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Childhood food allergy in the New Zealand context: An exploration of trends, prevalence, risk factors, and the impact on quality of life

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Centre for Longitudinal Research – He Ara ki Mua

A thesis submitted in fulfilment of the requirements for the degree of Doctor of Philosophy in Health Sciences
The University of Auckland
2015
Abstract

Introduction
Although food allergy has been extensively described, diagnosed, managed and researched, many questions still linger. For over 2000 years scientists have sought to understand the effects of food on the human body. Hippocrates, the Father of Medicine, had recognition that food can cause illnesses, disease and health concerns for some people. In 1906, Dr Clemens von Pirquet suggested the use of the word “allergy” to describe an inappropriate reaction to food or other substances not typically harmful or bothersome. Researchers are just beginning to recognise potential influences on the development and progression of food allergy. The prevalence and incidence rates of food allergies worldwide have been the subject of much debate in recent decades due to what many observe as a dramatic increase in childhood food allergies. A food allergy touches the life of not only the individual diagnosed but also their family, friends, health care providers, food producers, retailers, and schools. The epidemiology of food allergy in New Zealand has been incompletely described and there is minimal published data that allows for any estimation of the disease burden caused by food allergy in New Zealand.

Aims
Although New Zealand has a high prevalence of asthma, the epidemiology of other atopic disease has not been studied. Based on other studies completed around the world and the data available in New Zealand this thesis set out to better understand: (1) temporal trends in food allergy; (2) prevalence of peanut allergy and risk factors for peanut allergy; and (3) the impact of childhood food allergy on quality of life.
Methods
To meet the objectives of this thesis four projects made up of five studies were completed. Each project utilised a different data set to allow comparisons to be made of data from New Zealand with that which has been reported from other countries and to provide several perspectives on how food allergies are impacting New Zealand children.

1) To determine whether Emergency Department presentations can be used to describe temporal trends in food allergy presentations an audit was completed for all emergency department (ED) presentations from 1988 to 2011 of children (0 to 14 years old) to the public hospital ED in the Auckland District Health Board (ADHB) region, for which the ICD codes ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’, were assigned.

2) The new knowledge learnt from project one was then applied to the National Minimum Dataset (NMDS), a national collection of public and private hospital discharge information, temporal trends in emergency department (ED) presentations for food-related acute allergic reactions from 1988 to 2011 of children (0 to 14 years old) were investigated.

3) Utilising data from the Growing Up in New Zealand cohort study the prevalence of peanut allergy and factors associated with the presence of peanut allergy at age two years were investigated.

4) The impact of food allergy on quality of life was investigated through the use of reflexive photography, photo elicitation, and the autodriven interview with food allergic children and their families.
Results

*Understanding Administrative Coding of Emergency Department Visits for Unspecified Acute Allergic Reactions*

The aim of this project was to determine the proportion of ED visits coded as ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’ that are food-related allergic reactions. Food-related acute allergic reactions account for 29% of hospital presentations that were assigned a discharge code for ‘anaphylaxis, unspecified’ or ‘allergy, unspecified in the ADHB.

The ED presentation rate with food-related allergic reactions from 2004-2011 was almost twice as high as that which occurred from 1988-1995 (RR=1.98, 95%CI 1.10-3.72). By contrast, ED presentation rates for non-food-related allergic reactions did not change over these years. This apparent increase in New Zealand is consistent with observations using comparable data sources reported from Australia and the United States.

*Hospital Presentations Due to Acute Allergic Reactions Related to Food*

Between 1988 and 2011, 3,735 children 0 to 14 years old presented to a New Zealand hospital with an acute allergic reaction identified by ICD-9-CMA-II codes 692.5 (contact dermatitis and other eczema due to food in contact with), 693.1 (Dermatitis due to food taken internally), 995.0 (Anaphylactic reaction due to unspecified food), 995.3 (Allergy, unspecified), and 995.60-995.69 (Anaphylactic reaction due to food unspecified and specified). An average yearly increase of 8% in hospital presentations due to acute allergic reactions (p=<0.001) was observed.

In comparison to the 1988 time interval (annual rate 7.11/100,000), the rate of all acute allergic presentations was consistently higher from 1997 onwards with rate ratios increasing
from 1.43 and 1.55 in 1997 and 1998 to 6.06 and 5.33 in 2010 and 2011. Over the time interval from 1988 to 2011 there has been an average yearly increase of 9% in hospital presentations due to all of the identified acute allergic reaction diagnostic groups (p<0.001). A greater rate of increase was observed in the anaphylactic and allergic reactions group (average increase 11% per year, p<0.001) as compared to the skin related group (average increase 2% per year, p<0.001).

Peanut Allergy in the New Zealand Context

Based on parental reported data collected from Growing Up in New Zealand, a contemporary longitudinal birth cohort study, 162 (2.1%) children were identified as peanut allergic. Within this cohort, factors associated with the development of a peanut allergy were categorized based on the measurement of child, family & wider influences on disease in early childhood as defined by: child characteristics, proximal social environments, distal social environments, and macro environmental factors.

The odds of having parental reported peanut allergy at age two years were increased for boys, children diagnosed with eczema since 9 months, children whose mother had a history of atopic disease (eczema, hay fever, or food allergy), and mothers who identified as being of Asian ethnicity. The odds of having parental reported peanut allergy at age two years were decreased for children who had never tried nuts or peanuts, or whose mothers had no secondary qualifications or secondary school/NCEA 1-4.

Impact of Food Allergy on New Zealand Families

The impact a food allergy has on a family is influenced by environment and includes four levels home, school, community, and beyond the community. This is due, in part, to the amount of
control a food allergic family has within these environments. The ability to control the environment gradually decreases as a food allergic family moves away from the home.

Based on the impact a food allergy has on the food allergic family, three outcomes of living with a food allergy are evident: responsibility, exclusion, and resilience. The lessons learned from these families can be used to guide other food allergic families through education and advocacy. Based on the family’s description of the impact of a food allergy on their quality of life and management strategies, there is a need for consistent national school policy focused on food allergies and the provision of education and training to hospitality workers. These changes could positively impact food allergic families at multiple environmental levels.

**Discussion**

Consistent with what has been reported from several other countries the prevalence of childhood food allergy appears to have increased in recent decades. The rate of parental self-report of peanut allergy in New Zealand is similar to other countries. In New Zealand, children of male gender or who have eczema are at increased, and those who have never tried nuts or peanuts by age two years were at decreased risk of parental reported peanut allergy. In New Zealand children of atopic mothers or mothers of Asian ethnicity are at increased risk and children of less educated mothers are at decreased risk of parental reported peanut allergy. A food allergy impacts all members of a food allergic family based on the level of control within various environments. Food allergy is a complex condition and this thesis provides insight into the current state of food allergy in New Zealand.
Acknowledgements

On a warm sunny day in June 2012 I boarded a plane in San Francisco heading for a new adventure in the “land of the long white cloud.” Little did I know that my three years in New Zealand would be a life changing experience filled with memories that will last a lifetime. During this time I was fortunate to meet many passionate, kind, and generous people whom I will never forget. The completion of my thesis would not have been possible without my many supervisors, advisors, colleagues, and friends. To Cameron, Susan, and Laura – thank you for your support, encouragement, and positivity. I have been so fortunate to work with you all and hope to continue to work together in the future. Your joyful smiles and gracious words kept me going. To Mandy – thank you for your friendship and laughter. Our many gab sessions provided endless hours of comic relief. To the Growing Up in New Zealand team – thank you for all of your tireless work collecting data and connecting with families. To the Growing Up in New Zealand families – thank you for your continued participation in the study. Your contributions will be experienced by generations to come. To the families who shared their stories with me – thank you for opening your home and providing a glimpse into your daily lives.

Although separated by many miles, the love and support of my family and friends back home was never far away. To Mom and Dad - thank you for all of the care packages and long phone calls. You were always there to help and provide an encouraging word. To my first mentor Dr Bryant – thank you for taking a chance on me and kindling my passion for teaching and research. Last but not least, thank you to my husband Joe who agreed to move to New Zealand to embark on a new adventure. We did it! I will always carry New Zealand close to my heart, thanks for the memories.
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This form is to accompany the submission of any PhD that contains published or unpublished co-authored work. Please include one copy of this form for each co-authored work. Completed forms should be included in all copies of your thesis submitted for examination and library deposit (including digital deposit), following your thesis Acknowledgements. Co-authored works may be included in a thesis if the candidate has written all or the majority of the text and had their contribution confirmed by all co-authors as not less than 65%.

Please indicate the chapter/section/pages of this thesis that are extracted from a co-authored work and give the title and publication details or details of submission of the co-authored work.

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<td>data collection, analysis, and manuscript development</td>
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The undersigned hereby certify that:

1. the above statement correctly reflects the nature and extent of the PhD candidate's contribution to this work, and the nature of the contribution of each of the co-authors; and  
2. that the candidate wrote all or the majority of the text.

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<td>Susan Morton</td>
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<td>12/11/15</td>
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<td>Carlos A. Camargo, Jr.</td>
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<td>12-Nov-2015</td>
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<td>ADHB</td>
<td>Auckland District Health Board</td>
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<tr>
<td>DBPCFC</td>
<td>Double blind placebo-controlled food challenge</td>
</tr>
<tr>
<td>DHB</td>
<td>District Health Board</td>
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<tr>
<td>ECC</td>
<td>Early Childhood Centre</td>
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<tr>
<td>ED</td>
<td>Emergency Department</td>
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<tr>
<td>HRQL</td>
<td>Health related quality of life</td>
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<tr>
<td>ICD</td>
<td>International Classification of Diseases</td>
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<td>IgE</td>
<td>Immunoglobulin E</td>
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<tr>
<td>ISAAC</td>
<td>International Study of Asthma and Allergies in Childhood</td>
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<tr>
<td>NHI</td>
<td>National Health Index</td>
</tr>
<tr>
<td>NIAID</td>
<td>US National Institute of Allergy and Infectious Disease</td>
</tr>
<tr>
<td>NICE</td>
<td>UK National Institute of Health and Clinical Excellence</td>
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<tr>
<td>NMDS</td>
<td>National Minimum Dataset</td>
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<td>NZ</td>
<td>New Zealand</td>
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<tr>
<td>OFC</td>
<td>Oral food challenge</td>
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<td>RAST</td>
<td>Radioallergosorbent test</td>
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<td>SPT</td>
<td>Skin prick test</td>
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<tr>
<td>TLA</td>
<td>Territorial Local Authority</td>
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<td>UK</td>
<td>United Kingdom</td>
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Chapter 1: INTRODUCTION

Researchers are only beginning to recognise potential influences on the development and progression of food allergy. Food allergies can affect any individual; the disorder does not discriminate based on geography, socioeconomic status, race, ethnicity, gender or age. A food allergy touches the life of not only the individual diagnosed but also their family, friends, health care providers, food producers and retailers, schools and government regulatory departments. Living with a food allergy affects the whole family through social interactions, emotional demands and resources. The prevalence and incidence rates of food allergies worldwide have been the subject of much debate in recent decades due to what many observe as a drastic increase in childhood food allergies. It’s the intention of this thesis to explore the prevalence of food allergy, causal factors and the population effects in the New Zealand context.

The epidemiology of food allergy in New Zealand has been incompletely described. This thesis will contribute to the field of food allergy research through the investigation of childhood food allergy in New Zealand including temporal trends in incidence, identification of factors associated with the development of food allergy including the role of prenatal factors and quality of life issues faced by both children diagnosed with a food allergy and their families.

Aims

Although New Zealand has a high prevalence of asthma, the epidemiology of other atopic disease has not been studied. Based on other studies completed around the world and the data available in New Zealand this thesis set out to better understand: (1)
temporal trends in food allergy; (2) prevalence of peanut allergy and risk factors for peanut allergy; and (3) the impact of childhood food allergy on quality of life.

Research Questions
1. For what proportion of ED visits, in public hospital ED in the Auckland District Health Board (ADHB), coded as ICD-9 codes 995.0 (ICD-10 T78.2) (anaphylaxis, unspecified) and 995.3 (ICD-10 T78.4) (allergy, unspecified) is the ED presentation due to a food-related allergic reaction?
2. Based on the National Minimum Dataset (NMDS), a national collection of public and private hospital discharge information, has the rate of emergency department presentations for food-related acute allergic reactions from 1988 to 2011 of children (0 to 14 years old) changed over time?
3. Within the birth cohort study Growing Up in New Zealand what is the prevalence of peanut allergy?
4. Based on the data collected from the birth cohort study Growing Up in New Zealand, what factors contribute to the development of a peanut allergy in early childhood (up to age 2 years).
5. How does a food allergy impact a family’s daily life?
6. What strategies are needed for a New Zealand family to cope, adapt, and feel fully supported when living with a food allergic child?
Table 1. Description of thesis studies and corresponding objectives and hypotheses.

<table>
<thead>
<tr>
<th>Study</th>
<th>Objective</th>
<th>Hypotheses</th>
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<tr>
<td>A determination of the proportion of food-related acute allergic reactions in children (0 to 14 years old), presenting to the public hospital ED in the Auckland District Health Board (ADHB) region, assigned a discharge code for 'anaphylaxis, unspecified' or 'allergy, unspecified' that were food-related.</td>
<td>To establish that ICD-9-CM codes can be used to describe emergency department presentations for food-related allergic reactions in New Zealand.</td>
<td>Through a review of medical charts from a public hospital ED in the Auckland District Health Board (ADHB), it is possible to determine the proportion of food-related acute allergic reactions in children aged 0 to 14 years when the discharge codes for 'anaphylaxis, unspecified' or 'allergy, unspecified' are assigned. The rate of presentation of children aged 0 to 14 years to a public hospital ED in the ADHB due to food-related acute allergic reactions assigned the discharge codes for 'anaphylaxis, unspecified' or 'allergy, unspecified' has increased over time.</td>
</tr>
<tr>
<td>An investigation of national temporal trends in emergency department (ED) presentations for food-related acute allergic reactions from 1988 to 2011 of children (0 to 14 years old).</td>
<td>To utilise emergency department presentations for food-related allergic reactions to describe epidemiological trends in food allergy prevalence in New Zealand.</td>
<td>The rate of emergency department presentations for food-related acute allergic reactions in children aged 0 to 14 years has increased from 1988 to 2011 in New Zealand.</td>
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<tr>
<td>Study</td>
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<tr>
<td>An investigation of the prevalence of peanut allergy at age two within New Zealand’s contemporary child cohort study <em>Growing Up in New Zealand</em> cohort study.</td>
<td>To describe the prevalence of peanut allergy at the age of 2 years within a birth cohort study.</td>
<td>The prevalence of peanut allergy in <em>Growing Up in New Zealand</em> at age two years will be within the prevalence range of 1% to 3% based on data reported from the United States, Canada, France, United Kingdom, and Australia.</td>
</tr>
<tr>
<td>An investigation of risk factors for peanut allergy at age two completed within the <em>Growing Up in New Zealand</em> cohort study.</td>
<td>To determine factors associated with the development of a peanut allergy in early childhood.</td>
<td>Factors acting at different phases of the life course contribute to the development of a peanut allergy by the age of 2 years.</td>
</tr>
<tr>
<td>A description of quality of life issues, and issues related to the management of food allergy, reported by New Zealand families raising a food allergic child.</td>
<td>To describe the impact of a food allergy on the daily life of a food allergic family and how the management strategies employed.</td>
<td>Childhood food allergy has a substantial impact upon quality of life for both the food allergic child and their family; important themes will emerge from a qualitative analysis for this disorder.</td>
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Overview of Thesis

This thesis is presented in accordance with the 2011 University of Auckland PhD thesis regulations for PhD with publication.

The thesis consists of 8 chapters and five studies investigating related topics on childhood food allergy. A review of the relevant literature precedes the presentation of original data from each of the five studies. Each chapter begins with a statement of the chapter objectives and ends with a conclusion which summarises how each chapter objective was met. This introductory chapter (Chapter 1) provides an introduction to the thesis and an overview of the approach taken. Chapter 2 provides part one of a review of literature pertaining to the epidemiology of food allergy. Building upon this foundation, Chapter 3, which consists of two parts, reports on two related studies describing national trends of food allergy in New Zealand. Chapter 4 provides part two of a review of the literature pertaining to potential causal pathways that result in a child developing a food allergy. Chapter 5 reports on the corresponding study describing the prevalence of peanut allergy at age two years and the risk factors for the presence of peanut allergy at age two years within the Growing Up in New Zealand cohort study. Chapter 6 provides part three of a review of the literature pertaining to the impact a food allergy has on quality of life. Chapter 7 reports on the corresponding study in which I report the findings from my study in which New Zealand families described quality of life issues and management of a food allergy.

Finally, in chapter 8, implications of these findings are considered and conclusions are made.
A manuscript which reports the findings from one of the five studies that form this thesis has been published and is included exactly as submitted, apart from being reformatted to be consistent with the remainder of the thesis (rather than included in the submitted format). Hence pages, tables, figures, and references in this article have been renumbered so that numbering is continuous throughout the thesis. I am the lead author on the publication; I planned and carried out the study, wrote the text and reviewed it with co-authors and wrote the final accepted version of the manuscript.

In accordance with the University of Auckland PhD thesis regulations for PhD with publication, introductory and closing chapters provide context around the publication.
Chapter 2: LITERATURE REVIEW PART ONE - EPIDEMIOLOGY OF FOOD ALLERGY

Chapter objectives
In this chapter I review the literature pertaining to the epidemiology of food allergy. Specifically I focus on what a food allergy is, who a food allergy affects and the situations where food allergies are most common. Food allergy is a complex topic as there is: (1) not yet a universally-accepted definition; (2) variation in the defining clinical features; and (3) variability in diagnosis and management. While acknowledging these limitations I provide a global summary of food allergy prevalence and incidence.

Overview
For over 2000 years scientists have sought to understand the effects of food on the human body. Hippocrates, the Father of Medicine, recognised that food caused illnesses, disease, and health concerns for some people. In 1906, Dr Clemens von Pirquet suggested the use of the word “allergy” to describe an inappropriate reaction to food or other substances not typically harmful or bothersome.

Food allergies are a problem because they (1) cause a decrease in quality of life; (2) generate substantial medical costs associated with disease management; and (3) can be life threatening. Although food allergy has been extensively described, diagnosed, managed and researched many questions still remain. Indeed, food allergy does not yet have a universally-accepted definition. To advance that goal, an expert panel sponsored by the National Institute of Allergy and Infectious Disease (NIAID) recently stated that food allergy is defined as an “adverse immune response that occurs reproducibly on exposure to a given food and is distinct from
other adverse responses to food, such as food intolerance, pharmacologic reactions, and toxin-mediated reactions” (Chafen et al., 2010).

More specifically, food allergy can be defined as an immunologically mediated adverse reaction to food, triggered by either an IgE- or non-IgE mediated immune mechanism. Non-IgE mediated reactions, which can be characterized as food intolerance, involves a wide spectrum of other adverse food reactions and includes some for which the mechanisms are unknown (Anderson, 2000). Their incidence greatly exceeds IgE mediated food allergies in all age groups. For the purposes of this dissertation, food allergy characterized by atopic (IgE mediated) reactions rather than non-atopic will be the focus of the literature review.

For many individuals with IgE mediated food allergy, IgE antibodies to foods are detectable within the first two years of life. Serum concentrations of these antibodies may increase or decrease over time. The pathophysiology of IgE mediated food allergy is characterized by the failure of the immune system to develop tolerance, or by the breakdown of tolerance, to food proteins.

Through a process that is not fully understood, IgE-mediated allergic sensitization to a specific food occurs after an individual is exposed to a food protein. The exposure leads to an immune response that includes the production of IgE antibodies to that specific food protein. Once the IgE antibodies are formed, they bind to tissue mast cells, and, in some cases, circulating basophils. The mast cells and the basophils are the effector cells of the allergic response. These cells release either preformed chemical mediators or are able to induce the formation of mediators in the immediate tissue around the cell once stimulated. Re-exposure to the same
food protein results in release of the chemical mediators, which lead to the clinical symptoms and signs of food allergy (Figure 1) (Buchanan, Sicherer, Furuta, & Burks, 2005).

**Figure 1.** IgE-dependent activation of mast cells and basophils

Clinically, IgE mediated food allergy is generally rapid in onset, occurring within minutes to two hours. Variance is a defining clinical feature of food allergy. Food-related acute allergic reactions can range in severity from an urticarial rash to life threatening food-induced anaphylaxis. Anaphylactic reactions (IgE-dependent) affect one or more target organs: the skin (urticaria, angio-oedema), respiratory tract (rhinitis, asthma), gastrointestinal tract (pain, emesis, diarrhoea), and cardiovascular system (anaphylactic shock) (Sicherer, 2002).

Similarly, much variability exists in diagnosis and management. Multiple methods are used to diagnose food allergy including one or more of: medical history, diet diary, elimination diet, skin prick testing, and double-blind placebo-controlled food challenge (Buchanan et al., 2005). Upon diagnosis an individual is instructed to avoid the foods that cause a reaction, provided with an over-the-
counter antihistamine or, for severe cases, prescribed an epinephrine autoinjector.

The eight most common foods to which people become allergic are peanuts, tree nuts, fruits, shell fish, fish, milk products and eggs. Variation in causal foods occurs in different global regions. For example egg, milk and wheat are the most common food allergens in Japan, whereas in Korea soybean, buckwheat, and peanut are also common (Goh, Lau, Chew, Shek, & Lee, 1999; Kanagawa, Matsumoto, Koike, & Imamura, 2009). In France, rosaceous fruit is a common food allergen (Kanny et al., 2001). In both the United States and Australia milk, egg, and peanut are common allergens reported (Hill et al., 1997; Sicherer, 2002).

The cause of these geographical variations are likely multifactorial with potential contributors including different eating patterns, cultural and environmental influences, genetic and epigenetic factors, plus contributions from non-immunological reactions to food (Sicherer, 2002). For example, in Israel, sesame is a major cause of IgE-mediated food allergy (Dalal et al., 2002). Sesame is the equivalent ‘peanut butter’ of the Middle East.

Not infrequently, individuals have symptomatic reactions to several food allergens. For example, in a study of 1383 food allergic patients whose families were members of a national allergy network in Japan, 80% of the food allergic patients (n = 1115) presented with allergic reactions to multiple food allergens (Kanagawa et al., 2009).

The prevalence and incidence rates of food allergies worldwide have been the subject of much debate in recent decades due to what many observe as a dramatic increase in childhood food allergies.
For example, in the United States (US), emergency room visits for food-induced anaphylaxis more than doubled from 2001 to 2006 (Rudders, Banerji, Vassallo, Clark, & Camargo, 2010). Similar increases have been observed in Australia where, from 1994/1995 to 2004/2005, hospital admissions for food-induced anaphylaxis increased five-fold among those age 0 to 4 years old (Poulos, Waters, Correll, Loblay, & Marks, 2007). Although a consensus has not been reached among researchers world-wide regarding the number of children affected by food allergies, all do agree that there is a need to more precisely determine both incidence and prevalence rates, and to do this in a manner that allows for comparison between countries and within countries over time.

However, determining food allergy prevalence and incidence is not straight forward. There is considerable variability in the methods used to define the presence of food allergy and food-related allergic reactions. Double-blind placebo-controlled food challenge is the gold standard diagnostic tool; however this method is resource intensive and not available worldwide, requires access to a tertiary referral centre and for those with a history of peanut-induced anaphylaxis would be life threatening (Baral & Hourihane, 2005; Boyce et al., 2010; Hourihane et al., 2007). Relying solely on self or parental report to describe food allergy and/or food related allergic reactions is insufficient as it results in a gross overestimation of both the prevalence and incidence (Rona et al., 2007). Well-designed surveillance programmes are needed to improve understanding of the epidemiology of food-related allergic reactions (Clark, Espinola, Rudders, Banerji, & Camargo, 2011).

**Contemporary issues in the diagnosis of IgE mediated food allergy**

Multiple methods are used in the evaluation of food allergic reactions including: medical history, diet diary, elimination diet, skin
prick testing, serum specific IgE measurements and food challenges. Skin-prick tests (SPTs) measure the local release of histamine and other allergic mediators in response to local stimulation by a specific antigen. Blood sampling is used to measure total and specific serum IgE concentrations and determine if the concentrations of these antibodies are elevated relative to population norms. An oral food challenge involves feeding a patient gradually increasing amounts of a food that is suspected to cause a previously identified reaction. The challenge is completed under observation by a physician and may be spread over hours or days. An oral food challenge is often used when it is possible that a patient may be allergic to multiple foods, tests for specific IgE are positive and elimination of the foods in question has resulted in resolution of symptoms. The food(s) chosen for the oral food challenge are dependent on the individual’s clinical history. The challenge can be either open or blinded. In an open challenge the patient, under careful medical and nursing supervision, is provided with a small amount of the suspected food in its natural state.

A double blind placebo-controlled food challenge (DBPCFC) is the method least prone to bias. The patient receives a disguised form of the food on a given day and is then provided with a dummy challenge on another (Baral & Hourihane, 2005). Neither the patient nor doctor knows whether the food in question or dummy is provided each day. The food is provided to the patient in small quantities below a predetermined threshold based on clinical history (50-250 mg doubled every 15 minutes). In most cases, once 8-10g is tolerated, IgE mediated reactivity is generally ruled out. To rule out the rare false negative the food in question is then given to the patient in usual quantities. A food challenge can last a minimum of 2h and up to 4-days for non-IgE mediated reactions (Ives & Hourihane, 2002)
Medical professionals differ in their opinion on which method is the optimal diagnostic testing regime. The DBPCFC has been considered the gold standard for decades. The false negative rate of DBPCFC is about 3%, so negative challenges should always be followed by a supervised open feeding of a relevant portion of the tested food in its commonly prepared state. Others dispute the idea that DBPCFC is the “only” way to diagnose food allergy, writing that food challenges, SPTs and serum food-specific IgE all have a role to play in making the diagnosis but no one test has sufficient ease of use, or sensitivity or specificity to be recommended to the exclusion of all other tests.

The lack of accurate and generalizable data regarding incidence and prevalence of food allergy worldwide is due in large part to the challenge of an appropriate and reproducible standard diagnostic procedure. Without a universally-accepted standard, cases are examined and diagnosed based on the clinical presentation of the symptomatic individual and availability of diagnostic resources. In some cases, due to lack of resources, people who have food allergy may never be diagnosed by a health professional. Alternatively, symptoms attributed to food allergy are often due to other causes.

Guidelines recommending increased use of confirmatory tests in general and food challenges in particular have been disseminated. It is anticipated that these will lead to more accurate diagnosis in those with symptoms presenting to a clinician.

The US National Institute of Allergy and Infectious Disease (NIAID) sponsored expert panel developed diagnostic guidelines, based on a meta-analysis of current research. Recommended methods included medical history, physical examination, skin prick test,
intradermal tests, allergen-specific serum IgE, atopy patch test, use of combination food elimination diets and oral food challenges. Of these methods the expert panel concluded that there was insufficient evidence to support: (1) the measurement of total serum IgE levels as this is neither sensitive nor specific enough to diagnose food allergy; (2) atopy patch tests for the evaluation of non-contact food allergy; and (3) the use of SPTs, IgE tests and atopy patch tests in combination. By comparison, United Kingdom (UK) National Institute of Health and Clinical Excellence (NICE) guidelines recommended the use of medical history, physical examination, SPTs and allergen-specific serum IgE measurements as joint diagnostic tools. These guidelines concluded that neither atopy patch testing nor oral food challenges should be used to diagnose IgE mediated allergy in primary care or community settings as their accuracy varies widely and is less well standardised. The NICE guidelines stated that one diagnostic method should not determine the presence, or lack thereof, of a food allergy.

To measure the prevalence and incidence of food allergy worldwide, appropriate population surveillance methods are needed. Few data sources are available that can be used to make statistically robust, reliable estimates of food allergy prevalence among children, on a nationally representative basis. From a public health perspective, there is a need for population-based estimates of the prevalence of food allergies based on objective diagnostic criteria rather than subjective questionnaire or interview data which may overestimate the prevalence and contribute to further uncertainty among health professionals, health policy makers, food producers and retailers.

An obstacle to the investigation of trends over time on a world population scale, as defined by the International Study of Asthma
and Allergies in Childhood (ISAAC), had been the availability of a suitable and generally accepted method to measure prevalence and severity of food allergy, and of other atopic diseases (e.g. atopic asthma) in children. In 1991, ISAAC was developed to address this issue in addition to creating a coordinated research programme that would be able to collect and analyse comparative data globally.

ISAAC was formed to facilitate epidemiological research into asthma, allergic rhinitis and eczema by promoting a standardised methodology that could be utilised around the world. Although successful in collecting the aforementioned data on a worldwide scale, the prevalence of food allergy was measured in just three countries. In a French study, prevalence of food allergy and its associations with respiratory manifestations among schoolchildren was assessed (Pénard-Morand et al., 2005). The second study compared the prevalence of self-reported food allergy and IgE antibodies to food allergens in wheezing and non-wheezing Estonian and Swedish school children (Sandin et al., 2005).

**Spectrum of clinical presentation of food allergy**
Adverse food reaction symptoms vary based on the patient, allergen, route of exposure and whether the reaction is IgE or non-IgE mediated. The clinical severity of reactions that people experience varies enormously. Mild, moderate and severe food-related reactions can occur when a particular food is ingested by different individuals. Urticaria is an example of a mild reaction. Urticarial reactions are characterized by pale, localized swellings of the skin with a surrounding erythematous border. These pruritic lesions range from 1 to 3 mm to several centimetres in diameter, and each may be round to serpiginous in configuration.

Food induced anaphylaxis is an example of a severe reaction. Food induced anaphylaxis is an IgE-mediated, generalized, clinical
reaction to a food because of mast cell/basophil chemical mediator release after first sensitization and then re-exposure to the same food (Anderson, 2000; Boyce et al., 2010). Anaphylactic reactions (IgE-dependent) affect one or more target organs: the skin (urticaria, angio-oedema), respiratory tract (rhinitis, asthma), gastrointestinal tract (pain, emesis, diarrhoea), and cardiovascular system (anaphylactic shock) (Sicherer, 2002).

**Food allergy as a component of the atopy disease spectrum**
Atopy is a personal and/or familial tendency to become sensitized and produce IgE antibodies in response to ordinary exposures to allergens, usually proteins. Food allergy is an atopic disease and as such shares some similarities and overlaps with other atopic diseases; atopic asthma, atopic eczema and allergic rhinitis (Vickery, Chin, & Burks, 2011). Children with food allergy are two to four times more likely to have other related atopic conditions such as asthma and eczema, compared with children without food allergies. When co-occurring conditions such as food allergy and asthma are present, the individual is at an increased risk of having a fatal reaction following food allergen exposure (Bird & Burks, 2009).

It is not known how to prevent atopic disease and food allergy in particular. In some cases, childhood food allergies resolve. For decades, the prevention of food allergies focused on altering the maternal or infant diet. The three primary areas of focus include the maternal diet during pregnancy and lactation; the infant’s early exposure to breast milk or a commercial formula, if given; the type of formula; and the timing and types of complementary foods. The primary goal of food allergy prevention strategies using dietary means is to prevent sensitization and allow tolerance to develop.
To date no studies have shown a reduction in specific food allergies based on these proposed prevention strategies.

The lack of definitive data to support each of these preventive strategies may be due in part to study design limitations (Du Toit & Lack, 2011). Ethical issues arise with study design of food allergy prevention studies. For example, randomization of infants to anything but breast milk would be unethical. A second limitation to studies within the field includes the phenotypic description of food allergy. Although eczema and food allergy are strongly associated the two are not synonymous and therefore the use of eczema as a surrogate has in itself many limitations (Du Toit & Lack, 2011). In addition, selection bias and reverse causality afflict nutritional interventions. Food allergy as a health problem evolves over time, particularly in children in whom it frequently resolves within a relatively short space of time. Lastly, when conducting nutritional studies the use of placebo is not always practical, or safe.

Alternative hypotheses that seek to explain the aetiology of food allergy include the hygiene hypothesis, topical sensitization, caesarean section, antacid medication use by infants, food processing methods, vitamin D exposure, alterations in dietary fat consumption and delayed introduction of potentially allergenic foods (Mullins, Dear, & Tang, 2009). A review of the literature that considers the potential contribution of these to the current food allergy disease burden is presented in Chapter 6.

**Prevalence, incidence, and trends world-wide**
There are growing concerns worldwide that the disease burden from childhood food allergy is rising. However, estimating the true disease burden of food allergy is difficult. As discussed above a lack of accurate and generalizable data regarding incidence and prevalence of food allergy world-wide is due in large part to the
challenge of an appropriate and reproducible standard diagnostic tool.

To date, studies worldwide have used both subjective and objective measurement tools to describe population rates of food allergy. Methods that have been used include self-report, medical history, skin prick test, serum specific IgE measurements, oral food challenges and health care utilisation, acute allergic reactions (e.g. unscheduled office or clinic visits, emergency department visits or hospitalisations). There are strengths and limitations to methods currently available (Table 2).

**Table 2.** Description of food allergy data collection tools

<table>
<thead>
<tr>
<th>Measurement Tool</th>
<th>Strengths</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self-report</td>
<td>• Easier to establish a large sample size</td>
<td>• Recall bias</td>
</tr>
<tr>
<td></td>
<td>• Less expensive</td>
<td>• Lack of understanding of food allergy v food intolerance</td>
</tr>
<tr>
<td></td>
<td>• Quick to conduct</td>
<td></td>
</tr>
<tr>
<td>Self-report + clinical follow-up (e.g. SPT)</td>
<td>• Easier to establish a large sample size</td>
<td>• Without a food challenge or definitive clinical diagnosis cannot be confirmed</td>
</tr>
<tr>
<td></td>
<td>• Confirmation testing can be provided</td>
<td></td>
</tr>
<tr>
<td>Hospitalisation data</td>
<td>• Easier to establish a large sample size</td>
<td>• Cannot confirm food allergy diagnosis without follow-up data</td>
</tr>
<tr>
<td></td>
<td>• Inexpensive</td>
<td>• Only captures severe end of clinical spectrum</td>
</tr>
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</table>

Variation between measurement tools selected contribute to some of the reported differences in prevalence rates worldwide (Table 3). For example, in a meta-analysis published by Rona et al. in 2007, self-reported prevalence of food allergy varied from 1.2% to 17% for milk, 0.2% to 7% for egg, 0% to 2% for peanuts and fish, 0% to 10% for shellfish, and 3% to 35% for any food (Rona et al., 2007). On the other end of the spectrum lies the DBPCFC which has been considered the gold standard. Prevalence rates based on DBPCFC described in a review by Rona et al. showed less variability
between studies as compared to values from self-report. For example, the prevalence for milk allergy per DBCFC varied from 0% to 3%, for egg from 0 to 1.7% and for any food from 1% to 10.8%.

Table 3. Estimates of prevalence of food allergy and hospital presentations due to an adverse reaction to food over time by data collection tool: Self-report, self-report with clinical follow-up, and hospital presentation

<table>
<thead>
<tr>
<th>Self-report</th>
<th>Self-report with clinical follow-up (e.g. SPT)</th>
<th>Hospital presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Emmett, Angus, Fry, &amp; Lee, 1999) Great Britain, 0-14y, Prevalence: 0.61%</td>
<td>(Kristjansson et al., 1999) Iceland and Sweden, 18 month, Prevalence: 2.0%</td>
<td>(Low &amp; Stables, 2006) Auckland, New Zealand, anaphylaxis fatalities due to food 1985-2005: 2 (11% of all anaphylaxis fatalities)</td>
</tr>
<tr>
<td>(Sicherer &amp; Muñoz-Furlong, 1999) United States, &lt;18y, Prevalence: 0.4% (0.6% peanut and/or tree nut)</td>
<td>(Dalal et al., 2002) Israel, 0-2y, Prevalence: 1.2%</td>
<td>(Branum &amp; Lukacs, 2009) United States, &lt;18y: 116,000 food-related visits (1993-1997); 247,000 (1998-2002; 317,000 (2003-2006)</td>
</tr>
<tr>
<td>(Kanny et al., 2001) France, 1-15y, Prevalence: 3.24%</td>
<td>(Roehr et al., 2004) Germany, 0-17y, Prevalence: 3.5%</td>
<td>(Liew, Williamson, &amp; Tang, 2009) Australia, anaphylaxis fatalities due to food 1997-2005: 7 (6% of all anaphylaxis fatalities)</td>
</tr>
<tr>
<td>(Sicherer, Munoz-Furlong, &amp; Sampson, 2003) United States, &lt;18y, Prevalence: 0.8% (1.2% peanut and/or tree nut)</td>
<td>(Osterballe, Hansen, Mortz, Høst, &amp; Bindslev-Jensen, 2005) Denmark, 3y, Prevalence: 2.3%</td>
<td>(Rudders et al., 2010) United States, food-related anaphylaxis visits per 10,000 total ED visits &lt;18y; (2001-2002: 15.3); (2003-2003: 15.5); (2005-2006: 28.7)</td>
</tr>
<tr>
<td>(Rancé, Grandmottet, &amp; Grandjean, 2005) France, 2-14y, Prevalence: 6.7% (cumulative prevalence); 4.7% (point prevalence)</td>
<td>(Pereira et al., 2005) United Kingdom, 11y &amp; 15y, Prevalence: 2.3%</td>
<td>(Mullins, Dear, &amp; Tang, 2015) Australia, food-related anaphylaxis admissions per 100,000 population per year: (1998-1999: 2.0); (2004-2005: 4.5); (2005-2006: 5.6); (2011-2012: 8.2)</td>
</tr>
<tr>
<td>(Branum &amp; Lukacs, 2009) United States, &lt;18y, Prevalence: 3.3% (1997), 3.2% (1999), 3.5% (2001), 3.6% (2003), 4.0% (2005), 3.9% (2007)</td>
<td>(Branum &amp; Lukacs, 2009) United States &lt;18y, Prevalence: 9% (peanut); 7% (egg), 12% (milk), 5% (shrimp)</td>
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<tr>
<td>(Sicherer, Munoz-Furlong, Godbold, &amp; Sampson, 2010) United States, &lt;18y, Prevalence: 1.4% (2.1% peanut and/or tree nut)</td>
<td>(Kagan et al., 2003) Canada, 5-9y, Prevalence: 1.5%</td>
<td></td>
</tr>
<tr>
<td>(Ben-Shoshan et al., 2010) Canada, &lt;18y, Prevalence: 1.03% (peanut), 0.69% (tree nut), 0.06% (shellfish), 0.03% (sesame)</td>
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</table>

The currently available epidemiological data provides an incomplete description of childhood food allergy worldwide. In addition to the
uneven distribution of studies globally, design factors limiting completeness include sample size, response rates and response bias (those who respond are more likely to have a food allergy), highly selected populations, lack of completion of confirmatory testing, lack of subsequent testing and limitation of studies to a subpopulation of those with food allergy, for example as happens when only those children that have been hospitalised are studied. A narrative follows providing a more comprehensive description of the studies conducted to aid in the development of a picture of the worldwide prevalence of food allergy in children.

**Rates based on self-report**
Self-reported food allergy is likely to overestimate the true prevalence. Self-report data is exposed to several forms of measurement error including those caused by low response rates, selection bias, limitations of the survey tool to identify true allergy, inherent bias because of the subjectivity of the answers, recall bias and the possibility of missing out on identifying transitory food allergy. A telephone survey may over represent a higher socioeconomic population due to the exclusion of homes without telephones and increased likelihood of selecting homes with multiple voice lines, a decrease in participation due to privacy concerns and telemarketers, and due to the availability of a household member.

