divided into groups and used the identified factors to create causal maps and diagrams using either the software MentalModeler<sup>®</sup> or drawing them out on large sheets of butcher's paper.

Semi-structured interviews were also used to elicit information and data from key-informants who were unable to attend the problem structuring workshop. Like the workshops, interview participants were asked to draw on their professional experience to identify factors contributing to

*Cryptosporidium* transmission in South East Queensland. They were also asked to describe the possible ways in which the previously identified key components can influence the system.

Prior to the conclusion of the workshops and interviews, participants were asked to identify any known datasets that may be available to help populate a simulation model that is representative of the identified model structure and behaviour. This information was used request access for data from relevant data custodians for later use in the simulation model.

As this research involved conducting workshops and interviews, ethical approval to conduct this research was obtained from Queensland Health's Royal Brisbane and Women's Hospital Human Research and Ethics Committee (approval number: HREC/16/QRBW/509) and the University of Queensland's Human Research and Ethics Committee (approval number: 2016001630).

#### 4.2.3. Data Analysis

Factors and causal relationships included in the causal maps and diagrams created by workshop participants were initially combined in the Stella Architect (ISEE Systems<sup>®</sup>) modelling software to create a master causal loop diagram (CLD).

Data and information extracted from tape and video recordings of the workshops and interviews and researcher's field notes was then extracted and thematically analysed to identify any additional factors or system behaviours that had not been captured in the initial master diagram. This step involved open-coding, where key concepts (codes) from the data were identified and grouped together with other like-concepts. Codes were sourced both directly from the workshops and interviews, as well as from concepts and key terms identified in the literature. These were then added to refine the master causal loop diagram using an iterative process.

The resulting CLD provided a high-level conceptual overview of the stakeholders' understanding and assumption of the system's structure and behaviour. Variables and relationships in the combined CLD were validated against the research and grey literature, with particular focus giving to literature originating from Australia. A memo system was used to collate information from both the interviews and the literature that support and substantiate the inclusion of each variable and relationship that appeared in the diagram. Particular attention was given to finding confirmatory evidence from the research and grey literature to support the biological and physical plausibility of relationships identified in the CLD. None of the relationships identified by the stakeholders contradicted the existing body of research evidence. The CLD was then translated into a system dynamics stock-and-flow model. The memo system described above also included reference to any data or evidence that could be used to quantify the value of each variable and relationship. The model was then parameterised with available data based on the hierarchy show in Figure 4.5. In cases where data was not available, evidence from the literature (where available) was combined with local expert opinion to form a Beta-PERT distribution. The used of Beta-PERT distributions is described in more detail in Section 6.4.1.



Figure 4.5: Hierarchy of data and evidence used to parameterize the simulation model

A series of interventions that could be used to manage cryptosporidiosis in SEQ were created using themes identified in the workshops and interviews and then run as scenarios in the model to predict possible future outcomes and trajectories. Additional information about the creation and selection of scenarios is provided in section 7.2).

# **Chapter 5.** Conceptualizing the Problem Using Causal Loop Modelling

The main purpose of this chapter is to provide an overview of the first two stages of the system dynamics modelling process; problem structuring and qualitative causal loop mapping. The chapter is broken into three main sections. The first section describes the problem structuring process, including identifying the problem's reference mode, and establishing the scope and boundary of the system to be modelled. The second section describes the results of causal loop mapping and provides a detailed description of the main feedback loops within the system. The final section of this chapter outlines several initial policy-relevant insights that emerged from the causal mapping process.

The results described in this chapter are the outcomes of a series of workshops and interviews with key stakeholders (as described in Chapter 4.2.2) from the following sectors:

- Queensland Department of Health
- Public Health Units
- Local Government
- Aquatics Industry
- Infectious Disease/Public Health Research

#### 5.1. Problem structuring

#### 5.1.1. Reference mode

Characterising problems dynamically, that is, as a pattern of changing behaviour over time, is a central premise of system dynamic modelling. The term 'reference mode' is used to describe the graphical representation of the problem's dynamic pattern of behaviour and is used to guide the model-building process. From a practical perspective, the intent of the SD modelling process is to map, and later model, the causal structures and relationships that, when calibrated with local data, can reproduce the pattern of behaviour shown in the reference mode.

During the initial problem identification and scoping meeting, stakeholders identified the apparent cyclic trend in the weekly cryptosporidiosis notifications as the reference mode that captured the problematic system behaviour for which they sought an explanation (Figure 5.1). The pattern in

weekly cryptosporidiosis notifications was chosen to be the main reference mode for this project because it is the most readily available data on the incidence of cryptosporidiosis infections in the community. Additionally, it also represents the key source of information currently used by public health units to determine when interventions are necessary to control outbreaks.



**Figure 5.1:** Reference mode - Weekly notifications of cryptosporidiosis to the NCOS by PHU region, July 2007-2017

#### 5.1.2. Problem Articulation

As discussed in section 4.1.1.1, once the reference mode has been identified the first stage of the modelling process is problem structuring, which involves identifying the root problem to be modelled. In order to define the boundaries of the 'problem' that will be modelled, workshop and interview participants were asked to describe challenges and barriers to successful cryptosporidiosis prevention efforts in South East Queensland (SEQ) communities. Participants identified several recurrent themes related to these challenges, which were then broadly grouped according to the four main stakeholder groups for whom the challenges exist (Table 5.1).

Stakeholder Group	Challenges to disease prevention efforts	
Public Aquatic Facility Staff/ Operators	High bather load in peak season	
	Large number of incontinent patrons	
	Complexity of existing management standards and guidelines	
	• Staff and operator knowledge and training	
	Availability and design of hygiene facilities in existing aquatic facilities	
	Perceived low prevalence of cryptosporidiosis within the community	
	Highly seasonal workforce with large turnover	
	Financial viability of public swimming pools	
General Public	• Frequent contact with incontinent children	
	• Low awareness of cryptosporidiosis within the community	
	• Low knowledge of transmission and exposure risk factors	
	Confusion related to misinformation	
Public Health Units	Finite resource availability and allocation	
	Low priority of cryptosporidiosis relative to other notifiable conditions	
	• Cost and effectiveness of water sampling/testing	
	Difficulty detecting outbreaks	
	Delays within the surveillance system	
	Potential for politicised, reactionary interventions	
	Limited regulatory capacity	
	High levels of international travel within the population	
Medical Community	Provider familiarity with cryptosporidiosis	
	Providers' perceived prevalence of cryptosporidiosis within the community	
	Frequency of faecal testing	
	• Inconsistent provision of accurate transmission-related information to patients during pre- and/or post-test counselling	

# **Table 5.1:** Stakeholder-identified challenges to cryptosporidiosis prevention efforts in South East Queensland communities

## 5.1.3. Model scope and boundary

Subsystem diagrams are a useful tool for outlining the scope and boundary of the model, as well as capturing the high-level architecture of the system in which the problem sits. Key factors identified by stakeholders largely fit within one or more of the following categories: (1) disease dynamics, (2) population dynamics, (3) the primary healthcare system, (4) the public health sector, (5) public

aquatic facilities (PAFs), and (6) international travel. As seen in Figure 5.2, disease dynamics are directly affected by factors related to the interactions between individuals within the community (secondary transmission), as well as the management and operation of public aquatic facilities (environmental transmission). Overseas travel, which results in the introduction of overseas-acquired infections into the community, also has a direct causal link with local disease dynamics.



Figure 5.2: Subsystem diagram of the overall system structure of cryptosporidiosis in South East Queensland

Additionally, the primary healthcare and public health systems have indirect causal relationships with local disease dynamics. Symptomatic individuals seek medical attention from the healthcare system, which in turn leads to notification of confirmed cases to the public health system. The public health service then provides information to the public on how to modify their behaviour to prevent further disease transmission and advises/instructs the operators of implicated PAFs on how to remediate their facilities. The public health service also supplies information to primary healthcare providers to encourage doctors to give precautionary advice to patients on how to reduce the risk of transmitting their illness to others within the community.

While subsystem diagrams are a useful tool to understand the overall scope of the problem being modelled, they are not intended to comprehensively capture the causal structures and relationships that created the problem in the first place. The subsystem diagram shown in Figure 5.2 served as a starting point for more detailed causal loop diagrams used to convey stakeholders' collective dynamic hypothesis (i.e. their working theory of the systemic source of the problem).

#### 5.2. Causal loop modelling – development of a dynamic hypothesis

The causal loop diagram (CLD) in Figure 5.3 represents the combination of causal diagrams created by the workshop and interview participants, as well as causal relationships identified during the literature review that supported the information provided by local stakeholders. This diagram, which is explained in more detail in section 5.2.1, shows the non-linear causal relationships that contribute to the number of cryptosporidiosis cases in SEQ communities. Like the subsystem diagram, the CLD is organised into six categories, each shown in a different colour. Each category involves a different set of actors, all playing different roles in the problem.

Two reinforcing loops and eleven balancing loops were identified in the combined CLD, each representing a different transmission-related issue. Interestingly, the majority (9 out of 13) of loops identified involve variables from two or more sectors that highlights the multisectoral and interconnected nature of the problem. The section below describes each of these main causal loops and outlines the dynamics of the relationships within the loops. A more detailed explanation of the loops can be found in Appendix A.



Figure 5.3: Combined causal loop diagram of cryptosporidiosis dynamics in South East Queensland

#### 5.2.1. Analysis of the cryptosporidiosis conceptual model

#### Person-to-person transmission loop (R1) and PAF-related transmission loop (R2)

The first, and arguably core components of the dynamic hypothesis are the person-to-person transmission loop (R1) and the environmental (PAF-related) transmission loop (R2). These simple loops exhibit reinforcing feedback behaviour. In the cases of person-to-person transmission, the number of cryptosporidiosis cases in the community causes an increase in the number of healthy people exposed to *Cryptosporidium*. This has a reinforcing effect, causing the number of cases in the community to increase.



# Figure 5.4: Person-to-person transmission loop (R1) and public aquatic facility transmission loop (R2)

In the case of PAF-related transmission, an increase in the number of cryptosporidiosis cases in the community causes an increase in the number of infectious people swimming in local pools. As more infectious people swim in pools, the number of *Cryptosporidium* oocysts in the pools increases through contamination of the water by passive shedding of oocysts or by gross contamination in the form of an accidental faecal release (AFR). This increased oocyst load in pool water causes an increase in the number of healthy swimmers at risk of infection, which results in more cryptosporidiosis cases in the community. The combined reinforcing natures of these two feedback

loops is assumed to be the main driver of the growth of outbreaks within the community following an exposure event.

#### Use of healthcare services (loop B1 and loop B2)

The primary role of healthcare providers in the dynamics of cryptosporidiosis in SEQ is the education of infected individuals about the transmission risk they pose to others in the community because there is no approved treatment. This occurs most commonly after the patient's faeces have tested positive for *Cryptosporidium* (B2), or occasionally prior to a confirmed diagnosis (B1) (Figure 2.1).



Figure 5.5: Use of healthcare services (loop B1 and loop B2)

Participants noted that there are numerous delays between when a person becomes symptomatic, and when they are diagnosed with cryptosporidiosis. The likelihood of patients receiving correct information about their transmission risk is controlled by the doctor's awareness and knowledge of *Cryptosporidium* transmission. The outcome of any patients being aware of the risk of transmitting their illness is a reduction in the number of contacts they have with healthy individuals (B1a & B2a) and a reduction in the number of infectious people swimming in public pools (B1b & B2b).

#### Public Messaging campaigns (loop B3)

In Queensland, cryptosporidiosis is a notifiable disease. Therefore, a laboratory-confirmed case will be automatically notified to the state public health service. If the public health service observes

a significant increase in disease notifications in a given period suggesting an outbreak they may respond in several ways to limit transmission.



Figure 5.6 Public Messaging campaigns (loop B3)

The first control measure is public messaging campaigns to educate the general public about *Cryptosporidium* transmission (Figure 5.6). The intended effect of these campaigns is to increase awareness and knowledge of cryptosporidiosis within the community, with the goal of reducing infectious contacts between healthy and exposed people (B3a) and to reduce the number of infected people who swim while infectious (B3b).

### <u>Community awareness of crypto following public messaging campaigns leading to community</u> <u>members avoiding pools (Loop B7)</u>

In addition to causing infectious cases to avoid contact with swimming pools, public health messaging campaigns can have the effect of creating fear within the community (as seen in Figure 5.7). This, in turn, can potentially reduce transmission because fewer healthy swimmers are exposed to *Cryptosporidium* oocysts in pool water. An unintended consequence of this reaction is decreased revenue for the PAF operator. Study participants noted that the effect of this decreased revenue would not be uniform in the study area because some PAFs rely on admission fees as their main revenue source while other pools are publicly-funded and do not charge users an admission fee.



Figure 5.7: Community awareness of crypto following public messaging campaigns leading to community members avoiding pools (Loop B7)

#### Messaging directed at healthcare practitioners (loop R3 and loop B4)

Another control measure that is used following an outbreak is an awareness and information campaign directed at primary healthcare providers. The aim of this campaign is to raise awareness amongst doctors of the increased number of cases of in the community and to provide them with the contemporary messaging they should provide to their patients.

The effect of these campaigns creates both reinforcing and balancing feedback loops. A reinforcing loop (R3) is created when this increased awareness causes a temporary rise in the proportion of patients who are tested for cryptosporidiosis. A balancing feedback loop (B4) was created as the increased knowledge leads to an increase in the number of patients correctly counselled about their transmission risk, which in turn decreases the number of both secondary and PAF-associated cases of cryptosporidiosis.





It was unclear the degree to which (if at all) this activity was occurring in the community. Therefore, the model was constructed on the assumption that it was not currently a dominant loop in the system.

#### Swimming pool hyperchlorination at the request of the Public Health Service (Loop B5)

A third control measure implemented following the identification of a suspected PAF-associated outbreak is the closure and hyperchlorination of implicated swimming pools at the request of the Public Health Service. Additionally, if there is sufficient evidence to suggest that a particular PAF is implicated in an outbreak the current public health legislation (*Health Act 1937*) provides the authority to force the closure and remediation of the facility in the interest of protecting the health of the public (Queensland Health, 2004).



**Figure 5.9:** Swimming pool hyperchlorination at the request of the Public Health Service (loop B5) Study participants noted that in most cases it was difficult and time-consuming to isolate the source of cryptosporidiosis outbreaks. This creates a potentially significant time delay between the identification of a potential outbreak and implicating a specific swimming pool. Participants also noted that the difficultly in exposure source attribution for cryptosporidiosis outbreaks means that this feedback loop rarely occurs and PAF operators are unlikely to be directly made aware of ongoing outbreaks by the public health service.

#### Swimming pool hyperchlorination at the request of a patron (Loop B6)

Study participants from the aquatics industry noted that they are occasionally alerted by one of their patrons who is ill and suspects the pool is the source of their illness. These situations are frequently a source of confusion and frustration for both the PAF operator and the patrons as the pool has yet to be formally implicated. Stakeholders noted that these requests most often came from individuals who suspected they were infected with *Cryptosporidium* or another enteric pathogen but were not yet confirmed cases. Nevertheless, in these situations the pool operator may choose to proactively hyperchlorinate their pool, which creates a balancing feedback loop by inactivating any oocysts that may be present in the swimming pool.



Figure 5.10: Swimming pool hyperchlorination at the request of a patron (loop B6)

Unfortunately, in the system's current form, stakeholders indicated that swimming pool operators rely heavily on their patrons for awareness of cryptosporidiosis outbreaks within the community. The piecemeal and sporadic nature of this information, coupled with the relative rarity of outbreaks successfully attributed to specific aquatic facilities, means that swimming pool operators often have very poor situational awareness of cryptosporidiosis outbreaks when they are occurring. This low level of awareness has created a perception amongst many operators that cryptosporidiosis outbreaks are very rare within SEQ and not a significant concern for them.

#### Media-driven public awareness (loop B8) and policy change (loop B9)

Multiple stakeholders identified the media as playing a role in the prompting and amplifying the public health response to suspected outbreaks. Media outlets in the study area are known to monitor the weekly disease counts posted on the online publicly-accessible notifiable conditions surveillance reports and report any unusual changes that may indicate a disease outbreak. Additionally, Queensland Health posts information about confirmed outbreaks on a public website and social media to raise awareness of ongoing outbreaks.

Stakeholders noted that increased media coverage of outbreaks has two main effects on communitylevel cryptosporidiosis dynamics. The first (loop B8) is the creation of a balancing loop whereby increased media attention leads to increased public awareness of the presence of *Cryptosporidium*  in their community. This is assumed to lead to an overall increase in the public's (including symptomatic and asymptomatic cases) knowledge of transmission-prevention strategies, resulting in a decrease in the number of infectious cases contaminating local swimming pools. As the notifiable condition surveillance reports are only posted on a weekly basis, this loop is somewhat constrained by an information delay.





The second is the creation of an additional balancing loop whereby the increased public awareness of cryptosporidiosis within the community captures the attention of local and state politicians, who then call for additional guidelines and regulations to protect the health and safety of the public. In the case of cryptosporidiosis, this is likely to result in a call for additional guidance and training of PAF operators and staff, with the intention of improving the effectiveness of disinfection systems and thus reducing the number of *Cryptosporidium* oocysts in pool water. Loop B9 is also constrained by several major delays between public awareness of cryptosporidiosis in their community and improved effectiveness of disinfection systems. The first is a delay between increased public awareness and an increase in political will to more thoroughly tackle the problem. Politicians face many competing priorities and are assumed to respond in cases where there is significant public pressure to do so. This would require heavy and sustained media coverage and public attention. The second delay is a delay between an increase in political will and the creation of additional guidelines, resulting from the long and complex government policy-making processes.

The third major delay is a delay reflecting the amount of time stakeholders assumed it would take to implement any changes included in the new guidelines.

Some of the system behaviours associated with these two loops are based on stakeholders' experience with other similar notifiable conditions, as well as the outcome of outbreaks in other regions of Australia. This is because the relative lack of media and political interest in *Cryptosporidium*-related illness only rarely stimulates significant public health or political responses.

### Training and staff knowledge and experience (loop B10)

The PAF operations and maintenance section of the CLD contains two balancing loops. The first, shown in Figure 5.12, describes the relationship between training and staff knowledge and experience (loop B10). Stakeholders noted that due to the highly seasonal nature of employment in the aquatics industry, PAFs frequently experience high rates of staff turnover. As new and/or less experienced staff are hired during the busy season, the overall staff knowledge and experience at the facility drops. This drop is balanced out through increased training to upskill the new staff. A delay is present within this balancing loop to allow time for new staff to acquire the on-the-job experience necessary to competently manage the water quality of a PAF. Additionally, the ability to train new staff is also constrained by the financial resources of the PAF.



Figure 5.12: Training and staff knowledge and experience (loop B10) and financial disincentive to close pool for hyperchlorination (loop B11)

#### Financial disincentive to close pool for hyperchlorination (loop B11)

The second balancing loop in the PAF operations and maintenance section of the CLD describes the financial disincentive PAFs face in terms of routine hyperchlorination of swimming pools. The financial resources of PAFs is highly reliant on user fees, which requires sustained community attendance at the facility. As discussed in chapter 3.2.5, effective hyperchlorination of a swimming pool requires closure of the facility for between 12 and 25 hours. In the case of many swimming pools in the study area, this time exceeds the normal period that the PAF is normally closed each day. Therefore, hyperchlorination would require some swimming pools to be closed during their normal operating hours. This would reduce the user fees collected, which would provide PAF operators with a disincentive to practice hyperchlorination. Additionally, stakeholders noted that hyperchlorination of a pool immediately after detection of a loose or semi-solid AFR does not always occur because it requires closure of the pool and cancellation of all remaining activities for the day. This creates several logistical and financial implications for the pool operator, which is also a disincentive for proper hyperchlorination post-AFR.

#### 5.3. Initial insights from causal loop diagrams

Unlike other modelling techniques where the 'result' (i.e. output) of the modelling exercise takes the form of predictions, the primary aim of system dynamic models is not to produce predictions *per se* but to facilitate the development of an expanded theory of the relationship between system structure and behaviour. The outputs of this theory-driven exercise are commonly referred to within the system dynamics community as 'insights' (Stave et al., 2016). In the context of system dynamics, 'insights' also refer more generally to areas where the model provides information or an understanding that differs from currently held mental models of the system or the problematic behaviour it produces. While most of the insights originating from SD models come from quantitative simulation modelling, some initial insights, particularly those related to the broader conceptualisation of the problem, can also emerge from qualitative modelling.

The following section outlines a number of 'insights' produced as a result of the qualitative system mapping process and discusses their implications from a policy perspective.

#### Multi-sectoral nature of the problem

Of the thirteen feedback loops driving the transmission dynamics of cryptosporidiosis in SEQ identified by stakeholders, ten of them (71%) are 'multi-sectoral' (i.e. cross one or more sectoral boundaries). In particular, the CLD indicates that the majority of the balancing feedback loops

involve at least three sectors. Balancing feedback loops maintain balance within a system. In the case of infectious disease management, it is these loops that help keep individual exposure events from becoming major outbreaks. As with any cycle, feedback loops are only as strong as their weakest link. Despite identifying the numerous intersectoral linkages that exist within the system, there was broad consensus amongst stakeholders that there is insufficient communication and collaboration amongst different sectors involved in this problem. The cross-sectoral nature of these loops highlights the necessity of intersectoral collaboration to achieve greater balance within the system.

#### Role of compounding delays in the system

Several of the key feedback loops identified in the system were characterised by multiple material and/or information delays<sup>4</sup>. Numerous material delays were identified in the primary healthcare sector, including delays related to seeking care, being tested, testing positive, being notified of one's disease status, and notification of the positive result to the public health service/system. Each of these delays increases the time between the initial exposure event and the ability of any of the key actors to react. This is important because an infected individual may be infectious in the community for nearly their entire infectious period before they are made aware of their status. These material delays also compound information delays that are present in other areas of the system.

Sporadic cases of cryptosporidiosis are not routinely investigated by public health units (Queensland Health, 2015) and stakeholders indicated that there was no established threshold for how many notifications over a particular time period warrants an investigation. Therefore, it is largely discretionary when, and if, cases are investigated.

The amount of time that must pass before sufficient notifications are received by the public health units to prompt them to initiate an investigation is an information delay within the system. Similar information delays exist in relation to how long it takes the media and/or the general public to become sufficiently interested in an outbreak to bring about noticeable action. Awareness of the problem by infected individuals, the public health units, swimming pool operators, and the general public is key to reducing further transmission of the disease. In order for interventions to be successful, potential outbreaks need to be identified before they become community-wide events.

<sup>&</sup>lt;sup>4</sup> The term 'material delays' describes delays that modify the speed at which things flow in and out of stocks. 'Information delays' is the term used to describe delays in changes of people's perceptions or beliefs (Sterman, 2001b).

Early intervention for cryptosporidiosis requires significant reductions in the time delays identified in the system.

#### Leverage points within the system – System Archetypes

System archetypes are generic system structures that characterise patterns of behaviour seen in systems across a wide array of disciplines (Senge, 2006, Sterman, 2000a). These universal behaviours provide insight on the system structures creating problematic behaviour within a system. Analysing the archetypal structures within a system can help pinpoint potential leverage points for future action. (Senge, 2006). During the mapping process, behaviour associated with two main system archetypes were identified.

#### 1) Drifting goals – Outrage-driven action

Drifting goals describes how the gap between the actual conditions and the desired conditions (goal) can be achieved either through actions to adjust the actual conditions, or by lowering the goal (Kim, 1995). In cases where there is a significant delay between actions and a change in actual conditions, managers can take the easier path of simply lowering the goal to more rapidly close the gap between actual and desired conditions.

In the case of cryptosporidiosis in SEQ, there is gap between the number of cryptosporidiosis cases in the community and the acceptable threshold of cases within the community that are sufficient to justify an intervention (Figure 5.13). It should be noted that there is currently no specified or regulated 'acceptable threshold' of cases in the community. Nevertheless, there are unstated and unofficial thresholds that, when exceeded, will prompt decision-makers to call for action. Because these thresholds are informal, they can differ both geographically and temporarily. During outbreaks this gap may become sufficiently large to initiate actions to reduce the number of cases within the community, such as information and awareness campaigns (B1). Unfortunately, the effects of these interventions are difficult to measure and may take time to have an effect. Therefore, decision-makers may lose interest in the problem before the gap has been sufficiently closed, which in-turn decreases the time and resources dedicated to gap-closing actions (B2). This effectively raises the threshold for future interventions as the number of cases may still be above the previous threshold, yet the actions to reduce the gap have been curtailed.

There is one exception to this archetype behaviour. In some rare instances, an outbreak may receive significant media attention or public outrage calling for immediate action (essentially compelling decision-makers to lower their threshold for action) (B3). While this effect may be sudden and intense, it is typically short-lived. The reality of this behaviour is that the threshold for action is

controlled by the degree of outrage the problem is producing, rather than by evidence-based public health decision making.



Figure 5.13: Drifting goals archetype – Outrage-driven action

The key leverage point to the drifting goals archetype is to anchor the goal to an external framework or reference point, to stop it from drifting or being open to interpretation (Senge, 2006). In the context of this problem, this would involve transparently establishing a threshold for intervention that could be consistently applied across the region. This would also help counteract the effect of public outrage on the threshold for action by providing the public with a transparent rationale of when action is and isn't required.

#### 2) Growth and underinvestment archetype - Impact of situational awareness

A second system archetype behaviour that was identified within the system was the 'growth and underinvestment archetype'. This archetype characterises behaviour where success reaches a limit due to an underinvestment in the capacity needed to sustain continued performance. This behaviour will perpetuate itself as decreased performance is compensated for by decreasing performance standards, which in turn further decreases performance. (Senge, 2006).

Despite having a strong desire to do more to prevent cryptosporidiosis outbreaks in the community, low awareness of current outbreaks amongst PAF operators and staff was identified as being a major barrier to successful and sustained outbreak reduction. As outbreak reduction efforts become

more successful, the number of outbreaks in the community decreases (B1). As the number of outbreaks decreases, PAF staff become less and less aware of cryptosporidiosis as a potential issue that is relevant to them, which eventually undermines the outbreak reduction efforts (B2). In the case of cryptosporidiosis in SEQ, this decreased 'situational awareness' is further compounded by regular staff turnover, as new staff will have had little to no exposure to previous outbreaks. The less frequent exposure of staff to outbreaks or awareness campaigns will lead to a greater tendency to interpret cryptosporidiosis events as 'rare cases' (effectively increasing the number of incidents they need to be exposed to in order to change their belief that cryptosporidiosis outbreaks are rare). This cycle is only broken when the number or severity of outbreaks increases to such a level that they are compelled to act by an outside force.



Figure 5.14: Growth and underinvestment archetype - Impact of situational awareness

The key point of leverage in systems exhibiting growth and underinvestment archetypes is to identify and better understand the relationship between what is driving demand and performance, and what is the limiting factor. This can help sever the link between the driving and limiting factor. In the case of cryptosporidiosis in SEQ, there is need for greater communication between public health units and the operators of PAFs, particularly in keeping operators informed of increases in disease notifications in their region. Making them aware of disease trends, regardless of whether their facility is involved, may increase their appreciation of the magnitude of the problem at any given time, and encourage them to actively participate in interventions to reduce the severity of and prevent outbreaks.

#### 5.4. Conclusion

The causal mapping process presented in this chapter outlines a whole-of-systems approach to conceptualising our understanding of the community-level system structures driving the cryptosporidiosis in SEQ. This extends beyond simply presenting a list of factors, and instead focus on the dynamic relationships between factors that are producing the problematic system behaviour. The inclusion of diverse stakeholder perspectives produced a map of the problem extending beyond the typical view of cryptosporidiosis as a 'swimming pool management problem'. In particular, the role of international travel and primary healthcare, two sectors not typically involved in the management of cryptosporidiosis outbreaks, were identified as important drivers of the dynamics of this problem.

Beyond producing a more comprehensive description of this problem in SEQ, several insights emerged from the mapping process. Most of the balancing feedback loops in the system cross at least one sectoral boundary, indicating that the effective management of this problem will require a multi-sectoral approach. This was further highlighted by the insight that delays in one sector are likely undermining the effectiveness of interventions in adjacent sectors. Additionally, the lack of a consistent threshold for action is allowing the media/public outrage cycle to dictate the threshold for action, rather than evidence-based decision making. Finally, the lack of communication between the aquatics industry and the public health service has created a cycle where PAF staff incorrectly believe cryptosporidiosis is rare in the community, and therefore do not prioritise actions to prevent it.

# Chapter 6. Design, testing and validation of a system dynamics model of cryptosporidiosis dynamics in South East Queensland

The main purpose of this chapter is to provide an overview of the design, testing and validation of the stock-and-flow system dynamics simulation model that was constructed based on the qualitative causal loop diagrams presented in Chapter 5. The chapter is broken into five main sections. The first section provides a brief introduction to the model, and the modelling approach used. The second section describes the model's setup and constrains. The third section describes the structural and behavioural assumptions that form the basis of the model itself. The fourth section outlines the parameters that were used to populate the model, including how the model handles parameters with highly variable or uncertain values. The final section outlines the various tests used to validate the model, as well as their results.

#### 6.1. Introduction

As discussed in Chapter 5, qualitative systems thinking exercises such as causal loop diagramming (CLD) can provide significant insight on our collective assumptions about the systemic structures driving systemic behaviour. Nevertheless, an inherent limitation of CLDs is their inability to show the dynamic nature of causal relationships between system components. Because of that, they have limited utility as an operational tool to predict the how changes in one part of the system's structure and/or behaviour may affect the overall system's behaviour.

To operationalize the model of the system structures and relations uncovered during the qualitative modelling portion of the project, a system dynamics stock-and-flow simulation model was developed. The stock-and-flow model allows users to dynamically simulate the interactions between different elements within the system, as well as uncover potential leverage points for future action.

The overall modelling approach chosen for the cryptosporidiosis in South East Queensland (SEQ) problem was centred on the primary objective of endogenizing (i.e. find an internal explanation for) the transmission mechanisms associated with contact with contaminated recreational water at public aquatic facilities. The modelling approach therefore sought to outline the structures and feedback mechanisms that link infected people with public aquatic facilities, which eventually cause susceptible people to then become exposed. Specifically, using a modified Susceptible-Exposed-Infected-Recovered (SEIR) compartmental model, causal chains originating from the infectious
persons compartments were drawn, first to describe the processes driving how an infectious person come in contact with and contaminates the water at a PAF, followed by how a susceptible person then comes in contact with the contaminated water and becomes exposed to the pathogen.

The model was then expanded to include the relationships between several key sectors identified in the conceptual model, including overseas acquired cases, secondary transmission, and the public health service. Additionally, the role that the healthcare sector plays in this problem, particularly in closing the gap between the true disease incidence in the community and the number of disease notifications the public health service receives. The resulting composite structure formed the basis of the model used for scenario simulation.



Figure 6.1: Outline of causal structures included in the system dynamics simulation model.

Figure 6.1 outlines the relationship between the causal loop diagram described in Chapter 5 and the simulation model used for scenario simulation. Several factors identified in the causal mapping exercise were not explicitly included within the scope of the simulation model for the sake of model parsimony. Factors excluded from the explicit structure of the model were either implicitly included in the design of the scenarios used for policy analysis in section 7.2 (such as 'swimming pool staff knowledge and experience' and 'swimming pool operator training', which were key components of policy analysis scenarios 1A and 1B) or excluded due to a high level of uncertainty surrounding the factors impact on system behaviour ('political will', 'media attention' and 'swimming pool operator awareness of crypto in the community').

# 6.2. Model set-up and constraints

**Model purpose:** The purpose of this simulation model is to provide insight on some simple dynamic system behaviours, especially those related to public aquatic facilities, which are contributing to the overall trend of cryptosporidiosis notification in South East Queensland communities.

**Intended audience:** With the above-mentioned purposed in mind, the intended audience of this model are public health officers and managers in South East Queensland, as well as decision makers and facility operators in the region. While the model approaches the problem at a population level, some of the insights derived from the model may be of interest to the larger medical community.

**Spatial and temporal extent:** The model runs on a daily time-step over a period of 10 years, from July 1, 2007 to July 1, 2017. This amounts to a total of 3651 days.

The model represents the dynamics of *Cryptosporidium* transmission in South East Queensland, Australia, within the geographic boundaries of the Metro North Brisbane, Metro South Brisbane and Gold Coast Public Health Unit (PHU) boundaries, as defined by Queensland Health.

**Reference mode:** The primary reference mode for the model (i.e. the main variable whose behaviour over time is of greatest interest) is the daily count of case notifications for the entire SEQ study area, as seen in Figure 5.1, which have peak and trough pattern, with spikes ever 1-3 years (as discussed in section 5.1.1). There is also a slight overall upward trend of an increasing number of notifications over the study period. The desired system behaviour is a reduced number of daily case notifications in SEQ, particularly with shallower and less frequent peaks.

**Degree of aggregation**: The model examines the transmission dynamics of *Cryptosporidium* infections at the population level. It has been aggregated to the degree that there is no distinction between variations in individual persons, medical practices or practitioners, or public aquatic facilities. Public aquatic facilities are also aggregated in such a way that five shallow children's swimming pool and ten adult lap pools represent all the public aquatic facilities within each PHU region.

**Model boundaries:** The model's boundaries are designed to align with the boundaries of the causal loop diagrams discussed in Chapter 5. Several known factors related to *Cryptosporidium* transmission are considered outside the boundaries of this model. The simulation model focuses on the contribution of secondary transmission, international travel, and the use of public aquatic facilities to the population-level transmission dynamics of *Cryptosporidium* infections in the study

area. Other known sources of transmission such as contact with livestock and domestic animals, and contaminated drinking water are not included in this model. Additionally, only attendance at public aquatic facilities is addressed. The model does not account for individuals swimming in other sources of recreational water such as lakes, rivers, the ocean or private swimming pools. Weather and climate are considered exogenous to this model and are not accounted for other than the impact that various seasons have on attendance patterns at public aquatic facilities.

**Arrayed dimensions and elements:** System dynamics modelling allows for model structure and behaviour to be replicated across multiple dimensions, such as age or geographic region. These parallel model structures are called arrays (also known as subscripts). Each variable can be defined such that it differs in a pre-defined way for different arrays. Arrayed variables are show in the model structure as a three stacked variables (as seen in Figure 6.2) For example, the variable *proportion of people who swim* can be arrayed so that it has a unique value for different age groups and/or regions.



Figure 6.2: Example of three parallel stock and flow structures encapsulated within a single arrayed stock and flow structure.

This model has been arrayed across two main dimensions; by age, and by Public Health Unit (PHU). The dimension [Age] contains two elements, children 0-4 years old, and people  $\geq$ 5 years old. The distinction between these two age groups was chosen as children 0-4 years old are more likely to be faecally-incontinent. This increases their likelihood of coming into contact with faeces (their own or their peers'). They are also more likely to use specialized 'learn-to-swim' pools at public aquatic facilities.

The dimensions [PHU] contains three elements, Metro North, Metro South and Gold Coast. These three elements are consistent with the geographical boundaries of the three Public Health Units. The model was arrayed by PHU as the rate of cryptosporidiosis differs amongst the three regions.

The additional arrays have been used within the Public Aquatic Facility (PAF) Sector; pool size, pool type, and season. The [pool size] array divides the PAF sector model into large pools and small pools, to represent the differences in user profile and behaviour between users of lap-style swimming pool (large pools) and shallow learn-to-swim style pools (small pools). The large and small pools have further been divided into ten individual large pools ([large pool number] array) and five small pools ([small pool number] array) to represent the approximate number of council-owned swimming pools in each PHU region.

The last array is season, which was arrayed across two-dimensions, summer and winter. This array was used to differentiate between swimming pools that close in the winter from those that are open year-round.

**Software**: The software used to construct this model is Stella® Architect (version 1.7.1, ISEE Systems).

# 6.3. Model structure

The simulation model is made of six different interconnected sectors, the population sector (shown in green), secondary transmission sector (shown in aquamarine), healthcare sector (shown in red), Public Aquatic Facility (PAF) sector (shown in blue), imported cases sector (shown in purple) and public health sector (shown in orange), as seen in Figure 6.3. Each sector contains several causally-linked stocks, flows and converters (as shown in Figure 4.2 and described in section 4.1.1.3) that influence the way *Cryptosporidium* infections spread throughout the community. As variables from one sector may influence variables in several other sectors (and thus appear in the structure of other sectors), stocks and converters that appear in other sectors have been colour-coded to match the sector from which they originate (as per the colouring scheme described at the beginning of this paragraph). The following six sections describe the structural and behavioural assumptions used in each sector to collectively simulate the historical trends of cryptosporidiosis notifications in South East Queensland over the past 10 years.



Figure 6.3: High-level model structure

# 6.3.1. Population (SIER) Sector (Sector 1)

The Population Sector (shown in Figure 6.4) is both the starting point and the backbone of the entire system dynamics model. In this Sector, the population of each region, which are handled by seven stocks, move between different stages of the disease progression. The structure of this Sector follows a modified version of the SEIR compartmental model used in commonly epidemiology (Brauer, 2008). The classic SEIR model has been expanded to capture features specific to *Cryptosporidium* infections, such as the difference between symptomatic and asymptomatic infections, as well as symptom relapse in recovering symptomatic individuals.



Figure 6.4: Stock and flow structure of the Population Sector

# 6.3.1.1. Sector Summary

The first stock of the model, 'Susceptible People', contains the portion of the total population that is not infected with *Cryptosporidium*, nor are they immune to the parasite. These people represent the pool of people who have the potential to be exposed. The remainder of the people described in this model are considered "immune to exposure" because they are actively infected with parasite and therefore can't currently become infected. The number of susceptible people is influenced by population change (i.e. births, aging, deaths, immigration and emigration) over time. This effect is controlled by the rate variable 'daily population change'.

The remaining six stocks represent individuals at different stages of the natural progress of *Cryptosporidium* infections.

The second stock, **'Latently Infected People'** represents the number of people who are infected with *Cryptosporidium* but are in the incubation period. Individuals in this model can be exposed either through person-to-person contact or thought contact with contaminated water at a public aquatic facility (defined by the auxiliary variables **'new person to person cases'** and **'new swimming-related cases'** respectively). These individuals are not yet symptomatic, nor are they infectious. The mechanisms by which individuals are exposed to *Cryptosporidium* by person-to-person contact and contact with recreational water is discussed in sections 6.3.2 and 6.3.4 respectively. The average time spent in the latently infected stock is defined by the auxiliary delay parameter **'incubation period'**, i.e. infected but not yet symptomatic.

After the incubation period, a portion of people in this category move on to the third stock **'Symptomatic Infectious People'**, which contains the people who are currently experiencing

symptoms of cryptosporidiosis. People in this group are infectious, meaning they have the potential to transmit their infection to others. The amount of time where a person remain symptomatic is defined by the auxiliary delay variable **'duration of symptoms'**. Not all people who are infected with *Cryptosporidium* develop symptoms. The people without symptom instead move from the latently infected group to the fourth stock **'Asymptomatic Infectious People'**. Much like the people in the third stock, individuals in this stock have the potential to transmit their infection to others. The division of people between the third and fourth stock is determined by the auxiliary variable **'probability of being symptomatic given infection'**.

The fifth stock, '**Recovering Symptomatic Infectious People**', contains the symptomatic people who have ceased to display symptoms. These individuals remain infectious.

A fraction of people will move from the fifth stock to the sixth stock, '**Relapsed Symptomatic Infectious People'**. These individuals are people who experience a relapse of symptoms. The portion of people who fall into this category is determined by the auxiliary variable '**relapse rate**' and the amount of time that passes before the relapse occurs is controlled by the auxiliary delay variable '**reinfection delay'**. Individual in this stock return to the recovering symptomatic infectious people stock after a specified period, defined by the auxiliary delay variable '**relapse duration**'.

After a period defined by the auxiliary delay variable **'post-symptom infectious period'**, the people from both the symptomatic and asymptomatic infectious people stocks move to the seventh and final stock, **'Recovered People'**. Individuals in this stock are no longer infectious and are experiencing a period of temporary immunity (following their infection). The duration of time people remain in this stock is defined by the auxiliary delay variable **'immunity period'**. After their immunity period, the people contained within this stock will return to the **'Susceptible People'** stock at the beginning of the model.

