# The New Zealand Coeliac Health Survey

Kiri Patrice Hamilton Sharp

Student ID Number: 438095

Thesis submitted in partial fulfilment of the partial requirements of the Master of Dietetics program

The University of Otago

January 2013

**ABSTRACT**

Coeliac disease (CD) is a multi-system autoimmune enteropathy caused by an inappropriate immune response to certain storage plant proteins known as gluten. Gluten is found in wheat, barley, rye and triticale. The autoimmune response causes damage to the gut resulting in gastrointestinal and non-gastrointestinal symptoms. Untreated CD leads to malabsorption resulting in nutritional deficiencies and long term damage including an increased risk of intestinal lymphomas. The only treatment is removal of gluten from the diet, which in most cases, allows a full mucosal recovery and return to full functionality. In New Zealand the prevalence of CD is about 1%. However, there is little information on the health of people with CD in New Zealand. This study aims to fill that gap.

**Methods:** The eligible study population was members of Coeliac New Zealand Incorporated who had an email or postal address. 2,200 members were invited by email and 500 members by postal letter to complete a questionnaire online or a paper copy. The questionnaire was adapted from the Canadian Celiac Health Survey and consisted of 10 sections investigating demographics, health, diagnosis, diet, quality of life issues, associated medical conditions, CD and associated medical conditions among relatives and recommendations. Data from completed postal surveys were entered, and all results collated in Excel. The results were analysed using STATA. Proportions and means were calculated as appropriate.

**Results:** The response rate was 46.5%. Responses from 936 biopsy positive adults aged 16 years and over were analysed. 94% were New Zealanders of European descent. The ratio of males to females was 1:4.7. The mean age was 57 years for males and 53 years for females.

The mean age at diagnosis of CD was 43.5 years and the mean time between the reported onset of symptoms and a diagnosis of CD was 11.6 (SD -3.6 -26.8) years. At the time of diagnosis the most common symptoms were bloating, gas and abdominal pain (76%), extreme weakness or tiredness (67%), diarrhoea (64%) and anaemia (57%)., When diagnosed with CD, 82% had been referred to a nutritionist or dietitian. 96% found their health improved to some extent after commencing a gluten free diet (GFD). A large number of respondents continued to have ongoing symptoms despite 92% always following a GFD. The most common, and highly rated source of information about CD and its treatment was Coeliac New Zealand Incorporated. Three-quarters had received information from dietitians/nutritionist, but 19% considered the information to be fair or poor quality.

The two factors which participants believed would contribute the most to improving the lives of individuals with CD were earlier diagnosis of CD (55%) and better labelling of gluten-containing ingredients in foods.

**Conclusions:** CD is a common condition in New Zealand, but the disease is often misdiagnosed with the diagnosis being delayed for up to 12 years. Despite best efforts, accidental ingestion of gluten appears to be a problem with most people having ongoing symptoms to varying degrees. This may relate to inadequate dietary education, contaminated food products, or co-existing conditions. Given the significant delays in the diagnosis of CD, and the fact that a GFD is the only available treatment for CD, there needs

to be greater awareness among health professionals of CD symptoms and its atypical presentation, improved knowledge about CD and its dietary treatment among dietitians, and improved knowledge among those preparing and manufacturing gluten free foods.

## PREFACE

The author of this thesis, Kiri Sharp, was the primary researcher for this project. She conducted the research - wrote the ethics application, adapted a validated survey for a New Zealand audience, consulted with members of the Palmerston North coeliac support group, liaised with Coeliac New Zealand Incorporated to invite their members via email and post to participate in the survey, uploaded the survey online, entered the data from the paper surveys, contributed to the data analysis, interpreted the data, and wrote this thesis.

Dr. Kirsten Coppell, supervisor of this project developed the study idea, sought permission to use the Canadian Celiac Survey, consulted with members of the Dunedin coeliac support group, and oversaw all aspects of the study.

Heather Walker conducted all the statistical analyses.

## ACKNOWLEDGEMENTS

I would l like to acknowledge my supervisor Dr. Kirsten Coppell, Heather Walker the biostatistician for this project and Kent Sharp who helped with the data entry.

I would also like to acknowledge Coeliac New Zealand Incorporated, in particular Sue Clay and Melissa Rouse, for sending the questionnaire to all their membership. Their assistance in this study was enormously appreciated. The assistance and support from members of both the Coeliac New Zealand Incorporated Palmerston North and Dunedin support groups is also acknowledged.

I extend thanks to all the people who took the time to complete the survey.

I also thank the University of Otago Human Nutrition Department who provided some funding for project expenses, and Winsome Parnell for facilitating this Masters of Dietetics programme.

## TABLE OF CONTENTS

## LIST OF TABLES

## LIST OF FIGURES

## LIST OF ABBREVIATIONS

CD - Coeliac Disease

DH – Dermatitis Herpetiformis

EMA - anti-endomysium antibody

GF - Gluten-Free

GFD - Gluten-Free Diet

NZ - New Zealand

PHARMAC - Pharmaceutical Management Agency

TTG – Tissue Transglutaminase

EmA – Antiendomysium

TG2A – Antitransglutaminase type 2

## 1.0 INTRODUCTION

Coeliac disease (CD) is a multi-system autoimmune enteropathy caused by an inappropriate T-cell mediated immune response to ingestion of certain storage plant proteins. Collectively these are known as gluten. Only the prolamin fraction of the storage protein is toxic. The prolamins include gliadins and glutenins in wheat, horedein in barley and secalin in rye (1). Gliadins contain monomeric proteins and glutenins contain aggregated proteins (2). Gluten causes an immunologically mediated inflammation, villous atrophy and crypt hyperplasia in the proximal small intestinal mucosa. This damage may cause gastrointestinal symptoms and can result in malabsorption of macro- and micronutrients causing deficiencies in iron, folate, calcium, vitamin B12 and vitamin D. CD can also affect the nervous, reproductive and skeletal systems. CD occurs in genetically susceptible individuals, being strongly associated with the HLA allelles HLA-DQ2 and HLA-DQ8. CD has previously been known as coeliac sprue, nontropical sprue, gluten-sensitive enteropathy and idiopathic steatorrhea (3).The only treatment for this disease is to permanently remove the allergen by adhering to a strict gluten free diet. Elimination of gluten will, in most cases, allow a full mucosal recovery and return to full functionality.

CD appears to be increasing in prevalence around the world and in New Zealand. Little is known about the health and treatment of CD patients in New Zealand. For this reason the New Zealand Coeliac Health Survey will provide new information about how CD is managed in New Zealand and how it affects the lives of those who have it.

The main aim of this thesis is to describe the health of people with CD in New Zealand, and any problems associated with the only treatment, a gluten-free diet (GFD). The background section describes the prevalence of CD worldwide and in New Zealand and possible reasons for the increase in prevalence of this disease are discussed. The risk factors, clinical presentation in adults and children, complications, diagnosis and testing, and the management of CD are also described. The mainstay of treatment for CD, a GFD and how it is ideally adopted and managed with the expertise of a dietitian is described. The preparation of gluten free (GF) foods, including how the gluten levels of food are ascertained, the levels of gluten permissible, and how and when gluten contamination of GF foods occurs. The methods section describes how the New Zealand Coeliac Health Survey was undertaken. The results are presented, and these are explored and interpreted in the discussion section. This is followed by conclusions and recommendations.

# 2.0 LITERATURE REVIEW

## 2.1 Epidemiology of Coeliac Disease

### (i) Prevalence

The prevalence of CD is difficult to establish accurately as the gold standard diagnostic test for CD is a small intestine biopsy. Despite this limitation, overall the prevalence of CD appears to be increasing worldwide, and it is three times more common in women than men.

### Worldwide Prevalence of CD

The prevalence of CD varies worldwide. CD has a strong genetic component, and is found most commonly in European populations (4). Figure 1 highlights the variation in the prevalence of CD between countries in the Asia Pacific region, with countries with predominantly European populations having a higher prevalence. Overall the prevalence of CD in adult populations is about 1%, but it ranges from 0.6% (5) to 1.99% (6). In New Zealand and Ireland the incidence among adults is reported to be 1.59/100,000 (7) and 3.3/100,000 (8), respectively.

The prevalence in children has been found to range from 1:158 in Turkey (9) to 1:320 in America (10). The reported incidence among children varies between 0.19/100,000 (7) and 51/100,000 (11). A summary of the key CD incidence and prevalence studies undertaken worldwide is shown in Appendix 1.

**Figure 1: Prevalence of CD in countries in the Asia-Pacific region**

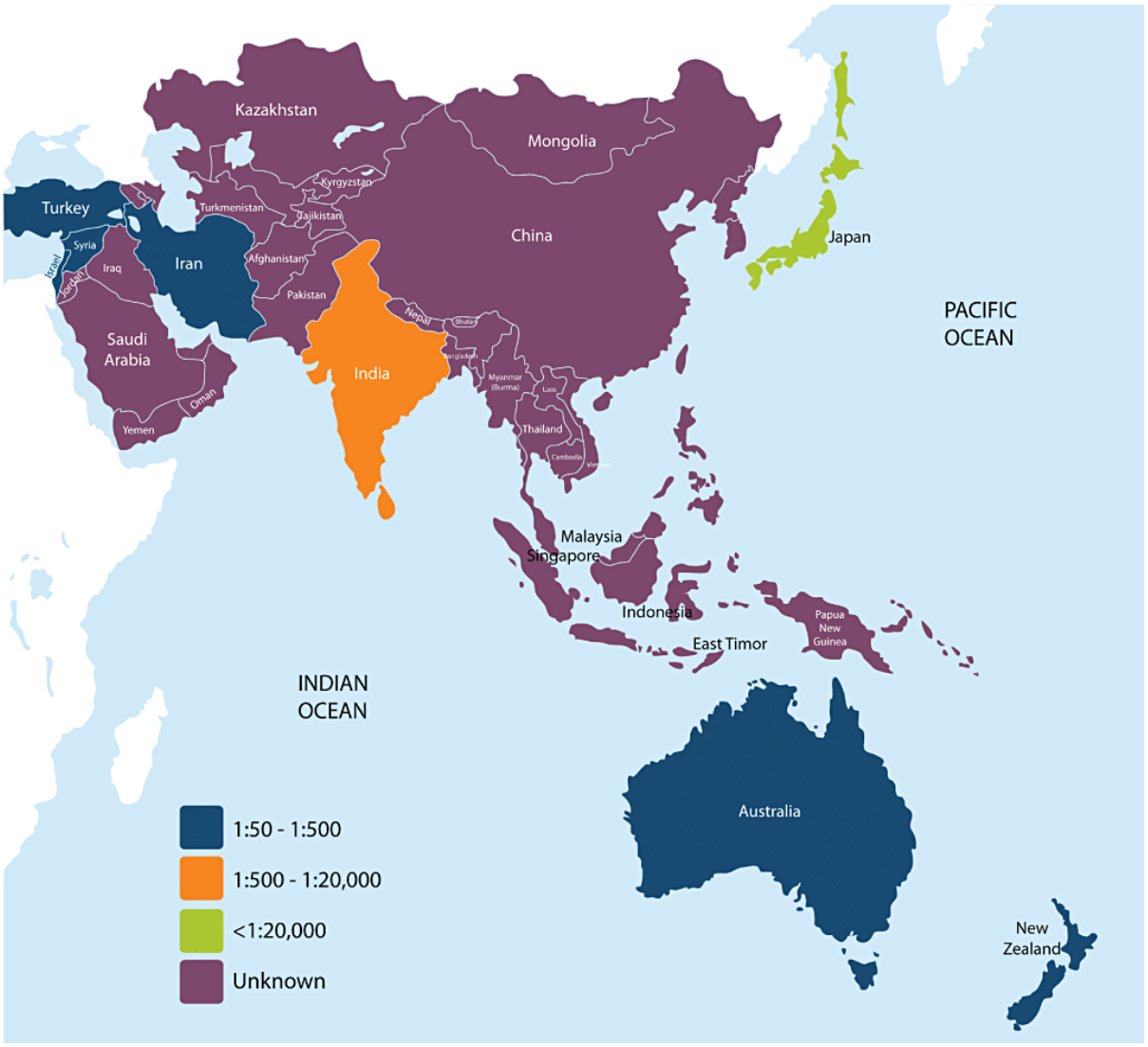



Image from: (12)

## Prevalence of Coeliac Disease in New Zealand

There are no national CD prevalence data for New Zealand but as observed in other countries, the incidence of CD in New Zealand appears to have risen (13). The few regional studies provide some good information.