In an epidemiologic survey in France, validation of the questionnaire used to determine the presence of food allergy was conducted in order to strengthen the data collection process (Kanny et al., 2001). This involved a pilot sample of 15 subjects to ensure that the questions were intelligible and comprehensible, verifying that the subjects understood the question correctly and the subjects’ situations were precisely described. At the conclusion of a more detailed second phase of the study that only included the 1129 of the 33,110 surveyed subjects in whom a food allergy was present,
researchers verified that there was no bias in the phase two sample as compared to phase one (Kanny et al., 2001).

The first phase of this study involved distributing a questionnaire to a representative sample of the French population. At the completion of this phase, 1129 persons with food allergy were selected to receive a second questionnaire. Corrections were made to the prevalence estimate from the phase one sample using the results of the follow-up telephone calls. Differences identified between the answers provided by subjects in phase 1 as compared to phase 2 led researchers to contact subjects to determine the reasons for these differences, 143 subjects participated (Kanny et al., 2001). The reported prevalence of present food allergy was 3.24%. Whether or not laboratory testing had been used to establish the presence of food allergy was determined. Further allergic testing had not been completed for 50% of the younger children with food reactions, however when allergy evaluation was performed a food allergy was confirmed in 85% of patients who self-reported food allergy. An additional French study investigated the prevalence of food allergies in a population of school age children in Toulouse (Rancé et al., 2005). Questionnaires were distributed to 150 classes in eight schools, and parents were asked to provide information pertaining to allergen testing similar to previously discussed study. However, this study included one phase with no additional follow-up and was targeted to school age children. Based on the information obtained from the questionnaires (78% return rate) the point prevalence for children diagnosed with a food allergy was reported as 4.0% (2-5 years), 6.8% (6-10 years), and 3.4% (11-14 years) (Rancé et al., 2005). Similar to the first study described, for a significant proportion (32%) of the children no laboratory evaluation of the food allergy had been completed.
The prevalence of food allergy was investigated in the United States (US) within the National Health Interview Survey, a nationally representative survey in which data are collected in a consistent manner each year from a large sample size. Prevalence rates of food allergy among all children (<18 y) increased significantly from 1997 (3.3%) to 2007 (3.9%), an 18% (p<0.01) increase in the proportion of children with a reported food or digestive allergy in the previous 12 months (Branum & Lukacs, 2009). An additional US study investigating trends over time of self-reported peanut, tree nut, and sesame allergy from 1997-2008 reported an increase in prevalence (Sicherer et al., 2010). This nationwide, cross-section random digit dial telephone survey sought to minimise selection bias related to availability by calling between the hours of 3 and 9pm (local time) Monday through Thursday and 10am until 5pm Eastern standard time on Saturday, a random sampling of telephone numbers were obtained for the 48 contiguous states. Participants were questioned regarding the presence of an allergy, and upon a positive result, questioning continued to determine details of the allergic reactions and history of other allergic diseases. Of those interviewed in 2008 (the 11th year of the study) the prevalence of peanut or tree nut allergy for children <18 years was 2.1% compared with 1.2% in 2002 (P=0.007) and 0.6% in 1997 (P<0.001) (Sicherer et al., 2010). This increase was primarily due to the increase in reported peanut allergy among children <18 years old. Whilst acknowledging the limitations associated with self-reported data these two studies from the US do provide data that allows temporal trends to be described in large and national representative samples.

**Rates based on self-report with clinical follow-up**

Studies in which both self-reported data and medical follow-up data are collected can allow for quantification of the overestimate of food
allergy prevalence in children that occurs when only self- or parent-reported data are used and thus can provide a more accurate prevalence estimate. Supportive clinical history (e.g. SPT, DBPCFC), as defined by each individual study, is collected prior to determining which children have a food allergy. An individual’s history is then corroborated with this confirmatory testing. However, in some instances where this approach has been used, there has been an underuse of confirmatory tests to both refute the self- or parent reported diagnosis of food allergy and incomplete availability of clinical and laboratory data to confirm the presence of food allergy. The precision and accuracy of food allergy estimates from such studies is also limited by reliance on laboratory tests which indicate sensitisation but which on their own do not establish a diagnosis of food allergy. Also variability in IgE cut-offs values used to define sensitisation adds additional uncertainty.

The discrepancy that occurs between estimates of the prevalence of food-allergy as defined by self-report compared with challenge-confirmed food hypersensitivity was demonstrated in a Danish cohort of 111 children <3 years old, 486 children 3 years old, 301 children older than 3 years of age and 936 adults (Osterballe et al., 2005). Almost a quarter (23%) of children were identified by parental self-reported as food hypersensitive. The prevalence of food hypersensitivity as defined by a positive oral food challenge was 2.3% for children aged 3 years, 1% of children older than 3 years and 3.2% for adults (Osterballe et al., 2005).

Using objective laboratory tests including skin prick tests and double blind food challenge tests, food allergy prevalence was confirmed in 2% of 18 month Icelandic and Swedish children enrolled in a prospective multicentre comparative study (Kristjansson et al., 1999). The study concluded that one can
expect to confirm food allergy in approximately one out of 15 children with reported adverse reactions to food. A similar study conducted in Germany collected data using a randomly sampled, cross-sectional population-based survey of children 0 to 17 years old. The study concluded that self-reported food allergy could be confirmed in around one out of 10 individuals, with clinical symptoms confirmed through skin prick tests, blood tests (RAST), and food challenges (both OFC and DBPCFC) in 3.5% of the participant population (Roehr et al., 2004).

A cross-sectional study involving kindergarten through grade 3 classrooms concluded a prevalence of peanut allergy at 1.5% (Kagan et al., 2003). Prevalence was confirmed with skin prick tests (SPTs), peanut-specific IgE measurement, and double-blind placebo-controlled food challenges (DBPCFCs) with peanut. 4,339 children provided information on the initial questionnaire at a mean age of 7 years (Kagan et al., 2003). Confirmatory objective tests (supportive clinical history, peanut specific IgE, or a positive DBPCFC result) were used to determine prevalence, therefore the number of peanut allergic children identified was considered to be an accurate and precise estimate. Through the technique of multiple imputation, both partial and non-responders were included in the analysis. These methods calculate the patient-specific probability of peanut allergy based on the available information, while taking into account the fact that the information is imperfect (Kagan et al., 2003). When those who had withdrawn prior to testing were included the prevalence of peanut allergy was estimated to increase to 1.8%. When including non-responders the estimated prevalence fell to 1.3% (Kagan et al., 2003).

Rates based on hospitalisation data
Due to difficulties inherent with the establishment of stable and repeatable measures of food allergy prevalence in the community or
primary care settings, hospital admissions for food allergy and ED visits for adverse reactions related to food have been used, for example Australia and the US, to examine changes in food allergy prevalence rates over time. In general, cases are identified by extensive chart review and/or the classification of ICD diagnostic codes. Using ICD codes for tracking patients has its limitations since symptoms and signs may not be correctly coded which may lead to under-estimation of the number of hospital visits. In addition, hospital admissions only represent a small proportion of symptomatic cases of these conditions. Data extracted via ICD codes can be used to report on prevalence of food allergy but only the prevalence of those allergies that result in severe adverse reactions necessitating hospital presentation. Hospitalisation data does provide some uniformity of measurement of severity over time, due to the fact an individual has to have a similar degree of illness to be hospitalised in different countries and across different time periods. Such data can provide an opportunity to investigate temporal trends in food allergy and adverse reactions due to food. A benefit of this approach is that data can be extracted over longer periods of time and used to describe trends over time in an efficient and economical manner. However, in some cases changes in visit frequency with time may be attributable to the variability in the use of general diagnostic codes in conjunction with other factors that influence hospital presentation more broadly. Underreporting does occur with the use of ICD-9-CM codes and chart review (Clark et al., 2004; Clark, Gaeta, Kamarthi, & Camargo, 2006; Ross et al., 2008). When interpreting data from such studies, it is also necessary to consider increased awareness of food allergy by physicians, other health care providers, and parents when considering whether changes in emergency room visits and hospital admissions are due to a true increase in the prevalence of this severe end of the disease spectrum.
In the US, based on the 1993-2006 National Hospital Ambulatory Medical Care Survey (NHAMCS) and National Ambulatory Medical Care Survey (NAMCS), Branum and Lukacs reported that hospitalisations with any recorded diagnoses related to food allergies increased from an average of 2600 discharges per year in 1998-2000 to 9500 discharges per year in 2004-2006 (P <0.01). In addition the average numbers of ambulatory care (emergency and outpatient departments and physician's offices) visits per year among children <18 years of age increased over a 15 year period; 116 000 (1993-1997), 247 000 (1998-2002) and 317 000 (2003-2006) (Branum & Lukacs, 2009). In a study conducted in Boston, US, a two fold increase in food-induced anaphylaxis visits by children to the Emergency Department of Boston Children’s Hospital was observed from 2001 to 2006 (Rudders et al., 2010).

In Australia from 1994/1995 to 2004/2005, hospital admissions for food-related anaphylaxis increased more than five-fold among children age 0 to 4 years old (Poulos et al., 2007). Within this age group a larger average annual increase was apparent amongst boys (18.0%) as compared to girls (10.7%) (Poulos et al., 2007). In a study conducted in Australia, food-related anaphylaxis admission rates (per 100,000 people/yr) increased amongst all ages over a 15 year period; 2.0 (1998-1999), 4.5 (2004-2005), 5.6 (2005-2006), and 8.2 (2011-2012) (Mullins et al., 2015). The largest increase over time was seen amongst children aged 0 to 4 (1.4-fold increase 2005/2006 to 2011/2012) with the largest proportionate increase over time in those aged 5 to 14 (2.1-fold increase) (Mullins et al., 2015).

Two studies were included that investigated fatalities due to food-related anaphylaxis based. The first study conducted in Auckland City Hospital, NZ, examined the coronial autopsy database to determine the number of anaphylaxis deaths between 1985 and
2005 (Low & Stables, 2006). Eighteen deaths due to anaphylaxis were identified with 2 (11%) determined to be due to food. A second study conducted in Australia, examined death certificate codes as part of the national database to determine the causes and demographics of deaths due to anaphylaxis between 1997 and 2005 (Liew et al., 2009). Over this 9 year time period 112 deaths were identified with 7 (6%) determined to be caused by food. Overall fatalities related to food-related anaphylaxis are quite rare.

**Rates based on cohort studies**
A cohort design, particularly one with a broad research agenda is able to gather information on potential exposures in earlier life prior to the onset of food allergy and also obtain a description of such exposures which is unlikely to be influenced by the child’s subsequent development of a food allergy. This study design however is not without its own set of limitations (Table 4).

**Table 4. Description of food allergy study design**

<table>
<thead>
<tr>
<th>Study Design</th>
<th>Strengths</th>
<th>Limitations</th>
</tr>
</thead>
</table>
| Cohort       | • Established methods can provide consistency between studies  
               • Confirmation testing can be provided | • Expensive  
               • Selection bias |

Cohort studies are labour, cost and time intensive, and if significant attrition occurs, may result in population loss to follow-up which may in turn lead to a study sample which becomes biased over time. However they allow for collection of data prospectively for a defined population sample and, in comparison with self-report, have the advantage of being less vulnerable to both selection and recall bias when assessing prevalence and identifying potential risk factors (Table 5).
Table 5. Estimates of prevalence of food allergy over time by study design: Cohort study

<table>
<thead>
<tr>
<th>Cohort study</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Hill et al., 1997) Australia, 0-2y, Prevalence:</td>
<td>3.2% (egg), 2.0% (milk), 1.9% (peanut)</td>
</tr>
<tr>
<td>(C Venter et al., 2010) United Kingdom, 3-4y, Prevalence:</td>
<td>0.5% (1989), 1.4% (1994-1996), 1.2% (2001-2002)</td>
</tr>
<tr>
<td>(Osborne et al., 2011) Australia, Prevalence:</td>
<td>10% of 12-month-olds</td>
</tr>
</tbody>
</table>

For example a UK study, conducted in the Isle of Wight, investigated trends over time in the prevalence of peanut allergy based on three cohort studies: Cohort A, Cohort B, Cohort C, with the children enrolled in each cohort born in 1989, 1994-96 and 2001-02 respectively. Less than 50% of children born between 1994 to 1996 (Cohort B) consented to the medical questionnaire, which was conducted when the children were 3 to 4 years old, thus they may be a highly selective population (Venter et al., 2010). In all three cohorts the children were assessed for peanut sensitisation and clinical symptoms, in addition to other study objectives. To confirm sensitisation children in all three cohorts were skin prick tested (SPT) and in the two more recent cohorts (Cohorts B and C), open food challenges (OFC) were conducted to confirm the diagnosis. Children born in 1989 (Cohort A) did not undergo any food challenges. In some instances confirmed sensitisation supported by clinical history resulted in diagnosis. Children with a positive reaction were invited to participate in a double-blind, placebo-controlled food challenge (DBPCFC) but none of the parents were prepared to consent to this (Venter et al., 2010). Although there are distinct differences between the three cohorts being compared it was still possible to investigate trends in food allergy prevalence over time. A significant increase in clinical peanut allergy was reported in children born from 1994 to 1996 (1.4%, p=0.02) as compared to 1989 (0.5%). A subsequent decrease, however not significant, was seen in children born between 2001
and 2002 (1.2%, p=0.85). Approximately 3,400 children participated over the 13 year period.

In the Melbourne Atopy Cohort Study (MACS), 620 Australian infants, who because their parents were atopic, were at increased risk for development of atopic disease were tested for food allergy by skin prick testing (Hill et al., 1997). As previously discussed, children with food allergy are two to four times more likely to have other related atopic conditions such as asthma and eczema, compared with children without food allergies. This study is an example of a selective study population which may lead to a biased estimate of food allergy prevalence. In this instance, data collected was utilised to extrapolate to a random community population in Australia and concluded a true prevalence of egg allergy of 3.2%, cow’s milk allergy of 2.0%, and peanut allergy of 1.9% at the age of two years (Hill et al., 1997). Extrapolation was validated through the use of additional data from a random population in Toowoomba, Queensland.

Using a sampling frame designed to recruit a representative population, the HealthNuts study in Melbourne, Australia set out to describe the prevalence of food allergy in infants (12-month-olds) through the use of multiple assessment tools including the gold standard oral food challenge (Osborne et al., 2011). Of those approached, 2848 agreed to participate with 10.4% (95% CI, 9.3-11.5) of participating infants identified as having a challenge-proven IgE-mediated food allergy (Osborne et al., 2011). Specifically, peanut allergy was identified in 2.9% (95% CI, 2.3-3.6). Similar to previous studies discussed, as compared to nonparticipants, participants in the HealthNuts study were more likely to be boys, have a history of eczema, and more often had a first-degree relative with a history of atopic disease. Findings from this study
are also in line with the high rate of other allergic disease, including asthma and eczema, in Australia (Asher et al., 2006).

**Chapter conclusions**

In summary, for the purposes of this thesis a food allergy is defined as an immunologically medicated adverse reaction to food triggered by an IgE mediated immune mechanism (Anderson, 2000). Food allergy is an atopic disease and as such shares some similarities and overlaps with other atopic diseases; atopic asthma, atopic eczema and allergic rhinitis. The manifestation of a food allergy, an acute allergic reaction, can range in severity from an urticarial rash to life threatening food-induced anaphylaxis. Adverse food reaction symptoms vary based on the patient, allergen, route of exposure and whether the reaction is IgE or non-IgE mediated. Defining and measuring food allergy prevalence and the temporal trends associated with adverse reactions to food is challenging. Variation in causal foods occurs in different global regions.

Based on the literature presented it is evident that there has been a change in the rates of childhood food allergy. The extent of this increase, the rate at which it has occurred, and current prevalence rates worldwide continue to be debated. All study designs used to assess the prevalence of food allergy have their limitations and strengths. For example, self- or parent reported data on peanut allergy shows much closer approximation to more robust prevalence measures than is evident for other food allergens, for example egg and dairy. In general, a consistent approach is needed; it is not possible to explore trends over time when multiple different methods are used by the studies being compared. Thus this highlights the need for international collaborative studies that employ standardised methods to minimize variation caused by response bias, methodology, and technology.
Currently there is limited published data in New Zealand regarding both trends over time and prevalence rates of food allergy. One study describes the prevalence of adverse reactions to food in New Zealand children age 0-5 years (Crooks et al., 2010). Data was collected from caregivers/parents of 110 children through a cross-sectional survey as part of the Royal New Zealand Plunket Society. Out of 44 children who had experienced an adverse reaction to food, only four were clinically evaluated and had undergone diagnostic testing (Crooks et al., 2010). An adverse food reaction is not an adequate measure of food allergy, however the results of this data indicate that in children with an adverse food reaction only four had been clinically evaluated and undergone diagnostic testing. Based on these results further research on food allergy in New Zealand is justified.

The lack of published data in New Zealand and the worldwide change in the rates of childhood food allergy provide strong support for the need for research in this area. As a first step hospital presentations for food-related acute allergic reactions were investigated at the national level. Presentations for acute allergic reactions have been used in other countries to investigate temporal trends.

In the next chapter, two studies are presented that provide a snapshot of trends over time for food-related acute reactions based on hospital presentations for children aged 0 to 14 years from 1988 to 2011.
Chapter 3: NATIONAL TRENDS IN HOSPITAL PRESENTATIONS FOR ACUTE ALLERGIC REACTIONS TO FOOD

Chapter objectives
In this chapter I describe temporal trends in New Zealand for hospital emergency department presentations with an allergic reaction to food. Due to difficulties inherent with the establishment of stable and repeatable measure of food allergy prevalence in the community or in primary care settings, hospital emergency department (ED) presentations for acute allergic reactions have been used in other countries to investigate temporal trends (Clark, Espinola, Rudders, Banerji, & Camargo, 2013; Clark, Espinola, Rudders, Banerji, & Camargo, 2011; Poulos, Waters, Correll, Loblay, & Marks, 2007; Rudders et al., 2010). While hospital event data only describes food-related allergic reactions at the severe end of the clinical spectrum, these data do allow for a measure of disease burden that is less subject to bias than those based upon self-report, or in the case of children, parental report.

My objectives for the projects reported in this chapter were to establish whether hospital event data could be used to describe temporal trends in allergic reactions to food in New Zealand and, if so, to describe trends over time in hospital presentations with allergic reactions to food.

New Zealand has a national collection of public and private hospital discharge information stored by the Ministry of Health in the National Minimum dataset (NMDS). The NMDS allows each person’s hospital presentation record over time to be investigated. Individual
hospital presentations are linked using the National Health Index (NHI) number, a unique identifier assigned to each person in NZ for use within health and disability support services.

To begin our investigation we obtained an anonymised data set from the NMDS of all emergency department (ED) presentations for food-related acute allergic reactions from 1988 to 2011 for children 0 to 14 years old. Upon initial analysis, it became evident that the majority of the episodes where a child presented to hospital with an acute allergic reaction it was coded as ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’. This led to the need for a better understanding of the use of these codes in NZ. Therefore this chapter is divided into two parts.

Part 1 describes a chart review of all presentations from 1988 to 2011 of children (0 to 14 years old) to the public hospital ED in the Auckland District Health Board (ADHB) region, for which the ICD codes for ‘anaphylaxis, unspecified’ or ‘allergy, unspecified, were assigned. Part 1 includes a manuscript, published in the peer reviewed literature, and is included exactly as submitted, apart from being formatted consistently with the remainder of the thesis (rather than included in the published format); pages, tables, figures, and references in this article have been renumbered so that numbering is continuous throughout the thesis (McMilin, Camargo, Morton, & Grant, 2015). Part 2 is an investigation of temporal trends throughout NZ in emergency department (ED) presentations for food-related acute allergic reactions from 1988 to 2011 of children (0 to 14 years old).
Abstract

Introduction
Emergency department (ED) visits for food-related acute allergic reactions enable estimation of temporal trends in food allergy prevalence. To use this approach in New Zealand requires an understanding of the proportion of ED visits coded as ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’ that are food-related allergic reactions.

Methods
We reviewed all ED presentations of children, coded as ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’, from 1988-2011 to the Auckland City Hospital ED. Charts were reviewed independently by two investigators to determine agreement on categorisation of presentations as being food-related acute allergic reactions. We compared ED presentation rates in different time intervals using rate ratios (RR) and 95% confidence intervals (CI).

Results
Sixty-five (29%) of the 221 ED presentations given a discharge code of ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’, were a food-related allergic reaction. Inter-observer agreement was very good (kappa >0.80). The ED presentation rate with food-related allergic reactions in 2004-2011 was 98% higher than in 1988-1995 (RR=1.98, 95%CI 1.10-3.72). By contrast, ED presentation rates for non-food-related allergic reactions did not change over these years.

Discussion
ED presentations for food-related allergic reactions are identifiable from within ED presentations coded as ‘anaphylaxis, unspecified’ or
‘allergy, unspecified’. ED presentations for food-related allergic reactions have increased over time in Auckland.

**Introduction**

Worldwide, food allergies have been the subject of much debate in recent decades due to what many observe as a dramatic increase in childhood food allergy prevalence, incidence, and severity (Moshe Ben-Shoshan, Turnbull, & Clarke, 2012; Burks et al., 2012; Sicherer, 2011; Sicherer & Sampson, 2014; Venter & Arshad, 2011). Despite New Zealand (NZ) having one of the highest prevalence of asthma worldwide, the epidemiology of other atopic diseases, including food allergy, has been poorly characterised (Asher et al., 2001; Asher, Stewart, Clayton, & Crane, 2008; The International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee, 1998).

Due to difficulties inherent with the establishment of stable and repeatable measures of food allergy prevalence in the community or in primary care settings, hospital emergency department (ED) presentations for food-related acute allergic reactions have been used in other countries to estimate temporal trends (Clark et al., 2013, 2011; Poulos et al., 2007; Rudders et al., 2010). Such an approach would seem particularly appropriate in NZ given that acute hospital based secondary care services are free, the hospital presentation data is stored and accessible at both a national and regional level, and each person having contact with health services in NZ is assigned a unique identifier: the national health index (NHI) number.

While hospital event data only describes food-related allergic reactions at the severe end of the clinical spectrum, these data do allow for a measure of disease burden that is less subject to bias than those based upon self-report, or in the case of children,
parental report. Rates of IgE-mediated food allergy can easily be
overestimated as people confuse IgE-mediated food allergy with
other, non-IgE-mediated, food intolerances. Rate estimates based
upon studies that use self-reported data are often times based upon
broad questions, for example ‘do you (or your child) have a food
allergy?’ (McGowan & Keet, 2013). A systematic review of studies
published from 1988 to 2009 showed that the prevalence of food
allergy estimated from self-report data is much higher than that
estimated when diagnosis is based upon skin prick testing, food
specific IgE determinations or food challenges (Chafen et al., 2010).
For example, based on the inclusion of 51 relevant studies, the
pooled estimate for cow’s milk allergy prevalence from self-report
was 3.5%, and from the other three methods was between 0.6%
and 0.9% (Chafen et al., 2010). Population estimates of food
allergy prevalence based upon self-reported data also vary widely
between studies, ranging from 1.2% to 17% for milk, 0.2% to 7%
for egg, 0% to 2% for peanuts and fish, 0% to 10% for shellfish,
and 3% to 35% for any food (Rona et al., 2007).

Episodes of acute allergic reactions that result in ED presentations
are identified by administrative codes based on the International
Classification of Diseases (ICD) system. These codes include both
those specific to food-related allergic reactions (e.g. contact
dermatitis due to food in contact with skin, dermatitis due to food
taken internally, anaphylactic shock due to peanuts) and those that
are more generic (e.g. anaphylaxis unspecified, allergy unspecified).

Inconsistent code assignment and lack of a universally-accepted
clinical definition for anaphylaxis have required that investigation of
ED presentations for food-related allergic reaction presentations
consider both visits coded as being food-related and visits coded as
being due to an anaphylactic or allergic reaction not further
specified (Clark et al., 2004, 2013, 2006; Gaeta, Clark, Pelletier, & Camargo, 2007; Rudders et al., 2010). In a study conducted across multiple United States EDs, relying solely on food specific ICD-9-CM codes resulted in identification of only 53% of patients presenting to the ED with a food-related allergic reaction whereas 87% of patients presenting with insect sting related allergic reactions were identified by the codes specific to insect sting related reactions (Clark et al., 2006). In another multi-site ED study from the United States, specifically of ED visits for food-related allergic reactions, 57% of patients were identified by using food-related allergic reaction codes and an additional 43% were identified from within less specific ICD-9 codes (Clark et al., 2004). Hence, the inclusion of only patients who are identified by ICD codes specific to food-related allergic reactions results in an underestimation of the true frequency of ED presentations (Clark et al., 2006; Poulos et al., 2007).

To describe temporal trends in food-related allergic reactions in NZ requires a more comprehensive understanding of the application of the commonly used administrative codes (i.e., ICD-9-CM-II and ICD-10-AM). Specifically, it is necessary to determine for what proportion of ED visits coded as ICD-9 codes 995.0 (ICD-10 T78.2) (anaphylaxis, unspecified) and 995.3 (ICD-10 T78.4) (allergy, unspecified) is the ED presentation due to a food-related allergic reaction?

Our objective was to complete a review of the ED presentations of children who presented to a public hospital ED between 1988 and 2011 with allergic reactions that were coded as either ‘anaphylaxis, unspecified’ and/or ‘allergy, unspecified’. From this review we sought to establish the proportion of children with an ED presentation, identified by the aforementioned ICD-9 and ICD-10
codes, in whom the presentation was caused by a food-related allergic reaction. Knowledge of this proportion is necessary before being able to utilise national ED presentation data to determine if there have been increases in food-related allergic reaction hospital presentations in NZ in recent decades.

**Methods**

We completed a chart review of all presentations from 1988 to 2011 of children (0-14 years old) to the public hospital ED in the Auckland District Health Board (ADHB) region, for which the ICD codes for ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’, were assigned. Although these codes only capture a subset of acute allergic reaction presentations, it is important to focus on these unspecified codes as they account for the majority of ED presentations that are not identified by food specific ICD codes. We chose this time period because it is the same as that for which data are available nationally. The ADHB operates NZ’s largest public hospital with almost two million patient contacts annually, serving more than 30% of the Auckland population (Auckland DHB Hospital and Related Services, 2015). The demographics of the ADHB population are broadly generalizable to the national population. Population estimates were based upon the national five-yearly census and intercensal estimates (Statistics New Zealand, 2014). Ethical and institutional approval for the project was granted by the ADHB Research Review Committee (Approval number A+ 6133).

Patient record data at ADHB is predominantly electronic. Once identified, patient charts were obtained from the ADHB. We reviewed these charts and extracted data to describe demographics and determine whether the hospital presentation was due to a food-related allergic reaction. Presentations were categorised as food-related, not food-related or due to an unknown cause (Table 6).
Table 6. Definitions used to categorise hospital presentations identified by ICD codes ‘anaphylaxis, unspecified*’ and ‘allergy, unspecified†’ in children (0-14 years).

<table>
<thead>
<tr>
<th>Food-related allergic reaction category</th>
<th>Features used to assign hospital emergency department visits to each category</th>
</tr>
</thead>
</table>
| Food-related                           | • History of atopic disease and generalised reaction shortly after consuming specified food  
Or                                      | • A previous episode of food-related allergic reaction with the reaction occurring in a location where food supervision was potentially not stringent and the history indicated timing in association with food consumption  
Or                                      | • A generalised reaction in association with food without documentation that exposure to a new food had occurred |
| Not food-related                       | • Non-food allergen identified  
• Localised reaction e.g. digit or eye |
| Unknown cause                          | • Generalised reaction not related to meal  
• No known allergen exposure or no allergen exposure documented  
• No past history of food-related allergic reaction |

* Anaphylaxis, unspecified = ICD-9 code 995.0 or ICD-10 code T78.2  
† Allergy, unspecified = ICD-9 code 995.3 or ICD-10 code T78.4

To ensure that data extraction was complete and allergic reaction categorisation was consistent, the two reviewers (CM and CCG) independently reviewed all of the patient charts and assigned each ED presentation as being due to a food-related allergic reaction, non-food-related allergic reaction or due to a reaction for which the cause was unknown. We then determined inter-observer agreement for this categorisation.

**Statistical analyses**
Agreement between investigators was calculated using a Kappa (κ) statistic. Kappa scores and 95%CI were calculated using SAS SAS-PC version 9.3 software. Kappa scores were defined as showing poor (κ ≤ 0.2), fair (κ >0.2 to ≤0.4), moderate (κ >0.4 to ≤0.6), good (κ >0.6 to ≤0.8) or very good (κ >0.8 to ≤1.0) agreement (Landis & Koch, 1977).
We compared the age of those for whom the ED presentation was or was not a food-related allergic reaction using the Wilcoxon-rank-sum test. We described the number of individuals in each year with an ED presentation that was food-related, not food-related or of unknown cause and determined whether the annual number of presentations in each of these three categories changed over time. We assumed that, in addition to being infrequent, presentations to the ED in each of these categories occurred independently of each other and at a constant rate (Sedgwick, 2014).

In view of the small number of ED presentations per year in each of the three categories (food-related, not food-related or unknown cause) and large year-to-year variability we grouped cases into eight-year intervals (i.e. 1988-1995, 1996-2003, and 2004-2011), and described the number of presentations per person-year in each interval. Using the first time interval as the reference period, we then determined if ED presentation rate in the other two subsequent eight year time intervals differed from this baseline interval, using rate ratios (RR) and 95% CIs. Because anaphylaxis is only identified in a proportion of patients making an ED presentation with anaphylaxis we combined ED presentations for which the code identified or did not identify the presence of anaphylaxis (Gaeta et al., 2007).

**Results**
Of the 248 records that met the inclusion criteria, 226 were reviewed by both reviewers. We included 221 (89%) in the analysis with the five excluded being duplicate presentations; we only counted the first event for the individuals who presented twice. None of the five presentations excluded were food-related acute allergic reactions. Of these 221 presentations 120 (54%) were coded as ‘anaphylaxis, unspecified’, and 101 (46%) as ‘allergy, unspecified’. Records reviewed by both investigators were
categorised with respect to the probability of the allergic reaction being due to a food allergen (Table 7).

**Table 7.** Categorisation of children aged 0 to 14 years presenting to the hospital emergency department with ‘anaphylaxis, unspecified*’ and ‘allergy, unspecified†’ from 1988 to 2011.

<table>
<thead>
<tr>
<th>Categorisation by investigator one n (%)</th>
<th>Food-related allergic reaction category</th>
<th>Food-related</th>
<th>Not food-related</th>
<th>Reaction to unknown cause</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food-related</td>
<td>58 (26)</td>
<td>2 (1)</td>
<td>3 (1)</td>
<td></td>
<td>63 (29)</td>
</tr>
<tr>
<td>Not food-related</td>
<td>5 (2)</td>
<td>125 (57)</td>
<td>13 (6)</td>
<td></td>
<td>143 (65)</td>
</tr>
<tr>
<td>Unknown cause</td>
<td>2 (1)</td>
<td>3 (1)</td>
<td>10 (4)</td>
<td></td>
<td>15 (7)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Categorisation by investigator two n (%)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food-related</td>
<td>65 (29)</td>
</tr>
<tr>
<td>Not food-related</td>
<td>130 (59)</td>
</tr>
<tr>
<td>Unknown cause</td>
<td>26 (12)</td>
</tr>
</tbody>
</table>

Total 221 (100)

* Anaphylaxis, unspecified = ICD-9 code 995.0 or ICD-10 code T78.2
† Allergy, unspecified = ICD-9 code 995.3 or ICD-10 code T78.4

Inter-observer agreement was very good for categorisation into a food-related allergic reaction versus all other visits (κ = 0.87) or only an allergic reaction not due to food (κ = 0.92) (Table 8).

**Table 8.** Agreement between researchers in categorising of hospital presentations

<table>
<thead>
<tr>
<th>Categories</th>
<th>Kappa score* (κ)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food-related vs. not food-related or unknown</td>
<td>0.87</td>
<td>0.80-0.94</td>
</tr>
<tr>
<td>Food-related vs. not food-related</td>
<td>0.92</td>
<td>0.85-0.98</td>
</tr>
</tbody>
</table>

* Inter-observer agreement classification: Poor (κ ≤ 0.2), fair (κ >0.2 to ≤0.4), moderate (κ >0.4 to ≤0.6), good (κ >0.6 to ≤0.8) or very good (κ >0.8 to ≤1.0) agreement.

Sixty-five (29%, 95% CI 24-36%) of the 221 hospital presentations with a discharge code of ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’, were identified as food-related allergic reactions. Of these 65 presentations 43 (19%) were coded as ‘anaphylaxis, unspecified’, and 22 (10%) as ‘allergy, unspecified’. Of the
remaining presentations, 130 (59%, 95%CI 52-65%) were not due to a food-related allergen and 26 (12%, 95%CI 8-17%) were due to an unknown allergen. Median age at presentation of those with a food-related allergic reaction (2.0 years) was less than those for whom the allergic reaction was not food-related (7.0 years, p<0.001) or was due to an unknown allergen (8.5 years, p = 0.02).

Change over time was evident for the rate of hospital presentations for food-related allergic reactions, but not for reactions that were non-food-related or for which the allergen was unknown (Figure 2). In comparison to the 1988-1995 time interval (average annual rate 0.85/100,000), the rate of presentation with food-related allergic reactions was lower from 1996-2003 (0.35/100,000, RR = 0.41) and higher from 2004-2011 (1.68/100,000, RR = 1.98) (Table 9).

**Table 9.** Comparison of rate of hospital presentations due to acute allergic reactions in Auckland that were coded as ‘anaphylaxis, unspecified*’ and ‘allergy unspecified†’: 1996-2003 and 2004-2011 versus 1988 to 1995.

<table>
<thead>
<tr>
<th>Time interval</th>
<th>Average population per year</th>
<th>Number of ED presentations</th>
<th>Rate ratio vs. 1988-95 (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Food-related</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1988-95</td>
<td>249270</td>
<td>17</td>
<td>1.00</td>
<td>---</td>
</tr>
<tr>
<td>1996-03</td>
<td>289047</td>
<td>8</td>
<td>0.41 (0.15-0.99)</td>
<td>0.03</td>
</tr>
<tr>
<td>2004-11</td>
<td>296841</td>
<td>40</td>
<td>1.98 (1.10-3.72)</td>
<td>0.02</td>
</tr>
<tr>
<td></td>
<td>Not food-related</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1988-95</td>
<td>249270</td>
<td>38</td>
<td>1.00</td>
<td>---</td>
</tr>
<tr>
<td>1996-03</td>
<td>289047</td>
<td>35</td>
<td>0.79 (0.49-1.29)</td>
<td>0.32</td>
</tr>
<tr>
<td>2004-11</td>
<td>296841</td>
<td>57</td>
<td>1.26 (0.82-1.95)</td>
<td>0.27</td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1988-95</td>
<td>249270</td>
<td>5</td>
<td>1.00</td>
<td>---</td>
</tr>
<tr>
<td>1996-03</td>
<td>289047</td>
<td>6</td>
<td>1.03 (0.26-4.29)</td>
<td>0.95</td>
</tr>
<tr>
<td>2004-11</td>
<td>296841</td>
<td>15</td>
<td>2.52 (0.87-8.86)</td>
<td>0.06</td>
</tr>
</tbody>
</table>

Abbreviation: ED, emergency department.
* Anaphylaxis, unspecified = ICD-9 code 995.0 or ICD-10 code T78.2
† Allergy, unspecified = ICD-9 code 995.3 or ICD-10 code T78.4
‡ CI = confidence interval
Figure 2. Trends in hospital presentations due to acute allergic reactions that were coded as ‘anaphylaxis, unspecified*’ and ‘allergy, unspecified†’

* Anaphylaxis, unspecified = ICD-9 code 995.0 or ICD-10 code T78.2
† Allergy, unspecified = ICD-9 code 995.3 or ICD-10 code T78.4

The proportion of presentations that were due to a food-related allergic reaction varied slightly between the 3 time intervals (p=0.04) however not in any directional way. Upon further investigation in which the proportions were grouped into six 4-yearly time intervals, the variance that was present was random with no statistically significant directional trend evident (p=0.07).


Discussion
Food-related acute allergic reactions accounted for 29% of hospital presentations that were assigned a discharge code for ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’. Identification of these cases permitted us to have a more complete picture of ED visits for
unspecified acute allergic reactions. In comparison with 1988-95, the ED presentation rate for food-related allergic reactions decreased during 1996-03 and then increased during 2004-11, with the average annual rate from 2004-11 being almost 100% higher than it had been from 1988-95. In contrast, there was no significant change over time in the hospital presentation rates for ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’ ED presentations that were not food-related or for which the allergen was unknown.

An unanticipated finding from this study was the decrease in food-related allergic reaction presentations during the time period 1996-03. A similar, but non-significant, reduction was also seen during this time interval in hospital presentation rates for acute allergic reactions that were not food-related. In July of 1995 (the start of the 1995/96 NZ financial year), NZ changed from using ICD-9-CM to ICD-9-CM-A discharge coding systems. As part of this change the National Coding Standards were introduced, including a coding standard for allergic reactions. These standards were introduced to improve consistency in applying the classifications across the hospital and health services of NZ. This change may have resulted in a more strict application of the ICD codes investigated in this study, potentially leading to a decrease in the number of ED presentations assigned these codes. Another factor that may potentially explain the decrease in ED presentations during this time period is that it was a period when the NZ health care system experienced some major structural changes which included the temporary introduction of part charges for public hospital presentations (Hornblow & Barnett, 2000).

Based on the data obtained from the ADHB, the code ‘anaphylaxis, unspecified’ (19%) was used for a larger proportion of the hospital presentations than was reported from a large United States study of
ED presentations for acute allergic reactions where <1% of all ED visits were coded as anaphylaxis (Gaeta et al., 2007). Based on other work by the authors of this US study (Clark et al., 2004; Clark, Long, Gaeta, & Camargo, 2005), which showed that approximately 51% of food-related allergic reactions and 31% of venom-related allergic reactions result in anaphylaxis, this very low proportion of ED presentations that were coded as anaphylaxis implies these codes cannot be used to reliably identify all ED presentations where anaphylaxis has occurred. Due to the variable documentation of presenting symptoms and signs in the Auckland records, we cannot determine from our study if the rate of hospital presentation with true anaphylaxis differs in NZ from that reported in the United States.

Our study represents the first attempt to estimate the frequency of hospital ED presentations of food-related allergic reactions in NZ. We have validated the methodology for identifying, at a national level, the food-related proportion of hospital presentations assigned a discharge code for ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’. Our data suggest that, consistent with observations reported from Australia and the US, the rate of such presentations has increased in NZ in recent years (Poulos et al., 2007; Rudders et al., 2010).

In comparison with other causes of ED presentations, food-related acute allergic reactions are relatively rare events. Our study only included a subset of these food-related allergic reactions as we were only investigating those ED presentations that were coded as ‘anaphylaxis, unspecified’ and ‘allergy, unspecified’. Because of these small numbers it was necessary to combine years to allow for temporal trends to be determined. As a result it was difficult to identify the true impact of events such as the 1995 change from using ICD-9-CM to ICD-9-CM-A codes. Nor were we able to
determine if there has been a plateauing in the rate of hospital presentations for food-related allergic reactions as has been reported recently from the US (Clark et al., 2013). A subsequent larger national study is likely to overcome the issues described as it will allow for the inclusion of the unspecified and specific food-related allergic reaction codes.