# 6.3.1.2. Sector Dynamics

While the structure of the system is conveyed visually by the stock and flow diagram, the value of a stock as it changes dynamically over time is expressed with an ordinary differential equation (ODE). To illustrate the connection between the visual depiction of a stock and flow structure and the associated mathematical equations that drive the dynamic nature of the model, the differential equations that describe the dynamic change of each stock within the Population Sector, are show below in equations (6.1) to (6.8), described using the symbols listed in Table 6.1. Additional

information about the dynamics of this, as well as the ODEs driving the dynamics of the remaining sectors, can be found in Appendix A.

$$\frac{dS}{dt} = -(\Phi + \Psi) + \frac{R}{\lambda} + \Delta p$$
(6.1)

where,

$$\Delta \boldsymbol{p} = \boldsymbol{p}_{t-1} - \boldsymbol{p}_t \tag{6.2}$$

$$\frac{d\mathbf{E}}{dt} = (\mathbf{\Phi} + \mathbf{\Psi}) - \left(\frac{s\mathbf{E}}{\iota}\right) - \left(\frac{(1-s)\mathbf{E}}{\iota}\right)$$
(6.3)

$$\frac{d\mathbf{I}_{S}}{dt} = \left(\frac{s\mathbf{E}}{\iota}\right) - \boldsymbol{\varphi}\mathbf{I}_{S}$$
(6.4)

$$\frac{d\mathbf{I}_{\mathbf{A}}}{dt} = \left(\frac{(1-s)\mathbf{E}}{\iota}\right) - \rho\mathbf{I}_{\mathbf{A}}$$
(6.5)

$$\frac{d\mathbf{I}_{SR}}{dt} = \varphi \mathbf{I}_{S} + \left(\frac{\mathbf{I}_{SS}}{\pi}\right) - \left(\frac{r \mathbf{I}_{SR}}{\mu}\right)$$
(6.6)

$$\frac{d\mathbf{I}_{SS}}{dt} = \left(\frac{\mathbf{r}\cdot\mathbf{I}_{SR}}{\mu}\right) + \left(\frac{\mathbf{I}_{SS}}{\pi}\right) \tag{6.7}$$

$$\frac{d\mathbf{R}}{dt} = \left(\frac{(1-r)\mathbf{I}_{SR}}{\rho}\right) + \left(\frac{\mathbf{I}_{A}}{\rho}\right) - \frac{\mathbf{R}}{\lambda}$$
(6.8)

Table 6.1: Variables used in the ordinary differential equations to describe the dynamics of the population Sector

Text description of model variable	Symbol used in ODEs
SUSCEPTIBLE PEOPLE	S
LATENTLY INFECTED (EXPOSED) PEOPLE	Ε
ASYMPTOMATICALLY INFECTED PEOPLE	IA
SYMPTOMATICALLY INFECTED PEOPLE	Is
RECOVERING SYMPTOMATIC PEOPLE	I <sub>SR</sub>
RELAPSED SYMPTOMATIC PEOPLE	Iss
<b>RECOVERED PEOPLE</b>	R
daily population change	$\Delta p$
population	$\mathcal{P}$
new person-to-person cases	Φ
new swimming-related cases	Ψ
incubation period	l
probability of being symptomatic given infection	&
duration of symptoms	arphi
re-infection delay	μ
relapse rate	1~
relapse duration	π
post-symptom infectious period	ρ
immunity period	λ

# 6.3.1.3. Sector assumptions and limitations

The purpose the stock and flow diagram of the system (shown in Figure 6.4) is to make explicit our understanding and assumptions of the system's structure and behaviour. Nevertheless, the current depiction of the model's structure includes several additional implicit assumptions. These implicit assumptions are over-simplifications of our understanding of reality but have nevertheless been retained in the model for model simplicity and for ease of understand. For the Population Sector, these implicit assumptions include:

- Individuals who enter or leave the system do so only from the 'susceptible people' stock (i.e. no one dies in any of the other stocks, and no one enters the system already infected).
  - In reality, individuals may die, immigrate or emigrate from the population at any stage of the disease. As the number of diseased individuals is only a small fraction of the total population, and the duration of infection is relatively short, it is expected

that individuals entering or exiting the system while infected represents a negligible fraction of the population.

- 2. There is no treatment or outside means that would decrease the symptomatic period or duration of infectiousness.
  - Treatment of cryptosporidiosis using the drug nitazoxanide is available in a number of countries including the United States of America (Fox and Saravolatz, 2005).
     However, Nitazoxanide is not currently registered in Australia and is only available via the Special Access Scheme (Zhu, 2018). Because it is not routinely available in Australia, the model has been built to assume that no treatment is available.
- 3. Previous infection has no impact on the outcome of future infections once an individual has returned to the susceptible people stock.
  - There is no conclusive evidence that long-lasting immunity is conferred by infection with *Cryptosporidium* (see section 3.2). Therefore, the model assumes that previous infection has no impact on the outcome of future infections.
- 4. Symptom onset and onset pathogen shedding (onset of infectious period) occur at the same time in symptomatic infectious people.
  - The model assumes that the duration of the prepatent period (period from exposure to presence of oocytes in faeces) and incubation period (period from infection to onset of symptoms) are the same. While a number of studies have found these periods vary slightly (+/- 1-2 days) depending on the exposure dose (Chalmers and Davies, 2010). As the time difference is relatively short, and for model simplicity it is assumed that the prepatent and incubation periods are of equal duration.
- 5. Cryptosporidium infections are not fatal.
  - In immunocompetent individuals, *Cryptosporidium* infections are self-limiting, with symptoms ceasing typically in less than a month. However, In immunocompromised individuals, (particularly those whose T-cell function is compromised such as those with acquired immune deficiency syndrome (AIDS) or severe combined immunodeficiency syndrome (SCIDS)), *Cryptosporidium* infections can become chronic, progressive and potentially life-threatening (Hunter and Nichols, 2002).

While present in Queensland, rates of HIV in the state are believed to be quite low<sup>5</sup>. As the number of individuals in the study area whose T-cell function is sufficiently compromised to lead to fatal cryptosporidiosis is assumed to be quite small. For model simplicity, it is assumed that no one dies of cryptosporidiosis.

## 6.3.2. Secondary Transmission Sector (Sector 2)

The Secondary Transmission sector outlines a key component of most disease transmission models; person-to-person transmission. Person-to-person transmission has been implicated in numerous cryptosporidiosis outbreaks (as outlined in Chapter 2), particularly those involving children. As person-to-person transmission is not the main area of focus for this model, a simplified structure has been used to describe the dynamics of this form of transmission (shown below in Figure 6.5).



Figure 6.5: Stock and flow structure of the Secondary Transmission Sector

# 6.3.2.1. Sector Summary

The structure of the Secondary Transmission Sector begins with the variable '**potential infectors**', which is the sum of the daily number of new infections (i.e. the sum of '**asymptomatic infection**' and '**symptomatic infection**' flows described in the Population sector (section 6.3.1.1)). The model assumes that only a fraction of potential infectors will transmit their infection to others at some point in their period of infectiousness ('secondary transmission rate'). As young children are thought to be more likely than adults to transmit their infection to others, the rates of transmission for adults and children have been separated into the 'adult secondary transmission rate' and the

<sup>&</sup>lt;sup>5</sup> The 2012-2015 average rate of new HIV diagnosis in Queensland is 4.5 cases/100,000 persons, with less than 10% of those cases being at an advanced stage at time of diagnosis (Queensland Health, 2015).

'child secondary transmission rate' respectively. The model further assumes that <sup>3</sup>/<sub>4</sub> of people infected by children are adults (presumably their parents or caregivers) whereas adults are assumed to be equally likely to infect both children and other adults. The people that they infect then become '**new secondary cases**'.

A stock and flow structure is used to determine the point of time during the primary infector's period of infectiousness when they infect a susceptible person. The 'new secondary cases' drive the stock's inflow. A 'conveyor-type' stock is used as they allow for flow prioritization (i.e. first in-first out). These cases remain within the '**Susceptible Contacted People**' stock, until a pre-defined period of time where they flow out of the stock and become '**New person to person cases**'. This period represents the average time after the infector's onset of infectiousness that they infect a susceptible person. This variable is necessary as secondary transmission can occur at any point within the infector's period of infectiousness, but '**potential infectors**' is calculated at the point of time when the infectors first become infectious. The stock and flow structure ensures that the 'new person to person cases' enter the exposure flow of the person to person sector (described in section 6.3.1.1) at the correct point in time.

Another key assumption of this sector is that it is possible to prevent secondary cases, even when contact between susceptible and infectious people occurs. The model assumes the number of prevented (avoided) cases is dependent on the primary case being sufficiently aware of their potential to transmit their infection to others, that they are able to use transmission-prevention behaviours. This is included in the model using a second outflow from the susceptible contacted people stock ('avoided cases').

# 6.3.3. Healthcare Sector (Sector 3)

The Healthcare Sector describes changes in healthcare-seeking behaviour of symptomatic infectious people, as well as the testing, diagnosis and information-provision patterns of physicians. These changes ultimately effect both the number of symptomatic infectious people who are able to implement measures to reduce the risk of disease transmission, but also the number of disease notifications the public health service receives (described in the Public Health Sector model in section 6.3.6).

# 6.3.3.1. Sector Summary

The Healthcare Sector is built around three stocks, 'People with Crypto at the Doctor', 'People with Crypto Tested' and 'Aware Infectious People'. The first two stocks are controlled by bidirectional flows (bi-flows). Furthermore, both stock and bi-flow structure in the healthcare sector model exhibits a similar goalseeking behaviour. The behaviour of each flow is controlled by the gap between the predicted value of the stock and the actual value of the stock, as controlled by a delay variable. When the predicted value exceeds the actual value, the gap becomes a positive number and the bi-flow behaves like an inflow. When the actual value exceeds the predicted value, the gap becomes negative and the biflow behaves like an outflow. This produces goal seeking behaviour and allows the delay between people becoming sick and seeking treatment by a doctor, and the delay between seeking treatment by a doctor and being tested for crypto, to be modelled. A more detailed description of these stocks, in the form of their equations, can be found in Appendix B.



Figure 6.6: Structure of the stock and flow model in the Healthcare Sector

This section provides an overview of the structural and behavioural assumptions used in the Healthcare Sector (shown in Figure 6.6) to represent the mechanisms by which symptomatic infectious people are diagnosed and given education of means to reduce the transmission of their infection to others.

# 6.3.3.2. Patients' Healthcare-Seeking Behaviour

The first portion of this sector (Figure 6.7) describes the model assumptions related to patients' health-seeking behaviour. The model assumes that a fraction of symptomatic people from the population sector will visit a doctor, with this typically occurring several days after the onset of their symptoms.



Figure 6.7: Structure used to model the number of symptomatic infectious people who go to the doctor

The 'healthcare seeking gap', which is the difference between the actual number of people at the doctor and the predicted number of people at the doctor, creates a goal-seeking behaviour which is modulated by the treatment seeking delay.



**Figure 6.8:** Estimated effect of the treatment-seeking delay on the dynamics of the number of people 5+ years old with crypto at the doctor in Metro North (day 500-800)

Using the [*Metro North, Over 5 Years Old*] arrays for days 500-800, Figure 6.8 compares the Stella Architect modelled dynamics of the 'Symptomatic Infectious People', 'People with Crypto at the Doctor', People with Crypto Tested' and 'Aware Infectious People' stocks. The figure demonstrates the effect of the three delay variables in the Healthcare Sector, with delays clearly shown between peaks in the number of symptomatic persons, and the corresponding peaks in the other variables. A vertical reference line on day 608 has been added to demonstrate the value of each stock when 'symptomatic infectious people' is at its highest.

# 6.3.3.3. Diagnostic Approach of the Healthcare Provider

The next section of the model describes the assumptions related to the diagnostic approach of the healthcare provider (Figure 6.9). Of the people with symptoms who visit a doctor, a fraction of them will be asked by their doctor to collect and submit a stool sample to the pathology lab for testing. This process may take a few days ('**test submission delay**'). Not all symptomatic infectious people will have their faeces tested for a variety of reasons, including parasites rarely being considered in the initial differential diagnosis of diarrhoea, an initial treatment of just supportive therapy, a perceived lack of specific treatment options for *Cryptosporidium* infections making testing unwarranted, poor practitioner awareness of *Cryptosporidium*, and/or the perceived rarity of cryptosporidiosis in the community (Attias et al., 2015).

Once the faecal sample has been submitted, it will be tested. The '**testing gap**' is the difference the actual number of people with symptoms have been tested and the predicted number of people with *Cryptosporidium* infections who have been tested. This creates a goal-seeking behaviour that is modulated by the faecal testing delay.



Figure 6.9: Stock and flow structure used to model the fraction of symptomatic infectious people who get their faeces tested for *Cryptosporidium*.

Depending on the sensitivity of the testing method used, a portion of the samples will correctly test positive for infection with *Cryptosporidium*. For the purpose of this model, two different diagnostic

techniques are used; microscopy/antigen detection and polymerase chain reaction (PCR) analysis<sup>6</sup>. Each type of analysis has a different sensitivity to *Cryptosporidium*, with PCR considered more sensitive than microscopy/antigen detection. Beginning in 2013, the pathology laboratories began transitioning from faecal microscopy to the more sensitive polymerase chain reaction (PCR)

analysis ('testing transition') which may influence the annual trend of cryptosporidiosis notification.

The 'testing transition' variable uses a graphical function (Figure 6.10) to describe the percent of faecal samples that are tested using Multiple PCR detection vs. microscopy or direct antigen. The behaviour graphical function represents the transition from microscopy and antigen testing to multiplex PCR, which began in January 2013 (point A at t=2010), and became widespread in from 2015 (point B at t= 2755) to 2017 (point C at t=3102).



Figure 6.10: Graphical function of the Testing Transition (TT) Variables

# 6.3.3.4. Transmission-risk awareness

Lastly, the final section of this sector models the number of people who become aware of the transmission-risk related to a diagnosis of cryptosporidiosis (shown in Figure 6.11). For this purposed of this model, "awareness" is defined as the patient knowing:

- they are, or potentially are, infected with Cryptosporidium;
- the transmission risk associated with being infected with Cryptosporidium; and
- the appropriate transmission control measures necessary to reduce risk of spreading *Cryptosporidium* to others, including abstaining from swimming for 2 weeks after their diarrhoea has ceased.

<sup>&</sup>lt;sup>6</sup> While microscopy and antigen detection tests are fundamentally different diagnostic technique, laboratory billing practices make it unclear which proportion of tests were done with each technique during the study period as they are both use the same billing code. Because of this, the sensitivity of microscopy and antigen detection have been combined into a single variable (**'microscopy sensitivity'**).

The model assumes "awareness" instigated by the healthcare sector occurs in one of two ways, either by the doctor counselling the patient once their faeces has tested positive for *Cryptosporidium* (**'predicted aware infectious people'**), by the doctor pre-emptively counselling the patient at their initial visit when cryptosporidiosis is suspected (**'people not tested but aware'**).

In the first case, the laboratory notifies the physician of the case of cryptosporidiosis following the positive faecal test result, who will then contact the patient and inform them of the transmission risk associated with their diagnosis (**'predicted aware infection people'**). For the sake of model simplicity, this variable used a constant value of 0.43 (43%) is based on the study from Attitas et al. (2015) which found that only 43% of physicians knew the correct advice to provide to patients regarding measures to prevent transmission of *Cryptosporidium* infections.



Figure 6.11: Structure used to model the number of symptomatic infectious people that have been made aware of their transmission risk by their doctor

The process from sample submission to becoming aware of their diagnosis (and the associated measures to reduce the risk of transmitting it to others) is assumed to take between 1 and 5 days to complete. This delay includes the time it takes for the laboratory to return the results to the physician, and the number of days it takes for the physician to contact the patient to provide the results.

In the second case, a fraction of the patients who are not tested will nevertheless be counselled by their physician about measures they can use to reduce the risk of them transmitting their illness to others. The size of this fraction is based on the number of people who seek medical attention but do not get tested, multiplied by the physician precautionary advice rate. While these individuals that fall into this category may be unaware of their diagnosis, they are sufficiently aware of the transmission potential to implement risk-reduction strategies. Patients who are pre-emptively counselled are not subjected to the numerous delays in the Healthcare Sector, as they do not need to wait to be tested to receive the risk-reduction information.

The **'healthcare messaging effect'** (from the Public Health Sector in Section 6.3.6) variable describes the potential effect that messaging targeted specifically at healthcare professionals can have on their likelihood to communicate correct transmission-prevention information to patients they suspect have cryptosporidiosis. If/when the public health sector releases messaging targeted at healthcare professionals, both variables increase by an amount specified by the healthcare messaging effect variable.

# 6.3.3.5. Sector Assumptions and Limitations

The design of the Healthcare Sector incorporates several additional implicit assumptions and/or limitation, including:

- 1. Symptomatic infectious individuals only visit the doctor a single time.
  - A more complete model may account for individuals who seek medical attention more than once, as the doctor's differential diagnosis (and associated likelihood to test the patients' faeces for *Cryptosporidium*) may change over time as their symptoms progress. For the sake of model simplicity, it is assumed that patients only visit their doctor once during their infection.
- 2. Each symptomatic infectious person is equally as likely to visit the doctor as any other symptomatic infectious person.
  - A more complete model may include the relationship between healthcare-seeking behaviour and symptom severity and duration. Individuals with more severe symptoms, or symptoms that have lasted longer, are more likely to present to a doctor than those with mild symptoms, or symptoms lasting less than 3 days (Scallan et al., 2006, Kirk et al., 2014). For the sake of model simplicity, it is assumed that all symptomatic infectious people are equally as likely to visit a doctor.
- 3. Infectious individuals only need to submit a single faecal sample;
  - A more complete model equation might consider the frequent necessity to submit multiple stool samples before a positive or negative diagnosis is made. It is recommend that up to three stool samples are submitted prior to a negative test result being reported (Centers for Disease Control and Prevention, 2016b) due to intermittent shedding of oocytes. For the sake of model simplicity, it is assumed that only one faecal sample is necessary for diagnosis.
- 4. Physicians order the correct faecal test capable of identifying the presence of *Cryptosporidium* in faeces.

- A more complete model may also consider the number of people tested with a diagnostic technique that is not capable of detecting *Cryptosporidium*. As the number of times this occurs is unknown, and it is believed that this does not routinely happen in Australia, this has not been included in the model.
- 5. There are no false-positive test results.
  - A more precise model might also consider the specificity of each test, as it is likely that a small number of false positive test results contribute to the predicted number of cryptosporidiosis cases. As the number of individuals with diarrhoea in the community who are tested but do not have cryptosporidiosis is considered exogenous to this model, the number of false positive tests cannot be calculated. For the sake of simplicity, the model only considers the sensitivity of the tests.

# 6.3.4. Public Aquatic Facility Sector (Sector 4)

In addition to exposure to *Cryptosporidium* through person-to-person contact (as described in section 6.3.2), contact with contaminated water at public aquatic facilities is another means of exposure that is addressed in this model. The intention of the Public Aquatic Facility (PAF) Sector is to describe the mechanisms by which infected individuals contaminate the water at public aquatic facilities, as well as how susceptible individuals who swim at contaminated PAFs can be exposed. These mechanisms are represented explicitly in the model to endogenise the effect of PAF management and patron behaviour in this systemic problem. The overall structure of the PAF Sector can be seen in Figure 6.12.

# 6.3.4.1. Sector Summary

The Public Aquatic Facility Sector is divided into two related sections (sub-models). The first models the faecal contamination of PAFs through accidental faecal releases (AFR) or bather shedding (*swimming pool sub-model*). The second models susceptible people becoming infected by swimming in the contaminated water (*contamination exposure sub-model*)

To account for differences in the way people interact with different types and sizes of PAFs, the model within the PAF Sector has been divided and arrayed in several ways. The first is by swimming pool type, with a division between large pools and small pools (described in the model as LP and SP respectively). For the purpose of this model, 'large pools' are considered lap-style swimming pools, primarily used for recreational and competitive swimming, as well as other aquatic-based sports/activities such as water polo and aqua aerobics. 'Small pools' are considered shallow pools (often referred to as 'wading pools' or 'learn-to-swim' pools) that are designed

primarily to be used to teach diaper-aged children to swim. As can be seen in Figure 6.12, the design of the swimming pool sub-model contains two identical structures, one for large pools (left) and one for small pools (right) adjacent to each other. While the structural assumptions for the contamination of large and small pool is assumed to be the same, the value of the auxiliary variables within the model have been tailored to reflect the differences in the way each pool is typically used.

Additionally, each pool type has been arrayed to contain several pools of various sizes.<sup>7</sup> The large and small pool model structures have been arrayed into 10 and 5 different pools respectively. The number of arrays and the 2:1 ratio of large to small pools represents the approximate breakdown of council-owned pools in each Public Health Unit area. The pools within these arrays have been varied by size (from a 16m x 6m x 0.75m learn-to-swim pool to a 25m x 50m x 2m Olympic-sized pool) to represent the various sizes of council pools within the regions (Table 6.2).

Large Pools		Small Pools			
Pool number	Pool volume (litres)	Closed in winter	Pool number	Pool volume (litres)	Closed in winter
LP1	375,000	Y	SP1	205,200	Y
LP2	375,000	Ν	SP2	205,200	N
LP3	375,000	Ν	SP3	116,600	N
LP4	468,750	Y	SP4	69,570	N
LP5	468,750	Ν	SP5	69,570	N
LP6	468,750	Ν			
LP7	2,500,000	Ν			
LP8	2,500,000	Ν			
LP9	2,500,000	Ν			
LP10	2,500,000	Y			

Table 6.2: Volume of pools contained in the large and small pool arrays

<sup>&</sup>lt;sup>7</sup> Pool sizes have also been arrayed by PHU, with each PHU containing the same number of number of small and large pools.



Figure 6.12: Stock and Flow diagram depicting the complete Public Aquatic Facility (PAF) Secto

The variables 'Users in each LP' and 'Users in each SP', as shown in Figure 6.13, is used to divide the users from each PHU into the various pools, with a higher proportion of swimmers using the higher volume pools than the smaller volume pools. As many of the PAFs in SEQ are closed during the winter months (mid-May to mid-August) the variables 'seasonal LP users' and 'seasonal SP users' switch the users in three of the large pools and one of the small pools to 0 during these months.



Figure 6.13: Stock and flow structure illustrating the division of infectious swimmers into large and small pools.

Within the two sub-models are four modules. Modules (depicted as white boxes with rounded corners, as seen in Figure 6.13) are self-contained 'mini-models' embedded within the main model and are used to simplify its appearance. The 'Sick swimmer' and 'Healthy swimmer' modules calculate the estimated number of infectious and healthy people swimming in each pool. There are then two identical copies of the 'AFR' module, one for each size of pool (small and large pool), that contain the mechanisms used to predict the frequency and severity of accidental faecal releases.

As the assumptions dictating the system structure of the large and small pool portions of the model are identical, the following summary will focus only on that of the large pool. All assumptions regarding the large pool portion of the model also apply to the small pool unless otherwise stated.

# 6.3.4.2. Swimming Pool sub-model

The intention of the *Swimming Pool sub-model* is to imitate the dynamic system behaviour that leads to infectious people contaminating the PAFs.

#### Sick Swimmers

The Sick Swimmers module (shown in figure 6.14) is the starting point for *the Swimming Pool submodel*. It outlines the assumptions related to the estimated fraction of infectious people in the population who are swimming in a PAF at any given time. The model assumes that only a certain percentage of the population, regardless of their health status, swims in public aquatic facilities (they are referred to hereafter as "swimmers"). To estimate the fraction of infectious people who are considered 'swimmers', the number of infectious people is multiplied by the proportion of the population who report swimming in the last year.

As one of the key transmission prevention strategies is for infectious individuals to abstain from swimming, the portion of infectious swimmers who are 'aware' of these strategies (as described in section 6.3.3.1) is excluded from the calculation of infectious swimmers.

The estimated number of infectious swimmers who are swimming on any given day is calculated by

dividing the total number of infectious swimmers by the average frequency of swimming (swim events per year). While South East Queensland has a sub-tropical climate, the model assumes that seasonality does influence the rate at which people swim. This is primarily because a large percentage of public aquatic facilities are outdoors, many of which close during the colder months. It is assumed that people are more likely to swim during the summer months, with pool attendance peaking in January (mid-summer) and falling during the colder months (June-September). It is also assumed that this effect is less pronounced for the 0-4 year old age group as the majority of swimming is done as part of 'learn-to-swim' classes, which typically occur in indoor heated pools.

As the model addresses small and large pools separately, the number of child swimmers and adult swimmers in each pool are calculated separately. The model assumes that most young children (90%) swim exclusively in the small pool, and the







majority of adults (95%) swim exclusively in the large pool. These proportions are multiplied by the number of swimmers in both age groups to determine the estimate number of adult and child swimmers in each pool per day.

The sick swimmers module also captures the effect that targeted messaging by PAF operator has on the number of infectious swimmers in the pools (Figure 6.15). For this model, it is assumed that this messaging is targeted at both increasing swimmer hygiene and promoting self-exclusion of individuals with a recent history of diarrhoea.



**Figure 6.15:** Stock and flow structure depicting PAF-led messaging

The model assumes that the operators of the PAFs may release periodic messaging to their patrons about the risk of transmitting cryptosporidiosis to other patrons if they swim while infectious and/or do not shower prior to entering the pool. The uptake of the message depends when the message is released and how effective the messaging campaign is (i.e. the proportion of people who will modify their behaviour as a result of the messaging campaign). This assumption is modelled with the **'PAF messaging'** variable, which represents the fraction of the population that will modify their behaviour in accordance with the message being delivered. This variable is a delay variable whose input is controlled by the 'messaging converter' and whose output is delayed by the "PAF messaging effectiveness delay'. The timing of when the message is released ('messaging converter' variable) is controlled by two variables. The first is a switch variable that determines whether the messaging occurs. The second is a counter variable that releases a value of 1 at predetermined intervals ('PAF messaging start period'). The model assumes that the effect of the messaging campaign decreases over time, with the effect only lasting a short duration. The 'PAF messaging effectiveness decay' variable controls the length of time people continue to modify their behaviour based on the messaging they receive.

When the PAF messaging switch variable is on, the number of infectious swimmers who decide to self-exclude based on the PAF messaging are removed from the calculation of the number of infectious people in the pool. PAF messaging switch variable is switched off in the model's base case.

# **Accidental Faecal Release**

The 'ARF in pool' module, shown below in Figure 6.16, calculates the predicted number of oocytes released into the pool via an accidental faecal release, given the number of infected swimmers in the pool. The intention of the ARF module is to imitate intermittent AFRs that occur seemingly at random.

As watery diarrhoea is one of the most common symptoms of cryptosporidiosis, the module begins with the assumption that there is a given probability of an ARF given infection with *Cryptosporidium*. Multiplying this probability by the number of infectious swimmers provides an estimated probability of AFR per pool per day. A binomial random number generator ('AFR LP'), which releases the value '0' or '1' based on that probability, is then used to simulate days where an AFR occurs in one or more of the swimming pools. This structure insures that AFRs occur as discrete pulse-like events lasting only 1 dt.



Figure 6.16: Stock and flow diagram illustrating the mechanisms used to calculate the frequency and size of AFRs per pool per day

To calculate the number of oocytes released per ARF event, the predicted weight of faeces (in grams) released in an AFR is multiplied by the predicted concentration of oocytes per gram of stool. As guidance provided by the US Centers for Disease Control states that formed (solid) faeces poses little-to-no risk of transmitting *Cryptosporidium* infections, the model assumes that all AFRs are diarrhoeic faeces (Centers for Disease Control and Prevention, 2016a).

The predicted oocyte concentration per AFR is then combined with the result of the AFR generator process described above, using an additional IF, THEN, ELSE statement shown in equation 6.9.

$$Large \ ARF \ released = IF \ (AFR\_LP=1) \ THEN$$

$$Oocyte \ concentration \ in \ an \ AFR \ ELSE \ 0$$

$$(6.9)$$

The resulting value is a discrete pulse of oocytes (**'oocytes released into LP'**) released into the pool when it is predicted than an AFR has occurred.

#### **Bather shedding**

In addition to contamination resulting from accidental faecal releases, *Cryptosporidium* oocytes also enter the swimming pool through bather shedding. The figure below (Figure 6.17) show the portion of the model relating to this effect.



Figure 6.17: Stock and flow diagram illustrating the mechanisms associated with infectious bathers shedding faeces containing *Cryptosporidium* oocytes into the swimming pool

This section begins with the assumption that all swimmers will have some amount of residual faecal matter on their perianal area, which will be shed upon entering the swimming pool. The amount of faeces shed per swimmer is calculated as the amount of faeces (in grams) that the average person has on their perianal area, multiplied by the oocytes per grams of faeces (**'oocytes in 1 grams of stool'**). The amount of faeces shed has been separated by age group as young children, particularly those in nappies, are believed to have significantly more residual faeces on their body than those 5+ years old. The value of the **'oocytes in 1 grams of stool'** is assumed to be highly variable and therefore has been given a continuous probability distribution with a large range to account for

periods of intermittent shedding<sup>8</sup>. The number of oocytes shed per swimmer is then multiplied by the number of infectious swimmers to determine the number of oocytes infected swimmers will shed into the pool.

Showering, including cleansing the perianal area with soap, is believed to effectively remove residual faeces from the body. The model assumes that individuals who shower prior to entering the pool have 80% less faeces on their bodies than those who do not shower.

While a small percentage of swimmers routinely shower prior to entering the swimming pool, the model assumes that patron showering is also prompted by hygiene messaging campaigns. During these messaging campaigns, the number of swimmers who shower increases in proportion to the effectiveness of the campaign. The predicted number of oocytes associated with the proportion of swimmers who shower is therefore factored out of the calculation of oocytes shed into the pool <sup>9</sup>.

# Contamination and decontamination of the swimming pool

The figure below (Figure 6.18) shows the part of the swimming pool sub-model that describes how contaminants (*Cryptosporidium* oocytes) enter and exit the swimming pool water. Twice a day ( $dt = \frac{1}{2}$  a day) the sum of the total number of oocytes shed by swimmers and oocytes released by AFRs enter the '**Oocytes in the Large Pool'** stock.

Oocytes are then removed from the pool by chlorine inactivation, which the model assumes can happens in one of four ways: standard chlorine inactivation, log-3 disinfection, routine hyperchlorination, or reactionary hyperchlorination.

It is well-established that *Cryptosporidium* oocytes are highly resistant to disinfectants such as chlorine, and that it can take up to a week for oocytes to be inactivated in a well-maintained swimming pool with the standard free chlorine concentration of 1-3 mg/L (referred to in this model as "standard chlorine inactivation"). For the purpose of this model, the process of standard chlorine inactivation is modelled with by 'removal LP' flow, with the period of time required for oocyte inactivation (2-7 days) controlled by the 'oocyte inactivation LP' variable.

<sup>&</sup>lt;sup>8</sup> Intermittent shedding refers to the process where the faeces of infectious individuals may alternate between containing large numbers of oocytes, to containing little to no oocytes.

<sup>&</sup>lt;sup>9</sup> For the purpose of this model, the 'percent of pool patrons who shower' variable only includes individuals who effectively shower with soap (including cleansing their perianal area) prior to entering the swimming pool.

The model has also included the option for both large and small pools to have a secondary disinfection system, such as UV or ozone disinfection, that is capable of rendering 99.9% of oocytes inactive ('**log-3 disinfection'**). The model assumes that the system inactivates all oocytes as they pass through the system. According to Gage and Bidwell's *Law of Dilution*, assuming that a treatment system is able to remove all of the target pathogen as water passes through the system, only 42% of the pathogens in the pool are removed per turnover cycle (Health Protection NSW, 2013). Because of this, it takes 7 turnovers of the pool water to ensure that 99.9% of the water within the pool is treated. A log-3 disinfection system reduces the amount of time viable oocytes are present in the pool, from the standard 2-7 days, to 0.6-2 days, depending on the size of the pool and the pool's turnover rate.



Figure 6.18: Stock and flow diagram illustrating the mechanisms of contamination and decontamination in the large pool

A binomial switch variable (**'disinfectant type switch'**) is used to control whether the pools have a log-3 secondary disinfection system. The base case of the model assumes that none of the pools currently have such a system.

The remaining two methods of chlorine inactivation describe the process of hyperchlorination (also known as 'shock chlorination' or 'shocking'), which involves raising the free chlorine concentration in the pool to 10-20 mg/L, and maintaining that concentration for 12-25 hours (respectively) to ensure total oocyte inactivation in the pool. As these concentrations of chlorine can be harmful to

human health, the pool must be closed during the hyperchlorination process. The process of hyperchlorination may be reactionary, as part of faecal incident (AFR) response initiated routinely as part of a routine disinfectant procedure.

The model assumes that reactionary hyperchlorination is directly controlled by the percentage of AFRs that are detected by PAF staff ('**AFR detection rate**'). When an AFR is detected ('**AFR detected**'), the staff will initiate hyperchlorination procedures. This corresponds with all of the oocytes in the 'oocytes in the large pool' stock being removed via the 'decontamination LP' outflow.

Routine hyperchlorination is modelled in a similar fashion, with the decision of whether it occurs controlled by the **'routine hyperchlorination switch LP'** variable, and the number of days between hyperchlorination events controlled by the **'routine hyperchlorination frequency LP'** variable.

As the pool must be closed during hyperchlorination, the **'contamination LP'** inflow has a value of 0 whenever reactionary or routine hyperchlorination is occurring. For the base-case, the model assumes that 30% of AFRs are detected, and that routine shocking does not occur.



Figure 6.19: Modelled behaviour of 'oocytes in large pool' stock in relation to standard inactivation (left), reactionary hyperchlorination (centre) and routine hyperchlorination (right) [Metro North, LP4]

Figure 6.19 illustrates the dynamic relationships between the number of oocytes in the pool and the three inactivation methods. When standard inactivation occurs (left), the number of oocytes in the pool decreases exponentially over a period of approximately a week.<sup>10</sup> When reactionary hyperchlorination occurs (centre), the quantity of oocytes in the pool does not spike, but rather falls

<sup>&</sup>lt;sup>10</sup> The count of oocytes in the pool rapidly increases due to an AFR on day 2715.

to 0 following the AFR (shown occurring on day 2749). When routine hyperchlorination occurs, the quantity of oocytes in the pool falls to 0 at pre-defined intervals.

# **Contamination Exposure sub-model**

The design of the *Contamination Exposure sub-model* is strongly based on concepts originating in the field of risk assessment. To calculate the daily number of new cases attributable to public aquatic facilities, the model multiplies the estimated number of susceptible people who swim each day with the risk per person per swim (i.e. the probability of infection for a single exposure to PAF water – adjusted daily). The section below provides an overview of the structural and behavioural assumptions used in the *Contamination Exposure sub-model* to represent the dynamics of susceptible people becoming infected through exposure to contaminated pool water.

The Contamination Exposure sub-model begins in a similar way to the Swimming Pool sub-model, with a module describing the fraction of people in the population who are swimming in a PAF at any given time. As with the Swimming Pool sub-model, this sub-model is also separated by swimming pool type, containing two identical structures for large and small pools respectively. For the sake of brevity, the description of this sub-model will focus only on the structures related to the large pools.

# **Healthy Swimmers Module**

The structural assumptions of the *Healthy Swimmers module* (shown to the right in Figure 6.20) are nearly identical to those of the previously discussed *Sick Swimmers module*. The model assumes that only a fraction of susceptible people are swimmers, and that on any given day a fraction of swimmers will swim. Of those that do swim, the majority of adults will swim in the large

# **HEALTHY SWIMMERS MODULE**



**Figure 6.20**: Stock and flow structure of the Healthy Swimmers module

pool, whereas the majority of young children will swim in the small pool. The resulting variables, 'healthy people in LP' and 'healthy people in SP', serve as the module's output variables.

Once outside the module, the number of healthy people in the large and small pools are divided amongst the different swimming pool arrays (i.e. LP1-LP10 and SP1-SP5) to represent the predicted number of healthy swimmers in each of the PHUs' different swimming pools.

# Risk

Risk, in the context of environmental exposures, can be calculated as:

# $Risk = f(Dose \times Hazard)$ Dose = f(Exposure × Rate Introduced)

The model therefor assumes that the risk of Cryptosporidium infection per person per swim is:

 $Risk_{crypto} = f(concentration of oocytes in the pool water \times quantity of water ingested$  $<math>\times$  dose response relationship for cryptosporidium)

Figure 6.21 illustrates the portion of the *Contamination Exposure sub-model* where risk of infection per person per swim is established. This portion of the model has been arrayed by *age*, *pool type*, and *PHU*, to imitate the different degrees of risk associated with different types of pools and poolusers. The model assumptions for this section are as follows.

# Exposure

The quantity of oocytes that all swimmers (collectively) are exposed to is a function of the number of oocytes in each pool per day (calculated in the *Swimming Pool sub-model*) and the volume of each pool (described in Table 6.2).

# Dose

Several factors, in addition to the concentration of oocytes in the water, are considered when predicting the ingested dose of *Cryptosporidium* per swim.

It is assumed that only a fraction of the oocytes in the pool are viable (capable of causing infection). It is also assumed that the quantity of pool water ingested per swim event varies both by the swimmer's age, and by the size/type of pool they are using. Children, on average consume over two times as much water per swim event as adults (Dufour et al., 2006). While dose for waterborne pathogen transmission typically includes an element of time, the average volume of water consumed per average swim event was used to account for variations in time spent in the pool.

The amount of water consumed by adults while in the small pool was considered negligible, as adults would typically be in the small pool to assist young children who are learning to swim. It is not expected that this activity would requires adults to routinely submerge their face.

# Hazard and Risk

Several plausible dose-response relationships for *Cryptosporidium* have been suggested in literature. Brower et al. (2017) compared various dose-response curves and determined that only exponential and beta–Poisson functions were appropriate for waterborne transmission of *Cryptosporidium*. Based on these findings, a single-hit exponential dose-response function was chosen for this model.

A single-hit exponential model assumes that each viable oocyte acts independently and has the sample probability of causing infection, that the minimum infective dose is 1 oocyte, and that the distribution of oocytes between doses is randomly distributed (World Health Organization, 2016).

The equation for risk ('probability of infection per swim event') that was used in this model is:





$$P_{\text{infection/swim}} = 1 - \exp^{(-rN)}$$
(6.10)

where r equals the dose-response parameter (slope of the dose-response curve) and N equals the average number of oocytes ingested per swim.

The dose-response parameter is a measure of pathogen infectivity, representing the probability of infection per oocyte consumed. A meta-analysis of human dose-response data conducted by Messner et.al (2001) combining the estimates for three different *Cryptosporidium* isolates (UCP, IOWA, and TAMU) and estimated the risk of infection, given one oocyst of unknown strain per volume ingested, of 0.028 (80% CI: 0.005-0.066). As the strains causing infection in South East Queensland are unknown, the Messner estimate was used in this model.

# New swimming-related cases

The final portion of the sub-model addressed how the risk of infection per person per swim translates into new cases (Figure 6.22). The model assumes that the probability of infection per swim event multiplied by the number of swimmers swimming each day results in the number of new *Cryptosporidium* infection cases per swimming pool.



Figure 6.22: Stock and flow structure illustrating the process of susceptible swimmers becoming infected through contact with PAFs.

The combined number of new infections from all of the large and small pools within each PHU region make up the variable 'New swimming-related cases'. This variable controls the inflow to the 'Latently Infected People' stock in the Population Sector.

6.3.4.3. Sector Assumptions and Limitations

In addition to the explicit assumptions previously mentioned, the design of the Public Aquatic Sector includes several implicit assumptions and limitations:

- 1. Cryptosporidium is an obligate parasite
  - The model is based on the assumption that *Cryptosporidium* can only breed with a host, and therefore does not multiply while in the environment (such as in the mechanical components of a swimming pool). Recent studies have called this into

question, indicating that *Cryptosporidium* may have the capacity to multiply extracellularly (particularly in biofilms) (Koh et al., 2013). A more detailed model would consider the possibility that, under certain conditions, the number of *Cryptosporidium* oocytes in the environment could increase. As these findings are still preliminary and require further investigation, the model has not incorporated the concept of extracellular multiplication.

- 2. Pool systems only use chlorine inactivation as the method of disinfection
  - Several commercially available secondary disinfection systems (ultraviolet light or ozone systems) are capable of inactivating *Cryptosporidium* oocytes in aquatic facilities more effectively and efficiently than systems that rely solely on chlorine disinfection. A more complete model could account for the dynamics of various disinfection systems. Stakeholders from the aquatic industry have indicated that these systems are uncommon in the study area as they are expensive to maintain and require a higher-level of training to operate. For that reason, and for the sake of model simplicity, it is assumed that all pools in the region only use chlorine inactivation as the method of disinfection.
- 3. All pools have an established faecal response procedure that is compliant with the established guidelines for managing faecal accidents
  - Consultations with stakeholders from the aquatic industry have indicated that several the PAFs in the region likely do not have established faecal response procedure and/or their procedures are not implemented consistently and effectively. This would result in incomplete inactivation of oocytes in the pool following an AFR. A more complete model may account for the presence and effectiveness of AFR response procedures on the effectiveness of reactionary hyperchlorination. For the sake of model simplicity, it is assumed that all detected AFRs are appropriately managed.
- 4. All hyperchlorination events result in complete inactivation of oocytes in the pool
  - For hyperchlorination to achieve a 99.9% kill rate, the free chlorine concentration in the pool must be maintained at 10-20 mg/L for 12-25 hours (respectively). The use of chlorine stabilisers or failing to maintain the free chlorine concentration for an appropriate amount of time, can result in an incomplete kill. Consultations with stakeholders from the aquatics industry indicated that instances of ineffective hyperchlorination event may be occurring, particularly in pools with larger volumes.

As there is insufficient information about the effectiveness of hyperchlorination events in the region, it is assumed that they all achieve a 99.9% kill rate.