A case review study conducted in the Canterbury region between 1970 and 1999 identified 416 people diagnosed with biopsy proven CD (13). Overall the annual incidence was 2.2/100,000/year. However, the incidence in the last 3 years of the study period (12.9/100,000 year) was 9 times higher than in the first three years of the study (1.4/100,000/year). The incidence of CD in adults started to rise in the mid 1980s, while the incidence in children started to rise in the early 1990s. Three-quarters (77%) of people diagnosed were aged between 10 and 59 years, and the ratio of women to men was 2.1:1.

Two earlier studies assessed the prevalence of CD in the Wellington and Otago regions. In the Wellington region, hospital medical records from 1985-1992 were used to estimate incidence (7). During the study period 38 people were diagnosed with CD, of whom four were children aged 12 years and under. The reported overall incidence was 1.8 per 100,000 and the overall prevalence was 70 per 100,000. Among children the prevalence was 14 per 100,000. The ratio of females to males was 3:1. The number of children identified was very low compared with other New Zealand and international studies. The authors postulated possible reasons were dietary differences, under-diagnosis or milder forms of CD presenting at a later age.

In the Otago region the prevalence of CD was assessed using Otago Hospital Board medical records for children and adults between 1975 and 1984 (14). The diagnostic codes for malabsorption, steatorrhoea, small intestinal biopsy and diagnosis of CD were used to identify cases. For the 9-year study period, the prevalence was 9 per 100,000 overall, and 35 per 100,000 among children. Both reported rates were considered to be relatively low, and probably due to under-diagnosis.

In a cross-sectional study conducted in Christchurch, 1064 randomly selected adults from the 1996 Christchurch electoral roll were screened using the anti-endomysium (EMA) antibody test for CD. Participants with a positive screening test were offered an intestinal biopsy test. Twelve adults tested positive for both tests and a further adult had known CD, giving an estimated prevalence of 1.2% (15). Although a random sample was used, the Canterbury based study population may not be representative of all groups in New Zealand, particularly in relation to genetic risk and ethnicity. The results of this study highlight another important point in relation to estimating prevalence; only 1 out of 13 adults with CD had previously been diagnosed. This suggests CD case finding studies underestimate the burden of disease.

More recently in 2009, the New Zealand Asthma and Allergy Cohort Study asked parents about whether their child had gluten intolerance or doctor diagnosed CD (16). Children participating in the study lived in Wellington or Christchurch. Overall 1% had doctor diagnosed CD, and the median age at diagnosis was 7.0 years. All these children had undergone blood testing but only 4 of the 9 children had had an intestinal biopsy.

**(ii) Reasons for the increase in the incidence and prevalence of CD**

The worldwide increase in the prevalence of CD is thought to be due to not only increased awareness and advances in screening and diagnosis, but also a true increase in prevalence.

Awareness of CD and its varying multisystem symptoms has heightened among the public and medical practitioners(13). Further, GF diets have become a nutritional trend, and with

this has come an abundance of misinformation. Nevertheless this trend translates to more people being aware of the symptoms of CD and investigating these with their doctor.

The relatively recent development of antibody tests (anti-gliadin antibodies, anti-endomysial antibodies, transglutaminase antibodies) has led to the widespread use of serological testing to screen for CD among those who are symptomatic but also asymptomatic high risk groups. Although a diagnosis of CD should be confirmed with a small bowel biopsy (17), serological screening tests can be more readily performed. Serological testing is quick, easy and affordable, and has opened the door to test many more patients with suspected CD.

Improved access to diagnostic facilities and improved diagnostic techniques in recent times is also likely to have contributed to the increased number of cases diagnosed. The change from capsule techniques to endoscopic small bowel biopsies has increased access and acceptance to biopsy tests (18). Further endoscopists are better skilled at recognising the macroscopic appearances of CD in the duodenum (19).

Despite these screening and diagnostic advances, a true increase in CD is thought to have occurred. Some consider it is possible that there has been a genetic shift in some populations with more people having the genes associated with CD (13). Another more likely reason is that the ingestion of gluten has increased in recent years. This is likely to have happened because the consumption of wheat products has increased and the gluten content of wheat has also increased. For technological reasons, wheat with a higher gluten content has been selected over time for food production (2). As exposure to gluten proteins in wheat, barley and rye (12) is a necessary factor to develop the disease, countries where

these grains are a staple, CD is more common. For example, many Caucasian populations have a staple diet of bread while Asian countries have a staple diet of rice which does not naturally contain gluten.

**(iii) Risk factors**

**Genetic predisposition**

CD is associated with the alleles of the HLA-DQ gene, the most common being HLA-DQ2 and HLA-DQ8. 90% of individuals with CD have the HLA-DQ2 serotype, while most of the remaining 10% have the HLA-DQ8 serotype (20, 21). This is not thought to be the complete story as many other genes also contribute to the development of the disease (2). Further, most people with HLA-DQ2 do not develop CD; in fact approximately 3% will (12). This suggests an environmental factor is required to trigger the disease.

The prevalence of the two alleles in a population pattern the prevalence of CD. Caucasian populations in western Europe have been shown to have a high frequency of HLA-DQ2 (20-30%) along with populations in northern and western Africa, the Middle East and eastern Asia (12). Low frequencies of HLA-DQ2 occur in South East Asia and Japan (12).

**Family History**

As CD has a strong genetic component, having a family history of CD is a risk factor. Having a sibling with CD is the single most important risk factor (22). The prevalence of CD among first degree relatives and second degree relatives of those with biopsy proven CD in the US is 4.5% and 2.6%, respectively, compared with 0.8% in the general asymptomatic population (10). However, a prevalence of up to 20% among first degree

relatives has been reported, with a higher prevalence being observed in families with multiple CD cases, particularly where the cases are male (23).

### Associated Autoimmune and Genetic Conditions

CD is associated with the following health conditions: type 1 diabetes, selective IgA deficiency, autoimmune thyroiditis, Downs syndrome, Williams syndrome, and Turners syndrome (24). For those with one of these conditions, the current recommendation is that the individual and their first degree family members be screened for CD. Short statue and dental enamel defects are also associated with CD in children (25).

## 2.2 Clinical Presentation

Both adults and children with CD may be symptomatic or asymptomatic at the time of diagnosis (24, 26). Those who are asymptomatic are most likely to be those who are screened because they have a family member with CD, or an associated autoimmune or genetic condition. Possible clinical manifestations of CD are listed in Table 1.

### (i) Adults

Typical gastrointestinal symptoms include steatorrhea (fatty stools), abdominal pain, abdominal bloating and distension, and excessive gas. Consequences of damage to the small intestine include lactose intolerance, iron deficiency anemia, and weight loss. Non-gastrointestinal symptoms can also occur and include, neurological symptoms and menstrual abnormalities (27). Other symptoms and conditions associated with CD include fertility problems and an increased risk of miscarriage, hyposplenism, abnormal liver function tests, (28), irritable bowel syndrome (IBS), osteopaenic bone disease (27).

**Table 1: Possible clinical manifestations of coeliac disease (27)**

**Table 1.** Possible Clinical Manifestations of CD

| Typical symptoms | Atypical symptoms | Associated conditions |
|---|---|---|
| Chronic diarrhea | Secondary to malabsorption | Possibly gluten dependent |
| Failure to thrive | Sideropenic anemia | IDDM |
| Abdominal distention | Short stature | Autoimmune thyroiditis |
| | Osteopenia | Autoimmune hepatitis |
| | Recurrent abortions | Sjögren syndrome |
| | Hepatic steatosis | Addison disease |
| | Recurrent abdominal pain | Autoimmune atrophic gastritis |
| | Gaseousness | Autoimmune emocytopenic diseases |
| | Independent of malabsorption | Gluten independent |
| | Dermatitis herpetiformis | Down syndrome |
| | Dental enamel hypoplasia | Turner syndrome |
| | Ataxia | Williams syndrome |
| | Alopecia | Congenital heart defects |
| | Primary biliary cirrhosis | IgA deficiency |
| | Isolated hypertransaminasemia | |
| | Recurrent aphthous stomatitis | |
| | Myasthenia gravis | |
| | Recurrent pericarditis | |
| | Psoriasis | |
| | Polyneuropathy | |
| | Epilepsy (with or without intracranial calcifications) | |
| | Vasculitis | |
| | Dilatative cardiomyopathy | |
| | Hypo/hyperthyroidism | |

### (ii) Children

The age of the child at the onset of CD may influence the type of symptoms they develop (27). Gastrointestinal symptoms in children include: diarrhoea, abdominal pain, vomiting, constipation, pseudo-obstruction, intussusceptions, abdominal distension, aphthous stomatitis and irritable bowel syndrome (IBS) (24, 27). Non-gastrointestinal tract symptoms include faltering growth, idiopathic short stature, dental enamel defects, osteoporosis and pathological fractures, delayed menarche, unexplained anaemia or iron deficient anaemia unresponsive to treatment, unexplained abnormal liver function tests, weakness, failure to thrive (FTT), muscle wasting and rickets. There may also be behavioural symptoms such as behavioural disturbances, and poor performance in school

(24, 27). Neurological symptoms are also seen in children with CD and include ataxia, muscle weakness, paraesthesias, sensory loss, epilepsy, and bilateral parieto-occipital calcification (27).

### 2.3 Complications of Coeliac Disease

#### (i) Nutritional Deficiencies

Damage to the small intestinal villi in CD causes a decrease in the functionality of the bowel to absorb a wide range of macro- and micronutrients. Normally, absorption of nutrients is divided into three phases; luminal, mucosal and post-absorptive. The *luminal phase* occurs in the lumen of the intestine; nutrients are broken down into smaller particles and bound to various compounds ready to be absorbed across the intestinal wall. The *mucosal phase* involves the transportation of the broken down nutrients across the brush border of the intestine into the intestinal cells. In the *post-absorptive phase* the nutrients reassemble and are transported via lymph or blood throughout the body to where they are needed. In CD the mucosal phase is predominantly affected, decreasing the capacity for nutrient absorption due to a loss of integrity of the brush border cells and a loss of cellular area available for absorption. Decreased absorption of carbohydrates may cause weight loss, muscle wasting and fatigue. Decreased absorption of fat also occurs leading to steatorrhoea (fatty stools), and possible deficiency of fat soluble vitamins. Other vitamin and mineral deficiencies that can commonly occur include iron (iron deficiency anemia), folate (megaloblastic anemia), vitamin $B_{12}$ (megaloblastic anemia) and vitamin K (abnormal coagulation). In the long term decreased absorption of vitamin D and calcium can affect bone health contributing to osteopenia and osteoporosis (27, 29).

Malabsorption in children and adolescents can also detrimentally affect growth (30). Failure to thrive may be evident along with growth deficiency of either slowed growth or a low height for age. In adolescents puberty may be delayed and girls may have delayed menarche or missed periods.

**(ii) Gastrointestinal**

In the long term untreated CD increases the risk of gastrointestinal cancer (adenocarcinoma and lymphoma), and ulcers in the bowel (ulcerative jejunitis) and narrowing leading to obstruction of the bowel (stricturing) (27, 31).

## 2.4 Diagnosis of Coeliac Disease

**(i) Why diagnosis is important**

Undiagnosed and untreated CD has serious health consequences, as indicated above, increasing morbidity and mortality (32), as well as significantly decreasing the quality of life (33-35). The length of time CD remains undiagnosed or untreated proportionally increases the risk of some health conditions. Exposure to gluten for people with CD increases the risk of developing other autoimmune diseases and intestinal lymphomas (36).