We showed very good inter-observer reliability in our assignment of ED presentations to the different food-related categories and to the food versus not-food related categories. No trend over time was observed in our estimate of the food-related proportion of hospital presentations coded as ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’. Thus it appears reasonable to infer that these observations are likely to apply to national data over the same time interval. It is important though to remain aware of the potential for the quality of such clinical data to change over time. For example, in 1992 the number of diagnosis codes for an event that were included in National Minimum Dataset reporting expanded from 4 to 25. Since this time they have been expanded even further to 99 codes which could lead to an increase in the number of codes used per event and thus potentially inflate the overall total number of presentations for which a food-related code was included.

Our study provides insight into the use of two unspecified ICD codes in one large NZ hospital ED and sets the foundation for future work on the epidemiology of food allergy and food allergy-related allergic reactions in NZ. We can now justify including a proportion of presentations from both ICD codes ‘allergy, unspecified’ and ‘anaphylaxis, unspecified’ in a national study of ED presentations of food-related allergic reactions. This will enable us to describe this aspect of food allergy epidemiology in a manner that will enable comparison with other countries.
Acknowledgments

We acknowledge Jan Sisley at the Auckland District Health Board (ADHB) for her assistance with obtaining both electronic and paper copies of the requested medical charts.
At the conclusion of the first study it was evident that when reporting on the hospital presentations due to acute allergic reactions related to food it would not be possible to identify a proportion of such presentations if we limited our analysis to hospital presentations identified solely by food specific ICD-9-CM codes. Our ADHB chart review showed us that a proportion of hospital presentations with acute allergic reactions related to food will have been coded as unspecified allergic or anaphylactic reactions. Therefore in part 2 of this chapter data presented from the NMDS pertaining to acute allergic reactions include an analysis of all such unspecified presentations.

When ICD codes ‘anaphylaxis, unspecified’ and/or ‘allergy, unspecified’ are included we know that only a proportion of these ED visits will have been for acute allergic reactions related to food. It is important to include these reactions though, as we saw in our validation study that the temporal trends in hospital presentations that were assigned these codes were determined by the visits that were due to food related allergic reactions rather than visits that were non-food-related or for which the allergen was unknown. Part 2 is a national investigation of temporal trends in emergency department (ED) presentations for food-related acute allergic reactions from 1988 to 2011 of children (0 to 14 years old).
Part 2: Hospital Presentations Due to Acute Allergic Reactions Related to Food

Abstract

Introduction
Emergency department (ED) visits for food-related acute allergic reactions enable estimation of temporal trends in food allergy prevalence. Hospital presentation admission data has been used in other countries, specifically Australia and the United States. These data allow for the investigation of changes in rates of hospital presentation with food-related acute allergic reactions over time and between population subgroups defined by other variables. Specifically, the objective of this study is to utilise emergency department presentations for food-related allergic reactions to describe epidemiological trends in food allergy prevalence in New Zealand children (0 to 14).

Methods
Using the National Minimum Dataset (NMDS), a national collection of public and private hospital discharge information, we examined 24 years (1988 – 2011) of data pertaining to acute allergic reactions that could be due to food for children 0 to 14 years old. We compared ED presentation rates in different time intervals using rate ratios (RR) and 95% confidence intervals (CI). Annual rates of hospital presentations were calculated using population estimates based upon the national five-yearly census and intercensal estimates. Temporal trends were described with Poisson regression.

Results
Between 1988 and 2011, 3,735 children 0 to 14 years old presented to a New Zealand hospital with an acute allergic reaction identified by ICD-9-CMA-II codes 692.5, 693.1, 995.0, 995.3, and 995.60-995.69. An average yearly increase of 8% in hospital presentations
due to acute allergic reactions (p<0.001) was observed. In comparison to the 1988 time interval (annual rate 7.11/100,000), the rate of all presentations was five times higher in 2011 (37.88/100,000, RR=5.33).

Discussion
Hospital presentations due to acute allergic reactions identified from pre-selected ICD-9-CMA-II codes have increased over the past two plus decades in New Zealand. This increase is seen throughout the country, in all ethnic groups, in both genders, and across the age range from 0 to 14 years.

Introduction
Data that describe hospital presentations for food-related acute allergic reactions has been used in other countries, specifically Australia and the United States to investigate the epidemiology of food allergy. The use of this data has allowed for the investigation of temporal changes (Branum & Lukacs, 2009; Clark et al., 2011, 2006; Clark, Espinola, Rudders, Banerji, & Camargo, 2013; Poulos et al., 2007; Rudders, Banerji, Corel, Clark, & Camargo, 2010). In the US, based on the 1993-2006 National Hospital Ambulatory Medical Care Survey (NHAMCS) and National Ambulatory Medical Care Survey (NAMCS), the average numbers of visits per year among children <18 years of age increased over a 15 year period; 116 000 (1993-1997), 247 000 (1998-2002), and 317 000 (2003-2006) (Branum & Lukacs, 2009). Unspecified codes were not included in this investigation, however the ICD codes identifying unspecified anaphylaxis were included in an Australian study (Poulos et al., 2007) and both unspecified codes were used in other US studies (Clark et al., 2011, 2006; Clark et al., 2013). In the study completed in Australia, from 1994/1995 to 2004/2005 hospital admissions for food-induced anaphylaxis increased more than five-fold among children 0 to 4 years old (Poulos et al., 2007).
Based on the findings reported from the aforementioned study on administrative coding, including presentations with both ICD codes ‘allergy, unspecified’ and ‘anaphylaxis, unspecified’ in a national study of ED presentations of food-related allergic reactions is appropriate. Based on the data obtained from the ADHB, the code ‘anaphylaxis, unspecified’ (19%) was used for a larger proportion of the hospital presentations in NZ than was reported from a large United States study of ED presentations for acute allergic reactions where <1% of all ED visits were coded as anaphylaxis (Gaeta et al., 2007). From these codes, food-related acute allergic reactions accounted for 29% of hospital presentations.

In our study of hospital presentations in the ADHB region of NZ food-related acute allergic reactions accounted for 29% of hospital presentations coded as ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’ (McMilin et al., 2015). In comparison with 1988-95, the average annual rate of food-related acute allergic reaction hospital presentations that were assigned these codes from 2004-11 was approximately 100% higher. In contrast, there was no significant change over time in the hospital presentation rates for ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’ hospital ED presentations that were not food-related or for which the allergen was unknown. The objective of this study therefore was to utilise the approach developed from the ADHB regional study to investigate hospital emergency department presentations for food-related allergic reactions at a national level and thus describe epidemiological trends in food allergy prevalence in New Zealand children 0 to 14 years old.

**Methods**

Using the National Minimum Dataset (NMDS), a national collection of public and private hospital discharge information, temporal trends in emergency department (ED) presentations for food-
related acute allergic reactions from 1988 to 2011 of children 0 to 14 years old were investigated. Presentations were identified by a hospital discharge during the study interval coded as general allergy attributable to food including but not limited to anaphylaxis. Based on a review of the literature a list of ICD codes was created that included all potential codes used worldwide to identify food-related acute allergic reactions, including anaphylaxis (Clark et al., 2004, 2011, 2006; Clark, Espinola, Rudders, Banerji, & Camargo, 2013; Gaeta et al., 2007; Poulos et al., 2007; Rudders et al., 2010).

From the codes reported to have been used in the peer reviewed literature a list of codes was created based on the needs of the study and appropriateness for the New Zealand context taking into account the ICD coding version used in NZ and the level of coding detail contained in the NMDS. This list was then applied to the National Minimum Dataset (NMDS).

As the data for this study was spread across multiple years and include multiple versions of ICD codes (i.e. ICD-9-CMA-II, ICD-10-AM-II, ICD-10-AM-III etc.) the NMDS backward maps to more recent ICD versions. For this study presentations were backwards mapped to ICD-9-CMA-II. Not all codes requested were present in the ICD-9-CMA-II and not all codes requested were present at the requested level of detail on the NMDS. In addition, the code V150 was not included in the analysis reported as this code, at times, was used in conjunction with other non-acute allergic reaction codes to denote history of allergy. The use of this code could have resulted in counting presentations that may not have been due to an acute allergic reaction. The final list used for analysis was therefore determined from both a review of the literature and the applicability to the New Zealand context (Table 10). A child who presented to the ED more than once during the established time frame was only counted once.
Upon extraction the codes were grouped into two categories, ‘skin-related’ reactions and ‘anaphylactic or allergic’ reactions. Anaphylactic shock due to unspecified or specified foods (995.60-995.69) were included in the ‘anaphylactic or allergic’ reaction group as the codes were only used for a few years in the 1990’s.

In addition to evaluating all presentations over time, subgroups were also investigated. Subgroups included those defined by gender, ethnicity, geographical location (city centres, and city versus urban versus rural), and age. Ethnicity groups were established based on the parameters defined by Statistics New Zealand. For analysis purposes, ethnic groups with small populations were categorized as other and included with European. The ethnicity groupings used in this study were European/Other, Maori, Pacific, and Asian. To investigate differences based on geographical location, two types of subgroups were created based on territorial local authority (TLA). The first grouping was created to allow for a comparison of children presenting to hospitals in the five largest NZ city centres; Auckland, Hamilton, Wellington, Christchurch, and Dunedin, which allowed for an investigation of differences in latitude from Auckland (37°S) to Dunedin (46°S). The second grouping was created to enable a comparison of children presenting to hospital in a city, urban, or rural area. Children presenting to hospital in a city centre (Auckland, Hamilton, Wellington, Christchurch, and Dunedin) were classified as city, children presenting to hospital in an area surrounding a city centre or designated urban area were classified as urban, and the remainder were classified as rural. Age groups were established based on the parameters set out by the NMDS. The age groupings used by the NMDS for children were 0 to 4 years, 5 to 9 years, and 10 to 14 years.
**Statistical analyses**
To determine annual rates of hospital presentations, census data from Statistics New Zealand was obtained for the following time periods: 1991, 1996, 2001, 2006, and 2013. Intercensal estimates were calculated for the years between census collections. For instances in which years are grouped together, the census collection closest to the year group are used as the denominator in rate calculations.

Presentation rates were compared in different time intervals using rate ratios (RR) and 95% confidence intervals (CI). For comparison between population subgroups defined by gender, ethnicity and geography, presentations were grouped into six 4-yearly time intervals due to low numbers in some categories. Statistical significance of temporal trends are determined using Poisson regression. Analyses were performed using SAS (Version 9.2) and StatsDirect. All P-values are two-tailed, with P<0.05 considered statistically significant.

**Results**
Between 1988 and 2011 there were 3,735 children aged 0 to 14 years who made hospital presentations that met the inclusion criteria. Of these presentations 772 (21%) were coded as food-related reactions affecting the skin (692.5, 693.1), 2877 (77%) were coded as unspecified acute allergic reactions including anaphylaxis (995.0, 995.3), and 86 (2%) were coded as anaphylaxis including a food related allergen (995.60-995.69). As the anaphylaxis specified codes (995.60-995.69) were only used from 1995 to 1999 they were grouped with the unspecified codes into an ‘anaphylactic or allergic’ group (Table 10).
**Table 10.** Total hospital presentations due to specified acute allergic reactions from 1988 to 2011 in children 0 to 14 years old.

<table>
<thead>
<tr>
<th>Definition</th>
<th>ICD-9-CM</th>
<th>Presentations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Skin related reactions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contact dermatitis and other eczema due to food in contact with</td>
<td>692.5</td>
<td>60 (2%)</td>
</tr>
<tr>
<td>Dermatitis due to food taken internally</td>
<td>693.1</td>
<td>712 (19%)</td>
</tr>
<tr>
<td><strong>Anaphylactic or allergic reactions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anaphylactic shock, not elsewhere classified</td>
<td>995.0</td>
<td>1912 (51%)</td>
</tr>
<tr>
<td>Allergy, unspecified</td>
<td>995.3</td>
<td>965 (26%)</td>
</tr>
<tr>
<td>Anaphylactic reaction due to unspecified food</td>
<td>995.60</td>
<td>7 (&lt;1%)</td>
</tr>
<tr>
<td>Anaphylactic reaction due to peanuts</td>
<td>995.61</td>
<td>25 (1%)</td>
</tr>
<tr>
<td>Anaphylactic reaction due to crustaceans</td>
<td>995.62</td>
<td>7 (&lt;1%)</td>
</tr>
<tr>
<td>Anaphylactic reaction due to fruits and vegetables</td>
<td>995.63</td>
<td>3 (&lt;1%)</td>
</tr>
<tr>
<td>Anaphylactic reaction due to tree nuts and seeds</td>
<td>995.64</td>
<td>5 (&lt;1%)</td>
</tr>
<tr>
<td>Anaphylactic reaction due to fish</td>
<td>995.65</td>
<td>2 (&lt;1%)</td>
</tr>
<tr>
<td>Anaphylactic reaction due to food additives</td>
<td>995.66</td>
<td>8 (&lt;1%)</td>
</tr>
<tr>
<td>Anaphylactic reaction due to milk products</td>
<td>995.67</td>
<td>15 (&lt;1%)</td>
</tr>
<tr>
<td>Anaphylactic reaction due to eggs</td>
<td>995.68</td>
<td>14 (&lt;1%)</td>
</tr>
<tr>
<td>Anaphylactic reaction due to other specified food</td>
<td>995.69</td>
<td>60 (&lt;1%)</td>
</tr>
</tbody>
</table>

Change over time was evident for the rate of hospital presentations for identified acute allergic reactions. There was a distinct difference between the rate of increase between the skin related and the anaphylactic or allergic reactions group (Figure 3).
Figure 3. Annual hospital presentations from 1988 to 2011 in children 0 to 14 years old due to skin related reactions to food, anaphylactic and allergic reactions (both unspecified and to specific foods), and all of these allergic reaction groups combined.

* Skin related presentations = ICD-9-CM code 692.5 or 693.1
† Anaphylactic or allergic = ICD-9-CM code 995.0, 995.3, or 9956.0-9956.9

In comparison to the 1988 time interval (annual rate 7.11/100,000), the rate of all presentations was consistently higher from 1997 onwards with rate ratios increasing from 1.43 and 1.55 in 1997 and 1998 to 6.06 and 5.33 in 2010 and 2011. For skin related reactions, in comparison with the 1988 rate, the rate of presentation was higher only in 1993, 2009, 2010 and 2011. For anaphylactic or allergic reactions, in comparison with the 1988 rate, the rate of presentation was higher in 1993 and 1994, and then from 1997 onwards (Table 11).

Over the time interval from 1988 to 2011 there has been an average yearly increase of 9% in hospital presentations due to all of the identified acute allergic reaction diagnostic groups (p<0.001). A
greater rate of increase was observed in the anaphylactic and allergic reactions group (average increase 11% per year, \( p < 0.001 \)) as compared to the skin related group (average increase of 2% per year, \( p < 0.001 \)).

With presentations grouped into six 4-yearly time intervals: 1988-91, 1992-95, 1996-99, 2000-03, 2004-07, and 2008-11, a statistically significant directional trend was evident, with a larger increase in rates in the first decade of the new millennium as compared to 1988-1991; 1992-95 (57% increase, \( p < 0.001 \)), 1996-99 (65% increase, \( p < 0.001 \)), 2000-2003 (310% increase, \( p < 0.001 \)), 2004-2007 (363% increase, \( p < 0.001 \)), and 2008-2011 (550% increase, \( p < 0.001 \)) (Table 12).

Upon stratification of the hospital presentations in these same six 4-yearly time intervals, change over time was evident amongst both genders, in all four ethnic groups, in children living in various geographic environments, and in children of different age groups. With the exception of age groups, presentations were grouped into six 4-yearly time intervals due to low numbers in some categories.
Table 11. Comparison of annual rates of hospital presentations from 1988 to 2011 in children 0 to 14 years old due to skin related reactions to food, anaphylactic and allergic reactions (both unspecified and to specific foods) and to all of these allergic reaction groups combined.

<table>
<thead>
<tr>
<th>Year</th>
<th>All</th>
<th>Skin</th>
<th>Anaphylactic or allergic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases</td>
<td>Rate (per 100,000)</td>
<td>RR (95% CI)</td>
</tr>
<tr>
<td>1988</td>
<td>56</td>
<td>7.11</td>
<td>1.00</td>
</tr>
<tr>
<td>1989</td>
<td>45</td>
<td>5.71</td>
<td>0.80 (0.53-1.21)</td>
</tr>
<tr>
<td>1990</td>
<td>60</td>
<td>7.61</td>
<td>1.07 (0.73-1.57)</td>
</tr>
<tr>
<td>1991</td>
<td>48</td>
<td>6.09</td>
<td>0.86 (0.57-1.28)</td>
</tr>
<tr>
<td>1992</td>
<td>65</td>
<td>8.15</td>
<td>1.15 (0.79-1.67)</td>
</tr>
<tr>
<td>1993</td>
<td>116</td>
<td>14.37</td>
<td>2.02 (1.46-2.83)</td>
</tr>
<tr>
<td>1994</td>
<td>98</td>
<td>12.00</td>
<td>1.69 (1.20-2.39)</td>
</tr>
<tr>
<td>1995</td>
<td>70</td>
<td>8.47</td>
<td>1.19 (0.82-1.71)</td>
</tr>
<tr>
<td>1996</td>
<td>74</td>
<td>8.85</td>
<td>1.25 (0.87-1.80)</td>
</tr>
<tr>
<td>1997</td>
<td>85</td>
<td>10.13</td>
<td>1.43 (1.01-2.03)</td>
</tr>
<tr>
<td>1998</td>
<td>93</td>
<td>11.04</td>
<td>1.55 (1.10-2.21)</td>
</tr>
<tr>
<td>1999</td>
<td>114</td>
<td>13.48</td>
<td>1.90 (1.37-2.66)</td>
</tr>
<tr>
<td>2000</td>
<td>119</td>
<td>14.02</td>
<td>1.97 (1.42-2.76)</td>
</tr>
<tr>
<td>2001</td>
<td>173</td>
<td>20.31</td>
<td>2.86 (2.10-3.94)</td>
</tr>
<tr>
<td>2002</td>
<td>187</td>
<td>21.84</td>
<td>3.07 (2.27-4.22)</td>
</tr>
<tr>
<td>2003</td>
<td>221</td>
<td>25.69</td>
<td>3.62 (2.69-4.94)</td>
</tr>
<tr>
<td>2004</td>
<td>207</td>
<td>23.94</td>
<td>3.37 (2.50-4.61)</td>
</tr>
<tr>
<td>2005</td>
<td>223</td>
<td>25.67</td>
<td>3.61 (2.68-4.93)</td>
</tr>
<tr>
<td>2006</td>
<td>202</td>
<td>23.14</td>
<td>3.26 (2.41-4.46)</td>
</tr>
<tr>
<td>2007</td>
<td>209</td>
<td>23.95</td>
<td>3.37 (2.50-4.61)</td>
</tr>
<tr>
<td>2008</td>
<td>275</td>
<td>31.53</td>
<td>4.44 (3.32-6.03)</td>
</tr>
<tr>
<td>2009</td>
<td>290</td>
<td>33.26</td>
<td>4.68 (3.51-6.35)</td>
</tr>
<tr>
<td>2010</td>
<td>375</td>
<td>43.03</td>
<td>6.06 (4.56-8.17)</td>
</tr>
<tr>
<td>2011</td>
<td>330</td>
<td>37.88</td>
<td>5.33 (4.01-7.21)</td>
</tr>
</tbody>
</table>

† CI = confidence interval
Table 12. Comparison of annual rates of hospital presentations from 1988 to 2011 in children 0 to 14 years old due to skin related reactions to food and anaphylactic and allergic reactions (both unspecified and to specific foods) combined by gender, ethnicity, city centre, and city/urban/rural.

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases</td>
<td>Rate (100,000/yr)</td>
<td></td>
<td>Cases</td>
<td>Rate (100,000/yr)</td>
<td>(95% CI)</td>
<td>Cases</td>
<td>Rate (100,000/yr)</td>
<td>(95% CI)</td>
</tr>
<tr>
<td>Total Presentations</td>
<td>209</td>
<td>6.63</td>
<td>1.00</td>
<td>349</td>
<td>10.44</td>
<td>1.57 (1.32-1.88)</td>
<td>366</td>
<td>10.95</td>
<td>1.65 (1.39-1.97)</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>122</td>
<td>7.57</td>
<td>1.00</td>
<td>209</td>
<td>12.17</td>
<td>1.61 (1.28-2.02)</td>
<td>227</td>
<td>13.21</td>
<td>1.74 (1.39-2.19)</td>
</tr>
<tr>
<td>Female</td>
<td>87</td>
<td>5.64</td>
<td>1.00</td>
<td>140</td>
<td>8.61</td>
<td>1.53 (1.16-2.02)</td>
<td>139</td>
<td>8.55</td>
<td>1.52 (1.15-2.00)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>158</td>
<td>7.36</td>
<td>1.00</td>
<td>239</td>
<td>11.84</td>
<td>1.61 (1.31-1.98)</td>
<td>219</td>
<td>10.85</td>
<td>1.47 (1.20-1.82)</td>
</tr>
<tr>
<td>Maori</td>
<td>34</td>
<td>5.21</td>
<td>1.00</td>
<td>64</td>
<td>8.15</td>
<td>1.56 (1.02-2.44)</td>
<td>75</td>
<td>9.55</td>
<td>1.83 (1.21-2.83)</td>
</tr>
<tr>
<td>Pacific</td>
<td>14</td>
<td>6.41</td>
<td>1.00</td>
<td>36</td>
<td>14.77</td>
<td>2.30 (1.21-4.63)</td>
<td>47</td>
<td>19.28</td>
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Table 12 Continued. Comparison of annual rates of hospital presentations from 1988 to 2011 in children 0 to 14 years old due to skin related reactions to food and anaphylactic and allergic reactions (both unspecified and to specific foods) combined by gender, ethnicity, city centre, and city/urban/rural.

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<th>2000-2003 Cases</th>
<th>Rate (100,000/yr)</th>
<th>RR (95% CI)</th>
<th>2004-2007 Cases</th>
<th>Rate (100,000/yr)</th>
<th>RR (95% CI)</th>
<th>2008-2011 Cases</th>
<th>Rate (100,000/yr)</th>
<th>RR (95% CI)</th>
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<tr>
<td>European/Other</td>
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<td>2.76 (2.29-3.33)</td>
<td>450</td>
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<td>623</td>
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<td>4.67 (3.91-5.59)</td>
</tr>
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<tr>
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<td>133</td>
<td>44.02</td>
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<td>8.89 (2.93-44.04)</td>
<td>166</td>
<td>46.58</td>
<td>15.16 (5.10-74.25)</td>
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<td>41</td>
<td>32.45</td>
<td>2.52 (1.35-5.01)</td>
<td>69</td>
<td>52.51</td>
<td>4.08 (2.27-7.85)</td>
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<tr>
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<td>35.02</td>
<td>3.61 (2.24-6.05)</td>
<td>79</td>
<td>29.84</td>
<td>3.07 (1.90-5.18)</td>
<td>65</td>
<td>26.58</td>
<td>2.74 (1.67-4.66)</td>
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<td>Dunedin</td>
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<td>9.49</td>
<td>1.42 (0.43-4.96)</td>
<td>16</td>
<td>19.72</td>
<td>2.95 (1.10-9.20)</td>
<td>20</td>
<td>25.30</td>
<td>3.78 (1.46-11.51)</td>
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<td>25.43</td>
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<td>675</td>
<td>38.26</td>
<td>6.10 (4.87-7.72)</td>
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<td>22.89</td>
<td>2.63 (2.00-3.49)</td>
<td>226</td>
<td>26.26</td>
<td>3.02 (2.31-3.99)</td>
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<td>36.76</td>
<td>4.23 (3.27-5.53)</td>
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<td>283</td>
<td>32.56</td>
<td>6.15 (4.53-8.51)</td>
</tr>
</tbody>
</table>
**Gender**
Over the time period of 1988-2011, the average annual rate amongst males was 7.57/100,000 and among females was 5.64/100,000. After adjustment for year of hospital presentation males presented to hospital and were assigned one of the identified acute allergic reaction codes 37% more often than girls (p=<0.001).

In comparison to the average annual rate in 1988-1991 time interval, the average annual rate of all presentations was higher in each subsequent time interval with the rate ratios increasing in subsequent time intervals. In comparison to the 1988-91 time interval, the average annual rate from 2008-2011 was 5.37 (95% CI 4.42-6.56) times greater for males and 5.69 (95% CI 4.53-7.22) times greater for females (Table 12).

**Ethnicity**
Children identified as European/Other presented to the hospital at an average annual rate of 7.36 per 100,000 children, as Maori presented with an average annual rate of 5.21/100,000, as Pacific presented with an average annual rate of 6.41/100,000 and as Asian presented with an average annual rate 3.07/100,000. In comparison with the rate in those of European/Other ethnicity the rate of presentation with an acute allergic reaction was 74% higher in children of Pacific ethnicity (p<0.001) and 27% higher in children of Asian ethnicity (p<0.001). The rate in children of Maori ethnicity did not differ from that in children of European/Other ethnicity (5% lower, p=0.27).

In comparison to the average annual rate in the 1988-1991 time interval, the average annual rate of all presentations increased in subsequent time intervals in each ethnic group. In comparison to
the 1988-1991 time interval, the average annual rate from 2008-2011 was 4.67 (95% CI 3.91-5.59) times greater for children of European/Other ethnicity, 6.80 (95% CI 4.76-10.02) times greater for children of Maori ethnicity, 9.52 (95% CI 5.53-17.75) times greater for children of Pacific ethnicity and, 15.16 (95% CI 5.10-74.25) times greater for children of Asian ethnicity (Table 12).

**Geographic Environments**

Five major city centres are located within New Zealand: Auckland, Hamilton, Wellington, Christchurch, and Dunedin (Figure 4). Researchers hypothesize that geography influences the development of a food allergy (Cochrane et al., 2009; Gray & Kung, 2012; Mullins, Clark, & Camargo, 2010; Prescott & Allen, 2011). In addition, increased access to care could also play a role in an increase in the rate of hospital presentations for any condition, including food-related acute allergic reactions. To gain a better understanding of the city centre effect in New Zealand, data was categorised by city centre based on Territorial Local Authority (TLA) boundaries.
Children living in Auckland presented to the hospital at an average annual rate of 4.59 per 100,000, children living in Hamilton presented with an annual average rate of 5.50/100,000, children living in Wellington presented with an annual average rate of 12.88/100,000, children living in Christchurch presented with an annual average rate of 9.71/100,000, and children living in Dunedin presented with an average annual rate of 6.69/100,000. In comparison with the rate of those children living in Auckland the rate of presentation due to an acute allergic reaction was 55% higher in children living in Wellington (p<0.001), 35% higher for children living in Christchurch (p<0.001), and 32% lower for children living in Dunedin (p=0.003). The rate in children living in Hamilton did not differ from that in children living in Auckland (12% lower, p=0.21) (Table 12).
In comparison to the average annual rate in the 1988-1991 time interval, the average annual rate in 2011 was 8.73 (95% CI 6.32-12.38) times greater for children living in Auckland, 6.73 (95% CI 2.68-21.71) times greater for children living in Hamilton, 4.08 (95% CI 2.27-7.85) times greater for children living in Wellington, 2.74 (95% CI 1.67-4.66) times greater for children living in Christchurch, and 3.78 (95% CI 1.46-11.51) times greater for children living in Dunedin (Table 12) (Figure 5).

**Figure 5.** Annual hospital presentations, by city centre, from 1988 to 2011 in children 0 to 14 years old due to skin related reactions to food, anaphylactic, and allergic reactions (both unspecified and to specific foods).

Children living in rural areas presented to the hospital at an average annual rate of 5.29 per 100,000, children living in urban areas presented with an annual average rate of 8.70/100,000, and children living in cities presented with an average annual rate of 6.27/100,000. In comparison with the rate of those children living in rural areas the rate of presentation with an acute allergic reaction was 33% higher in children living in urban areas (p<0.001) and 33% higher in children living in cities (p<0.001).
In comparison to the average annual rate in the 1988-1991 time interval, the average annual rate of all presentations increased in subsequent years in rural, urban and city areas. In comparison to the 1988 time interval, the average annual rate in 2011 was 6.15 (95% CI 4.53-8.51) times greater for children living in rural areas, 4.23 (95% CI 3.27-5.53) times greater for children living in urban areas, and 6.10 (95% CI 4.87-7.72) times greater for children living in cities (Table 12).

**Age**

Children 0 to 14 years presented to the hospital at an average annual rate of 12.90 per 100,000, children 5 to 9 years presented with an annual average rate of 3.96/100,000, and as children 10 to 14 years presented with an average annual rate of 3.90/100,000. In comparison with the rate of those aged 10 to 14 years the rate of presentation with an acute allergic reaction was three times higher in children 0 to 4 years (p<0.001). The rate in children 5 to 9 years did not differ from that of children 10 to 14 years (10% higher, p=0.07) (Figure 6).

In comparison to the average annual rate in the 1988 time interval, the average annual rate of all presentations increased in subsequent years in each age group. In comparison to the 1988 time interval, the average annual rate in 2011 was 4.93 (95% CI 3.43-7.26) times greater for children 0 to 4 years old, 7.19 (95% CI 3.72-15.55) times greater for children 5 to 9 years old, and 5.59 (95% CI 2.85-12.21) times greater for children 10 to 14 years old (Table 13).
Table 13. Comparison of annual rates of hospital presentations from 1988 to 2001 in children 0 to 14 years old due to skin related reactions to food and anaphylactic and allergic reactions (both unspecified and to specific foods) combined by age groups.

<table>
<thead>
<tr>
<th>Year</th>
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<th>10 to 14 years</th>
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<tbody>
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<td>Cases</td>
<td>Rate per 100,000</td>
<td>Cases</td>
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</table>

Figure 6. Annual hospital presentations, by age group, from 1988 to 2011 in children 0 to 14 years old due to skin related reactions to food, anaphylactic, and allergic reactions (both unspecified and to specific foods).
Discussion

Principal findings
An increase in hospital presentations due to acute allergic reactions identified from pre-selected ICD-9-CMA-II codes over the past two plus decades was observed. An average yearly increase of 8% in hospital presentations due to acute allergic reactions (p<0.001) was evident. In comparison to the 1988 time interval (annual rate 7.11/100,000), the rate of all presentations was higher in 2011 (37.88/100,000, RR=5.33). The largest increase can be seen in the last decade; 2000-2003 (20.54/100,000, RR=3.10), 2004-2007 (24.09/100,000, RR=3.63) and 2008-2011 (36.47/100,000, RR=5.50). This increase is evident in both males and females, all ethnic groups, throughout the country, and in all age groups.

In both males and females the average annual rate from 2008-2011 was over 5 times greater than 1988-1991. Ethnic differences are apparent, specifically the very large increases in hospital presentations by children of Asian ethnicity. The average annual rate of hospital presentations amongst children of Asian ethnicity was 15 times greater in 2008-2011 as compared to 1988-1991. Based on geography, children living in Wellington (12.88/100,000) and Christchurch (9.71/100,000) presented with higher annual average rates than children living in Auckland. However the overall increase seen over the two plus decades was greatest in children living in Auckland (RR=8.73). In addition, children living in both urban (8.70/100,000) and city areas (6.27/100,000) presented to the hospital at a higher rate than those living in rural areas (5.29/100,000). However the rate of change over time was greatest in children living in rural areas (RR=6.15) and children living in cities (RR=6.10). As expected, younger children (0 to 4 years) presented to the hospital more often (12.90/100,000) than children 5 to 9 years (3.96/100,000) and children 10 to 14 years
(3.90/100,000). However the rate of change over time was greater in children 5 to 9 years (RR=7.19) and children 10 to 14 (RR=5.59) than in those 0 to 4 years old (RR=4.93).

Based on this analysis it is evident that there has been in an increase in hospital presentations due to acute allergic reactions and therefore most likely an increase in the overall rates of food allergy in children 0 to 14 years old living in all areas of New Zealand.

A dramatic increase, specifically over the last decade, in hospital presentations is evident in the Asian population which can partly be explained by the changing ethnic make-up of New Zealand and more specifically Auckland, with Asians being a more recent migratory group. This Asian component also potentially plays a role in the greater increase of hospital presentations seen in children living in Auckland, the NZ city where the majority of Asian immigrants reside.

The increase in hospital presentations amongst Asian children may also be due to gene-environment interactions. Evidence exists that supports the role of dietary factors, long considered one of the most important causal exposures, playing a role in the development of food allergy. Although the overall prevalence of food allergy in Asia is similar to the West, the types of food allergy differ in order of relevance (Lee, Thalayasingam, & Lee, 2013). For example, shellfish is the most common allergen in Asia whereas peanut prevalence is extremely low compared to the West for reasons unknown (Lee et al., 2013). Based on a questionnaire administered to local and expatriate Singapore and Philippine schoolchildren (23,435), respondents of either Asian or white subjects born in Western countries were at higher risk of peanut and tree nut allergy.
compared with those born in Asia (Shek et al., 2010). In the population-based HealthNuts study, compared to infants with two Australian-born parents, peanut allergy was more common among infants with at least one parent born in East Asia but not those with at least one parent born in the UK/Europe (Koplin et al., 2014). However rates of allergic disease were lower among Asian parents and therefore the high peanut allergy prevalence among infants of Asian-born parents appears to have occurred in a single generation.

While higher rates of hospital presentations were evident in both Christchurch and Wellington, no clear latitudinal effect was evident. Vitamin D status has been hypothesised as the reason for why latitudinal differences in food allergy prevalence have been observed in some countries (Camargo, Clark, Kaplan, Lieberman, & Wood, 2007; Mullins & Camargo, 2011; Mullins, Clark, & Camargo, 2010; Mullins & Camargo, 2012; Osborne, Ukoumunne, Wake, & Allen, 2012). In New Zealand, vitamin D status at birth varies more so by season of birth and ethnicity than by latitude (Camargo et al., 2010). Consistent with the literature, higher average annual rates of hospital presentations amongst children living in both urban and city areas of New Zealand were apparent. The hygiene hypothesis, exposure to nature, and access to care could all play a role in the higher rates.

Food allergy most frequently presents and is diagnosed early in childhood. Therefore the higher rates of hospitalisation in children 0 to 4 years old is to be expected. The greater increase over time in the annual rate of hospital presentation amongst children 5 to 9 and 10 to 14 years could potentially be due to the fact that fewer children are growing out of their food allergy and thus reactions may continue to occur at an older age.
**Limitations**

Using ICD codes for tracking patients has its limitations since symptoms and signs may not be correctly coded which may lead to under-estimation of incidence (Clark et al., 2006; Vetander et al., 2012). In addition, hospital admissions only represent a small proportion of incident cases of these conditions (Poulos et al., 2007). Because of these data issues we limited the objectives of this study to an investigation of trends over time pertaining to hospital presentations not incidence or prevalence of food allergies in New Zealand children. Although there may not be a direct connection between hospital presentations by children and prevalence or incidence, our case definition does appear to be stable over time (McMilin et al., 2015).

The high proportion of unspecified codes used in New Zealand differs from what is seen in other countries. As a large proportion of presentations were coded as unspecified the data presented here is an overestimation of hospital presentation due to a food-related allergic reactions. Based on the ADHB chart review results presented in part one we estimated that from 24% to 36% of the 51% of these hospital presentations which had an unspecified allergic or anaphylactic reaction code are due to food. Although this will impact the overall rate of presentations it is unlikely to have biased the temporal trends reported from this study as the chart review reported an increase over time in food-related acute allergic reactions only.

**Research implications**

The findings from this study provide a foundation for future research and evidence for the need of a more comprehensive study of food allergy prevalence in NZ children. Findings from this study should also be considered nationally at a policy level to support
medical professionals in the management of this increasingly prevalent problem.

**Opportunities for further research**
The large proportion of unspecified codes used is unique to the New Zealand context. The current study did not allow for food-related acute allergic reactions to be confirmed from the unspecified ICD-9 codes. Therefore future studies should extrapolate hospital presentations from the unspecified codes based on the chart review study (part one) to determine more accurate annual rates of hospitalisations due to a food-related acute allergic reaction.

**Chapter conclusions**
The two studies described in this chapter provide the first description of national trends on food allergy prevalence in New Zealand and demonstrate dramatic increases in hospital presentations for food related allergic reactions. Hospital presentations serve as a proxy measure for prevalence of food allergy and these data provide no explanation for why such large recent temporal changes have occurred.

The next chapter reviews the literature describing potential causal pathways leading to the development of childhood food allergy.
Chapter 4: LITERATURE REVIEW PART TWO - CAUSATION OF FOOD ALLERGY

Chapter objectives
In this chapter I review the literature pertaining to potential causal pathways of the development of a food allergy. Specifically focusing on factors that have been associated with the development of a food allergy, those specific to the host and those that are a result of the environment within which the child develops and the components of that environment to which the child is exposed. Both prenatal and postnatal determinants are considered to be important. Interaction between host and environmental factors is potentially as important as the specific host and environmental factors themselves.

Overview
Can food allergy be prevented? We are just beginning to recognise potential genetic, environmental, and immunologic influences on the development of an allergy to peanuts, a food acknowledged as one of the leading causes of life threatening and lifelong reactions. It is now appreciated that the immunological processes that lead to food allergy begin very early in life and, in at least some cases, prior to birth. Gene-environment interactions appear central to the development of food allergy.

Within the first three years of life, children are at greatest risk of developing atopic diseases, with food allergy and atopic dermatitis as the typical first manifestations. Of the atopic diseases, food allergy early in life seems to be a distinct predictor for developing other atopic diseases later in childhood. It remains unknown whether the apparent increase in food allergy denotes a discrete
and independent predictor of a subsequent increase in other allergic manifestations later in life.

In some cases, within the first 3-5 years of life, children lose their sensitivity to most allergenic foods for example, egg, milk, wheat and soya. Although the mechanism for this tolerance is not understood, it may involve maturation of the gut, resulting in reduced systemic absorption and maturation of immune responses. In contrast, the resolution of allergy to peanut and tree nut occurs less frequently. In more than 80% of children with peanut or tree nut allergy, the allergy persists into adult life. This natural history, and the relatively high frequency of these nut allergies, makes them a particularly important focus for epidemiologic food allergy related research.

Currently it is not known how to prevent food allergy. Factors related to the development of a food allergy in childhood can be considered as those that are intrinsically components of the host; those that are components of the environment within which the person and their family lives; and those that are due to interactions between the host and the environment (Figure 7) (Tan, Ellis, Saffery, & Allen, 2012).

Host factors include those described by familial patterns of atopy, genes that are specifically associated with the development of components of allergic disease and with, development of the immune system, and the contributions made by gut bacteria and interactions between these bacteria and the host. Within the environment early life exposure to infections, exposure to pollutants, adequate vitamin D status, the use of antibiotics, paracetamol, and antacids all have been proposed to play a potential role in the development of food allergy. Evidence exists
that supports the role of dietary factors, long considered one of the most important causal exposures, playing a role in the development of food allergy including: omega-3 fatty acids, antioxidants, and folate; the use of prebiotics and probiotics; child feeding patterns (breastfeeding, complimentary, and allergen avoidance); and maternal allergen avoidance.