- 5. Minimum infective dose of Cryptosporidium oocytes is one (i.e. no threshold)
  - The use of a single-hit exponential dose-response model implies that infection is possible following ingestion of a single oocyte. This is not truly accurate for *Cryptosporidium spp. as* studies examining the dose-response relationship have found that the minimum dose is more likely ~10 oocytes (World Health Organization, 2016). A more complete model would therefore consider the minimum dose. Nevertheless, a single-hit exponential dose-response model is the approach recommended by the World Health Organization (World Health Organization, 2016). For this reason, the model assumes there is no dose-response threshold.
- 6. The dose-response relationship is equal within the population
  - The dose-response parameter used in this model is based on studies of the infection response mounted in healthy adult volunteers (Messner et al., 2001), which is potentially not representative of the response in all members of the population, particularly young children and the immunocompromised. As no child-specific data exists, the difference in risk between young children and the rest of the population was address through differences in exposure. As the model does not account for differences in immune status, it is acknowledged that the model likely underestimates the risk posed to immunocompromised persons.

# 6.3.5. Imported Cases Sector (Sector 5)

Although it is likely that most cases of cryptosporidiosis originate from within the study area, a small portion of cases are expected to be acquired when residents of SEQ travel overseas. Overseas travel is a known risk-factor for acquiring a *Cryptosporidium* infection, with some studies identifying it as one of the highest risk factors (Roy et al., 2004). From a population perspective, the number of overseas-acquired cryptosporidiosis cases depends primarily on the number of residents who travel overseas, and the travellers' destination of travel. The risk of acquiring *Cryptosporidium* infection overseas varies greatly by region of travel, with North America, Western and Central Europe, Japan, Singapore, Australia and New Zealand considered 'low risk' destinations, and travel to other areas of the world is considered 'high risk'. South-central Asia, a common travel destination for SEQ residents, is considered one of the highest-risk areas for travel-acquired cryptosporidiosis. (Weitzel et al., 2006).

The Imported Cases Sector (shown in purple in Figure 6.23) describes the contribution that imported cases play to the population-level disease dynamics in SEQ.





# 6.3.5.1. Sector Summary

This section provides an overview of the structural and behavioural assumptions used in the Imported Cases Sector to represent SEQ residents acquiring *Cryptosporidium* infections while travelling overseas, and then re-entering local population.

The movement of people departing for, and returning from, temporary overseas travel is modelled with the two **'Travellers'** and **'Exposed Travellers'**.

Each day a number of healthy people travel overseas by exiting the population model and flowing to the **'Travellers'** stock. As overseas arrivals and departures data (**'daily departures'**) from Australia's Department of Immigration and Boarder Protection were only available at the state level, the data were divided by the fraction of the state population living in each study regions (**'SEQ fraction'**).

The number of people travelling overseas from Queensland (shown below in Figure 6.24) varies considerably by passenger age and time of travel, with the highest rates of travel typically occurring during Queensland's summer months. Throughout the study period, there has been a steady overall
increase in the number of overseas departures, though this is likely due to population growth in the region.



**Figure 6.24:** Number of 'daily departures' for persons 0-4 years old (left) and persons 5+ years old (right) over the period of July 1, 2007 to July 1, 2017. (Source: (Australian Bureau of Statistics, 2017))

After a delay representing their duration of overseas travel ('length of travel'), overseas travellers return home, with a small fraction of them returning infected. This is represented by two outflows from the 'traveller' stock. Travellers who have not been infected flow back into the 'Susceptible People' stock, while those that have been infected flow to the 'Exposed Travellers' stock.

The model assumes that the likelihood of returning infected depends on their travel destination and the likelihood of exposure for each region. Data from Australia's Department of Immigration and Boarder Protection were also used to determine the ratio of low-risk to high-risk travellers (Figure 6.25). While the ratio varied considerably over time for persons 0-4 years old, the 5+ years old

People within the 'exposed travellers' stock are presumed to be infected, flowing back into the population model in either the 'asymptomatic infectious people' stock, or 'symptomatic infectious people' stock, based on the rate of asymptomatic carriage in overseas travellers. It is estimated that approximately 1/3 of overseas-acquired cases of cryptosporidiosis are asymptomatic (ten Hove et al., 2009).



Figure 6.25: Ratio of travellers returning from low-risk and high-risk countries for persons 0-4 years old (left) and persons 5+ years old (right) over time

# 6.3.5.2. Sector Assumptions and limitation

The following implicit assumptions and limitation result from the Imported Cases Sector of the model:

- 1. Overseas visitors do not contribute to the regional transmission dynamics
  - The model does not account for cases where overseas or interstate visitors transmit their infection to local residents. While it is acknowledged that overseas or interstate visitors may contribute slightly to the regional transmission dynamics, they were deemed exogenous from this model as non-residents are not routinely captured in disease surveillance systems. It is also assumed that interactions capable of transmitting infection between locals and visitors are sufficiently uncommon that they have not been included in the model.
- 2. The fraction of people who travel from each PHU region is equal
  - The model relies on state-level travel data, multiplied by the fraction of the population that lived within each part of the study region to calculate the estimated number of daily departures. This calculation assumes that the fraction of the population who travel overseas is equal within all regions of the state. It is acknowledged that this may not reflect the true distribution of travellers throughout the state.

0

- 3. All travellers are equally likely to acquire an infection overseas
  - A more complete model may account for the differences in risk of infection by reason for travel. Recent studies have shown that tourists are more likely to acquire travel-related illness than travellers for business or education (Angelo et al., 2017). For the sake of model simplicity, it is assumed that all travellers have equal likelihood of infection.
- 6.3.6. Public Health Sector (Sector 6)

The public health sector in South East Queensland is the sector primarily responsible for conducting disease surveillance and implementing population-level disease prevention and management measures. In the case of cryptosporidiosis, poor public awareness of disease, combined with the complexity and difficultly associated with identifying crypto outbreaks, means that the current prevention approach has centred primarily on public health messaging campaigns.



Figure 6.26: Structure of the stock and flow model in the Public Health Sector

The Public Health Sector (shown below in Figure 6.26) describes the public health surveillance system, as well as two different types of messaging campaigns run by the public health units in the study area.

## Sector Summary

#### Infectious case awareness

The model assumes that one of the key mechanisms to prevent the transmission of cryptosporidiosis infections in the community is through a certain proportion of infectious cases becoming aware of strategies to prevent transmitting their infections to others (**'aware infectious people'**). As described in section 6.3.3.4, this awareness can occur when physicians counsel their patients pre- or post-diagnosis.

The model further assumes that this 'awareness' can occur in two additional ways. The first is through infectious travellers being screened out and counselled upon arrival in the country ('**proportion of symptomatic travellers screened out**'). As only symptomatic returning travellers would self-identify as being potentially infected, the model assumes that no asymptomatic travellers would be affected by these actions.

The second additional way infectious cases can become 'aware' is by infected individuals who suspect they have cryptosporidiosis becoming aware of their transmission-risk though public messaging campaigns from the public health sector ('**Messaging Effect'**). This is modelled by multiplying the total number of symptomatic people (from the Population Sector) by the '**messaging behaviour change proportion'**.

The model assumes that once aware, individuals remain in the in **'aware infectious people'** stock for the average duration of their period of infectiousness.

#### **Disease Surveillance System**

The first portion of the Public Health Sector describes the dynamics of the disease surveillance system for cryptosporidiosis in the study area. As of 2001 cryptosporidiosis has been a notifiable condition in Queensland. In Queensland, laboratories must report all cases where there is laboratory definitive evidence that a patient has cryptosporidiosis to the state's Notifiable Conditions System (NOCS). Once in the register, public health units are made aware of the cases and can use this information to make decisions about disease control and prevention.

A simple goal-seeking stock-and-flow structure was used to model the surveillance system, using a similar structure to those described in the healthcare sector. The behaviour the biflow is controlled by the gap between the '**positive crypto cases**' (from the healthcare sector as described in section 6.3.3) and the actual value of the '**notified crypto cases**' stock, as controlled by the 'notification

delay' variable. The notification delay represents the average amount of time between faecal sample submission to the laboratory, and the case being notified to the NCOS.

## **Public Health Messaging**

The second portion of the sector describes the use of proactive messaging to change the behaviour of the general public and primary healthcare practitioners (i.e. general practitioners / family doctors). While the messaging targeted at the two audiences is modelled separately, the structure used to model them is nearly identical. For that reason, only the structure of the general public messaging will be discussed in detail.



**Figure 6.27:** Stock and flow structure describing preventative public health messaging campaigns As seen in Figure 6.27, the main variable within this portion of the model is the '**messaging behaviour change proportion**'. This variable is used to describe the proportion of infectious, on any given day, who become 'aware' (as described in section 6.3.3.1) as a result of contact with materials/messaging from the public health units (e.g. media releases, flyers, etc.). The model assumes that this proportion is primarily controlled by: 1) whether or not the PHU conducts a messaging campaign ('**PH Public Messaging switch'**); 2) when during the year the PHU releases the information ('**routine messaging start date'**); and 3) how effective the campaign is as a whole at reaching and informing potentially infectious individuals ('**Routine messaging effectiveness'**). It is assumed that the impact of the messaging is greatest right after it has been released and diminishes in an exponential-like manner over a given period of time ('**routine messaging effectiveness decay'**).

In other words, the model structure pulses the value of the '**routine messaging effectiveness**' variable, divided by the '**routine messaging effectiveness decay**' variable at predefined periods, which then decays exponentially. For example, when the messaging effectiveness is set at 5% and the decay is set at 15 days, the value of the behaviour change proportion will be 0.05/15. Over the

next ~14 days the behaviour proportion will decay such that the sum of the values over the 15 day period will equal the routine messaging effectiveness. This structure was chosen because the value **'messaging behaviour change proportion'** is then multiplied by the number of infectious persons on any given day (in the Healthcare Sector) and is added to the 'awareness rate' inflow of the **'Aware Infectious People'** stock. As this structure feeds into an inflow, a summative effect is created.

The assumptions driving the structure of the messaging directed at doctors is the same as the above discussed structure with one minor variation. As the 'Healthcare messaging effect' modifies auxiliary variables instead of a flow, there is no additive effect on its result. Because of that, instead of initially pulsing the effectiveness/decay, the full value of the effectiveness variable is pulsed on the first day. As with the other structure, the 'healthcare messaging effect' then decays exponentially.

For the base case scenario, the model assumes that the public health unit has an annual public messaging campaign that begins in mid-November. The messaging campaign results in 5% of the infectious population modifying their behaviour, and this effect occurs over 15 days. The model assumes that the public health unit is not currently providing messaging to doctors.

# 6.4. Model Parameters

In addition to the stock and flow structures that define the causal relationships within the system, each sector contains a number of model parameters. These parameters, in the form of auxiliary variables, transmit actions and information throughout the sector and ultimately affect the way stocks change over time. From a modelling perspective, the information used to parameterise these of these variables can take several forms. They can take the form of a series of functional relationships that define the variable, a pre-defined point estimate, a graphical function, or a function based on a pre-defined probability distribution.

## 6.4.1. Parameterising variable or uncertain parameters

Modelling population health outcomes comes with the challenge of parameters whose values inherently have a high-level of variability or include significant uncertainty. Deterministic approaches to model these variables would fail to capture both this variability (range of possible and/or reasonable values), and the uncertainty created by using expert opinion and non-local research data. For that reason, a stochastic modelling approach was used.

Variables in this model were parameterized with input functions that generate random values according to predefined distributions. These distributions are routinely used in risk assessment to capture variability amongst different member of a given population (expressed as a frequency distribution), random variables (expressed as a probability distribution), and uncertainty that exists around a parameter that is known to have a fixed value, but where little is known about its true value (Vose, 2000).

The following types of functions are used in this model to parameterize several auxiliary variables:

**Uniform distributions:** Uniform distribution functions assign equal probability to all values between a defined minimum and maximum value. Uniform distributions were used in cases where there is little to no available data for a variable, and plausible maximum, minimum or mean values are unknown. The probability density function of a uniform distribution is shown in equation (6.11).

$$f(x) = \frac{1}{max - min} \tag{6.11}$$

**Triangular distributions:** Triangular distribution functions assign a value from a continuous distribution with a specified lower bound, mode, and upper bound. Triangular distributions were used in cases where there is little to no available data for a particular variable, but where plausible maximum, minimum or mean values could be established. The probability density function of a triangular distribution is shown in equation (6.12).

$$f(x) = \begin{cases} \frac{2(x - min)}{(max - min)(mode - min)} & \text{for } min \le x \le mode \\ \frac{2(max - x)}{(max - min)(max - mode)} & \text{for } mode \le x \le max \end{cases}$$
(6.12)

**Log-Normal distributions:** The log-normal distribution was used as it is ideal for modelling naturally-occurring normally distributed variables that begin at 0 and extend to  $\infty$ . The probability density function for a log-Normal distribution is shown in equation (6.13).

$$f(x) = \frac{1}{x\sqrt{2\pi\sigma^2}} e^{-\frac{(\ln x - \mu)^2}{\sqrt{2\sigma^2}}}$$
(6.13)

**Beta distributions**: The Beta distribution function describes a probability distribution (bounded between 0 and 1) that can take several shapes, according to the equation (6.14).

$$f(x) = \frac{(x)^{\alpha - 1} (1 - x)^{\beta - 1}}{B(\alpha, \beta)}$$
(6.14)

where  $B(\alpha, \beta)$  is a Beta function.

While the BETA distribution is a probability distribution, and therefore bounded between 0 and 1, it can easily be rescaled to model variables that range from min to max using the following formula:

$$\mathbf{x} = \mathbf{Beta}(\boldsymbol{\alpha}, \boldsymbol{\beta}) \times (\mathbf{max} - \mathbf{min}) + \mathbf{min}$$
(6.15)

**PERT distribution**: In cases where auxiliary variables were parameterized based on expert opinion or literature-based evidence, a PERT distribution was used. PERT distributions, which follow the structure of BETA distributions, are one of the most commonly used distributions for quantifying expert opinion, particular as the permit non-normal and asymmetrical distribution (Kirk et al., 2014). The distribution is parameterised using a *minimum value* (pessimistic estimate), a *mode* (most likely estimate) and a *maximum value* (optimistic value). In the absence of these values, a PERT distribution can be specified using a median value and 95% confidence intervals.

The software used to create this model, Stella Architect, does not have a built-in function for PERT distributions. Therefore PERT distributions were converted to BETA distributions according to the classic methods described in Malcolm et al. (1959) and modified by Vose (2000) (modified PERT distribution), as shown in equations (6.16) to (6.19).

$$f(x) = \frac{(x - \min)^{\alpha - 1} (\max - x)^{\beta - 1}}{B(\alpha, \beta) (\max - \min)^{\alpha + \beta - 1}}$$
(6.16)

where,

$$\alpha = (k+2) \left( \frac{\mu - \min}{\max - \min} \right)$$
(6.17)

and,

$$\boldsymbol{\beta} = (\boldsymbol{k} + 2) \left( \frac{max - \mu}{max - min} \right)$$
(6.18)

with,

$$\mu = \frac{(\min + k(mode) + \max)}{(k+2)} \tag{6.19}$$

and

 $B(\alpha, \beta)$  is a Beta function

where,

min: minimum value (pessimistic estimate or lower value of 95% confidence intervals)mode: mode (or median)

max: maximum value (optimistic estimate or high value of 95% confidence interval)

k: scale parameter (set to a default value of 4 for PERT distributions)

μ: mean

6.4.2. Input Variables used in the Model

A list of variables and their model input functions is provided in Table 6.3. The mean, median, 5<sup>th</sup> percentile and 95<sup>th</sup> percentile values have been included in the table to demonstrate how each distribution reflects the variability and/or uncertainty within that parameter.

# Table 6.3: Value of model input variables

Variabla nama	ariable name Unit Distribution		Moon	Madian	5 <sup>th</sup>	95 <sup>th</sup> porcontilo
Population Sector	Unit	Distribution	Wican	Wiculan	percentine	percentile
Regional population	Persons	Empirical distribution				
Incubation period	Davs	$BFTA(3 25 2 75 1 12)^{a}$	7.00	7.04	3.98	9.93
Probably of being symptomatic given	Days Persons/	$BETA(2.16, 3.84, 0.5, 0.88)^{a}$	0.64	0.63	0.54	0.76
infection	Exposed	BETA(2.10, 5.04, 0.3, 0.06)	0.04	0.05	0.54	0.70
Duration of symptoms	Days	BETA(2.04, 3.96, 1, 28) <sup>a</sup>	11.31	10.93	3.45	20.51
Reinfection delay	Days	BETA(1.5, 4.5, 2, 10) <sup>a</sup>	4.02	3.79	2.30	6.56
Relapse rate	Persons/ Infected	BETA(1.68, 4.32, 0.18, 0.95) <sup>a</sup>	0.37	0.36	0.22	0.58
Relapse duration	Days	BETA(1.29, 4.71, 1, 15) <sup>a</sup>	4.02	3.60	1.29	8.35
Post-symptom infectious period	Days	BETA(2.69, 3.31, 1, 15) <sup>a</sup>	6.84	6.78	2.94	10.91
Secondary transmission sector						
Adult secondary transmission rate	Persons/ Infected	UNIFORM(0, 0.05) <sup>b</sup>	2.53E-02	2.49E-02	2.47E-03	4.79E-02
Child secondary transmission rate	Persons/ Infected	BETA(3.19, 2.81, 0, 0.31) <sup>a</sup>	1.63E-01	1.64E-01	6.73E-02	2.59E-01
Susceptible Contacted People (transit time)	Days	BETA(1.67, 4.33, 1, 43) <sup>a</sup>	12.27	11.16	3.09	25.02
Healthcare sector						
Health seeking fraction	Persons/ Symptomatic	BETA(3.25, 2.75, 0.137, 0.24) <sup>a</sup>	0.19	0.19	0.16	0.22
Treatment seeking delay	Days	BETA(1.31, 4.69, 1, 27) <sup>a</sup>	6.61	5.82	1.64	14.05
Fraction of people tested	Persons/ Treated	BETA(3.08, 2.92, 0.062, 0.283) <sup>a</sup>	0.18	0.18	0.11	0.24
Fraction of tests submitted	Persons/ Tested	BETA(4, 2, 0.91, 0.95) <sup>a</sup>	0.93	0.93	0.92	0.95
Faecal testing delay	Days	UNIFORM(1, 5) <sup>b</sup>	3.03	3.02	1.22	4.84
Microscopy sensitivity		BETA(3.26, 2.74, 0.33, 1) <sup>a</sup>	0.70	0.70	0.49	0.89
PCR sensitivity		BETA(3.76, 2.24, 0.8, 0.939) <sup>a</sup>	0.89	0.89	0.84	0.93
Awareness delay	Days					
[Under 5 Years Old]		BETA(1.66, 4.33, 1, 7) <sup>a</sup>	2.66	2.51	1.28	4.51
[Over 5 Years Old]		BETA(1.89, 4.11, 1, 10) <sup>a</sup>	1.32	1.29	1.07	1.65

					5 <sup>th</sup>	95 <sup>th</sup>
Variable name	Unit	Distribution	Mean	Median	percentile	percentile
Public Aquatic Facility Sector						
Daily Swimming frequency	Swims/Day	BETA(1.51, 4.49, 0.008, 0.8) <sup>a</sup>	0.21	0.18	0.04	0.45
Percent of patrons who shower LP	Persons/ Person	Point-estimate	0.15			
Percent of patrons who shower LP	Persons/ Person	Point-estimate	0.15			
Probability of AFR given infection	AFR/Swim					
[Under 5 years old]		BETA(1.44, 4.56, 0.005, 0.05) <sup>a</sup>	1.13E-02	9.87E-03	1.97E-03	2.53E-02
[Over 5 years old]		BETA(2.78, 3.22, 0.001, 0.01) <sup>a</sup>	5.22E-03	5.18E-03	2.46E-03	8.10E-03
Weight of faeces shed	Grams					
[Under 5 years old]		BETA(1.1, 4.9, 0.01, 5) <sup>a</sup>	0.88	0.70	0.08	2.34
[Over 5 years old]		UNIFORM(0.001, 0.1)	0.05	0.05	0.01	0.10
Weight of faeces in AFR	Grams					
[Under 5 years old]		TRIANGULAR(30, 50, 70)°	49.93	49.98	36.60	63.73
[Over 5 years old]		TRIANGULAR(100, 150, 200)°	1.49 E+02	1.49 E+02	1.16 E+02	1.84 E+02
Oocytes in 1 gram of stool	Oocytes	UNIFORM(50, 10 <sup>6</sup> ) <sup>b</sup>	4.93E+05	4.85E+05	5.32E+04	9.42E+05
Oocyte inactivation	Days	UNIFORM(2, 7) <sup>b</sup>	4.52	4.55	2.24	6.77
Pool waster ingested per swim	Litres					
[Under 5 Years Old, Large pool]		BETA(1.96, 4.04, 0, 0.154) <sup>a</sup>	5.14E-02	4.90E-02	1.35E-02	9.85E-02
[Under 5 Years Old, Small pool]		BETA(1.96, 4.04, 0, 0.154) <sup>a</sup>	5.02E-02	4.74E-02	1.36E-02	9.78E-02
[Over 5 Years Old, Large pool]		BETA(2.21, 3.79, 0, 0.053) <sup>a</sup>	1.96E-02	1.87E-02	6.49E-03	3.52E-02
[Over 5 Years Old, Small pool]		UNIFORM(0, 0.01) <sup>b</sup>	5.06E-03	5.12E-03	5.22E-04	9.51E-03
Percent of oocytes viable		BETA(2.94, 3.06, 61.1, 100) <sup>a</sup>	60.47	60.31	35.75	85.68
Dose response parameter		BETA(2.55,3.45, 0.005,0.066) <sup>a</sup>	3.09E-02	3.07E-02	1.28E-02	5.07E-02
Large Pool Volume	Litres					
[LP1, LP2, LP3,]		Point-estimate	3.75E+05			
[LP4, LP5, LP6,]		Point-estimate	4.87E+05			
[LP7, LP8, LP9, LP10]		Point-estimate	2.50E+06			
Small Pool Volume	Litres					
[SP1, SP2]		Point-estimate	2.05E+05			

					5 <sup>th</sup>	95 <sup>th</sup>
Variable name	Unit	Distribution	Mean	Median	percentile	percentile
[SP3, SP4]		Point-estimate	1.17E+05			
[SP5]		Point-estimate	6.96E+04			
Seasonal LP Users [Summer]	Persons/					
[LP1, LP2, LP3, LP4, LP5, LP6]	Person/Day	Point-estimate	0.066			
[LP/,LP8,LP9,LP10]		Point-estimate	0.15			
Seasonal LP Users [Winter]	Persons/	Doint actimate	0.1			
[LF2, LF3, LF4, LF3] $[IP7 IP8 IP0]$	Person/Day	Point estimate	0.1			
[LP] I.P6 I.P101		Point-estimate	0.2			
Seasonal SP Users [Summer]	Persons/					
[SP1, SP2]	Person/Day	Point-estimate	0.3			
[SP3]		Point-estimate	0.2			
[SP4, SP5]		Point-estimate	0.1			
Seasonal SP Users [Winter]	Persons/					
[ <i>SP1</i> ]	Person/Day	Point-estimate	0			
[ <i>SP2</i> ]		Point-estimate	0.5			
[SP3]		Point-estimate	0.3			
[SP4, SP3] Log 2 disinfection LD	Dava	POINt-estimate $PETA(2.47.2.52, 1.16, 2.62)$	0.1			
Log-2 disinfection SP	Days	DETA(2.071, 2.020, 0.58, 1.7)	1.//	1.70	1.55	2.24
Log-5 disinfection SP	Days	BE1A(3.0/1,2.929, 0.38, 1.7)	1.15	1.10	0.80	1
Imported Cases Sector	D					
Daily Departures	Days	Empirical distribution				
SEQ fraction						
[Under 5 Years Old, Metro North]		Point-estimate	0.19			
[Under 5 Years Old, Metro South]		Point-estimate	0.24			
[Under 5 Years Old, Gold Coast]		Point-estimate	0.11			
[Over 5 Years Old, Metro North]		Point-estimate	0.20			
[Over 5 Years Old, Metro South]		Point-estimate	0.22			
[Over 5 Years Old, Gold Coast]		Point-estimate	0.12			
Proportion of low risk travellers	persons	Empirical distribution				
Low-risk infection rate	Infections/	UNIFORM(0,0.009) <sup>b</sup>				
	Traveller		0.004	0.004	0.000	0.009

					5 <sup>th</sup>	95 <sup>th</sup>
Variable name	Unit	Distribution	Mean	Median	percentile	percentile
High-risk infection rate	Infections/ Traveller	UNIFORM(0, 0.014) <sup>b</sup>	0.007	0.007	0.001	0.013
Rate of Asymptomatic Travellers	Persons/ Exposed	BETA(2.16, 3.84, 0.5, 0.88) <sup>a</sup>	0.64	0.63	0.54	0.76
Length of travel	Days	UNIFORM(2, 30) <sup>b</sup>	15.85	15.67	3.40	28.68
Public Health Sector						
Notification delay	Days	LOGNORMAL(6.29, 4.65) <sup>d</sup>	6.36	5.10	1.67	15.06
Routine Messaging Effectiveness	Persons/ Person/Day	Point-estimate	0.05			
Routine Messaging Effectiveness Decay	Days	Point-estimate	15			
Healthcare Messaging Effectiveness	Persons/ Person/Day	Point-estimate	0.05			
Healthcare Messaging Effectiveness Decay	Days	Point-estimate	15			

<sup>a</sup> Beta ( $\alpha$ ,  $\beta$ , min, max) <sup>b</sup> UNIFORM(min, max)

<sup>c</sup> TRIANGULAR(min, mode, max) <sup>d</sup> LOGNORMAL(mean, standard deviation)

# 6.4.3. Framework for model parameters

Using a modified version of the framework presented by David Lane (Lane, 2014), each parameter was categorised into one of six categories using the 3X2 matrix shown in Table 6.4. The value of a parameter could either be locally-known, broadly-known, or uncertain. Locally-known parameters are parameters relevant to the local context and are known or available. Broadly-known parameters are parameters where locally-relevant knowledge or information is not available, but sufficient information from other contexts, or a broader context, exist to be used in its place. Uncertain parameters are parameters where there is little to no information available about its value.

Variable were then further characterised by whether they are fixed or adjustable. Parameters with fixed values are either a fixed attribute of nature (such as climate) or are outside the scope or interest of the intended users of this model (exogenous control).

	Fixed Value	Adjustable Value
Locally-known value	Fixed, Locally-known	Adjustable, Locally-known
Broadly-known value	Fixed, Broadly-known	Adjustable, Broadly-known
Uncertain value	Fixed, Uncertain	Adjustable, Uncertain

Table 6.4: 3 x 2 matrix used to classify model parameters

This framework was used for several reasons. Characterising parameters based on whether they can be adjusted allows for the identification of variables that, in principle, can be altered to modify system behaviour. These are the first variables to be tested using sensitivity analysis to determine whether they can be leverage points of action. It is important to explicitly recognise what is and isn't within the potential realm of action. Sensitivity analysis conducted on the variables within the 'adjustable value' category is discussed in detail in Chapter 7.

Characterising the variables by the degree to which their value is known is important to identify future points of inquiry or research. Models are never complete, and continuously evolve as our understanding of the system grows and improves. Identifying uncertain variables helps chart an agenda for future research and inquiry into the problem. Future actions to improve our understanding of the problem, and subsequently the strength of the model, should be targeted at moving parameters towards the top of the chart.

# 6.4.3.1. Categorized model parameters

The table below applies the parameter characterization matrix to the parameters within each sector model. Parameters have been colour-coded based off of the sector from which they originate.

	Fixed Value	Adjustable Value
	Testing transition	Notification delay
Locally-known	Daily Departures	
value	SEQ fraction	
	Proportion of low risk travellers	
	Incubation period	Fraction of people tested
	Probability of being symptomatic	
	Re-infection delay	
	Relapse rate	
	Relapse duration	
	Post-symptomatic infectious period	
	Duration of symptoms	
	Health-seeking fraction	
	Fraction of test submitted	
	Microscopy sensitivity	
	PCR sensitivity	
Broadly-known value	Adult secondary transmission rate <sup>1</sup>	
value	Child secondary transmission rate <sup>1</sup>	
	Secondary transmission delay	
	Daily Swimming Frequency	
	Portion of population who swim	
	Weight of faeces shed by children <sup>1</sup>	
	Weight of faeces shed by adults <sup>1</sup>	
	Oocytes per gram of stool	
	Oocyte die-off	
	Pool water ingested per swim	
	Percent of oocytes viable <sup>1</sup>	
	Dose-response parameter <sup>1</sup>	

 Table 6.5: Characterization of parameters used in each sector of the model

	Fixed Value	Adjustable Value
	Length of immunity	Fraction of infectious people made
	Treatment-seeking delay	aware
	Faecal testing delay	Physician precautionary advice fraction
	Awareness delay	Routine messaging start date
	Length of travel	Routine messaging effectiveness
	High risk infection rate	Healthcare messaging start date
Uncertain	Low risk infection rate	Healthcare messaging effectiveness
value	Rate of asymptomatic travellers	PAF messaging cycle period
	Routine messaging decay	PAF messaging effectiveness
	Healthcare messaging effectiveness	Percent of PAF users who shower
	decay	Routine shock frequency
	PAF messaging effectiveness decay	AFR detection rate
	Seasonal converter	
	Probability of AFR given infection	
	Seasonal LP users	

<sup>1</sup> Information related to this variable is highly variable, context specific, or quite limited. Further context-specific inquiry is highly warranted.

# 6.5. Model Validation

Once system dynamics models have been constructed, but prior to them being used for their intended purpose (i.e. policy analysis), it is necessary for them to undergo "validation". While real "validation" of models is a fallacy, as models simply represent our understanding of a problem and are therefore inherently flawed (Sterman, 2000b), a number of tests can be used to assess if a model is fit-for purpose.

As mentioned at the beginning of this chapter, the purpose of this model is to:

'provide contextual insight on system structures and dynamics behaviours, especially those related to public aquatic facilities, that are contributing to the overall trend of cryptosporidiosis notification in South East Queensland communities.'

With that purpose in mind, the validation tests used sought to establish whether the model presented sufficient contextual validity, structural validity, and behavioural validity, to be fit-for-purpose.

# 6.5.1. Boundary Adequacy

The boundary adequacy test ensures that the model's boundaries fit the purpose of the model. Ensuring the model's boundaries are appropriately scaled to match the purpose of the model is necessary to build confidence in the model's outputs (Sterman, 2000a). The primary method I used to ensure boundary adequacy was continual referral back to the causal loop diagrams constructed in the first portion of this study. The causal loop diagrams (CLD) provided a basis for defining the desired scope of the model. Actively including the intended users of the model in the construction of the CLDs provided additional confidence that the boundaries of the CLDs reflected the desired purpose and scope of the simulation model. All variables identified during the modelling workshops and interviews that fit within the boundary of the problem (as defined by the CLDs) were considered for inclusion in the final model. Additionally, a substantial review of the literature was then conducted (as described in Chapter 2) to ensure that any potentially important variables within the boundary of the model were not omitted. The final model was then compared with the original CLDs to ensure that important concepts that the participants deemed necessary to address the problem were endogenous to the model.

#### 6.5.2. Dimensional Consistency

The test for dimensional consistency involves comparing the units of each variable within the model to ensure no inconsistencies exist. The dimensional analysis tool built in to the Stella Architect program was used to ensure all equations within the model had logical and consistent units. The results of the dimensional consistency test indicated that there were no inconsistent units within the model. Additionally, the model was built in such a way that dimensional consistency could be maintained without the use of arbitrary scaling factors or dummy variables.

#### 6.5.3. Structural Assessment

Structural assessment tests ensure that the structure and behaviour of the model is consistent with current knowledge of the real system. This includes ensuring that the model respects know physical and natural laws, decision rules, and does not include inappropriate assumptions.

The following two methods were used to evaluating the structure of the model:

1. As all of the elements represented by stocks in the model are elements that cannot naturally have negative values (such as number of infectious people, or *Cryptosporidium* oocytes in a swimming pool), the values of all stocks were reviewed to ensure they did not produce negative values in the model. This was also accomplished by ensuring that all stocks in the model had a first order negative feedback loop restricting the outflow of the stock. This results in the outflow of the stock approaching zero as the value of the stock approaches zero.

2. The final simulation model was compared to the CLDs from the first portion of the study to ensure the model's structure reflected the relationships between key variables identified by local experts. This also involved ensuring that the polarity of those relationships was retained in the simulation model.

# 6.5.4. Extreme Conditions test

The extreme conditions test attempts to ensure the model is capable of producing rational and realistic behaviour, even when variables within the system take on extremely high or low values (Sterman, 2000a). If the model can provide realistic outputs under these extreme conditions, it is an indication that the model's structure likely reflects reality.

For this test, several variables from different sectors were modified to simulate extreme conditions. Extremely low conditions were simulated by giving each of the test variables a value of 0. Extremely high conditions were simulated by giving each test value their maximum value within their distribution. The effect of the variables taking their extreme values was evaluated using their impact on the mean daily number of notified cases, total infectious people, oocytes in the large pool, and oocytes in the small pool.

The results of the extreme conditions test can be seen in Table 6.6.

Across all variables, the model produced results that make sense in the context of the modified variable. For example, when the health care seeking fraction was set to 0, one would expect the number of infectious people to increase because less people receive medical treatment or advice, leading to an increase in the transmission of the disease and more infected people. The model predicted an increase in the number of infectious people when the healthcare seeking fraction was set to 0, so the model behaved as expected.

		Notified cases <sup>a</sup>	Total infectious people <sup>a</sup>	Oocytes in large pool <sup>a</sup>	oocytes in small pool <sup>a</sup>
Base C	Case	2.84	330.30	105,960	298,680
Second	dary Transmission <sup>1</sup>	0			
Low	0%	2.26	263.39	81,880	221,850
High	Adult: 5% Child: 31%	5.89	685.19	264,570	727,900
Overse	eas-acquired infect	ion rate <sup>c</sup>			
Low	0%	0.00	0.31	30	120
High	High risk: 0.9% Low risk: 1.5%	5.69	667.00	270,350	600,940
Proba	bility of being asyn	nptomatic <sup>d</sup>			
Low	0%	5.35	556.67	192,550	636,250
High	50%	2.47	298.96	94,680	263,020
Health	care-seeking fracti	ion			
Low	0%	0.00	944.42	396,250	1,026,720
High	24%	3.30	309.24	92,940	263,770
Daily s	swim frequency	1			
Low	0	1.59	188.45	0	0
High	80%	621.64	74,016.05	43,838,180	46,640,330

Table 6.6: Mean daily values for extreme conditions test

<sup>*a*</sup> mean daily value

<sup>b</sup> values for the adult secondary transmission rate and the child secondary transmission rate were both modified as part of the extreme means test

<sup>c</sup> values for low risk infection rate and the high-risk infection rate were both modified as part of the extreme means test

<sup>d</sup> the value if 1 minus the probability of being symptomatic was used

# 6.5.5. Period Comparison

Period comparison tests aim to ensure that the model is able to replicate the location and duration of any cyclical components of the natural behaviour being modelled. As the main variable of interest in this problem's reference model (number of cryptosporidiosis notifications) appears to exhibit repetitive oscillating behaviour, assessing whether the model can reproduce the period of the reference mode's oscillation is an important part of model validation. As part of his multi-step model validation procedure, Barlas (1989) recommends using the autocorrelation function for period comparison. Autocorrelation is a statistical measure of affiliation between each data point in a series and future values within the same series, after a given time interval. Autocorrelation has the advantage of being able to filter out cyclical components of time-series data, even in the presence of significant noise. Autocorrelation assigns a value of +1 to strong positive associations between variables, -1 to strong negative associations, and 0 to no association.



**Figure 6.28:** Period comparison of correlograms of actual and predicted (modelled) weekly cryptosporidiosis notifications in South East Queensland (95% confidence bands shown in grey)

Figure 6.28 shows correlograms of the result of the autocorrelation period comparison of the actual and predicted (modelled) weekly count of cryptosporidiosis notifications in SEQ. The actual data shows a statistically significant positive autocorrelation for lags 1 to 15 and 41 to 59 (weeks), whereas the predicted data shows statistically significant positive autocorrelation for lags 1 to 15 and 38 to 59. Both areas of significant autocorrelation have a similar amplitude. While minor differences do exist in the amplitude of the autocorrelation functions between the two data series, these differences occur at lags not considered significant. The results of both correlograms point to cryptosporidiosis notifications having a strong annual cycle, but do not support the hypothesis that there are statistically significant 2-, 3-, or 4-year cycles in cryptosporidiosis notifications in SEQ.

The high-level of similarity in the periods of the autocorrelation functions for the actual and predicted data validates the model's ability to replicate the oscillation of the reference mode.

#### 6.5.6. Trend Analysis, Discrepancy Coefficient and Family Member Test

The final step of model verification was trend analysis and discrepancy coefficient. The discrepancy coefficient was an index proposed by Barlas (1989) to summarize the overall fit of system dynamics simulation models to historical data. The discrepancy coefficient is measured between 0 (perfect predictions) and 1 (worst predictions). Values less than 0.6 are considered to have at least 'good' fit.

The equation for Barlas' discrepancy coefficient (U) is:

$$U = \frac{\sqrt{\sum(S_i - \bar{S} - A_i + \bar{A})^2}}{\sqrt{\sum(A_i - \bar{A})^2} + \sqrt{\sum(S_i - \bar{S})^2}}$$

Where:

A = historical (actual) data (i.e.  $A_1, A_2...A_i$ )

 $S = simulated (predicted) data (i.e. S_1, S_2...S_i)$ 

#### 6.5.6.1. South East Queensland

Figure 6.29 below shows the results of the trend comparison between the historical daily count of cryptosporidiosis notifications in South East Queensland, and the results of the base case of the simulation model. Both the historical and simutated trends show a similar cyclical pattern of outbreaks throughout the study period. Barlas' discrepancy coefficient for the base case of daily cryptosporidiosis notification is 0.36, which indicates that it is a good-fitting model. While there are minor variations in the amplitude of the peaks within the simulated trend, the overall shape and temporal distribution of the peaks is quite similar to that of the historical data.



Figure 6.29: Trend Analysis - Daily Cryptosporidiosis notifications in South East Queensland (initial base case)

The base case of the simulation model does indicate a systemic displacement of the silmuated curve, with the model overestimating the daily notification count by approximately 0.71 persons/day. This may be due to an overestimation of the healthcare-seeking fraction in the region. This would happen because a higher than average healthcare-seeking fraction would result in the detection and reporting or more cases of cryptosporidiosis cases compared to what would otherwise be detected and reported. The value of the healthcare seeking fraction variable used in base-case, which has a BetaPERT distribution with a minimum value of 13.7%, a mean value of 19.5% and a maximum value of 24%, is based on non-Australian values reported in the literature.



Figure 6.30: Trend Analysis - Daily Cryptosporidiosis notifications in South East Queensland (updated base case with 5% reduction in healthcare seeking fraction)

The systemic displacement of the curve is virtually elimiated when there is a 5% decrease in the healthcare seeking rate, as shown in Figure 6.30. Barlas' discrepancy coefficient for the revised base case is 0.31, which indicates better model fit. As no locally-relevant data about the healthcare seeking rate of SEQ residents is available, and the revised mean still fits within the range of values reported in the literature, the final base case of the model was modified to assume that the revised value of the healthcare seeking fraction variable.

#### 6.5.6.2. Public Health Unit Regions

The overall trends seen in South East Queensland can be examined in greater detail by looking at the model outputs for each of the Public Health Unit (PHU) regions.

Figure 6.31 below compares the historical and simulated trends of cryptosporidiosis notifications in Metro North PHU. Barlas' discrepancy coefficient for the Metro North PHU simulation is 0.39, which indicate good model fit.



Figure 6.31: Trend Analysis - Daily Cryptosporidiosis notifications in Metro North PHU (final base case)

Figure 6.32 below compares the historical trend of daily cryptosporidiosis notifications in Metro South PHU with the simulated trend. Barlas' discrepancy coefficient for the Metro South PHU simulation is 0.35, which also indicates good model fit.



Figure 6.32: Trend Analysis - Daily Cryptosporidiosis notifications in Metro South PHU (final base case)

Lastly, Figure 6.33 below compares the historical trend of daily cryptosporidiosis notifications in the Gold Coast PHU region with the model's simulated trend. Barlas' discrepancy coefficient for the Gold Coast PHU simulation is 0.35 which indicates good model fit.



Figure 6.33: Trend Analysis - Daily Cryptosporidiosis notifications in Gold Coast PHU (final base case)

In view of the results of the trend analysis and the results of the discrepancy coefficient, both for SEQ as a whole and for each of the PHU regions, it is reasonable to conclude that the base trend produced by the simulation model is a good fit to available historical data.