**(ii) Screening for Coeliac Disease**

Serological tests are now available to screen for CD, and have improved the diagnostic pathway because they are cheap, easy, accessible and repeatable compared with small bowel biopsy. High risk groups can be more readily assessed. The serological tests measure the autoimmune reaction to gliadin in gluten and endomysium (a structure of smooth

muscle tissue in the small bowel) (37). The tests vary in specificity and sensitivity, but the more recently developed tests, antiendomysium (EmA) and antitransglutaminase type 2 (TG2A) (38) have replaced the earlier tests as they extremely sensitive and specific (39). It is recommended that those with positive serological screening tests are usually referred for biopsy to confirm CD (40).

**(iii) Diagnostic tests for Coeliac Disease**

The accepted diagnostic test for CD is a small bowel biopsy taken from the distal part of the duodenum during an upper endoscopy before a gluten free diet (GFD) has been initiated (4). In people with CD the biopsy shows flat small intestinal mucosa. Damage to the bowel is assessed by the Marsh criteria (See Appendix 2). Sometimes the diagnosis is confirmed by repeating the biopsy following withdrawal of gluten from the diet to assess recovery of the mucosa (41), but this is not usual practice. Damage to the small bowel is not uniform throughout. (24) As damage is usually more pronounced in the proximal small bowel, samples are also taken from the distal part so the area of least damage is assessed (32). Thus, it is recommended that at least four biopsy samples are taken at different sites along the duodenum (32). An additional reason for multiple samples is that sometimes samples may be damaged or too small to assess. Even with best practice standards, there is a small possibility of a false negative. Thus, if there is still a strong clinical suspicion of CD even with a negative or ambiguous biopsy result, a repeat biopsy may be necessary (32). When a diagnosis of CD is unclear, genetic testing for the HLA genotype can also be done (32).

### (iv) Criteria for diagnosis of Coeliac Disease

Clear guidelines regarding the diagnosis of CD are not universally available or even nationally available. However, the diagnosis of CD does follow very similar standards and practices throughout the world, and is based on three key parameters (36):

1. Case Identification
2. Screening Tests
3. Definitive Tests

Because of increasing awareness of atypical presentation of CD, and advances in methods and practices for screening and testing for CD, Fasano and Catassi have updated and simplified the diagnostic criteria (40). They suggest if four out of the following five criteria are met, CD can be diagnosed:

1. Typical symptoms of CD
2. Positivity of serum CD IgA class autoantibodies at high titre
3. HLA-DQ2 and/or HLA-DQ8 genotypes
4. Celiac enteropathy found on small bowel biopsy
5. Response to a GFD

## 2.5 Management of Coeliac Disease

The aim of treatment is to restore small intestinal damage and to correct malabsorptive macro- and micronutrient deficiencies, and to maintain a well balanced nutritious diet. Currently, the only treatment for CD is strict lifelong elimination of gluten from the diet.

Future therapeutic options are in the early stages of development. While these offer hope for future treatment of CD being easier to manage and possibly including some gluten in the diet, a gluten-free diet (GFD) remains the mainstay of treatment in the mean time.

Monitoring is an important internationally recommended part of management, and includes assessment of the degree of recovery, and adherence to a GFD, as well as screening for associated disorders. Serological tests are used to monitor CD. The Celiac Disease Guideline Committee recommends measurement of TTG after 6 months of consuming a GFD (4, 24). This test should also be performed when people have persistent or recurrent coeliac symptoms. (36). If there is a rise in TTG the individual may be consuming gluten accidentally or non-accidentally. Coeliac sprue or non-responsive coeliac disease may also need to be considered (25).

**(i) Potential Future Treatments**

Increases in the knowledge of the pathological mechanisms in CD have led to new therapeutic options being developed to manage CD without the requirement for strict adherence to a GFD. This potentially allows individuals with CD to consume a less restrictive diet.

Potential treatments aim to correct any of the four main pathological disturbances in the bowel (42): (i) lack of the digestive enzyme intestinal glutaminase, (ii) antibody production to the gluten proteins or fragments of them, (iii) increased permeability of the bowel to macromolecules (including antigenic proteins and their fragments), and (iv) increased production and release of mediators such as histamine, serotonin, kinins, prostaglandins, interferons and interleukins (43). Much work continues on the development of both drugs

and vaccines for the treatment of CD, with a vaccine (Nexvax2) about to be assessed in a Phase 1b clinical trial (44).

Other therapeutic options include the production of non-toxic grains, that is cereals which contain very little or none of the immunotoxic sequences. These potential new grains have a higher baking quality than traditional gluten-free grains giving better nutritional quality to a GF diet. (43).

**(ii) Alternative Treatments**

While potential conventional therapies are being investigated, 'natural' therapies are also proposed. Although not validated in the scientific literature, herbal remedies are sometimes used to treat CD, and more commonly to treat the effects of CD such as gastrointestinal disturbances. Herbal remedies are used to soothe the gastrointestinal damage and irritation. This includes Roberts Formula (45), agrimony tincture (45), silica (45), dandelion saffron and yellow dock teas (helps cleanse blood) (45, 46), pickled ginger (reduces inflammation) (45, 46), primrose oil (46), medicinal clay (46), green food supplement (46), unrefined sea salt in water (46), alimony concentrate (46), liquorice root (47), marshmallow root (47), methylsulfonylmethane (MSM) (promotes structural repair) (47), aloe vera (47), and mucin (47). Anecdotally paprika has been reported to help in CD (47). Herbal remedies are thought to also help stimulate intestinal repair and repopulate the intestine with healthy flora composition, and control the amount of candida growth in the intestine. Herbal remedies believed to suppress candida production include: colloidal silver, garlic, hydrogen peroxide, grapefruit seed extract, baking soda, cayenne pepper, oregano oil, chamomile, wormwood, and extra virgin coconut oil (47).

## 2.6 The gluten free diet and associated problems

A gluten free diet (GFD) means elimination of wheat, barley, rye and triticale containing products from the diet. Whether oats should also be excluded from the diet is controversial, but in New Zealand the current recommendation is to also exclude oats from the diet (48). Adopting such a diet is a huge change nutritionally, physiologically, socially and financially. It involves ongoing effort to recognise and avoid gluten in everyday life, and relies on preparation of uncontaminated GF food.

The dietetic treatment of CD and associated malabsorption importantly requires a correct diagnosis. Gluten containing foods should not be unnecessarily eliminated from the diet without good reason, as a GFD tends to be lower in some nutrients and fibre. Following diagnosis, there are likely to be some nutritional deficiencies or low nutrient stores due to malabsorption. These deficiencies need to be identified as part of assessing a new patient with CD and ongoing monitoring. As the gut recovers, the diet can be adjusted accordingly taking into account correction in nutritional deficiencies. In some cases dietary supplementation may be necessary, for example iron and vitamin D (48).

### (i) Approaches to adopting a Gluten-Free Diet

When CD is diagnosed, patient dietary education needs to be provided by a health professional, ideally a registered dietitian, regarding the adoption of a GFD, coping skills, the consequences of not following a GFD, and support group and follow-up information. The first step in adopting and adjusting to a GFD is patient education. The person diagnosed with CD needs to know how to recognise gluten containing or gluten contaminated foods and food that will be safe to eat, as well as strategies for coping with

this fundamental dietary change. For a person with CD to avoid gluten, they must have knowledge of what foods do and don't contain gluten and be able to identify whether a food product contains gluten from the label.

Case (2005) proposed that there are five aspects to the successful management of CD (49):

1. *A team approach*. A multi-disciplinary team is involved in the diagnosis and treatment of CD. This includes doctors, gastroenterologists, dietitian or nutritionist and most importantly, the person, their family or caregivers and friends.

2. *An individualised approach*. The person's personality, age, life situation, income must be taken into account when strategies are put in place for GF eating.

3. *An understanding of quality of life issues*. How the person feels at the present time will dictate how motivated they are to put energy into changing their diet and lifestyle.

4. *Use of evidence-based, current information and resources*. Information given must be the most up-to-date and accurate and based on sound scientific evidence. The information base and educational resources should be constantly evolving as new evidence comes to light and new treatments are developed.

5. *Regular follow-up* to monitor compliance and nutritional status, and to offer ongoing support and education. In regard to GFD education, multiple visits should be booked to adequately cover all that patients need to know. The initial visit should cover the basics of a GFD and survival skills. Follow up visits should be used to explain the GFD in more detail, skills for handling a GFD, and making sure the whole diet is nutritionally adequate. It

would be very hard for a person to learn and understand all the details they need to know about a GFD in one visit. Spacing the visits allows small dietary changes to be accumulated and reduces the likelihood of a patient becoming overwhelmed. Following initial education, annual follow up appointments would be the ideal (50).

**(ii) Implementing a GFD as an individual with CD or as a parent to a child with CD**

Adopting a lifelong GFD may seem quite straightforward with the current labeling standards but can be a challenge to follow due to hidden sources of gluten, non-food sources of gluten and gluten contamination. This can be a particular challenge when a parent is trying to implement a GF lifestyle for a child. The child may not understand where gluten is found and may not yet have the intellectual capacity to identify and avoid gluten in their diet themselves. Within the home this may be able to be managed effectively, but during school, visits to friends and family, and birthday parties, the parents will have to rely on their child's supervisor at that location.

**(iii) The content of GFD Education**

1. Firstly the individual or family must realise that any product that can be ingested or comes into contact with the individual with CD must be GF (36). This is not limited to just food, but includes non-dietary sources which are or may be ingested. Non-dietary sources of gluten can include toothpaste, medication, suntan lotion, chewing gum, lipstick and playdough. Avoiding gluten can be particularly difficult with small children who frequently put things in their mouths without thinking about it.

2. Secondly dietary education to recognise gluten containing grains and potential hidden sources of gluten in products. Label reading is crucial to identify products containing or at risk of containing gluten. This will include but is not limited to education regarding the following:

- *Natural GF food choices.* These are single ingredient foods which are inherently GF. These include egg, fish, meat, 100% fruit juices, almonds, peanuts, milk, fresh fruit without waxing, lentils and butter.

- -*GF carbohydrate sources*. These naturally GF carbohydrate sources should be safe for those with CD to eat. They include corn, potatoes, rice, tapioca, amaranth, arrowroot, millet, motina, lupin, quinoa, sorghum, teff, taro, chia seed, yam, soybean and some nut flours.

- *Gluten containing grains and ingredients.* Knowing and identifying natural gluten containing grains is essential information for the person or parents of a child newly diagnosed with CD. These are wheat, rye, barley and triticale. In New Zealand it is recommended oats be avoided also (51). Even though they are inherently GF, contamination can occur during harvesting and processing as oats are often grown in close proximity to gluten containing cereals.

- -*Gluten contamination.* Knowing and identifying foods and ingredients that are at high risk of gluten contamination and therefore should be avoided.

- *Label reading education*. Understanding food labeling, including health claims and allergen warnings People with CD need to be familiar with the names and

alternative names of GF and gluten containing foods. When they pick up a product they need to be able to identify from the food or the label whether it is GF, whether the ingredients are GF, if the additives are GF, if it is processed on a GF line, and whether it is likely to be contaminated. Wheat-free is not the same as GF. Any mulit-ingredient product has the potential to be contaminated with gluten, thus necessitating an understanding of cross-contamination issues. Once the person is familiar with label reading there shouldn't be too much confusion as all food products must be labeled with allergens so will therefore be easy to identify foods to avoid.

- *Verifying gluten free status of food* products. If there is any confusion or uncertainty as to whether a food product contains gluten, people with CD need to know how and who to contact to verify the gluten-free status of a food. This will be the company or manufacturer as specified on the food label. An assertive approach needs to be taken when contacting manufacturers to clarify GF status of products.

3. Avoiding cross-contamination in the home

- *Cleanliness.* To avoid contamination of GF products with gluten, clean utensils, plates, bowls, cutting boards must be used. A clean bench is also essential. A separate toaster and toasted sandwich maker must be used for GF bread. GF foods should be packaged and stored separately from gluten containing foods.