**Figure 7.** Potential genetic, epigenetic, and environmental factors contributing to an increase in IgE – mediated food allergy, in prenatal, perinatal, and postnatal phases.

**Child characteristics**

**Immunological mechanisms underlying food allergy**
IgE-mediated food allergy is an immunological phenomenon in which an atopic response occurs to a food antigen. The T-helper lymphocyte is believed to play a central role in the development of food allergy as it does in all of the clinical manifestations of atopy. This T-helper cell differentiation into a Th2 phenotype is believed to
be a key differentiating point in the development of either tolerance or allergic sensitization (Vickery, Chin, & Burks, 2011).

**Genetic and epigenetic mechanisms**

Both genetic factors and epigenetic modification of gene expression may play a role in the development of food allergy. Epigenetic factors are believed to act during prenatal and perinatal periods via modification of the development of the immune system and gut bacteria.

**Genetics and food allergies**

*Family history of atopy*

The atopic march describes the natural history of atopic manifestations characterised by a typical sequence of progression of clinical signs of atopic disease, with some signs of atopic disease becoming more prominent while others subside. Although population dependent, children born into atopic families have a high risk of developing allergy, 60-80 per cent (Kjellman, 1998; Prescott, 2011; Zeiger et al., 1992). However the risk is not 100 per cent even if both parents have allergies, therefore genetics cannot explain all allergy (Kjellman, 1998; Prescott, 2011; Zeiger et al., 1992). In addition the risk of developing an allergy when neither parent has any history of allergies, is still more than 25 per cent and this risk appears to be increasing (Kjellman, 1998; Prescott, 2011; Zeiger et al., 1992). The direct effects of the mother in addition to genetic inheritance is evident as allergy in the mother, compared to allergy in the father, is a much stronger risk factor (Litonjua, Carey, Burge, Weiss, & Gold, 1998). The role of both genetics and the environment is evident in twins, if one identical twin has a food allergy there is a very high chance (over 60 percent), but not a guarantee, that the other twin will also have a food allergy (Prescott, 2011). If they are non-identical twins, the chance of both developing a food allergy is less than 10 per cent.
Allergy genes
Underlying genetic factors are likely to play a role in the development of food allergy. A number of gene polymorphism(s) have been implicated in the development of atopy including those in the Major Histocompatibility Complex (MHC) human leukocyte antigen (HLA) class II gene family including HLA-DRB1, HLA-DQB1 and HLA-DB1, cluster of differentiation 14 (CD14), forkhead box P3 (FOXP3), signal transducer and activator of transcription 6 (STAT6), serine protease inhibitor Karzal type 5 (SPINK5), interleukin-10 (IL-10) and interleukin-13 (IL-13) (Tan et al., 2012). Studies that have investigated each of these gene polymorphisms separately have shown each of them to be associated with the incidence or the severity of food allergy.

Epigenetics and food allergies
In addition to these genetic associations, studies also point to epigenetic mechanisms, defined as changes in gene expression that are the result of causes other than gene mutations. Immune development is under epigenetic regulation and therefore investigation of relationships between epigenetic alterations and allergic phenotypes are an important area of contemporary research into the cause of food allergy. Current understanding of environmental epigenetics supports the hypothesis that epigenetic alterations are one of the mechanisms mediating the effect of pre- and postnatal environmental exposure on the development of food allergy.

Microbiota
More than 100 trillion bacteria make up the human gastrointestinal tract, and together with archae, fungi, and viruses, form the gut microbiota (Tilg & Moschen, 2015). Diet can affect inflammatory processes, the immune system, and is probably the most relevant factor associated with the composition of the microbiota. The typical Western diet comprised of refined grains, alcohol, salt,
certain oils, corn-derived fructose, fatty domesticated meats, and other foods frequently consumed in caloric excess promote inflammation and atherosclerosis through specific fatty acids and degradation products such as trimethylamine N-oxide (Tilg & Moschen, 2015). Whereas diets rich in vegetables and fruits can have anti-inflammatory effects, however the Western diet can be further characterized by a reduced consumption of these foods.

Dysbiosis, changes in the composition of the microbiota, has been hypothesized as leading to a loss of protective bacterial signals which can cause both allergic and inflammatory diseases (Feehley & Nagler, 2014). Beginning at birth and continuing throughout life, these signals provide an education for the immune system. However it is not well understood which signals are required from the bacteria to prevent allergic diseases (Feehley & Nagler, 2014). A variety of signals, most likely from the microbiota, must be integrated within the intestine creating a balance between activation and tolerance, disruption of this balance can induce sensitisation. Exposure to breast milk for example, may be important for promoting ‘immune tolerance’ through the gut as it contains substances that promote favourable colonisation of friendly bacteria (Prescott, 2011).

There is evidence both in mouse models and human studies of the influence of the microbiota on the development of allergic disease but how this occurs is not fully understood (Feehley & Nagler, 2014). Therefore a need for a better understanding of the relationship among food, immunity, and the microbiota is critical as it is currently unclear whether changes in the microbiome cause allergic disease or occur as a consequence of the allergic disease (Tilg & Moschen, 2015).
Reduced early life exposure to infectious pathogens and the development of allergic disease: the hygiene hypothesis

The hygiene hypothesis, initially proposed in 1989 in relation to hay fever and, to a lesser extent, eczema, proposed that these atopic diseases were prevented by increased exposure to infections in early childhood (Strachan, 1989). Interestingly, considering the prominence that this hypothesis has had in the field of atopy search, the only measure of infectious disease exposure included in the original analysis was number of other children in the household and the outcome measure “hay fever or allergic rhinitis in the past 12 months” was based upon parental and self-report at ages 12 and 23 years respectively.

It has been proposed that the gastrointestinal tract, and thus gut flora, may be important to the interrelationship between infection and allergy. In a Canadian study examining the influence of education level, immigrant status, and geographic location, food allergies (to shellfish) were found to be most prevalent in the more educated, those born in Canada and living in urban settings. Immigrants were hypothesised to have a reduced risk of food allergy due to both genetic factors and environmental influences. In this instance, education level may act as a proxy for excessive hygiene but could also indicate more awareness of food allergy as a cause of symptoms and/or better access to care and hence a greater likelihood of diagnosis. Similar results were found in a study conducted in Australia which showed that socio-economic advantage and urban dwelling may be risk factors for developing childhood food allergy and anaphylaxis but again cannot reliably inform on the mechanism of this relationship.
Feeding patterns

Breastfeeding
Breast milk contains many immune factors including antibodies and immune cells, cytokines, and other nutrients all of which are important for both protection from infection and promotion of immune tolerance (Prescott, 2011). As previously mentioned breast milk favours colonisation of the gut with friendly bacteria. Therefore clues may exist within this natural tolerance-inducing food to aid with the understanding of how to promote tolerance and prevent immune and allergic disease in the early postnatal period (Prescott, 2011).

Complimentary feeding
Advice regarding the introduction of complementary foods has changed over the years as a greater understanding of allergic disease has emerged. There is evidence of a reduction in early allergic disease outcomes in children who have not been exposed to complementary foods before four months of age, however the benefits do not extend beyond this time point (Filipiak et al., 2007). To the contrary, avoidance beyond six months has been associated with increased risk of allergic diseases such as food allergy, eczema, and asthma (Filipiak et al., 2007; Poole, 2006; Zutavern et al., 2004). As described by Prescott in her book ‘The Allergy Epidemic: A Mystery of Modern Life’ the majority of experts in industrialised countries recommended that complimentary foods are introduced ‘from around 4-6 months of age’ and that these should not be delayed beyond six months of age (Prescott, 2011). However it is suggested that parents consider introducing a new food every 2 to 3 days according to what the family usually eat, regardless of whether or not the food is perceived to be highly allergenic. By giving foods one at a time reactions can be more clearly identified and it is unlikely that a child will develop a new
allergy to a food that is already tolerated if eaten regularly (Prescott, 2011).

**Environmental exposure**
As the risk of developing an allergy is not 100 per cent even if both parents have allergies, environmental factors therefore must play a role (Prescott, 2011). For the purposes of this thesis these factors, or exposures, occur at multiple levels: proximal social environments, distal social environments, and macro environments. These levels of influence are defined within the conceptual approach that underpins *Growing Up in New Zealand* and only factors applicable to the research described as part of this thesis in the New Zealand context are included. Proximal social environments include the child’s home physical and social environment; parental health, parental and family socioeconomic indicators; and ethnic identity. Distal social environments include the neighbourhood (physical location, local engagement, proximity to services, informal support available). Macro environmental factors include maternity care and continuity of access to primary health care services, and costs.

**Proximal social environments**
Proximal social environments include the child’s home physical and social environment; parental health, parental and family socioeconomic indicators; and ethnic identity.

**Maternal smoking**
Avoidance of cigarette smoke is recommended for disease prevention, both during pregnancy and after birth. This is due to the effect of smoking on the immune development of the foetus (Noakes et al., 2006; Noakes, Holt, & Prescott, 2003). In addition a change in gene expression through epigenetic effects has been attributed to cigarette smoke exposure (Adcock, Tsaprouni, Bhavsar, & Ito, 2007).
**Maternal diet**

As previously described the typical Western diet is comprised of refined grains, alcohol, salt, certain oils, corn-derived fructose, fatty domesticated meats, and other foods frequently consumed in caloric excess which not only can promote inflammation but has also led to a change in the consumption of omega-3 and omega-6 polyunsaturated fatty acids (PUFA). The increasing intake of omega-6 PUFA has led to an increased exposure to a more inflammatory fatty acid profile by the foetus and young infant, which could be contributing to the growing risk of inflammatory diseases such as allergy (West, Videky, & Prescott, 2010). However the effects of omega-3 PUFA include a suppression of inflammatory responses of many immune cells including T cells, therefore an omega-3 rich diet may be beneficial (Calder, 1998; Prescott, 2011). However, this is one piece of the complexity of the development of food allergy and therefore the protective effects may not be strong enough to overcome other environmental pressures promoting allergic disease.

An additional outcome of the Western diet is the declining intake of soluble fibre which in addition to fermentable dietary carbohydrates, have been recognised as important in the maintenance of gut health. The decline in consumption of soluble fibre is a concern and it has been proposed to contribute to a reduction in immune tolerance leading to pro-inflammatory and an increased risk of allergic diseases (Arslanoglu et al., 2008; Prescott, 2011).

Prebiotics can selectively stimulate the growth and activity of bacteria in the colon and there is evidence to support direct anti-inflammatory effects of these on the immune system (Prescott, 2011; Schouten et al., 2009; Van Hoffen et al., 2009). A Cochrane database review concluded that the use of prebiotics as an allergy
prevention strategy provided some evidence for eczema prevention however there is a need for further research before routine use (Osborn & Sinn, 2007).

As gene expression is epigenetically controlled by, for example, changes in gene methylation, research has focused on dietary factors that can alter gene expression (Prescott, 2011). For example, folate provides the ’methyl’ groups for various metabolic functions, including DNA methylation for gene silencing (Miller & Ho, 2008). The evidence to date for the contribution of alterations in gene expression to atopic disease has only been reported for allergic airway disease and asthma (Håberg, London, Stigum, Nafstad, & Nystad, 2009; Prescott, 2011; Whitrow, Moore, Rumbold, & Davies, 2009).

**Maternal allergen avoidance**
It is important to note here the rapid changes and reversals that have occurred in recent decades in strategies for the prevention of food allergy, for example dietary avoidance during pregnancy and early childhood. There is minimal reproducible evidence that manipulation of the maternal diet during pregnancy and/or breastfeeding has any protective effect on the development of food allergy, nor that early introduction of foods increases the risk of a child having food allergy. To the contrary, avoidance strategies have been shown to potentially compromise the nutritional well-being of both mother and child (Du Toit & Lack, 2011). Child health policy development agencies in many countries, for example the American Academy of Paediatrics and the UK Government Department of Health, have now withdrawn recommendations of avoidance even for at-risk families (Du Toit & Lack, 2011).

**Vitamin D**
Vitamin D deficiency has been identified as a prevalent population health issue in many countries in recent decades. This apparent re-
The emergence of vitamin D deficiency as a public health problem has occurred over the same time interval that food allergy prevalence has increased. Pregnant women are a population group at higher risk of vitamin D deficiency. Globally it is estimated that 54% of pregnant women and 75% of newborns have vitamin D deficiency as defined by a serum 25-hydroxyvitamin D (25(OH)D) concentration <50 nmol/L (Saraf, Morton, Camargo, & Grant, 2015).

Sources of vitamin D include dietary (both natural and fortified foods), supplements and ultraviolet B exposure. In humans ultraviolet B exposure is the principal source of vitamin D, being the source of more than 90% of vitamin D in most populations (Paxton et al., 2013). Exposure of the body, in a bathing suit, to one minimal erythema dose (the amount of UVB which would produce redness 24 hours after exposure) is equivalent to taking between 10,000 and 25,000 IU of vitamin D orally. Serum 25(OH) D concentrations, the biomarker used to define vitamin D status, decrease with increasing latitude, and also vary with ethnicity, dietary patterns, local climate and elevation.

Since the discovery of vitamin D receptors on the majority of tissues and cells within the body, a new understanding has developed regarding the function of this vitamin. Low levels of 25-hydroxy vitamin D (25(OH) D) at birth are associated with a range of adverse health outcomes. The 2011 IOM report suggests a serum 25(OH) D level of >50nmol/L (20 ng/ml) is sufficient to optimize bone mineral density in vast majority of population. However, the IOM focused on 25(OH) D as a marker of skeletal health and it is unclear whether their recommendation is valid for all of the putative non-skeletal actions of vitamin D. One of the more
important extra skeletal roles of vitamin D is as a modulator of immune function.

Vitamin D is believed to have anti-inflammatory and immune-modulatory effects in the body, which is the potential mechanism for its role in the development of allergy. Expression of vitamin D receptors (VDRs) occurs on many cells within both the innate and adaptive components of the immune system: T and B lymphocytes, neutrophils, macrophages, and dendritic cells. The active form of vitamin D, 1,25-(OH)₂D, binds to the vitamin D receptor (VDR) which is expressed by many cells of the immune system, including activated B and T cells, monocytes, and dendritic cells (DCs) (Chambers & Hawrylowicz, 2011). These T cells can suppress as well as enhance immune responses (Chambers & Hawrylowicz, 2011). This creates the potential for vitamin D to modulate both the innate and adaptive components of the immune system. Thus the immune-modulatory properties of vitamin D, including its capacity to promote regulatory T-cells (Treg populations), has become an area of recent interest in our understanding of environmental influences on immune-mediated diseases (Chambers & Hawrylowicz, 2011).

In terms of the effects of vitamin D on effector T-cell responses and antigen-presenting cell function, it is likely that 1,25-(OH)₂D, influence DC phenotype and activation and thus antigen presentation and therefore the nature of the T-cell response to the antigen. In addition, 1,25-(OH)₂D may promote a Treg phenotype by acting directly on T-cells, for example through the promotion of peripheral tolerance via inhibition of inflammatory responses and induction of Tregs (Chambers & Hawrylowicz, 2011). Vitamin D therefore has the capability to promote – both directly and indirectly – regulatory or suppressor T-cell populations leading to the
inhibition of inappropriate immune responses that can cause disease (Chambers & Hawrylowicz, 2011).

As the development of atopy is believed to begin early in life, including perhaps in-utero, vitamin D status during pregnancy is a potential determinant of the development of food allergy and of the increase in food allergy prevalence that has occurred in recent decades. The central role of vitamin D in modulating the immune response to antigen presentation provides biological plausibility for such a role. However, preliminary evidence indicates that the relationship between vitamin D status and the development of food allergy is unlikely to be a simple linear population wide relationship. For example, both high and low cord blood concentrations of 25(OH) D are associated with an increased risk of a child being atopic (Rothers, Wright, Stern, Halonen, & Camargo, 2011).

**Maternal use of antibiotics, paracetamol, antacids**

Albeit limited, there is evidence supporting a relationship between allergic disease and the use of: antibiotics during the first year of life; paracetamol in pregnancy; and acid-suppressive medications in pregnancy (McKeever et al., 2002; Persky et al., 2008; Shaheen et al., 2005). This area represents potentially avoidable risk factors that require further investigation (Prescott, 2011).

**Distal environmental exposures**

Distal social environments include the neighbourhood (physical location, local engagement, proximity to services, informal support available).

**Rurality**

Where a child lives may play a role in the development of a food allergy based on the hygiene hypothesis. Exposure to pets, rural environments, and/or agricultural settings may reduce the risk for allergic diseases. In comparison to rural settings, living in a major
city may be a risk factor for developing childhood food allergy and anaphylaxis as described by an Australian study (Mullins et al., 2010).

**Macro environmental exposures**
Macro environmental factors include maternity care and continuity of access to primary health care services, and costs.

**Maternity care**
Building on the hygiene hypothesis, the infant is first exposed to maternal gut bacteria as it passes through the birth canal. Therefore the rise in asthma and allergic disease in relation to rates of non-vaginal birth (caesarean section) has become a growing area of interest (Bager, Wohlfahrt, & Westergaard, 2008; Metsälä et al., 2008; Prescott, 2011; Thavagnanam, Fleming, Bromley, Shields, & Cardwell, 2008; Tollånes, Moster, Daltveit, & Irgens, 2008). In addition, a baby may be exposed to antibiotics during this early period if born via caesarean section. Although it is likely to be only one of several contributors to the allergy epidemic, there is a growing body of evidence to support an increased risk of allergic disease in children born via caesarean section (Bager, Wohlfahrt, & Westergaard, 2008).

**Chapter conclusions**
Food allergy is a complex condition with multiple potential causal pathways. Specifically factors associated with the development of a food allergy can be categorized as those specific to the host and those that are a result of the environment within which the child develops and the components of that environment to which the child is exposed. To further investigate the epidemiology of food allergy using peanut allergy at age 2 years as an example, factors associated with the development of peanut allergy will be identified in the next chapter, using data collected within New Zealand’s contemporary child cohort study *Growing Up in New Zealand*. 
Chapter 5: CHILDHOOD PEANUT ALLERGY IN NEW ZEALAND CONTEXT

Chapter objectives
As previously described, food allergy is a complex condition. I therefore decided to focus on one food allergen for the purpose of measuring prevalence and determining the factors associated with the development of a food allergy in early childhood. I chose peanut as the allergen of interest for several reasons. Firstly, it is evident in the literature that of the self- or parent reported data on food allergy, that which has been reported on peanut allergy shows much closer approximation to more robust prevalence measures than is evident for other food allergens, for example egg and dairy. Second, peanuts are the food allergen most frequently associated with life threatening or fatal allergic reactions. Third, relative to other food allergies, peanut allergy is more likely to persist throughout life. Therefore in this chapter I describe the prevalence of peanut allergy, based on self-report, at the age of 2 years within the birth cohort study Growing Up in New Zealand, and using this self-reported measure of prevalence. I describe factors associated with the development of this allergy in early childhood. Having emphasised the significance of bias that is introduced by the self-reported estimates of food allergy prevalence I also present a description of my validation of this self-reported data.

Abstract

Introduction
Growing Up in New Zealand is a longitudinal study providing a contemporary and population relevant picture of what it is like to be a child growing up in New Zealand in the 21st century. Data has been collected from both the child’s mothers and their partners at
various time periods before and during their child’s life to better understand the complex interplay of all the factors that lead to child outcomes including growth, health, behaviour and cognitive development.

Methods
Utilising data collected from *Growing Up in New Zealand*, up to age two, potential factors associated with the development of a peanut allergy in early childhood were investigated. The presence of an association between variables describing exposures with the presence of peanut allergy was determined using the chi-square test and then univariable and multivariable logistic regression analyses. Independent associations were described using adjusted odds ratios (OR) and 95% confidence intervals (CI).

Associations with the presence of peanut allergy were categorized based on the measurement of child, family and wider influences on disease in early childhood as defined by *Growing Up in New Zealand* conceptual model that incorporates a life course approach and the interaction over time of the individual’s biology with the environment in which they develop.

Results
Based on parental self-report of doctor diagnosis, 162 (2.6%, 95% CI 2.2-3.0%) of the cohort children were identified as peanut allergic. Thirty-two variables were analysed with nine being included in the final multivariate model.

The odds of having parental reported peanut allergy at age two years were increased for boys (OR=1.59, 95% CI 1.13-2.26), children diagnosed with eczema since 9 months (OR=10.72, 95% CI 7.26-16.31), children whose mother had been history of atopic disease (eczema, hay fever, or food allergy) (OR=1.40, 95% CI
1.00-1.97), and mothers who identified as Asian (OR=2.27, 95% CI 1.48-3.43). The odds of having parental reported peanut allergy at age two years were decreased for children who had never tried nuts or peanuts (OR=0.56, 95% CI 0.33-0.89), or whose mothers had no secondary qualifications (OR=0.35, 95% CI 0.11-0.89) or secondary school/NCEA 1-4 (OR=0.27, 95% CI 0.14-0.51).

Discussion
The rate of parental self-report of children were identified as peanut allergic within the Growing Up in New Zealand longitudinal birth cohort is comparable to rates observed worldwide. The independent associations with the risk of peanut allergy that were evident for child gender and presence of eczema, maternal ethnicity and atopic history imply independent biological contributions to the aetiology of peanut allergy. The independent associations with the risk of peanut allergy that were evident for child eczema, maternal ethnicity, lack of exposure to peanuts and maternal education imply independent environmental or host-environmental interaction factors in the aetiology of peanut allergy.
Introduction
In 2004 the NZ Ministry of Social Development released a request for proposals for a new longitudinal study of New Zealand Children and Families (Health Research Council of New Zealand, 2004). Growing Up in New Zealand was created in response to this request. The essential design features of Growing Up in New Zealand were that the study would begin during pregnancy, would enrol a sample that reflected the ethnic and socioeconomic diversity of NZ and that included both the children’s mothers and their partners (Morton et al., 2012).

The study was designed to allow for investigation of the determinants of health in a generalizable NZ population sample and therefore key ethnic and socio-demographic characteristics of the recruited main cohort families are similar to those of families having children in New Zealand today (Morton et al., 2014). The conceptual framework for Growing Up in New Zealand takes a life course approach to child development, thus recognizing the dynamic interactions between children and their environments across a broad range of influences from their immediate family environments to their wider societal contexts over time (Morton et al., 2013). The cohort size and diversity provides adequate explanatory power to examine complex developmental life course outcomes across the whole population and more specifically population subgroups, for example ethnic identity.

Strong links with policy makers while working closely with multiple government agencies and related stakeholders allows for the translation of findings into policy development and refinement. A key finding to date highlights the diversity of the new generation of New Zealand children and thus the importance of considering this diversity when developing a better understanding of the experience
of growing up in New Zealand during the 21st Century (Morton et al., 2013). For example, when the cohort children were aged two years, multiple ethnicities were identified for 32% of the cohort (Morton et al., 2014).

**Methods**

**Study setting**

During 2009 and 2010, pregnant women and their partners were recruited to participate in a longitudinal study to provide a contemporary and population relevant picture of what it is like to be a child growing up in New Zealand in the 21st century. Pregnant women were eligible for enrolment if they were resident in a geographical region defined by the three contiguous district health board regions of Auckland, Counties-Manukau and Waikato and had an estimate delivery date between 25th April 2009 and 25th March 2010. There was no other inclusion or exclusion criteria. The study region was chosen because its demographic profile indicated that a random sample recruited from this region would reflect the diversity of the contemporary national birth cohort with respect to ethnicity, socioeconomic status and rurality. Ethical approval was obtained from the Ministry of Health Ethics Committee. Written informed consent was obtained from all participating women and partners. Partners were only approached with the consent of each recruited woman.

A child cohort of 6853 was created following the recruitment of 6822 pregnant women and 4404 partners. The child cohort was defined as those children alive at six weeks of age. The cohort includes 11% of the children born in NZ during the recruitment period. Broad generalisability of the enrolled cohort to the national birth cohort from 2007 to 2010 has been shown (Morton et al., 2014). The statistically significant small differences in the
proportion of the cohort children compared to all births nationally that were born low birth weight (4.9% vs. 6.1%, \( p<0.0001 \)) or preterm (6.4% vs. 7.4%, \( p=0.001 \)) is a reflection, at least in part, of the requirement for survival to age six weeks in order to become a cohort participant.

To date *Growing Up in New Zealand* has collected information from both mothers and their partners from before their children were born, and subsequently undertaken several further data collection waves, most recently completing data collection at age 54 months.

**Study population and sample**
Of the 6853 children in the *Growing Up in New Zealand* cohort, information for 6476 (95%) children was collected at the nine month data collection wave and information of the 6327 (92%) was collected at the two year data collection wave. Two year interviews were completed with 6242 mothers and 3804 of their partners. There are 164 children from whom two year data is available for even though their mothers had skipped the previous data collection wave (the nine month interview). Consent to link was obtained from 6682 (98%) and was established for 6674 (97%) of the children.

**Data collection**
Four *Growing Up in New Zealand* data collection waves were conducted within the first two years of the children’s lives (Morton et al., 2014). To date longitudinal information collected includes that from face-to-face interviews, telephone interviews, and data linkage (Table 14).

Data collection within *Growing Up in New Zealand* seeks age-appropriate information across six inter-connected domains: family and whanau, societal context and neighbourhood, education, health
and wellbeing, psychological and cognitive development, and culture and identity. Other key issues that guide the development of methods and specific tools used for each data collection wave include the relevant constructs to be measured at specific time points and transitions, policy-relevance and the overarching longitudinal research questions and objectives (available at www.growingup.co.nz).

For the study described here data up to age two years will be described (Table 14). Dave have been collected through face-to-face computer assisted interviews completed independently with each mother and partner antenatally and when the children were nine months and two years old. Computer assisted brief telephone interviews were completed with each mother when the children were 6 weeks, 35 weeks, 16 months and 23 months old (Morton et al., 2013).

In addition linkage to health care records has allowed access to data which describes hospital presentations, beginning from the birth event and also permission to access other health records, including community laboratory investigation and primary care records. The primary linkage variable used was the National Health Index Number (NHI), a unique identifier assigned to each person in New Zealand upon their first contact with the health care system. Linkage to routinely collected perinatal health records was completed in 2011, providing information about the latter stages of pregnancy, birth records and immediate neonatal outcomes. Linkage was established using unique identifiers created for each family, mother, enrolled partner and each cohort child. With respect to the project described here, linkage to the perinatal data set provided information on gestation, birthweight, and mode of delivery.
Access to linked data remained incomplete at the time of this thesis submission. Written informed consent had only been obtained for access to hospital records up to age 12 months, and only limited numerical data on inpatient records were available. The process for identification, collection and linkage of all primary care and community laboratory records and of hospital outpatient records for the entire cohort had not yet been established.

**Table 14.** Data collection methods used in *Growing Up in New Zealand*

<table>
<thead>
<tr>
<th>Child age</th>
<th>CAPI*</th>
<th>CATI†</th>
<th>Child‡</th>
<th>Data linkage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antenatal</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perinatal</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>6 weeks</td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>35 weeks</td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 months</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 months</td>
<td></td>
<td></td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>16 months</td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>23 months</td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 years</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
</tbody>
</table>

* CAPI computer assisted personal interview  
† CATI computer assisted telephone interview  
‡ Child measurement

Data obtained at data collection waves up to when the cohort children were two years old were utilised to examine associations between potential risk factors and self-reported peanut allergy.

**Measurements**

**Definition of peanut allergy**
During the two year data collection, 6297 participants provided a response to the question “Has your doctor ever told you {NAME} has an allergy lasting six months or more?”. Of those who answered yes, a follow-up question was then asked “Looking at this
showcard, could you tell me what {NAME} is allergic to?” (Figure 8).

Figure 8. Growing Up in New Zealand 2 year data collection showcard

Potential risk factors associated with the development of childhood food allergy, and more specifically peanut allergy were identified through a review of the literature. Factors were categorized based on the measurement of child, family & wider influences on disease in early childhood as defined by Growing Up in New Zealand: child characteristics and environmental exposures which were categorised as those measuring proximal social environments, distal social environments and macro environmental factors (Table 15).
Table 15. Measurement of child, family & wider influences on diseases in early childhood

<table>
<thead>
<tr>
<th><strong>Child characteristics</strong></th>
<th>CAPI*</th>
<th>CM†</th>
<th>DL‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early life infectious disease events</td>
<td>✓</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Size at birth and perinatal health</td>
<td></td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Child anthropometry</td>
<td></td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Early feeding patterns</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immunisation</td>
<td></td>
<td>✓</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Proximal social environments</strong></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Family structure, including parents, siblings and extended family</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child’s home physical and social environment</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parental health, parental and family socioeconomic indicators</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethnic identity</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Distal social environments</strong></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Neighbourhood (physical location, local engagement, proximity to services, informal support available)</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transport and access to local services</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early childhood well child care</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Interaction with social services</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Macro environmental factors</strong></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternity care and continuity of access to primary health care services, &amp; costs</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parental leave policies and impact on parents returning to work after their children are born</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Provision of early childhood child-care</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family support measures including any family taxation relief or benefits</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*CAPI Computer assisted personal interview
†CM Child measurement
‡Data linkage
Thirty-two variables were identified based on a review of the literature and were evaluated statistically, using univariate analyses and multivariate regression analyses (Table 16).

**Table 16.** Organisation of variables based on influences on diseases in early childhood defined by *Growing Up in New Zealand*

<table>
<thead>
<tr>
<th>Category</th>
<th>Variables</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Child characteristics</strong></td>
<td></td>
</tr>
<tr>
<td>Early life infectious disease events</td>
<td>Skin infections 9 months</td>
</tr>
<tr>
<td></td>
<td>Skin infection since 9 months</td>
</tr>
<tr>
<td></td>
<td>Eczema or dermatitis 9 months</td>
</tr>
<tr>
<td></td>
<td>Eczema since 9 months</td>
</tr>
<tr>
<td></td>
<td>Respiratory infections 9 months</td>
</tr>
<tr>
<td></td>
<td>Respiratory infections since 9 months</td>
</tr>
<tr>
<td></td>
<td>Asthma up to child age 2 years</td>
</tr>
<tr>
<td></td>
<td>Antibiotics up to child age 2 years</td>
</tr>
<tr>
<td>Size at birth and perinatal health</td>
<td>Gestational Age</td>
</tr>
<tr>
<td></td>
<td>Birthweight</td>
</tr>
<tr>
<td>Child anthropometry</td>
<td>Gender</td>
</tr>
<tr>
<td>Early feeding patterns</td>
<td>Breastfeeding</td>
</tr>
<tr>
<td></td>
<td>Hypoallergenic Formula</td>
</tr>
<tr>
<td></td>
<td>Child never tried nuts or peanuts</td>
</tr>
<tr>
<td><strong>Proximal social environments</strong></td>
<td></td>
</tr>
<tr>
<td>Child’s home physical &amp; social environment</td>
<td>Smokers in the home</td>
</tr>
<tr>
<td>Parental health, parental and family socioeconomic indicators</td>
<td>Eczema – Mother</td>
</tr>
<tr>
<td></td>
<td>Hay fever – Mother</td>
</tr>
<tr>
<td></td>
<td>Food Allergy – Mother</td>
</tr>
<tr>
<td></td>
<td>Asthma - Mother</td>
</tr>
<tr>
<td></td>
<td>Smoking – During pregnancy</td>
</tr>
<tr>
<td></td>
<td>Smoking – At child age 9 months</td>
</tr>
<tr>
<td></td>
<td>Anti-reflux medication 1st trimester</td>
</tr>
<tr>
<td></td>
<td>Avoid nuts - Mother</td>
</tr>
<tr>
<td></td>
<td>Body mass index (BMI) - Mother</td>
</tr>
</tbody>
</table>
Age - Mother
Highest Education – Mother

<table>
<thead>
<tr>
<th>Ethnic identity</th>
<th>Ethnicity - Mother</th>
</tr>
</thead>
</table>

Distal social environments

| Neighbourhood (physical location, local engagement, proximity to services, informal support available) | Rurality |

Macro environmental factors

<table>
<thead>
<tr>
<th>Maternity care and continuity of access to primary health care services, and costs</th>
<th>Delivery Method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamins, Multi, Minerals since 1st trimester</td>
<td></td>
</tr>
<tr>
<td>Folate 1st trimester</td>
<td></td>
</tr>
<tr>
<td>Folate after 1st trimester</td>
<td></td>
</tr>
</tbody>
</table>

Statistical analyses
The proportion of the cohort with peanut allergy were determined with the denominator being the number of cohort children for whom data were obtained at the two-year face-to-face interview to the food allergy specific questions (n=6297). Confidence intervals for this proportion (p) were calculated using the normal approximation to the binominal distribution, with the standard error (se) of the proportion (p) being se(p)=√(p/(1-p)/n) and the 95% confidence interval being ± 1.96 x se, where n= the sample size (Altman, 1991).

To begin with a chi-square test was performed to determine if the association of each independent variable with the dependent variable (presence of peanut allergy) was statistically significant (sum=observed/expected; null=no significant difference between observed values and expected frequencies). Each identified significant variable was then included in a univariate analysis. With the univariate analysis we assumed that the response variable is
influenced only by one other factor (i.e. peanut allergy vs. eczema). Each significant variable was then considered for the multivariate analysis. In some instances variables were grouped together (i.e. mother with atopic history = mother with eczema or hay fever or allergy; antibiotic use was grouped into yes/no in place of ordinal 1-4 variables). In some instances variables were dropped: (1) hypoallergenic formula because there were a small number of hypoallergenic formula users in the peanut group; (2) skin infection because of the associated with antibiotic use; (3) multivitamins because of missing values (10%); (4) rurality due to small numbers; (5) BMI due to missing values; and (6) folate and anti-reflux due to missing values.

Logistic regression analyses were performed to allow associations to be described using odd ratios (OR) and 95% confidence intervals (CI). Independent associations were described using adjusted OR and 95% CI generated from multivariable logistic regression models. Analyses were conducted using SAS software (version 9.4, SAS Institute, Cary, NC, US). A two-sided P-value of <0.05 was considered statistically significant.

**Results**

Within the *Growing Up in New Zealand cohort*, history of a doctor diagnosed allergy was reported for 651 (10%) children at two years of age (Morton et al., 2014). The most common specific allergens described were egg (36%, 235 children), dairy (36%, 234 children), peanuts (25%, 162 children), and house dust mites (15%, 95 children). In addition, other atopic illnesses were common for children at two years of age. These include 1655 (26%) of children had been told by a doctor that they had eczema and 725 (12%) of children had been told by a doctor that they had asthma (Morton et al., 2014).
Based on parental self-report 162 (2.6%, 95% CI 2.2-3.0%) children were identified as having a history of a doctor diagnosed peanut allergy.

**Univariate analyses**
Results are categorized based on the measurement of child, family & wider influences on disease in early childhood as defined by *Growing Up in New Zealand*: child characteristics, proximal social environments, distal social environments, and macro environmental factors (Table 16).

**Child characteristics**
From the univariate analysis eight variables related to child characteristics were identified that were independently associated with the presences of parental reported peanut allergy at age two years (Table 17). The odds of having parental reported peanut allergy at age two years were increased for boys compared with girls (OR 1.70, CI 1.24-2.37).

Early life infectious disease events increased the odds for having parental reported peanut allergy at age two years specifically for children whose parents reported a skin infection at age nine months (OR 1.74, CI 1.06-2.72), and since age nine months (OR 2.04, CI 1.40-2.92), as compared to children who had never had an infection; for children whose parents reported eczema or dermatitis at nine months (OR 9.80, CI 6.60-15.08), and eczema since nine months (OR 12.34, CI 8.46-18.55), as compared to children who had never had eczema or dermatitis; and for children who were prescribed either 1-2 courses (OR 1.77, CI 1.11-2.93), 3-4 courses (OR 2.19, CI 1.32-3.74), or 5+ courses (1.73, CI 0.96-3.12) of antibiotics by two years as compared to those who had never received antibiotics.
Early feeding patterns related to the use of hypoallergenic formula increased the odds of having parental reported peanut allergy at age two years for children who were fed hypoallergenic formula (OR 7.54, CI 4.17-12.84) as compared to those who were not. Children who had not tried nuts or peanuts (OR 0.49, CI 0.30-0.76) had decreased odds of having parental reported peanut allergy at age two years as compared to children who had tried nuts or peanuts.