# Chapter 7. Risk-management strategies for Cryptosporidium transmission in South East Queensland

The aim of this chapter is to identify and explore realistic policy strategies to curb the transmission of *Cryptosporidium* in South East Queensland (SEQ) communities. The first part of the chapter begins with an overview of sensitivity analysis in the context of system dynamics models and outlines the results of the analysis conducted on the *Cryptosporidium* in South East Queensland model. presents and discusses the results of the simulation of different policy strategies to reduce *Cryptosporidium* transmission in SEQ. The final part of the chapter explores the strategies in terms of their ability to harness potential areas of leverage within the system

# 7.1. Sensitivity Analysis

The purpose of sensitivity analysis is to test the robustness of the outputs of an SD model to uncertainty or variability in the data or assumptions upon which the model was built. In other words, sensitivity analysis allows you to assess if changes in model assumptions produces changes in either the numerical values of model outputs, or the behavioural patterns the model produces (Sterman, 2000a). Sensitivity analysis also allows for the identification of key leverage points, where small changes in system input produce large changes in system output.

As nearly all the variables in this model are characterised by a high degree of natural variability and uncertainty, their value has been parameterised with input functions that generate random values according to predefined distributions (as described in section 6.4.1). Therefore, the sensitivity analysis of this model will focus on testing its sensitivity in terms of the changes in system behaviour generated by changes in the underlying assumptions.

While there exist several different approaches to perform sensitivity analysis of system dynamics models, a modified version of the following four step process, as proposed by Maani and Cavana (Maani and Cavana, 2007) was used:

"a) Select those parameters or groups of parameters that are considered most likely to affect the behaviour of the model, or whose estimation was based on more imprecise or uncertain information than that of other parameters.

*b) Modify the value of each separate group of parameters by a given percentage (say 10%) at a time, and conduct the corresponding simulation runs.* 

*c) Identify those parameters that, when changed, significantly affect the model behaviour.* 

*d)* Analyse and interpret whether the behavioural changes are justified using existing knowledge or common sense."

In this study, each of the chosen input parameters were individually increased and decreased by 10% (except for 'physician precautionary advice fraction' and 'routine messaging effectiveness' which were only adjusted by  $\pm$  5% as their original value was only 5% prior to adjustment) from their original value, while holding all other values in the model constant at their base-case value. The daily count of cryptosporidiosis notifications in SEQ was used as the dependant variable because disease notifications are the primary unit of measure of disease burden. In addition, the sensitivity of several proposed interventions that modify the current structure and behaviour of the system was tested. These interventions, while not part of the current system, represent several potential system additions proposed by stakeholders during the consultation period.

As the modelled system produces recurring overshoot and collapse behaviour, the standard sensitivity analysis approach of comparing the final values of the dependent variable under different scenarios could not be used (Hekimoglu and Barlas, 2010). An alternative approach, using four different behaviour pattern measures, was used to estimate the relative influence of various system parameters on the dependant variable (case notification). As one of the key features of the system's behaviour is a cycle between outbreak and non-outbreak periods, the analysis focused on assessed behaviour of the dependant variable in the context of major disease outbreaks. For the purpose of this sensitivity analysis, 'major outbreaks' were triggered when at any point in

time where there was a rapid increase ( $\geq 3\%^{11}$ ) in the weekly *trend*<sup>12</sup> of disease notifications.

<sup>&</sup>lt;sup>11</sup> There is no established 'outbreak trigger' for cryptosporidiosis surveillance in Queensland. Values of 1%, 2%, 3% and 4% were tested using the model and visually assessed to establish which value correctly identified the beginning of the major peaks in disease notifications, without also flagging minor fluctuations in the trend. A value of 3% was chosen as the value most closely matching this requirement under the 'base case' scenario.

<sup>&</sup>lt;sup>12</sup> The weekly trend is based on the first order exponential average of the input daily cryptosporidiosis notifications, and a 7 day exponential averaging time. It is expressed as the fractional change in input per unit time.



**Figure 7.1:** Behaviour pattern measures used for the sensitivity analysis The four measures used in the sensitivity analysis that are displayed in Figure 7.1, are:

- Days above the non-outbreak trend: The number of days where the count of disease notifications was above the daily non-outbreak trend (Fig. 7.1) was used as a measure of the influence each parameter on the overall number of cryptosporidiosis notifications per day and not just during outbreaks. As the daily notification count for days not identified as being part of a 'major outbreak' were observed to follow a slight upward trend, the data were fitted using an exponential trend line<sup>13</sup>. The percent difference in the mean number of days above the non-outbreak trend line was used to compare the relative influence of each parameter.
- **Outbreak duration**: The period (i.e. duration) of a major outbreak is defined as the amount of time between the days the outbreak starts and ends. The start date of a 'major outbreak' was defined as the first day before the 'outbreak trigger' where the *trend* of disease notifications became positive in a sustained<sup>14</sup> manner. The end date of a 'major outbreak' was defined as the last day after the 'outbreak trigger' where the *trend* of disease notifications is negative in a sustained manner. The percent difference in the mean outbreak period was used to compare the relative influence of each parameter.
- **Outbreak Intensity**: The intensity (i.e. amplitude) of the 'major outbreaks' was defined as the difference between the number of disease notifications on the start date of the outbreak

<sup>&</sup>lt;sup>13</sup> y =  $1.0671e^{0.0003x}$ , R<sup>2</sup> = 0.55

<sup>&</sup>lt;sup>14</sup> Defined as  $\geq$ 7 days.

period, and the date within the outbreak period with the greatest number of disease notifications. The percent difference in the mean outbreak amplitude was used to compare the relative influence of each parameter.

• **Outbreak frequency**: The outbreak frequency was defined as the number of 'major outbreaks' experienced during the study period (2007-2017). The absolute difference in the number of outbreaks was used to compare the relative influence of each parameter.

# 7.1.1. The Base Case

Prior to running any simulation experiments using the model, a 'base case' scenario is run to simulate a 'business as usual' situation. The primary aim of the base case is to serve as a baseline for sensitivity analysis, and a benchmark against which alternative scenarios are compared. A secondary aim of the base case is to provide insights into parts of the system that may not be easily observed in the real world, or for which there is data or information available

Table 7.1 below outlines the values for key parameters that were used in the 'base case' scenario. These parameters represent the variables within the model that are considered potentially modifiable by decision- and policy-makers in the public health sector. The remaining model variables, while critical to the overall dynamics of the system, are not considered within the control of decision- and policy-makers in the public health sector. These variables were therefore held constant at the values described in Table 6.3.

Variable	Value in 'base case'
Physician precautionary advice fraction	5%
Percent of patrons who shower	15%
Fraction of positive cases made aware	43%
Routine Messaging effectiveness	5%
AFR detection and proper management rate	30%
PAF messaging effectiveness	0%
Percent of symptomatic travellers screened out	0%
Healthcare Messaging effectiveness	switched off
Routine hyperchlorination frequency	switched off
3-log secondary disinfection system	switched off

**Table 7.1:** Parameter values used in the base case scenario

# 7.1.1.1. Base Case Results

Table 7.2 shows the results of the model for the variable '**notified crypto cases**' when run using the base case scenario. The model identifies 8 unique outbreaks over the course of the study period. The behaviour '**notified crypto cases**' variable over the study period when run at the base case can be seen in Chapter 6 in Figure 6.29.

Table 7.2: Results of the sensitivity analysis measure under	r the	'base-case'	scenario	for the
cryptosporidiosis in Queensland system dynamic model.				

	Measure	<b>Base Case</b>
	mean	8.44
Peak value (daily notifications)	median	8.60
	max	20.37
	mean	6.58
Amplitude (daily notifications)	median	6.94
	mean	157.63
Total period (days)	median	159.00
	count	2024.00
Days above non-outbreak mean	%	55.45%
Number of outbreaks	count	8.00

The number of daily new infectious cases, by source of exposure under the 'base case' scenario is shown in Figure 7.2. Over the course of the 10-year study period, the model estimates that overseas acquired cases are responsible for 58.7% of total infectious individuals in the community, whereas PAFs and secondary transmission are responsible for 30.7% and 10.6% respectively.

The model demonstrates that overseas-acquired cases provide the region with a low but steady source of cases. This is in stark contrast to the current estimation of the fraction of overseas acquired cases of 1% to 8% reported in the National Notifiable Diseases Surveillance System (Kirk et al., 2014), and 5% to 20% reported in other countries (Havelaar et al., 2008, Adak et al., 2002, Scallan et al., 2011). This discrepancy is most likely due to the reliance on data generated from the investigation of outbreaks associated with public aquatic facilities. While the model demonstrates that overseas-acquired cases are implicated in seeding many of these outbreaks, they are not typically involved in the resulting spike of cases associated with the outbreak itself. They would, therefore, likely make up only a small percentage of individuals identified during outbreak investigations.



Figure 7.2: Daily new infectious cases in South East Queensland, by source of exposure.

Using the NHMRC *Guidelines for Managing Risks in Recreational Water*, the number of days over the study period (per PHU region) where the risk of infection exceeded 1% of swimming events<sup>15</sup>, was calculated. The number was calculated when looking at users of small and large pools separately, as well as pool users as a whole. Over the entire study period (3650 days) Metro North, Metro South and Gold Coast had 197, 215.5, and 51.5 days where the water quality of their small pools fell below the Level A criteria (Table 7.3). Overall, this represented less than 6% of days during the study period. Water quality in large pools only fell below these criteria for 1.5 days and 0.5 days in Metro North and South respectively.

<sup>&</sup>lt;sup>15</sup> The NHMRC Guidelines for Managing Risks in Recreational Water define waterbodies with a Category A (very good) ranking as those that have an estimated average probability of gastrointestinal illness of less than 1 case in every 100 exposures.

Deel		Level A (<1% risk of GI Illness)		Level A (<1% risk of Level B (1-5% Level C (5 GI Illness) risk of GI risk of Illness) Illnes		C (5-10% Level D (> < of GI risk of ( ness) Illness		D (>10% of GI ness)	
type	Region	Count	(%)	Count	(%)	Count	(%)	Count	(%)
	Metro North	3,453.5	(94.62%)	184.5	(5.05%)	9.5	(0.26%)	2.5	(0.07%)
Small pools	Metro South	3,434.5	(94.10%)	203.5	(5.58%)	10	(0.27%)	2.0	(0.05%)
	Gold Coast	3,598.5	(98.59%)	49.0	(1.34%)	2.5	(0.07%)	0	(0%)
	Metro North	3,649.5	(99.99%)	0.5	(0.01%)	0	(0%)	0	(0%)
Large pools	Metro South	3,648.5	(99.96%)	1.5	(0.04%)	0	(0%)	0	(0%)
	Gold Coast	3,650.0	(100.00%)	0	(0%)	0	(0%)	0	(0%)

**Table 7.3:** Simulated cumulative count of days spent in each of the four NHMRC recreational water quality microbial risk criteria categories, for large and small pools under 'base case' conditions, by PHU region

Alternatively, if the WHO reported tolerable risk threshold for drinking water of 1 infection per 10,000 population<sup>16</sup> per year is used, the threshold is exceeded in all but one year in Metro North and Metro South, and six out of 10 years in the Gold Coast, (Table 7.4)

**Table 7.4:** Simulated water-related infections per 10,000 population per year under 'base case' conditions, by PHU region

Year	Metro North	Metro South	Gold Coast
2007-2008	0.7	0.8	0.4
2008-2009	18.2	21.4	6.8
2009-2010	1.6	2.0	0.5
2010-2011	1.7	4.1	1.3
2011-2012	15.6	16.1	8.3
2012-2013	2.5	6.5	0.9
2013-2014	2.1	2.3	0.6
2014-2015	11.7	12.1	7.6
2015-2016	28.5	28.9	5.3
2016-2017	24.0	24.1	2.5

<sup>&</sup>lt;sup>16</sup> As reported in Hunter and Fewtrell (2001)

Under the base case scenario, the model produces a mean ratio of notified cases to actual cases<sup>17</sup> of 1 to 8.4 (s.d.: 1.78), which is consistent with the mean ratio of 1 to 7.4 (s.d.: 2.38). reported Kirk et al. (2014).

## 7.1.2. Results of sensitivity analysis

The results of the sensitivity analysis are shown in Figure 7.3. Across all four sensitivity measures decreasing the physician precautionary advice fraction from its base case value of 5% to 0% results in the greatest increase in the number of notified cases. Increasing the fraction to 10% also resulted the greatest decrease in the overall number of days above the non-outbreak mean, and the mean outbreak intensity.

This may suggest that even at low levels (5%), physicians providing patients who they suspect are infected with *Cryptosporidium* (but have not confirmed) with advice to avoid risky contact behaviour and avoid swimming has a noteworthy dampening effect on the number of cases that result from outbreaks, making it a high-leverage variable. This is not surprising because providing patients suspected of having *Cryptosporidium* with information at their first point of contact with the healthcare system, as opposed to waiting for a confirmed diagnosis, greatly reduces the time delay between onset of symptoms and the patient becoming aware that they have the disease. This in turn reduces the number of days of potential infectious contact per infectious cases, which decreases the amplification potential of each outbreak.

Similar, yet much less pronounced effects were seen when the fraction of people tested, and the fraction of positive cases made aware were adjusted. The logic remains the same that the more infectious people who are away of their transmission risk, the fewer infectious people in the community who are spreading the disease. The smaller effect relative to that found with advice provided to pre-confirmed cases is likely due to the time delay required for testing and being notified of the result. These variables are likely of low-to-moderate leverage value.

A pronounced protective effect was also noted across all four sensitivity measures when the AFR detection and proper management rate was increased by 10% in relation to small pools only. This is not unexpected as small pools have an increased density of patrons who are both at higher risk of fouling the pool (i.e. young children) and consuming pool water, and the pools have a lower water

<sup>&</sup>lt;sup>17</sup> Ratio is calculated using the inflow of new cases at time -11 days to account for the average time delay between exposure and testing positive.

volume, which increases the concentration of oocytes/litre following an AFR. Conversely, large pools have high water volumes and older patrons who are less likely to swallow less water (i.e. adults). Furthermore, reducing the number of AFRs that are unmanaged is of greater importance in small pools than large pools. As a result, the AFR detection and proper management rate in small and large pools could be considered of high and moderate leverage values respectively.

The percent of patrons who shower had minimal effect on measures directly related to outbreaks but did have a notable impact on the days above the non-outbreak mean. As showering only decreased the pathogen-load related to bather shedding and not AFRs, this implies that bather shedding does have an impact on the overall number of cases of cryptosporidiosis in the community, and to a lesser degree on the risk of an outbreak.

The effectiveness of routine public messaging provided by the public health units had little-to-no effect on any of the sensitivity measures. All the proposed system interventions, with the exception of PAF messaging (i.e. messaging put out by the operators of swimming pools), had a moderate or significant negative impact on case notifications. Much like the public messaging provided by the public health units, PAF messaging had little effect on the dependant variable across all four sensitivity measures. Conversely, the effectiveness of healthcare messaging (i.e. messaging directed at medical practitioners) did have a moderate effect on the mean intensity of outbreaks and the overall number of days above the non-outbreak mean. This is likely due to the impact healthcare messaging has on the high-leverage physician precautionary advice fraction variable.

The intervention with the greatest independent effect on case notifications was the introduction of 3-log disinfection systems (e.g. UV or ozone disinfection) to all small pools



Figure 7.3: Results of sensitivity analysis on cryptosporidiosis notifications in South East Queensland

The likely mechanism of this is similar to improving the AFR detection and proper management rate, i.e. minimising the presence of infectious oocysts from pool water. The use of 3-log disinfection systems for only high-risk small pools<sup>18</sup> or large pools. This is likely to be attributable to the subtle differences that each type of facility plays in the overall dynamics of the outbreaks. Small pools (figure 7.4) tend to be the source of both the outbreak itself and the driver of the duration of the outbreak through repeated spread and re-contamination. Conversely, while outbreaks are unlikely to be initiated in large pools they tend to be associated with amplification of the outbreak and the high total number of cases.



**Figure 7.4:** Predicted effect of installing 3-log secondary disinfection systems in only high-risk swimming pool or all large pools on new cryptosporidiosis cases attributed to all small and large swimming pools in SEQ

While the installation of 3-log disinfection systems in both large and small pools demonstrated a significant improvement in the number of cryptosporidiosis cases in the community, the cost of

<sup>&</sup>lt;sup>18</sup> For the purpose of this analysis, 'high-risk' small pools defined as pools with a total volume  $\leq 100,000$  litres. These pools are considerably smaller than other pools used for similar purposes.

these systems and the level of sophistication required to properly manage these systems means that these scenarios are unlikely to be feasible in SEQ at this time. For that reason, only the use of 3-log disinfection systems in high-risk small pools has been carried forward to the policy analysis stage.

Much like the AFR detection and proper management rate, routine hyperchlorination had a marked effect on reducing case notification when applied to both large and small pools, thought the effect was more pronounced when applied to small pools. The degree of effect was highly dependent on the frequency of hyperchlorination, with little-to-no effect when done quarterly, moderate effect when done monthly, and a large effect when done fortnightly. While fortnightly hyperchlorination appears to lead to significant improvement in the number of cryptosporidiosis cases in the community, the impact of this activity and the associated cost is unlikely to be feasible in all large pools. For that reason, fortnightly hyperchlorination of large pools has not been carried forward to the policy analysis stage.

## 7.1.3. Combined contribution of model sectors

In addition to examining the sensitivity of case notifications to changes in individual system components, I also examined the sensitivity of the case notification variable to changes in the output of the three sectors that contribute new *Cryptosporidium* infections to the population (i.e. secondary transmission sector, imported cases sector, and PAF sector). The purpose of this was to compare the relative contribution of different forms of transmission to the overall disease dynamics in SEQ.



Figure 7.5: The sensitivity of daily number of cryptosporidiosis notification to changes in the daily number of new secondary infections
Figure 7.5 shows the change in the number of notifications of cryptosporidiosis to changes in the number of new secondary infections. Reducing the number of secondary infections, even to the point of complete elimination, has little effect on the frequency or duration outbreaks, and only a minor effect on the intensity of durations. There is also no noticeable tipping point with this variable.



Figure 7.6 Sensitivity of daily number of cryptosporidiosis notification to changes in the daily number of new overseas-acquired infections (OAIs) entering the community.

Similarly, notable changes in outbreak intensity and frequency are evident when the number of overseas acquired infections (OIA) is adjusted (shown in Figure 7.6). Interestingly, almost no effect is seen on outbreak duration. This could indicate that OAIs play a significant role in starting outbreaks by seeding the community with infectious cases but play a lesser role in sustaining them. A striking threshold effect is seen when OIAs are reduced by 60%. At this level of reduction, the model predicts that all but one of the outbreaks would have been avoided. As well, a complete elimination of OAIs results in a near elimination of cryptosporidiosis cases in SEQ. This indicates that overseas travel is likely responsible for the endemic levels of cryptosporidiosis within the region.

In contrast, the adjusting the number of cases originating from the PAF sector (shown in Figure 7.7) has a noticeable effect on both the intensity, frequency and magnitude of outbreaks. A 60% reduction in PAF-related cases causes the intensity of all outbreaks to be greatly diminished, and an 80% reduction virtually eliminates all outbreaks. It is worth noting that while the model predicts that eliminating PAF-related transmission of *Cryptosporidium* can eliminate the outbreaks

experienced in SEQ, it does not eliminate cases all-together because of the presence of secondary transmission and overseas-acquired infections that maintain an endemic pattern of cryptosporidiosis cases





The results of the sensitivity analysis will be used to inform the policy analysis below.

#### 7.2. Policy Analysis

#### 7.2.1. Scenario development

Based on the results of the sensitivity analysis, along with feedback provided by workshop and interview participants, the following eight scenarios were developed to simulate the effect of a combination of different interventions on the outbreak dynamics of cryptosporidiosis in SEQ. The following variables were found to be moderately or highly influential variables during the sensitivity analysis and were therefore carried over to the policy analysis stage:

Physician precautionary advice fraction	Percent of symptomatic travellers screened out	Percent of patrons who shower
Fraction of positive cases made aware	3-log secondary disinfection system	AFR detection and proper management rate
Routine hyperchlorination frequency	Routine hyperchlorination frequency	

Model parameters used for each scenario are provided in Table 7.5.

#### 7.2.1.1. Scenario 1 – International traveller intervention

This scenario represents an intervention targeted at screening incoming international travellers to identify individuals who may have been exposed to *Cryptosporidium* while overseas and providing suspected infectious cases with transmission-prevention advice. Due to the large volume of incoming travellers that arrive in SEQ, and the non-specific nature of cryptosporidiosis symptoms, this scenario assumes that only 20% of incoming travellers with symptomatic *Cryptosporidium* infections will receive this advice.

This scenario is likely to pose several logistical and financial challenges to implement as there are currently no systems in place to screen incoming passengers for infectious diseases.

#### 7.2.1.1. Scenario 2 – PAF infrastructure intervention

This scenario represents an infrastructure-focused intervention that requires all public aquatic facilities to install a secondary disinfection system, such as a UV or ozone treatment, capable of rendering 99.9% of oocytes inactive (i.e. log-3 disinfection) within 7 turnovers of the pool water. This scenario is likely to be difficult to implement because of the high cost of these systems and the resultant financial burden on PAFs. It is included to demonstrate the effects of an infrastructure-focused intervention, and because it is the most effective individual intervention.

#### 7.2.1.2. Scenario 3 – PAF awareness intervention

Two scenarios were created that represent the implementation of two education and awareness interventions targeted at the staff of public aquatic facilities

<u>Scenario 3A – Faecal Accident Awareness</u>: Scenario 3A represents the implementation of an operator and staff education intervention with specific focus on the operators of small (<250,000 litres) swimming pool. It reflects a continuation of the current management approach of regulating the operation of public aquatic facilities but adds specific emphasis on improving the capacity of PAF operators to appropriately identify and remediate faecal accidents that happening in and around the swimming pool. As the sensitivity analysis indicated that the AFR detection and management rate offered more leverage in small pools than large pools, this intervention is targeted primarily at small pools. A modest increase in the proportion of pool patrons who shower was also included in this scenario to reflect a minor amount of carry-over of *Cryptosporidium*-related knowledge from swimming pool operators to pool patrons that may result from more knowledgeable staff.

		Scenario 1	Scenario 2	Scen	ario 3	Scena	ario 4	Scen	ario 5
		Traveller intervention	PAF infrastructure intervention	PAF awareness intervention		Healthcare intervention		Combined intervention	
Variable	'base case'			3A	3В	4A	4B	5A (3A+4A)	5B
Physician precautionary advice fraction	5%	(NC)	(NC)	(NC)	(NC)	10% (+5%)	30% (+25%)	10% (+5%)	30% (+25%)
Fraction of positive cases made aware	43%	(NC)	(NC)	(NC)	(NC)	75% (+32%)	60% (+17%)	75% (+32%)	60% (+17%)
Percent of symptomatic travellers screened out	0%	20% (+20%)	(NC)	(NC)	(NC)	(NC)	(NC)	(NC)	(NC)
Percent of patrons who shower	15%	(NC)	(NC)	25% (+10%)	20% (+5%)	(NC)	(NC)	25% (+10%)	20% (+5%)
AFR detection and proper management rate	30%	(NC)	(NC)	SP:60% (+30%) LP:40% (+10%)	SP:40% (+10%) LP:40% (+10%)	(NC)	(NC)	SP:60% (+30%) LP:40% (+10%)	SP:40% (+10%) LP:40% (+10%)
Routine hyperchlorination frequency	switched off	(NC)	(NC)	(NC)	Monthly	(NC)	(NC)	(NC)	(NC)
3-log secondary disinfection system	switched off	(NC)	Switched on – all pools	(NC)	(NC)	(NC)	(NC)	(NC)	(NC)

 Table 7.5: Model parameters used for each policy scenario

Numbers in brackets denote change from 'base case' scenario NC: No change from 'base case' scenario <u>Scenario 3B – Routine Hyperchlorination</u>: Scenario 3B represents a strengthening of the current Swimming Pool and Spa guidelines with a requirement that all PAFs hyperchlorinate their swimming pools monthly. A monthly interval was chosen for this scenario as the sensitivity analysis found that quarterly hyperchlorination was not a sufficiently sensitive interval, and the stakeholders indicated that fortnightly or weekly hyperchlorination is unlikely to be feasible for many facilities in the study area. This scenario also includes modest increases to the rates of patron showering and faecal accident detection, which would likely accompany strengthen guidelines.

It is anticipated that Scenario 3B would likely place a greater financial and resource burden on the operators and staff of PAFs than Scenario 3A, due to the cost associated with routinely hyperchlorinating a swimming pool, and the extended duration that the facility must be closed during treatment.

#### 7.2.1.3. Scenario 4 – Healthcare interventions

This scenario represents two interventions targeted at improving the way medical practitioners communicate prevention-related information to suspected and confirmed cases of cryptosporidiosis.

- Scenario 4A Post-diagnosis transmission-prevention advice: Scenario 4A is based on a stakeholder-proposed intervention to include transmission prevention-related advice on the pathology results forms that healthcare providers receive when their patient tests positive for *Cryptosporidium*. This scenario assumes that providing doctors with this information at the moment they are communicating the results to the patient, will result in at least 75% of confirmed cases receiving the correct preventative advice. This scenario also includes a modest increase (+10%) in the number of suspected cases who receive prevention-related advice prior to diagnosis. It is assumed that as medical practitioners become more familiar with the correct preventative advice to give to patients with confirmed *Cryptosporidium* infections, they will also begin providing this advice to patients with suspected cryptosporidiosis.
- <u>Scenario 4B Precautionary pre-diagnosis transmission-prevention advice</u>: Scenario 4B represents an intensive campaign targeted at educating medical practitioners about cryptosporidiosis symptoms and transmission, with the goal of encouraging them to provide suspected cases with transmission-prevention related advice when they first present to their clinic (prior to having a laboratory-confirmed diagnosis). This scenario assumes that providing doctors with this information will result in 30% (+25%) of symptomatic cases

receiving prevention-related advice prior to diagnosis. This scenario also includes a moderate increase (+17%) in the number of suspected cases who receive prevention-related advice post-diagnosis, as it is assumed that educating doctors about *Cryptosporidium* infections will also result in more laboratory-confirmed cases receiving the correct advice.

#### 7.2.1.4. Scenario 5 – Combined intervention

This scenario represents two different combined strategies using the interventions discussed in scenario 3 and 4. Interventions from scenarios 1 and 2 were not included in the combined interventions due to the high financial and logistical requirements associated with these strategies.

<u>Scenario 5A – Faecal Accident Awareness + Post-diagnosis transmission-prevention</u> <u>advice</u>: Scenario 5A represents a situation where both faecal accident awareness education at PAF (scenario 3A) and modifying the pathology results forms to include transmission prevention-related advice (scenario 4B) are implemented concurrently. This represents a combined scenario that would require a moderate amount of resource investment to achieve.

 <u>Scenario 5B – Strengthened PAF guidelines + Precautionary pre-diagnosis</u> <u>transmission-prevention advice:</u> Scenario 5B represents a situation where the intensive campaign targeted at educating medical practitioners about cryptosporidiosis symptoms and transmission (scenario 4B) is implemented, as well as minor strengthening of the swimming pool guidelines (modified scenario 3B). This scenario includes all the changes listed on scenario 3B except for the monthly hyperchlorination, as that intervention represents a significant burden to operators of PAFs in the study area, and its inclusion in this scenario resulted in a negligible decrease in the number of cryptosporidiosis notification over the study period<sup>19</sup>. This represents a combined scenario that would require a higher amount of resource investment to achieve.

#### 7.2.1.5. Results of the analysis of scenario simulations

All six scenarios achieved a reduction in total number of cryptosporidiosis notifications when simulated over the study period (Figure 7.8). The use of secondary disinfection systems in scenario 2 produced the greatest reduction in total cases (percent difference in total cases over study period:

<sup>&</sup>lt;sup>19</sup> Including monthly hyperchlorination in scenario 5B resulted in a 0.81% reduction in the total estimated number of cryptosporidiosis notifications over the 10-year study period when compared to scenario 5B without monthly hyperchlorination.

48.25%), while international traveller screening (scenario 1) produced the smallest reduction (percent difference: -13%).

Scenario 2 (PAF infrastructure intervention) produced the largest reduction when compared to the base-case scenario across all four sensitivity measures. This scenario was excluded from the prioritization of the strategies because it is considered too expensive to be feasible. The main purpose for its inclusion is to provide a best-case scenario as a contrast with less effective intervention scenarios.



## Figure 7.8: Cumulative total number of cryptosporidiosis notifications over the study period (2007-2017) under each policy scenario

In terms of days above of the non-outbreak mean, scenario 5B (combined intervention) produced the greatest reduction from the base-case (Table 7.6), with only an estimated 29.8% of days during the study period with notified cases of cryptosporidiosis over the non-outbreak mean compared to the base-case of 55.5% of days. Scenario 4B (pre-diagnosis advice) represented the single sector intervention that resulted in the greatest reduction in overall days above the non-outbreak mean.

Days above non-outbreak mean		
Scenario	Count (% diff from base case)	
Base Case	2024	
Scenario 2	075	(70.09%)
(PAF - Infrastructure)	915	(-70.078)
Scenario 5B	1000	(60.10/)
(modified $3B + 4B$ )	1089	(-60.1%)
Scenario 5A	1100	(-52.5%)
(3A + 4A)	1182	
Scenario 4B	1269	(-45.9%)
(Pre-diagnosis advice)		
Scenario 3B	1404	(-36.2%)
(routine hyperchlorination)	1404	
Scenario 3A	1505	( 20, 40/)
(faecal accident awareness)	1505	(-29.4%)
Scenario 4A	1575	(-25.0%)
(Post-diagnosis advice)	13/3	
Scenario 1	1630	( 21 (0/)
(International travellers)		(-21.0%)
	ScenarioBase CaseScenario 2(PAF - Infrastructure)Scenario 5B(modified 3B + 4B)Scenario 5A(3A + 4A)Scenario 4B(Pre-diagnosis advice)Scenario 3B(routine hyperchlorination)Scenario 3A(faecal accident awareness)Scenario 4A(Post-diagnosis advice)Scenario 1(International travellers)	ScenarioDays above non-Count (% diff from Count (% diff from Count (% diff from Count 2Base Case2024Scenario 2975(PAF - Infrastructure)975Scenario 5B1089(modified 3B + 4B)1089Scenario 5A1182(3A + 4A)1182Scenario 4B1269(Pre-diagnosis advice)1404Scenario 3B1404(routine hyperchlorination)1505Scenario 4A1575(Post-diagnosis advice)1575Scenario 11630

Table 7.6: Scenario	priority bas	sed on days above	ve the non-outbreak mean
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Scenario 5B (combined intervention) also produced the greatest reduction in outbreak intensity when compared to the base case scenario (Table 7.7). Single-sector interventions in scenario 4B and scenario 3B both produced greater reduction in outbreak intensity than the combined intervention in scenario 5A by a small margin.

		Outbreak intensity <sup>1</sup>		
Priority				(% difference
order	Scenario	Mean	stdev	base case)
	Base Case	6.6	6.0	
*	Scenario 2	1 /	1.0	(121.20/)
	(PAF - Infrastructure)	1.4	1.0	(-131.270)
1	Scenario 5B	26	1.8	(-85.8%)
1	(modified $3B + 4B$ )	2.0		
2	Scenario 4B	2.8	3.4	(-53.2%)
۷	(Pre-diagnosis advice)	5.0		
2	Scenario 3B	4.0	2.7	(-49.4%)
3	(PAF routine hyperchlorination)	4.0		
1	Scenario 5A	4.0	4.0	(-48.3%)
-	(3A + 4A)	4.0		
5	Scenario 3A(PAF faecal accident	4.0	5.2	(-29.8%)
5	awareness)	4.9		
6	Scenario 4A	5.0	4.2	( 26 50/)
0	(Post-diagnosis advice)	5.0		(-20.370)
7	Scenario 1	5.2	4.5	(22,20/)
/	(International travellers)	5.5		(-22.3%)

 Table 7.7: Scenario priority based on outbreak intensity (amplitude)

<sup>1</sup>Outbreak intensity (amplitude) is measured as the difference between the peak number of daily cryptosporidiosis notification and number of notifications on the day prior to the beginning of the outbreak.

Both combined scenarios (5A and 5B) produced moderate decreases in mean outbreak duration (Table 7.8), with reductions of 22.7 days and 21.2 days respectively. A modest synergistic effect was produced in scenario 5A, where the combination of interventions 3A and 4A produced an overall reduction in mean outbreak duration of 21.2 compared to 16.9 days and 3 days respectively. Scenario 1 (international travellers) had a negligible impact on outbreak duration.

Duiovity		Outbreak duration			
order	Scenario			(% difference	
oruci		Mean	stdev	base case)	
	Base Case	157.6	73.5		
*	Scenario 2	106 7	56.5	(38.6%)	
	(PAF - Infrastructure)	100.7	50.5	(-38.0%)	
1	Scenario 5B	13/ 0	59.2	(-15.6%)	
1	(modified $3B + 4B$ )	134.9			
2	Scenario 5A	126.2	65.0	(-14.5%)	
2	(3A + 4A)	130.3			
3	Scenario 3A	140 7	72.7	(-11.4%)	
	(PAF faecal accident awareness)	140.7			
4	Scenario 4B	141 4	69.1	(-10.8%)	
4	(Pre-diagnosis advice)	141.4			
5	Scenario 3B	148.8	54.3	(-5.7%)	
5	(PAF routine hyperchlorination)				
6	Scenario 4A	1546	70.6	(1.00%)	
	(Post-diagnosis advice)	134.0	/0.0	(-1.970)	
7	Scenario 1	157.0	72.5	(0.40/)	
	(International travellers)	157.0		(-0.4%)	

 Table 7.8: Scenario priority based on outbreak duration (period)

All scenarios, except for scenario 4A and 1, resulted in the occurrence of at least one less outbreak over the study period (Table 7.9). Interventions related to the operation and maintenance of PAFs produced the greatest reduction in the number of outbreaks, which is unsurprising as PAFs are most often the initial source exposure for major outbreaks.

Priority		Number of Outbreaks
order	Scenario	Count (% diff from base case)
	Base Case	8
*	Scenario 2	6 ( 29 60/)
	(PAF - Infrastructure)	0 (-28.070)
1	Scenario 3A	(28.60/)
1	(PAF faecal accident awareness)	0 (-28.0%)
1	Scenario 3B	
	(PAF routine hyperchlorination)	0 (-28.0%)
1	Scenario 5A	
	(3A + 4A)	0 (-28.0%)
4	Scenario 5B	7 (12.29/)
	(modified $3B + 4B$ )	/ (-13.3%)
4	Scenario 4B	7 (12.20/)
	(Pre-diagnosis advice)	/ (-13.370)
6	Scenario 4A	8 (09/)
	(Post-diagnosis advice)	ð (U70)
6	Scenario 1	8 (09/)
	(International travellers)	8 (0%)

Table 7.9: Scenario priority based on total number of outbreaks

The radar plot in Figure 7.9 demonstrates the outcomes of each scenario across the four sensitivity measures and the estimated total number of cases over the study period. The base-case scenario is represented by the black exterior perimeter of the pentagon. Scenarios with values closer to the centre indicate greater reduction in that measure compared to the base-case scenario. This plot also demonstrates the degree of change that is achievable across the 5 different measures. Outbreak amplitude and days above the non-outbreak mean are the two measures where the greatest percent change from the base-case could be achieved, with maximum possible reductions of -86% and - 60%, respectively. Outbreak period and total number of outbreaks were the measure that was least changed by all of the interventions, achieving maximum possible reductions of only -16% and - 29%, respectively.



Figure 7.9: Performance of each scenario, measured in percent difference from base case (a smaller polygon indicates greater reduction across the five measures)

To rank the scenarios based on overall performance two different measures were used; 'impact effect' and 'impact symmetry'. For this study, 'impact effect' refers to the ability of the intervention(s) in each scenario to produce the greatest combined percent difference from the base case scenario across the five criteria of outbreak intensity, duration, frequency, days above the non-outbreak mean, and the estimated total number of cases over the study period. To calculate the 'impact effect' for each scenario, the total summed percent difference from the base case scenario was calculated. Table 7.10 summarized the overall performance in terms of 'impact effect' of the seven scenarios for cryptosporidiosis reduction in SEQ.

Priority order	Scenario	Total percent difference across all measures
1	Scenario 5B (Pre-diagnosis advice + strengthened PAF guidelines)	-218.4%
2	Scenario 5A (PAF faecal accident awareness + Post-diagnosis advice	-180.2%
3	Scenario 4B (Pre-diagnosis advice)	-157.1%
4	Scenario 3B (PAF routine hyperchlorination)	-152.9%
5	Scenario 3A (PAF faecal accident awareness)	-124.2%
6	Scenario 4A (Post-diagnosis advice)	-70.0%
7	Scenario 1 (International travellers)	-57.3%

**Table 7.10:** Overall scenario priority based on 'impact effect' (total combined percent difference across all five criteria)

For this study, 'impact symmetry' refers to the relative performance of each scenario across all five criteria, compared to the best performing scenario (scenario 2: 3-log secondary disinfection systems). While scenario 2 is unlikely to be feasible given current conditions, it represents the 'best case' scenario in terms of what could reasonably be achieved given sufficient investment. Relative performance was calculated as:

$$RP = \left(\frac{\sum \left(\frac{scenario\ value}{best\ performing\ scenario\ value}\right)}{5}\right) \times 100$$

The relative performance for each scenario across the five different measures is summed together to get a score of overall 'impact symmetry'. A larger summed relative performance indicates a smaller deviation from the best performing scenario, which therefore indicates better overall performance across the five impact measures. As each of the measures are given equal weighting in this equation, scenarios whose impact is more symmetrical across the different measures will result in better score than measures that performed very well across a limited number of measures and moderately or poorly across the remaining measures. Table 7.11 provides a summary of the overall performance in terms of 'impact symmetry' of the seven simulated scenarios.

Priority order	Scenario	Total percent difference across all measures
1	Scenario 5B	65 75%
1	(Pre-diagnosis advice + strengthened PAF guidelines)	03.7370
2	Scenario 5A	64 059/
Z	(PAF faecal accident awareness + Post-diagnosis advice)	04.9370
3	Scenario 3B	51 510/
	(PAF routine hyperchlorination)	54.5170
4	Scenario 4B	50 220/
4	(Pre-diagnosis advice)	30.23%
5	Scenario 3A	40.220/
	(PAF faecal accident awareness)	49.22%
6	Scenario 4A	10 000/
	(Post-diagnosis advice)	19.0070
7	Scenario 1	15 100/
	(International travellers)	13.1970

**Table 7.11:** Overall scenario priority based on 'impact symmetry' (relative performance of each scenario compared to the best performing scenario)

Scenario 5B is the best performing scenario both in terms of impact effect and impact symmetry<sup>20</sup>, with scenario 5A also performing well across both measures. Interestingly, healthcare-related scenario 4B (pre-diagnosis advice) outperformed the two PAF-related scenarios in terms of total impact effect. It's 4<sup>th</sup> place ranking in terms of impact symmetry is like due to it only reducing the number of outbreaks by one, as compared to the PAF-related interventions which had a greater effect on the outbreak frequency measure. Conversely, while scenario 3B (PAF routine hyperchlorination) had slightly lower overall impact than scenario 4B, its effect was symmetric, , indicating that it is likely to have a beneficial effect across more of the criteria than scenario 4B. Scenario 4A and 1 both performed quite poorly across both measures, indicating that they are likely poor investments if applied independently.

#### 7.3. Insights and policy implications

AS mentioned in chapter 5, the primary aim of system dynamics models is not to produce predictions per se, but rather to generate an expanded theory of the relationship between system structure and behaviour in the form of 'insights'. In addition to 'insights' where the model provides information or an understanding that differs from currently held mental models of the system or the

<sup>&</sup>lt;sup>20</sup> Recognising that the relative importance of each of the 5 criteria may not be equal, a more robust measure would be to weight the criteria in according to stakeholder feedback on each criteria's relative importance prior to calculating the overall effect. While outside the scope of this project, this potential limitation could be addressed in future studies.

problematic behaviour it produces, insights can also be the identification of previously unexplored leverage points. In other words, while the results of the scenario analysis discussed in the previous section provide important information on the strength of various leverage points within the system, they in and of themselves are not the true results of the research.

The following sections outline a number of 'insights' produce as a result of the simulation modelling process and discusses their broader implications from a policy perspective.

#### Nature of the 'cyclic' pattern of cryptosporidiosis notifications in SEQ

Prior to the commencement of this project there had been widespread speculation amongst stakeholders that the temporal pattern of cryptosporidiosis notifications was following a 2-3 year cycle (i.e. oscillating trend). This infers that the probability of a large outbreak occurring in any given year is not independent of previous outbreaks. The thorough search of the literature conducted as part of this project (detailed in section 3.1) was unable to identify evidence to support this theory, nor a biologically plausible explanation for this pattern of behaviour.

The results of the model, as well as the outcome of the autocorrelation analysis, suggest that the apparent temporal outbreak pattern observed during the study period is not cyclic, but rather a series of sequential, yet independent, instances of overshoot and collapse behaviour (as shown in Figure 7.10). Overshoot and collapse is a common behaviour observed in infectious disease outbreaks, where the number of cases rises exponentially while the ratio of exposed to susceptible cases who have infectious contact with each other is high (i.e. when  $R_0 > 1$ )<sup>21</sup>. At a certain threshold, the number of susceptible cases who have contact with infected cases (or the contaminated environment) will reach a point of saturation, and then fall exponentially.