4. Coping skills

- *Social situations* will now need an awareness of what food is offered and what it contains. While it may seem awkward, enquiries will often need to be made to the host or caterer or the person providing the food as to what the food contains and how it has been prepared, in order to avoid gluten.

- *Eating out* will entail much the same as other social situations. The person who is avoiding gluten or the caregiver making sure a child avoids gluten will need to become familiar with what to look for and how and who to contact to verify gluten-free status of a restaurant or take-away food.

- *Special needs for* children will require networking with teachers, staff, other parents or any other adult who will be responsible for a child with CD at any stage will be important to ensure a GF environment for the child. This may involve removing items containing gluten, supervising the child more closely to make sure they don't come into contact with gluten by for example sharing food, or playing with conventional play dough. Having a reference list of instructions and gluten-containing products can be a good idea to give to people responsible for children with CD, in case they forget certain details.

5. Support Information

- *Support* Groups. There are a lot of support groups and information available for people with CD or parents and carers of children with CD. It is important that people newly diagnosed with CD are referred to the correct groups and

organisations as these are an important source of social support and will help make the adjustment to a GFD much easier.

- *Special Authority Number and Disability Allowance*. Because of additional processing, gluten free foods are usually more expensive than gluten containing foods. This additional cost can be subsidised in New Zealand through a special authority number or a disability allowance. The disability allowance can be a child disability allowance or a disability allowance available to both children and adults. A child disability allowance is paid to a primary carer of a child that needs extra care for a serious disability. It is not income tested and an application must be accompanied by a doctor's medical certificate. A disability allowance is available to anyone who has extra cost due to a disability. This is income tested. A special authority number partially subsidises a small number of GF foods which are traditionally more expensive than the gluten containing counterparts. These are GF flour and pasta. Except for a child disability allowance, the subsidies are only available to those with biopsy proven (52).

**2.7 Testing for gluten in foods – What is the standard?**

People with CD are reliant on manufacturers of food products to provide true gluten free food. For manufacturers to label a product 'gluten free', they have to test or calculate the gluten content of a product. This is difficult to do accurately as there are many different methods for measuring gluten in foods and there is no set standard for assessing gluten contamination (53). This is especially important for wheat starch based gluten free products, where the product is processed efficiently enough so that the gluten is removed, is

as safe as naturally GF foods. Provided the products are adequately processed and tested, the consumption of wheat starch based gluten free products appears to have no or minimal effect on the intestine (54-57). However, a standardised system for the measurement of gluten in food is needed so people have confidence in the product when purchasing 'gluten-free' foods. Thompson et al (2008) states "The degree of confidence that can be placed in a manufacturer's assertion that a product is gluten-free is based on the assay used to determine the gluten content and the specific food analysed" (1).

**(i) Testing**

The organisations which lead the way for gluten standards for the rest of the world currently endorse different methods. The Association of Analytical Communities endorses the sandwich ω-gliadin enzyme-linked immunosorbent assay (ELISA), whereas the Codex Alimentarius Commission endorses the sandwich R5 ω-gliadin enzyme-linked immunosorbent assay (R5 ELISA) (1). Both of these methods have strengths and weaknesses (See Appendix 3).

To recap gluten is a water-soluble protein found in wheat, and consists of an alcohol-soluble fraction, gliadin, and an insoluble fraction, glutenin. The gliadin fraction can be further subdivided into α, β, γ and ω fractions, and collectively known as prolamins. They are termed gliadin in wheat, hordein in barley, secalin in rye and avenin in oats. Both ELISA and R5 ELISA quantify native and heated gluten, but are unable to accurately quantify hydrolysed gluten. The R5 ELISA overestimates barley hordein, while the ELISA method is unable to accurately detect and quantify barley prolamins, as the prolamins from

barley are in the form of hordein and the ELISA only measures the prolamin gliadin. Due to this restriction the ELISA can over and underestimate gluten content of foods (1).

**(ii) Safe gluten threshold in foods**

Ideally people with CD should consume no gluten whatsoever, but some food products may still contain residual gluten. Regular accurate testing for gluten in 'gluten-free' products is required to be sure GF food products are free of gluten or within the safe level for gluten contamination. For safe management of CD, thresholds for allowable safe gluten levels in food products are specified. Determining a safe allowable level of gluten in food products involves investigating at what levels gluten causes not only symptoms but also the intestinal mucosal damage.

The current standard of the Codex Alimentary Commission GFD 1981 threshold value is 0.05g nitrogen per 100g dry matter, because at the time the standard was established methods to measure gluten weren't available (58). Subsequent studies this to establish safe threshold levels for food have shown variable results (59-62). This is complicated by the fact that individuals with CD react differently to varying amounts of gluten and any reaction can occur within different timeframes, from almost immediately up to a day or two later (63). Also, at what point is the inflammatory response to gluten causing harm to the person? And what levels of harm are permissible? That is, what level is acceptable because it does not cause permanent damage or does not interrupt activities of daily living?

Further sensitivity levels can differ markedly between children and adults. Adolescence further complicates sensitivity to gluten as it is during this period that CD can sometimes goes into remission (64). A number of studies have explored gluten sensitivity in children

which have usually involved a gluten challenge (59, 65-67). From these studies, it is apparent that the amount of gluten intake seems to have a dose dependant effect, particularly on the intestinal intraepithelial lymphocyte count. Also, smaller amounts of gluten than previously thought are needed to cause damage to the small intestine, and these microscopic changes may occur before symptoms occur.

Clearly there is much to consider when establishing a safe gluten threshold in foods for CD patients, and much work has been to achieve this. Collin et al. (2004) took the approach that if CD is currently being treated effectively then the gluten levels in GF foods must be safe for long term use (68). They used the ELISA method to measure gluten levels in the most widely used natural gluten free products, wheat starch based gluten free flours and baked products. The detection limit for gluten was 10ppm. They also used food records to estimate daily intake of flour. Physiological effects from the gluten were measured by small intestine biopsy and serum immunoglobulin A (IgA) class endomysial antibodies. Seven products were found to contain >100ppm gluten, but less than 200ppm. The median daily intake of flours was 80g in adults and 60g in children. This had no correlation with intestinal mucosal histology or levels of anti-endomysial antibodies. Thus, in consideration with other findings, Collin et al. (2004) recommended that an acceptable threshold for gluten in GF food products could be set at 100ppm (68). This level takes into account the practical aspect of GF diet compliance and allows reasonable variety within a GF diet. However 100ppm is not the widely adopted threshold, and 20ppm is the threshold for gluten levels adopted by many countries, including most European countries, Canada, Australia and New Zealand (69-72). This lower level of gluten in foods has been found to

be very safe for people with CD, and the chance of an adverse reaction is very low (70). The level of 100ppm is used as a threshold to label products as “low gluten”.

### 2.8 Gluten contamination of foods

Foods can become contaminated by gluten at any stage from when naturally occurring gluten free cereals are grown to when foods are served (73). Gluten contamination can occur when crops are grown with or rotated with other crops in the same soil. During the harvesting and processing of foods contamination can occur by harvesting and processing gluten containing and GF crops and foods together or by using the same equipment for both and not cleaning it adequately between the two food groups. When GF food is handled or prepared using the same (inadequately washed) equipment for gluten containing food, there is a risk GF foods can be contaminated. This may occur during packaging, transportation, storage, food preparation, cooking and serving.

In the USA inherently GF grains, seeds and flours (not labelled GF) had gluten levels ranging from 8.5 to 2,925.0ppm (73). Around one third of the samples (32%) had mean gluten levels ≥20 ppm. Under the proposed FDA rule for gluten-free labelling (that proposes that inherently GF grains, seeds, and flours no longer need to be labelled as GF), these would not meet the GF standard.

People with CD often eat GF substitutes for wheat containing foods, such as substituting wheat flour bread with GF bread. A European study evaluated the risk of mucosal damage in those with CD due to gluten consumption via these products (74). The gluten content of the products in this study was between <5 and 27.8 mg/kg (ppm). 99.5% of the foods had a

gluten concentration <20 ppm with 94% being below 5 ppm. They found a very low risk of mucosal damage from eating these products.

Oats are an inherently GF product that are prone to gluten contamination. Both New Zealand and the USA recommend those with CD do not consume commercial oat products as they may be contaminated with wheat, barley, or rye during harvesting, transporting, milling, and processing (75). In the USA Thompson (2004) found 75% of the 12 different oat brands tested had gluten levels above 20ppm ranging from 23 – 725ppm (75).

Gluten contamination has been shown to be a problem in the USA, but less so in European. However, this flags the issue of whether GF substitute products and inherently GF foods are consistently GF in New Zealand.

Very little research exists detailing whether gluten contamination of GF foods in New Zealand is a problem, and what the nature and extent of the problem is.

Under the current food standards set by FSANZ, for food to be labelled as 'gluten-free' it must contain no detectable gluten and no oats or malted gluten containing cereals or their products, and for a food to be classed as low-gluten it must have gluten levels less than 200ppm (71). Currently it is not recommended that people with CD consume low gluten foods (71).

### 2.9 Sources of information about gluten free foods in New Zealand

Previously, GF foods in New Zealand were listed annually on the Manufactured Food Database (MFD). This database ceased to operate in 2012. The listed foods relied on self-report by manufacturers. Other resources to aid in choosing GF foods are available through

the New Zealand Coeliac Society (51) or through a dietitian or doctor. Contacting the manufacturer directly about ingredients, processing and risk of contamination may be helpful in determining the risk of contamination of a product.

## 3.0 OBJECTIVE STATEMENT

About 1% of New Zealanders have CD, but very little is known about their health. A gluten free diet (GFD) is the only treatment, but little is known about the quality of GF food in New Zealand, and whether this has an impact on patients with CD. Thus, the aims of this study were:

1. To describe the health of people with CD in New Zealand and the impact of a gluten free diet.

2. To determine the prevalence of on-going symptomatic CD.

# 4.0 METHODS

The New Zealand Coeliac Health Survey was a cross sectional study administered in New Zealand. This study was given ethical approval by the Multi-region Ethics Committee on June 5 2012. The study reference number is MEC/12/EXP/072 (See Appendix 4 & 5).

The study population consisted of all members of Coeliac New Zealand Incorporated (Inc). who had an email or postal address, and included all ages, as well as parents or guardians of children. The number of members in July 2012 was 2720. Coeliac New Zealand is a not for profit organisation supporting those diagnosed with coeliac disease, dermatitis herpetiformis and those on a gluten free diet. They support those adjusting to and maintaining a GF diet, through support groups, resources and education. They also collaborate with gluten free manufacturers, medical professionals and support research (51).

## 4.1 Administering the survey

The New Zealand Coeliac Health Survey (See Appendix 6) was primarily administered as an online survey using Survey Monkey Software (www.surveymonkey.net). A link to the survey, along with a study invitation and information sheet (See Appendix 7 - 11), was emailed to 2383 members of the Coeliac New Zealand Inc. The study invitation and information sheet stated participants could forward the email containing the link to other people they knew who had confirmed CD. This created a snowball effect expanding the sample size and including people with CD who were not members of Coeliac New Zealand Inc. for whatever reasons. Paper surveys with a return stamped envelope were sent via post

to 337 Coeliac New Zealand Inc members who did not have an email address but had a postal address.

All surveys were sent out by Coeliac New Zealand Inc to maintain anonymity of the participants. If participants preferred to fill out a paper survey, rather than complete the survey online, they emailed the investigator who sent paper copies of the information and the survey to them.

Participants were given a month to complete and return the survey. A reminder email was sent to the email participants after 2 weeks and 3 weeks to increase response rate (See Appendix 12). The survey was also advertised at a local Coeliac New Zealand meeting in Palmerston North and Dunedin prior to the email and postal invitations being sent out to enhance the response rate. A thank you was sent to all participants after the study closing date (See Appendix 13).