No associations with the presence of parent reported peanut allergy were evident for variables describing respiratory infections since 9 months, asthma in the child ever at 2 years, gestational age, birthweight or exclusive breastfeeding.
Table 17. Univariable associations of child characteristics with presence of parent-reported peanut allergy at age two years

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total n (%)</th>
<th>Peanut Allergy</th>
<th>p-value</th>
<th>Univariable odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Yes (n=162)</td>
<td>No (n=6135)</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td>0.001</td>
</tr>
<tr>
<td>Male</td>
<td>3532 (52)</td>
<td>104 (64)</td>
<td>3140 (51)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>3321 (48)</td>
<td>58 (36)</td>
<td>2981 (49)</td>
<td></td>
</tr>
<tr>
<td>Skin Infections 9 months - Child</td>
<td></td>
<td></td>
<td></td>
<td>0.019</td>
</tr>
<tr>
<td>Yes</td>
<td>534 (8)</td>
<td>21 (12)</td>
<td>480 (8)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>5940 (92)</td>
<td>138 (87)</td>
<td>5485 (92)</td>
<td></td>
</tr>
<tr>
<td>Skin Infections since 9 months</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Yes</td>
<td>869 (14)</td>
<td>39 (24)</td>
<td>825 (13)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>5457 (86)</td>
<td>123 (76)</td>
<td>5309 (87)</td>
<td></td>
</tr>
<tr>
<td>Eczema or dermatitis 9 months - Child</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Yes</td>
<td>2151 (33)</td>
<td>130 (82)</td>
<td>1913 (32)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>4310 (67)</td>
<td>28 (18)</td>
<td>4038 (68)</td>
<td></td>
</tr>
<tr>
<td>Eczema since 9 months - Child</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Yes</td>
<td>1655 (26)</td>
<td>129 (80)</td>
<td>1509 (25)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>4664 (74)</td>
<td>32 (20)</td>
<td>4619 (75)</td>
<td></td>
</tr>
<tr>
<td>Respiratory Infections 9 months - Child</td>
<td></td>
<td></td>
<td></td>
<td>0.612</td>
</tr>
<tr>
<td>Never</td>
<td>4731 (73)</td>
<td>121 (76)</td>
<td>4348 (73)</td>
<td></td>
</tr>
<tr>
<td>1-3 Times</td>
<td>1551 (24)</td>
<td>33 (21)</td>
<td>1440 (24)</td>
<td></td>
</tr>
<tr>
<td>4+ Times</td>
<td>191 (3)</td>
<td>5 (3)</td>
<td>174 (3)</td>
<td></td>
</tr>
<tr>
<td>Respiratory Infections since 9 months</td>
<td></td>
<td></td>
<td></td>
<td>0.276</td>
</tr>
<tr>
<td>Never</td>
<td>3822 (60)</td>
<td>88 (55)</td>
<td>3719 (61)</td>
<td></td>
</tr>
<tr>
<td>1-3 Times</td>
<td>2041 (32)</td>
<td>61 (39)</td>
<td>1968 (32)</td>
<td></td>
</tr>
<tr>
<td>4+ Times</td>
<td>457 (7)</td>
<td>12 (7)</td>
<td>442 (7)</td>
<td></td>
</tr>
<tr>
<td>Asthma - Child ever 2 years</td>
<td></td>
<td></td>
<td></td>
<td>0.107</td>
</tr>
<tr>
<td>Yes</td>
<td>726 (11)</td>
<td>25 (15)</td>
<td>695 (11)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>5594 (89)</td>
<td>137 (85)</td>
<td>5432 (89)</td>
<td></td>
</tr>
<tr>
<td>Antibiotics - Child 2yrs</td>
<td></td>
<td></td>
<td></td>
<td>0.028</td>
</tr>
<tr>
<td>Never</td>
<td>1432 (23)</td>
<td>22 (14)</td>
<td>1406 (23)</td>
<td></td>
</tr>
<tr>
<td>1-2 Courses</td>
<td>2645 (42)</td>
<td>71 (44)</td>
<td>2562 (42)</td>
<td></td>
</tr>
<tr>
<td>3-4 Courses</td>
<td>1306 (21)</td>
<td>43 (27)</td>
<td>1256 (21)</td>
<td></td>
</tr>
<tr>
<td>5+ Course</td>
<td>919 (15)</td>
<td>24 (15)</td>
<td>888 (15)</td>
<td></td>
</tr>
<tr>
<td>Gestational Age</td>
<td></td>
<td></td>
<td></td>
<td>0.210</td>
</tr>
<tr>
<td>&lt;37 weeks</td>
<td>436 (6)</td>
<td>6 (4)</td>
<td>395 (6)</td>
<td></td>
</tr>
<tr>
<td>37-41 weeks</td>
<td>6234 (91)</td>
<td>154 (95)</td>
<td>5565 (91)</td>
<td></td>
</tr>
<tr>
<td>&gt;41 weeks</td>
<td>166 (2)</td>
<td>2 (1)</td>
<td>150 (2)</td>
<td></td>
</tr>
<tr>
<td>Birthweight</td>
<td></td>
<td></td>
<td></td>
<td>0.161</td>
</tr>
<tr>
<td>&lt;2500 grams</td>
<td>336 (5)</td>
<td>4 (2)</td>
<td>309 (5)</td>
<td></td>
</tr>
<tr>
<td>2500-4000 grams</td>
<td>5396 (79)</td>
<td>125 (77)</td>
<td>4803 (79)</td>
<td></td>
</tr>
<tr>
<td>&gt;4000 grams</td>
<td>1110 (16)</td>
<td>33 (20)</td>
<td>1003 (16)</td>
<td></td>
</tr>
<tr>
<td>Exclusive Breastfeeding</td>
<td></td>
<td></td>
<td></td>
<td>0.424</td>
</tr>
<tr>
<td>&lt;=1 Month</td>
<td>1139 (18)</td>
<td>33 (21)</td>
<td>1025 (18)</td>
<td></td>
</tr>
<tr>
<td>2-4 Months</td>
<td>2138 (35)</td>
<td>47 (30)</td>
<td>1961 (34)</td>
<td></td>
</tr>
<tr>
<td>5-9 Months</td>
<td>2912 (47)</td>
<td>77 (49)</td>
<td>2728 (48)</td>
<td></td>
</tr>
<tr>
<td>Hypoallergenic Formula</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Yes</td>
<td>108 (2)</td>
<td>16 (10)</td>
<td>87 (1)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>6345 (98)</td>
<td>143 (90)</td>
<td>5862 (99)</td>
<td></td>
</tr>
<tr>
<td>Child never tried nuts or peanuts</td>
<td></td>
<td></td>
<td></td>
<td>0.002</td>
</tr>
<tr>
<td>Yes</td>
<td>1503 (23)</td>
<td>21 (12)</td>
<td>1416 (24)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>4948 (77)</td>
<td>138 (87)</td>
<td>4529 (76)</td>
<td></td>
</tr>
</tbody>
</table>

*Fisher’s Exact Test
Proximal social environments
From the univariate analyses nine variables related to proximal social environments were identified that were independently associated with the presence of parental reported peanut allergy at age two years (Table 18). These variables described parental health, parental and family socioeconomic indicators, and ethnic identity.

Based on parental health the odds of having a parental reported peanut allergy at age two years were increased for children whose mothers had ever been diagnosed with eczema (OR 1.80, CI 1.23-2.58), hay fever (OR 1.48, CI 1.06-2.06), and/or food allergy (OR 1.72, CI 0.99-2.79) as compared to those who had not been diagnosed with each of these conditions; for children whose mothers used anti-reflux tablets within the first trimester (OR 1.97, CI 1.09-3.29) as compared to those who had not; for children whose mothers had avoided nuts during their pregnancy (OR 3.84, CI 0.91-10.92) as compared to those who had not; and for children whose mothers were underweight (OR 2.27, CI 1.22-3.92) as compared to those whose mothers were of normal weight.

The odds of having a parental reported peanut allergy at age two years were decreased for children whose mothers had smoked within the first nine months of the child’s life (OR 0.46, CI 0.23-0.82) as compared to those who abstained from smoking and for children whose mother was overweight (OR 0.64, CI 0.40-0.99) or obese (OR 0.63, CI 0.37-1.02) as compared to those whose mother were of normal weight.

With respect to ethnic identity the odds of having a parental reported peanut allergy at age two years were increased for
children whose mothers who self-identified as Asian (OR 2.93, CI 1.99-4.30) compared with European ethnicity.

With respect to maternal education the odds of having a parental reported peanut allergy at age two years were decreased for children whose mothers had no secondary school qualifications (OR 0.42, CI 0.16-0.93) or secondary school/NCEA 1-4 (OR 0.27, CI 0.14-0.49) as compared to those with a higher degree.

No associations with the odds of parent allergy were evident for variables describing smokers in the home, the mother having asthma, smoking during pregnancy, or maternal age.
Table 18. Univariable associations of proximal social environments with presence of parent-reported peanut allergy at age two years

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total</th>
<th>Peanut Allergy</th>
<th>p-value</th>
<th>Univariable odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>Yes (n=162)</td>
<td>No (n=6135)</td>
<td></td>
</tr>
<tr>
<td>Smokers in Home</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1895 (29)</td>
<td>36 (22)</td>
<td>1704 (29)</td>
<td>0.102</td>
</tr>
<tr>
<td>No</td>
<td>4572 (71)</td>
<td>123 (77)</td>
<td>4259 (71)</td>
<td>0.73 (0.50-1.05)</td>
</tr>
<tr>
<td>Eczema – Mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>987 (15)</td>
<td>39 (25)</td>
<td>918 (15)</td>
<td>0.002</td>
</tr>
<tr>
<td>No</td>
<td>5480 (85)</td>
<td>119 (75)</td>
<td>5046 (85)</td>
<td>1.80 (1.23-2.58)</td>
</tr>
<tr>
<td>Hayfever – Mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1732 (27)</td>
<td>56 (35)</td>
<td>1611 (27)</td>
<td>0.019</td>
</tr>
<tr>
<td>No</td>
<td>4735 (73)</td>
<td>102 (65)</td>
<td>3533 (73)</td>
<td>1.00</td>
</tr>
<tr>
<td>Food Allergy – Mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>429 (7)</td>
<td>17 (11)</td>
<td>391 (7)</td>
<td>0.037</td>
</tr>
<tr>
<td>No</td>
<td>6038 (93)</td>
<td>141 (87)</td>
<td>4496 (93)</td>
<td>1.72 (0.99-2.79)</td>
</tr>
<tr>
<td>Asthma-Mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>5325 (82)</td>
<td>26 (16)</td>
<td>5065 (82)</td>
<td>0.650</td>
</tr>
<tr>
<td>No</td>
<td>1142 (18)</td>
<td>132 (87)</td>
<td>4999 (82)</td>
<td>1.00</td>
</tr>
<tr>
<td>Smoking- Pregnancy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Continues</td>
<td>657 (11)</td>
<td>8 (6)</td>
<td>547 (10)</td>
<td>0.114</td>
</tr>
<tr>
<td>Stopped</td>
<td>617 (10)</td>
<td>10 (7)</td>
<td>534 (10)</td>
<td>0.67 (0.33-1.23)</td>
</tr>
<tr>
<td>Non Smoker</td>
<td>4972 (80)</td>
<td>124 (87)</td>
<td>4456 (80)</td>
<td>1.00</td>
</tr>
<tr>
<td>Smoking-9 months</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>912 (14)</td>
<td>11 (7)</td>
<td>828 (14)</td>
<td>0.012</td>
</tr>
<tr>
<td>No</td>
<td>5557 (86)</td>
<td>148 (93)</td>
<td>5137 (86)</td>
<td>0.46 (0.23-0.82)</td>
</tr>
<tr>
<td>Anti-reflux 1st trimester</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>355 (6)</td>
<td>15 (11)</td>
<td>314 (6)</td>
<td>0.014</td>
</tr>
<tr>
<td>No</td>
<td>5899 (94)</td>
<td>127 (89)</td>
<td>5228 (94)</td>
<td>1.97 (1.09-3.29)</td>
</tr>
<tr>
<td>Avoid Nuts - Mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>36 (1)</td>
<td>3 (2)</td>
<td>30 (&lt;1)</td>
<td>0.052*</td>
</tr>
<tr>
<td>No</td>
<td>6903 (92)</td>
<td>159 (98)</td>
<td>6105 (100)</td>
<td>3.84 (0.91-10.92)</td>
</tr>
<tr>
<td>BMI - Mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;18.5 (underweight)</td>
<td>259 (4)</td>
<td>14 (10)</td>
<td>204 (4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>18.5 - &lt;25 (normal weight)</td>
<td>3295 (54)</td>
<td>90 (61)</td>
<td>2792 (55)</td>
<td>2.27 (1.22-3.92)</td>
</tr>
<tr>
<td>25 - &lt;30 (overweight)</td>
<td>1372 (23)</td>
<td>24 (16)</td>
<td>1247 (23)</td>
<td>0.64 (0.40-0.99)</td>
</tr>
<tr>
<td>&gt;=30 (obese)</td>
<td>1124 (19)</td>
<td>19 (13)</td>
<td>991 (18)</td>
<td>0.63 (0.37-1.02)</td>
</tr>
<tr>
<td>Mothers Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;25 years</td>
<td>1340 (19)</td>
<td>20 (12)</td>
<td>1117 (18)</td>
<td>0.085</td>
</tr>
<tr>
<td>25-34 years</td>
<td>3835 (55)</td>
<td>91 (56)</td>
<td>3415 (56)</td>
<td>0.67 (0.40-1.07)</td>
</tr>
<tr>
<td>35+ years</td>
<td>1740 (25)</td>
<td>51 (31)</td>
<td>1586 (26)</td>
<td>1.00</td>
</tr>
<tr>
<td>Highest Education - Mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No secondary school qualifications</td>
<td>501 (7)</td>
<td>6 (4)</td>
<td>395 (6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Secondary school/NCEA 1-4</td>
<td>1646 (24)</td>
<td>14 (9)</td>
<td>1430 (23)</td>
<td>0.42 (0.16-0.93)</td>
</tr>
<tr>
<td>Diploma/trade certificate/NCEA 5-6</td>
<td>2110 (31)</td>
<td>52 (32)</td>
<td>1859 (30)</td>
<td>0.27 (0.14-0.49)</td>
</tr>
<tr>
<td>Bachelor’s degree</td>
<td>1563 (23)</td>
<td>54 (33)</td>
<td>1420 (23)</td>
<td>0.77 (0.50-1.20)</td>
</tr>
<tr>
<td>Higher degree</td>
<td>1076 (16)</td>
<td>36 (22)</td>
<td>996 (16)</td>
<td>1.05 (0.69-1.63)</td>
</tr>
<tr>
<td>Ethnicity - Mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>European</td>
<td>3667 (53)</td>
<td>66 (41)</td>
<td>3448 (57)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Maori</td>
<td>961 (14)</td>
<td>20 (12)</td>
<td>818 (13)</td>
<td>1.00</td>
</tr>
<tr>
<td>Pacific</td>
<td>1012 (15)</td>
<td>23 (14)</td>
<td>803 (13)</td>
<td>1.28 (0.75-2.08)</td>
</tr>
<tr>
<td>Asian</td>
<td>1013 (15)</td>
<td>46 (28)</td>
<td>919 (13)</td>
<td>1.50 (0.91-2.38)</td>
</tr>
<tr>
<td>Other</td>
<td>243 (4)</td>
<td>7 (4)</td>
<td>212 (3)</td>
<td>2.93 (1.99-4.30)</td>
</tr>
</tbody>
</table>

*Fisher’s Exact Test
**Distal social environments**
In a univariate analysis there was no association of rurality with the presence of parental reported peanut allergy at age two years (Table 19).

**Table 19.** Univariable associations of distal social environments with presence of parent-reported peanut allergy at age two years

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total</th>
<th>Peanut Allergy</th>
<th>Univariable odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>Yes (n=162) n (%)</td>
<td>No (n=6135) n (%)</td>
</tr>
<tr>
<td>Rurality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-rural</td>
<td>6439 (93)</td>
<td>156 (96)</td>
<td>5667 (93)</td>
</tr>
<tr>
<td>Rural</td>
<td>476 (7)</td>
<td>6 (4)</td>
<td>451 (7)</td>
</tr>
</tbody>
</table>

**Macro environmental factors**
From the univariate analyses one variable that described macro environmental factors was identified as associated with the presence of parent reported peanut allergy. The odds of having a parental reported peanut allergy at age two years were increased for children who were delivered through a caesarean section (OR 1.47, CI 1.04-2.04) as compared to those delivered vaginally.

No associations with parent reported food allergy were present for variables describing vitamin, multivitamin or mineral use since the 1st trimester, folate use in the first trimester, and folate taken after the first trimester (Table 20).
Table 20. Univariable associations of macro environmental factors with presence of parent-reported peanut allergy at age two years

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total</th>
<th>Peanut Allergy</th>
<th>Univariable odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>Yes (n=162) n (%)</td>
<td>No (n=6135) n (%)</td>
</tr>
<tr>
<td>Delivery Method</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vaginal</td>
<td>5180 (76)</td>
<td>111 (69)</td>
<td>4645 (76)</td>
</tr>
<tr>
<td>C-Section</td>
<td>1605 (24)</td>
<td>51 (31)</td>
<td>1455 (24)</td>
</tr>
<tr>
<td>Vitamins, Multi, Minerals since 1st trimester</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3261 (52)</td>
<td>87 (61)</td>
<td>2961 (53)</td>
</tr>
<tr>
<td>No</td>
<td>3005 (48)</td>
<td>55 (39)</td>
<td>2591 (47)</td>
</tr>
<tr>
<td>Folate 1st trimester</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>4995 (80)</td>
<td>124 (87)</td>
<td>4546 (82)</td>
</tr>
<tr>
<td>No</td>
<td>1269 (20)</td>
<td>18 (13)</td>
<td>1004 (18)</td>
</tr>
<tr>
<td>Folate – After 1st trimester</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3460 (55)</td>
<td>95 (67)</td>
<td>3123 (56)</td>
</tr>
<tr>
<td>No</td>
<td>2802 (45)</td>
<td>47 (33)</td>
<td>2425 (44)</td>
</tr>
</tbody>
</table>

Multivariate regression analyses
For the purposes of the multivariable analysis some simplification of the variables identified as potentially important in the univariable analyses was performed. Antibiotic use before age 2 years was re-categorised as never versus at least 1 course; a maternal history of eczema, hay fever or food allergy were combined into one variable which described whether any or none of these manifestations of atopic disease were present.

From the multivariable analysis six variables were identified that were independently associated with the presence of parental reported peanut allergy at age two years (Table 21). The odds of having parental reported peanut allergy at age two years were increased for boys compared with girls (OR 1.59, CI 1.13-2.26); for children whose parents reported a doctor diagnosis of eczema since age nine months compared with children with no diagnosis of eczema since age nine months (OR 10.72, CI 7.26-15.31); for children whose mothers were of Asian ethnicity compared with European ethnicity (OR 2.27, CI 1.48-3.43); and for children whose mothers had a history of atopy (eczema, hay fever or food allergy) compared with children whose mothers did not have a history of atopy (OR 1.40, CI 1.00-1.97). Variables independently associated
with a decreased odds of parent-reported peanut allergy were lower maternal education (no secondary qualifications (OR 0.35, 0.11-0.89) or secondary school/NCEA 1-4 (OR 0.27, CI 0.14-0.51)) compared with mothers who had received a higher degree and for children who had never tried nuts or peanuts as compared to those who had tried nuts or peanuts (OR 0.56, CI 0.33-0.89).

No independent associations with the presence of parent reported peanut allergy at age two years were evident for variables describing maternal smoking, child antibiotic use or delivery method.
Table 21. Independent associations of variables describing child characteristics, proximal and distal social environments and macro-environmental factors with the odds of development of a peanut allergy in early childhood based on self-report data from *Growing Up in New Zealand*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Child characteristics</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1.59 (1.13-2.26)</td>
</tr>
<tr>
<td>Female</td>
<td>Reference</td>
</tr>
<tr>
<td>Eczema since 9 months - Child</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>10.72 (7.26-16.31)</td>
</tr>
<tr>
<td>Antibiotics – Child 2yrs</td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>Reference</td>
</tr>
<tr>
<td>At least 1 course</td>
<td>1.50 (0.95-2.47)</td>
</tr>
<tr>
<td>Child never tried nuts or peanuts</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>0.56 (0.33-0.89)</td>
</tr>
<tr>
<td></td>
<td>Proximal social environments</td>
</tr>
<tr>
<td>Eczema, Hayfever or Food Allergy – Mother</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1.40 (1.00-1.97)</td>
</tr>
<tr>
<td>Smoking-9 months</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>0.64 (0.31-1.21)</td>
</tr>
<tr>
<td>Highest Education - Mother</td>
<td></td>
</tr>
<tr>
<td>No secondary qualifications</td>
<td>0.35 (0.11-0.89)</td>
</tr>
<tr>
<td>Secondary school/NCEA 1-4</td>
<td>0.27 (0.14-0.51)</td>
</tr>
<tr>
<td>Diploma/Trade Certificate/NCEA 5-6</td>
<td>0.70 (0.44-1.14)</td>
</tr>
<tr>
<td>Bachelor’s Degree</td>
<td>0.87 (0.56-1.38)</td>
</tr>
<tr>
<td>Higher Degree</td>
<td>Reference</td>
</tr>
<tr>
<td>Ethnicity - Mother</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>Reference</td>
</tr>
<tr>
<td>Maori</td>
<td>1.36 (0.77-2.30)</td>
</tr>
<tr>
<td>Pacific</td>
<td>1.64 (0.94-2.77)</td>
</tr>
<tr>
<td>Asian</td>
<td>2.27 (1.48-3.43)</td>
</tr>
<tr>
<td></td>
<td>Macro environmental factors</td>
</tr>
<tr>
<td>Delivery Method</td>
<td></td>
</tr>
<tr>
<td>Vaginal</td>
<td>Reference</td>
</tr>
<tr>
<td>C-Section</td>
<td>1.29 (0.89-1.84)</td>
</tr>
</tbody>
</table>

**Confirmation of parental report**

As previously mentioned linkage to health care records allowed access to data describing hospital presentations and other health records, including community laboratory investigation and primary care records. To confirm sensitisation based on parental reported peanut allergy, laboratory results and hospital records were reviewed. From the 162 parental reported cases of peanut allergy,
consent to link to health records was received from 140 parents and caregivers. Laboratory results were then collected from community providers, where available, and interpreted based on the serum specific IgE grade reported and SPT results predicting chance of reaction at challenge >95% (Sinclair et al., 2013). Positive results were defined as SPT≥ 4mm and serum specific IgE≥ grade 1. Where there were no lab results available, hospital medical records were reviewed. By reviewing hospital medical records, additional lab test results were obtained and in some instances documentation from the allergy clinic was included as evidence of sensitisation. There was evidence to confirm sensitisation amongst 81 (50%) of the total parental reported peanut allergic children (Figure 9).

**Figure 9.** Process to confirm sensitisation based on parental reported peanut allergy
Discussion

Principal findings
Based on parental report 162 (2.6%) of children were identified as peanut allergic within the Growing Up in New Zealand (GUiNZ) longitudinal birth cohort. This is comparable to rates observed worldwide as described by Rona et al. in a meta-analysis where self-reported prevalence of peanut allergy varied from 0% to 2% (Rona et al., 2007). Additionally, this prevalence is comparable to the HealthNuts study in Australia (2.9%) (Osborne et al., 2011). Study designs differ in that HealthNuts recruited a representative population from childhood immunization sessions in Melbourne to specifically investigate the prevalence of food allergy in infants (12-month-olds). The study used multiple assessment tools including the gold standard oral food challenge as opposed to parental report.

From the univariate analysis eight variables related to child characteristics were identified that were associated with the development of a peanut allergy in early childhood. Early life infectious disease events increased the odds for having parental reported peanut allergy at age two specifically for children whose parents reported a skin infection at either age nine months and/or since nine months; eczema or dermatitis at nine months, eczema since nine months and for children who were prescribed at least 1 course of antibiotics by the age of two years. Associations with parent reported food allergy at age two years were present for variables describing early feeding patterns, in particular, an increased odds association with the use of hypoallergenic formula, and a decreased odds associated with no consumption of nuts or peanuts.

Nine variables related to proximal social environments were identified that were associated with the presence of parental
reported peanut allergy at age two. Specifically the odds of having parent reported peanut allergy were increased for children whose mother had a history of an eczema, hay fever and/or food allergy diagnosis; for children whose mother used anti-reflux tablets within the first trimester; for children whose mother avoided nuts during their pregnancy; for children of underweight mothers; and for children whose mother was of Asian ethnicity. The odds of parent report peanut allergy were decreased for children whose mother had smoked within the first nine months of the child’s life; or whose mother had only secondary school qualifications.

There was no significant association with the presence of parental reported peanut allergy at age two and distal social environments.

One variable related to the macro environment was identified as associated with the development of a parental reported peanut allergy at age two specifically for children who were delivered through a caesarean section.

From these analyses a multivariate model was developed. From the multivariable analysis six variable were identified that were independently associated with the presence of parental reported peanut allergy at age two years. The odds for having parental reported peanut allergy at age two increased for children who were male; children who had a doctor diagnosis of eczema since age nine months; and children born to mothers who identified as Asian. The odds for having parental reported peanut allergy at age two decreased for children born to mothers with no maternal education or only secondary school/NCEA 1-4 and children who had never tried nuts or peanuts. Children who hadn’t been knowingly exposed to nuts or peanuts were included in the analyses because although participants were asked about their feeding practices related to
peanuts, there is no guarantee that the children haven’t been exposed to nuts or peanuts. For example, this exposure could have occurred through skin contact with a peanut product or by sharing food in a day care setting. In addition, only one member of the family (many times the mother) was asked the question about peanut exposure. This leaves open the possibility that they may not have been aware of all food exposures. With the ADHB chart review we saw that children were mistakenly given a food they were allergic to, this also could have occurred with a child that does not have an as yet diagnosed allergy and therefore would not have realized they had been exposed. Thus there are too many unknown factors to justify the exclusion of the children who had not been exposed to peanuts or nuts in the analysis.

Within the international context, study findings related to eczema are consistent with data reported from other countries. HealthNuts participants who were sensitized to foods were also more likely to have a history of eczema (26.7%, 95% CI, 25.0-28.4) (Osborne et al., 2011). There is an established positive relationship between food allergy and eczema risk and infants who already have eczema are at higher risk of food allergies. In some instance eczema is one of the earliest signs of allergy (Leung, Boguniewicz, Howell, Nomura, & Hamid, 2004; Prescott, 2011). Factors implicated in the aetiology of both conditions include altered permeability of the gut, predisposing genes, and an abnormal immune response (Prescott, 2011).

Additionally, as a result of eczema, the skin is more permeable than usual to allergens which can allow for allergens from the environment to penetrate the skin and induce an allergic response which may contribute to such conditions such as food allergy. The increased risk of peanut allergy when preparations containing
peanut oil are used on the skin in the first 6 months of life provides supportive evidence for the potentially important role that skin integrity plays in the development of peanut allergy (Lack, Fox, Northstone, & Golding, 2003).

Contributing to the breakdown in skin barrier integrity, once an allergic reaction is initiated more immune cells are recruited into the skin to produce many more inflammatory products (Cork et al., 2009). A vicious cycle ensues, making it difficult to determine directional and causal relationships particularly given the variance between individuals in other genetic and environmental risk factors. Food allergy can trigger eczema flares and, in an IgE-mediated food allergic episode, the immediate symptoms of food allergy are usually obvious but can then give way to the more chronic symptoms of eczema (Prescott, 2011).

The changing ethnic make-up of New Zealand and more specifically Auckland, is a dominant demographic feature. Asians have been one of the largest more recent immigrant groups. A greater increase of hospital presentations in children living in Auckland, where the majority of Asian immigrants reside, was evident in the previous study investigating national temporal trends. The increased likelihood of children born to mothers who identified as Asian may be related to a change in diet or other environmental exposures. As previously described the typical Western diet is comprised of refined grains, alcohol, salt, certain oils, corn-derived fructose, fatty domesticated meats, and other foods frequently consumed in caloric excess which not only can promote inflammation and atherosclerosis but has also led to a change in the consumption of omega-3 and omega-6 polyunsaturated fatty acids (PUFA) (Prescott, 2011; Tilg & Moschen, 2015). In addition the
declining intake of soluble fibre has been recognised in the promotion of gut health.

As previously described, based on data collected from local and expatriate Singapore and Philippine schoolchildren, respondents of either Asian or white subjects born in Western countries were at higher risk of peanut and tree nut allergy compared with those born in Asia (Shek et al., 2010). In the population-based HealthNuts study, compared to infants with two Australian-born parents, peanut allergy was more common among infants with at least one parent born in East Asia (OR 3.4, CI 2.2-5.1) but not those with at least one parent born in the UK/Europe (OR 0.8, CI 0.4-1.5) (Koplin et al., 2014). This finding is similar to that presented within the Growing Up in New Zealand data where the odds of having parental reported peanut allergy at age two years were increased for children whose mothers were of Asian ethnicity compared with European ethnicity (OR 2.27, CI 1.48-3.43).

The decreased odds for having parental reported peanut allergy at age two for children born to mothers with no maternal education or only secondary school/NCEA 1-4 and children who had never tried nuts or peanuts may be due to the exposure to peanut. The cost of peanuts may be inhibitory and therefore it is unknown if their child has a food allergy as they have never tried nuts or peanuts. In addition the lack of maternal education could result in a barrier to access health care and therefore they are less likely to get a doctor diagnosis.

The findings related to absence of peanut exposure as a protective factor are contradictory to those recently published from the Learning Early about Peanut Allergy (LEAP) trial. Based on the randomized trial of infants at risk for peanut allergy, findings
concluded that the early introduction of peanuts can significantly reduce the frequency of the development of peanut allergy at age 60 months, 13.7% in the avoidance group and 1.9% in the consumption group (Du Toit et al., 2015). When the study period was extended beyond the initial time frame to include 12 months of peanut avoidance, no association with an increase in the prevalence of peanut allergy was observed (Du Toit et al., 2016). It is important to note key differences between the GUiNZ population and those enrolled in the LEAP trial. As previously mentioned GUiNZ is longitudinal birth cohort study that included a nationally representative population with the purpose of tracking the development of New Zealand children. Whereas the LEAP trial recruited high risk infants - defined as having severe eczema, egg allergy or both – to consume or avoid peanuts until 60 months of age (Du Toit et al., 2013). The differences in the participants between the two studies could account for the differences observed in the outcome related to the risk associated with early introduction of peanut.

Study Limitations
Although the data presented is based on self-report it is evident in the literature that self-report data is least variable amongst those with a peanut allergy as compared to other allergens for example egg and dairy.

The Growing Up in New Zealand study design created some limits to the variables that could be considered. For example, although maternal dietary intake was measured during pregnancy and child dietary practices were described at age 9 months and two years these descriptions were made using a food frequency questionnaire. This creates limits to the degree of dietary detail that can be investigated and to the quantification of dietary exposures both by amount and time. In order to minimise study attrition a biological
sampling component was not included at birth with the exception of the collection of cord blood samples on a small subset of the cohort (n=131) (Morton et al., 2014). However, with the cohort’s engagement with the study having now been established Growing Up in New Zealand has recently begun to develop a biological sampling component of the study. A component of this will be the measurement of biomarkers on dried blood spots collected at birth as part of the national newborn screening programme (Ministry of Health, 2011). We intend to measure dried blood spot 25(OH)D concentration and hence will have the opportunity to subsequently consider the relationship of vitamin D status at birth with the development of peanut allergy in early childhood (Eyles et al., 2010).

Our capacity to confirm parental reported food allergy was incomplete. Importantly we have very high consent rates for health care record linkage and the capacity to create reasonably complete health care records because of the existence of a unique identifier, the National Health Index number. As our processes for obtaining data from the linkage with health care records becomes more fully developed we will be able to establish more robust case definitions for subsequent food allergy related projects.

Research implications
The Growing Up in New Zealand data allows for reflection of the New Zealand population as a whole. The cohort size and diversity provides adequate explanatory power to examine complex developmental life course outcomes.

Opportunities for further research
Food allergy most frequently presents and is diagnosed early in childhood. Thus there is a potential for the prevalence rate of peanut allergy within the Growing Up in New Zealand cohort to
increase at 45 months, during this data collection wave food allergy was assessed in more detail than was considered at age two years.

Therefore the findings from this study provide a foundation for future research and evidence for the need for additional investigation of: (1) parental report of peanut allergy versus confirmed peanut allergy; (2) potential change in rate of peanut allergy between data collection waves (i.e. 45 months); (3) prevalence of other food allergies (i.e. egg and milk); and (4) contribution of genetic and epigenetic factors that require biomarker description for their characterisation.

**Chapter conclusions**

The study described in this chapter provides the first description of prevalence of peanut allergy within the New Zealand context and demonstrates a rate comparable to that seen worldwide. Several independent associations of child characteristics and proximal social environments with the presence of parental reported peanut allergy at age two years were identified.

The next chapter reviews the literature describing the physical, social, and emotional burden of food allergy on the family.
Chapter 6: LITERATURE REVIEW PART 3 - THE IMPACT OF A FOOD ALLERGY ON FOOD ALLERGIC FAMILIES

Chapter objectives
In this chapter I review the literature pertaining to the physical, social, and emotional impact of a food allergy on a food allergic family. Children diagnosed with a food allergy and their families are faced with multiple life challenges, or burdens, including but not limited to: fear, anxiety, depression, bullying, harassment, shame, medical costs, need for increased vigilance, lack of accessible resources, and isolation.

Overview
The term quality of life is comprised of multiple factors including an individual’s health, financial security, standard of living, family and friends, and spiritual contentment (Graham & Blaiss, 2000; Meltzer, 2001). The impact a food allergy has on the quality of life of the food allergic child, their caregivers, and in some instances their siblings has been investigated across several continents - Australia, North America, and Europe - using a variety of qualitative and quantitative methods and instruments.

Although quality of life has been studied worldwide, there is limited published data in New Zealand (NZ). Currently there is one piece of literature in which quality of life issues faced by NZ families raising a food allergic child is described (McBride, McBride-Henry, & van Wissen, 2010).

Qualitative methods include questionnaires, interviews, and visual methods. Quantitative measures have been predominantly used and
the impact of food allergy has been investigated through both generic and disease specific health-related quality of life (HRQL) instruments. HRQL instruments have been developed and validated for use worldwide and include a measurement of the impact of food allergy on parents, teens, and children. Based on a review of HRQL studies, the impact that a health condition may have on an individual can be categorized into three major aspects of overall health: physical, social, and emotional or psychological well-being (Lieberman & Sicherer, 2011). These aspects have been used to shape the literature review and subsequent study (Figure 10).

Healthcare, financial resources, and the ability to purchase and prepare allergen free foods will impact a food allergic family’s physical health. The impact of a food allergy on the food allergic family’s emotional health can be further described through mental health and the need for continuous vigilance. The ability to eat outside the home, management of the food allergy in a childcare and school setting, and social support received when living with a food allergy will impact the social health and well-being of the food allergic family. Each aspect of overall health is further described throughout the literature review and subsequent study.
Figure 10. Food allergy’s impact on the family’s overall health and well-being
Food allergy’s impact on the family’s physical health

Healthcare, financial resources, and the ability to purchase and prepare allergen free foods will impact a food allergic family’s physical health (Figure 11). The ability to receive a timely diagnosis, appropriate follow-up care, and manage acute allergic reactions primarily impacts the food allergic child’s health but can involve multiple members of the family. Financial resources needed are included in this category as the financial costs of a food allergy are seen in both healthcare and food costs. Purchasing and preparing allergen free foods can have a greater impact on the overall family in terms of their nutrition and time spent shopping for appropriate foods.

Figure 11. Food allergy’s impact on the family’s physical health

Healthcare
A food allergy differs from many other chronic diseases; children are generally healthy (although they may suffer from other problems such as eczema or asthma). The food allergic child does not have daily symptoms and does not experience deterioration in physical function, pain, physical restrictions or disability (Pitchforth et al., 2011). This can have potential implications regarding public
perception, which will be discussed within a food allergy’s social impact on a family later in this literature review. Although the food allergic child typically ‘looks healthy’ families face multiple challenges within the area of health including obtaining a timely diagnosis, appropriate follow-up care, support, and management of a reaction.

In a recent article published in the New Zealand Medical Journal, authors provide a summary of the diagnosis and management of IgE-mediated food allergy in New Zealand children (Sinclair et al., 2013). Allergy testing, primarily Skin Prick (SPT) and RAST is used to confirm the cause of an allergic reaction. However in some instances a convincing history suggesting IgE-mediated food allergy will not be confirmed with an investigation (test) (Sinclair et al., 2013). Thus demonstrating the time consuming and potentially frustrating process of food allergy diagnosis. Recommendations stress that testing a small range of common allergens may be considered as opposed to screening for a large group of allergens; and in cases in which children have already tolerated a food testing should not be conducted (Sinclair et al., 2013).

Sinclair et al describes the investigation of the patient’s history as the key to interpretation of testing results. The presence of a specific IgE can be measured through testing however positive results vary by food allergen and age (Sinclair et al., 2013). Thus demonstrating a challenge of food allergy diagnosis. For a child who has had symptoms highly suggestive of an IgE-mediated reaction followed by a positive SPT or RAST test, a diagnosis can be confirmed with some confidence. Whereas to confirm diagnosis in a child whose history is less clear or there is not history of exposure then a more robust laboratory measure is needed. It is important to confirm food allergy as this will aid in ongoing management but
this can lead to a time consuming and resource intensive diagnostic process.

As food allergy is a complex condition, referral to a specialist is needed in some instance and should occur in any child with anaphylaxis, allergy to more than one food allergen, or where the primary care practitioner is not confident about diagnosis, test interpretation, or management (Sinclair et al., 2013). Upon diagnosis, advice about allergen avoidance and a written management plan should be provided to children and young people. In addition regular follow-up, and reassessment, is important as many food allergies are not persistent.

The impact of a food allergy on a family at the healthcare level includes reaching a diagnosis of food allergy; follow-up care associated with a food allergy; and issues related to acute allergic reactions.

**Reaching a diagnosis of food allergy**
As described above diagnosing a food allergy can be challenging, time consuming, and resource intensive for the food allergic family. They face these challenges and often times become frustrated with the issues created by: having to access resources; make multiple visits to a variety of different health care professionals; experience delays between referrals; and the overall delay in a timely diagnosis of food allergy (McBride et al., 2010; Obeng & Vandergriff, 2008; Pitchforth et al., 2011).

However in some instances families describe the processes of seeking and receiving health care as straightforward, including a description of helpful medical staff involved in the process – for example in the UK study of families (Pitchforth et al., 2011). As the diagnosis of a food allergy is complex and not well understood by all
physicians, multiple studies recommend increased awareness amongst general practitioners (Mandell, Curtis, Gold, & Hardie, 2005; McBride et al., 2010; Pitchforth et al., 2011). This may aid in a families ability to access timely and appropriate health care thus helping to alleviate feelings of anxiety and fear, and providing clarity about how to manage childhood food allergy safely.

In a study of 26 families, a peanut allergy diagnosis had both practical and social consequences (Pitchforth et al., 2011). For example, caregivers can create a safe place at home where they exclude nuts completely. In contrast, when parents are unable to control an environment, they may/can adopt a number of strategies: having an epinephrine autoinjector on hand at all times; careful reading of product labels; restrictions on eating and shopping habits, food preparation and storage; limiting activities that might involve eating in uncontrolled conditions; direct supervision of the child; and seeking to educate others about the risks posed (Mandell et al., 2005; Pitchforth et al., 2011).

**Follow-up care associated with a food allergy**

The impact a food allergy has on the family can continue even after a diagnosis is confirmed. This post-diagnosis period creates issues with accessing follow-up care, dietary support, pharmaceutical resources, and social support. However families feel more confident in managing their child’s food allergy when a physician provides a referral to an anaphylaxis organisation, takes an active role, and communicates with parents in a respectful way parent (Mandell et al., 2005).

Follow-up care reaches beyond health care professionals as caregivers of children with a food allergy seek support and management strategies from a variety of sources including other parents, friends and relatives, the child with a food allergy, the
internet, books, and non-profit organizations (Mandell et al., 2005; McBride et al., 2010). For example, understanding cross-contamination, food labelling issues, and the risk of trace contaminants is imperative for families when managing food allergies and often requires the collection of information from a variety of sources (Mandell et al., 2005).

**Issues related to acute allergic reactions**
The impact a food allergy has on the family includes avoidance of food allergens and management of acute allergic reactions. Avoidance remains central to the prevention of an allergic food reaction. However even with the best efforts by parents, children are still likely to come in contact with allergens which can lead to a reaction.

Reactions can occur anywhere including home, childcare setting, school, restaurants, and/or public settings (i.e. park, swimming pool). In a study of 83 peanut allergic children, 115 documented adverse reactions caused by accidental exposure to peanuts were reported by 50/83 (60%) (Vander Leek, Liu, Stefanski, Blacker, & Bock, 2000). Reactions occurred in a variety of settings including childcare and school.

First time reactions, which by definition cannot be anticipated, pose a particular challenge. In some instances first-time reactions occur at schools and are not limited to settings such as school cafeterias, which are often the sites of focus for prevention strategies. For example, in a US study, 25% of reactions were first time and the majority of reactions from peanuts or tree nuts in the school setting were caused by food used in class projects (Sicherer, Noone, & Munoz-Furlong, 2001).
As food allergies, especially milk and egg, occur in children prior to attending school reactions can also occur in other childcare settings. Of 132 food allergic children (aged 3-19 years old) almost two thirds had accidental ingestions in the preceding 2 years (Nowak-Wegrzyn, 2001). Milk was the most common cause of reactions in preschools (32%); and peanuts the most common cause of reactions in elementary or primary schools (29%) (Nowak-Wegrzyn, 2001).

Allergic reactions to food are more likely to occur outside of the home, where there is less control of the environment. Dining at a restaurant is a situation where there is an increased risk of a food allergic reaction. This increased risk is due to many factors including a lack of understanding of food allergies by restaurant staff; a low level of knowledge regarding ingredients in the food items available; cross contamination; and a lack of communication between the food allergic individual and the restaurant staff who serve them (Sicherer et al., 2001). Often times (50%) when a reaction occurs it is due to ‘hidden’ foods, for example, in dressings, egg rolls, or sauces (Sicherer et al., 2001).