The assumption that cryptosporidiosis outbreaks are driven by a 2-3 year cyclic is likely due to the clustering illusion, which is a common cognitive bias of identifying the inevitable common patterns, clusters or trends that occur in small samples drawn from random distributions as non-random events (Gilovich, 1991).

<sup>&</sup>lt;sup>21</sup>The basic reproductive number (denoted  $R_0$ ) refers to the number of new infections each infectious case produces. When >1, infection will continue to spread through the population. When <1, the outbreak will decline and eventually cease.

On the other hand, the autocorrelation analysis did identify a statistically significant yearly cycle of notifications, supporting the hypothesis that cryptosporidiosis notifications are seasonally dependent.



Figure 7.10: Generic structure of overshoot and collapse behaviour (source: Breierova (1997))

The implication of this insight for decision-makers is that the number of cases in previous year should not be viewed as in indicator of the timing or likelihood of future outbreaks, but it likely safe to assume that the peaks and troughs of notification will come at approximately the same time each year.

#### Ratio of notified cased to true cases in the community

While it is widely accepted that cryptosporidiosis, like many other acute gastrointestinal illnesses, is largely underdiagnosed and underreported in the community, the degree to which this is occurring is unclear. Knowing the degree to which this is occurring is critical for effective outbreak detection and management, as it is the key to establishing the true scope of the problem within the community. As it is nearly impossible to identify all cases within the community, public health surveillance systems use an 'under-reporting' and 'under-diagnosis' multiplier to estimate how many cases exist in the community for each case that is captured by the surveillance system. A wide range of 'under-diagnosis' and 'under-reporting' multipliers are being used for cryptosporidiosis in surveillance systems around the world, ranging from 7.4 in Australia, 10.4 in the United Kingdom, and 98.6 in the United States and New Zealand (Cressey and Lake, 2011, Kirk et al., 2014). The order of magnitude different between these multipliers produce vastly different estimates of true cases in community. Under the base case scenario, the model produces an under-diagnosis multiplier of 8.4 (s.d.: 1.78), which is quite consistent with the multiplier that is currently

being used in Australia (7.4 - as reported Kirk et al. (2014)), and lends support to the idea that the proportion of true cases that result in disease notifications is far greater than the American/ New Zealand estimate would suggest.

#### Larger-than expected role of overseas acquired cases in local disease dynamics

Prior to this project, little consideration has been given to the role of imported cases in local disease dynamics. Studies that have identified international travel as a strong risk factor for *Cryptosporidium* infections often question or downplay its role. For example, despite identifying overseas travel as the risk factor with the second highest odds ratio<sup>22</sup> in a case-control study of sporadic cryptosporidiosis cases in two Australian cities (Melbourne and Adelaide), the authors of the study concluded that the their finding of overseas travel being a strongly associated risk factor for sporadic cryptosporidiosis is likely due to ascertainment bias instead of a true incidence in the community (Robertson et al., 2002). The results of the model developed in this project support the finding that overseas travel is a strong risk factor for sporadic cases of cryptosporidiosis. Imported cases not only represent a far greater proportion of cases present in the community than expected, but that they also play a critical role in the local disease dynamics, by seeding local transmission. The role of imported cases seeding local disease transmission is not unique to *Cryptosporidium* infections, as studies have found imported cases have been responsible for outbreaks of diseases such as Ebola, measles and Middle East Respiratory Syndrome, Influenza, and Zika virus (Gomes et al., 2014, Koenig et al., 2016).

Despite the model indicating the importance of imported cases in the local disease dynamics, the results of the scenario analysis suggest that screening and educating incoming passengers is a low leverage intervention and is not recommended.

The larger-than expected role of overseas-acquired cases in local disease dynamics has several policy and research implications. The first is that while the number of imported cases is not a strong policy lever, they never the less deserve attention in the management of the disease. Controlling the presence of these cases may not be something that can easily be done but mitigating their impact is potentially more achievable. Decision-makers should consciously and explicitly

<sup>&</sup>lt;sup>22</sup> Overseas travel and consuming unboiled water, ice cubes or salad overseas was second only to immune system illness.

account for the fact that imported cases will likely continue to seed outbreaks in the community when designing interventions.

The second implication of this insight is that attention should be paid to the drivers of disease dynamics both during and outside of outbreak periods. Outbreak investigations are likely to significantly underestimate the total number of overseas acquired cases in the community as these cases only make up a small percentage of cases during major outbreaks. Therefore, sources of exposure identified during major outbreaks may not be representative of sources of exposure during non-outbreak periods because waterborne and foodborne illness outbreaks are typically investigated with the goal of source attribution, instead of contact tracing. The intent of source attribution is to identify the reservoir within the community driving the outbreak (i.e. where/what is causing the outbreak), rather than identifying the index case (i.e. who started the outbreak).

#### Effectiveness of management techniques applied to small vs. large public swimming pools

Several differences between the effectiveness of PAF-related interventions in small and large PAFs emerged during the sensitivity and scenario analysis of the model. Across nearly all PAR-related interventions (3-log disinfection systems, routine hyperchlorination, ARF detection and propermanagement rate), interventions targeted at small pools had a much greater effect on reducing the overall burden of outbreaks than those targeted at large pools. Additionally, interventions targeted solely at small pools were still able to achieve high overall reduction in case notifications, even in the absence of any interventions targeted at large pools. This finding is not surprising as small pools tend to be used by individuals who are at a high-risk of both transmission and exposure (i.e. incontinent young children who swallow a lot of water while swimming). The insight that emerges from this finding is that it may not be necessary to target all PAFs within the community with PAF-related interventions to achieve an acceptable reduction in case notifications. Achieving cost savings by focusing interventions at all small PAF pools may still be sufficient to greatly reduce the burden of cryptosporidiosis in the community.

#### Unexpected role of primary care in the dynamics of Cryptosporidium transmission in SEQ

The current narrative surrounding *Cryptosporidium* in SEQ points to the overarching notion that *Cryptosporidium* is a 'swimming pool problem' or perhaps a 'water problem'<sup>23</sup>. Unsurprisingly,

<sup>&</sup>lt;sup>23</sup> Cryptosporidium is also viewed as a zoonosis problem in many regions but is not considered as such in SEQ due to the local urban population's limited contact with livestock.

this has led to the operation and maintenance of PAFs being the main target of interventions within SEQ. The results of the simulation model demonstrate that cryptosporidiosis is a far more complex and interdisciplinary problem.

The results of the simulation model supported the findings from chapter 5, which highlighted that community-level disease dynamics are associated with the interactions between people and primary healthcare providers. The model demonstrated empirically that if doctors provide patients with advice to reduce their likelihood of transmission, particularly when they first present for medical care, the number of new cases of cryptosporidiosis in the community will be significantly reduced. It was surprising to observe that healthcare-related variables, in particular medical practitioners providing advice to suspected (unconfirmed) cases, were higher leverage variables than many of those within the PAF sector.

There is considerable evidence that interventions targeted at educating doctors lead to direct improvements in patient outcomes for several health problems. For example, a study assessing the quality of care delivered by doctors to patients who smoke found that healthcare providers who received training in smoking cessation, as well as reminders and prompts to provide advice to their patients, were more effective than doctors without the training and prompts (odds ratio 1.35) (Silagy et al., 1994). Similar effects were found in a meta-analysis of interventions related to immunisation and cancer screening (Stone et al., 2002). While smoking and gastroenteritis differ in many ways, they are similar in terms of the role doctors play in providing guidance to symptomatic patients. Targeted continuing education interventions, in conjunction with electronic reminders, may present a novel strategy to reduce the transmission of *Cryptosporidium* within the community, especially in high risk periods of the year.

# <u>Rethinking the role of primary and secondary outbreak prevention in the design of intervention strategies</u>

The current goal of the public health intervention for cryptosporidiosis is primary outbreak prevention<sup>24</sup> by eliminating or reducing the risk of an initial exposure to *Cryptosporidium*. While

<sup>&</sup>lt;sup>24</sup> Definitions of primary and secondary prevention vary greatly within the field of environmental health, and largely depend on what is trying to be prevented (e.g. individual illness, outbreaks, etc.). For the purpose of this study, primary outbreak prevention refers to interventions targeted at eliminating the hazard (cryptosporidium) within the community, and thus preventing outbreaks from occurring. Secondary outbreak prevention refers to interventions targeted at containing outbreaks to reduce the magnitude of their effect.

primary outbreak prevention is the front line of community-level infectious disease control and management, the realities of cryptosporidiosis dynamics in SEQ, particularly the consistent inflow of imported cases, makes primary outbreak prevention difficult. Indeed, the outputs of this model showed that the frequency of major outbreaks was one of the least modifiable outcome measures. Even under the best-case scenario the frequency of outbreaks could only be reduced by up to 29%.

The results of the model indicate that, for the most part, individuals with overseas acquired *Cryptosporidium* infections are seeding community outbreaks. The realities of globalization and increasing population mobility mean that the consistent flow of individuals with overseas acquired *Cryptosporidium* infections into the community is unlikely end. The implication of this is that as long as the importation of overseas-acquired infections remains exogenous to the system, outbreaks are likely to continue regardless of primary prevention efforts in the community.

Additionally, the system exhibits delays and variability that undermine the effectiveness of interventions targeted at preventing outbreaks, particularly those related to public aquatic facilities. Complex and expensive secondary disinfection systems, often promoted as being a 'solution' to the *Cryptosporidium* problem, still required a significant period of time to achieve complete removal of oocytes in the water due to the large size of most public pools. Completed detection and management of ARFs as an outbreak prevention strategy is also effective, but likely unachievable due to the complexity and difficulty of managing busy swimming pool, particularly with a highly seasonal workforce. Additionally, visual detection as a means of identifying accidental faecal releases into swimming pool will only be possible for gross faecal contamination events. Regardless of the diligence or training of the swimming pool operator, small faecal leakage events are likely to go undetected by staff unless they are directly notified by the patron.

The result of the scenario simulations showed that outbreak intensity was the most modifiable outcome measure. Interventions targeted at modify outbreak intensity are exclusively secondary disease prevention. These interventions acknowledge that outbreaks are inevitable and that activities are designed to minimise the number of people exposed to infection, which reduces the scale of an outbreak.

In the context of the system in the case study, shifting from a goal of primary prevention to a goal of secondary prevention would require a paradigm shift. *Cryptosporidium* infections are typically self-limiting, lead to short-term incapacity, and are unlikely to be fatal. Because of this, it is possible that such a paradigm shift will be within the confines of what is socially acceptable public health practice.

In her seminal essay "*Leverage Points: Places to Intervene in a System*" Donella Meadows identified a hierarchy of 12 different points within complex systems, ranging from low leverage single variable changes, to very high leverage system-wide paradigm shifts (Meadows, 1997). The second highest point of leverage within a system identified by Meadows is the goal of the system, the driving reason for which the system exists. She argues that shifting the goal of the system changed all the system components, effectively reshuffling the system.

Shifting the goal of the cryptosporidiosis system would require the realignment of health policy priorities and reallocation resources. Doing so would require the state health authority to weigh the costs and benefits of spending public funds on enforcement versus education, as well as determine the degree to which the financial burden associated with *Cryptosporidium* management should be placed on the private sector.

The current focus on preventing outbreaks has largely put the resource cost on the shoulders of PAF owners and operators. These interventions come at significant capital and staffing costs, yet the model indicates that they are likely to be of limited effectiveness. Stakeholders in the aquatics industry indicated that the profit margins on PAFs are typically quite small, particularly in the case of community pools, which raises the question about the economic feasibility of PAF owners independently shouldering the burden while potentially reaping limited reward (Hunter and Fewtrell, 2001). Additionally, placing further emphasis or legislative requirements on PAF-focused interventions will likely require increased investment, by both local government and the state health department, on enforcement. It is widely recognised that enforcement is a crucial, yet very costly, element of the long-term effectiveness of public health guidelines (Costich and Patton, 2013).

Education-focused interventions, particularly those targets at GPs, are also not without their costs. Studies of the effectiveness of GP education campaigns have found that active education campaigns that took place in multiple interventions over time were more effective at changing GP behaviour than passive interventions or those that occurred only once (Mansouri and Lockyer, 2007). However, active multi-stage interventions are likely to be more expensive than simpler passive education campaigns.

#### Unclear threshold for action for swimming pool water quality issues

There is widespread recognition that complete elimination of exposure to pathogens in water is not possible, and thus when it comes to managing the safety of water resources, a risk-based approach must be adopted (Fewtrell and Bartram, 2001). Figure 7.11 below shows a simplified version of an iterative water quality risk management framework developed by the WHO. The basic premise of

this framework is that the assessment of microbial risk is the starting point for the development of health targets, which guide risk management activities and ultimately affect public health status.



Figure 7.11: Simplified risk-based water management framework (source: Fewtrell and Bartram (2001))

The use of health targets to guide the design, implementation, and evaluation risk management activities is a key element of this process. As seen in Figure 7.11, health targets are not based solely on the absolute risk posed by the contaminant, but also on the threshold of acceptable risk. The use of an acceptable (or tolerable) risk threshold is especially relevant for waterborne pathogens, as complete elimination of pathogens from water is typically not feasible. The NHMRC Guidelines for managing risk in recreational water emphasize this point stating:

"These guidelines require that risk be reduced to a tolerable level rather than being eliminated altogether (complete elimination of risk is impossible). For most healthy people, water conforming to the guideline value will pose only a minimal increase in daily risk. However, water conforming to the guidelines may still pose a potential health risk to high-risk user groups such as the very young, the elderly and those with impaired immune systems."(NHMRC, 2008)

Assessing the combined absolute and acceptable risk posed by a water source allows decisionmakers to establish the 'goal posts' (i.e. health targets) for risk management activities. There are currently no established standards in Queensland or Australia for the acceptable risk of swimming pool water. The model highlights the challenges associated with the lack of this standard.

The results of the model indicate that under the 'base case' scenario, *cryptosporidium*- infection risk associated with swimming pool water falls outside of Category A (very good) less than 6% of the time, and only in small swimming, according to the NHMRC Recreational Microbial Water Quality guidelines. If swimming pool water is considered comparable to other forms of recreational water, this would suggest that the risk associated with exposure to swimming pool water is well within the range of 'tolerable/acceptable' the clear majority of the time, even under status quo (base case)

conditions. Conversely, when compared to the drinking water quality tolerable risk threshold of 1 illness per 10,000 population per year, the risk associated with swimming pools exceeds the tolerable risk threshold almost every year in Metro North and Metro South, and 60% of the time in the Gold Coast region.

There are several policy-relevant insights associate these findings. The risk management strategy, if any, required to reduce the risk of *cryptosporidium* infection in SEQ swimming pools depends on whether swimming pool water is considered recreational water, drinking water, or something in between. If considered drinking water, the consistent exceedance of tolerable infection risk suggests that risk management activities are not only warranted, but necessary. If considered recreational water, the relative rarity of days when infection risk exceeds the tolerable risk suggests that little to no additional risk management activity is necessary beyond the status quo.

It is likely that the threshold of tolerable risk is somewhere in between. Without an established infection risk threshold, there is no clear goal to guide risk management activities for cryptosporidium in SEQ. This has both economic and health implications. If an overly conservative risk threshold is assumed, private and public funds may be spent on unnecessary interventions. If an overly relaxed risk threshold is assumed, an unacceptable number of illnesses may occur, affecting not only the overall health of the community, but also potentially undermining public confidence in both the public health service and the aquatics industry.

The overall insight from this finding is that it is not clear whether or not the current water quality situation in SEQ is above or below a level of 'tolerable' risk threshold. Without an established tolerable risk threshold for swimming pool water, decision makers are left to guess whether the risk is sufficiently managed or not. A secondary key insight from this finding is that it is quite possible that the currently status quo is below that tolerable risk threshold, and no additional action is required.

#### Value-added as a decision support tool

Prior to exploring this problem using system dynamics modelling, potential leverage points for the effective management of cryptosporidiosis in South East Queensland were elusive due to the problem being plagued with high levels of problem uncertainty. After applying this method to the specific case of cryptosporidiosis in South East Queensland, I have identified several ways that the system dynamics model process helped reduce three main types of uncertainty; aleatoric uncertainty, epistemic uncertainty, and process (deep) uncertainty. A broader insight that emerged from this modelling process is the many ways that this process could add value as a decision-

support tool by reducing the amount of uncertainty decision-makers must contend with. The following section outlines how the model helps reduce each of the three main types of uncertainty.

#### Aleatoric uncertainty (variability and heterogeneity)

The system dynamics model allowed variables that are characterised by significant natural variability and/or heterogeneity to be parameterised with Beta-PERT. These distributions leveraged existing research evidence by combined values from the literature (where available) with local expert opinion, to identify maximum, minimum and most likely values for each uncertain parameter. The model also adds value by producing a graphical output for each variable, which makes the variability that characterises each variable more explicit to users of the model. Additionally, using the system dynamics model to simulate this problem allowed discrete semi-random events, such as the timing of when an infected patron has an AFR in the pool, to be captures as part of wider system behaviour. When run over several iterations, the model adds value in terms of its ability to show decision-makers the array of potential behaviours the system can produce when natural variability is accounted for.

#### Epistemic uncertainty (lack of information)

The problem of cryptosporidiosis in SEQ is plagued by a lack of locally-specific decision-relevant knowledge and information about both the parameters in the system, and the causal relationship that form the architecture of the system. Traditionally, there is a tendency to omitting concepts or variables from decision support tools in circumstances where there is no (or insufficient) numerical data to discern a representative numerical value. Jay Forrester describes the problem with this saying: "To omit such variables is equivalent to saying they have zero effect—probably the only value that is known to be wrong!" (Forrester, 1968).

In relation to uncertainty caused by a lack of knowledge (i.e. 'we roughly know' and/or 'we know what we do not know') the system dynamics model adds value in two ways. Much like the way Aleatoric uncertainty can be reduced by parameterizing the model with beta-PET distributions, factors whose value are unknown were also parameterised with this technique, but using expert opinion. This allowed the assumed system structures and relationships associated with these variables to be captured and made explicit despite the lack of research evidence.

Additionally, calibrating the model based on the historic behaviour of known variables allowed the values of variables for which there is no research evidence, such as number of asymptomatic cases or the probability of having an AFR given infect, to be estimated. While these estimates are not a substitute for local data or research evidence, the reality of decision-making is that time and

resources often limit the ability of this information to be collected and analysed prior to a decision being made. These calibrated values likely provide a substantial improvement over estimates produced by the mental models of decision-makers.

#### Process (deep) uncertainty

The most difficult form of uncertainty to reduce is that related to ignorance or conflicting evidence regarding the structure of the system itself (i.e. 'we do not know what we do not know' and/or 'we do not know what we know'). Reducing this form of fundamental uncertainty is one of the areas where system dynamics modelling has the potential to have the greatest value.

One of the key ways that the SD modelling process reduced this form of uncertainty in relation cryptosporidiosis in SEQ is by elucidating, capturing and integrating the mental models of stakeholders and actors across different sector into the model. Because of the complex nature of this problem, each actor holds a limited, yet unique, understanding of the interplay between and amongst human and environmental systems that is creating this problem. By forcing each actor to make their mental models explicit, and then combine the collective knowledge held within each of these mental models, a broader and more complete image of the problem emerged. It is through this process that uncertainties that were unknown were revealed. For example, the effect of physicians providing advice to infectious individuals, and more specifically the timing of that advice, on the community-level outbreak dynamics was a previously unknown relationship. Had the mental models of individuals in the water quality sector not been combined with those doing front-line public health investigations, and those of physicians, this causal relationship would have remained unknown.

More broadly, the system dynamics model adds value in the way that it packages the information it contains. From the most basic level, the model reduces the conceptual complexity of the cryptosporidiosis problem by presenting a colour-coded visual map of the assumed causal structures that make up the problem. By making everyone's assumptions explicit in one diagram, it creates a common language for discussing the problem. The model interface also communicates the nature of the relationships used to simulate the model in a simpler and more explicit way that can be more easily understood by users with varying levels of domain knowledge. At a higher level, the model communicates decision-specific actionable information to about the likely outcomes of a series of potential future interventions. The model itself was structured in such a way that decision makers could design and test potential future interventions in a virtual world prior to implementing these

interventions (as shown in section 7.2). By presenting its results in a decision-ready format, it increased the value of the model as a decision-support tool.

#### 7.4. Summary

The results of the sensitivity analysis and scenario analysis discussed in this chapter highlight the potential usefulness of system dynamics simulation modelling in providing policy-relevant insights for complex environmental health problems like *cryptosporidium*. Key insights from the model include the greater-than-anticipated effect of overseas-acquired infections and the timing of advice provided by general practitioners on transmission dynamics. Additionally, the model highlighted the importance of a tolerable risk threshold on prioritising risk management activities and suggested the need for a paradigm shift from primary to secondary outbreak prevention. Overall, the use of system dynamics modelling to explore the problem of cryptosporidiosis in South East Queensland provided a clearer understanding of the problem by uncovering some previously unknown or poorly understood elements of the system.

More broadly, insights can also be obtained from the overall process of applying system dynamics to a complex environmental health problem like *Cryptosporidium*. The modelling highlighted the importance of tapping into the knowledge and experience of a wide range of stakeholders when trying to develop a conceptual understanding of complex problems. While the idea of engaging stakeholders in the policy development process is not new, there is a lack of tools to facilitate this. The structured engagement process used in this project, particularly at the problem formulation and dynamic hypothesis stages, provided information that was simply not captured in traditional data bases and information collection channels.

There is also an ever-growing narrative about the need for bigger and better linked datasets to support infectious-disease related policy and research (Moore and Blyth, 2018). The results of this system dynamics modelling process demonstrated how this method allows for the integration of non-traditional forms of evidence, such as expert opinion and incomplete datasets, into a working simulation model. This model was built entirely on publicly-available datasets and research evidence, demonstrating that it is possible to gain a detailed understanding of complex disease dynamics using readily available data Additionally, this highlights how system dynamics modelling sits at the intersection between epidemiology, data science, public health decision making. System dynamics models of infectious disease management can integrate fundamental theories and insights from epidemiology about infectious disease transmission, distribution and control, with the linked

datasets emerging from the field of data science, in a manner that is understandable, applicable and approachable to public health decision makers.

This chapter demonstrates the potential usefulness of system dynamics modelling, not just for understanding cryptosporidiosis dynamics in SEQ, but also complex environmental health problems in general.

## Chapter 8. Conclusions

The aim of this project was to develop a tool that incorporated both qualitative and quantitative information to help decision-makers identify the underlying environmental and social feedback mechanisms that contribute to the transmission of cryptosporidiosis in South East Queensland (SEQ), Australia.

The qualitative systems map discussed in Chapter 5 combined mental models from actors in several different sectors into a collective conceptualisation of the systemic drivers of the problem. On its own, this system mapping exercise can support decision-makers achieve a better and more comprehensive understanding of the problems they are attempting to manage. The process of creating the causal loop diagram (CLD) served as a platform to integrate stakeholder perspectives into a common vision of the problem. It also helped communicate the importance of key feedback loops within the system that are contributing to outbreaks of cryptosporidiosis in SEQ.

The quantitative system dynamics model simulated the system behaviour produced by the relationships identified in the CLD. The simulation model is an additional tool that enables decision makers to design, test and evaluate possible strategies to reduce the disease burden of cryptosporidiosis in SEQ.

#### 8.1. Key findings in response to research questions

This central aim of this project lead to the formulation of three research questions. The following section summarises the key findings in relation to each of the research questions.

## Research Question 1: What are the population-level drivers of *Cryptosporidium* transmission in South East Queensland, and how do these drivers dynamically interact to create the trends in notified cases of cryptosporidiosis observed in the region?

A review of the academic and grey literature was conducted to obtain a comprehensive understanding of the current state of knowledge of the drivers of *Cryptosporidium* transmission in general, as well as in SEQ (see as discussed in Chapter 3). The list of drivers identified in the literature was then refined during consultations with local stakeholders. While not an exhaustive list, the following key population level factors were identified as contributing to *Cryptosporidium* transmission dynamics in SEQ (as presented in Chapter 5):

- Seasonally-driven community attendance at public aquatic facilities;
- Hygiene-related behaviours of swimming pool patrons;
- Person-to-person contact between infected and susceptible individuals, particularly those in childcare;
- Case management and diagnostic practices of local doctors;
- Timing, nature and quality of transmission-related preventative advice that doctors provide to suspected and confirmed cases of cryptosporidiosis;
- Timing of public health response to suspected cryptosporidiosis outbreaks;
- Attention given to cryptosporidiosis outbreaks by the media;
- Political will to address cryptosporidiosis in the community;
- Community knowledge and awareness of cryptosporidiosis as a problem in their community;
- Public aquatic facility (PAF) operator and staff awareness of cryptosporidiosis as a problem in their community;
- PAF operator and staff training with regards the management of water quality and hygiene;
- High turnover of seasonal PAF staff; and
- Infectious international travellers returning to the community.

Interestingly, commonly-cited drivers of *Cryptosporidium* transmission, such as contaminated drinking water and contact with livestock, were not deemed to be relevant in the context of SEQ.

The system dynamic simulation model (as presented in Chapter 6 and 7) identified several key dynamic relationships within the system that contributed to the trends in notified cases of cryptosporidiosis that have been observed in the region. Unsurprisingly, the timing of outbreaks observed in the region appears to coincide with seasonal attendance patterns at public aquatic facilities, which peak in the warmer summer months. Beyond this, several subtler dynamic relationships of importance were identified.

The observation that the timing of when doctors provide infectious individuals with advice to refrain for swimming or avoiding contact with other people was found to be a key driver of overall dynamics was unexpected. The standard practice of providing advice upon confirmation of infection (post-diagnosis) rather than before testing (pre-diagnosis) was shown to be problematic.

This is because the delay in preventive behaviours by infectious people significantly increases the risk of secondary infection and contamination of pool water.

The constant inflow of international travellers returning home with overseas-acquired infections was also a surprising observation enabled by the model. The system dynamic simulation model showed that if the inflow of infectious international travellers was prevented/removed then there would be no outbreaks in SEQ because local transmission dynamics are not sufficient to maintain an endemic state. This finding suggests that the consistent low level of 'sporadic' cases of cryptosporidiosis in SEQ is largely associated with the influx of overseas-acquired cases that "seed" the larger outbreaks observed. Therefore, the interplay between overseas-acquired cases seeding outbreaks and the rapidity with which infections spread through public aquatic facilities, is largely what is causing the outbreak dynamics observed in the region. The dominance of post-diagnosis advice provided by doctors, which results is poor awareness amongst infectious individuals of their risk of transmitting their infection to others, is likely to be contributing to the intensity and persistence of these outbreaks.

### **Research Question 2: What policies or interventions could be used to more effectively reduce** the incidence of cryptosporidiosis in South East Queensland?

Following the construction of the system dynamic simulation model (Chapter 6), sensitivity analyses were run to identify the modifiable factors within the model that had the greatest influence on the pattern of cryptosporidiosis outbreaks in SEQ (leverage).

Within the healthcare sector, the proportion of infected individuals who receive pre- versus postdiagnosis transmission prevention advice was the highest leverage variable. In the PAF sector, high leverage variables included the proportion of accidental faecal releases (AFR) that are detected and properly managed by pool staff, the frequency of routine hyperchlorination, and the use of 3-log secondary disinfection systems. Pre-swim showering and screening of incoming international travellers were also identified as leverage points with lower levels of influence.

The high leverage variables were then used to create nine policy/intervention options (as described in Table 7.5). These were then simulated using the system dynamic simulation model and ranked based on their effect on the dynamics of cryptosporidiosis in SEQ using 5 metrics. The most effective scenario modelled the installation of 3-log disinfection systems in all PAF facilities. While this option had the greatest effect, it is not deemed feasible due to the high installation and running cost.

The next most effective scenario represented a campaign to educate General Practitioners about cryptosporidiosis symptoms and transmission, with the goal of increasing the number of suspected cryptosporidiosis cases receiving pre-diagnosis advice from their doctor by 25% and the number receiving post-diagnosis advice from their doctor 17%. This scenario also includes minor improvements in adherence to the existing swimming pool water quality guidelines, representing an increase in bather showering by 5%, and an increase in AFR detection of 10% in both large and small pools.

Overall, while better pool disinfection systems would be effective in minimising cryptosporidiosis outbreaks, it would be difficult to impellent and enforce due to the high cost that the pool operators would have to bear. Better education of doctors was found to have almost the same effect on reducing cryptosporidiosis outbreaks. It is possible that doctor-education interventions would be easier to implement because the cost, which would primarily be the responsibility of the public sector, is likely smaller.

## <u>Research Question 3: Can system dynamics modelling add value as a decision-support tool for</u> <u>environmental public health decision-making processes, in particular in the management of</u> <u>cryptosporidiosis in South East Queensland?</u>

The insights outlined in Chapter 7 provide an example of the many ways that system dynamic modelling has the potential to add value to decision-making processes. The first is by providing decision-makers with an expanded and more explicit theory of what is causing the cryptosporidiosis outbreak pattern observed over the last ten years.

The systems mapping exercise identified previously unexplored causal relationships and archetypal behaviour that add clarity to the conceptualisation of the problem. This expanded conceptualisation of the problem can help to overcome policy resistance. Policy resistance is caused by our mind's inability to capture and understand the array of feedback loops that exist within a system (Sterman, 2000a). The process of describing explicit mental models and combining them with the mental models of others, can help overcome this common cause of policy resistance.

The added value of the system dynamic simulation is its ability to reduce uncertainty surrounding the problem, particularly process uncertainty. This form of uncertainty is the most difficult to overcome because it is difficult for decision-makers to account for system behaviour originating from uncharted parts of a system. As with the systems mapping process, reducing the uncertainty surrounding the problem has the value of expanding a decision-maker's understanding of the feedback loops driving the problem, and thus reducing the likelihood of policy resistance. The system dynamic model lead to the identification of cryptosporidiosis scenarios that were not being targeted to currently management strategies. Moreover, the system dynamic model showed that the current management strategies were targeting low leverage points, and therefore where not likely to reduce outbreaks

Lastly, this research demonstrated that a system dynamic model can provide actionable information to decision-makers. The system dynamic model was used to predict the long-term impact of a range of scenario on cryptosporidiosis outbreaks. Other decision-support tools, such as risk assessment, do not provide users with an easy way to compare the outcomes of different scenarios, nor do they assist in identifying potential unanticipated or indirect effects of interventions.

#### 8.2. Limitations and suggestions for future research

#### "all models are wrong, but some models are useful" – George E.P. Box

Despite the comprehensiveness of this model, and the compelling nature of the results, this model is not perfect. Sterman (2002) expands upon the famous George Box quote, explaining it is because "a model is a simplification, an abstraction, a selection". In order to create a model that is functional and workable, imposing boundaries on the model was necessary. The model presented in this thesis is a representation constrained to the boundary described at the beginning of Chapter 6 and represents an interpretation of the mental models of the specific stakeholders who were consulted as part of the model-building process. A limitation of the system dynamics modelling process is that it is inherently subjective, and the model's structure and behaviour is largely based off of the feedback from the stakeholders who were consulted. It is possible that broader consultation with additional stakeholder, particularly those in the primary healthcare sector and the aquatics industry, should be a focus of future research.

Additionally, the model was not designed to capture all potential factors contributing to *cryptosporidium* transmission, but rather focused on the ones identified by local stakeholders as being the most important. A limitation of this is that the model's structure is based on the assumption that the fundamental system structure does/has not changed during the study period and that the stakeholders have correctly identified the key contributors to *cryptosporidium* transmission. Because of this, the model is only valid in the context for which it was built, and the representativeness of the model's findings in other geographical, temporal or contextual areas has not been explored.

Furthermore, it is important when interpreting the results of the model to keep in mind the overarching intention and purpose of system dynamic models. System dynamic models are theoretical models, meaning that their primary purpose is to explore and expand upon the theory of what causes problematic behaviours in a system. Conversely, the purpose of System dynamic models is not to be a predictive model, in that they are not designed to formulate absolute statements such point-estimates, or 'predication'. Rather, they are used to explore and compare potential system trajectories under different policy scenarios. It is for that reason that the model presented in this thesis was intentionally designed such that it provided only retrospective findings. While system dynamics models are capable of forecasting into the future, this model was limited to a retrospective lens so as to not mislead users about its ability to predict the exact timing of future outbreaks. The limited ability of system dynamics models to produce accurate predictions of future behaviour is a limitation of the system dynamics models and users about is a whole.

A limitation of all models, including system dynamics models is that the results of the model may be sensitive to variations in certain model inputs and parameters. The system dynamics model presented in this thesis is parameterised with probability density functions to partially address this limitation. Nevertheless, the scarcity of locally-produced data means that it is possible that the model is sensitive to variables that have been calibrated to non-local data, which could lead to variations in the model's results.

An additional limitation of the system dynamics approach used in this thesis is that many of the functional relationships within the model are based on theory that has not been empirically tested, particularly in the context of South East Queensland. While great effort was made to use local data and evidence to support decisions related to model structure and behaviour, the paucity of local data meant that this was not always possible. Future empirical studies verifying the theory-based relationships identified in this model is necessary to confirm the model's findings.

The model developed in this thesis does not include economic drivers of system behaviour, nor does it evaluate the financial cost of any of the proposed scenarios, as this would be a very data-intensive exercise. Incorporating a measure of the financial and resource costs associated with each management scenario would be a future focus of the research as it would add an additional degree of decision-relevance to the model's findings.

This study stops short of fully capturing the final stage of Maani and Cavana's five stage modelbuilding process; implementation and organisational learning (Maani and Cavana, 2007). While this study has shown how system dynamic modelling can support environmental health decisionmaking, additional research is needed to assess the degree to which the modelling process can lead to meaningful organisational learning. Such an endeavour could provide insight on ways to improve the consultation and engagement process for future applications of system dynamic modelling in this context.

Additionally, this study did not include the implementation and evaluation phase of the recommended interventions. Extending this research program to implement and evaluate the effectiveness of the proposed scenarios would add additional insight to the effectiveness and value of using system dynamic modelling as a decision support tool for managing environmental health problems.

## References

- ACKOFF, R. L. 1979. The Future of Operational Research is Past. *The Journal of the Operational Research Society*, 30, 93-104.
- ACKOFF, R. L. 1994. Systems thinking and thinking systems. System Dynamics Review, 10, 175-188.
- ADAK, G. K., LONG, S. M. & O'BRIEN, S. J. 2002. Trends in indigenous foodborne disease and deaths, England and Wales: 1992 to 2000. *Gut*, 51, 832-41.
- ÁINE GORMLEY, S. P., SOPHIE ROCKS 2011. Guidelines for Environmental Risk Assessment and Management. *In:* DEPARTMENT FOR ENVIRONMENT, F. A. R. A. (ed.). United Kingdom.
- ALUM, A., ABSAR, I. M., ASAAD, H., RUBINO, J. R. & IJAZ, M. K. 2014. Impact of Environmental Conditions on the Survival of Cryptosporidium and Giardia on Environmental Surfaces. *Interdisciplinary Perspectives on Infectious Diseases*, 2014, 7.
- AMBURGEY, J. E. & ANDERSON, J. B. 2011. Disposable swim diaper retention of Cryptosporidium-sized particles on human subjects in a recreational water setting. *J Water Health*, 9, 653-8.
- AMODIO, E., COSTANTINO, C., ASCIUTTO, R., DINO, C., BIANCO, A., MARINGHINI, G., MAMMINA, C. & CALAMUSA, G. 2014. Knowledge, risk perception and behaviours in swimming pool users of Palermo city, Sicily. *Eur J Sport Sci*, 14 Suppl 1, S51-6.
- AMORÓS, I., ALONSO, J. L. & CUESTA, G. 2010. Cryptosporidium oocysts and Giardia cysts on salad products irrigated with contaminated water. *Journal of Food Protection*®, 73, 1138-1140.
- ANGELO, K. M., KOZARSKY, P. E., RYAN, E. T., CHEN, L. H. & SOTIR, M. J. 2017. What proportion of international travellers acquire a travel-related illness? A review of the literature. *Journal of Travel Medicine*, 24, tax046-tax046.
- ARMAH, F. A., YAWSON, D. O. & PAPPOE, A. A. N. M. 2010. A Systems Dynamics Approach to Explore Traffic Congestion and Air Pollution Link in the City of Accra, Ghana. *Sustainability*, 2, 252-265.
- ARNOLD, R. D. & WADE, J. P. 2015. A Definition of Systems Thinking: A Systems Approach. *Procedia Computer Science*, 44, 669-678.
- ASHBOLT, N. J., SCHOEN, M. E., SOLLER, J. A. & ROSER, D. J. 2010. Predicting pathogen risks to aid beach management: the real value of quantitative microbial risk assessment (QMRA). *Water Res*, 44, 4692-703.
- ASHBOLT, R. H., COLEMAN, D. J., MISRACHI, A., CONTI, J. M. & KIRK, M. D. 2003. An outbreak of cryptosporidiosis associated with an animal nursery at a regional fair. *Commun Dis Intell Q Rep*, 27, 244-9.
- ATTIAS, E., CZINN SJ HARRO, C., MUNOZ, F. & SOCKOLOW, R. 2015. Emerging Issues in Managing Pediatric Parasitic Infections: An Assessment of Clinical and Epidemiological Knowledge of Giardiasis and Cryptosporidiosis. *Pediatrics & Therapeutics*, 5.
- AUSTRALIAN BUREAU OF STATISTICS 2011. 2011 Census of population and housing. Australia.
- AUSTRALIAN BUREAU OF STATISTICS. 2012. Feature Article: International Movements [Online]. Available:

http://www.abs.gov.au/ausstats/abs@.nsf/Previousproducts/3401.0Feature%20Article1Dec%202012 ?opendocument&tabname=Summary&prodno=3401.0&issue=Dec%202012&num=&view= [Accessed January 3 2017].

- AUSTRALIAN BUREAU OF STATISTICS 2014. Participation in Sport and Physical Recreation, 2013-14. Australia.
- AUSTRALIAN BUREAU OF STATISTICS 2015a. National Health Survey, 2014-15. Australia.
- AUSTRALIAN BUREAU OF STATISTICS 2015b. Population Estimates by Age and Sex, Regions of Queensland (ASGS 2011), 2010 and 2015 Australia.
- AUSTRALIAN BUREAU OF STATISTICS. 2015c. Regional Population Growth, Australia, 2013-14 [Online]. Queensland, Australia. Available:

http://www.abs.gov.au/AUSSTATS/abs@.nsf/Previousproducts/3218.0Main%20Features302013-14?opendocument&tabname=Summary&prodno=3218.0&issue=2013-14&num=&view=# [Accessed Spetember 26 2016].

AUSTRALIAN BUREAU OF STATISTICS 2016. 2016 Census of population and housing. AUSTRALIAN BUREAU OF STATISTICS 2017. Overseas Arrivals and Departures, Australia, 2007-2017.

- AUSTRALIAN DEPARTMENT OF INFRASTRUCTURE REGIONAL DEVELOPMENT AND CITIES 2018. Monthly Airport Traffic Data for top twenty airports: January 2009 to 2018. *In:* AUSTRALIAN DEPARTMENT OF INFRASTRUCTURE, R. D. A. C. (ed.).
- AUSTRALIAN INDIGENOUS HEALTHINFONET. 2008. Review of the impact of housing and healthrelated infrastructure on Indigenous health [Online].

<u>http://www.healthinfonet.ecu.edu.au/determinants/physical-environment/reviews/our-review</u>. Available: <u>http://www.healthinfonet.ecu.edu.au/determinants/physical-environment/reviews/our-reviews/our-reviews/our-reviews/lacessed March 12 2017]</u>.