**4.2 The questionnaire**

Permission was obtained from Dr. Mohsin Rashid for Professional Advisory Board and Board of Directors Canadian Celiac Association to use the Canadian Celiac Health Survey as a basis for this study (See Appendix 14). The reason for using this study is that the Canadian Celiac Health Survey is a reputable survey that has been successfully used in previous studies (76, 77). This questionnaire was developed by the Professional Advisory Board of the Canadian Celiac Association in collaboration with the department of Epidemiology and Community Medicine of the University of Ottawa. Dialogos Educational consultants were employed to help develop this survey (77). The survey was pretested by

members of the Canadian Celiac Association Board of Directors and pretested formally on 14 Canadian Celiac Association members for readability and ease of completion (76).

A pilot study was conducted on members of the Ottawa Chapter of the Canadian Celiac Association using a Modified Dillmans' Total Design method for mail surveys (76). There was a 76% response rate to this survey (n=315), but data were used from 64% of the original sample (n=266) due to the exclusion of participants who did not have biopsy-proven celiac disease. Feedback about the questionnaire was used to further improve the survey (76). This pilot study confirmed the feasibility of a national survey (76).

The Canadian questionnaire was adapted slightly to fit a New Zealand audience. Permission was given to modify the questionnaire to suit the needs of the New Zealand study. One section however was removed from the Canadian Celiac Health Survey (the generic quality of life, section 4, questions 21 to 32) as this section was evaluated using the SF-12$^{TM}$ (78) health survey for which The Canadian Celiac Association did not have the copyright for. Overall, the survey was kept as close as possible to the original. Questions were added in order to obtain data on ethnicity, whether the person completing the survey was a member of Coeliac New Zealand Inc or not and whether the person received a financial subsidy or not. A PHARMAC special authority number and a disability allowance are the two channels for financial support for people with CD in New Zealand. A question was also added to investigate how often the participant accidently consumed gluten. Comment boxes were also added to enquire about reasons why it may have been difficult to follow a GFD, and to provide additional comments describing their present GFD, and any other comments, information or suggestions regarding any CD related topic included in the

survey. Postal code was replaced with region in the demographics section. Some of the questions were country specific to Canada so these were changed so that they were specific to New Zealand. These included: information sources about coeliac disease, question investigating whether family members were also members of the local coeliac society, and the maximum level of educational achievement. To decrease the length of the survey in order to increase participation, Sections 7 and 8 were made optional. These two sections investigated CD in family members and other associated diseases among participants and their first degree relatives. Lastly we offered an incentive of a chance to win a fifty dollar book voucher to increase participation in the survey. Participants could elect to enter the draw for this or not. The survey was pilot tested on a few New Zealanders and the survey was modified according to their feedback.

The survey was uploaded online using the software Survey Monkey (www.surveymonkey.net). Due to the limitations of the software nineteen questions with multiple parts were split into multiple questions but overall the format was kept as close as possible to the paper survey version.

The postal surveys were entered into Survey Monkey by the primary researcher and an assistant using the same Survey Monkey software that participants who completed the survey online. Data entry was double checked for accuracy. Following data entry, the data were exported from Survey Monkey to Microsoft Excel for statistical analysis. Before data analysis the data were cleaned and checked for errors. Any nonsensical data were deleted before analysis.

4.3 Statistical AnalysisThe data were analysed using STATA Data Analysis and Statistical Software. For each question an absolute value and proportion, or mean, as appropriate, were calculated. Long answer and open answer questions were analysed separately. Text answers were reviewed and the most recurring themes identified.

## 5.0 RESULTS

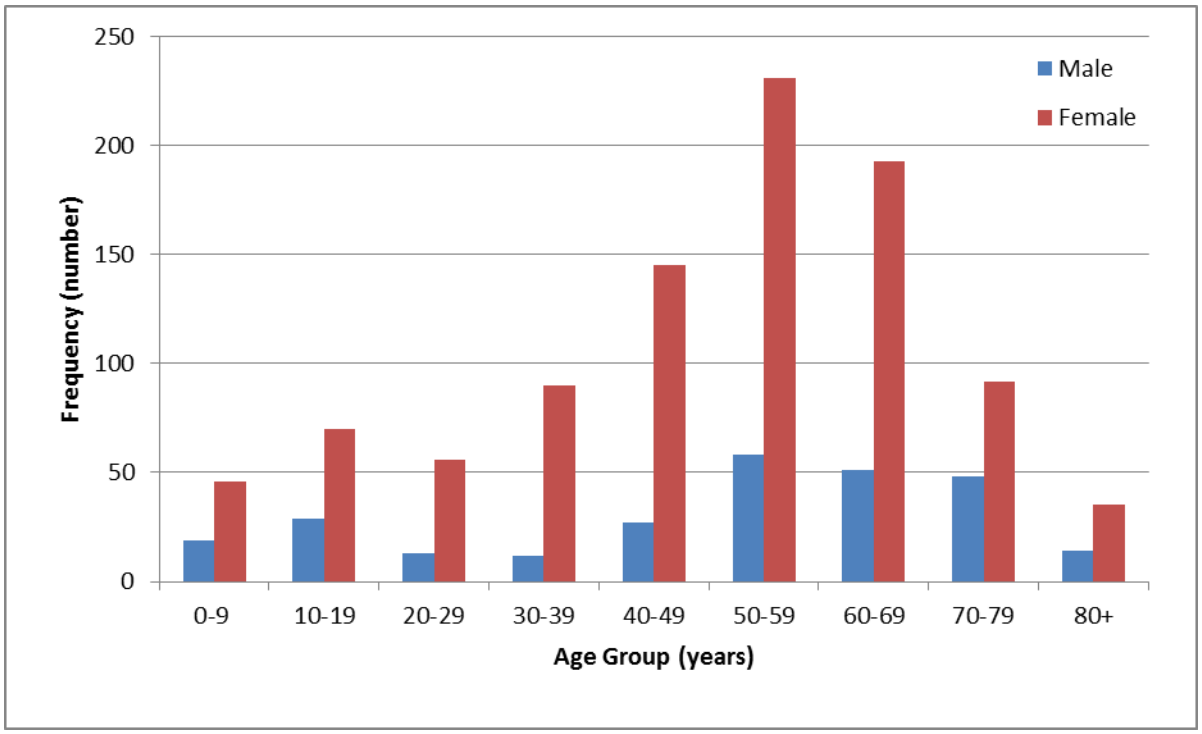
The New Zealand Coeliac Health Survey had 1264 responses giving a response rate of 46.5%. Ten responses were excluded due to minimal or no data being provided. A further 29 were excluded because their date of birth or sex was missing leaving 1225 complete responses for the analysis (46.1%).

The age range for all respondents was 0 - 95 years. The age distribution of all respondents is shown in figure 4. Overall 1044 (224 males and 820 females) had biopsy proven CD or biopsy proven DH. Among the paediatric population aged <16 years, 108 had biopsy proven CD and 23 had biopsy proven DH.

For the purpose of this thesis, the results for the adult population (age ≥ 16 years) with a positive biopsy are reported (n= 936). There were 199 males and 737 females giving a male:female ratio of 1:4.7. The mean age was 57 years for males and 53 years for females (54 years for the total study population).

The majority of participants (94%) were New Zealanders of European descent. 13 were New Zealanders of Maori descent, 1 was a Pacific Islander, and 55 self-identified with other nationalities, predominantly European based, and included Australian, American, Austrian, British (English), Canadian, Dutch, Indian, Irish, Italian, Jewish, Lebanese, Scottish, South African and Spanish. Most (82%) participants had completed high school, and 34% had completed a university or post graduate degree.

**Figure 2: The proportion of males and females with biopsy proven CD by 10 year age groups**

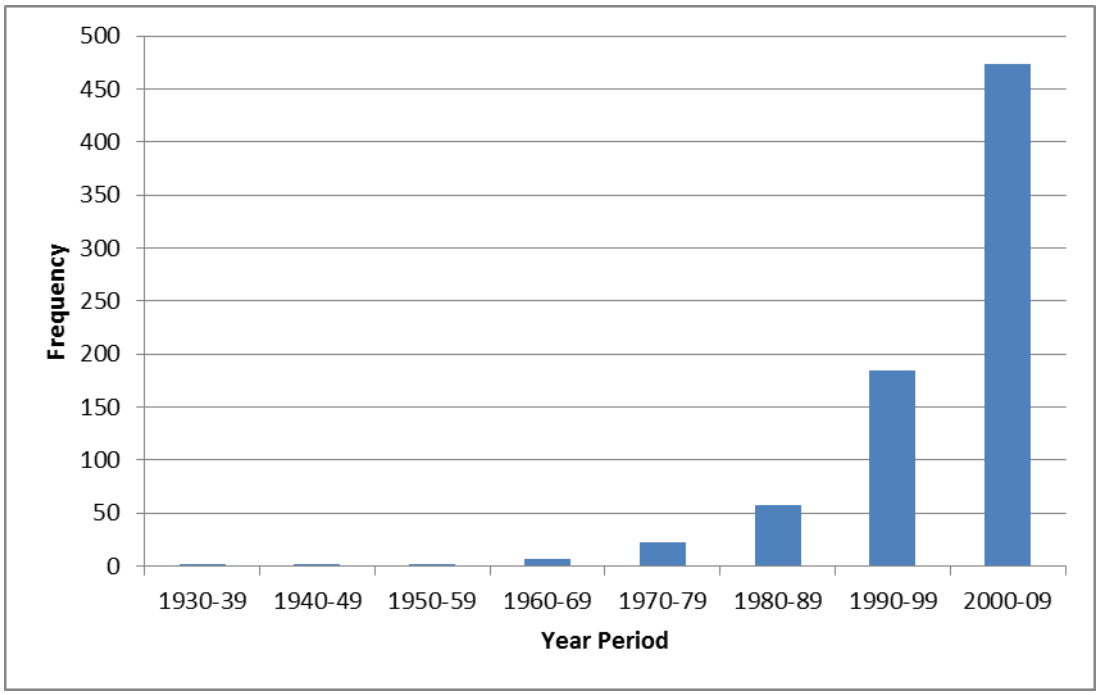



## Duration of Diagnosed Coeliac Disease

The mean age at diagnosis of CD was 43.5 years (42.8 years for females and 45.9 years for males). The range for duration of diagnosed CD was 0 – 80 years, and the mean duration of CD for people in this study was 10.3 years (SD 0.76 – 19.76).

Figure 3 shows the number of respondents reported to be diagnosed with CD each decade from 1930-1990. The earliest diagnosis was in 1932, but most people (87%) were diagnosed over the last 20 years, with 60% diagnosed in the last 10 years. A further 171 participants were diagnosed between 2010 and the end of the study period, May 2012.

**Figure 3: The frequency of diagnosis of CD by decade, 1930-2009.**




## Clinical Symptoms and Recovery from Symptoms

Table 2 details the many different symptoms that participants experienced prior to being diagnosed with CD. Participants experienced a range of gastrointestinal and non-gastrointestinal symptoms. A combination of bloating, gas and abdominal pain was the most common symptom (76%), followed by extreme weakness or tiredness (67%), diarrhoea (64%) and anaemia (57%). Other gastrointestinal symptoms included large, pale foul-smelling stools, constipation, nausea or vomiting, lactose intolerance, and hypoglycaemia. Other non-gastrointestinal symptoms included mood swings/depression, itchy skin, bone/joint pain, mouth ulcers, easy bruising of skin, migraine headaches, muscle cramps, eczema, swollen extremities, and poor growth, all of which were reasonably common.