With the establishment that a child has a food allergy and the recognition that subsequent allergic reactions to food are likely to occur, it becomes necessary for families to develop the ability to recognize the signs of such a reaction and to have in place strategies that enable them to provide emergency treatment at all levels of severity (Miles, Fordham, Mills, Valovirta, & Mugford, 2005). As the most severe cases are likely to result in anaphylaxis, caregivers report a high level of burden associated with a visit to the emergency room (Springston et al., 2010). Appropriate management requires several components to be in place. The food allergic child needs to become responsible for carrying their
epinephrine auto injectors, in addition to other medicines, which for some children provides them a sense of safety while others are fearful (Valentine & Knibb, 2011).

**Financial Resources**
The physical impact a food allergy has on the family includes the financial resources necessary to manage a food allergy in addition to the financial loss that a food allergy family can incur. In the characterisation of the financial impact of food allergies globally, costs are broken down into direct, indirect, and intangible costs (Alanne, Maskunitty, Nermes, Laitinen, & Pekurinen, 2012; Gupta et al., 2013; Miles et al., 2005; Patel, Holdford, Edwards, & Carroll, 2011). The intangible costs, which relate to quality of life, are addressed in both the sections below describing the social and emotional impact a food allergy has on food allergic families.

The costs associated with a food allergy impact the whole family. These include household and health care costs. The additional household costs incurred are created by the family’s need to change, and in some instances maintain, their lifestyle in order to prevent a food related reaction from occurring, for example purchasing allergen free substitute foods or seeking childcare that includes a peanut free environment (McBride et al., 2010; Miles et al., 2005). The length of time these costs are incurred are dependent on the persistence of the food allergy. Allergen free substitute foods are often expensive, and some have argued that this is partly due to the costs of development and the fact that manufacturers who are seen as socially responsible can charge a higher price (Miles et al., 2005). Healthcare system costs are incurred at multiple levels including diagnosis, support, and ongoing education. Household costs have been found to outweigh healthcare costs in all but the most severe cases (Miles et al., 2005).
Direct costs of childhood food allergy
The physical impact a food allergy has on the family includes direct health care costs which are study dependent and can include: the use of emergency services, visits to and by medical professionals, medication, hospitalisation, diagnostic and other laboratory testing, and therapy (such as immunotherapy) (Miles et al., 2005). As previously described in Chapters 2 and 3, only the most severe food allergic reactions result in hospitalisation. Direct costs are further defined by each study described below.

Direct healthcare costs experienced by the family vary between countries as they are dependent upon the healthcare system of each country and the extent to which health care is based upon a user-pays or a state funded model. For example, families may take on the burden of purchasing medications like epinephrine autoinjectors in New Zealand, whereas families living in Australia, Canada, Italy, Portugal, and the United Kingdom have access to subsidised funding of epinephrine autoinjectors (Simons, 2009). Such differences in health care system organisation make it difficult to compare costs directly between countries or to use cost data from one country to estimate potential costs in another country. Contemporary examples of the health care cost of childhood food allergy are provided using data from Finland and the United States, countries with very different financial structuring of their health care system (Alanne et al., 2012; Gupta et al., 2013; Patel et al., 2011).

A Finnish study reported that within the first two years, food allergy led to a median cost of €3182 euros (mean €4348), with 41% of these costs occurring in the first year (Alanne et al., 2012). In this group, the largest proportions of direct costs were made up of hospital outpatient care, including food challenges, formula for infants with cow’s milk allergy, disability allowances and indirect
costs from travel costs. In Finland the public insurance system paid a significant proportion of the costs due to food allergy and thus alleviates the cost burden to the families (Alanne et al., 2012).

Based on retrospective data from 2006/2007, a US study reported the annual direct medical costs caused by food allergy and anaphylaxis was estimated at US$225 million (Patel et al., 2011). Direct costs included inpatient, ED visits, office visits, and outpatient department costs. Children accounted for 47% of the total inpatient costs, 32% of the ED visit costs, 67% of the office visit costs, and 98% of the total outpatient department visit costs. However this may be an underestimate as these costs were based on cases identified through the use of an epinephrine autoinjector as a result of a reaction and not all cases could be identified using ICD-9 codes (Patel et al., 2011).

More recently, a US study described the direct costs including both medical and household costs borne for the diagnosis, treatment, and prevention of childhood food allergy. Based on the figure of 5.9 million US children with food allergies, an annual cost of US$24.8 billion or US$4184 per child was reported. These costs are comprised of direct medical costs (US$4.3 billion) and household costs (US$20.5 billion) (Gupta et al., 2013). Direct medical costs include hospitalisations (US$1.9 billion), outpatient visits to allergists (US$819 million), ED visits (US$764 million), and paediatrician visits (US$543 million) (Gupta et al., 2013). Household costs were estimated at US$5.5 billion (US$931 per child) and included insurance co-payments, medications, and special diets (US$1.7 billion) and special childcare arrangements (US$857 million) (Gupta et al., 2013).
Very minimal data exist currently on the direct cost of childhood food allergy in New Zealand. The costs will include those for the purchase of epinephrine autoinjectors (ca NZ$150) and are not currently subsidised by the NZ public healthcare system (McBride et al., 2010). Families can qualify for the Work and Income New Zealand Child Disability Allowance, to assist them in the costs associated with having a child with a disability, however individuals need to be aware of their eligibility and therefore many may missing out on this financial assistance. This allowance is made to the main carer of a child or young person with a serious disability as recognition of the extra care and attention needed for that child ("Work and Income," n.d.). The child disability allowance as of April 2015 is a weekly, non-taxable, rate of NZ$46.49 ("Work and Income," n.d.).

**Indirect costs of childhood food allergy**

The physical impact a food allergy has on the family includes indirect costs and are largely made up of loss of productivity which impacts both the food allergic child and their caregiver. Loss of productivity is due to an interruption in the caregivers’ workday or inability to complete work responsibilities. For example, a food allergic child may be more likely to miss school as a result of their condition and thus caregivers may also miss work; or they may find it hard to work regular hours or overtime due to additional responsibilities at home resulting from the management of a food allergy (i.e. medical visits, attending school events, responding to an emergency) (Mandell et al., 2005; Miles et al., 2005). In some countries, a child with a severe food allergy has to eat lunch at home due to school policies (Miles et al., 2005). In a US study, lost labour productivity costs associated with caregivers accompanying their food allergic child to medical visits totalled US$773 million (US$130 per child) (Gupta et al., 2013). The majority of these
visits were to allergists, followed by paediatricians, and visits to the ED.

Total opportunity costs in the US were estimated by multiplying the percentage of caregivers reporting lost opportunity in the labour market by the mean caregiver-reported cost and the number of US children with food allergies; resulting in a loss of annual opportunity costs totalling an estimated US$14 billion or US$2399 per child (Gupta et al., 2013).

**Food and Nutrition**
The physical impact a food allergy has on the family includes food and nutrition as there is an increased demand placed on food allergic families in terms of purchasing and preparing foods to prevent accidental ingestion of an allergen.

A routine trip to the grocery store can lead to an increased level of anxiety, stress, and frustration as time is devoted to reading labels, finding appropriate allergen free foods, and travelling to multiple stores (Miles et al., 2005; Valentine & Knibb, 2011). Parents of food allergic children describe a sense of injustice and discrimination due to lack of availability of foods for their allergic child as compared, for example, to a child who is a vegetarian or has other dietary restrictions (Pitchforth et al., 2011).

A family’s home can allow for a greater sense of control, for example many choose to keep this environment allergen free. To ensure the child has an allergen free diet research suggests parents of food allergic children often focus on foods that they know are safe to eat, adapt recipes, make appropriate substitutions, and prepare many meals from scratch (Muñoz-Furlong, 2003; Pitchforth et al., 2011). Because this does require additional time for meal preparation, developing routines is paramount (Springston et al.,
In some instances, parents may impose rules of restriction to manage the child’s eagerness to consume ‘forbidden’ food (Pitchforth et al., 2011). The amount of risk a family is willing to take differs as some choose to introduce an allergen into the home to allow for the opportunity to teach the child how to avoid certain foods (Muñoz-Furlong, 2003). Although some food allergic children must adhere to a restricted diet, an unintended positive consequence of preparing meals from scratch is that food allergic families tend to consume healthier diets as they are less likely to eat calorie dense, pre-packaged and takeaway meals (Valentine & Knibb, 2011).

Another management strategy utilised by food allergic families – food allergic child, siblings, and parents - is label reading, however this is an arduous task and food labels are not always easy to decipher and are constantly changing (Muñoz-Furlong, 2003; Obeng & Vandergriff, 2008). For example, the use of advisory statements has increased dramatically over the past decade (Muñoz-Furlong, 2003; Pitchforth et al., 2011). Companies have adopted these voluntary statements, of which there are more than a dozen versions, to indicate that there may be a risk of cross-contact with an allergen in another product and/or that the allergen is present somewhere in the manufacturing plant (Muñoz-Furlong, 2003). The use of these labels is widespread and has led to frustration for many families as they question whether there truly is a risk of cross-contact, especially when it is a food they have been consuming for years. A growing number of families have chosen to ignore the advisory statements (Muñoz-Furlong, 2003; Pitchforth et al., 2011).
Food allergy’s impact on the family’s emotional health
Mental health and the need for continued vigilance will impact a
food allergic family’s emotional health (Figure 12). The fear, worry,
and guilt that accompanies a food allergy is expressed by multiple
members of the food allergic family. In addition the need for
continued vigilance leads to an increased amount of responsibility
placed on the food allergic child, though not solely.

Figure 12. Food allergy’s impact on the family’s emotional health

Mental health
The emotional impact a food allergy has on the family is its
association with an increased risk of developing a mood or anxiety
disorder. A Canadian study reported that individuals with self-
reported food allergy were more likely to suffer from mood and
anxiety disorders (major depressive disorder, panic disorder, and
bipolar disorder) than the community population (Patten & Williams,
2007).

Caregivers with a food allergic child suffer from increased levels of
stress, anxiety, and guilt. In a study investigating mental health
access, 70 percent of caregivers reported that mental health
support would have been helpful, but only 23 percent had seen a
mental health professional (Annunziato et al., 2012). That such a high proportion of caregivers of food allergic children perceive the need for mental health support is noteworthy given that such children are not expected to be suffering from a diagnosable mental health disorder. That such a small proportion of those who perceived the need actually accessed mental health support was due to the fact that they did not think about it; were too overwhelmed/busy; were not recommended to do so, and/or it was perceived as too costly. This unmet need for mental health support is a particularly important issue because we know from other research that isolation is a common consequence of a food allergy, impacting not only the food allergic child but also their family (McBride et al., 2010).

Caregivers struggle with feelings of worry and guilt when their child has been diagnosed with a food allergy (Miles et al., 2005; Muñoz-Furlong, 2003; Valentine & Knibb, 2011). Caring for a child with a food allergy creates a day-to-day burden that needs to be managed and can lead to an emotional strain on the caregiver of the food allergic child (Valentine & Knibb, 2011). The impact is variable, with some caregivers being more impacted by food allergy than others. For example poor quality of life was significantly more likely to be reported by caregivers whose children had been to the ED in the past year, were more knowledgeable about food allergy, had multiple food allergies, or where the allergy was to specific foods (Springston et al., 2010).

Within the families of peanut-allergic children, mothers described more anxiety and stress than fathers, and children with compared to their siblings without peanut allergy report worse health related quality of life (HRQL) scores regarding physical health, general quality of life, and greater separation anxiety (King, Knibb, &
Hourihane, 2009). When compared with non-allergic children, those children with a nut allergy have poorer social, emotional, and psychosocial HRQL scores (Cummings et al., 2010). However, the anxiety of both the mother and food-allergic child are lower when the food-allergic child has versus has not been prescribed an epinephrine auto-injector.

A child who has experienced a severe reaction can develop disordered eating, become withdrawn, or fearful it is important for the caregivers to achieve a balance between protecting their child against the seriousness of their food allergy and building confidence in their child’s ability to manage their condition and lead as normal a life as possible. Parents need to be able to appreciate that providing the food allergic child with management strategies will lead to a feeling of empowerment and enable their child to feel more confident, and therefore be less likely to make mistakes, take unnecessary risks, and more likely to return to normal health in a timely manner after the reaction (Muñoz-Furlong, 2003).

Constant Vigilance
The need for constant vigilance is an emotional impact of a food allergy felt by some food allergic families. The need for constant vigilance in all environments (e.g. school, camp, cafes etc.) over time takes an emotional toll on the food allergic family and leads to feelings of stress and anxiety (Lieberman & Sicherer, 2011). A ‘fear of food’ can develop over time although no food has been ingested and can be a source of stress to the family (Miles et al., 2005; Obeng & Vandergriff, 2008; Sicherer, Muñoz-Furlong, Murphy, Wood, & Sampson, 2003). High levels of anxiety were present after initial diagnosis and again after allergic episodes (Mandell et al., 2005).
Compared to non-allergic children, parents constantly monitor their child’s diet and behaviour, which can lead to overprotective parenting (Miles et al., 2005). Anxiety levels decrease when families are able to obtain sufficient information and concrete suggestions about avoidance and risk management in addition to adopting suitable strategies (Mandell et al., 2005). The anxiety and fears of children and parents were most intense in the middle childhood age group (age 6-11 years), when the child’s autonomy and comprehension of the dangers steadily increase but the capacity for self-protective responsibility is not yet completely developed. An appropriate level of anxiety may be a necessary for families coping with anaphylaxis in a child (Mandell et al., 2005).

The disease-specific health related quality of life (HRQL) of children with food allergy was first reported in 2003. In this first study children with peanut allergy were compared to children with insulin-dependent diabetes mellitus (IDDM) (Avery, King, Knight, & Hourihane, 2003). The HRQL was lower in the children with peanut allergy. In comparison with the children with IDDM, children with peanut allergy experienced more fear of an adverse event associated with eating, more anxiety about eating in general, and were more restricted in their physical activities (Avery et al., 2003). In addition peanut allergic children had greater awareness that their condition might be fatal as compared to children with IDDM despite the greater longer-term implications of IDDM. In comparison with the children with IDDM, compliance with specific aspects of management plans, was greater for food allergic children (Avery et al., 2003). It was hypothesised that this was a consequence of the children having greater disease-specific anxiety.

Due to the need for constant vigilance while living with a food allergy, an increase in the need for responsibility and perception of
needing to keep oneself safe is felt by some food allergic families. From a young age children with food allergy are trained to remain vigilant and scan for risk across a range of environments, including mobilising urgent and immediate support from their parents (Pitchforth et al., 2011).

**Food allergy’s impact on the family’s social health and well-being**

The ability to eat outside of the home, management of the food allergy within a childcare or school setting, and the social support received when living with a food allergy will impact the social health and well-being of the food allergic family (Figure 13). The ability to interact with peers outside of the home impacts multiple members of the food allergic family. The policies enacted within the school setting in regard to food allergies in addition to the possibility of the food allergic child being bullied or feeling excluded within this setting all impact the social health of the food allergic child. Social support is a key determinant of quality of life and can include public perception. Food allergy has been described as an ‘invisible’ condition which is related to the public’s perception of severity which can impact a food allergic family’s social health and well-being.

**Figure 13.** Food allergy’s impact on a family’s social health and well-being
**Eating Outside the Home**

A food allergy impacts the family socially as it adds a level of complexity to eating outside of the home. This may include eating outside of the home with family members or with friends. Food allergic children may be given the responsibility of inquiring about potential allergenic foods when eating outside the home as there is an increased risk for accidental ingestion of hidden allergies, due to the exclusion of ingredient statements; confusion with ingredient labels; and staff who may lack the knowledge and training to assist food-allergic individuals (Miles et al., 2005; Muñoz-Furlong, 2003; Obeng & Vandergriff, 2008; Pitchforth et al., 2011). For example, hospitality staff may not be aware of the need to wash their hands after handling nuts. In a comparison study, peanut allergic children are more afraid of eating peanuts than diabetics were of having a hypoglycaemic attack (Avery et al., 2003). A large proportion of peanut allergic children (85%), as compared to diabetics (50%), feel that they always have to be careful about what they eat, especially in restaurants (Avery et al., 2003).

The inability of food allergic children to eat outside of the home leads to limitations to engage in social or peer interactions (Obeng & Vandergriff, 2008; Pitchforth et al., 2011). For example many children attend events like birthday parties, sleep overs, playing with friends, school events, camps, and going on holiday. A food allergy can impact attendance, and in some instances invitation, to these events which has been shown to result in lower quality of life (Obeng & Vandergriff, 2008; Pitchforth et al., 2011; Primeau et al., 2000; Springston et al., 2010). For example in a comparison study of diabetic and peanut allergic children; peanut allergic children reported significantly higher anxiety and lower quality of life when on holiday, at birthday parties and using public transport (Avery et al., 2003; Obeng & Vandergriff, 2008).
The impact of these limitations reaches beyond the food allergic child as caretakers report that the difficulties in eating out also influence their quality of life negatively (Valentine & Knibb, 2011). This may be due in part to the pressure placed on parents to convince hospitality staff that their child’s food allergies are real (Muñoz-Furlong, 2003). Beyond the café and restaurant setting, when travelling, an airline is unable to guarantee a peanut-free flight as they may have peanut ingredients in their meals or other passengers may carry peanuts on the plane with them (Muñoz-Furlong, 2003). Families of peanut allergic children avoid eating food served on flights, as ingredient lists are not usually available.

A second comparison study based on parental report in Canada, Primeau et al reported that peanut allergic children had significantly more disruption in their daily activities, including more disruption in their familial – social interactions as compared to children with a rheumatologic disease (Primeau et al., 2000). For some families, social limitations can lead to a food allergic family feeling excluded and may disproportionately impact caregivers (Springston et al., 2010; Valentine & Knibb, 2011).

**Childcare and schools**
A food allergy’s social impacts include childcare and school settings. These setting were included as children spend a large proportion of their young lives in a childcare and school setting where they are exposed to their peers in many situations involving food. Similar challenges present themselves in this setting as they do in others (i.e. home, cafes, friend’s house etc.). Families are relying on others to consistently maintain a safe environment for their food allergic children and caregivers often describe feelings of discomfort and anxiety in dealing with schools and childcare (Springston et al., 2010).
Childcare and school policies
To manage food allergies within the school system, the best line of defence is to provide training to staff to minimize risks, recognize symptoms, and act quickly once a reaction occurs (Muñoz-Furlong, 2003). Schools therefore are in need of policies and protocols to adopt strategies for the management of food allergies. However in some instances a national policy is lacking and therefore putting children at risk.

Within the New Zealand education system there currently is no legislative protection or national policy to support families, including formal protocols or guidance provided to schools (McBride et al., 2010). In contrast, in the United States federal law provides this support and schools cannot exclude children with food allergies from participating in school functions because of their food allergies (Food Allergy & Anaphylaxis Network, 1995; Muñoz-Furlong, 2003). In addition schools are required to administer a child’s medications, including epinephrine autoinjectors, during a reaction, even if they do not have a full-time nurse (Food Allergy & Anaphylaxis Network, 1995; Muñoz-Furlong, 2003).

Beyond central government mandates schools can adopt policies at the local level. For example, in the US, schools have adopted policies that require that only commercially prepared food containing pre-printed ingredient statements may be sent to school (Muñoz-Furlong, 2003). Thus allowing the teacher or caregiver to read the ingredient list to determine whether the food is safe. This policy still requires that the teacher be provided with the proper education to be able to decipher the ingredient labels and does place an added responsibility on teachers. A more practical policy to adopt non-edible treat policies or class parties and celebrations (i.e. stickers, colourful pencil erasers, sport cards, and other child-
friendly collectibles) is being chosen by many schools (Muñoz-Furlong, 2003).

Caregivers report that their anxieties would ease during school hours if the school setting adopted a no peanut or no nut products policy (Obeng & Vandergriff, 2008). Additionally, to ensure safety many parents elect to provide their child’s food not only for lunchtime but also during celebrations or snack time. To combat feelings of exclusion and prevent stigma, caregivers send enough snacks or party fare to school for their food allergic child to share, therefore eating the same food as classmates (Muñoz-Furlong, 2003). In some instances caregivers choose to attend an event within the school setting to ensure the safety of their food allergic child (Obeng & Vandergriff, 2008).

Within the school setting, Valentine et al. reported the greatest impact on quality of life was restrictions forced upon children in terms of school lunches. Different school policies, or in some instances no policy, have been adopted in regards to the lunch room setting, for example providing allergen free tables or no nut policies. In 2009 allergists were advised by key researchers in the field – Young, Munoz-Furlong, and Sicherer – that children should not be separated from their friends during school lunches and that if ‘allergen-safe’ tables and classrooms are used, friends with safe lunches should also be allowed to sit at the table (Valentine & Knibb, 2011).

Within the United Kingdom this advice has not been implemented in the education system and both the food allergic child and their caregiver reported that segregation and restrictions on school lunches had a negative impact on their quality of life (Valentine & Knibb, 2011). Additional research in schools described that where
nuts have not been banned from packed lunches, a food allergic child has to decide on a safe place to sit which can lead to enacted stigma, including being teased and taunted (Muñoz-Furlong, 2003; Pitchforth et al., 2011).

**Bullying, harassment, and exclusion**
Food allergic children describe feelings of anxiety and exclusion in the school setting when eating treats in the classroom; eating in a group setting; eating food served by the school system; and school trips (Mandell et al., 2005; Obeng & Vandergriff, 2008; Pitchforth et al., 2011; Valentine & Knibb, 2011). In addition food allergic children described being bullied, teased, or harassed due to their food (Lieberman, Weiss, Furlong, Sicherer, & Sicherer, 2010). Both physical - allergen thrown at them or having their food purposely contaminated with an allergen - and nonphysical acts - verbal teasing and taunting - were reported. Non-physical acts were more common (Lieberman et al., 2010). Quality of life can be improved when staff at the food allergic child’s school are helpful and supportive (Valentine & Knibb, 2011).

An unintended consequence of a food allergy reported by Flokstra-de Blok et al., was that food allergic children and adolescents reported fewer limitations in schoolwork or activities with friends due to behavioural problems. As described throughout this literature review, a food allergy demands that the child be conscious of their behaviour, especially regarding situations involving food, emphasizing avoidance of impulsive behaviour. The demands of managing this condition may result in more consciousness about behaviour in other situations as well, resulting in less behavioural problems than in children of the general population (Flokstra-de Blok et al., 2010).
Overall, the school environment is manageable and can facilitate a safe environment for a food allergic child. However, a consistent approach across all schools is needed to allow for all food allergic children to receive the same levels of care. In addition support services offered to children with other chronic health conditions within the education environment need to be provided to children diagnosed with a food allergy (McBride et al., 2010). By providing schools with support at a national level they will be more equipped to adopt management strategies; provide a safe environment for children to learn in; and will ease the caregiver anxiety and stress. Living in a supportive environment can be a significant tool in managing a food allergy. Examples provided by the literature include other family members; participation in support groups; health care professionals; school personnel; parents of food allergic children; advocacy organizations; and increased public awareness (Annunziato et al., 2012; Mandell et al., 2005). In some instances parents of children with anaphylaxis report relying heavily on school and day care personnel, relatives, neighbours, and friends not only to act swiftly and properly in case of an unpredictable emergency, but also to participate in restrictive avoidance strategies (Mandell et al., 2005).

**Social support**
A food allergy’s social impacts include the importance of social support and public perception. For some caregivers, family and social support are described as key determinants of quality of life (Pitchforth et al., 2011; Valentine & Knibb, 2011). For example, Sicherer et al., reported that parents of food-allergic children had significantly poorer HRQL for general health perception, emotional impact on the parent, and limitation on family activities compared with previously established norms (Lieberman & Sicherer, 2011).
However an unexpected outcome was that the food allergic family achieve higher scores in the measure of family cohesion.

Siblings have been labelled as a source of support and in some instances they have described feelings of resentment and fear (Miles et al., 2005; Muñoz-Furlong, 2003). Siblings have taken a high level of personal responsibility for their food allergic brother or sister’s safety; which may include avoiding the allergenic food itself outside of the home (Mandell et al., 2005).

Caregivers of food allergic children may perceive a difference between the supports available to their food allergic child as compared to children with other chronic health conditions. For example in Britain, children with diabetes can attend specific camps, diabetes clinics, and often have access to diabetes nurse specialists, psychologists, and dieticians (Avery et al., 2003). Based on these findings, similar services do not appear to be so widely available to families with severe food allergies. These types of resources could help to address major issues of anxiety and lifestyle adaptation in families affected by peanut allergy (Avery et al., 2003). In addition, enabling education opportunities for families to meet is an important way of providing support and information, sharing ‘tips’, bolstering confidence, promoting reflection, and enabling carer and family members to feel included (Pitchforth et al., 2011).

**Public perception**

Food allergic children and their families can face many challenges and they develop management strategies to combat them. However, one area that may pose the most significant challenge to manage is that of public perception.
Families living in England (UK) with a peanut allergic child, based on confirmed diagnosis, described through interviews that they often feel as though there is a stigma attached to having a child with this allergy (Pitchforth et al., 2011). Families reported major obstacles to coping including lack of public understanding; willingness to accommodate; even hostility; inconsistent medical information; mislabelled foods and inconsistent labelling practices; and the presence of multiple food allergies (Mandell et al., 2005).

Both food allergic children and their caregivers encounter individuals, including family and friends, who are quick to judge and do not believe that their allergy is a serious and life-threatening condition (McBride et al., 2010; Muñoz-Furlong, 2003; Pitchforth et al., 2011; Springston et al., 2010). Caregivers are often treated as neurotic, over-anxious, fussy, attention seeking, and a self-indulgent fad when they raised concerns, asked for special arrangements, or requested information. Caregivers describe unsympathetic responses from those who felt that their behaviour was excessive and unnecessary (Pitchforth et al., 2011).

In part this is due to a food allergy being described as an ‘invisible’ condition. To provide the child with a safe environment caregivers have to make the allergy public. Which in turn exposes parents to risks of stigma and discomfort (Pitchforth et al., 2011). The majority of the time family members and friends are described as supportive however in some instances they do not understand the severity of an allergy (Pitchforth et al., 2011). In some instances those who do not believe the food allergy diagnosis may attempt to give the child the restricted food, not infrequently causing a reaction (Muñoz-Furlong, 2003; Pitchforth et al., 2011).
Chapter conclusions
In this chapter I reviewed the literature pertaining to the physical, social, and emotional burden on the family living with a food allergy. A food allergy impacts a family’s health, financial security, and standard of living. However families exhibit a lack of self-pity, and emphasize coping and management strategies while striving for an optimal balance between vigilance and normal daily living (Mandell et al., 2005). Recognizing the severity of food allergies at the local, national, and international level can lend support to policy, clinical practice guidelines, and improved public awareness (McBride et al., 2010).

Although the food allergic child typically ‘looks healthy’ families face multiple challenges within the domain of health including obtaining a timely diagnosis, appropriate follow-up care, support, and management of a reaction. Not only is diagnosing a food allergy challenging, time consuming, and resource intensive but the transition period after diagnosis poses issues with accessing follow-up care, dietary support, pharmaceutical resources, and social support. This leads to direct health care costs of food allergy that include the use of emergency services, visits to and by medical professionals, medication, hospitalisation, diagnostic and other laboratory testing, and therapy (such as immunotherapy) (Miles et al., 2005).

Beyond healthcare and costs, an additional physical burden is the need for avoidance as this remains central to the prevention of a food-related acute allergic reaction. Through a search of the literature, the home was established as a place where families have greatest control, and many choose to keep this environment allergen free. To ensure the child has an allergen free diet parents of food allergic children focus on foods that they know are safe to
eat, adapt recipes, make appropriate substitutions, and prepare many meals from scratch (Muñoz-Furlong, 2003; Pitchforth et al., 2011).

Socially, caregivers describe unsympathetic responses from those who felt that their behaviour was excessive and unnecessary (Pitchforth et al., 2011). In part this is due to a food allergy being described as an ‘invisible’ condition. This in turn leads to the likelihood of that a caregiver’s social relationship will suffer. In addition the food allergic child suffers from feeling of anxiety and exclusion, specifically in the school setting. Beyond the school setting, the inability of food allergic children to eat outside of the home leads to limitations to engage in social interactions (Obeng & Vandergriff, 2008; Pitchforth et al., 2011). A significant tool in managing a food allergy is living in a supportive environment. This environment is made up of other family members; participation in support groups; health care professionals; school personnel; parents of food allergic children; advocacy organizations; and increased public awareness (Annunziato et al., 2012; Mandell et al., 2005).

At the emotional level, living with a food allergy leads to an increase in responsibility and an increased perception of needing to keep oneself safe. In addition, having a food allergy is associated with an increased risk of developing a mood or anxiety disorder not only for the food allergic child but also their caregivers are at an increased risk. This is due, in part, to the need for constant vigilance in all environments (e.g. school, camp, cafes etc.) over time takes an emotional toll on the food allergic family and leads to feelings of stress and anxiety (Lieberman & Sicherer, 2011).
To date there is limited published data regarding the quality of life and food allergic children, including their families, in New Zealand. To better understand the impact of a food allergy on the daily life of a food allergic New Zealand family, and management strategies employed, the following chapter presents a study in which seven food allergic families were asked to capture photographs that describe their experiences of living with a food allergy.
Chapter 7: THE IMPACT OF A FOOD ALLERGY ON NEW ZEALAND FOOD ALLERGIC FAMILIES

Chapter objectives
In this chapter the results of a qualitative study with seven New Zealand families with a food allergic child are presented. The impact of a food allergy on the families’ quality of life is explored through reflexive photography and autodriven group interviews. The families’ strategies for managing their child’s allergy are also discussed.

Abstract
Introduction
Living with a food allergy can present multiple challenges for individuals with allergies and their families including: fear, anxiety, depression, bullying, harassment, shame, medical costs, a need for increased vigilance, a lack of accessible resources, isolation, and exclusion. Food allergy can also have a significant impact on the way families interact with relatives, friends, health care providers, food producers, retailers, and schools. Currently there is limited published data describing quality of life issues, and management of a food allergy, by New Zealand families raising a food allergic child.

Methods
Seven families were recruited to participate in a project utilising reflexive photography to tell their story. Participants were asked to take simple photographs that “tell stories” about living with a food allergy. These photographs were then used as a foundation for discussion about the impact of food allergy on the food allergic family including management strategies.

Results
The impact a food allergy has on a family is strongly influenced by place, or the environment, including: home, school, community,
and beyond the community. This is due, in part, to the amount of control a food allergic family has within these different locations. The ability to control the environment gradually decreases as a food allergic family moves away from the home. Management strategies - safe food storage systems, label reading, asking what is in a food, and carrying a medical kit at all times - have been developed and employed at each environmental level to provide a safe and healthy environment for their children to thrive. Within the home responsibility is shared amongst all members of the family. However outside of the home, when the food allergic child is on their own, the responsibility shifts solely to themselves.

Discussion
This study provides a snap-shot of seven families’ daily lives and the impact food allergies have. Three outcomes of living with a food allergy are evident throughout all environmental levels are responsibility, exclusion, and resilience. The results of this study will add to the growing body of food allergy literature, specifically in the New Zealand context. In addition the lessons learned from these families can be used to guide other food allergic families through education and advocacy. Based on the family’s description of the impact of a food allergy on their quality of life and management strategies, there is a need for: consistent national school policy focused on food allergies and the provision of education and training to hospitality workers. These changes could positively impact food allergic families at multiple environmental levels.
**Introduction**
The term quality of life is comprised of multiple factors including an individual’s health, financial security, standard of living, family and friends, and spiritual contentment (Graham & Blaiss, 2000; Meltzer, 2001). Food allergy can impact the individual diagnosed and their family, friends, health care providers, food producers, retailers, and schools. Studies spanning multiple continents – North America, Europe, and Australia - have described experiences of food allergic children and their families: fear, anxiety, depression, bullying, harassment, shame, increased medical costs, need for increased vigilance, lack of accessible resources, isolation, and feelings of exclusion (Flokstra-de Blok et al., 2008; Pinczower et al., 2013; Primeau et al., 2000; Shemesh et al., 2013; Sicherer, Noone, & Munoz-Furlong, 2001). Living with a food allergy affects the whole family through social interactions, emotional demands, and resources. From working to get a diagnosis to providing a safe environment at home; navigating social interactions, the grocery store, and school; a food allergy is with a child and in many cases their family 24-hours a day, 365 days a year and for some this is lifelong.

Currently there is one piece of literature in which quality of life issues faced by New Zealand families raising a food allergic child is described (McBride et al., 2010). McBride and colleagues reported parents of children living with medically diagnosed severe food allergy (MDSFA) in New Zealand experienced isolation, significant burden of accessing resources, and increased costs associated with food allergy. As this research provides us the perspective of a limited number of parents, there is a need to further understand the impact on the family unit – food allergic children, siblings, and parents. This would allow for an understanding of unmet needs in addition to providing other food allergic families with information.
pertaining to management strategies. Therefore a focus of the study described here is to provide the experience faced by each member of the family. In addition, based on the family’s contributions, the following study provides a description of how to improve public awareness and build a stronger, more inclusive, and adaptable community and school support system to fit the needs of food allergic families.

**Methods**

**Engaging children in qualitative research: Using visual methods**

Three visual methods were blended to ensure that the participant was engaged throughout the study: reflexive photography, photo elicitation, and the autodriven interview. The use of reflexive photography allowed for the participant to capture simple photographs that “tell stories” about living with a food allergy. Photo elicitation allowed for the use of the photographs to provide the researcher with the opportunity to learn more about participants’ daily activities. The autodriven interview ensured the participant was “driving” the interview, questions were not constructed prior to meeting with the food allergic families.

Reflexive photograph, photo elicitation, and autodriven interview were utilised due to their flexible and interactive nature which are beneficial characteristics when working with children (Avery et al., 2003; Harper, 2002; O’Connell, 2013). Children may be more comfortable in expressing themselves through a visual approach of representation as it allows for active participation and keeps them focused on the task at hand (Buckingham, 2009; Croghan, Griffin, Hunter, & Phoenix, 2008; Dell Clark, 1999; Mauthner, 1997). The narratives provided in response to the images presented, act as an insider’s guide, through their eyes, to their individual experiences over time (Riley, 2004). To gain this perspective it was important
to include all members of the immediate family unit – food allergic children, siblings, and caretakers.

With the integration of visual methods, a child is able to convey their story to the researcher in a comfortable setting, as they are familiar with the model of show and tell; children can use props to convey messages (Dell Clark, 1999). Visual methods utilising props, or photographs, encourages storytelling (Bugos et al., 2014).

Although cliché, a picture is worth a thousand words and it allows for the child to remember what they want to tell the researcher and provides them with a guide for their narrative. The use of visual, or creative, methods is not a new phenomenon. These methods have been used in numerous settings through various techniques - including mapping, photovoice, photo elicitation, autodriven interview, and reflexive photograph - to gain better insight into a participant’s life (Avery et al., 2003; Buckingham, 2009; Croghan et al., 2008; Dell Clark, 1999; Harper, 2002; Mauthner, 1997; O’Connell, 2013; Purcell, 2007; Wang & Burris, 1997). This study incorporated the use of reflexive photography (the participant taking the photos), photo elicitation (insertion of photographs into a research interview), and the autodriven interview.

Reflexive photographs, a term coined by Harper over two decades ago, are photographs produced by research participants and are used as the foundation for a discussion (Harrington & Schibik, 2003). The use of reflexive photography is framed by two theoretical understandings: (1) individual-environmental interaction theories, which have been developed and applied to the higher education settings; and (2) symbolic interactionism, allowing for participant photographs objects that act as symbols of their social interactions (Harrington & Schibik, 2003).
Once the photographs had been taken by the participants a blending of photo elicitation and the autodriven interview was used with each food allergic family to allow them to describe their photographs. These techniques were blended to ensure that the participants were driving the interview, questions were not constructed prior to meeting with the food allergic families.

Photo elicitation provides the researcher with the opportunity to learn more about a participants daily activities through the use of photographs (Purcell, 2007). Based on the four concentration areas of photo elicitation, this technique was chosen for this project as a tool to better understand participant’s identity, how they are marked by illness (food allergy) (Harper, 2002).

To ensure the participant is in a sense “driving” the interview, food allergic families explained their photographs in an unrestricted environment (Clark, 1999). The photos form a basis for the discussion of their experience. The photographs become a part of the interview and allow for participants to self-select relevant events, pertaining to food allergy, from their own lives. This technique was chosen as it increases the researcher’s access to youthful experience in interviews (Clark, 1999). Specifically, the use of this technique allows for the use of photographs to bring about the reality of the needs of food allergic children and their family members. In addition, photographs helped to break the ice when interviewing, particularly the children in the food allergic families. As the photos guide the conversation there was minimal prompting, the picture served as “projective stimuli” (Clark, 1999).
Lastly by utilising this technique record, through the photographs, will be established which can be used in analysis, dissemination, and future project development.
Participants and recruitment
The goal of the study was to recruit seven to ten families to describe for us what strategies are needed for their family to manage, adapt, and feel fully supported while raising a child with a food allergy. A strategy may also be defined as resources. Family was defined as consisting of at least one food allergic child aged 8-12 years and one parent/guardian; a family may consist of additional members. Working with families, through the use of participatory visual methods this project was designed to begin the conversation.

Recruitment occurred through collaboration with Allergy New Zealand a national charity providing reliable information, education, and support to families (Allergy New Zealand, 2010). Allergy New Zealand is a national membership-based society who for over three decades has been committed to all those living in New Zealand with allergies. In addition they advocate to government, policy makers and the media, while providing information and guidance to the health, education and food sectors, and support research (Allergy New Zealand, 2010). A flyer (Appendix B, page 181) was provided and circulated throughout the Allergy New Zealand network via email, Facebook, Twitter, and their website. The sample recruited for this study is a convenience sample focused on the Auckland area and we relied on Allergy New Zealand’s reach to recruit from. Potential participants then contacted the lead researcher for more information and completed a brief demographics survey.
Research approach

Figure 14. Impact of food allergy on New Zealand families: research process

Session 1: Meeting and consent
Once confirmed, families took part in a one-hour introduction and training session. This session involved obtaining consent, discussing privacy concerns in regard to taking pictures, and a brief camera training. The camera training included how to operate the disposable camera, what to photograph, and a reminder about privacy and confidentiality. Due to confidentiality issues, families were asked to not take photographs that included people’s faces. The discussion around what to photograph was left open ended and allowed participants to ask basic questions. Simply stated they were instructed to take simple photographs that ‘tell stories’ about living with a food allergy. The importance of each member of the family taking at least one photo was stressed. Each family was then provided with one disposable camera. A copy of the session one training discussion guide is located in Appendix B (pages 187-189).
Reflexive Photography
Food allergic families were given two weeks to take all of the exposures on their disposable camera. In most cases all exposures were taken however if a family felt they could not think of any more pictures to take that was ok and the camera was collected.

Session2: Family interviews
A follow-up interview to share the photographs was scheduled for each family with the lead researcher. At the beginning of the interview each family was provided with the hard copies of the photographs. This was the first time they had seen their pictures. They were asked to select at least six to seven photos however if a family member, especially a child, was having a tough time choosing between pictures they were allowed to include them all. This strategy was decided upon, as it was not the goal of the project to compare the number of photos discussed between families or even within a family. The photographs were not the end product but more a means to facilitate a discussion. As some of the photographs were not clear enough to be used it was necessary to remain flexible. The discussion was initiated with the interviewer posing broad questions such as “what do you see in the picture?” and “what is happening in the picture?”.