- BARLAS, Y. 1989. Multiple tests for validation of system dynamics type of simulation models. *European journal of operational research*, 42, 59-87.
- BERTALANFFY, L. V. 1968. *General system theory: Foundations, development, applications*, George Braziller New York.
- BERTALANFFY, L. V. 1972. The history and status of general systems theory. *Academy of Management Journal (pre-1986)*, 15, 407.
- BERTALANFFY, L. V. & WOODGER, J. H. 1934. *Modern Theories of Development,* Oxford, Oxford University Press.
- BLACK, M. & MCANULTY, J. 2006. The investigation of an outbreak of cryptosporidiosis in New South Wales in 2005. *N S W Public Health Bull*, 17, 76-9.
- BLACKBURN, B. G., MAZUREK, J. M., HLAVSA, M., PARK, J., TILLAPAW, M., PARRISH, M., SALEHI, E., FRANKS, W., KOCH, E. & SMITH, F. 2006. Cryptosporidiosis associated with ozonated apple cider. *Emerg Infect Dis*, 12, 684-686.
- BOEHMER, T. K., ALDEN, N. B., GHOSH, T. S. & VOGT, R. L. 2009. Cryptosporidiosis from a community swimming pool: outbreak investigation and follow-up study. *Epidemiol Infect*, 137, 1651-4.
- BONINI, M., BODINA, A., BONALI, D., BASCUCCI, B., PELLINO, P. & CASTALDI, S. 2011. Investigation and comparison of behaviours of adults and children in swimming pools. *Annali di igiene : medicina preventiva e di comunità*, 23, 319-328.
- BOUCKENOOGHE, A. R., JIANG, Z. D., DE LA CABADA, F. J., ERICSSON, C. D. & DUPONT, H. L. 2002. Enterotoxigenic Escherichia coli as cause of diarrhea among Mexican adults and US travelers in Mexico. *J Travel Med*, 9, 137-40.
- BOUZID, M., HUNTER, P. R., CHALMERS, R. M. & TYLER, K. M. 2013. Cryptosporidium pathogenicity and virulence. *Clin Microbiol Rev*, 26, 115-34.
- BRAUER, F. 2008. Compartmental models in epidemiology. Mathematical epidemiology. Springer.
- BREIEROVA, L. 1997. Generic Structures: Overshoot and Collapse. *In:* MASSACHUSETTS INSTITUTE OF TECHNOLOGY (ed.) *MIT System Dynamics in Education Project.* Boston, USA.
- BRIGGS, A. D. M., WOLSTENHOLME, J., BLAKELY, T. & SCARBOROUGH, P. 2016. Choosing an epidemiological model structure for the economic evaluation of non-communicable disease public health interventions. *Population Health Metrics*, 14, 17.
- BRIGGS, D. 1999. Coping with complexity in environmental health management and policy. *Environmental Health for All.* Springer.
- BRIGGS, D. J., SABEL, C. E. & LEE, K. 2009. Uncertainty in epidemiology and health risk and impact assessment. *Environmental Geochemistry and Health*, 31, 189-203.
- BRISBANE CITY COUNCIL. n.d. *Council Pools* [Online]. Available: <u>https://www.brisbane.qld.gov.au/facilities-recreation/sports-leisure/council-pools</u> [Accessed May 7 2018].
- BROOKHART, M. A., HUBBARD, A. E., VAN DER LAAN, M. J., COLFORD, J. M., JR. & EISENBERG, J. N. 2002. Statistical estimation of parameters in a disease transmission model: analysis of a Cryptosporidium outbreak. *Stat Med*, 21, 3627-38.
- BROUWER, A. F., WEIR, M. H., EISENBERG, M. C., MEZA, R. & EISENBERG, J. N. S. 2017. Doseresponse relationships for environmentally mediated infectious disease transmission models. *PLoS Comput Biol*, 13, e1005481.
- BROWN, J. D. 2004. Knowledge, uncertainty and physical geography: towards the development of methodologies for questioning belief. *Transactions of the Institute of British Geographers*, 29, 367-381.
- BRYDON-MILLER, M., GREENWOOD, D. & MAGUIRE, P. 2003a. Why Action Research? Action Research, 1, 9-28.

- BRYDON-MILLER, M., GREENWOOD, D. & MAGUIRE, P. 2003b. Why action research? : Sage Publications.
- BUSS, B. F., SAFRANEK, T. J., MAGRI, J. M., TÖRÖ, T. J., BEACH, M. J. & FOLEY, B. P. 2009. Association between swimming pool operator certification and reduced pool chemistry violations-Nebraska. 2005-2006. *Journal of Environmental Health*, 71, 36-40.
- CASMAN, E., FISCHHOFF, B., SMALL, M., DOWLATABADI, H., ROSE, J. & MORGAN, M. G. 2001. Climate Change and Cryptosporidiosis: A Qualitative Analysis. *Climatic Change*, 50, 219-249.
- CASMAN, E. A., FISCHHOFF, B., PALMGREN, C., SMALL, M. J. & WU, F. 2000. An integrated risk model of a drinking-water-borne cryptosporidiosis outbreak. *Risk Anal*, 20, 495-511.
- CASTOR, M. L. & BEACH, M. J. 2004. Reducing illness transmission from disinfected recreational water venues: swimming, diarrhea and the emergence of a new public health concern. *Pediatr Infect Dis J*, 23, 866-70.
- CAUSER, L. M., HANDZEL, T., WELCH, P., CARR, M., CULP, D., LUCHT, R., MUDAHAR, K., ROBINSON, D., NEAVEAR, E., FENTON, S., ROSE, C., CRAIG, L., ARROWOOD, M., WAHLQUIST, S., XIAO, L., LEE, Y. M., MIREL, L., LEVY, D., BEACH, M. J., POQUETTE, G. & DWORKIN, M. S. 2006. An outbreak of Cryptosporidium hominis infection at an Illinois recreational waterpark. *Epidemiol Infect*, 134, 147-56.
- CENTERS FOR DISEASE CONTROL AND PREVENTION 1998. Foodborne outbreak of cryptosporidiosis--Spokane, Washington, 1997. *MMWR Morb Mortal Wkly Rep*, 47, 565-7.
- CENTERS FOR DISEASE CONTROL AND PREVENTION 2001. Prevalence of parasites in fecal material from chlorinated swimming pools--United States, 1999. *MMWR Morb Mortal Wkly Rep*, 50, 410-2.
- CENTERS FOR DISEASE CONTROL AND PREVENTION 2012. Promotion of healthy swimming after a statewide outbreak of cryptosporidiosis associated with recreational water venues--Utah, 2008-2009. *MMWR Morb Mortal Wkly Rep,* 61, 348-52.
- CENTERS FOR DISEASE CONTROL AND PREVENTION 2016a. 2016 Model Aquatic Health Code.

CENTERS FOR DISEASE CONTROL AND PREVENTION. 2016b. Laboratory diagnosis of cryptosporidiosis [Online]. Available:

https://www.cdc.gov/dpdx/resources/pdf/benchAids/crypto\_benchaid.pdf [Accessed January 13 2018].

- CHAIDEZ, C., SOTO, M., GORTARES, P. & MENA, K. 2005. Occurrence of Cryptosporidium and Giardia in irrigation water and its impact on the fresh produce industry. *International journal of environmental health research*, 15, 339-345.
- CHALMERS, R. M., CAMPBELL, B. M., CROUCH, N., CHARLETT, A. & DAVIES, A. P. 2011. Comparison of diagnostic sensitivity and specificity of seven Cryptosporidium assays used in the UK. *J Med Microbiol*, 60, 1598-604.
- CHALMERS, R. M. & CASEMORE, D. P. 2004. Epidemiology and Strain Variation of Cryptosporidium. *In:* STERLING, C. R. & ADAM, R. D. (eds.) *The Pathogenic Enteric Protozoa: Giardia, Entamoeba, Cryptosporidium and Cyclospora*. Boston, MA: Springer US.
- CHALMERS, R. M. & DAVIES, A. P. 2010. Minireview: Clinical cryptosporidiosis. *Experimental Parasitology*, 124, 138-146.
- CHAPPELL, C. L., OKHUYSEN, P. C., LANGER-CURRY, R. C., AKIYOSHI, D. E., WIDMER, G. & TZIPORI, S. 2011. Cryptosporidium meleagridis: infectivity in healthy adult volunteers. *Am J Trop Med Hyg*, 85, 238-42.
- CHAPPELL, C. L., OKHUYSEN, P. C., STERLING, C. R., WANG, C., JAKUBOWSKI, W. & DUPONT, H. L. 1999. Infectivity of Cryptosporidium parvum in healthy adults with pre-existing anti-C. parvum serum immunoglobulin G. *Am J Trop Med Hyg*, 60, 157-64.
- CITY OF GOLD COAST. n.d. *Aquatic Centres* [Online]. Available: <u>http://www.goldcoast.qld.gov.au/thegoldcoast/swimming-aquatic-centres-966.html</u> [Accessed May 7 2018].
- COFFEY, R., CUMMINS, E., CORMICAN, M., FLAHERTY, V. O. & KELLY, S. 2007. Microbial exposure assessment of waterborne pathogens. *Human and Ecological Risk Assessment*, 13, 1313-1351.
- COMMITTEE ON DECISION MAKING UNDER UNCERTAINTY, BOARD ON POPULATION HEALTH AND PUBLIC HEALTH PRACTICE & INSTITUTE OF MEDICINE 2013. Environmental Decisions in the Face of Uncertainty. Washington DC.
- CONRAD, C. C., STANFORD, K., NARVAEZ-BRAVO, C., CALLAWAY, T. & MCALLISTER, T. 2016. Farm Fairs and Petting Zoos: A Review of Animal Contact as a Source of Zoonotic Enteric Disease. *Foodborne Pathog Dis.*
- COPE, J. R., PROSSER, A., NOWICKI, S., ROBERTS, M. W., ROBERTS, J. M., SCHEER, D., ANDERSON, C., LONGSWORTH, A., PARSONS, C., GOLDSCHMIDT, D., JOHNSTON, S., BISHOP, H., XIAO, L., HILL, V., BEACH, M. & HLAVSA, M. C. 2015. Preventing communitywide transmission of Cryptosporidium: a proactive public health response to a swimming poolassociated outbreak--Auglaize County, Ohio, USA. *Epidemiol Infect*, 143, 3459-67.
- CORDELL, R. L. & ADDISS, D. G. 1994. Cryptosporidiosis in child care settings: a review of the literature and recommendations for prevention and control. *Pediatr Infect Dis J*, 13, 310-7.
- COSTICH, J. F. & PATTON, D. 2013. Enforcement: Linking Policy and Impact in Public Health. *Jurimetrics*, 53, 293-305.
- CRESSEY, P. & LAKE, R. 2011. Estimated incidence of foodborne illness in New Zealand: Application of overseas models and multipliers. *Christchurch, New Zealand: New Zealand Government*.
- CRUICKSHANK, R., ASHDOWN, L. & CROESE, J. 1988. Human cryptosporidiosis in North Queensland. *Aust N Z J Med*, 18, 582-6.
- CURRIE, D. J., SMITH, C. & JAGALS, P. 2018. The application of system dynamics modelling to environmental health decision-making and policy a scoping review. *BMC Public Health*, 18, 402.
- DALE, K., KIRK, M., SINCLAIR, M., HALL, R. & LEDER, K. 2010. Reported waterborne outbreaks of gastrointestinal disease in Australia are predominantly associated with recreational exposure. *Australian and New Zealand Journal of Public Health*, 34, 527-530.
- DESAI, N. T., SARKAR, R. & KANG, G. 2012. Cryptosporidiosis: An under-recognized public health problem. *Trop Parasitol*, 2, 91-8.
- DIAZ, R., BEHR, J., JENG, A., LU, H. & LONGO, F. Analyzing the effects of policy options to mitigate the effect of sea level rise on the public health and medically fragile population: A system dynamics approach. Emerging M and S Applications in Industry and Academia Symposium 2012, EAIA 2012 2012 Spring Simulation Multiconference, 2012. 47-54.
- DIAZ, R., BEHR, J. & TULPULE, M. Energy portfolio simulation considering environmental and public health impacts. Emerging M and S Applications in Industry and Academia Symposium 2011, EAIA 2011 - 2011 Spring Simulation Multiconference, 2011. 38-45.
- DIEMERT, D. J. 2006. Prevention and self-treatment of traveler's diarrhea. Clin Microbiol Rev, 19, 583-94.
- DIETZ, V. J. & ROBERTS, J. M. 2000. National surveillance for infection with Cryptosporidium parvum, 1995-1998: what have we learned? *Public Health Rep*, 115, 358-63.
- DIXON, B., PARRINGTON, L., COOK, A., POLLARI, F. & FARBER, J. 2013. Detection of Cyclospora, Cryptosporidium, and Giardia in ready-to-eat packaged leafy greens in Ontario, Canada. *J Food Prot*, 76, 307-13.
- DOMJAHN, B. T., HLAVSA, M. C., ANDERSON, B., SCHULKIN, J., LEON, J. & JONES, J. L. 2014. A survey of U.S. obstetrician-gynecologists' clinical and epidemiological knowledge of cryptosporidiosis in pregnancy. *Zoonoses Public Health*, 61, 356-63.
- DONOVAN, G. H. & BROWN, T. C. 2007. Be careful what you wish for: the legacy of Smokey Bear. *Frontiers in Ecology and the Environment*, 5, 73-79.
- DUFOUR, A. P., EVANS, O., BEHYMER, T. D. & CANTU, R. 2006. Water ingestion during swimming activities in a pool: a pilot study. *J Water Health*, 4, 425-30.
- DUPONT, H. L., CHAPPELL, C. L., STERLING, C. R., OKHUYSEN, P. C., ROSE, J. B. & JAKUBOWSKI, W. 1995. The infectivity of Cryptosporidium parvum in healthy volunteers. *N Engl J Med*, 332, 855-9.
- EISENBERG, J. N., DESAI, M. A., LEVY, K., BATES, S. J., LIANG, S., NAUMOFF, K. & SCOTT, J. C. 2007. Environmental determinants of infectious disease: a framework for tracking causal links and guiding public health research. *Environ Health Perspect*, 115, 1216-23.
- EISENBERG, J. N. S., LEI, X., HUBBARD, A. H., BROOKHART, M. A. & COLFORD, J. M. 2005. The Role of Disease Transmission and Conferred Immunity in Outbreaks: Analysis of the 1993 Cryptosporidium Outbreak in Milwaukee, Wisconsin. *American Journal of Epidemiology*, 161, 62-72.
- ERDOZAIN, G., KUKANICH, K., CHAPMAN, B. & POWELL, D. 2013. Observation of public health risk behaviours, risk communication and hand hygiene at Kansas and Missouri petting zoos--2010-2011. *Zoonoses Public Health*, 60, 304-10.

- FEINGOLD, B. J., VEGOSEN, L., DAVIS, M., LEIBLER, J., PETERSON, A. & SILBERGELD, E. K. 2010. A niche for infectious disease in environmental health: rethinking the toxicological paradigm. *Environ Health Perspect*, 118, 1165-72.
- FEWTRELL, L. & BARTRAM, J. 2001. Water quality: guidelines, standards and health: assessment of risk and risk management for water-related infectious diseases.
- FORD, A. 2009. Modeling the Environment, Second Edition, Island Press.
- FORRESTER, J. W. 1958. Industrial Dynamics A Major Breakthrough for Decision Makers. *Harvard Business Review*, 36, 37-66.
- FORRESTER, J. W. 1968. Principles of Systems, Waltham, MA., Pegasus Communications.
- FOX, L. M. & SARAVOLATZ, L. D. 2005. Nitazoxanide: a new thiazolide antiparasitic agent. *Clin Infect Dis*, 40, 1173-80.
- FROST, F. J., CALDERON, R. L., MULLER, T. B., CURRY, M., RODMAN, J. S., MOSS, D. M. & DE LA CRUZ, A. A. 1998. A two-year follow-up survey of antibody to Cryptosporidium in Jackson County, Oregon following an outbreak of waterborne disease. *Epidemiol Infect*, 121, 213-7.
- GALLE, F., DALLOLIO, L., MAROTTA, M., RAGGI, A., DI ONOFRIO, V., LIGUORI, G., TONI, F. & LEONI, E. 2016. Health-Related Behaviors in Swimming Pool Users: Influence of Knowledge of Regulations and Awareness of Health Risks. *Int J Environ Res Public Health*, 13.
- GARRO, C. J., MORICI, G. E., UTGÉS, M. E., TOMAZIC, M. L. & SCHNITTGER, L. 2016. Prevalence and risk factors for shedding of Cryptosporidium spp. oocysts in dairy calves of Buenos Aires Province, Argentina. *Parasite Epidemiology and Control*, 1, 36-41.
- GERBA, C. P. 2000. Assessment of Enteric Pathogen Shedding by Bathers during Recreational Activity and its Impact on Water Quality. *Quantitative Microbiology*, 2, 55-68.
- GILOVICH, T. 1991. *How we know what isn't so: The Fallibility of Human Reason in Everyday Life*, New York, USA, Free Press.
- GOH, S., REACHER, M., CASEMORE, D. P., VERLANDER, N. Q., CHALMERS, R., KNOWLES, M., WILLIAMS, J., OSBORN, K. & RICHARDS, S. 2004. Sporadic cryptosporidiosis, North Cumbria, England, 1996-2000. *Emerg Infect Dis*, 10, 1007-15.
- GOMES, M. F. C., PASTORE Y PIONTTI, A., ROSSI, L., CHAO, D., LONGINI, I., HALLORAN, M. E.
   & VESPIGNANI, A. 2014. Assessing the International Spreading Risk Associated with the 2014 West African Ebola Outbreak. *PLoS Currents*, 6, ecurrents.outbreaks.cd818f63d40e24aef769dda7df9e0da5.
- GOVERNMENT OF AUSTRALIA 2005. Communicable diseases surveillance. Highlights for 2nd quarter, 2005. Commun Dis Intell Q Rep, 29, 316-35.
- GOVERNMENT OF AUSTRALIA 2013. NNDSS Fortnightly summary notes 2013 *In:* DEPARTMENT OF HEALTH (ed.).
- GOVERNMENT OF AUSTRALIA 2016. Number of notifications of Cryptosporidiosis, received from State and Territory health authorities in the period of 1991 to 2015 and year-to-date notifications for 2016. *National Notifiable Diseases Surveillance System,*. 14 September, 2016 ed.: Government of Australia.
- GRACZYK, T. K., GRIMES, B. H., KNIGHT, R., DA SILVA, A. J., PIENIAZEK, N. J. & VEAL, D. A. 2003. Detection of Cryptosporidium parvum and Giardia lamblia carried by synanthropic flies by combined fluorescent in situ hybridization and a monoclonal antibody. *The American journal of tropical medicine and hygiene*, 68, 228-232.
- HALL, A. D. & FAGEN, R. E. 1956. Definition of system. General systems, 1, 18-28.
- HAQUE, R., ROY, S., SIDDIQUE, A., MONDAL, U., RAHMAN, S. M., MONDAL, D., HOUPT, E. & PETRI, W. A., JR. 2007. Multiplex real-time PCR assay for detection of Entamoeba histolytica, Giardia intestinalis, and Cryptosporidium spp. *Am J Trop Med Hyg*, 76, 713-7.
- HARPER, C. M., COWELL, N. A., ADAMS, B. C., LANGLEY, A. J. & WOHLSEN, T. D. 2002. Outbreak of Cryptosporidium linked to drinking unpasteurised milk. *Commun Dis Intell Q Rep*, 26, 449-50.
- HASHIM, A., MULCAHY, G., BOURKE, B. & CLYNE, M. 2006. Interaction of Cryptosporidium hominis and Cryptosporidium parvum with primary human and bovine intestinal cells. *Infect Immun*, 74, 99-107.
- HAVELAAR, A. H., GALINDO, A. V., KUROWICKA, D. & COOKE, R. M. 2008. Attribution of foodborne pathogens using structured expert elicitation. *Foodborne pathogens and disease*, 5, 649-659.
- HEALTH PROTECTION NSW 2013. Public swimming pool and spa pool advisory document. Sydney, Australia: NSW Ministry of Health.

- HEIJBEL, H., SLAINE, K., SEIGEL, B., WALL, P., MCNABB, S. J., GIBBONS, W. & ISTRE, G. R. 1987. Outbreak of diarrhea in a day care center with spread to household members: the role of Cryptosporidium. *Pediatr Infect Dis J*, 6, 532-5.
- HEKIMOGLU, M. & BARLAS, Y. 2010. Sensitivity Analysis of System Dynamics Models by Behavior Pattern Measures.
- HELLARD, M. E., SINCLAIR, M. I., FAIRLEY, C. K., ANDREWS, R. M., BAILEY, M., BLACK, J., DHARMAGE, S. C. & KIRK, M. D. 2000a. An outbreak of cryptosporidiosis in an urban swimming pool: why are such outbreaks difficult to detect? *Aust N Z J Public Health*, 24, 272-5.
- HELLARD, M. E., SINCLAIR, M. I., HOGG, G. G. & FAIRLEY, C. K. 2000b. Prevalence of enteric pathogens among community based asymptomatic individuals. *J Gastroenterol Hepatol*, 15, 290-3.
- HENNESSY, T. W., MARCUS, R., DENEEN, V., REDDY, S., VUGIA, D., TOWNES, J., BARDSLEY,
   M., SWERDLOW, D. & ANGULO, F. J. 2004. Survey of physician diagnostic practices for patients with acute diarrhea: clinical and public health implications. *Clin Infect Dis*, 38 Suppl 3, S203-11.
- HITCHINS, D. 2009. What are the general principles applicable to systems? Insight, 12, 59-63.
- HOCHBERG, M. E. 1991. Non-linear transmission rates and the dynamics of infectious disease. *Journal of theoretical biology*, 153, 301-321.
- HOMER, J. B. & HIRSCH, G. B. 2006. System dynamics modeling for public health: Background and opportunities. *American Journal of Public Health*, 96, 452-458.
- HOPPER, M. & STAVE, K. A. Assessing the effectiveness of systems thinking interventions in the classroom. Proceedings of the 26th International Conference of, 2008.
- HU, W., MENGERSEN, K. & TONG, S. 2010. Risk factor analysis and spatiotemporal CART model of cryptosporidiosis in Queensland, Australia. *BMC Infect Dis*, 10, 311.
- HUNTER, P. R. & FEWTRELL, L. 2001. Acceptable risk. *Water Quality: Guidelines, Standards and Health. Risk assessment and management for water-related infectious disease. London: IWA Publishing*, 207-227.
- HUNTER, P. R., HUGHES, S., WOODHOUSE, S., NICHOLAS, R., SYED, Q., CHALMERS, R. M., VERLANDER, N. Q. & GOODACRE, J. 2004a. Health Sequelae of Human Cryptosporidiosis in Immunocompetent Patients. *Clinical Infectious Diseases*, 39, 504-510.
- HUNTER, P. R., HUGHES, S., WOODHOUSE, S., SYED, Q., VERLANDER, N. Q., CHALMERS, R. M., MORGAN, K., NICHOLS, G., BEECHING, N. & OSBORN, K. 2004b. Sporadic cryptosporidiosis case-control study with genotyping. *Emerg Infect Dis*, 10, 1241-9.
- HUNTER, P. R. & NICHOLS, G. 2002. Epidemiology and Clinical Features of Cryptosporidium Infection in Immunocompromised Patients. *Clinical Microbiology Reviews*, 15, 145-154.
- INSTITUTE OF MEDICINE 2009. Environmental Health Sciences Decision Making: Risk Management, Evidence, and Ethics: Workshop Summary. Washington, DC: The National Academies Press.
- JAGAI, J. S., CASTRONOVO, D. A., MONCHAK, J. & NAUMOVA, E. N. 2009. Seasonality of cryptosporidiosis: A meta-analysis approach. *Environ Res*, 109, 465-78.
- JELINEK, T., LOTZE, M., EICHENLAUB, S., LÖSCHER, T. & NOTHDURFT, H. D. 1997. Prevalence of infection with Cryptosporidium parvumand Cyclospora cayetanensis among international travellers. *Gut*, 41, 801-804.
- JOHANSEN, O. H., HANEVIK, K., THRANA, F., CARLSON, A., STACHURSKA-HAGEN, T., SKAARE, D. & ROBERTSON, L. J. 2015. Symptomatic and asymptomatic secondary transmission of Cryptosporidium parvum following two related outbreaks in schoolchildren. *Epidemiol Infect*, 143, 1702-9.
- JOHNSON, D. C., ENRIQUEZ, C. E., PEPPER, I. L., DAVIS, T. L., GERBA, C. P. & ROSE, J. B. 1997. Survival of giardia, cryptosporidium, poliovirus and salmonella in marine waters. *Water Science and Technology*, 35, 261.
- JOKIPII, L. & JOKIPII, A. M. M. 1986. Timing of Symptoms and Oocyst Excretion in Human Cryptosporidiosis. *New England Journal of Medicine*, 315, 1643-1647.
- KARANIS, P., KOURENTI, C. & SMITH, H. 2007. Waterborne transmission of protozoan parasites: a worldwide review of outbreaks and lessons learnt. *J Water Health*, 5, 1-38.
- KENDALL, M. E., CRIM, S., FULLERTON, K., HAN, P. V., CRONQUIST, A. B., SHIFERAW, B., INGRAM, L. A., ROUNDS, J., MINTZ, E. D. & MAHON, B. E. 2012. Travel-associated enteric infections diagnosed after return to the United States, Foodborne Diseases Active Surveillance Network (FoodNet), 2004-2009. *Clin Infect Dis*, 54 Suppl 5, S480-7.
- KENT, L., MCPHERSON, M. & HIGGINS, N. 2015. A positive association between cryptosporidiosis notifications and ambient temperature, Victoria, Australia, 2001-2009. *J Water Health*, 13, 1039-47.

KIM, D. H. 1995. Systems archetypes as dynamic theories. The Systems Thinker, 6, 6-9.

- KIRK, M., GLASS, K., FORD, L., BROWN, K. & HALL, G. 2014. Foodborne illness in Australia: Annual incidence circa 2010. Canberra: Commonwealth of Australia.
- KOENIG, K. L., ALMADHYAN, A. & BURNS, M. J. 2016. Identify-Isolate-Inform: A Tool for Initial Detection and Management of Zika Virus Patients in the Emergency Department. *West J Emerg Med*, 17, 238-44.
- KOH, W., CLODE, P. L., MONIS, P. & THOMPSON, R. C. 2013. Multiplication of the waterborne pathogen Cryptosporidium parvum in an aquatic biofilm system. *Parasit Vectors*, 6, 270.
- KOLB, D. A. 1984. *Experiential learning: experience as the source of learning and development*, Englewood Cliffs, NJ, Prentice Hall.
- KREUTER, M. W., DE ROSA, C., HOWZE, E. H. & BALDWIN, G. T. 2004. Understanding wicked problems: a key to advancing environmental health promotion. *Health Educ Behav*, 31, 441-54.
- KRIEGER, N. 2008. Proximal, distal, and the politics of causation: what's level got to do with it? *American journal of public health,* 98, 221-230.
- LABONTE, R. & ROBERTSON, A. 1996. Delivering the goods, showing our stuff: The case for a constructivist paradigm for health promotion research and practice. *Health Education & Behavior*, 23, 431-447.
- LADYMAN, J., LAMBERT, J. & WIESNER, K. 2013. What is a complex system? *European Journal for Philosophy of Science*, **3**, 33-67.
- LAL, A., CORNISH, L. M., FEARNLEY, E., GLASS, K. & KIRK, M. 2015. Cryptosporidiosis: A Disease of Tropical and Remote Areas in Australia. *PLoS Negl Trop Dis*, 9, e0004078.
- LAM, S., SIVARAMALINGAM, B. & GANGODAWILAGE, H. 2014. Cryptosporidium outbreaks associated with swimming pools. *Environmental Health Review*, 57, 3-8.
- LANE, D. 2014. Modelling the foodborne transmission mechanisms for norovirus. London: Food Standards Agency.
- LAUPLAND, K. B. & CHURCH, D. L. 2005. Population-based laboratory surveillance for Giardia sp. and Cryptosporidium sp. infections in a large Canadian health region. *BMC Infectious Diseases*, 5, 72.
- LEARMONTH, J., IONAS, G., PITA, A. & COWIE, R. 2001. Seasonal shift in Cryptosporidium parvum transmission cycles in New Zealand. *J Eukaryot Microbiol*, Suppl, 34s-35s.
- LEARMONTH, J. J., IONAS, G., EBBETT, K. A. & KWAN, E. S. 2004. Genetic characterization and transmission cycles of Cryptosporidium species isolated from humans in New Zealand. *Appl Environ Microbiol*, 70, 3973-8.
- LEITCH, G. J. & HE, Q. 2012. Cryptosporidiosis-an overview. J Biomed Res, 25, 1-16.
- LEMMON, J. M., MCANULTY, J. M. & BAWDEN-SMITH, J. 1996. Outbreak of cryptosporidiosis linked to an indoor swimming pool. *Med J Aust*, 165, 613-6.
- LEWIN, K. 1951. Field theory in social science: selected theoretical papers. New York: Harper.
- LEWIS, L., CHEW, J., WOODLEY, I., COLBOURNE, J. & POND, K. 2015. The application of computational fluid dynamics and small-scale physical models to assess the effects of operational practices on the risk to public health within large indoor swimming pools. *J Water Health*, 13, 939-52.
- LIGUORI, G., CASTALDI, S., SIGNORELLI, C., AUXILIA, F., ALFANO, V., SACCANI, E., VISCIANO, A., FANTI, M., SPINELLI, A. & PASQUARELLA, C. 2007. Hygienic risks in swimming pool: knowledge and behaviours of consumers of three structures in Crema, Parma and Naples. *Annali di igiene : medicina preventiva e di comunità*, 19, 325-335.
- LIU, J., GRATZ, J., AMOUR, C., KIBIKI, G., BECKER, S., JANAKI, L., VERWEIJ, J. J., TANIUCHI, M., SOBUZ, S. U., HAQUE, R., HAVERSTICK, D. M. & HOUPT, E. R. 2013. A laboratory-developed TaqMan Array Card for simultaneous detection of 19 enteropathogens. *J Clin Microbiol*, 51, 472-80.
- LOUIE, K., GUSTAFSON, L., GILL, I., LAURA & MACDOUGALL 2004. An outbreak of Cryptosporidium parvum in a Surrey pool with detection in pool water. *Canada Communicable Disease Report*, 60, 61-66.
- LUCIO-FORSTER, A., GRIFFITHS, J. K., CAMA, V. A., XIAO, L. & BOWMAN, D. D. 2010. Minimal zoonotic risk of cryptosporidiosis from pet dogs and cats. *Trends Parasitol*, 26, 174-9.
- MAANI, K. E. & CAVANA, R. Y. 2007. Systems Thinking, System Dynamics: Managing Change and Complexity, Pearson Education New Zealand.
- MAANI, K. E. & MAHARAJ, V. Systemic Thinking and Complex Problem Solving. A Theory Building Empirical Study. Proceedings of the 19 th International Conference of the System Dynamics Society 2001, 2001. 101.

- MACKENZIE, W. R., KAZMIERCZAK, J. J. & DAVIS, J. P. 1995a. An outbreak of cryptosporidiosis associated with a resort swimming pool. *Epidemiology & Infection*, 115, 545-553.
- MACKENZIE, W. R., SCHELL, W. L., BLAIR, K. A., ADDISS, D. G., PETERSON, D. E., HOXIE, N. J., KAZMIERCZAK, J. J. & DAVIS, J. P. 1995b. Massive outbreak of waterborne cryptosporidium infection in Milwaukee, Wisconsin: recurrence of illness and risk of secondary transmission. *Clin Infect Dis*, 21, 57-62.
- MAJOWICZ, S. E., EDGE, V. L., FAZIL, A., MCNAB, W. B., DORE, K. A., SOCKETT, P. N., FLINT, J. A., MIDDLETON, D., MCEWEN, S. A. & WILSON, J. B. 2005. Estimating the under-reporting rate for infectious gastrointestinal illness in Ontario. *Can J Public Health*, 96, 178-81.
- MALCOLM, D. G., ROSEBOOM, J. H., CLARK, C. E. & FAZAR, W. 1959. Application of a technique for research and development program evaluation. *Operations research*, 7, 646-669.
- MANHEIM, D., CHAMBERLIN, M., OSOBA, O. A., VARDAVAS, R. & MOORE, M. 2016. Decision Support Using Models. *Improving Decision Support for Infectious Disease Prevention and Control*. RAND Corporation.
- MANSOURI, M. & LOCKYER, J. 2007. A meta-analysis of continuing medical education effectiveness. J Contin Educ Health Prof, 27, 6-15.
- MAYNE, D. J., RESSLER, K. A., SMITH, D., HOCKEY, G., BOTHAM, S. J. & FERSON, M. J. 2011. A community outbreak of cryptosporidiosis in sydney associated with a public swimming facility: a case-control study. *Interdiscip Perspect Infect Dis*, 2011, 341065.
- MCCANN, R., JONES, R., SNOW, J., CLEARY, P., BURGESS, S., BOTHRA, V. & CHALMERS, R. M. 2014. An outbreak of cryptosporidiosis at a swimming club--can rapid field epidemiology limit the spread of illness? *Epidemiol Infect*, 142, 51-5.
- MCCLAIN, J., BERNHARDT, J. M. & BEACH, M. J. 2005. Assessing parents' perception of children's risk for recreational water illnesses. *Emerg Infect Dis*, 11, 670-6.
- MCKERR, C., ADAK, G. K., NICHOLS, G., GORTON, R., CHALMERS, R. M., KAFATOS, G., COSFORD, P., CHARLETT, A., REACHER, M., POLLOCK, K. G., ALEXANDER, C. L. & MORTON, S. 2015. An Outbreak of Cryptosporidium parvum across England & Scotland Associated with Consumption of Fresh Pre-Cut Salad Leaves, May 2012. *PLoS One*, 10, e0125955.
- MCKNIGHT, U. S. & FINKEL, M. 2013. A system dynamics model for the screening-level long-term assessment of human health risks at contaminated sites. *Environmental Modelling & Software*, 40, 35-50.
- MCLAUCHLIN, J., PEDRAZA-DIAZ, S., AMAR-HOETZENEDER, C. & NICHOLS, G. L. 1999. Genetic characterization of Cryptosporidium strains from 218 patients with diarrhea diagnosed as having sporadic cryptosporidiosis. *J Clin Microbiol*, 37, 3153-8.
- MCMILLIAN, M., DUNN, J. R., KEEN, J. E., BRADY, K. L. & JONES, T. F. 2007. Risk behaviors for disease transmission among petting zoo attendees. *J Am Vet Med Assoc*, 231, 1036-8.
- MEADOWS, D. 1997. Places to Intervene in a System. Whole Earth, 91, 78-84.
- MEISEL, J. L., PERERA, D. R., MELIGRO, C. & RUBIN, C. E. 1976. Overwhelming watery diarrhea associated with a cryptosporidium in an immunosuppressed patient. *Gastroenterology*, 70, 1156-60.
- MESSNER, M. J., CHAPPELL, C. L. & OKHUYSEN, P. C. 2001. Risk assessment for Cryptosporidium: a hierarchical Bayesian analysis of human dose response data. *Water Res,* 35, 3934-40.
- MILLARD, P. S., GENSHEIMER, K. F., ADDISS, D. G., SOSIN, D. M., BECKETT, G. A., HOUCK-JANKOSKI, A. & HUDSON, A. 1994. An outbreak of cryptosporidiosis from fresh-pressed apple cider. *Jama*, 272, 1592-1596.
- MOORE, H. & BLYTH, C. 2018. Optimising the use of linked administrative data for infectious diseases research in Australia. *Public Health Research & Practice*.
- MORIN, C. A., ROBERTS, C. L., MSHAR, P. A., ADDISS, D. G. & HADLER, J. L. 1997. What do physicians know about cryptosporidiosis? A survey of Connecticut physicians. *Arch Intern Med*, 157, 1017-22.
- MORROW, A. L., TOWNSEND, I. T. & PICKERING, L. K. 1991. Risk of enteric infection associated with child day care. *Pediatr Ann*, 20, 427-33.
- MOSIER, D. A. & OBERST, R. D. 2000. Cryptosporidiosis. A global challenge. *Ann N Y Acad Sci*, 916, 102-11.
- NAUMOVA, E. N., CHRISTODOULEAS, J., HUNTER, P. R. & SYED, Q. 2005. Effect of precipitation on seasonal variability in cryptosporidiosis recorded by the North West England surveillance system in 1990-1999. *J Water Health*, 3, 185-96.

- NAUMOVA, E. N., JAGAI, J. S., MATYAS, B., DEMARIA, A., JR., MACNEILL, I. B. & GRIFFITHS, J. K. 2007. Seasonality in six enterically transmitted diseases and ambient temperature. *Epidemiol Infect*, 135, 281-92.
- NETT, R. J., TOBLIN, R., SHEEHAN, A., HUANG, W. T., BAUGHMAN, A. & CARTER, K. 2010. Nonhygienic behavior, knowledge, and attitudes among interactive splash park visitors. *J Environ Health*, 73, 8-14.
- NG-HUBLIN, J. S., HARGRAVE, D., COMBS, B. & RYAN, U. 2015. Investigation of a swimming poolassociated cryptosporidiosis outbreak in the Kimberley region of Western Australia. *Epidemiol Infect*, 143, 1037-41.
- NG, J., EASTWOOD, K., DURRHEIM, D., MASSEY, P., WALKER, B., ARMSON, A. & RYAN, U. 2008. Evidence supporting zoonotic transmission of Cryptosporidium in rural New South Wales. *Exp Parasitol*, 119, 192-5.
- NHMRC 2008. Guidelines for Managing Risks in Recreational Water. Canberra: Australian Government.
- OKHMATOVSKAIA, A., VERMA, A. D., BARBEAU, B., CARRIERE, A., PASQUET, R. & BUCKERIDGE, D. L. 2010. A simulation model of waterborne gastro-intestinal disease outbreaks: description and initial evaluation. AMIA Annu Symp Proc, 2010, 557-61.
- OKHUYSEN, P. C., CHAPPELL, C. L., STERLING, C. R., JAKUBOWSKI, W. & DUPONT, H. L. 1998. Susceptibility and serologic response of healthy adults to reinfection with Cryptosporidium parvum. *Infect Immun*, 66, 441-3.
- ORGANIZATION., W. H. 2002. Protozoan Parasites (Cryptosporidium, Giardia, Cyclospora). *Guidelines* For Drinking-Water Quality, 2nd ed. Geneva: World Helath Organization.
- OSEWE, P., ADDISS, D. G., BLAIR, K. A., HIGHTOWER, A., KAMB, M. L. & DAVIS, J. P. 1996. Cryptosporidiosis in Wisconsin: a case-control study of post-outbreak transmission. *Epidemiol Infect*, 117, 297-304.
- OZFOODNET WORKING GROUP 2003. Foodborne disease in Australia: incidence, notifications and outbreaks. Annual report of the OzFoodNet network, 2002. *Communicable diseases intelligence quarterly report*, 27, 209.
- PATERSON, J. & GOLDTHORPE, I. 2006. Managing a cluster of cryptosporidiosis associated with a public swimming pool. *N S W Public Health Bull*, 17, 80.
- PERZ, J. F., ENNEVER, F. K. & LE BLANCQ, S. M. 1998. Cryptosporidium in tap water: comparison of predicted risks with observed levels of disease. *Am J Epidemiol*, 147, 289-301.
- PETTOELLO-MANTOVANI, M., DI MARTINO, L., DETTORI, G., VAJRO, P., SCOTTI, S., DITULLIO, M. T. & GUANDALINI, S. 1995. Asymptomatic carriage of intestinal Cryptosporidium in immunocompetent and immunodeficient children: a prospective study. *Pediatr Infect Dis J*, 14, 1042-7.
- PINTAR, K. D., FAZIL, A., POLLARI, F., CHARRON, D. F., WALTNER-TOEWS, D. & MCEWEN, S. A. 2010. A risk assessment model to evaluate the role of fecal contamination in recreational water on the incidence of cryptosporidiosis at the community level in Ontario. *Risk Anal*, 30, 49-64.
- PLATE, R. & MONROE, M. 2014. A structure for assessing systems thinking. *The 2014 Creative Learning Exchange*, 26, 1-12.
- POLAGE, C. R., STODDARD, G. J., ROLFS, R. T. & PETTI, C. A. 2011. Physician use of parasite tests in the United States from 1997 to 2006 and in a Utah Cryptosporidium outbreak in 2007. *J Clin Microbiol*, 49, 591-6.
- PREISER, G., PREISER, L. & MADEO, L. 2003. An Outbreak of Cryptosporidiosis Among Veterinary Science Students Who Work With Calves. *Journal of American College Health*, 51, 213-215.
- PRÜSS-ÜRSÜN, A. & CORVALÁN, C. 2006. Preventing disease through healthy environments. Towards an estimate of the environmental burden of disease. SciELO Brasil.
- PRÜSS-ÜSTÜN, A. & NEIRA, M. 2016. Preventing disease through healthy environments: a global assessment of the burden of disease from environmental risks, World Health Organization.
- PUBLIC HEALTH LABORATORY NETWORK. 2017. Cryptosporidiosis Laboratory Case Definition (LCD) [Online]. Available: <u>http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-phlncd-cryptosporidiosis.htm</u> [Accessed February 5 2018].
- PUECH, M. C., MCANULTY, J. M., LESJAK, M., SHAW, N., HERON, L. & WATSON, J. M. 2001. A statewide outbreak of cryptosporidiosis in New South Wales associated with swimming at public pools. *Epidemiology & Infection*, 126, 389-396.
- QUEENSLAND GOVERNMENT STATISTICIAN'S OFFICE 2015. Population growth highlights and trends, Queensland regions, 2015 edition. *In:* TREASURY, Q. (ed.). Queensland, Australia.