**Table 2: Clinical symptoms prior to diagnosis**

| Symptom | Present at diagnosis n (%) | Fully Recovered n (%) |
|---|---|---|
| Bloating, gas, abdominal pain | 714 (76.3) | 372 (52.1) |
| Extreme weakness or tiredness | 630 (67.3) | 328 (52.1) |
| Diarrhoea | 602 (64.3) | 344 (57.1) |
| Anaemia | 530 (56.6) | 360 (67.9) |
| Weight loss | 460 (49.1) | 349 (75.9) |
| Large, pale, foul-smelling stools | 389 (41.6) | 262 (67.4) |
| Mood swings/depression | 374 (40.0) | 152 (40.6) |
| Itchy skin | 333 (35.6) | 102 (30.6) |
| Constipation | 305 (32.6) | 131 (43.0) |
| Bone/joint pain | 304 (32.5) | 89 (29.3) |
| Mouth ulcers | 296 (31.6) | 176 (59.5) |
| Easy bruising of skin | 276 (29.5) | 78 (28.3) |
| Nausea or vomiting | 270 (28.8) | 188 (69.6) |
| Migraine headaches | 268 (28.6) | 118 (44.0) |
| Muscle cramps | 265 (28.3) | 105 (39.6) |
| Eczema | 190 (20.3) | 70 (36.8) |
| Lactose intolerance | 166 (17.7) | 63 (38.0) |
| Swollen hands/feet/ankles | 119 (12.7) | 52 (43.7) |
| Poor growth | 118 (12.6) | 67 (56.8) |
| Hypoglycemia | 71 (7.6) | 34 (47.9) |

Table 2 also shows the proportion who experienced the different symptoms and reported having fully recovered from the symptom after the diagnosis of CD. Many people reported ongoing symptoms. Those who experienced skin problems, or joint or bone pain were less likely to report having fully recovered from these symptoms. About two-thirds of those with these symptoms at diagnosis continued to have them. There were only four symptoms where more than two-thirds of participants reported complete recovery from; weight loss, nausea and vomiting, anaemia, and large pale foul smelling stools.

For those who had not fully recovered from any of these symptoms, this was most commonly attributed to an unknown cause (n=405), followed by being unaware of sources of gluten (n=243), and other food allergies (n=118). Only a small proportion attributed ongoing symptoms to be due to problems adhering to a gluten free diet (n=60).

**Previous diagnoses and possible triggers**

Before diagnosis, many signs and symptoms seen in CD can be attributed to other conditions. About one-third (35%) reported they were diagnosed with anaemia, 21% with stress/nerves/depression, and 21% with irritable bowel syndrome. Table 3 shows a range of other diagnoses participants were advised they had prior to being diagnosed with CD. The relatively common diagnoses were vitamin deficiencies, acid reflux, chronic fatigue syndrome, menstrual problems, and a food allergy, while stomach or duodenal ulcers, hiatus hernia, gallbladder, and diverticulitis were less frequent diagnoses.

**Table 3: Diagnoses given to participants prior to a correct diagnosis of CD (n=936)**

| **Diagnosis** | **n (%)** |
|---|---|
| Anaemia | 329 (35.1) |
| Stress/nerves/depression | 201 (21.5) |
| Irritable bowel syndrome | 199 (21.3) |
| Vitamin deficiencies | 178 (19.0) |
| Acid Reflux | 147 (15.7) |
| Chronic fatigue syndrome | 103 (11.0) |
| Menstrual problems | 101 (10.8) |
| Food Allergy | 95 (10.1) |
| Stomach or duodenal ulcers | 58 (6.2) |
| Hiatus hernia | 47 (5.0) |
| Gall bladder | 34 (3.6) |
| Diverticulitis | 33 (3.5) |

Major medical and psychological events can trigger the onset of CD. Of the 936 respondents, 21% identified that they had severe stress within 6 months prior to the onset of symptoms of CD. A further 9% had a pregnancy, 9% had a severe intestinal infection, 7% had major surgery, and 4% had severe influenza a few months prior to diagnosis of CD.

**Table 4: Possible triggers of CD (n=936)**

| **Possible trigger** | **n (%)** |
|---|---|
| Severe Stress | 192 (20.5) |
| Pregnancy | 87 (9.3) |
| Severe intestinal infection | 81 (8.7) |
| Major Surgery | 63 (6.7) |
| Severe influenza | 42 (4.5) |

**Delay in diagnosis**

The mean time between the reported onset of symptoms and a diagnosis of CD was 11.6 years (SD -3.6 -26.8) (Males: 10.4 years, females: 11.9 years). 59% of respondents had symptoms for between <1 year and 10 years before diagnosis. 12% had symptoms for between 11 and 20 years before diagnosis.

**Table 5: Self-reported time between onset of symptoms and diagnosis of CD**

| Duration of symptoms before diagnosis (years) | n (%) |
|---|---|
| ≤1 | 205 (21.9) |
| ≤2 | 92 (9.8) |
| ≤3 | 61 (6.5) |
| ≤4 | 30 (3.2) |
| <5 | 2 (0.2) |
| 5-10 | 159 (17.0) |
| 11-20 | 108 (11.5) |
| 21-30 | 55 (5.9) |
| 31-40 | 34 (3.6) |
| 41-50 | 32 (3.4) |
| 51+ | 22 (2.4) |

**Medical consultations prior to diagnosis of Coeliac Disease**

The most commonly consulted medical practitioner about symptoms prior to diagnosis of CD was a general practitioner (GP) with 93% of respondents consulting at least one. The second most commonly consulted medical practitioner was a gastroenterologist with 65% consulting a gastroenterologist/s. 63% of respondents saw both a GP and a gastroenterologist. Table 6 shows the wide range of other medical and health practitioners who were consulted prior to the diagnosis of CD. Often multiple health practitioners were consulted, with 34% consulting two or more family doctors, 16% consulted two or more physicians, 11% consulted two or more gastroenterologists, and 38% consulted 2 or more alternative medical practitioners.

The most common health professional to diagnose CD was a gastroenterologist. 58.2% reported being diagnosed by a gastroenterologist, and 17.6% by a GP. A further 2.1% reported both a GP and gastroenterologist contributed to their diagnosis of CD (n=20). A

wide range of other health professionals were reported to diagnose CD including a paediatrician, endocrinologist, surgeon, dermatologist, gynaecologist, haematologist, endoscopist, bone specialist, naturopath, neurologist, oncologist, allergist, consultant, ENT specialist, eye doctor, hepatologist, and homoeopath.

**Table 6: Medical practitioners who made the diagnosis of CD (n=936)**

| Type of Doctor | n (%) |
|---|---|
| Gastroenterologist | 545 (58.2) |
| GP | 165 (17.6) |
| Specialist | 32 (3.4) |
| Physician | 28 (3.0) |
| Gastroenterologist & GP | 20 (2.1) |
| Doctor | 19 (2.0) |
| Paediatrician | 17 (1.8) |
| Endocrinologist | 16 (1.7) |
| Surgeon | 10 (1.1) |
| Dermatologist | 7 (0.7) |
| Self | 5 (0.5) |
| Gynaecologist | 4 (0.4) |
| Haematologist | 4 (0.4) |
| Endoscopist | 3 (0.3) |
| General Surgeon | 3 (0.3) |
| Bone specialist | 2 (0.2) |
| Hospital | 2 (0.2) |
| Naturopath | 2 (0.2) |
| Neurologist | 2 (0.2) |
| Oncologist | 2 (0.2) |
| Allergist | 1 (0.1) |
| Consultant | 1 (0.1) |
| ENT specialist | 1 (0.1) |
| Eye doctor | 1 (0.1) |
| Heptologist | 1 (0.1) |
| Hospital Gastronomy Dept | 1 (0.1) |
| House Surgeon | 1 (0.1) |
| Laproscopic Surgeon | 1 (0.1) |
| Orthopaedic Surgeon | 1 (0.1) |
| Physician & Dermatologist | 1 (0.1) |
| Professor | 1 (0.1) |
| Homoeopathic doctor & specialist | 1 (0.1) |
| Specialist & Surgeon | 1 (0.1) |

**Initial Dietary Management**

Most (94%) participants reported they were told they needed to follow a gluten-free diet (GFD) for life at the time of diagnosis, while a few (0.7%) reported that they were not told. Of those that were told they needed to follow a GFD for life, 82% (n=764) were referred to a nutritionist or dietitian for information about adopting a GFD.

Most (92%) participants reported always following a GFD from diagnosis. A small proportion (4%) of participants followed a GFD sometimes. No one indicated that they did not follow a gluten free diet, but 78 respondents did not answer this question.

Most (96%) found their health improved to some extent after commencing a GFD. Three-quarters (74%) found their health improved a lot, while 18% found their health moderately improved. A further 5% (n=46) found their health improved only a little, and 2% (n=16) found their health did not improve at all.

Although most (88%) participants described their diet as being strictly gluten free, 6% (n=60) described their diet as being partially gluten free and a small number (n=3) described their diet as not being gluten free. Following a GFD was described as being very difficult for 6%, moderately difficult for 30%, a little difficult for 38%, and not difficult at all for 24%. Reasons were provided as to why it was or wasn't difficult to follow a GFD and these are summarised in the section below, "Challenges with adopting and following a gluten free diet".

**Sensitivity to gluten and accidental ingestion of gluten**

80% have a reaction after accidentally consuming gluten-containing food. A further 14% were not sure if they have a reaction to consuming gluten containing foods and 6% reported not having a reaction. Table 7 shows the symptoms experienced following the accidental consumption of gluten-containing foods. The most common symptoms were gastrointestinal including abdominal discomfort (65%), diarrhoea (59%), bloating/gas (48%), and nausea and vomiting (33%).

**Table 7: Symptoms experienced following accidental consumption of gluten-containing foods (n=936)**

| Symptom | n (%) |
|---|---|
| No reported reaction | 53 (6) |
| Stomach/abdominal discomfort | 607 (65) |
| Diarrhoea | 554 (59) |
| Bloating/gas | 448 (48) |
| Fatigue | 337 (36) |
| Nausea/vomiting | 313 (33) |
| Headache | 208 (22) |
| Itcchy skin | 148 (16) |
| Constipation | 131 (14) |
| Mouth ulcers | 84 (9) |
| Insomnia | 80 (9) |

The mean time for the first symptoms to appear after accidentally consuming gluten was 4.9 hours (SD 3.3 -13.1). Most commonly the onset of symptoms occurred within one to two hours, but symptoms were reported to occur almost immediately and up to 72 hours following ingestion.

The reported frequency of accidental consumption of gluten varied. Three-quarters (76%) rarely or occasionally (less frequently than monthly, but at least annually) were aware that they had accidentally consumed gluten. 12% were aware they accidentally consumed gluten monthly and 5% at least weekly.

**Education and Quality of information on Coeliac Disease and Gluten Free Diet**

The most common source of information about CD and its treatment was Coeliac New Zealand Incorporated (91%). Other common sources of information were dietitian/nutritionist (75%), cook books (69%), the internet (58%), newspapers/magazines (52%) and other people with CD (51%). Other sources of information included family doctor, regional coeliac group, medical books, alternative medical professional and gastroenterologist.

The perception of the quality of the information from these sources was generally high. The most highly rated source of information was Coeliac New Zealand Incorporated. Of the 855 people sourcing information from Coeliac New Zealand Incorporated, 93% considered it to be excellent or very good. Three-quarters of people sourced information from a dietitian or nutritionist and half from a GP, and the quality of information provided was considered to be fair or poor by 19% for dietitians and 30% for GPs. Few people sought information from an alternative medical professionals, many of whom were considered to provide fair (11%) or (34%) poor quality information.

**Table 8: Sources of information and perceived quality of information**

| Source of Information | Received | Quality of Information (%) | | | | |
|---|---|---|---|---|---|---|
| | n (%) | Excellent | Very good | Good | Fair | Poor |
| Coeliac New Zealand Incorporated | 855 (91.3) | 66.8 | 25.6 | 5.7 | 0.9 | 0.2 |
| Dietitian/nutritionist | 698 (74.6) | 36.0 | 25.6 | 22.2 | 12.5 | 7.4 |
| Cook books | 647 (69.1) | 23.6 | 41.0 | 28.4 | 5.3 | 1.1 |
| Gastroenterologist | 572 (61.0) | 42.7 | 31.3 | 22.4 | 8.9 | 6.6 |
| Internet | 543 (58.0) | 22.3 | 42.5 | 26.5 | 4.6 | 1.7 |
| Newspapers/magazines | 488 (52.1) | 13.7 | 35.9 | 37.7 | 11.1 | 2.7 |
| Another person with coeliac disease | 480 (51.3) | 34.6 | 38.3 | 21.9 | 2.5 | 3.1 |
| Family doctor | 457 (48.8) | 26.3 | 34.6 | 31.9 | 20.8 | 20.1 |
| Regional coeliac group | 341 (36.4) | 48.4 | 31.7 | 17.6 | 6.7 | 7.3 |
| Medical books | 176 (18.8) | 34.1 | 40.3 | 27.3 | 8.0 | 15.9 |
| Alternative medical professional | 66 (7.1) | 27.3 | 37.9 | 42.4 | 21.2 | 65.2 |

## Quality of Life Issues

Table 9 shows the proportion of participants who had difficulties and the degree of difficulty with specific aspects of following a GF diet.