To ensure each member of the family had a chance to provide input, they were each asked to explain their photograph on their own followed by the opportunity for others in the family to comment on an individual photograph. A few families identified photographs they wish they could have taken or feelings they felt they were unable to capture in a photograph. In addition families were asked a series of follow-up questions at the end of each interview to share what advice they would give to other families and what they wish the public knew about food allergy. A copy of the session two interview discussion guide is located in Appendix B (pages 190-
The interviews were subsequently transcribed verbatim and uploaded to NVivo 10 qualitative analysis software.

**Ethical Considerations**

All participants, including children, were provided study details as well as age appropriate participant information sheets (Appendix B). The information provided to participants during recruitment was again provided at session one and participants had the opportunity to ask additional questions. At this time participants were also advised of their right to withdraw from the study if they are uncomfortable at any time. Although participants were provided with the opportunity to withdraw their interview responses, there was not an opportunity to provide edits. Participants completed a consent form and for children under the age of 16, their parent/guardian provided consent on their behalf. Participants could withdraw from the study at any time.

Family interviews took place on a one-on-one format to limit the nervousness of revealing sensitive information that would be present if the discussion was held in a group format; children were accompanied by an adult. To protect participant’s identities, all transcripts were de-identified for coding and participants cannot be identified from the results presented. Only the researcher had access to the audio recordings and any written notes. Participants completed a copyright release form for their selected photos used when presenting the results (Appendix B).

Participants understand that the results of the research will be included in a PhD thesis, but individuals will not be identified. The study was approved by the University of Auckland Human Participants Ethics Committee on 25 January 2014 (Reference #010922).
**Data Analysis**
Photographs and discussion provided by the participants were then categorized using a standardized coding scheme developed specifically for this study (Figure 15). The researcher reviewed complete interview transcripts and developed a list of approximately 25 codes to characterize themes (level 1). Based on similarities between codes these were then collapsed into 13 categories (level 2). Upon further review of the 13 categories it became evident that place played a large role in the food allergic families lives. This leading to the grouping of themes according to four environmental levels; home, school, community, and beyond the community (Figure 15).

![Environmental levels](image)

**Figure 15.** Environmental levels

The 13 categories were then collapsed into three overarching categories for classification of participants’ experiences: finding, storing, and preparing food; potential hazards leading to reactions; and social interactions (level 3). These overarching categories (level 3) were then explored at each environmental level (Figure
16). Specifically I was searching for the impact a food allergy has on the family’s daily life and strategies needed for a New Zealand family to cope, adapt, and feel fully supported when living with a food allergic child. The impact on daily life and management strategies were evident across all environments and at each level: finding, storing, and preparing food; potential hazards leading to reactions; and social interactions.

**Figure 16.** Levels of code developed to characterize themes

Toward the end of the interview each family was asked to reflect on (1) advice they would provide to other families and (2) what they wish the public knew about food allergies.
Not all photographs are included in the following pages, quotes and photographs were selected that exemplified patterns observed in the data and photographs of good quality.

**Results**

**Description of Families**
Using photographs, seven families ranging from three to five members, voiced their experiences regarding the impact food allergy has on their daily lives. Each family included at least one child (8-12 years of age) with a parent-reported, doctor-diagnosed food allergy, one caregiver, and one sibling (Table 22).

**Table 22.** Description of participant families including: members, age of children, age of doctor diagnosis, and foods currently allergic to.

<table>
<thead>
<tr>
<th>Family</th>
<th>Foods Currently Allergic to</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family one: mother, father, seven year old girl, and nine year old boy who was doctor diagnosed with a food allergy at age 5 months.</td>
<td>Dairy, Egg, Sesame, Wheat, Rye, Barley, Oats, Kiwifruit, Banana, Ginger</td>
</tr>
<tr>
<td>Family two: mother, father, thirteen year old boy, nine year old girl, and eleven year old girl who was doctor diagnosed with a food allergy at age 2.</td>
<td>All nuts, Diary, eggs, Shellfish, Sesame Seeds, Kiwifruit, Soy</td>
</tr>
<tr>
<td>Family three: mother, father, thirteen year old boy, and ten year old girl who was doctor diagnosed with a food allergy at age 8 months.</td>
<td>Peanut, Cashew, Walnut, Pistachio, Kiwifruit (Past: Milk)</td>
</tr>
<tr>
<td>Family four: mother, seven year old girl, and nine year old girl who was doctor diagnosed with a food allergy at age 6 months.</td>
<td>Peanuts, Tree Nuts</td>
</tr>
<tr>
<td>Family five: mother, father, seven year old boy, and eleven year old boy who was doctor diagnosed with a food allergy at less than 12 months of age.</td>
<td>Egg, Nuts</td>
</tr>
<tr>
<td>Family six: mother, father, ten year old girl, and twelve year old girl who was doctor diagnosed with a food allergy at age 6 months.</td>
<td>Milk, Eggs, Tree Nuts, Kiwifruit</td>
</tr>
<tr>
<td>Family seven: mother, father, seven year old girl, ten year old girl who was doctor diagnosed with a food allergy at age 3.</td>
<td>Peanuts, Cashew Nuts, Chick Peas, Pistachio Nuts, Soy Protein</td>
</tr>
</tbody>
</table>

Families ranged in the number of food allergens identified. As the sample is a convenience sample it is expected that children with more severe food allergies would be more likely to be part of the
Allergy New Zealand community and are more likely to participate in a research study.

In addition to demographic information parents were asked to provide information pertaining to reactions their food allergic child has had at any point in their lives (Table 23).

Table 23. Reactions suffered by food allergic child identified by parental report.

<table>
<thead>
<tr>
<th>Reaction</th>
<th>Family</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dizziness, feeling your heart beat fast, loss of vision, inability to stand, light-headedness, collapse, and loss of consciousness/passing</td>
<td>X</td>
</tr>
<tr>
<td>Tightening throat, difficulty swallowing, hoarseness/hoarse voice, difficulty breathing in, and shortness of breath, wheezing, cough.</td>
<td>X X X X X X X</td>
</tr>
<tr>
<td>Sick to your stomach, stomach cramps, vomiting, and diarrhoea.</td>
<td>X X X X X X X X</td>
</tr>
<tr>
<td>Itchy skin, red rash, hives, worsening eczema, selling of the skin.</td>
<td>X X X X X X</td>
</tr>
<tr>
<td>Other symptoms like oral allergy, swollen tongue or lips, symptoms of the nose or eyes.</td>
<td>X X X X X X X X</td>
</tr>
</tbody>
</table>

It is important to note that based on parental report all food allergic children participating in this study have had a previous reaction and in most instances a severe reaction. This supports parental self-report of a doctor diagnosed food allergy and provides a foundation for the credibility of the impact a food allergy has on the family.

The participating families had been raising a food allergic child for many years and therefore some of the challenges discussed may be different to those who have been newly diagnosed.
...it’s been our life for 10 years.

This is evident in the paucity of the discussion points around accessing healthcare. A minority of families mentioned that finding appropriate healthcare and getting a diagnosis was challenging, a finding echoed in the literature, but they felt that this has probably changed over time, as allergies are relatively new (McBride et al., 2010; Obeng & Vandergriff, 2008; Pitchforth et al., 2011).

Accessing the information proved to be difficult as well, however once they found the right person or in some instances place, Starship Immunology Clinic (Starship Children’s Hospital), a whole new world opened up. This was not something that was included in photographs but came out of the discussion pertaining to resources.

So yea we were really lost at the beginning...

When she was little it was like getting blood out of the stone.

In addition, it was not unexpected that some of the children who were part of this study commented on other medical issues that they have including other allergies, asthma, eczema, and celiac disease. It is quite common for these chronic health conditions to accompany food allergies and in some instances they can exacerbate food allergic reactions and vice versa. Overall the results presented here identify both the impact on daily life and management strategies.
..it’s annoying having a food allergy because I’m not allowed some stuff.

**Food allergy and environment**
The families discussion indicate that the environment – whether at home, school, out in the local community, and beyond the community – influences how a food allergy impacts on a family’s daily life. This impact is evident across three activities: finding, storing, and preparing food; managing potential hazards leading to reactions; and negotiating social interactions. For example: the storage of allergenic foods at home is easier to manage than in a school setting; the need for an epinephrine autoinjector is present across all environmental levels; and the level of vigilance within a social setting will differ between the environmental levels. Therefore food allergic families have developed and rely on management strategies corresponding to the challenges faced at each environmental level. These strategies help families provide a safe and healthy environment for the food allergic child to grow and develop as any other child their age. The following results are organised first by the environmental level represented and second by the activities presented above. Interwoven within each activity
the impact the food allergy has on daily life, the challenges faced by food allergic families, and management strategies are discussed.

**Managing food allergy at home**
Home is defined by some as a comfort zone, however the environment is not without challenges. Food allergic families are faced with how to: store food safely in the home; choose safe foods to bring into the home; prevent and manage reactions in the home; and provide food allergic children with the opportunity to feel included amongst their peers by inviting them over to their home. Through strategies both developed and learnt, the food allergic family is able to manage and control the home environment more than any other.

To develop the home into the comfort zone families describe it to be, they commonly referred to the creation of systems to minimize risk of a potential reaction. These systems include the use of rules regarding food storage, hand washing regimens, shared responsibility of label reading, rules in regard to the foods allowed in the home, and preparing meals at home from scratch. All of these factors are more easily controlled in the home environment, as compared to the other three environmental levels, which provides a sense of security and reduced anxiety for the entire food allergic family.

*So we’ve just sort of got all of our systems in place so that took a while but now we’re sort of quite sussed.*

**Finding, storing, and preparing foods**
The responsibility of the systems is shared amongst all members of the family. For example, safe foods are identified and stored in a way that makes them easily accessible to the food allergic child or individual who is preparing a meal at their home. Foods are identified as safe - or not - through stickers, specific shelves in the
fridge or panty, and in some instances a separate pantry devoted to safe foods. In some instances the food allergic child helps the parent label the foods with the appropriate stickers or explains to a visitor how the food storage system works. Although the systems differed amongst families, the majority of them had a food storage system in place.

The red sticker is I’m not allowed it and the green sticker I’m allowed it and it’s so nannies don’t get mixed up.

The sense of security in the home described by the food allergic families, is also due to the ability to control not only the foods kept in the home but also preparation. Food is prepared in a systematic way to decrease probability of cross contamination, which can prevent a reaction. For example, separate cookware is used when preparing foods for a child with a severe food allergy.

When living with a food allergy, meal preparation as would be expected, can be quite challenging. Families often times commented that dairy and egg allergies were the most challenging
to manage. Milk and egg allergies are most commonly grown out of
and when this occurred families found that more foods became
available making life a lot easier. Having a food allergic child really
forces you to learn to cook and try new things and thus a positive
consequence of living with a food allergic child is that the majority
of families prepare meals at home.

Before I had [allergic child] the only recipes I ever used were
on the back of the Magi packet or a Continental packet.

In some instances cooking is a family affair as the allergic child
and/or sibling have grown a love for cooking, possibly out of
necessity. Families report that the use of allergy friendly
cookbooks, in addition to websites, enables them to prepare
delicious family friendly recipes. Many times safe foods are simply
substituted for allergenic foods, i.e. soymilk for milk. In one
instance a mum who runs a support group described a cooking class
that they offer. This seems like a great way to not only teach safe
cooking practices and provide tasty recipes, but also builds social
cohesion and a sense of community for families.
As previously mentioned, the home is a comfort zone for food allergic families. In part this is due to the ability to control the food products that enter the home. Over the years, due to the recognition of food allergies, there have been numerous changes within the food industry. An increase in the number of allergy tailored food products available have made life easier for food allergic families. Although there are more products available today as compared to a decade ago these new products are not always available or affordable. Families still find it challenging to locate substitute foods and it is time consuming to travel to a variety of shops, order specific foods online, and in some instances convince stores to keep certain food items or try to stock additional foods. To increase efficiency families have staple, go to substitute foods.

One family commented that they though it was getting harder to find foods which was contradictory to the other families comments. It may have been due to the fact that their staple food items were being discontinued, harder to find, or there was a change in the product. It is especially frustrating and disappointing for all members of the family when substitute foods are discontinued, as there is a lot of trial and error involved to find a tasty substitute foods.

*Because you know these things [substitute foods] become really important to you..*

As previously mentioned the affordability of substitute products can be out of reach for many. The cost of the products can be a barrier as they are more expensive then ‘normal’ foods.

*..so this cheese this soy cheese works out at $50 a kilogram, $50 a kilogram.*
On the opposite end of the spectrum, new products lead to feelings of joy and excitement. It was evident across the board that the mother typically completes the majority of the food shopping and therefore described the amount of time spent looking for safe foods and remaining optimistic for the development or stocking of new products.

*So finding new products is just like the best, the biggest buzz you can get as an allergy family I think.*

**Managing potential hazards leading to reactions**

Although the home is a safety zone for many, risks remain and for some families having allergenic foods in the home for others to enjoy is too much of a risk, this is somewhat dependent on severity of the allergy. Family’s opinions of allergenic foods in the home differ as some believe with the appropriate vigilance it is safe to have said foods in the home while others believe it is too risky. These opinions change over time as well; the older a child is in some instance the more risk a family takes on.

To prevent reactions, safe foods are purchased, and to ensure foods are safe reading labels becomes a part of life in a food allergic family and will remain as such until the allergy is outgrown (or not). This responsibility does not lie solely with the mother or parent; the whole family reads ingredient lists on a continuous basis as foods are always changing. At an early age food allergic children take on the added responsibility of reading labels, something other children most likely rarely consider. From early on they know what key terms to look for, based on the foods they are allergic to, and after many years of reading labels they become experts.

*I had to learn to actually look at the small print and see what’s in it.*
...[allergic child] who is also amazing at reading labels and knowing what her limitations are...

Label reading has also changed over the years, as recently there has been a shift by manufacturers to include the phrase, ‘may contain traces’ on the food label, or one similar to it (Muñoz-Furlong, 2003; Pitchforth et al., 2011). This causes frustration and in some instances families take the risk and eat the food anyways (Muñoz-Furlong, 2003; Pitchforth et al., 2011). This decision is somewhat dependent on the severity of the allergy. For families who view this as too risky, they may ring the manufacturer to better understand the production process to better understand the risk of cross contamination within their facility. For those who view the label warning as too risky they choose to not purchase the food if it contains this statement or something similar.

...the thing I most actually find most difficult or frustrating is actually the default seems to be by all manufacturers of foods to say may contain traces of nuts...
...it’s really annoying when there are traces because it’s like oh this is good, oh it’s not.

An unexpected finding whilst talking with the families was that in some instances the food allergic child’s tastes change as a result of the food allergy, specifically in regard to egg and milk allergy. For example, even after growing out of an egg allergy the food allergic child continues to not eat egg, as they don’t like the taste. An interesting item of note is that siblings often times don’t consume or like foods that the allergic child avoids. This may be a way of supporting their food allergic sibling or could be due to the fact that they aren’t exposed to the food and therefore do not develop a taste for it.

Negotiating social interactions
As previously mentioned the home is considered to be a safe zone, one that can be controlled. Therefore for many children social
interactions with friends happens more often in their own home as opposed to spending time at another’s house. As there is a strong tie between food and emotion, feelings of exclusion are first discussed here. A food allergic child may feel left out or different when they are unable to participate in the family ritual of consuming takeaways. To combat feelings of exclusion, kid friendly foods and “homemade takeaways”, are created to support allergic kids.

*It’s all homemade but there is so much, she doesn’t miss out really. We try really hard to make stuff safe for her all the time.*

When we step to the next environmental level of school we hear and learn more about these feelings of exclusion and how the challenges shift and are managed.

**Managing food allergy at school**

School is the first level outside of the safety of the home, the comfort zone. Food allergic families are still able to have some control of the food allergic child’s environment however there are now outside influences beyond their control. Children spend the majority of their time in school and therefore it is no surprise that all families described the impact a food allergy has on attending school and the barriers and facilitators within the school. Different experiences were expressed amongst the families ranging from their food allergic child attending a school with a very supportive environment to them attending a school with little or no support. Although unknown in the greater New Zealand context, food allergic families described a lack of consistency between schools and there support of food allergic children as some schools are nut-free, others used to be nut-free, and in some instances the policies in place are not enforced.
Finding, storing, and preparing foods
As to be expected the majority of children with a food allergy in this study reported that they pack their lunch at home to ensure that it contains safe foods. School canteens may provide allergy friendly foods however this was not discussed by any of the families. Although food is brought from home this does not guarantee that the child feels safe and supported, as they still have to remain vigilant of their surroundings. The level of comfort eating around others differed amongst the food allergic children in this study. One food allergic child described how they prefer to eat away from the other children.

..we normally sit away from everybody else at lunch time just because their food can flick around in places and get in my lunch box.

Managing potential hazards leading to reactions
As the school environment is not as controlled as the home environment there is the potential for additional reaction causing
hazards to be present. There are multiple opportunities throughout the day that the food allergic child may be exposed to food and there are management plans in place for the treatment of the reaction (i.e. accessibility of an epinephrine autoinjector), even more so then the prevention of a reaction. A food allergic child and their family suggested implementing a hand washing policy/procedure before and after morning tea, lunch etc. as a prevention strategy. The majority of families and food allergic children mentioned having an emergency kit at school that contains antihistamines, an auto injector, inhaler, and other child specific medicines. These emergency medical kits go everywhere with them and they also have a kit stored at school and in some instances medication in their classroom.

So say if somebody has an allergy they would put a box in there with an epipen, an inhaler, and some antihistamine I think [Mom: mhmm]. And they have it in there just in case anything happens and we need to take them with us on school trips if we go anywhere which sometimes can get a bit annoying with the big box.

One constant described by the majority of families was that yearly trainings were provided to school staff on how to use an auto injector.
Negotiating social interactions
As opposed to the home environment, social interactions were discussed more at the environmental level of school. For example, shared lunches are very popular amongst this age group and families indicated that these present challenges for food allergic children. These challenges are not only reaction based but also include a social component as they are unable to participate or are only able to eat the item they bring from home. Although not expressed by all families, for some this can lead to feelings of exclusion and isolation, as they feel different to their peers?

..like it’s really hard to have shared lunches at school because I’m not allowed some things..

Parents of food allergic children attending what they described as a supportive school described high levels of communication within the school and other families within the school community about food allergy. Not all of the families expressed similar experiences in within schools. For example, some schools provided all families with newsletters reminding parents to be mindful of food allergic children. Newsletters were not sent out at all of the schools and in many instances this notice was only included in the first newsletter of each school year. Families described the newsletter as one example of a way to facilitate communication about a food allergy to everyone within the school. Additionally, a food allergic child and their family described how the food allergic child educates their classmates about their food allergy. This was viewed as a great way to facilitate the discussion, empower the food allergic child, and educate their peers.

Managing food allergy in the local community
Finding, storing, and preparing foods
As we move out an additional environmental level to the wider community, vigilance among the families increases and additional
challenges have to be managed. In today’s society many meals are eaten outside of the home, often times in social settings. Outside of the home is defined as a café, takeaway, neighbourhood BBQ, and/or the home of a friend or extended family member. All of the families discussed some aspect of eating outside the home and the many challenges it presents. For some the challenges of finding a safe place to eat not only within Auckland but also throughout New Zealand and/or Australia seemed too great to overcome and therefore chose to stay close to home. McDonald’s was discussed repeatedly by the families as a preferred destination as many trust it and because they are located around the world. The consistency that they offer provides families a sense of security. In addition to McDonald’s, individual families identified trusted cafes and takeaways in their communities and in many instances food allergic children order the same items each time. Families described café workers as helpful, as in they tried to answer questions regarding potential allergens in food products or were cautious about cross contamination, but in some instances they did not know the ingredients of a specific items or were unaware of all potential opportunities for cross contamination. This seems like an appropriate area to promote training due to cafes being a high risk setting (Sicherer et al., 2001).
..they [café workers behind the counter] don’t have training in food allergies, it is really hard to take them on their word.

As previously mentioned, there is a strong social and emotional component of eating. Food is something to be shared and enjoyed amongst family and friends. When families are able to enjoy meals outside the home together or partake in family traditions it brings with it joy and a sense of belonging.

One of the things that’s been really cool is that if we are out somewhere we can now find somewhere that has it [coconut based ice cream] and she can have an ice cream at the same time as us.

Managing potential hazards leading to reactions
As not all meals consumed outside of the home are done so in a café or from a takeaway shop, eating at a friends and/or extended family member’s house was also discussed. Hazards can include cross contamination, contact with allergen (i.e. grocery store, in a park, at school etc.), and allergenic foods in the home. One of the biggest issues is that of the potential for cross contamination as one can never be sure of an individual’s food preparation practice.
..and if someone does cook for you, you have to quiz them on what they did.

Oftentimes social gatherings include food which can make it challenging for food allergic families to participate in birthday parties, block parties, or neighbourhood barbeques for example. The majority of families recognized the challenges and in some cases talked with the individual organizing the event beforehand to ensure a safe space while others have a select group that they prefer to socialize with. However in most cases the food allergic family provided their own food at the gatherings to reduce risk.

When eating outside of the home not only the food allergic child, but also the whole family, take responsibility for asking people what ingredients a food may contain when offered.

...if he ever got given any food he had to come and check with one of us as to whether um he could eat it so that we would check that and that we drilled it into [him] and we drilled it into his brother...

The level of risk attached to these hazards again varies between families and constant vigilance is again part of daily life. Having a few trusted go to homes and cafés provides a sense of security. Informing others is part of life for these families and the response they receive is a mixed bag. Some feel supported by friends, families, and neighbours while others are met with fear.

Although vigilance is a way of life for families reactions still happen and the majority of families described at least one event, or in some instance multiple events, that have occurred. A reaction may be due to accidental ingestion, accidental contact, and/or cross contamination.

One bite is enough to set it off.
For example, an unexpected area of concern is the grocery story, specifically the bulk bins. For a food allergic child with a severe allergy, these bins are hazardous not just because of what they contain, and the potential for cross contamination, but also because of the potential to come into contact with the allergen if spilled on the floor. In addition, the bulk bins tend to contain many kid type snacks, which again can lead to feelings that they are missing out. This theme continues to run throughout the discussions.

These reactions have led many to seek treatment from a healthcare provider, many times the emergency department, and at times have resulted in the use of their child’s auto injector.

She’s had an anaphylactic reaction from me kissing her on the cheek after eating peanut butter so she’s got quite a severe allergy..

As families are aware that reactions can happen at any time, strategies are in place in case of an emergency. The majority of families, in most instances the food allergic child, described their emergency medical kits and their contents which may include auto epinephrine auto injectors, antihistamines, inhalers, and action plans/instructions.

In addition some food allergic children wear medic alert bracelets and families are members of St. John’s Ambulance, which provides a lower cost ambulance trip. When the child is not accompanied by family out in the community the responsibility of their allergy shifts more fully onto themselves. Having a food allergy requires the food allergic child to take responsibility of their condition and in most cases they expressed that they are responsible for remembering to take their emergency kit with them when they leave the house. All of these items provide a sense of security.
I have to take it with me everywhere so I’m always carrying a bag, that can get annoying.

A few times I forget it but hardly ever.

Although cost rarely came up in the family discussions a few families mentioned the cost of auto injectors and the lack of government funding of these potentially lifesaving devices.

...epipens cost quite a bit of money to replace.

Negotiating social interactions
It was evident that the impact of food allergy on the food allergic child’s social wellbeing differed between children. However, the majority would agree that birthday parties are quite challenging and are ever present.

..in fact birthday parties are the worst.

A consistent message expressed included the importance of support networks for both the food allergic child and their parent(s). Some of the food allergic children described close friends who invite them over and their parents are comfortable feeding them, or they may take their own food with them. While others were not as fortunate and they may not be invited over as their friend’s parents find it hard to care for an allergic child. Again the discussion lent itself to feelings of exclusion and isolation, this is a constant in the life of a food allergic child.

Families describe that in some instances people feel uncomfortable when it comes to feeding other people’s allergic children.
...well they can’t feed her so that’s uncomfortable for people for a start but I have to train them in adrenaline and that’s scary so um yea so that’s definitely something that affects quality of life that I don’t have any photo of..

Parents understand that it may be uncomfortable for their child to be in another family’s home, it is a big responsibility for a parent to watch over any child let alone one with a food allergy. It’s finding the balance between explaining to the family limitations of what the child can eat along with how to use the items in the medical kit, with not overwhelming or frightening them.

I mean it’s a big responsibility taking someone else’s child who has a food allergy and having an epipen.

Managing food allergy beyond the local community
Many New Zealanders travel; it is a way of life and includes both domestic and international holidays. The fourth environmental level includes places outside the family’s local community. Although not as heavily discussed as the other three environmental levels, it is important to consider the challenges and management strategies at this level. Travelling with food allergies presents additional challenges, however they are similar to those at the other three levels. Families described destination limitations due to both the availability of safe foods and adequate health care.

Finding, storing, and preparing foods
The ability to find safe foods begins once the family leaves their house. For example, when travelling both domestically and internationally, food must be brought on planes, as airlines don’t often provide allergy free foods. Once abroad families must acquaint themselves with the foods that are available. In English speaking countries this is easier as they are able to communicate with café workers for example and read labels, this is why many
families in this study indicated that they choose to travel only to these countries.

But we love Australia so that’s good, that’s lucky.

Families also mentioned that the availability of chain restaurants worldwide, for example McDonald’s, allows for an easy option when travelling. One family translated documents that could be used to alert someone to an emergency in a non-English speaking country. They also mentioned the benefit of having an experienced tour guide that could point them in the right direction in regards to safe foods and the ingredients included in the local cuisine.

Managing potential hazards leading to reactions
Again cross contamination and exposure to an allergen are the biggest risks while travelling as the level of control a family has on this environment is minimal compared to the other three environmental levels. For example, one family indicated that when travelling domestically and staying at someone else’s vacation home all of the dishes may need to be washed to ensure traces of an allergen were not left behind. Many families with children who do
not have a food allergic child may take for granted the cleanliness of dishes for example. Whereas for a food allergic family this is a potential area for cross contamination and thus they must be concerned with the cleanliness.

As can be expected families differ on the risk they are willing to take with travelling and this may be dependent on the severity of the allergy and/or other chronic health conditions they may have. A holiday is meant to be a time for relaxing however some families voiced that vigilance is heightened during this time as the introduction of a new environment is challenging.

*Negotiating social interactions*
In terms of social interactions, this area was not discussed at this environmental level. Travelling is inherently social however at this age it is done as a family and therefore the lack of discussion around the impact on social interactions while beyond the family’s local community is not out of the ordinary. The impact would be that if a food allergic child were unable to travel beyond their local community this could influence the potential to interact with other cultures for example.

*Advice for Other Families and Public Awareness*
Toward the end of the interview each family was asked to reflect on (1) advice they would provide to other families and (2) what they wish the public knew about food allergies. Therefore these discussion points came out in the photos taken and through the additional questions that were asked.

When asked about what advice they would give to others the majority of families found this question tough as they have been managing a food allergy for a long time now. The advice was varied and included logistical items like the importance of setting up a safe environment at home, being diligent about hand washing,
cooking and trying new things, utilising the resources available on
the internet, and seeing a specialist. A few families stressed that
you can keep allergenic foods in the home safely. Additionally, they
provided emotional advice like remain vigilant, don’t make a big
deal about it, and get on with it.

Um…I don’t know I suppose I’d give people advice you know
um, don’t panic really, you can cope.

Be vigilant. When you are talking about one nut that can take
your daughter’s life away from her you know it’s not minor
stuff to deal with. Its anaphylaxis and you can easily die from
it.

…don’t make a big deal about it and you know there’s no
point in feeling guilty about it. You know it’s nothing we’ve
done to give them a nut allergy or whatever allergy, it is what
it is and you can’t change it so you just have to adapt your
life accordingly and actually you don’t really need to adapt it
that much.

As there has been an increase in food allergy worldwide the
condition has received more attention than in previous decades.
This attention has led to misinformation and in some instances a
lack of understanding in the public forum. The majority of families
would like the public to be more educated about what an allergy is
and how they can help prevent a reaction; focus on issues of cross
contamination, hand washing, and foods in public places. For
examples, if a peanut butter sandwich is consumed at a park and
then the child touches the swings this provides a potential hazard
for a child with a severe nut allergy.

I think um there’s a lot of people that sort of don’t get it don’t
get what allergies are and they don’t really know enough and
they form an opinion about it without sort of fully
understanding, so education people would be more helpful.
Um and yea there’s that whole education piece that people just make assumptions which cause they don’t know.

Additionally it is necessary to provide education to café workers, thus empowering them to provide accurate information in regard to potential allergens in the food which they are serving. General awareness of allergies in eating spaces would also be beneficial. Better public awareness may also help decrease the fear felt by some in terms of providing a safe environment for an allergic child.

And you can’t expect the world to change for one child but it would be nice if people were more aware.

Conclusion
Although frustrating at times to everyone within the family, a resounding comment made by most is that it’s just part of life, we’re used to it, and we just get on with things. The management strategies employed throughout their daily lives have lead the families to this point. As described above they have developed and implemented strategies related to: finding, storing and preparing food; preventing and managing reactions; and navigating social interactions to ensure safety. These management strategies in part have been developed by the families discussed here based on the impact food allergy has had on their daily lives.

It’s all doable. I mean it’s all liveable. It can be frustrating. It’s not too big a mountain to climb.

..cause I’ve been doing it all my life. It doesn’t really hinder us because you know we just go on get on with it.

They expressed that it’s important to be aware of it but not to dwell on the issue.
...you can’t change it you just have to get on with it and don’t make a big deal out of it.

...they can live a pretty normal life they just have to be aware of it...

Providing safe foods that make a child feel more inclusive and having safe treats can facilitate a sense of normalcy, they fit in, however feelings of exclusion do persist.

When living with any chronic condition, support can be the biggest facilitator or barrier to achieving a high quality of life and can include a variety of tools (Mandell et al., 2005; McBride et al., 2010). It was reiterated by all families that the biggest support they have is each other-family support. Immediate family members are there for one another and are looking out for the food allergic child.

So ya he does look out for her even though he pretends he doesn’t.

In some instances this does not reach beyond the immediate family to extended family, while others find their extended family equally supportive.

Outside of family parents found support through blogs, websites, online communities, physical support groups, books, and magazines. Surprisingly healthcare was rarely mentioned as a support. Allergy New Zealand was also mentioned as a place to find information. Although it may be hard to find, the support is out there. Families felt like this has likely become easier over time as there are more tools access.
...it is a picture of the computer with the allergy support network Facebook page which was not around when she [allergic child] was little but I so wish it was because it is so helpful.

Some allergic children described supportive friends and mentioned how they help them out and keep them safe. Additionally some families mentioned supportive family friends who make the allergic child feel included and provide a safe environment for them. The sentiment echoed by all members of the family was that food allergies don’t affect everything; kids still get to be kids. Specifically the food allergic children were quick to point out that they can still participate in their favourite activities like soccer, riding their scooter, and playing outside.
Yea you can do soccer even if you’ve got allergies and no one needs to know about it yea.

Discussion
This exploratory qualitative study is an important first step in adding to the very limited published literature investigating the impact a food allergy has on a food allergic family in New Zealand. It provides insights about the strategies used by food allergic families to cope, adapt, and feel fully supported. This research also contributes new knowledge about the experience of food allergy from the perspectives of all members of a family whereas other New Zealand based literature’s main focus was on the parent’s point of view (McBride et al., 2010). Beyond the New Zealand context the results presented confirm findings expressed worldwide.

Principal findings
It is evident that the impact a food allergy has on a family is influenced by their environment including: home, school, community, and beyond the community. Most notably, the amount of control a food allergic family has within these environments is seen to gradually decrease as they move away from the home
leading to variability in the degree of impact the food allergy has on the food allergic family.

Based on the impact a food allergy has on the food allergic families discussed above, in addition to the management strategies described, three outcomes of living with a food allergy are evident: responsibility, exclusion, and resilience. As described in the literature, a challenge faced by some food allergic children is that they bare more responsibility than the average child, they have to manage their food allergy on a daily basis (Miles et al., 2005; Muñoz-Furlong, 2003; Obeng & Vandergriff, 2008; Pitchforth et al., 2011). The families included in this study described that their food allergic children are responsible for managing their food allergy by: asking what is in a particular food they are given; remembering to take their emergency kit with them when they are away from home; label reading; knowing how to use their epinephrine autoinjector; and informing others of their allergy. Within the home, responsibility is shared amongst all members of the family. However outside of the home, when the food allergic child is on their own the responsibility shifts solely to themselves. Vigilance, including being aware of potential hazards in the environment, is a key piece of responsibility and unlike other studies, children did not express a ‘fear of food’ (Miles et al., 2005; Obeng & Vandergriff, 2008; Sicherer et al., 2003).

A second outcome of living with a food allergy described by food allergic families was the feeling of exclusion felt primarily by their food allergic child. In some instances food allergic children and/or their parent described feelings of exclusion due to the fact that the food allergic child is: unable to eat certain foods; not invited over to a friend’s house due to their allergy; or unable to participate in school based activities involving food. Although at times food
allergic children expressed feelings of exclusion through both photographs taken and as part of the interview, there was no mention of being bullied, teased, or harassed due to their food allergy as discussed in the literature (Lieberman et al., 2010). In some instances the parents were more concerned with exclusion than their food allergic children; for example the food allergic child was not aware that they hadn’t been invited over to a friend’s due to their food allergy. To manage this challenge across all environments parents are constantly working to help them feel as ‘normal’ as possible to prevent those feelings. This common concern expressed by parents, and echoed in the literature, led to parents cooking homemade takeaways, providing safe snacks for school, and preparing child-friendly meals (Muñoz-Furlong, 2003; Valentine & Knibb, 2011).

A third outcome of living with a food allergy was that of resilience, described by some as ‘getting on with things’. Although a food allergy is challenging and frustrating at times to everyone within the food allergic families in this study, they are resilient and treat it as part of everyday life and their attitude reflects the fact that they ‘get on with things’. As discussed above this attitude can be attributed, in part, to the development and success of daily management strategies.

The families in this study stressed the importance of not focusing on the food allergy and they remind their kids that their allergy doesn’t hold them back from achieving their goals. Based on the family’s discussion, they tend to spend time together shopping for allergen free foods, preparing meals, label reading, eating at home, remaining vigilant when eating outside of the home, and supporting one another. It was evident after talking with the families and analysing the data that they have developed strong family bonds. A
strong family bond has been described in the literature as a key determinant of quality of life for some families (Lieberman & Sicherer, 2011; Pitchforth et al., 2011; Valentine & Knibb, 2011). All members of the food allergic families in this study described the roles siblings play in regard to looking out for their food allergic brother or sister. Similar to examples in the literature: hand washing, label reading, and alerting their sibling of a potential hazard were all shared as examples of the responsibility taken on by the sibling (Mandell et al., 2005).

**Study limitations**
This study included a convenience sample in Auckland and therefore potential bias could have been introduced as underserved and underrepresented communities were not purposely recruited nor was recruitment held at a national level.

Coding of the results was carried out by one researcher with the input of a second. Cross checking of coding strategies, and interpretation of at least a portion of the data, by both researchers would have strengthened the methodology. However codes were discussed which added value to the refining of coding frames. A systematic process was followed and is described in the methods.

The study did include three similar methods of data collection – reflexive photography, photo elicitation, and the autodriven interview - to ensure the participant’s voice was heard. Although valuable, respondent validation was not utilised as participants were not involved with the project in an ongoing basis.

The use of visual methods resulted in high levels of engagement however lessons were learnt in regard to capturing photographs. Using a disposable camera did not allow for participants to review them prior to printing which resulted in pictures being of poor
quality. However the outcome of the study was not the photographs themselves as they were used as props to convey messages, to drive the interview.

Although rich data was collected, it is important to note that a food allergy may have an additional impact not described in this study due to the inability to capture it in a photograph. For example, families were asked to not take photographs of a person’s face, for confidentiality reasons, leading to some frustration as families expressed that they were unable to capture specific emotions or feelings. As described by one mother, if able to include faces they would have had their daughter stand in front of the bulk bins with a sad face. A second mother expressed frustration with how isolating a food allergy can be for her daughter, as she is not invited to other people’s houses very often. She wasn’t sure how to capture this in a photograph but felt it had a large impact on quality of life. She stated, “taking a photo of exclusion is hard”. Although photographs of this challenging nature were not included families were able, and encouraged, to share such comments and they were included in the results.

**Research implications**
There are two key implications arising from this research that could lead to a discussion nationally of strategies to better support food allergic families.

First, the need for the introduction of a national level school policy and procedure for food allergy management, in which accountability is included. Each of the seven participating families described a variety of policies and procedures, or lack thereof, within the school setting. If this was explored beyond these seven families it is most likely that we would see an even greater number of differences in schools. Therefore a national school policy, in which schools are
held accountable, regarding the treatment of a food allergy within the primary and secondary school system could improve the school environment. This finding supports previous work expressing the need for a consistent approach across all schools (McBride et al., 2010). A large responsibility of care rests on childcare services and school staff and in some instances a food-related allergic reaction can first present in these settings. Currently Allergy New Zealand and the New Zealand Ministry of Education have developed guidelines for early childhood centres (ECC) and schools (Allergy New Zealand, 2011; Ministry of Education, 2006). These guidelines provide information to help ECC and schools review, adapt, and/or develop key policies and procedures to support children and young people with health conditions to ensure the health and safety of children and students with allergies while in their care. In addition to policies, a family recommended utilising food allergic children to educate their peers on food allergies as this would not only make the food allergic child feel more comfortable but would also have the unintended consequence of a more educated and aware public, expanding to the community level.

Utilising the tools mentioned above, based on the guidelines published by Allergy New Zealand, a national school policy should include: (1) the requirement to obtain medical information about children at risk; (2) educating all those responsible for the care of children concerning the risk of food allergy; (3) implementation of practical strategies to avoid exposure to known triggers, for example hand washing; (4) a safe place for food allergic children to eat; and (5) age-appropriate food allergy education for all children. Once more accountability would need to be included to ensure consistency amongst schools.
Second, the need for an educated hospitality workforce. Thus food allergic family may feel more comfortable eating away from home. Currently the Ministry of Primary Industries provides information on the topics of: the seriousness of allergens; foods that trigger allergies; how to identify allergens in food; managing food allergens in your business; and how to prevent cross-contamination (Ministry for Primary Industry, 2013). In addition Allergy New Zealand has developed posters for the food service industry focused on general food allergy awareness and steps to take if a customer has a food allergy (Allergy New Zealand, 2010). Food companies can become a professional member of Allergy New Zealand which include multiple benefits including access to education seminars and programmes for health and education professionals.

In addition to above mentioned education opportunities, the ServSafe© program in the United States provides a food safety training program in which a section is devoted specifically to food allergies in addition to their ServSafe Allergens ™ Online Course (ServSafe, 2015). Requiring all hospitality workers to complete an education and training program would impact food allergic families as they would feel more comfortable eating away from home.

Opportunities for further research
As this study included a convenience sample in Auckland, it would be beneficial to repeat it in other areas of New Zealand, including underserved and underrepresented communities. Specifically investigating potential differences between the experience and impact of a food allergy in Maori and Pasifica communities, as well as lower socioeconomic status populations. Specifically the impact on families’ resilience, ability to accommodate their child, expense, and utilisation of health care. For example, within this study there was minimal discussion regarding the burden of cost of epinephrine autoinjectors. Understanding why that is and what impact this cost
may have on other families could be explored in future studies. In addition a larger sample size would allow for comparison between families, allergy severity, and time since diagnosis. Overall this study has provided a foundation for future research.