- QUEENSLAND HEALTH 2004. Queensland Health Swimming and Spa Pool Water Quality and Operational Guidelines. Queensland, Australia: Queensland Government.
- QUEENSLAND HEALTH 2015. Cryptosporidiosis Queensland Health Guidelines for Public Health Units. *In:* STATE OF QUEENSLAND (ed.).
- QUIROZ, E. S., BERN, C., MACARTHUR, J. R., XIAO, L., FLETCHER, M., ARROWOOD, M. J., SHAY, D. K., LEVY, M. E., GLASS, R. I. & LAL, A. 2000. An outbreak of cryptosporidiosis linked to a foodhandler. *Journal of Infectious Diseases*, 181, 695-700.
- REASON, P. & BRADBURY, H. 2001. *Handbook of action research: Participative inquiry and practice*, Sage.
- REUTER, H., JOPP, F., BLANCO-MORENO, J. M., DAMGAARD, C., MATSINOS, Y. & DEANGELIS, D. L. 2010. Ecological hierarchies and self-organisation – Pattern analysis, modelling and process integration across scales. *Basic and Applied Ecology*, 11, 572-581.
- RICHARDSON, G. P. 2011. Reflections on the foundations of system dynamics. *System Dynamics Review*, 27, 219-243.
- RICHMOND, B. 1994. Systems thinking/system dynamics: Let's just get on with it. *System Dynamics Review*, 10, 135-157.
- RNA. 2016. 2016 RNA Annual Report [Online]. Available: <u>https://www.rna.org.au/media/881637/2016%20rna%20annual%20report.pdf</u> [Accessed May 7 2018].
- ROBERTSON, B., SINCLAIR, M. I., FORBES, A. B., VEITCH, M., KIRK, M., CUNLIFFE, D., WILLIS, J. & FAIRLEY, C. K. 2002. Case-control studies of sporadic cryptosporidiosis in Melbourne and Adelaide, Australia. *Epidemiol Infect*, 128, 419-31.
- ROBERTSON, L. J., CAMPBELL, A. T. & SMITH, H. V. 1992. Survival of Cryptosporidium parvum oocysts under various environmental pressures. *Applied and Environmental Microbiology*, 58, 3494-3500.
- ROBERTSON, L. J. & CHALMERS, R. M. 2013. Foodborne cryptosporidiosis: is there really more in Nordic countries? *Trends in Parasitology*, 29, 3-9.
- ROBINSON, R. A. & PUGH, R. N. 2002. Dogs, zoonoses and immunosuppression. *J R Soc Promot Health*, 122, 95-8.
- ROSE, J. B., HUFFMAN, D. E. & GENNACCARO, A. 2002. Risk and control of waterborne cryptosporidiosis. *FEMS Microbiol Rev*, 26, 113-23.
- ROSSLE, N. F. & LATIF, B. 2013. Cryptosporidiosis as threatening health problem: A review. Asian *Pacific Journal of Tropical Biomedicine*, 3, 916-924.
- ROY, S. L., DELONG, S. M., STENZEL, S. A., SHIFERAW, B., ROBERTS, J. M., KHALAKDINA, A., MARCUS, R., SEGLER, S. D., SHAH, D. D., THOMAS, S., VUGIA, D. J., ZANSKY, S. M., DIETZ, V. & BEACH, M. J. 2004. Risk factors for sporadic cryptosporidiosis among immunocompetent persons in the United States from 1999 to 2001. *J Clin Microbiol*, 42, 2944-51.
- RYAN, U., LAWLER, S. & REID, S. 2017. Limiting swimming pool outbreaks of cryptosporidiosis the roles of regulations, staff, patrons and research. *J Water Health*, 15, 1-16.
- RYU, H., ALUM, A., MENA, K. D. & ABBASZADEGAN, M. 2007. Assessment of the risk of infection by Cryptosporidium and Giardia in non-potable reclaimed water. *Water Sci Technol*, 55, 283-90.
- SAVIGNY, D. D., ADAM, TAGHREED (ed.) 2009. Systems Thinking for Health Systems Strengthening: World Health Organization
- SAYERS, G. M., DILLON, M. C., CONNOLLY, E., THORNTON, L., HYLAND, E., LOUGHMAN, E., O'MAHONY, M. A. & BUTLER, K. M. 1996. Cryptosporidiosis in children who visited an open farm. *Commun Dis Rep CDR Rev*, 6, R140-4.
- SCALLAN, E., HOEKSTRA, R. M., ANGULO, F. J., TAUXE, R. V., WIDDOWSON, M. A., ROY, S. L., JONES, J. L. & GRIFFIN, P. M. 2011. Foodborne illness acquired in the United States--major pathogens. *Emerg Infect Dis*, 17, 7-15.
- SCALLAN, E., JONES, T. F., CRONQUIST, A., THOMAS, S., FRENZEN, P., HOEFER, D., MEDUS, C.
   & ANGULO, F. J. 2006. Factors associated with seeking medical care and submitting a stool sample in estimating the burden of foodborne illness. *Foodborne Pathog Dis*, 3, 432-8.
- SCALLAN, E., MAJOWICZ, S. E., HALL, G., BANERJEE, A., BOWMAN, C. L., DALY, L., JONES, T., KIRK, M. D., FITZGERALD, M. & ANGULO, F. J. 2005. Prevalence of diarrhoea in the community in Australia, Canada, Ireland, and the United States. *Int J Epidemiol*, 34, 454-60.

- SCHETS, F. M., ENGELS, G. B. & EVERS, E. G. 2004. Cryptosporidium and Giardia in swimming pools in the Netherlands. *J Water Health*, 2, 191-200.
- SCHOEFER, Y., ZUTAVERN, A., BROCKOW, I., SCHAFER, T., KRAMER, U., SCHAAF, B., HERBARTH, O., VON BERG, A., WICHMANN, H. E. & HEINRICH, J. 2008. Health risks of early swimming pool attendance. *Int J Hyg Environ Health*, 211, 367-73.
- SCHOLL, H. J. J. Action research and system dynamics: can they benefit from each other? 37th Annual Hawaii International Conference on System Sciences, 2004. Proceedings of the, 5-8 Jan. 2004 2004. 11 pp.
- SEMENZA, J. & NICHOLS, G. 2007. Cryptosporidiosis surveillance and water-borne outbreaks in Europe. *Eurosurveillance*, 12, 13-14.
- SENGE, P. 2006. The fifth discipline (Revised ed.). New York: Currency Doubleday.
- SEQWATER. 2012. Annual Report 2011-2012 [Online]. Available: http://www.seqwater.com.au/sites/default/files/PDF%20Documents/Publications/20130205-
  - Seqwater-WD-WQMP-Annual-Report-.pdf [Accessed].
- SEQWATER. 2013. Response to submission of the SEQ Water Grid Manager [Online]. Available: <u>http://www.qca.org.au/getattachment/41abf143-ba00-4802-8237-1ff81a1b4e02/Seqwater-2012-13-</u> <u>Submission-Response-to-SEQ-Water.aspx</u> [Accessed May 7 2018].
- SHIELDS, J. M., GLEIM, E. R. & BEACH, M. J. 2008a. Prevalence of Cryptosporidium spp. and Giardia intestinalis in swimming pools, Atlanta, Georgia. *Emerg Infect Dis*, 14, 948-50.
- SHIELDS, J. M., HILL, V. R., ARROWOOD, M. J. & BEACH, M. J. 2008b. Inactivation of Cryptosporidium parvum under chlorinated recreational water conditions. *J Water Health*, 6, 513-20.
- SILAGY, C., LANCASTER, T., GRAY, S. & FOWLER, G. 1994. Effectiveness of training health professionals to provide smoking cessation interventions: systematic review of randomised controlled trials. *Quality in Health Care*, 3, 193-198.
- SIOKOU, C., MORGAN, R. & SHIELL, A. 2014. Group model building: a participatory approach to understanding and acting on systems. *Public Health Research & Practice*, 25.
- SKYTTNER, L. 1996. Decision Making and Decision Aids. General Systems Theory. Springer.
- SMITH, R. P., CHALMERS, R. M., MUELLER-DOBLIES, D., CLIFTON-HADLEY, F. A., ELWIN, K., WATKINS, J., PAIBA, G. A., HADFIELD, S. J. & GILES, M. 2010. Investigation of farms linked to human patients with cryptosporidiosis in England and Wales. *Prev Vet Med*, 94, 9-17.
- SNEL, S. J., BAKER, M. G., KAMALESH, V., FRENCH, N. & LEARMONTH, J. 2009. A tale of two parasites: the comparative epidemiology of cryptosporidiosis and giardiasis. *Epidemiol Infect*, 137, 1641-50.
- SOLLER, J. A., SCHOEN, M. E., BARTRAND, T., RAVENSCROFT, J. E. & ASHBOLT, N. J. 2010. Estimated human health risks from exposure to recreational waters impacted by human and nonhuman sources of faecal contamination. *Water Res*, 44, 4674-91.
- SOONAWALA, D., VAN LIESHOUT, L., DEN BOER, M. A., CLAAS, E. C., VERWEIJ, J. J., GODKEWITSCH, A., RATERING, M. & VISSER, L. G. 2014. Post-travel screening of asymptomatic long-term travelers to the tropics for intestinal parasites using molecular diagnostics. *Am J Trop Med Hyg*, 90, 835-9.
- SORVILLO, F. J., FUJIOKA, K., NAHLEN, B., TORMEY, M. P., KEBABJIAN, R. & MASCOLA, L. 1992. Swimming-associated cryptosporidiosis. *Am J Public Health*, 82, 742-4.
- STAFFORD, R., NEVILLE, G., TOWNER, C. & MCCALL, B. 2000. A community outbreak of Cryptosporidium infection associated with a swimming pool complex. *Commun Dis Intell*, 24, 236-9.
- STARK, D., ROBERTS, T., ELLIS, J. T., MARRIOTT, D. & HARKNESS, J. 2014. Evaluation of the EasyScreen<sup>™</sup> Enteric Parasite Detection Kit for the detection of Blastocystis spp., Cryptosporidium spp., Dientamoeba fragilis, Entamoeba complex, and Giardia intestinalis from clinical stool samples. *Diagnostic Microbiology and Infectious Disease*, 78, 149-152.
- STAVE, K. A., ZIMMERMANN, N. & KIM, H. 2016. Exploring the nature of insight in System Dynamics. 34th Conference of the International System Dynamics Society. Delft, Netherlands.
- STEFANOGIANNIS, N., MCLEAN, M. & VAN MIL, H. 2001. Outbreak of cryptosporidiosis linked with a farm event. *N Z Med J*, 114, 519-21.
- STERMAN, J. D. 2000a. *Business dynamics: systems thinking and modeling for a complex world*, Irwin/McGraw-Hill Boston.
- STERMAN, J. D. 2000b. Business dynamics: systems thinking and modeling for a complex world.

- STERMAN, J. D. 2001. System Dynamics Modeling: Tools for Learning in a Complex World. *California Management Review*, 43, 8-25.
- STERMAN, J. D. 2002. All models are wrong: reflections on becoming a systems scientist. *System Dynamics Review: The Journal of the System Dynamics Society*, 18, 501-531.
- STONE, E. G., MORTON, S. C., HULSCHER, M. E., MAGLIONE, M. A., ROTH, E. A., GRIMSHAW, J. M., MITTMAN, B. S., RUBENSTEIN, L. V., RUBENSTEIN, L. Z. & SHEKELLE, P. G. 2002. Interventions that increase use of adult immunization and cancer screening services: a meta-analysis. *Ann Intern Med*, 136, 641-51.
- SUPPES, L. M., CANALES, R. A., GERBA, C. P. & REYNOLDS, K. A. 2016. Cryptosporidium risk from swimming pool exposures. *Int J Hyg Environ Health*.
- SUSMAN, G. I. & EVERED, R. D. 1978. An Assessment of the Scientific Merits of Action Research. *Administrative Science Quarterly*, 23, 582-603.
- SWEENEY, L. B. & STERMAN, J. D. 2000. Bathtub dynamics: initial results of a systems thinking inventory. *System Dynamics Review*, 16, 249-286.
- SYSTEM DYNAMICS SOCIETY. 2015. Introduction to System Dynamics [Online]. Available: <u>http://www.systemdynamics.org/what-is-s/</u> [Accessed 08-13 2015-].
- TAM, C. C., RODRIGUES, L. C., VIVIANI, L., DODDS, J. P., EVANS, M. R., HUNTER, P. R., GRAY, J. J., LETLEY, L. H., RAIT, G., TOMPKINS, D. S. & O'BRIEN, S. J. 2012. Longitudinal study of infectious intestinal disease in the UK (IID2 study): incidence in the community and presenting to general practice. *Gut*, 61, 69-77.
- TEN HOVE, R. J., VAN ESBROECK, M., VERVOORT, T., VAN DEN ENDE, J., VAN LIESHOUT, L. & VERWEIJ, J. J. 2009. Molecular diagnostics of intestinal parasites in returning travellers. *Eur J Clin Microbiol Infect Dis*, 28, 1045-53.
- UTSI, L., SMITH, S. J., CHALMERS, R. M. & PADFIELD, S. 2016. Cryptosporidiosis outbreak in visitors of a UK industry-compliant petting farm caused by a rare Cryptosporidium parvum subtype: a case-control study. *Epidemiol Infect*, 144, 1000-9.
- VALDERRAMA, A. L., HLAVSA, M. C., CRONQUIST, A., COSGROVE, S., JOHNSTON, S. P., ROBERTS, J. M., STOCK, M. L., XIAO, L., XAVIER, K. & BEACH, M. J. 2009. Multiple risk factors associated with a large statewide increase in cryptosporidiosis. *Epidemiol Infect*, 137, 1781-8.
- VALLY, H., HALL, G., SCALLAN, E., KIRK, M. D. & ANGULO, F. J. 2009. Higher rate of cultureconfirmed Campylobacter infections in Australia than in the USA: is this due to differences in healthcare-seeking behaviour or stool culture frequency? *Epidemiol Infect*, 137, 1751-8.
- VAN DEN BOSSCHE, D., CNOPS, L., VERSCHUEREN, J. & VAN ESBROECK, M. 2015. Comparison of four rapid diagnostic tests, ELISA, microscopy and PCR for the detection of Giardia lamblia, Cryptosporidium spp. and Entamoeba histolytica in feces. *J Microbiol Methods*, 110, 78-84.
- VANDENBERG, O., ROBBERECHT, F., DAUBY, N., MOENS, C., TALABANI, H., DUPONT, E., MENOTTI, J., VAN GOOL, T. & LEVY, J. 2012. Management of a Cryptosporidium hominis outbreak in a day-care center. *Pediatr Infect Dis J*, 31, 10-5.
- VENNIX, J. A. 1999. Group model-building: tackling messy problems. *System Dynamics Review: The Journal of the System Dynamics Society*, 15, 379-401.
- VICTORIAN GOVERNMENT 2008. Pool operators handbook. Melbourne, Australia: Department of Human Services,.
- VOSE, D. 2000. *Risk analysis, a quantitative guide, 2nd edition*. [Online]. Available: <u>https://www.vosesoftware.com/riskwiki/PERTdistribution.php</u> [Accessed].
- WALKER, W. E., HARREMOËS, P., ROTMANS, J., VAN DER SLUIJS, J. P., VAN ASSELT, M. B. A., JANSSEN, P. & KRAYER VON KRAUSS, M. P. 2003. Defining Uncertainty: A Conceptual Basis for Uncertainty Management in Model-Based Decision Support. *Integrated Assessment*, 4, 5-17.
- WEESE, J. S., MCCARTHY, L., MOSSOP, M., MARTIN, H. & LEFEBVRE, S. 2007. Observation of practices at petting zoos and the potential impact on zoonotic disease transmission. *Clin Infect Dis*, 45, 10-5.
- WEITZEL, T., WICHMANN, O., MÏHLBERGER, N., REUTER, B., HOOF, H. D. & JELINEK, T. 2006. Epidemiological and clinical features of travel-associated cryptosporidiosis. *Clinical Microbiology* and Infection, 12, 921-924.
- WHEELER, C., VUGIA, D. J., THOMAS, G., BEACH, M. J., CARINES, S., MAIER, T., GORMAN, J., XIAO, L., ARROWOOD, M. J., GILLISS, D. & WERNER, S. B. 2007. Outbreak of cryptosporidiosis at a California waterpark: Employee and patron roles and the long road towards prevention. *Epidemiology and Infection*, 135, 302-310.

- WIANT, C. 2011. A Snapshot of Swimmer Hygiene Behavior. International Journal of Aquatic Research and Education, 5.
- WILLOWS, R., REYNARD, N., MEADOWCROFT, I. & CONNELL, R. 2003. *Climate adaptation: Risk, uncertainty and decision-making. UKCIP Technical Report*, UK Climate Impacts Programme.

WOOLHOUSE, M. 2008. Exemplary epidemiology. Nature, 453, 34-34.

- WOOLHOUSE, M. 2011. How to make predictions about future infectious disease risks. *Philos Trans R Soc Lond B Biol Sci*, 366, 2045-54.
- WORLD HEALTH ORGANIZATION 2006. Guidelines for safe recreational water environments Volume 2: Swimming pools and similar Environments. Geneva Switzerland: WHO Press.
- WORLD HEALTH ORGANIZATION. 2015. *Health Topics Environmental Health* [Online]. Available: <u>http://www.who.int/topics/environmental\_health/en/</u> [Accessed, 2015 June 3].
- WORLD HEALTH ORGANIZATION 2016. *Quantitative microbial risk assessment: application for water safety management,* Geneva, Switzerland, WHO Press.
- XIAO, L., BERN, C., LIMOR, J., SULAIMAN, I., ROBERTS, J., CHECKLEY, W., CABRERA, L., GILMAN, R. H. & LAL, A. A. 2001. Identification of 5 types of Cryptosporidium parasites in children in Lima, Peru. J Infect Dis, 183, 492-7.
- XIAO, L. & FAYER, R. 2008. Molecular characterisation of species and genotypes of Cryptosporidium and Giardia and assessment of zoonotic transmission. *Int J Parasitol*, 38, 1239-55.
- YODER, J. S., HLAVSA, M. C., CRAUN, G. F., HILL, V., ROBERTS, V., YU, P. A., HICKS, L. A., ALEXANDER, N. T., CALDERON, R. L., ROY, S. L. & BEACH, M. J. 2008. Surveillance for waterborne disease and outbreaks associated with recreational water use and other aquatic facilityassociated health events--United States, 2005-2006. MMWR Surveill Summ, 57, 1-29.
- ZHU, F. X. 2018. Anti-Infection Handbook eBook, Elsevier Health Sciences.

Appendix A: Loop breakdown in causal loop diagram

Loop ID	Variables involved	Key concept
Reinford	zing statut s	
R1	Crypto cases in the community $\rightarrow$ healthy people exposed to infectious people $\rightarrow$ Crypto cases in the	Person-to-person transmission
	community	
R2	Crypto cases in the community $\rightarrow$ Infectious swimmers in the pool $\rightarrow$ <i>Cryptosporidium</i> oocytes in	Infections acquired at public
	the pool $\rightarrow$ Healthy swimmers exposed to crypto oocytes in the pool $\rightarrow$ Crypto cases in the	aquatic facilities
	community	
Balancir	lg	
B1a	Crypto cases in the community $\rightarrow$ People seeking medical care $\rightarrow$ suspected cases who receive	New secondary cases (B1a)
&	precautionary transmission-prevention advice $\rightarrow$ People knowledgeable about transmission-	and swimming-related cases
B1b	prevention strategies $\rightarrow$ (B1a: healthy people exposed to infectious people) or (B1b: Infectious	(B1b) prevented by infectious
	swimmers in the pool $\rightarrow$ <i>Cryptosporidium</i> oocytes in the pool $\rightarrow$ Healthy swimmers exposed to	cases receiving transmission-
	crypto oocytes in the pool) $\rightarrow$ Crypto cases in the community	prevention advice prior to
		diagnosis
B2a	Crypto cases in the community $\rightarrow$ people seeking medical care $\rightarrow$ people who have been tested for	New secondary cases (B2a)
&	crypto $\rightarrow$ people who test positive for crypto $\rightarrow$ people who receive precautionary advice at time of	and swimming-related cases
B2b	diagnosis → people knowledgeable about transmission-prevention strategies → (B2a: healthy people	(B2b) prevented by infectious
	exposed to infectious people) or (B2b: Infectious swimmers in the pool → <i>Cryptosporidium</i> oocytes	cases receiving transmission-
	in the pool $\rightarrow$ Healthy swimmers exposed to crypto oocytes in the pool) $\rightarrow$ Crypto cases in the	prevention advice at time of
	community	diagnosis

B3a	People who test positive for crypto $\rightarrow$ Notified crypto cases in the community $\rightarrow$ control measures	Transmission awareness and
&	from public health service $\rightarrow$ people knowledgeable about transmission-prevention strategies $\rightarrow$	prevention messaging to the
B3b	(B3a: healthy people exposed to infectious people) or (B2b: Infectious swimmers in the pool $\rightarrow$	general public
	<i>Cryptosporidium</i> oocytes in the pool $\rightarrow$ Healthy swimmers exposed to crypto oocytes in the pool) $\rightarrow$	
	Crypto cases in the community $\rightarrow$ people seeking medical care $\rightarrow$ people who have been tested for	
	crypto $\rightarrow$ people who test positive for crypto	
B4a	People who test positive for crypto $\rightarrow$ Notified crypto cases in the community $\rightarrow$ control measures	Transmission awareness and
&	from public health service $\rightarrow$ Doctors' crypto awareness and knowledge $\rightarrow$ suspected cases who	prevention messaging to
B4b	receive precautionary transmission-prevention advice $\rightarrow$ (B4a: healthy people exposed to infectious	general practitioners
	people) or (B4b: Infectious swimmers in the pool $\rightarrow$ <i>Cryptosporidium</i> oocytes in the pool $\rightarrow$ Healthy	
	swimmers exposed to crypto oocytes in the pool) $\rightarrow$ Crypto cases in the community $\rightarrow$ people	
	seeking medical care $\rightarrow$ people who have been tested for crypto $\rightarrow$ people who test positive for	
	crypto	
B5	People who test positive for crypto $\rightarrow$ Notified crypto cases in the community $\rightarrow$ control measures	Swimming pool
	from public health service $\rightarrow$ Swimming pool operator awareness of crypto in the community $\rightarrow$	hyperchlorination at the
	hyperchlorination of the swimming pool $\rightarrow$ <i>Cryptosporidium</i> oocytes in the pool $\rightarrow$ Healthy	request of the PHS
	swimmers exposed to crypto oocytes in the pool $\rightarrow$ Crypto cases in the community $\rightarrow$ people seeking	
	medical care $\rightarrow$ people who have been tested for crypto $\rightarrow$ People who test positive for crypto	
<b>B6</b>	Crypto cases in the community $\rightarrow$ people seeking medical care $\rightarrow$ suspected cases who receive	Swimming pool operator
	precautionary advice $\rightarrow$ people who receive precautionary advice at time of diagnosis $\rightarrow$ people	become aware of crypto in the
	knowledgeable about transmission-prevention strategies $\rightarrow$ Swimming pool operator awareness of	community by members of the
	crypto in the community $\rightarrow$ hyperchlorination of the swimming pool $\rightarrow$ <i>Cryptosporidium</i> oocytes in	general public

	the pool $\rightarrow$ Healthy swimmers exposed to crypto oocytes in the pool) $\rightarrow$ Crypto cases in the	
	community	
<b>B7</b>	People who test positive for crypto $\rightarrow$ Notified crypto cases in the community $\rightarrow$ control measures	Increased community
	from public health service $\rightarrow$ people knowledgeable about transmission-prevention strategies $\rightarrow$	awareness of crypto following
	Public awareness of crypto within the community $\rightarrow$ Community attendance at the pools $\rightarrow$ Healthy	public messaging campaigns
	swimmers exposed to crypto oocytes in the pool)  Crypto cases in the community  people seeking	leading to community
	medical care $\rightarrow$ people who have been tested for crypto $\rightarrow$ People who test positive for crypto	members avoiding pools
<b>B8</b>	Notified crypto cases in the community $\rightarrow$ Media attention $\rightarrow$ Public awareness of crypto within the	Media-driven public awareness
	community $\rightarrow$ People knowledgeable about transmission-prevention strategies $\rightarrow$ (B1a: healthy	
	people exposed to infectious people) or (B1b: Infectious swimmers in the pool $\rightarrow$ <i>Cryptosporidium</i>	
	oocytes in the pool $\rightarrow$ Healthy swimmers exposed to crypto oocytes in the pool) $\rightarrow$ Crypto cases in	
	the community $\rightarrow$ people seeking medical care $\rightarrow$ people who have been tested for crypto $\rightarrow$ People	
	who test positive for crypto -> Notified crypto cases in the community	
<b>B9</b>	Notified crypto cases in the community $\rightarrow$ Media attention $\rightarrow$ Public awareness of crypto within the	Media attention spurring
	community $\rightarrow$ Guidelines and regulations $\rightarrow$ Swimming pool operator training $\rightarrow$ swimming pool	political will to change policy
	staff knowledge and experience → (B9a: Proper management of faecal accidents ) or (B9b:	
	Effectiveness of disinfection system $\rightarrow$ <i>Cryptosporidium</i> oocytes in the pool $\rightarrow$ Healthy swimmers	
	exposed to crypto oocytes in the pool) $\rightarrow$ Crypto cases in the community $\rightarrow$ people seeking medical	
	care $\rightarrow$ people who have been tested for crypto $\rightarrow$ People who test positive for crypto $\rightarrow$ Notified	
	crypto cases in the community	
B10	Swimming pool operator training $\rightarrow$ swimming pool staff knowledge and experience $\rightarrow$ Swimming	Training and staff knowledge
	pool operator training	and experience

B11	Hyperchlorination of the swimming pool $\rightarrow$ community attendance at the swimming pool $\rightarrow$	Financial disincentive to close		
	swimming pool resources $\rightarrow$ Hyperchlorination of the swimming pool	pool for hyperchlorination		

**Appendix B: Model Parameters and Equations** 

## PAF Sector

Variable	Unit	Equation/value	Ref
New_LP_cases_by_pool [PHU, Large_Pool_number]	persons/ day	SUM(New_cases_from_LP [PHU,Large_Pool_number,*])	
new_sp_cases_by_pool [PHU, Small_Pool_number]	persons/ day	SUM(New_cases_from_SPs [PHU,Small_Pool_number,*])	
AFR_LP.AFR_LP [PHU, Large_Pool_number, Age]	DMNL	BINOMIAL(1, AFR_per_day_in_Large_pool)	
AFR_LP.AFR_per_day_in_Large_pool [PHU, Large_Pool_number, Age]	AFR/ day	.Sick_swimmers_in_each_LP [ <i>PHU</i> , <i>Large_Pool_number</i> , <i>Age</i> ] ×(.Probability_of_AFR_given_infection_LP [ <i>Age</i> , <i>Large_Pool_number</i> ] +(.Probability_of_AFR_given_infection_LP [ <i>Age</i> , <i>Large_Pool_number</i> ] × (AFR_sensitivity_converter [ <i>Age</i> ] /100)))	
AFR_LP.Large_ARF_released [PHU, Large Pool number, Age]	oocytes/ day	IF (AFR_LP [ <i>PHU</i> , <i>Large Pool number</i> , <i>Age</i> ] =1) THEN Oocyte concetration in an AFR [ <i>Age</i> ] ELSE 0	
AFR_LP.number_oocytes_per_unit_we ight	oocytes/ gram	UNIFORM(50, 10 <sup>6</sup> )	(Pintar et al., 2010),(G erba, 2000)
AFR_LP.Oocyte_concetration_in_an_A FR [Age]		weight_of_AFR [Age] ×number_oocytes_per_unit_weight	
AFR_LP.oocytes_released_into_LP [PHU, Large_Pool_number]	oocytes/ day	<pre>SUM(Large_ARF_released [PHU,Large_Pool_number, *])</pre>	
<b>AFR_LP.weight_of_AFR</b> [Under_5_Years_Old]	grams	TRIANGULAR(30, 50, 70)	(Pintar et al., 2010)
<b>AFR_LP.weight_of_AFR</b> [Over_5_Years_Old]	grams	TRIANGULAR(100, 150, 200)	(Pintar et al., 2010)
AFR_SP.AFR_in_Small_Pool [PHU, Small_Pool_number, Age]	DMNL	BINOMIAL(1,AFR_per_day_in_small_pool)	
<b>AFR_SP.AFR_per_day_in_small_pool</b> [PHU, Small_Pool_number, Age]	AFR/ day	Sick_swimmers_in_each_SP×(.Probability_of_AFR_given_infection_ SP [ <i>Age,Small_Pool_number</i> ]	

		+(.Probability_of_AFR_given_infection_SP [ <i>Age,Small_Pool_number</i> ] × (AFR sensitivity converter SP [ <i>Age</i> ] /100)))	
AFR_SP.number_oocytes_per_unit_we ight	oocytes/ day	UNIFORM(50, 10 <sup>6</sup> ,)	(Pintar et al., 2010),(G erba, 2000)
AFR_SP.Oocyte_concetration_in_an_A FR [Age]	oocytes/ day	weight_of_AFR [Age] ×number_oocytes_per_unit_weight)	
AFR_SP.oocytes_released_into_SP [PHU, Small_Pool_number]	oocytes/ day	<pre>SUM(Small_ARF_released [PHU, Small_Pool_number, *])</pre>	
AFR_SP.Small_ARF_released [PHU, Small_Pool_number, Age]	DMNL	(AFR_in_Small_Pool [ <i>PHU</i> , <i>Small_Pool_number</i> , <i>Age</i> ] =1) THEN Oocyte_concetration_in_an_AFR [ <i>Age</i> ] ELSE 0	
AFR_SP.weight_of_AFR [Under_5_Years_Old]	grams	TRIANGULAR(30, 50, 70)	(Pintar et al., 2010)
<b>AFR_SP.weight_of_AFR</b> [Over_5_Years_Old]	grams	TRIANGULAR(100, 150, 200)	(Pintar et al., 2010)
Healthy_Swimmers.Daily_susceptible_ Swimmers [PHU, Age]	persons/ day	Daily_Swimming_frequency×(Susceptible_swimmers [ <i>PHU, Age</i> ] ×.Seasonal_converter [ <i>Age</i> ] )	
Healthy_Swimmers.healthy_adults_in_ LP [PHU]	persons/ day	susceptible_adult_swimmers [PHU] ×(1-percent_of_adults_using_SP)	
Healthy_Swimmers.Daily_susceptible_ Swimmers [PHU, Age]	persons/ day	.Daily_Swimming_frequency×(Susceptible_swimmers [ <i>PHU</i> , <i>Age</i> ] ×.Seasonal_converter [ <i>Age</i> ] )	
Healthy_Swimmers.healthy_adults_in_ LP [PHU]	persons/ day	susceptible_adult_swimmers [PHU] ×(1-percent_of_adults_using_SP)	
Healthy_Swimmers.Healthy_adults_in_ SP [PHU]	persons/ day	susceptible_adult_swimmers [PHU] ×(percent_of_adults_using_SP)	
Healthy_Swimmers.healthy_children_i n_LP [PHU]	persons/ day	susceptible_child_swimmers [PHU] × percent_of_children_using_LP	
Healthy_Swimmers.Healthy_children_i n_SP [PHU]	persons/ day	susceptible_child_swimmers [PHU] ×(1- percent_of_children_using_LP)	
Healthy_Swimmers.Healthy_People_in _LP [PHU, Under 5 Years Old]	persons/ day	healthy_children_in_LP [PHU]	
Healthy_Swimmers.Healthy_People_in _LP [PHU, Over_5_Years_Old]	persons/ day	healthy_adults_in_LP [PHU]	

Healthy_Swimmers.percent_of_adults_ using_SP	DMNL	0.05
Healthy_Swimmers.percent_of_childre n_using_LP	DMNL	0.25
Healthy_Swimmers.susceptible_adult_s wimmers [PHU]	persons/ day	Daily_susceptible_Swimmers [PHU,Over_5_Years_Old]
Healthy_Swimmers.susceptible_child_s wimmers [PHU]	persons/ day	Daily_susceptible_Swimmers [PHU,Under_5_Years_Old]
Healthy_Swimmers.Susceptible_swimm ers [PHU, Age]	persons	.SUSCEPTIBLE_PEOPLE [ <i>PHU</i> , <i>Age</i> ] ×.Portion_of_population_who_swim [ <i>Age</i> ]
Sick_swimmers.Adults_in_LP [PHU]	persons/ day	(infectious_adult_swimmers [ <i>PHU</i> ] ×(1-PAF_Messaging [ <i>Large pool</i> ] )) × (1-percent_of_adults_using_SP)
Sick_swimmers.Adults_in_SP [PHU]	persons/ day	(infectious_adult_swimmers [ <i>PHU</i> ] ×(1-PAF_Messaging [ <i>Small pool</i> ] )) × (percent_of_adults_using_SP)
Sick_swimmers.children_in_LP [PHU]	persons/ day	infectious_child_swimmers [ <i>PHU</i> ] × (1-PAF_Messaging [ <i>Large pool</i> ] )) ×percent_of_children_using_LP
Sick_swimmers.children_in_SP [PHU]	persons/ day	(infectious_child_swimmers [ <i>PHU</i> ] × (1-PAF_Messaging [ <i>Small pool</i> ] )) × (1-percent_of_children_using_LP)
Sick_swimmers.daily _infectious_swimmers [PHU, Age]	persons/ day	Infectious_swimmers [ <i>PHU</i> , <i>Age</i> ] ×(.Daily_Swimming_frequency×.Seasonal_converter [ <i>Age</i> ] )
Sick_swimmers.infectious adult_swimmers [PHU]	persons/ day	daily_infectious_swimmers [PHU,Over_5_Years_Old]
Sick_swimmers.infectious _child_swimmers [PHU]	persons/ day	daily_infectious_swimmers [PHU,Under_5_Years_Old]
<b>Sick_swimmers.Infectious_swimmers</b> [PHU, Age]	persons	(.Total_Infectious_People [ <i>PHU</i> , <i>Age</i> ] × .Portion_of_population_who_swim [ <i>Age</i> ] ) × (1- "Percent_of_infectious_swimmers_who_self-exclude" [ <i>PHU</i> , <i>Age</i> ] )
Sick_swimmers.Messaging_Converter [Pool_type]	DMNL	IF Pool_messaging_time_cycle = Messaging_start_date AND PAF_messaging_switch = 1 THEN PULSE(PAF_Messaging_effectiveness, Messaging_start_date) ELSE 0
Sick_swimmers.Messaging_start_date	DMNL	140
Sick_swimmers.PAF_Messaging [Pool_type]	DMNL	DELAY1((Messaging_Converter [ <i>Pool_type</i> ] ×PAF_Messaging_Effectiveness_Decay), PAF_Messaging_Effectiveness_Decay)

Sick_swimmers.PAF_messaging_cycle_	days	365
period [Large_pool]		
Sick_swimmers.PAF_messaging_cycle_	days	365
period [Small_pool]		
Sick_swimmers.PAF_	DMNL	0.1
Messaging_effectiveness		
Sick_swimmers.PAF_Messaging_	days	15
Effectiveness_Decay		
Sick_swimmers.PAF_messaging_switch	DMNL	0
[Pool type]		
Sick_swimmers.percent_of	DMNL	0.05
_adults_using_SP		
Sick swimmers.percent of	DMNL	0.25
_children_using_LP		
Sick swimmers."Percent of infectious	DMNL	AWARE INFECTIOUS PEOPLE [Age,PHU] /
swimmers who self-exclude" [PHU,		.Total Infectious People [PHU,Age]
Age]		
Sick_swimmers.Pool_CycleStartTime	days	INIT(TIME)
[Pool_type]		
Sick_swimmers.Pool_messaging_time_	days	COUNTER(Pool_CycleStartTime,
cycle [Pool_type]		Pool_CycleStartTime+PAF_messaging_cycle_period)
Sick_swimmers.Sick_People_in_LP	persons/	children_in_LP [PHU]
[PHU, Under_5_Years_Old]	day	
Sick_swimmers.Sick_People_in_LP	persons/	Adults in LP [PHU]
[PHU, Over_5_Years_Old]	day	
Seasonal_converter	DMNL	GRAPH(Time_cycle)(1.0, 0.750), (35.0, 0.750), (63.0, 0.750), (98.0,
[Under_5_Years_Old]		1.000), (126.0, 1.000), (154.0, 1.200), (189.0, 1.500), (217.0, 1.200),
		(245.0, 1.000), (280.0, 1.000), (308.0, 1.000), (343.0, 0.750), (365.0,
		0.750)
Seasonal converter	DMNL	GRAPH(Time cycle)(1.0, 0.400), (35.0, 0.400), (63.0, 0.500), (98.0,
[Over 5 Years Old]		0.750), (126.0, 1.000), (154.0, 1.100), (189.0, 1.500), (217.0, 1.200),
,		(245.0, 1.100), (280.0, 1.000), (308.0, 0.750), (343.0, 0.500), (365.0,
		0.400)
AFR detected LP	DMNL	IF (RANDOM(0,100) <
[Large_Pool_number]		AFR_detection_and_proper_manAgement_rate_LP) THEN 1 ELSE 0

AFR_detected_SP	DMNL	= IF (RANDOM(0,100) <	
[Small_Pool_number]		AFR_detection_and_proper_man <i>Age</i> ment_rate_SP) THEN 1 ELSE 0	
AFR_detection_and_proper_	DMNL	30	
management_rate_LP			
AFR_detection_and_proper_	DMNL	30	
management_rate_SP			
Contamination_LP	oocytes/	IF PREVIOUS(Reactionary_hyperchlorination_LP	
[PHU, Large_Pool_number]	day	$[PHU,Large\_Pool\_number], 0) = 1 \text{ OR}$	
		PREVIOUS(Routine_hyperchlorination_LP	
		$[PHU,Large\_Pool\_number], 0) = 1$ THEN 0 ELSE	
		(Oocytes_shed_intoLP [PHU,Large_Pool_number]	
		+AFR_LP.oocytes_released_into_LP [ <i>PHU</i> , <i>Large_Pool_number</i> ])	
Contamination_SP	oocytes/	IF PREVIOUS(Reactionary_hyperchlorination_SP	
[PHU, Small_Pool_number]	day	$[PHU,Small_Pool_number], 0) = 1 \text{ OR}$	
		PREVIOUS(Routine_hyperchlorination_SP	
		[PHU,Small Pool number], 0) = 1 THEN 0 ELSE	
		Oocytes_shed_into_SP [PHU,Small_Pool_number]	
		+AFR_SP.oocytes_released_into_SP [PHU,Small_Pool_number]	
Crypto_dose_in_LPs	oocytes/	(oocytes_per_Litre_in_LPs [ <i>PHU</i> , <i>Large_Pool_number</i> ] ×	
[PHU, Age, Large_Pool_number]	person	(Percent_of_oocytes_viable/100)) × Pool_waster_ingested_per_swim	
Compte dese in SDs	ooortos/	[Age,Large pool]	
[DHU] Aga Small Bool number]	norgen	(bocytes_per_Life_in_SPS [PHU, Small_Pool_number] >	
[FHU, Age, Sman_Fool_number]	person	[ <i>Age</i> .Small pool]	
Daily Swimming frequency	persons/	$BETA(1.5, 4.5) \times 0.79 + 0.008$	(Australi
[PHU, Age]	person		an
	/day		Bureau
	•		of
			Statistics,
			2014)
Decontamination_LP	oocytes/	IF Reactionary_hyperchlorination_LP [ <i>PHU</i> , <i>Large_Pool_number</i> ] = 1	
[PHU, Large_Pool_number]	day	THEN ((Oocytes_in_the_large_pool [PHU,Large_Pool_number] /DT)	
		+ (Contamination_LP-Removal_LP [PHU, Large Pool number] ))	
		ELSE IF Routine_hyperchlorination_LP [ <i>PHU</i> , <i>Large_Pool_number</i> ] =	
		1	

		THEN ((Oocytes_in_the_large_pool [ <i>PHU</i> , <i>Large_Pool_number</i> ] /DT)+(Contamination_LP-Removal_LP [ <i>PHU</i> , <i>Large_Pool_number</i> ]	
		)) ELSE 0	
Decontamination_SP [PHU, Small_Pool_number]	oocytes/ day	IF Reactionary_hyperchlorination_SP [ <i>PHU</i> , <i>Small_Pool_number</i> ] = 1 THEN ((Oocytes_in_the_small_pool [ <i>PHU</i> , <i>Small_Pool_number</i> ] /DT)+(Contamination_SP-Removal_SP [ <i>PHU</i> , <i>Small_Pool_number</i> ] )) ELSE IF Routine_hyperchlorination_SP [ <i>PHU</i> , <i>Small_Pool_number</i> ] = 1 THEN ((Oocytes_in_the_small_pool [ <i>PHU</i> , <i>Small_Pool_number</i> ] /DT)+(Contamination_SP-Removal_SP [ <i>PHU</i> , <i>Small_Pool_number</i> ] )) ELSE 0	
Disinfectant_type_switch_LP	DMNL	0	
Disinfectant_type_switch_SP	DMNL	0	
Dose_response_parameter [PHU]	persons/ oocytes/ day	BETA(2.55,3.45) ×0.061+0.005	(Messner et al., 2001), (Brouwer et al., 2017), (Ryu et al., 2007)
Grams_of_faeces_shed_by_adults	grams	UNIFORM(0.001, 0.1)+0.1	(Gerba, 2000)
Grams_of_faeces_shed_by_children	grams	$BETA(1.05, 4.95) \times 4.99 + 0.01$	(Gerba, 2000)
healthy_swimmers_in_each_LP	persons/	Healthy_Swimmers.Healthy_People_in_LP [PHU, Age]	
[PHU, Large_Pool_number, Age]	day	×Users_in_each_LP [ <i>Large_Pool_number</i> ]	
healthy_swimmers_in_each_SP	persons/	Healthy_Swimmers.Healthy_People_in_SP [PHU, Age]	
[PHU, Small_Pool_number, Age]	day	×Users_in_each_SP [Small_Pool_number]	
"Log-3_disinfection_LP"	days	$BETA(2.47, 3.53) \times 1.465 + 1.16$	
[PHU, Large_Pool_number]			
"Log-3_disinfection_SP_		UNIFORM(2,10)	
highest_risk_only"		BETA(3.071,2.929) ×1.12+0.58	
[Small_Pool_number]			