### Purchasing gluten free food

Finding GF foods was difficult at some stage for 78% of respondents, but for most this was not a frequently occurring problem. A few (2%) found it difficult all the time, 7% most of the time, and 69% some of the time. The quality of GF food was considered to be a problem. One-fifth (20%) of respondents found it difficult to find good quality GF foods all or most of the time, and 59% found it difficult some of the time. More than three-quarters (78%) experienced some difficulty identifying whether foods were GF or not from food

labels. While 85% reported that they never had to compromise a GFD due to the cost of food, 6% ate gluten containing foods occasionally due to cost.

A PHARMAC Special Authority number enables subsidised GF food (flour and pasta) via prescription and is available for people with biopsy proven CD. At least 51% of the respondents had a special authority number. Some people (n= 126) reported that they were not sure whether they had a special authority number or not.

**Travel**

About one-third (34%) of respondents avoided travelling due to CD at least some of the time, with 1% all the time and 8% most of the time. To make travelling easier many people (89%) took their own GF food with them at least some of the time, while 5% never took GF foods.

**Dining out**

Almost one-quarter (24%) never ate at restaurants all or most of the time due to CD, and one-half avoided restaurants some of the time. Two-fifths (39%) of participants reported not being invited out for meals due to their CD at least some of the time. Being hospitalised and relying on hospital food was a concern for all or most of the time for 12%.

**Table 9: Adult special coeliac quality of life issues**

| | Number of responses | All of the time n (%) | Most of the time n (%) | Some of the time n (%) | Never n (%) |
|---|---|---|---|---|---|
| I (we) avoided restaurants because of coeliac disease. | 880 | 40 (4.6) | 185 (21.0) | 463 (52.6) | 192 (21.8) |
| I (we avoided travelling because of coeliac disease. | 864 | 13 (1.5) | 71 (8.2) | 230 (26.6) | 550 (63.7) |
| I (we) brought gluten-free food when I (we) travelled. | 873 | 372 (42.6) | 240 (27.5) | 211 (24.2) | 50 (75.8) |
| I was (we were) not invited out for meals because of coeliac disease. | 857 | 12 (1.4) | 52 (6.1) | 296 (34.5) | 497 (58) |
| I (we) worried about staying in hospital because of coeliac disease. | 835 | 53 (6.4) | 57 (6.8) | 141 (16.9) | 584 (69.9) |
| I (we) found it difficult to find gluten-free food in the stores. | 876 | 19 (2.2) | 63 (7.2) | 647 (73.9) | 147 (16.8) |
| I (we) found it difficult to find good quality gluten-free food. | 868 | 34 (3.9) | 153 (17.6) | 550 (63.4) | 131 (15.1) |
| I ate gluten-containing food because I (we) could not afford gluten-free food. | 861 | 4 (0.5) | 3 (0.4) | 55 (6.4) | 799 (92.8) |
| I (we) found it difficult to determine if foods were gluten-free from reading their labels. | 877 | 15 (1.7) | 69 (7.9) | 642 (73.2) | 151 (17.2) |

**Challenges with adopting and following a gluten free diet**

Most participants provided additional comments regarding the challenges of their current diet, and these are consistent with the quantitative data and are summarised as follows. Following a GFD required significant changes to household food stores with the restocking of foods that were gluten free at the time of diagnosis being an expensive process. A greater awareness about food and risk of gluten contamination was required, particularly when eating out or travelling. Thus people tended to prepare their own food and eat at home. Family support was considered important for maintaining a GFD.

A GFD was described to include more natural and basic foods, with pre-prepared processed GF foods being described as being more refined, calorie dense and lower in fibre. Some people reported eating a diet free of lactose or dairy also. While few people had gluten containing treats occasionally, the majority went by the motto “If in doubt, leave it out”, the health consequences and symptoms of consuming gluten were such that people did their best to avoid gluten.

Common difficulties that were described included, locating and identifying GF products, difficulty in eating out either at restaurants or friends houses, and the cost of GF food. Many people also commented that it was very difficult to follow a GFD in the past due to poor availability and less choice of GF products, but it is less difficult these days.

Issues relating to purchasing GF food products other than the cost were limited food choices in supermarkets and restaurants, trouble with finding uncontaminated GF products, hidden gluten in products labelled GF, substandard food products, a lack of fibre in GF food products, label reading and working out if a product was GF or not and the taste of GF products.

Many people stated that eating out was the main reason that following a GFD was difficult. There were many reasons for this. Firstly when eating out there is often a lack of GF options (sometimes none). Participants emphasised that lunch options tended to be the worst when eating out, and GF options tended to be more expensive. Secondly, when there are gluten free options available people have to trust that these have been prepared in the correct manner and with the correct ingredients to avoid gluten contamination. When manufacturers and cooks do not have a correct understanding of CD and GF food preparation and cooking this can be a problem. Eating out is often necessary when travelling and when attending social gatherings. Many people mentioned travelling away from home is difficult as there is a reliance on eating out and there can be limited options or a lack of GF food available. Socially when eating at friends' houses there is a often difficulty if the hosts do not understand a GFD or do not prepare food in such a way to avoid contamination. At social occasions or functions some people commented that they choose to bring their own food, but this takes time to organise and advance planning.

Factors that made eating a GF diet less difficult were family, college and partner support, and accepting that having CD was a fact of life and required adjusting their dietary intake accordingly. People found it easier to make their own food at home, in particular bread. Advice from appropriately knowledgeable health professionals, knowing how to read ingredient labels and increasing their own knowledge through support groups and books helped make adapting to this lifestyle easier.

**Associated Medical Conditions**

Table 10 lists the associated medical conditions that those with CD also suffer from. Despite this being an optional section in the questionnaire some participants chose to answer it. The most

frequently reported diseases were migraine headaches (21.3%), depression (16.9%), eczema (15.7%) and asthma (15.3%), and the most frequently reported nutrient deficiencies were iron deficiency anaemia (39.7%),. Vitamin B12 deficiency anaemia (18.7%), and folate deficiency anaemia (15.9%). While there was no questions about Vitamin D status and calcium status, 28% reported that they had had a previous fractured bone. The most common fracture site was wrist (44%), followed by feet and ankles (39%). About one-fifth had osteopaenia or osteoporosis. Among these people, the most common medications taken for these conditions were vitamin D (15%) and calcium (13%). Other medications taken were Alendronate, Etidronate, Calcitonin, and Hormone Replacement Therapy.

**Vitamin Deficiencies**

The vitamin and mineral deficiencies commonly observed in populations with CD were also observed in this study. These included iron deficiency anemia (39.7%), folate deficiency anemia (15.9%), vitamin B12 deficiency anemia (18.7%) and vitamin K (abnormal coagulation) (not investigated in this study). In the long term decreased absorption of vitamin D and calcium can occur bone health was investigated as an effect of this.

**Table 10: The prevalence of other diseases among those with CD**

| MEDICAL CONDITIONS | n (%) |
|---|---|
| **GASTROINTESTINAL AND URINARY** | |
| Colitis | 30 (3.2) |
| Crohn's disease | 10 (1.1) |
| Primary billiary cirrhosis | 4 (0.4) |
| Collagenous colitis | 6 (0.6) |
| Irritable bowel syndrome | 115 (12.3) |
| Stomach/duodenal ulcers | 41 (4.4) |

| | | |
|---|---|---|
| Lactose Intolerance | 111 | (11.9) |
| **CANCER** | | |
| Mouth | 1 | (0.1) |
| Oesophagus | 0 | |
| Stomach | 3 | (0.3) |
| Small Intestine | 3 | (0.3) |
| Colon | 13 | (1.4) |
| Lymphoma | 4 | (0.4) |
| Skin | 33 | (3.5) |
| Melanoma | 19 | (2.0) |
| Lung | 0 | |
| Breast | 22 | (2.4) |
| **BENIGN TUMOR OF GASTROINTESTINAL TRACT** | | |
| Colon polyps | 84 | (9.0) |
| **IMMUNE SYSTEM** | | |
| Auto-immune hepatitis | 13 | (1.4) |
| IgA deficiency | 37 | (4.0) |
| Multiple sclerosis | 1 | (0.1) |
| Lupus | 12 | (1.3) |
| Refractory coeliac disease | 29 | (3.1) |
| Sjogren's syndrome | 8 | (0.9) |
| Food allergies | 136 | (14.5) |
| **BLOOD** | | |
| Iron deficiency anaemia | 372 | (39.7) |
| Folate deficiency anaemia | 149 | (15.9) |
| Vitamin B12 deficiency anaemia | 175 | (18.7) |
| Hyposlenism | 3 | (0.3) |
| **ENDOCRINE** | | |
| Addison's disease | 1 | (0.1) |
| Type 1 diabetes | 16 | (1.7) |
| Type 2 diabetes | 15 | (1.6) |
| Hypothyroidism | 67 | (7.2) |
| Thyroiditis | 34 | (3.6) |
| Turner's syndrome | 1 | (0.1) |
| **NERVOUS SYSTEM** | | |
| Migraine headaches | 199 | (21.3) |

| | | |
|---|---|---|
| Peripheral neuropathy | 18 | (1.9) |
| Epilepsy | 11 | (1.2) |
| Depression | 158 | (16.9) |
| Alzheimer's disease | 0 | |
| Attention deficit disorder (ADHD/ADD) | 1 | (0.1) |
| Multiple sclerosis | 1 | (0.1) |
| Schizophrenia | 9 | (1.0) |
| Autism | 0 | |
| Down's syndrome | 4 | (0.4) |
| Ataxia | 3 | (0.3) |
| **MUSCULOSKELETAL** | | |
| Fibromyalgia | 26 | (2.8) |
| Sarcoidosis | 5 | (0.5) |
| Osteoarthritis | 79 | (8.4) |
| Rheumatoid arthritis | 28 | (3.0) |
| Osteopaenia | 52 | (5.6) |
| Osteoporosis | 92 | (9.8) |
| Raynaud's syndrome | 41 | (4.4) |
| Sacroileitus | 5 | (0.5) |
| Scleroderma | 1 | (0.1) |
| **PEDIATRIC** | | |
| Failure to thrive | 62 | (6.6) |
| Short stature | 72 | (7.7) |
| Dental enamel defects | 112 | (12.0) |
| Delayed puberty | 33 | (3.5) |
| **OTHER** | | |
| Dermatitis herpetiformis9 | 84 | (9.0) |
| Alopecia areata (patchy hair loss) | 26 | (2.8) |
| Eczema | 147 | (15.7) |
| Psoriasis | 52 | (5.6) |
| Pneumonia | 53 | (5.7) |
| Asthma | 143 | (15.3) |
| Cystic fibrosis | 1 | (0.1) |
| High Cholesterol | 115 | (12.3) |
| High blood pressure | 124 | (13.2) |
| Loss of vision | 57 | (6.1) |

### Coeliac Disease Symptoms among First-Degree Relatives

Symptoms suggestive of CD were common among respondents' families. 14% had a mother, 12% had a father, 24% had a sibling, 20% had a child, 14% had a grandparent and 7% had a grandchild with symptoms suggestive of CD. Overall 20% of participants had at least one relative, and 8% had multiple relations who consumed a GF diet due to having CD symptoms. It was not possible to ascertain how many relatives had biopsy proven CD or DH, because it was clear from the completed questionnaires that many participants answered the question by listing all first degree relative, not only those with biopsy proven CD or DH as asked.