Chapter conclusions
This study provided a snap shot of seven families’ daily lives and the impact food allergies have. The results of this study will add to the growing body of food allergy literature, specifically in the New Zealand context. In addition the lessons learned from these families can be used to guide other food allergic families through education and advocacy. Further research, with a larger and more diverse sample size, is needed in this area. It is evident that the impact a food allergy has on a family is influenced by environment and includes four levels: home, school, community, and beyond the community. However it is unknown if this holds true for other populations in New Zealand.

The next chapter includes a discussion of all four studies and how together they provide a contemporary context about childhood food allergy in New Zealand and identify several key areas to consider in order to improve our understanding of this complex condition and its prevention and management.
Chapter 8: DISCUSSION

In this thesis I aimed to describe: (1) temporal trends in food allergy; (2) prevalence of peanut allergy and risk factors for peanut allergy; and (3) the impact of childhood food allergy on quality of life. Although highly debated, a dramatic increase in childhood food allergies has been observed worldwide yet to date the epidemiology of food allergy in New Zealand has been incompletely described and there is minimal published data that allows for any estimation of the disease burden caused by food allergy in New Zealand. Therefore this was an area where research was needed. To begin to better understand childhood food allergy in the New Zealand context four research projects, utilising different data sets, were undertaken.

Principal findings
First to determine whether Emergency Department (ED) presentations could be used to describe temporal trends in food allergy presentations I completed an audit within the public hospital ED in the Auckland District Health Board (ADHB) region for all ED presentations from 1988 to 2011 of children (0 to 14 years old), for which the ICD codes ‘anaphylaxis, unspecified’ or ‘allergy, unspecified’, were assigned. Food-related acute allergic reactions account for 29% of hospital presentations that were assigned one of these codes. Over the past 20+ years the ED presentation rate with food-related reactions has almost doubled. By contrast, ED presentation rates for non-food-related allergic reactions did not change over these years. This apparent increase in New Zealand is consistent with observations using comparable data sources reported from Australia and the United States. Based on these findings it was evident that when reporting on the hospital presentations due to acute allergic reactions related to food it would not be possible to identify a proportion of such presentations if we limited our analysis to hospital presentations identified solely by
food specific ICD-9-CM codes. The ADHB chart review showed us that a proportion of hospital presentations with acute allergic reactions related to food will have been coded as unspecified allergic or anaphylactic reactions.

I then applied lessons learnt from this initial project to the National Minimum Dataset (NMDS), a national collection of public and private hospital discharge information, temporal trends in emergency department (ED) presentations for food-related acute allergic reactions from 1988 to 2011 of children (0 to 14 years old) were investigated. Hospital presentations due to acute allergic reactions identified from pre-selected ICD-9-CMA-II codes have increased over the past two plus decades in New Zealand. In comparison to the 1988 time interval (annual rate 7.11/100,000), the rate of all presentations was five times higher in 2011 (37.88/100,000, RR=5.33). An average yearly increase of 8% in hospital presentations due to acute allergic reactions (p<0.0001) was observed. Additionally the severity of these reactions has increased at a faster rate, anaphylactic reactions average increase 11% per year (p<0.001). Hospital presentations serve as a proxy measure for prevalence of food allergy and these data provide no explanation for why such large recent temporal changes have occurred. Therefore it was important to investigate the prevalence of food allergy within a subpopulation, in this instance a longitudinal birth cohort study.

Third to better understand prevalence of food allergy, specifically peanut allergy, I utilised data from the Growing Up in New Zealand cohort study to determine both the prevalence of parental report peanut allergy and factors associated with the presence of peanut allergy at age two years. Based on parental reported data collected from Growing Up in New Zealand, a contemporary longitudinal birth
cohort study, 162 (2.6%) children were identified as peanut allergic. The odds of having parental reported peanut allergy at age two years were increased for boys, children diagnosed with eczema since 9 months, children whose mother had a history of atopic disease (eczema, hay fever, or food allergy), and mothers who identified as Asian. The odds of having parental reported peanut allergy at age two years were decreased for children who had never tried nuts or peanuts, or whose mothers had no secondary qualifications or secondary school/NCEA 1-4.

The final component of the research involved utilising visual methods to learn about the impact of a food allergy on the quality of life of seven New Zealand families. This included a description of strategies needed for a New Zealand family to cope, adapt, and feel fully supported when living with a food allergic child. The impact a food allergy has on a family is influenced by environment and includes four levels: home, school, community, and beyond the community. This is due, in part, to the amount of control a food allergic family has within these environments. The ability to control the environment gradually decreases as a food allergic family moves away from the home. Based on the impact a food allergy has on the food allergic families, three outcomes of living with a food allergy are evident: responsibility, exclusion, and resilience.

**Limitations**
When reporting the hospital presentations due to acute allergic reactions related to food it was not possible to determine the proportion of confirmed food-related acute allergic reactions from the National Minimum Dataset. However limiting the analysis to hospital presentations identified solely by food specific ICD-9-CM codes would have resulted in grossly underreporting the results. The large proportion of unspecified codes used is unique to the New Zealand context.
A limitation of the study utilising data from Growing Up in New Zealand is the use of parental self-report data as it can result in over reporting. However parental self-report is least variable amongst those with a peanut allergy as compared to other allergens for example egg and dairy. In addition, while a cohort study allows for consideration of inferences across the life course, it inevitably cannot collect complete detail on all potential exposures. My study has identified some important areas where there remains potential for the cohort to be more informative in regard to the additional data that can be collected, namely by biomarker measurement and access to the data contained in each child’s health care record.

The final study was carried out in a small convenience sample in Auckland and therefore potential bias could have been introduced as underserved and underrepresented communities were not purposely recruited nor was recruitment held at a national level.

**Research implications**
Consistent with what has been reported from several other countries the prevalence of childhood food allergy appears to have increased in recent decades. The rate of parental self-report of peanut allergy in New Zealand is similar to other countries. A food allergy impacts all members of a food allergic family based on the level of control within various environments.

Findings from this thesis should be considered at the national level as there is a need to better understand food allergy in the New Zealand context. The Growing Up in New Zealand study design allows the findings to be broadly generalizable to the New Zealand population as a whole. In addition, the study was specifically designed to have adequate explanatory power to allow for complex analyses within the main ethnic and socioeconomic subgroups of
New Zealand. My thesis provided an example of the importance of such subgroup specific statistical power with the clear need now for a detailed consideration of the determinants of food allergy within the Asian subset of the cohort.

Findings from this thesis could be utilised in an advocacy context providing support for the need to fund epinephrine autoinjectors and support for medical professionals in the management of this increasingly prevalent problem. In addition lessons learnt from the seven New Zealand families could provide other food allergic families with management strategies.

**Opportunities for further research**
The findings from this thesis provide a foundation for future research. As previously mentioned the inability to confirm food-related acute allergic reactions from the unspecified ICD-9 codes resulted in an over reporting of acute allergic reactions. Therefore future studies should extrapolate hospital presentations from the unspecified codes based on the ADHB chart review to determine more accurate annual rates of hospitalisations due to a food-related acute allergic reaction.

Food allergy most frequently presents and is diagnosed early in childhood. Thus there is a potential for the prevalence rate of peanut allergy within the *Growing Up in New Zealand* cohort to increase at 45 months, the following next data collection wave in which this issue was assessed. Therefore the findings from this study provide a foundation for future research and evidence for the need for additional investigation of: (1) parental report of peanut allergy versus confirmed peanut allergy; (2) potential change in rate of peanut allergy between data collection waves (i.e. 45 months); and (3) prevalence of other food allergies (i.e. egg and milk).
In terms of continuing the work of understanding the impact a food allergy has on New Zealand families, it would be beneficial to repeat the qualitative study in other areas of New Zealand, including underserved and underrepresented communities. Specifically investigating potential differences between the experience and impact of a food allergy in Maori and Pacifica communities, as well as lower socioeconomic status populations. Specifically the impact on families’ resilience, ability to accommodate their child, expense, and utilisation of health care.

In conclusion, food allergy is a complex condition and this thesis provides insight into the current state of food allergy in New Zealand. The chart review provided justification for including unspecified codes in the national study. In addition the first study demonstrated that the method could be used to identify hospital presentations and provided justification for moving forward with the second study. In the second study, in which hospital presentations were analysed, an increase over time was observed however hospital presentations are only the tip of the iceberg and these findings cannot be generalized to the full clinical spectrum of food allergic reactions. Consistency is evident between the data collected as part of the national hospital presentation study and the longitudinal birth cohort GUiNZ study. Food allergies are increasing worldwide and many families within New Zealand are managing them every day. The final study conducted provided a better understanding of their experience. Based on the data collected it is apparent that food allergy is a major issue and more public health interventions are needed.

Food allergy is a contemporary issue that needs to be addressed and done so in a way that takes into account the ethnic distribution
of the problem and not rely solely on Eurocentric solutions. Researchers continue to investigate the prevalence and incidence rates of food allergies worldwide; potential influences on the development and progression of food allergy; and the impact a food allergy has on those living with it on daily basis with the goal of working towards prevention and a cure.
Appendices
Appendix A: Ethics Approval Chart Review

(Chapter 3)
Appendix B: Ethics Approval Quality of Life
(Chapter 7)
What Image Describes How Food Allergy Affects Your Family?

Is there someone between the ages of 8-12 living in your household who has been diagnosed with a food allergy?

Are you a family who lives in the Auckland region?

If so, we want YOUR help!

We are conducting a food allergy research study to learn about quality of life issues faced by both the children diagnosed with a food allergy and their families.

What is involved in participating?

- Taking simple photographs that “tell stories” about living with a food allergy
- A one-hour introduction and training session
- A one hour family interview to talk about your families’ photographs
- Completing a short 15 minute questionnaire

How will you benefit?

- Your family will learn from others who are affected by food allergies
- Your family will receive one $75 gift card at the completion of the study

If you want to learn more about taking part in this study please call or email:

Colleen McMilin, PhD Candidate Centre for Longitudinal Research – He Ara ki Mua School of Population Health, University of Auckland
64 9 373 7599 ext. 88385   Email: c.mcmilin@auckland.ac.nz
## Camera and Meeting Tracking Form

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Name: ____________________________________

Phone number: _____________________________

Email: _____________________________________
Release of Photographs’ Copyright

I give my permission for Colleen McMilin (PhD Candidate) at the University of Auckland, Centre for Longitudinal Research (School of Population Health) to include ________ (number) of my photographs in their analyses and reporting of results of the Quality of Life project, including possible publication of the photographs in journals. I understand that I will not be identified by name as photographer or as a participant in the research.

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As part of the Quality of Life project I give my permission for Allergy New Zealand to include _______ (number) of my photographs for means of advocacy upon completion of the project provided by the researcher (Colleen McMilin). I understand that I will not be identified by name as photographer or as a participant in the research.

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Session One: Training Discussion Guide

Note: Sections written in Italic font are to be read to participants.

**Introduction and Informed Consent** (approximately 10 minutes)
As participants arrive, greet them and invite them to sit at the table. After an initial welcome, the researcher will briefly describe the purpose of the two sessions, emphasizing the following Points:

“We have invited you to take part in an exercise that includes taking photographs and discussing them in terms of what you think about a health issue. We invite you to share your personal views and experiences, as they will help us better understand this health issue.”

Next, we will obtain consent. Read the consent script. Provide the participants with a paper copy of the consent and ask if they have any questions. Once all questions have been answered, ask the participants to sign the consent forms. Then the researcher will collect the completed consent forms.

You will be taking photographs in the community and then discussing some of them within your family group. To protect the privacy of other people not part of this study, you the photographer must do one of two things:

1. Not include any identifiable person in a photograph, or
2. Get the signed written permission of every identifiable person in a photograph.

Any photographs you take will belong to you. We will ask you to sign a release for a copy of the photographs you select to discuss so that we may include copies of them in our analyses and reports of the study.

We will be recording the interview session. We don’t want to miss any of your comments. Only members of the research team will have access to the tapes. If anyone is uncomfortable with being recorded, please say so. You are free to leave if you would prefer. The recordings will be transcribed without any names or other identifying information, and will be kept in a locked cabinet. Once the tapes have been transcribed they will be destroyed. The typed versions will also be kept in a locked cabinet or a secure computer. In any reports of the findings, we will not use anyone’s name. We also ask that each of you keep what others say here today and throughout the interview session confidential. Also, please do not identify any of our participants outside of this group. What is said here should stay here.

**Disease specific HRQL questionnaire (HRQL – PB, HRQL – CF)**
After obtaining consent, give each participant (excluding siblings) the appropriate HRQL questionnaire to complete (have pencils and pens available).
Training

What to photograph
We are trying to learn what families in New Zealand need to cope, adapt and feel fully supported when living with a food allergic child. Don’t let this list restrict your thinking, but here are some ideas to get you ready to take photos:

- Things that make it easier or harder to cope with a food allergy
- People that help you live with a food allergy
- Messages related to living with a food allergy

Privacy and confidentiality
We encourage you to be creative and use your imagination. There is, however, one constraint. If you take a photograph that includes enough of a person to identify him or her, then you must get written permission from that person, which is a nuisance and can be difficult. Here are several ways to avoid that:

- Don’t take photographs of identifiable people
- “Cut off their heads” – for example, take photographs of someone’s hands holding something
- Take photographs of groups in which you cannot identify a singer person

How to use the camera
Demonstrate how to turn on and off, how to advance the film, how to center and focus, etc.

Returning the camera and developing the photographs
When you have finished taking photographs or at the end of two weeks, whichever comes first, please place the camera in the envelope provided to you and place in your local post box. Once the researcher has received the film it will be developed and you will be contacted to schedule a time for your interview. Photographs will be provided to you at the interview session which will be scheduled based on your needs.

Discussion session
We will meet again within a month’s time to return the developed photographs to you and discuss those you choose to talk about. At that time each child within your family who has been diagnosed with a food allergy will choose three photos and the other family members will choose three photos (six photos total) to talk about. The discussion will include why you took that photograph, what it means, how it relates to food allergy in general or in your family or community, and anything else you think is important.
Ownership of photographs and release for analysis and reporting

*The photographs you take belong to you. So that we can include those photographs that you select in this research study and possibly in published reports of the study, including a thesis, we will ask you to give us written permission to do so.*

Hand out cameras. Confirm method and timing of returning cameras. Then collect contact information from participants to track cameras, return and send (or call) reminders for interview session. Answer any questions.

Thank each participant.
Session Two: Interview Discussion Guide

**Ground Rules**
Before we begin our discussion, let me make a few requests of you. First, speak up so that everyone can hear you and let’s try to have just one person speak at a time. Please say exactly what you think. Don’t worry about what I think or others in your family might think. There are no right or wrong answers. Everyone’s ideas and experiences are important. Everyone does not have to agree; we are interested in hearing all opinions.

As we said during the training session, we will audio record the discussion. We don’t want to miss any of your comments. Only the researcher will have access to the recordings. If anyone is uncomfortable with being recorded, please say so. You are free to leave if you would prefer. The recordings will be transcribed without any names or other identifying information, and will be kept in a locked cabinet. Once the recordings have been transcribed they will be destroyed. The typed versions will also be kept in a locked cabinet or a secure computer. In any reports of the findings, we will not use anyone’s name. We also ask that each of you keep what others say in this group confidential. What is said here should stay here.

**Selecting Photographs**
Hand out the photographs to the family. The researcher will spend a few minutes with the family looking at the developed photographs and selecting a total of six of them for discussion. Selected photographs must 1) not contain an identifiable person, 2) be “croppable” to exclude an identifiable person, or 3) be accompanied by signed permission from the identifiable person.

**Discussion**
The researcher will then lead a discussion about the selected photographs, guided by the SHOWED technique. Make sure that everyone gets a chance to talk about her/his photographs.

You have chosen up to six photographs to talk about. We are interested in hearing the stories about your photographs, for example, why you chose these particular subjects; how you think they relate to food allergy; and resources that are needed. Now we’ll spend time talking more about these photographs, first giving each person a chance to talk about her/his photographs. It’s fine to ask questions and add your own comments to others’ stories. To help you describe your photographs, you may want to consider the following “SHOWED” questions:

- **S=See:** What do you **SEE** in the picture?
- **H=Happening:** What is **HAPPENING** in the picture?
- **O=Our:** How does this relate to **OUR** lives and how do we feel about them?
- **W=Why:** **WHY** does this problem/condition/strength exist?
- **E=Educate:** How can this image **EDUCATE** the community, policy makers, others?
• **Do**: What can we DO about these issues in our lives?

**Wrap-Up**

Before we end our discussion, I’d like to know if there is anything you would like to add. Are there things that we didn’t discuss that you think are important for us to know about living with a food allergy?

Thank you very much for taking the time to talk with us. Your input will be very helpful. Again, if you have any questions at any time about this project, please feel free to contact me (provide participants with researcher’s contact information).

**Release of photographs for analysis and reporting**

Ask each participant to sign the release statement giving the researcher permission to include their selected photographs in analysis and reporting of results.

**Distribute voucher**

Thank each participant.
**Family Demographics**
To be completed by all families interested in participating in the study based on inclusion/exclusion criteria.

1. Name ____________________________________________
   Address ____________________________________________
   ____________________________________________
   Phone Number_____________________________________
   Email ____________________________________________

2. Please complete the following for each person living in the household and who would be participating in the study (please include siblings if possible)

<table>
<thead>
<tr>
<th>Name</th>
<th>Gender</th>
<th>Age</th>
<th>Doctor Diagnosed Food Allergy (Yes/No)</th>
<th>Age Diagnosed</th>
<th>Foods Currently Allergic To</th>
</tr>
</thead>
<tbody>
<tr>
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</tbody>
</table>
3. For each child living in the household with a doctor diagnosed food allergy please answer the following:

<table>
<thead>
<tr>
<th>Reaction</th>
<th>Child's Name:</th>
<th>Child's Name:</th>
<th>Child's Name:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dizziness, feeling your heart beat fast, loss of vision, inability to stand, light headedness, collapse, and loss of consciousness/passing out?</td>
<td>Yes / No</td>
<td>Yes / No</td>
<td>Yes / No</td>
</tr>
<tr>
<td>Tightening throat, difficulty swallowing, hoarseness/hoarse voice, difficulty breathing in, shortness of breath, wheezing, cough?</td>
<td>Yes / No</td>
<td>Yes / No</td>
<td>Yes / No</td>
</tr>
<tr>
<td>Sick to your stomach, stomach cramps, vomiting, and diarrhoea?</td>
<td>Yes / No</td>
<td>Yes / No</td>
<td>Yes / No</td>
</tr>
<tr>
<td>Itchy skin, red rash, hives, worsening eczema, selling of the skin?</td>
<td>Yes / No</td>
<td>Yes / No</td>
<td>Yes / No</td>
</tr>
<tr>
<td>Other symptoms like oral allergy, swollen tongue or lips, symptoms of the nose or eyes?</td>
<td>Yes / No</td>
<td>Yes / No</td>
<td>Yes / No</td>
</tr>
</tbody>
</table>
Participant Information Sheet (PIS)

**Researcher/Study Contact:**
Colleen McMilin, MPH | PhD Candidate
Centre for Longitudinal Research - He Ara ki Mua | School of Population Health
Faculty of Medical and Health Sciences | The University of Auckland | New Zealand
Tel 64 9 373 7599 ext. 88385 | Email c.mcmilin@auckland.ac.nz

**Study Title:** What’s life got to do it with it? Exploring quality of life issues amongst New Zealand food allergic children and their families.

Your family is invited to participate in a research project about how a food allergy impacts your daily lives and in particular what strategies are needed for a New Zealand family to cope, adapt and feel fully supported when living with a food allergic child. A food allergy touches the life of not only the individual diagnosed but also their family, friends, health care providers, food producers and retailers, schools and government regulatory departments. As I am sure you have experienced, living with a food allergy affects the whole family through social interactions, emotional demands and resources. From working to get a diagnosis to providing a safe environment at home, navigating social interactions, the grocery store and school cafeteria, a food allergy is with a child and in many cases their family 24-hours a day, 365 days a year and for some this is lifelong. It is important that we understand the affect food allergy has on quality of life, as there may be supports that can be utilised to improve it. We strive to describe quality of life issues faced by both the child in your family who has been diagnosed with a food allergy and others members of your family. We will be asking you to describe for us what strategies are needed for your family to cope, adapt and feel fully supported while raising a child with a food allergy.

This study is being undertaken by Colleen McMilin; a PhD Candidate at the University of Auckland’s School of Population Health, supervised by Dr Cameron Grant (Associate Professor in Paediatrics, University of Auckland) and advised by Dr Laura Wilkinson-Meyers (Lecturer in Health Services Research, University of Auckland). This project is a piece of a larger thesis that is working to fill gaps in the knowledge of childhood food allergy in New Zealand and ultimately guide strategies for: (1) prevention of food allergy in children in New Zealand and globally; (2) prevention of food induced reactions; and (3) diagnosis and management of childhood food allergy.

**Participant selection and role:** You have been invited to take part in the research through established communication pathways within Allergy New Zealand which may have included the Allergy New Zealand website, newsletter mailings, Twitter and Facebook platforms. We would like to recruit seven to ten families. A family must include at least one child (8-12 years of age) living in their home with a doctor diagnosed food allergy and a caregiver; it would be great if a family consisted of additional members as we are interested in hearing from multiple caregivers and siblings. We are interested in sampling a diverse population and therefore have the following inclusion criteria: date of birth (child diagnosed with a food allergy), food(s)
allergic to, ethnicity and gender. With these criteria it is hoped that the sample will include food allergic children aged 8-12 years of age, with varied food allergens, include multiple ethnic groups and include both boys and girls. We are asking each family to complete a brief demographics form to determine eligibility. After the demographics questionnaire has been received by the researcher, it will then be reviewed based on inclusion criteria. If the participants meet the inclusion criteria they will then be sent, through the post, a packet of information including the consent form and outline for the project. Families selected to participate will be asked to (1) attend a one-hour introduction and training session, (2) take simple photographs that “tell stories” about living with a food allergy, (3) participate in a one hour family interview to talk about your families’ photographs and (4) complete a short 15 minute questionnaire.

The introduction and training session will take place at the main office of Allergy New Zealand. The family interview can take place at a location of your choice, which can include Allergy New Zealand. Sites will be agreed upon with participants to reduce potential transportation issues. Participants will be asked to take pictures of any themes and topics that evolve from the discussion and that relate to food allergy quality of life issues (e.g. social isolation, fear of food, high grocery bill etc.). The participants will then have two weeks to take pictures. Participants will be required to return the cameras to study personnel for development. Each family will be provided with a self-addressed stamped envelope to return the camera to the researcher. The researcher will follow-up with the family weekly. Once the camera has been returned a follow-up appointment will be made with the family to discuss the photos. If you agree to take part in this study, we will ask you to do the following things:

- We will give you and six to nine other families’ simple cameras and training concerning how to use the cameras and how to protect the privacy of any person who might appear in a photograph. This session will take about an hour.
- We will tell you how to return the cameras so that we can develop the photos. We will make two copies and one electronic copy of the photographs.
- We will ask you to attend a second, family interview session during which your family will select up to 7 of the photos and describe each photo, explain why you chose that subject, and discuss what the photo means to you related to food allergy. This session will take about one hour. Your comments are important to us. We will audio record our conversation.
- We will ask you to sign a release form for the 7 photos you select and your family select so that we may include them in research analysis and subsequent publications. All other copies of the photographs will belong to you.
- In addition, before we begin our talk, we will ask you to complete a HRQL (health related quality of life) questionnaire. We have a questionnaire that has been designed for caregiver(s) and one for children who have been diagnosed with a food allergy.
- You will not put your name on the questionnaire.
- We will only use the questionnaire information to describe characteristics of the group as a whole.

- We will collect contact information from you only in order to keep track of the cameras and follow-up discussion groups. That information will be kept separate from other study materials and will be destroyed as soon as we complete the discussion groups. No personal identifying information will be included in any analysis or report.

**Your choice and your rights:** Taking part in this study is voluntary. Whether you take part in our study is up to you. You have the right to choose not to take part in this study. If you choose to take part, you can leave the group at any time. You can decide not to answer any question. All family interviews will be audio-recorded and then transcribed. You will not be provided with a copy of your interview transcript or recording, but if you would like to remove any part of your response, you can contact the researcher within 2 weeks of the interview to withdraw a piece, or the whole interview from the study.

**Protecting your anonymity and confidentiality:** Data collected from participants’ will be de-identified for protection. Each participant will be assigned an identifying number for use in the analysis comparing data from other participants and identifying recurring themes in the data to answer research questions, the same holds true for the photographs. De-identified quotations will be used in reports or publications of this study.

During group discussions others might hear what you say, and it is possible that they could tell someone else. Because we will be talking in a group setting during session one, we cannot guarantee that what a participant discusses will remain private. But we expect that all group members will respect the privacy of everyone in the group. Family interviews will take place on a one-on-one format (one researcher talking with one family) and therefore should limit the nervousness of revealing sensitive information.

You will not be identified by name on any digital recording or written notes, and only the researcher will be able to hear any audio recording or view any written notes. You will not be identified by name on the questionnaire form.

Data will be stored securely in a locked cabinet at the University of Auckland Centre for Longitudinal Research, by the researcher for six years. Results of the research activity will be disseminated within the study community, at meetings and conferences, will be published in scholarly journals and included in a PhD thesis. Photographs that include identifiable images of other people will not be used in this public manner unless we receive consent from the photograph’s subject(s) to use the image publicly. You will not be identified in any way. The only people with access to
this material will be the researchers named below. All material, including audio-
recordings, will be destroyed after six years.

**Benefits of participation for your family and sharing study findings:**
You will have the opportunity to voice your experience regarding the impact food allergy has on your daily life. Potential benefits to participation include a greater awareness within their family, and possibly their community at large, regarding how a food allergy affects their quality of life on a daily basis. Others may benefit through your participation as findings may lead to a greater knowledge of the physical, emotional and psychological effect a food allergy has on both the child and their family. This knowledge may contribute to a better understanding of the resources needed by families and may result in more support within communities including better public awareness, support in schools and support from food producers and retailers for example. At the completion of the family interview (session two) your family will receive a $75 voucher in appreciation of your time and contribution to the research. All families will be referred to their family general practitioner and Allergy New Zealand for further resources as a provision should there be adverse consequences or physical or psychological risks. It may be possible that the research could give rise to incidental findings. For example, during the family interview a food allergic child may share that he/she has been bullied at school due to their food allergy. If this, or any other psychological, emotional or physical risk is identified the family will be referred to their family general practitioner and Allergy New Zealand for further resources. Results of the research activity will be disseminated within the study community, at meetings and conferences, will be published in scholarly journals and included in a PhD thesis.

**Who to contact if you have concerns about participating:** If you have any queries or concerns regarding your participation, you can contact the researchers listed below. For any queries regarding ethical concerns you may contact the Chair, University of Auckland Human Ethics Committee, The University of Auckland, Office of the Vice Chancellor, Private Bag 92019, Auckland 1142, telephone 373 7599 extn 83711. Please contact Colleen McMilin or Dr Laura Wilkinson-Meyers if you have any questions about this study.

**Researcher/Study Contact:**
Colleen McMilin, MPH | PhD Candidate
Centre for Longitudinal Research - He Ara ki Mua | School of Population Health
Faculty of Medical and Health Sciences | The University of Auckland | New Zealand
Tel 64 9 373 7599 ext. 88385 | Email c.mcmilin@auckland.ac.nz

**Supervisor Contact:**
Dr. Cameron Grant FRACP PhD | Associate Professor in Paediatrics
The University of Auckland | Starship Children’s Health
Park Road | Auckland | New Zealand
Tel 09 373 7599 ext. 86192 | Email cc.grant@auckland.ac.nz
Advisor Contact:
Laura Wilkinson - Meyers, PhD | Lecturer in Health Services Research
Health Systems Section | School of Population Health
Faculty of Medical and Health Sciences | The University of Auckland | New Zealand
Tel 64 9 373 7599 ext. 89143 | Email l.wilkinson-meyers@auckland.ac.nz

Head of Department:
Susan Morton BSc(Hons) MBChB PhD FAFPHM FNZCPHM | Associate Professor | Research
Director, Growing Up in New Zealand | Director, Centre for Longitudinal Research - He Ara ki Mua
School of Population Health | Faculty of Medical and Health Sciences | The University of Auckland | New Zealand
Tel 64 9 373 7599 ext. 89268 | Email s.morton@auckland.ac.nz

For any concerns regarding ethical issues you may contact:
Chair | University of Auckland Human Participants Ethics Committee
The University of Auckland | Research Office
Private Bag 92019 | Auckland | 1142 | New Zealand
Tel 64 9 373 7599 ext. 87830/83761 | Email humanethics@auckland.ac.nz
Child Information Sheet (PIS)

Study Title: What’s life got to do with it? Exploring quality of life issues amongst New Zealand food allergic children and their families

My name is Colleen McMilin and I am a student researcher at the University of Auckland. I want to know what it is like to have a food allergy. I am doing a study to learn more about what it is like to be you. Some kids with a food allergy can’t eat a lot of foods and some can eat almost anything they want. Some kids get really sick when they eat certain foods and some do not. Some kids feel that it isn’t fair that they are different and can’t eat the same foods as their friends. I want to learn more about this.

You are invited to participate with your family in this study. If you choose to participate, these are the things that will happen:

- You and your family will attend a talk with other families to learn more about the study. This will last up to one hour.
- I will ask you to answer questions on paper about how having a food allergy makes you feel. I will not ask you to put your name on this paper and I will not know which answers are yours. This will last up to 15 minutes.
- You and your family will be given a camera and you will be asked to take pictures of what it is like to be you living with a food allergy.
- I will then print the pictures and meet up with you and your family to talk about the pictures you took. I will ask you to select three of them to talk to me about. This will last up to one hour.

**Important Information**

1. Before you start, your parent/caregiver has to say that it is ok for you to take part in my project. You will never be talking to me alone; your family will be part of the discussion.

2. You don’t have to talk with me or take pictures. If you choose to help me it is your choice.

3. While we are talking if you decide that you don’t want to participate that’s ok, you just need to let me know.

4. I will record what everyone says during the talk with your family. After this you can ask me to delete something you’ve said if you don’t want me to keep it. I will try my best to do this.

5. I am not allowed to use your real name when I’m writing or talking about your work later on.
Thank you for choosing to share your stories with me.

If you or your parents/caregivers have any other questions or want to know more about my project, you can contact me at any time:

*Researcher/Study Contact:*
Colleen McMilin, MPH | PhD Candidate
Centre for Longitudinal Research - He Ara ki Mua | School of Population Health
Faculty of Medical and Health Sciences | The University of Auckland | New Zealand
Tel 64 9 373 7599 ext. 88385 | Email c.mcmilin@auckland.ac.nz

*Advisor Contact:*
Laura Wilkinson - Meyers, PhD | Lecturer in Health Services Research
Health Systems Section | School of Population Health
Faculty of Medical and Health Sciences | The University of Auckland | New Zealand
Tel 64 9 373 7599 ext. 89143 | Email l.wilkinson-meyers@auckland.ac.nz
Child Information Sheet (PIS) – Sibling(s)

Study Title: What’s life got to do with it? Exploring quality of life issues amongst New Zealand food allergic children and their families

My name is Colleen McMilin and I am a student researcher at the University of Auckland. I want to know what it is like to have a brother or sister with a food allergy. I am doing a study to learn more about what it is like to be you. Some kids with a food allergy can’t eat a lot of foods and some can eat almost anything they want. Some kids get really sick when they eat certain foods and some do not. Some kids feel that it isn’t fair that they are different and can’t eat the same foods as their friends. I want to learn more about how this affects you.

You are invited to participate with your family in this study. If you choose to participate, these are the things that will happen:

- You and your family will attend a talk with other families to learn more about the study. This will last up to one hour.
- You and your family will be given a camera and you will be asked to take pictures of what it is like to be you living with a brother or sister with a food allergy.
- I will then print the pictures and meet up with you and your family to talk about the pictures you took. I will ask you to select two of them to talk to me about. This will last up to one hour.

**Important Information**

6. Before you start, your parent/caregiver has to say that it is ok for you to take part in my project. You will never be talking to me alone; your family will be part of the discussion.

7. You don’t have to talk with me or take pictures. If you choose to help me it is your choice.

8. While we are talking if you decide that you don’t want to participate that’s ok, you just need to let me know.

9. I will record what everyone says during the talk with your family. After this you can ask me to delete something you’ve said if you don’t want me to keep it. I will try my best to do this.

10. I am not allowed to use your real name when I’m writing or talking about your work later on.
Thank you for choosing to share your stories with me.

If you or your parents/caregivers have any other questions or want to know more about my project, you can contact me at any time:

Researcher/Study Contact:
Colleen McMilin, MPH | PhD Candidate
Centre for Longitudinal Research - He Ara ki Mua | School of Population Health
Faculty of Medical and Health Sciences | The University of Auckland | New Zealand
Tel 64 9 373 7599 ext. 88385 | Email c.mcmilin@auckland.ac.nz

Advisor Contact:
Laura Wilkinson - Meyers, PhD | Lecturer in Health Services Research
Health Systems Section | School of Population Health
Faculty of Medical and Health Sciences | The University of Auckland | New Zealand
Tel 64 9 373 7599 ext. 89143 | Email l.wilkinson-meyers@auckland.ac.nz
Study Title: What’s life got to do with it? Exploring quality of life issues amongst New Zealand food allergic children and their families.

Consent: This form will be held for six years

Project Description: You are being asked to take part in a research study to learn what affect food allergy has on quality of life as there may be supports that can be utilised to improve it. We strive to describe quality of life issues faced by both the child in your family who has been diagnosed with a food allergy and others members of your family. We will be asking you to describe for us what strategies are needed for your family to cope, adapt and feel fully supported while raising a child with a food allergy.

You are being asked to be in this study this project is a piece of a larger thesis that is working to fill gaps in the knowledge of childhood food allergy in New Zealand and ultimately guide strategies for: (1) prevention of food allergy in children in New Zealand and globally; (2) prevention of food induced reactions; and (3) diagnosis and management of childhood food allergy.

1. I have read the Participant Information Sheet. I have understood the nature of the research and why my family and I have been selected as a participant. I have had the opportunity to ask questions about the study and my participation and I am satisfied with the answers provided.

2. I understand that taking part is my choice and that I can withdraw from the study at any time up until the 1st of May, 2014 without giving a reason.

3. I agree to my family interview being recorded with a digital voice recorder.

4. I understand that even if I agree to being recorded, I do not have to answer all questions, and I may choose to have the recorder turned off at any time.

5. I understand that I will not receive a copy of my recording but I will have two weeks from the completion of my interview to contact the researcher and request to erase any sections.

6. I understand that I am agreeing to: (1) attending a one-hour introduction and training session, (2) taking simple photographs that “tell stories” about living with a food allergy, (3) participating in a one hour family interview to talk about your families’ photographs and (4) completing a short 15 minute questionnaire. Specifically I will:

   o Be provided with a simple camera and training concerning how to use the camera and how to protect the privacy of any person who might appear in a photograph. This session will take about an hour.

   o Return the cameras so that the file can be developed. I understand that two copies and a digital copy of each photograph will be created.
o Attend a second, family interview session during which my family and I will select up to 7 of the photos and describe each photo. I will explain why I chose that subject, and discuss what the photo means to me related to food allergy. I understand that this session will take about one hour. I understand that the audio from this session will be recorded.

o Sign a release form for the second copy of the 7 photos that I select so that the researcher may include them in research analysis and subsequent publications. All other copies of the photographs will be provided to me.

o Complete the appropriate HRQL (health related quality of life) questionnaire. I understand that my name will not be on the questionnaire and that the information will be used to describe characteristics of the group as a whole. No one will be able to identify my answers.

7. I understand that my contact information will be collected in order to keep track of the camera provided to my family and allow researchers to contact me regarding a follow-up session (family interview). I understand that the contact information will be kept separate from other study materials and will be destroyed as soon as we complete the discussion groups.

8. I understand that all study material will be stored on password protected computer databases, or in locked cabinets at the University of Auckland. Consent forms will be stored separately from other material. The only people with access to this material will be the research team. All material, including audio recordings, will be destroyed after six years.

9. I consent to information provided by me appearing in reports of this study. I understand that to protect my identity, all data that I provide will be de-identified and given a unique code. I understand that de-identified quotations will be used in reports of this study. I understand that no personal identifying information will be included in any analysis or report.

10. I understand that the data provided will not be used for any other purpose or shared with others without my written consent.

11. I understand the study and its possible risks and benefits.

12. I understand that participating in this study is voluntary and that I can stop at any time or decide not to answer a question.

13. I agree to respect the privacy of everyone in the group.

14. I wish / do not wish to receive the summary of findings.
15. I agree to participate in this study.

16. I agree that my
child(ren)______________________________________________________
can participate in this study.

Name______________________________________________ [Please print]
Signature ___________________________________________
Date____________________

Email address (if you would like to receive a summary of the findings)
___________________________________________________________________

Thank you for choosing to participate in this worthwhile project. Your insight is
greatly appreciated!

Researcher/Study Contact:
Colleen McMilin, MPH | PhD Candidate
Centre for Longitudinal Research - He Ara ki Mua | School of Population Health
Faculty of Medical and Health Sciences | The University of Auckland | New Zealand
Tel 64 9 373 7599 ext. 88385 | Email c.mcmilin@auckland.ac.nz

Advisor Contact:
Laura Wilkinson - Meyers, PhD | Lecturer in Health Services Research
Health Systems Section | School of Population Health
Faculty of Medical and Health Sciences | The University of Auckland | New Zealand
Tel 64 9 373 7599 ext. 89143 | Email l.wilkinson-meyers@auckland.ac.nz
Consent to use a photograph containing an image of you

We are conducting a research study to learn what affect food allergy has on quality of life as there may be supports that can be utilised to improve it. We also want to understand quality of life issues faced by both the child in your family who has been diagnosed with a food allergy and others members of your family. We are asking families to describe for us what strategies are needed for to help them cope, adapt and feel fully supported while raising a child with a food allergy.

As part of the project, we have asked families to take photographs that “tell stories” about living with a food allergy. We asked these families in general not to take photographs of people who could be identified in the photographs. You have been included in one photograph, however, and the photographer believes that photograph is important.

We would like your permission to use the photograph for further study, exhibition and publication. We believe this picture will communicate a strong message about the needs of families living with a food allergic child in communities to policy makers and others who are trying to improve the lives of those touched by a food allergy. Your name and any other identifying information will NOT be shared. If you do not give your permission, we will not use the picture publicly.

If you have any questions or concerns about this study, or the use of your photograph, please contact the researcher listed above. This study was approved by a committee that works to protect human rights and welfare.

If you agree, please tick the box and both print and sign your name:

[ ] I give permission for the researcher, Colleen McMilin (PhD candidate, University of Auckland) to use this photograph, which contains an identifiable image of me, for further study, exhibition or publication.

____________________________
Printed Name of Photograph Subject

_______________________    ___________________
Signature of Photograph Subject     Date
References


Allergy New Zealand. (2011). *Allergy and anaphylaxis guidelines for early childhood services and schools.*


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Ministry for Primary Industry. (2013). *Allergens can kill - lives are in your hands.*