New_cases_from_LP	persons/	Probability_of_infection_per_swim_event_LP [PHU,	
[PHU, Large_Pool_number, Age]	day	<i>Large_Pool_number</i> , <i>Age</i> ] ×healthy_swimmers_in_each_LP [ <i>PHU</i> ,	
		Large Pool number, Age]	
New cases from SPs	persons/	Probability of infection per swim event SP [PHU,	
[PHU, Small Pool number, Age]	dav	Small Pool number, Age ] × healthy swimmers in each SP [PHU.	
	5	Small Pool number. Age]	
New swimming related cases [PHI]	nersons/	(SUM(New cases from SPs $[PHU * Age]$ )	
Agol	dav	+SUM(New cases from $IP[PHU * Age]$ )	
1150	day	+ $((SIIM(New cases from SPs [PHI] * Aga])$	
		$+SUM(New eases from LD[DHU * (as1)) \times$	
		+SUM(New_cases_nom_LF [FII0, ·, Age])) ×	
		(Swimming_cases_sensitivity_parameter/100))	
Oocyst_sned_by_LP_users	oocytes/	Sick_swimmers_in_each_LP×Oocytes_from_shedding_swimmers	
[PHU, Large_Pool_number, Age]	day	[ <i>PHU</i> , <i>Age</i> ] - ((Oocytes_from_shedding_swimmers [ <i>PHU</i> , <i>Age</i> ] × 0.8)	
		× (Sick_swimmers_in_each_LP×	
		((Percent_of_patrons_who_shower_LP [Age]	
		/100)+Sick_swimmers.PAF_Messaging [Large_pool] )))	
Oocyst_shed_by_SP_users	oocytes/	Sick_swimmers_in_each_SP×Oocytes_from_shedding_swimmers	
[PHU, Small_Pool_number, Age]	day	[ <i>PHU</i> , <i>Age</i> ] - ((Oocytes_from_shedding_swimmers [ <i>PHU</i> , <i>Age</i> ] × 0.8)	
		× (Sick_swimmers_in_each_SP×	
		((Percent of patrons who shower SP [Age]	
		/100)+Sick swimmers.PAF Messaging [Small pool] )))	
oocyte inactivation LP	DMNL	UNIFORM(2,10)	(Ryan et
[PHU, Large Pool number]			al., 2017)
oocyte inactivation SP	DMNL	UNIFORM(2,10)	(Ryan et
[PHU, Small Pool number]			al., 2017)
<b>Oocytes from shedding swimmers</b>	oocytes/	Grams of faeces shed by children×oocytes in 1 gram of stool	
[PHU, Under 5 Years Old]	person		
<b>Oocytes from shedding swimmers</b>	oocytes/	Grams of faeces shed by adults×oocytes in 1 gram of stool	
[PHU, Over 5 Years Old]	person	$[PHU] \qquad \qquad$	
oocytes in 1 gram of stool	-	UNIFORM(50, 10^6)	(Castor
(PHU)			and
			Beach.
			2004)
Oocytes in the large pool	oocvtes	Oocytes in the large pool [PHU, Large Pool number] (t - dt) +	
[PHU, Large Pool number] (t)		(Contamination LP [PHU Large Pool number] - Removal LP	
[,,,, (v)			

		[ <i>PHU</i> , Large_Pool_number] - Decontamination_LP [ <i>PHU</i> , Large_Pool_number] ) × dt	
Oocytes_in_the_small_pool [PHU, Small_Pool_number] (t)	oocytes	Oocytes_in_the_small_pool [PHU, Small_Pool_number] (t - dt) + (Contamination_SP [PHU, Small_Pool_number] - Removal_SP [PHU, Small_Pool_number] - Decontamination_SP [PHU, Small_Pool_number] ) × dt	
oocytes_per_Litre_in_ <i>LPs</i> [PHU, Large_Pool_number]	oocytes/ litre	Oocytes_in_the_large_pool [ <i>PHU</i> , <i>Large_Pool_number</i> ] /LP_volume [ <i>Large Pool number</i> ]	
oocytes_per_Litre_in_SPs [PHU, Small_Pool_number]	oocytes/ litre	Oocytes_in_the_small_pool [ <i>PHU</i> , <i>Small_Pool_number</i> ] /SP_volume [ <i>Small_Pool_number</i> ]	
Oocytes_shed_into_SP	oocytes/ day	<pre>SUM(Oocyst_shed_by_SP_users [PHU,Small_Pool_number,*])</pre>	
Oocytes_shed_intoLP [PHU, Large_Pool_number]	oocytes/ day	<pre>SUM(Oocyst_shed_by_LP_users [PHU,Large_Pool_number, *])</pre>	
Percent_of_oocytes_viable	DMNL	$BETA(3.06, 2.95) \times 38.9 + 61.1$	(Schets et al., 2004)
<b>Percent_of_patrons_who_shower_LP</b> [Age]	DMNL	15	
<b>Percent_of_patrons_who_shower_SP</b> [Age]	DMNL	15	
Pool_waster_ingested_per_swim [Under_5_Years_Old, Large_pool]	Litre/ person	BETA(2.26,4.66) ×0.154	(Dufour et al., 2006)
Pool_waster_ingested_per_swim [Under 5 Years Old, Small pool]	Litre/ person	BETA(2.26,4.66) ×0.154	,
<b>Pool_waster_ingested_per_swim</b> [Over 5 Years Old, Large pool]	Litre/ person	BETA(2.71,4.66) ×0.053	
<b>Pool_waster_ingested_per_swim</b> [Over 5 Years Old, Small pool]	Litre/ person	UNIFORM(0, 0.01)	
Portion_of_population_who_swim [Age]	DMNL	= 0.0596	(Australi an Bureau of Statistics, 2014)

<b>Probability_of_AFR_given_infection_L</b> <b>P</b> [Under 5 Years Old]	DMNL	BETA(1.44, 4.56) $\times 0.045 + 0.0005$	
<b>Probability_of_AFR_given_infection_L</b> <b>P</b> [Over 5 Years Old]	DMNL	BETA(2.77, 3.22) ×0.009+0.001	
<b>Probability_of_AFR_given_infection_S</b> <b>P</b> [Under_5_Years_Old]	DMNL	BETA(1.44, 4.56) $\times 0.045 + 0.0005$	
<b>Probability_of_AFR_given_infection_S</b> <b>P</b> [Over_5_Years_Old]	DMNL	BETA(2.77, 3.22) $\times 0.009 + 0.001$	
Probability_of_infection_per_ swim_event_LP [PHU, Large_Pool_number, Age]	DMNL	1-EXP(-Crypto_dose_in_LPs [ <i>PHU,Age,Large_Pool_number</i> ] ×Dose_response_parameter [ <i>PHU</i> ])	(Messner et al., 2001), (Brouwer et al., 2017), (Ryu et al., 2007)
Probability_of_infection_per_ swim_event_SP [PHU, Small_Pool_number, Age]	DMNL	1-EXP(-Crypto_dose_in_SPs [ <i>PHU</i> , <i>Age,Small_Pool_number</i> ] ×Dose_response_parameter [ <i>PHU</i> ] )	(Messner et al., 2001), (Brouwer et al., 2017), (Ryu et al., 2007)
Reactionary_hyperchlorination_LP [PHU, Large_Pool_number]	DMNL	IF AFR_LP.oocytes_released_into_LP [ <i>PHU</i> , <i>Large_Pool_number</i> ] >0 AND AFR_detected_LP [ <i>Large_Pool_number</i> ] >0 THEN 1 ELSE 0	, ,
Reactionary_hyperchlorination_SP [PHU, Small_Pool_number]	DMNL	IF (AFR_SP.oocytes_released_into_SP [ <i>PHU</i> , <i>Small_Pool_number</i> ] >0 AND AFR_detected_SP [ <i>Small_Pool_number</i> ] >0) THEN 1 ELSE 0	
Removal_LP [PHU, Large_Pool_number]	oocytes/ day	IF Disinfectant_type_switch_LP = 1 THEN (Oocytes_in_the_large_pool [ <i>PHU</i> , <i>Large_Pool_number</i> ] /"Log-	

		3_disinfection_LP") ELSE (Oocytes_in_the_large_pool
		[PHU,Large_Pool_number] /oocyte_inactivation_LP
		[PHU,Large_Pool_number])
Removal_SP	oocytes/	IF Disinfectant_type_switch_SP= 2 THEN
[PHU, Small_Pool_number]	day	(Oocytes_in_the_small_pool [PHU,Small_Pool_number] /"Log-
		3_disinfection_SP") ELSE IF Disinfectant_type_switch_SP = 1 THEN
		(Oocytes_in_the_small_pool [PHU,Small_Pool_number] /"Log-
		3_disinfection_SP_highest_risk_only" [Small_Pool_number] ) ELSE
		(Oocytes_in_the_small_pool [PHU,Small_Pool_number]
		/oocyte_inactivation_SP [PHU,Small_Pool_number])
Routine_hyperchlorination_frequency_	days	90
LP		
Routine_hyperchlorination_frequency_	days	60
SP		
Routine_hyperchlorination_LP	DMNL	PULSE (Routine_hyperchlorination_switch_LP,
[PHU, Large_Pool_number]		Routine_hyperchlorination_frequency_LP,
		Routine_hyperchlorination_frequency_LP)) ×DT
Routine_hyperchlorination_SP	DMNL	PULSE (Routine_hyperchlorination_switch_SP,
[PHU, Small_Pool_number]		Routine_hyperchlorination_frequency_SP,
		Routine_hyperchlorination_frequency_SP)) ×DT
Routine_hyperchlorination_switch_LP	DMNL	0
Routine_hyperchlorination_switch_SP	DMNL	0
Sick_swimmers_in_each_LP	persons/	Sick_swimmers.Sick_People_in_LP [PHU, Age] × Users_in_each_LP
[PHU, Large_Pool_number, Age]	day	[Large_Pool_number]
Sick_swimmers_in_each_SP	persons/	Sick_swimmers.Sick_People_in_SP [PHU, Age] ×Users_in_each_SP
[PHU, Small_Pool_number, Age]	day	[Small_Pool_number]
Users_in_each_LP	DMNL	IF Time_cycle <45 OR Time_cycle>300 THEN Seasonal_LP_users
[Large_Pool_number]		[Large_Pool_number,Winter] ELSE Seasonal_LP_users
		[Large_Pool_number,Summer]
Users_in_each_SP	DMNL	IF Time_cycle <45 OR Time_cycle>300 THEN Seasonal_SP_users
[Small_Pool_number]		[Small_Pool_number,Winter] ELSE Seasonal_SP_users
		[Small Pool number,Summer]

Healthcare Sector

Variable	Unit	Equation/value	Ref
faecal_testing_delay	days	UNIFORM(1, 5)	
faecal_testing_rate [Age, PHU]	persons/ day	testing_gap [Age,PHU] /faecal_testing_delay	
fraction_of_people_tested [Age, PHU]	DMNL	BETA(3.08, 2.91) ×0.2208	(Scallan et al., 2005),(OzFoodNet Working Group, 2003), (Kirk et al., 2014), (Vally et al., 2009)
fraction_of_positive_cases _made_aware [Under_5_Years_Old]	DMNL	43	
fraction_of_positive_cases_ made_aware [Over_5_Years_Old]	DMNL	43	
fraction_of_tests_submitted	DMNL	$BETA(4, 2) \times 0.04 + 0.91$	(Kirk et al., 2014)
health_seeking_fraction [PHU, Age]	DMNL	$BETA(3.25, 2.75) \times 0.103$	(Vally et al., 2009), (Scallan et al., 2005)
healthcare_seeking_gap [Age, PHU]	persons	predicted_people_with_crypto_going_to_doctor [ <i>Age,PHU</i> ] - PEOPLE_WITH_CRYPTO_AT_THE_DOCTOR [ <i>Age,PHU</i> ]	
healthcare_seeking_rate [Age, PHU]	persons/ day	healthcare_seeking_gap [Age,PHU] /treatment_seeking_delay	
microscopy_sensitivity [PHU]	DMNL	BETA(3.25, 2.74) ×0.67+0.33	(ten Hove et al., 2009), (Stark et al., 2014), (Van den Bossche et al., 2015), (Chalmers et al., 2011)
PCR_sensitivity [PHU]	DMNL	BETA(3.72, 2.23) × 0.139+0.8	(ten Hove et al., 2009), (Stark et al., 2014), (Haque et al., 2007)

people_not_tested_but_aware [Age, PHU] PEOPLE_WITH_CRYPTO_AT_ THE_DOCTOR	persons/ day persons	PEOPLE_WITH_CRYPTO_AT_THE_DOCTOR [ <i>Age,PHU</i> ] × (1-(fraction_of_people_tested × fraction_of_tests_submitted ))) × (physician_precautionary_advice_fraction [ <i>Age</i> ] /100)+(Healthcare_Messaging_effect [ <i>PHU,Age</i> ] )) PEOPLE_WITH_CRYPTO_AT_THE_DOCTOR [ <i>Age, PHU</i> ] (t - dt) + (healthcare_seeking_rate [ <i>Age_PHU</i> ]) × dt	
[Age, PHU] (t)		al) (neutricule_seeking_lute [rige, 1 110]) at	
PEOPLE_WITH_CRYPTO_TES TED [Age, PHU] (t)	persons	PEOPLE_WITH_CRYPTO_TESTED [ <i>Age</i> , <i>PHU</i> ] (t - dt) + (faecal_testing_rate [ <i>Age</i> , <i>PHU</i> ]) × dt	
physician_precautionary _advice_fraction [Age]	DMNL	5	(Attias et al., 2015)
positive_crypto_cases [Age, PHU]	persons	((PEOPLE_WITH_CRYPTO_TESTED [ <i>Age,PHU</i> ] ×testing_transition) × PCR_sensitivity [ <i>PHU</i> ] ) +((PEOPLE_WITH_CRYPTO_TESTED [ <i>Age,PHU</i> ] × (1- testing_transition) × microscopy_sensitivity [ <i>PHU</i> ] ))	
<pre>predicted_aware_infectious_peop le [Age, PHU]</pre>	persons	positive_crypto_cases [ <i>Age,PHU</i> ] × ((fraction_of_positive_cases_made_aware [ <i>Age</i> ] /100) +Healthcare_Messaging_effect [ <i>PHU,Age</i> ] )	
predicted_people_with_ crypto_going_to_ doctor [Age, PHU]	persons	SYMPTOMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ] × health_seeking_fraction [ <i>PHU</i> , <i>Age</i> ]	
predicted_people_with_ crypto_tested [Age, PHU]	persons	(PEOPLE_WITH_CRYPTO_AT_THE_DOCTOR [ <i>Age,PHU</i> ] × fraction_of_people_tested [ <i>Age,PHU</i> ] ) × (fraction_of_tests_submitted)	
testing_gap [Age, PHU]	persons	predicted_people_with_crypto_tested [ <i>Age,PHU</i> ] - PEOPLE WITH CRYPTO TESTED [ <i>Age,PHU</i> ]	
testing_transition	DMNL	GRAPH(TIME)(1, 0.000), (2009, 0.000), (2010, 0.050), (2206, 0.100), (2393, 0.150), (2571, 0.200), (2755, 0.300), (2936, 0.500), (3102, 0.700), (3486, 0.800), (4000, 0.850)	
treatment_seeking_delay	days	BETA(1.3, 4.69)×26+1	(Vally et al., 2009), (Scallan et al., 2005),(Valderrama et al., 2009)

Internationally-acquired case sector

Variable	Unit	Equation/value	reference
asymptomatic_returning	persons/	EXPOSED_TRAVELLERS [PHU, Age] $\times$ (1-	
[PHU, Age]	day	Rate_of_symptomatic_travellers)	
Daily_departures	persons/	GRAPH(TIME)	
[Under_5_Years_Old]	day		
Daily_departures	persons/	GRAPH(TIME)	
[Over_5_Years_Old]	day		
departing	persons/	Daily_departures [Age] × SEQ_fraction [PHU, Age] )+((Daily_departures	
[PHU, Age]	day	$[Age] \times SEQ$ _fraction $[PHU, Age] ) \times (Travel_multiplier/100))$	
EXPOSED_TRAVELLERS	persons	EXPOSED_TRAVELLERS [PHU, Under_5_Years_Old] (t - dt) + ("high-	
[PHU, Under_5_Years_Old] (t)		risk_exposure" [PHU, Under_5_Years_Old] - symptomatic_returning [PHU,	
		Under 5 Years Old] - asymptomatic returning [PHU, Under 5 Years Old])	
		× dt	
EXPOSED TRAVELLERS	persons	EXPOSED TRAVELLERS [PHU, Over 5 Years Old] (t - dt) + ("high-	
[PHU, Over 5 Years Old] (t)		risk exposure" [PHU, Over 5 Years Old] - symptomatic returning [PHU,	
		Over 5 Years Old] - asymptomatic returning [PHU, Over 5 Years Old]) ×	
		dt	
high risk infection rate	DMNL	UNIFORM(0, 0.014)	ten Hove
° <b>_ _ _</b>			RJ et al.
			2009
"high-risk exposure"	persons/	((TRAVELLERS [PHU, Age] × (1-proportion of low risk travellers [Age])	
[PHU, Age]	day	× (high risk infection rate))+(TRAVELLERS $[PHU,Age]$	
	•	× proportion of low risk travellers [Age] ×	
		(low risk infection rate)))/length of travel [PHU]	
length of travel	days	UNIFORM(2, 30)	
	•		
low risk infection rate	DMNL	UNIFORM(0, 0.009)	(ten Hove
			et al.,
			2009)
proportion_of_low_risk_travell	DMNL	GRAPH(TIME)	
ers [Under_5_Years Old]			
proportion_of_low_risk_travell	DMNI	GRAPH(TIME)	
	DIVINL		

Rate_of_symptomatic_traveller s	DMNL	BETA(2.16, 3.84) ×0.38+0.5	(ten Hove et al., 2009)
SEQ_fraction	DMNL	$[Metro_North, Under_5_Years_Old] = 0.1935$ $[Metro_North, Over_5_Years_Old] = 0.2009$ $[Metro_South, Under_5_Years_Old] = 0.2404$ $[Metro_South, Over_5_Years_Old] = 0.2282$ $[Gold_Coast, Under_5_Years_Old] = 0.1134$ $[Gold_Coast, Over_5_Years_Old] = 0.1216$	(Australian Bureau of Statistics, 2015c)
symptomatic_returning [PHU, Age]	persons/ day	(EXPOSED_TRAVELLERS [ <i>PHU</i> , <i>Age</i> ] × Rate_of_symptomatic_travellers)	
TRAVELLERS [PHU, Age] (t)	persons	TRAVELLERS [ <i>PHU</i> , <i>Age</i> ] (t - dt) + (departing [ <i>PHU</i> , <i>Age</i> ] - returning_uninfected [ <i>PHU</i> , <i>Age</i> ] - "high-risk_exposure" [ <i>PHU</i> , <i>Age</i> ] × dt	

## Population Sector

Variable	Unit	Equation/value	reference
Asymptomatic_infection [PHU, Age]	persons/ day	LATENTLY_INFECTED_PEOPLE [ <i>PHU</i> , <i>Age</i> ] × (1- Probably_of_being_symptomatic_given_infection))/Incubation_Period	
ASYMPTOMATIC_INFECTI OUS_PEOPLE [PHU, Age] (t)	persons	ASYMPTOMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ] (t) = ASYMPTOMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ] (t - dt) + (Asymptomatic_infection [ <i>PHU</i> , <i>Age</i> ] + asymptomatic_returning [ <i>PHU</i> , <i>Age</i> ] - Asymptopatic_recovery [ <i>PHU</i> , <i>Age</i> ] ) × dt	
Asymptopatic_recovery [PHU, Age]	persons/ day	ASYMPTOMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ] /"Post- symptom infectious period"	
daily_population_change [PHU, Age]	persons/ day	regional_population-(PREVIOUS(regional_population)	
duration_of_symptoms	days	BETA(2.04,3.31, 4) ×27+1	(Hunter et al., 2004a), (Jokipii and Jokipii 1986),(Heijbel et al., 1987),(Johansen et al., 2015), (Millard et al., 1994)

Exposure [PHU, Age]	persons/ day	New_swimming_related_cases [ <i>PHU</i> , <i>Age</i> ] +New_person_to_person_cases [ <i>Age</i> , <i>PHU</i> ]	(Jokipii and Jokipii 1986), (Millard et al., 1994),(Chalmers and Davies, 2010)
Incubation_Period	days	$BETA(4.3, 3.6) \times 11+1$	
LATENTLY_INFECTED_PE OPLE [PHU, Age] (t)	persons	LATENTLY_INFECTED_PEOPLE [ <i>PHU</i> , <i>Age</i> ] (t - dt) + (Exposure [ <i>PHU</i> , <i>Age</i> ] - Symptomatic_infection [ <i>PHU</i> , <i>Age</i> ] - Asymptomatic_infection [ <i>PHU</i> , <i>Age</i> ] ) × dt	
length_of_immunity	days	1	
Post- symptom_infectious_period	days	$BETA(2.69,3.31) \times 13+1$	(Jokipii and Jokipii 1986)
Probably_of_being_symptomati c_given_infection	DMNL	BETA(2.16, 3.84) ×0.38+0.5	(Okhmatovskaia et al., 2010),(Soller et al., 2010, Heijbel et al., 1987),
RECOVERED_PEOPLE [PHU, Age] (t)	persons	RECOVERED_PEOPLE [ <i>PHU</i> , <i>Age</i> ] (t - dt) + (Asymptopatic_recovery [ <i>PHU</i> , <i>Age</i> ] + symptomatic_recovery [ <i>PHU</i> , <i>Age</i> ] - Waning_immunity [ <i>PHU</i> , <i>Age</i> ] ) × dt	
RECOVERING_SYMPTIMAT IC _INFECTIOUS_PEOPLE [PHU, Age] (t)	persons	RECOVERING_SYMPTIMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ] (t - dt) + (symptoms_waning [ <i>PHU</i> , <i>Age</i> ] + Relapse_recovery [ <i>PHU</i> , <i>Age</i> ] - symptomatic_recovery [ <i>PHU</i> , <i>Age</i> ] - Relapsing [ <i>PHU</i> , <i>Age</i> ] ) × dt	
regional_population [PHU, Age]	persons/ day	GRAPH(TIME)	
Reinfection_delay	days	BETA(1.5,4.5) $\times$ 8+2	(MacKenzie et al., 1995b)
relapse_duration	days	$BETA(1.28, 4.71) \times 14+1$	(MacKenzie et al., 1995b)
Relapse_Rate	DMNL	BETA(1.84, 4.06) $\times 0.62 + 0.18$	(Okhuysen et al., 1998),(Hunter et

			al., 2004b),(Boehme r et al., 2009),(MacKen zie et al., 1995a)
Relapse_recovery [PHU, Age]	persons/ day	RELAPSED_SYMPTIMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ] /relapse_duration	
RELAPSED_SYMPTIMATIC_ INFECTIOUS_PEOPLE [PHU, Age] (t)	persons	RELAPSED_SYMPTIMATIC_INFECTIOUS_PEOPLE [PHU, Age] (t - dt) + (Relapsing [PHU, Age] - Relapse_recovery [PHU, Age]) × dt	
Relapsing [PHU, Age]	persons/ day	RECOVERING_SYMPTIMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ] ×Relapse_Rate/Reinfection_delay	
Returning_uninfected [PHU, Age]	persons/ day	((TRAVELLERS [ <i>PHU</i> , <i>Age</i> ] × proportion_of_low_risk_travellers [ <i>Age</i> ] × (1-low_risk_infection_rate))+((TRAVELLERS [ <i>PHU</i> , <i>Age</i> ] × (proportion_of_low_risk_travellers [ <i>Age</i> ]) × (1- high_risk_infection_rate))))/length_of_travel [ <i>PHU</i> ]	
SUSCEPTIBLE_PEOPLE [PHU, Age] (t)	persons	SUSCEPTIBLE_PEOPLE [ <i>PHU</i> , <i>Age</i> ] (t - dt) + (Waning_immunity [ <i>PHU</i> , <i>Age</i> ] + returning_uninfected [ <i>PHU</i> , <i>Age</i> ] + Daily_population_change_rate [ <i>PHU</i> , <i>Age</i> ] - Exposure [ <i>PHU</i> , <i>Age</i> ] - departing [ <i>PHU</i> , <i>Age</i> ] ) × dt	
<b>Symptomatic_infection</b> [PHU, Age]	persons/ day	LATENTLY_INFECTED_PEOPLE [ <i>PHU</i> , <i>Age</i> ] ×Probably_of_being_symptomatic_given_infection)/Incubation_Perio d	
SYMPTOMATIC_INFECTIO US_ PEOPLE [PHU, Age] (t)	persons	SYMPTOMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ] (t - dt) + (Symptomatic_infection [ <i>PHU</i> , <i>Age</i> ] + symptomatic_returning [ <i>PHU</i> , <i>Age</i> ] - symptoms_waning [ <i>PHU</i> , <i>Age</i> ] ) × dt	
symptomatic_recovery [PHU, Age]	persons/ day	(LATENTLY_INFECTED_PEOPLE [ <i>PHU</i> , <i>Age</i> ] ×Probably_of_being_symptomatic_given_infection)/Incubation_Perio d	
SYMPTOMATIC_INFECTIO US_ PEOPLE [PHU, Age] (t)	persons	SYMPTOMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ] (t - dt) + (Symptomatic_infection [ <i>PHU</i> , <i>Age</i> ] + symptomatic_returning [ <i>PHU</i> , <i>Age</i> ] - symptoms_waning [ <i>PHU</i> , <i>Age</i> ] ) × dt	
symptomatic_recovery [PHU, Age]	persons/ day	(RECOVERING_SYMPTIMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ] × (1-Relapse_Rate)/"Post-symptom_infectious_period"	

symptoms_waning	persons/	SYMPTOMATIC_INFECTIOUS_PEOPLE [ <i>PHU</i> , <i>Age</i> ]
[PHU, Age]	day	/duration_of_symptoms
Waning_immunity [PHU, Age]	persons/ day	RECOVERED_PEOPLE [PHU, Age] /length_of_immunity

Public Health Sector

Variable	Unit	Equation/value	Ref
Aware_diagnosed_cases [Age, PHU]	persons/ day	predicted_aware_infectious_people [Age,PHU] /awareness_delay [Age] )	
Aware_imported_cases	persons/	symptomatic_returning [PHU,Age] ×	
[Age, PHU]	day	(Percent_of_sympotmatic_travellers_screened_out/100)	
AWARE_INFECTIOUS_PEO	persons	AWARE_INFECTIOUS_PEOPLE [ <i>Age</i> , <i>PHU</i> ] (t - dt) +	
PLE [Age, PHU] (t)		(Aware_diagnosed_cases [Age, PHU] + Aware_suspected_cases [Age, PHU] +	
		aware_unconsulted_cases [Age, PHU] + Aware_imported_cases [Age, PHU] -	
		Recovery_rate [Age, PHU]) $\times$ dt	
Aware_suspected_cases	persons/	people_not_tested_but_aware	
[Age, PHU]	day		
aware_unconsulted_cases	persons/	((total_symptomatic_people [PHU,Age] -AWARE_INFECTIOUS_PEOPLE	
[Age, PHU]	day	[Age,PHU]) × Messaging_behaviour_change_proportion [PHU,Age])	
awareness_delay	days	$BETA(1.66, 4.33) \times (7-1)+1$	
[Under_5_Years_Old]			
awareness_delay	days	$BETA(1.89, 4.11) \times (10-9)+1$	
[Over_5_Years_Old]			
Healthcare_Messaging_	person/	IF Time_cycle = Healthcare_Messaging_start_date AND	
Converter	person/d	Healthcare_messaging_switch = 1 THEN	
[PHU]	ay	PULSE(Healthcare_Messaging_effectiveness [PHU],	
		Healthcare_Messaging_start_date) ELSE 0	
Healthcare_Messaging_effect	person	DELAY1((Healthcare_Messaging_Converter [PHU]	
[PHU, Age]	/person/	×Healthcare_Messaging_Effectiveness_Decay),	
	day	Healthcare_Messaging_Effectiveness_Decay)	
Healthcare_Messaging	person/	0.05	
_effectiveness	person/d		
[PHU]	ay		
Healthcare Messaging	days	15	

_Effectiveness_Decay		
Healthcare_Messaging_start_	day	140
date		
Healthcare_messaging_switch	DMNL	0
Messaging_behaviour_	person/	DELAY1(Routine_Messaging_Converter [PHU],
change_ proportion	person/d	Routine_Messaging_Effectiveness_Decay)
[PHU, Age	ay	
Notification_Gap	persons	positive_crypto_cases [Age, PHU] -NOTIFIED_CRYPTO_CASES [PHU,Age]
[PHU, Age]		
NOTIFIED_CRYPTO_CASE	persons	NOTIFIED_CRYPTO_CASES [PHU, Age] (t - dt) + (notifying [PHU, Age])
$\mathbf{S}$ [PHU, Age] (t)		$\times$ dt
Notifying	person/d	Notification_Gap [PHU,Age] /notification_delay
[PHU, Age]	ay	
Percent_of_sympotmatic_	DMNL	0
travellers_ screened_out		
PH_Public_messaging_switch	DMNL	1
Recovery_rate	persons/	AWARE_INFECTIOUS_PEOPLE [ <i>Age</i> , <i>PHU</i> ]
[Age, PHU]	day	/(duration_of_symptoms+"Post-symptom_infectious_period")
Routine_Messaging_Converter	person/	IF (Time_cycle = Routine_Messaging_start_date
[PHU]	person/d	AND PH_Public_messaging_switch = 1)
	ay	THEN PULSE(Routine_Messaging_effectiveness,
		Routine_Messaging_start_date) ELSE 0
Routine_Messaging_	person/p	0.05
effectiveness	erson/da	
	у	
Routine_Messaging_Effectiven	days	15
ess_Decay		
Routine_Messaging_start_date	days	140
total_symptomatic_people	persons	RECOVERING_SYMPTIMATIC_INFECTIOUS_PEOPLE +
[PHU, Age]		RELAPSED_SYMPTIMATIC_INFECTIOUS_PEOPLE +
		SYMPTOMATIC_INFECTIOUS_PEOPLE

Secondary Transmission Sector

Variable	Unit	Equation/value	reference
Adult_secondary_ transmission_ rate [PHU]	DMNL	(UNIFORM(0, 0.05))	(MacKenzie et al., 1995b)
Avoided_cases [Age, PHU]	persons/ day	LEAKAGE OUTFLOW LEAKAGE FRACTION=Proportion_of_cases_avoided [Age,PHU]	
Child_secondary_transmission _ rate [PHU]	DMNL	(BETA(3.19) ×0.31)	(Boehmer et al., 2009), (Heijbel et al., 1987),(Goh et al., 2004), (Causer et al., 2006)
New_person_to_person_cases [Age, PHU]	persons/ day	Secondary_transmission [Age,PHU]	
New_secondary_cases [Under_5_Years_Old, PHU]	persons/ day	((Potential_infectors [ <i>Under_5_Years_Old</i> ,Metro_North] × Child_secondary_transmission_rate [ <i>PHU</i> ] ) × 0.25)+((Potential_infectors [ <i>Over_5_Years_Old</i> , <i>PHU</i> ] × Adult_secondary_transmission_rate [ <i>PHU</i> ] ) × 0.5)	(Johansen et al., 2015), (Boehmer et al., 2009)
New_secondary_cases [Over_5_Years_Old, PHU]	persons/ day	((Potential_infectors [ <i>Under_5_Years_Old,PHU</i> ] × Child_secondary_transmission_rate [Metro_North] ) × 0.75)+((Potential_infectors [ <i>Over_5_Years_Old,PHU</i> ] × Adult_secondary_transmission_rate [Metro_North] ) × 0.5)	(Johansen et al., 2015), (Boehmer et al., 2009)
<b>Potential_infectors</b> [Age, PHU]	persons/ day	Total_new_infections [PHU,Age]	
"pre-infection_contacts" [Age, PHU]	Persons/ day	New_secondary_cases [Age,PHU]	
<b>Proportion_of_cases_avoided</b> [Age, PHU]	DMNL	(AWARE_INFECTIOUS_PEOPLE [ <i>Age</i> , <i>PHU</i> ] /Total_Infectious_People [ <i>PHU</i> , <i>Age</i> ] )	
Secondary_transmission [Age, PHU]	persons/ day	CONVEYOR OUTFLOW	
SUSCEPTIBLE_CONTACTE D _PEOPLE	persons	SUSCEPTIBLE_CONTACTED_PEOPLE [ <i>Age</i> , <i>PHU</i> ] (t - dt) + ("pre-infection_contacts" [ <i>Age</i> , <i>PHU</i> ] -	

[Age, PHU] (t)		Secondary_transmission [Age, PHU] - Avoided_cases [Age, PHU] ) × dt TRANSIT TIME = BETA(1.7, 4.3) ×40+1
Total_new_infections [PHU, Age]	persons/ day	SUM(Asymptomatic_infection [*,*]) + SUM(Symptomatic_infection [*,*]) + asymptomatic_returning + symptomatic_returning
**Appendix C: Human Ethics Approval Letters** 



Metro North

Hospital and Health Service

Royal Brisbane & Women's Hospital Human Research Ethics Committee

 
 Enquiries to:
 Ann-Maree Gordon Coordinator

 Telephone:
 07 3646 5490

 Facisimile:
 07 3646 5849

 File Ref:
 HREC/16/QRBW/509

 Email:
 RBWH-Ethics@health.qld.gov.au

Dr Paul Jagals School of Public Health The University of Queensland Level 2, Public Health Building (887) Cnr Herston Road and Wyndham Streets Herston Qld 4006

## Dear Dr Jagals,

## Re: Ref Nº: HREC/16/QRBW/509: A system dynamics approach to informing environmental health decision-making: the case of cryptosporidiosis in South East Queensland, Australia

Thank you for submitting the above research project for single ethical review. This project was received by the Royal Brisbane & Women's Hospital Human Research Ethics Committee (RBWH HREC) (EC00172) on 11 October 2016 and was considered by a sub-Committee of the HREC. The research project meets the requirements of the National Health and Medical Research Council's (NHMRC) National Statement on Ethical Conduct in Human Research (2007).

I am pleased to advise that the sub-Committee has approved of this low risk project. This approval will be noted by the RBWH Human Research Ethics Committee at its 14 November 2016 meeting.

The nominated participating sites for this project are:

- Environmental Protection Unit, Queensland Department of Health
- Metro North Public Health Unit, Metro North Hospital and Health Service
- Metro South Public Health Unit, Metro South Hospital and Health Service
- · Gold Coast Public Health Unit, Gold Coast Hospital and Health Service

Note: If additional sites are engaged prior to the commencement of, or during the research project, the Coordinating Principal Investigator is required to notify the RBWH HREC. Notification of withdrawn sites should also be provided to the RBWH HREC in a timely fashion.

Royal Brisbane & Women's Hospital Level 7 Block 7 Butterfield Street, Herston Qld 4029 Australia Telephone +61 7 3646 5490 Facsimile +61 7 3646 5840 www.health.qld.gov.au/metronorth/research/ ethics-governance/default.asp This letter constitutes ethical approval only. This project cannot proceed at any site until separate research governance authorisation has been obtained from the CEO or Delegate of the institution under whose auspices the research will be conducted at that site.

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Document	Version	Date
Covering Letter	1	10 October 2016
Low or Negligible Risk Research Application (Submission Code: AU/10/FA39218)	1.0 (2011)	05 October 2016
Research Protocol	1	05 October 2016
Recruitment Email - Workshop		
Recruitment Email - Interview		
Participant Information Sheet & Consent Form - Workshop		
Participant Information Sheet & Consent Form - Interview		
Participant Questionnaire (pre-participation)		
Participant Questionnaire (post-participation)		
Curriculum Vitae of Paul Jagals		06 October 2016
Curriculum Vitae of Dr Carl Smith		06 October 2016
Curriculum Vitae of Danielle Currie		07 October 2016

The approved documents include:

Approval of this project from the RBWH HREC is valid from 24.10.2016 to 24.10.2019 subject to the following conditions being met:

- The Coordinating Principal Investigator will immediately report anything that might warrant review of ethical approval of the project.
- The Coordinating Principal Investigator will notify the RBWH HREC of any event that
  requires a modification to the protocol or other project documents and submit any
  required amendments in accordance with the instructions provided by the HREC. These
  instructions can be found at <u>https://www.hcalth.qld.gov.au/metronorth/research/ethicsgovernance/hrec-approval/default.asp.</u>
- The Coordinating Principal Investigator will submit any necessary reports related to the safety of research participants in accordance with the RBWH HREC policy and procedures. These instructions can be found at <u>https://www.health.qld.gov.au/metronorth/research/ethics-governance/post-approvalreporting/default.asp.</u>

- In accordance with Section 3.3.22 (b) of the National Statement the Coordinating Principal Investigator will report to the RBWH HREC annually in the specified format, the first report being due on 24.10.2017 and a final report is to be submitted on completion of the study. These instructions can be found at <u>https://www.health.qld.gov.au/metronorth/research/ethicsgovernance/post-approval-reporting/default.asp.</u>
- The Coordinating Principal Investigator will notify the RBWH HREC if the project is discontinued before the expected completion date, with reasons provided.
- The Coordinating Principal Investigator will notify the RBWH HREC of any plan to extend the duration of the project past the approval period listed above and will submit any associated required documentation. Instructions for obtaining an extension of approval can be found at <u>https://www.health.qld.gov.au/metronorth/research/ethics-governance/hrecapproval/default.asp.</u>
- The Coordinating Principal Investigator will notify the RBWH HREC of his or her inability to continue as Coordinating Principal Investigator including the name of and contact information for a replacement.
- A copy of this ethical approval letter together with completed Site Specific Assessment (SSA) and any other requirements must be submitted by all site Principal Investigators to the Research Governance Office at each participating institution in a timely manner to enable the institution to authorise the commencement of the project at its site/s.
- Should you have any queries about the RBWH HREC's consideration of your project
  please contact the HREC Coordinator on 07 3646 5490. The RBWH HREC's Terms of
  Reference, Standard Operating Procedures, membership and standard forms are available
  from <a href="https://www.health.qld.gov.au/metronorth/research/ethics-governance/hrec-approval/membership/default.asp">https://www.health.qld.gov.au/metronorth/research/ethics-governance/hrec-approval/membership/default.asp</a>.

The RBWH HREC wishes you every success in your research.

Yours sincerely,

Suply

Dr Conor Brophy Chairperson RBWH Human Research Ethics Committee Metro North Hospital and Health Service 24.10.2016

This HREC is constituted and operates in accordance with the National Health and Medical Research Council's (NHMRC) National Statement on Ethical Conduct in Human Research (2007). The processes used by this HREC to review multi-centre research proposals have been certified by the National Health and Medical Research Council.



Human Ethlos Research Office

Cumbrae-Stewart Building #72 The University of Queensiand St Lucia, QLD 4072

CRICOS PROVIDER NUMBER 000258

6 December 2016

Dr Paul Jagals School of Public Health

Dear Dr Jagals,

Clearance Number: 2016001630 / (HREC/16/QRBW/209) Project Title: "A System Dynamics Approach to Informing Environmental Health Decision-Making: the Case of Cryptosporidiosis in South East Queensland, Australia"

Following administrative review of the human research ethics approval from the RBWH HREC dated 24/10/2016 (HREC/16/QRBW/209), I am pleased to advise that, as the University of Queensland's authorised delegate for the University of Queensland's Human Research Ethics Committees A & B, approval is granted for this project.

The approved documents include:

Document	Version	Date
Covering Letter		10/10/2016
LNR Application (AU/10/FA39218)	1.0	05/10/2016
Research Protocol	1	05/10/2016
Recruitment Email – Workshop		
Recruitment Email – Interview		
PISCF – Workshop		
PISCF – Interview		
Participant Questionnaire (pre-participation)		
Participant Questionnaire (post-participation)		
CV Dr Paul Jagals		06/10/2016
CV Dr Carl Smith		06/10/2016

Address. Human Research Ethics Office Cumbrae-Stewart Building #72 The University of Queensland St Lucia, QLD 4072 E humanethics@research.uq.edu.au W <u>www.uo.edu.au/research/inteority-</u> compliance/human-ethics



This project is approved until 24 October 2019

We would like to take this opportunity to remind you that, should any modifications be made to this project, they will need to be approved by the lead human research ethics committee prior to being forwarded to the University of Queensland's human ethics office for administrative review and approval.

Please keep a copy of this document for your records.

Yours sincerely,

Nicale Shively

Nicole Shively Deputy Director, Research Management Office Research Ethics Operations The University of Queensland

Address. Human Research Ethics Office Cumbrae-Stewart Building #72 The University of Queensland St Lucia, QLD 4072 E humanethics@research.uq.edu.au W www.uq.edu.au/research/integritycompliance/human-ethics