### Patient Recommendations

Participants were asked to choose what two items (out of five) would contribute most to improving the lives of those with CD. Table 11 shows the most common items that were selected. The two factors which participants believed would contribute the most to improving the lives of individuals with CD were earlier diagnosis of CD (55%) and better labelling of gluten-containing ingredients in foods. One-quarter felt better dietary counselling, particularly at the time of diagnosis would be beneficial.

**Table 11: Recommendations from those with CD**

| Recommendation | n (%) |
|---|---|
| Earlier Diagnosis of coeliac disease | 519 (55.4) |
| Better labeling of gluten-containing ingredients in foods | 451 (48.2) |
| More gluten-free foods in the supermarket | 403 (43.1) |
| Gluten-free choices on restaurant menus | 352 (37.6) |
| Better dietary counseling, especially for newly diagnosed coeliac patients | 233 (24.9) |

When asked for other suggestions, the following themes were identified. First and foremost cheaper GF food was wanted or more financial assistance or subsidies on GF foods, particularly cereal foods such as cereal, pasta and bread, as GF food is more expensive than gluten containing foods. Better quality, range, and availability of GF foods was also desired. In particular, places such as airports and train stations were reported to be lack GF food options. The next most common issue was the fact there is a general lack of awareness and understanding about CD and GF diets. Better understanding by doctors, the food industry, family, friends, workmates, and in particular foodservice chefs and staff was often suggested. Many problems arose when eating out due to incorrect preparation of food, gluten contamination, being served incorrect food, gluten containing food being added to a gluten free meal, and a misunderstanding about how much gluten was permissible for those who request GF meals. Better labelling, including bigger print was suggested.

In terms of diagnosis and treatment, screening for CD in families where CD is already present was suggested, and increasing efforts to shorten the length of time to diagnosis of CD in those who have it and are yet undiagnosed. Better dietary counselling when first diagnosed was recommended along with better follow up. Those who had seem a dietitian or nutritionist reported that they already knew the information that they received, as often their initial appointment was a considerable time after their diagnosis, and they had had to seek necessary information from elsewhere. A particular point made by a couple of people was that education is needed about not commencing a GF diet until a firm diagnosis has been made as this can lead to false biopsy test results. Lastly a cure or a vaccine appeared to be much anticipated by the coeliac community.

## 6.0 DISCUSSION

The New Zealand Coeliac Survey is the first national survey of people with CD in New Zealand. It was timely to do this survey as the prevalence of CD is increasing worldwide and knowledge about this multisystem disorder, its varying clinical presentations and gluten free food production has increased substantially over the last 10-15 years. As the treatment of CD is entirely dietary, the results of this survey are highly relevant to dietary practice in New Zealand. This thesis sought to describe the health of people with CD in New Zealand, the impact of a gluten free diet, the extent of on-going symptomatic CD and reliable sources of information and support.

The mean age at diagnosis of CD in this study was 43.5 years. Most participants presented with classical symptoms of CD, but there was a considerable delay between the onset of symptoms and the diagnosis of CD. CD can be confused with other conditions and misdiagnosed, and this study showed CD was commonly misdiagnosed as anaemia, stress/nerves/depression, Irritable Bowel Syndrome, and vitamin deficiencies as well as some less common disorders. Gastroenterologists and GPs were consulted and contributed synergistically to a diagnosis of CD. Coeliac New Zealand Incorporated was the most important source of information and support for those with CD as expected, as the study population were members of this society. The only treatment for CD is nutritional, but not all respondents in this study were referred to a dietitian or nutritionist upon diagnosis, and a few reported not even being told they needed to follow a GFD at the time of diagnosis. Despite this, all respondents followed a GFD to some extent and this resulted in the improvement of health for almost all people. CD impacts on quality of life, but overall appeared to be manageable. Key issues identified that would improve quality of life were

more available good quality GF food for purchasing and when eating out. Key participant recommendations were earlier diagnosis of CD, and better labelling of gluten containing ingredients in foods.

### 6.1 Incidence and prevalence of CD

The incidence and prevalence of CD appears to be increasing worldwide (2, 6, 12, 13). In the New Zealand Coeliac Survey the number of people diagnosed with CD each year appeared to increase over time, particularly over the last 20 years. While this observation may reflect a true increase in the incidence of CD in New Zealand, this study was not designed to assess either the incidence or prevalence of CD. The study population was a self-selected population who chose to be members of Coeliac New Zealand Incorporated. While a few study participants were not members of Coeliac New Zealand Incorporated, an unknown number of people with CD are not members of this society. Thus, it was not possible to ascertain all cases of CD in New Zealand in this study. Further, among respondents the self-reported year of diagnosis was not validated, which would have required checking medical records. Nevertheless, the suggested increase in incidence is consistent with international observations (12).

There were more females than males, which is consistent with CD being more prevalent in females than in males. However, the ratio of females to males was higher than expected. In this survey the ratio was 4.7:1, whereas other New Zealand studies have found the ratio to be about 3:1 (7) and 2.1:1 (13). International studies from the USA and Canada have found similar ratios of 2.9:1 (79) and 3:1 (77). In this study most participants were members of Coeliac New Zealand Incorporated, whose 2720 membership has a greater proportion of women (76%) than men (24%). The membership reflects the widely reported ratio of 3:1, and thus a higher proportion of

women completed this survey than men. Also women may be more likely to seek social support for managing illness in their lives (80).

The number and proportion of Maori and Pacific Island peoples was very low compared with the general New Zealand population. This is consistent with the prevalence of CD being higher in European populations compared with most other ethnic groups (4). As Coeliac New Zealand Incorporated membership has low numbers of Maori and Pacific Island peoples, these survey results may reflect this.

### 6.2 Diagnosis of coeliac disease

A Gastroenterologist most frequently made the diagnosis of CD. In the New Zealand survey 58% of participants reported a gastroenterologist made their diagnosis of CD, whereas the proportion was higher in the Canadian and US surveys (77% in both) (77, 79). As a duodenal biopsy is required to make a diagnosis of CD, and only patients with biopsy proven CD were included in all 3 studies, it is surprising that more New Zealand participants did not report a gastroenterologist making their diagnosis. This less than expected proportion may reflect the increasing use of screening tests (36), and other health professionals making a diagnosis of CD based on serological testing with a gastroenterologist confirming the diagnosis.

#### Age at diagnosis

The mean age at diagnosis in the New Zealand Coeliac Health Survey was 43.5 years, which was three years younger than that found in the Canadian Coeliac Health Survey (46 years) (77). Males were older when they were diagnosed with CD (45.9 years) compared with females (42.8

years). This observation is consistent with the Canadian survey, but Canadian participants were on average 2 years older (males - 48 years; females - 45 years). Results of a survey of 1138 US adults aged 18 years and over with biopsy proven CD did not state the mean age at diagnosis, but most people were diagnosed between 40 and 60 years (79).

**Delay in diagnosis**

The mean time delay from when symptoms first appeared to when CD was diagnosed was 11.6 years. This is similar to findings from Canada (77) and the USA (10), where the mean time delay in diagnosis was 11.7 years and 11.0 years, respectively. The mean time delay was more in the UK (13.2 years), (81) and a little less in Germany (10.1 years) (82) and Sweden (9.7 years). (83). In a relatively old case study report of two New Zealand patients, one experienced a delay of 10 years and the other 36 years in diagnosis (84). Despite an increased awareness of CD, the Canadian survey found there was no difference in the time delay in diagnosis between those diagnosed during the 5 year period, 1998 - 2003, and those diagnosed before 1998.

Most of the delay in diagnosis occurs following consultation with a doctor. A German study found that while people with symptoms consulted a doctor, the time taken for the doctor to reach a diagnosis was the greatest proportion of the time delay when compared with the time from onset of symptoms to the first visit to a doctor (82). The mean time delay for a diagnosis was 10.1 years, with the mean time to seek medical attention being 2.2 years, and the mean time between first doctor consultation and diagnosis being 8 years. This finding has also been observed in Sweden where the mean delay to diagnosis from the onset of symptoms was 9.7 years, and from the first doctor visit to diagnosis was 5.8 years. (83). Patients with CD can be

referred to hospital clinics multiple times, and received incorrect diagnoses before a correct diagnosis is eventually made (85).

A common conclusion about this problem of delayed diagnosis of CD is that it has been considered a rare disease in the past, and so may not be at the forefront of a physician's mind when making a diagnosis. However, with increasing prevalence and the availability of serological tests, this situation is likely to change. There is some recent evidence that suggests the time from the onset of symptoms to diagnosis has decreased over time. In a recent study in the USA, the delay in diagnosis before 1981 was 11 years and it had decreased to 4 years in patients diagnosed after 2000 (63).

Another factor likely to contribute to delays in diagnosis is the atypical presentation (63, 86, 87). CD presents with a wide range of non-gastrointestinal symptoms, for example neurological symptoms (86), (77), and many other diseases may be considered before CD. In this study CD was frequently misdiagnosed as anaemia, severe stress, nerves or depression, irritable bowel syndrome, or a vitamin deficiency. These five diagnoses were also the most common diagnoses made prior to a correct diagnosis of CD in both Canada and the USA (77, 79). Further, if a patient initiates a GF diet prior to a diagnosis, this may further complicate the process of diagnosis (86).

Timely diagnosis of CD is important as untreated CD results in poor health-related quality of life, (81, 83). Although the New Zealand CD survey did not assess health-related quality of life, the delay in diagnosis was considered to be the top priority to be addressed by participants with 55% reporting an earlier diagnosis would have made their lives better. A similar proportion of Canadians (61%) also considered earlier diagnosis to be important (77). Quality of life improves

notably after initiating a GFD (81, 88). General health and wellbeing, anxiety and depression often markedly improve after 12 months despite the challenges of adopting a GFD (33, 35, 88, 89). In general, improvements in quality of life seem to be much more marked in those with clinical CD as opposed to those with asymptomatic CD (89), although Mustakahti (2002) noted quality of life improvements in both groups (88).

In addition to quality of life during the pre-diagnosis period, timely diagnosis is important because the longer a person has untreated CD, the more gastrointestinal damage occurs and the greater the risk of future health complications, in particular other autoimmune diseases and malignancies. The prevalence of autoimmune disorders in those with CD has been found to be related to the duration of exposure to gluten, as the prevalence of autoimmune disorders, including type 1 diabetes and thyroid disease, increases with increasing age at diagnosis of CD (90). In this study thyroid disease was relatively common with 10% reporting either hypothyroidism or thyroiditis. Few people had type 1 diabetes (2%). There is also an increased risk of malignancies (particularly intestinal lymphoma) in people with CD compared with people without CD, and most of these cancers occur before the diagnosis of CD (84, 91). Malignancy was found in 9.3% of patients in a USA study (63). The 35 malignancies were diagnosed before CD was diagnosed, 5 malignancies were diagnosed during diagnosis of CD and 15 were diagnosed after the diagnosis of CD. Lymphoma was reported by four people and a small intestinal cancer was reported by three people in this study. Other cancers were relatively common, but comparisons with the general population were not undertaken in this study.

### 6.3 Presenting symptoms

The most common presenting symptoms of CD in this survey were bloating, gas and abdominal pain (76%), extreme weakness or tiredness (67%), diarrhoea (64%), anaemia (56%), and weight loss (49%). These symptoms were also the most common symptoms reported in the Canadian Celiac Health Survey (77) and a UK survey (81), and are consistent with the classical presentation of CD (27). Some atypical symptoms were also relatively common in each of the New Zealand, Canadian and UK surveys. In these 3 surveys between 25% and 40% of participants reported each of the following symptoms: joint pain, skin rashes, mood swings or depression, and headaches or migraines.

Other studies have reported similar results. In the United States, in a case review the clinical features of 227 people with biopsy-proven CD were investigated (87). Overall, at diagnosis 62% had classical gastrointestinal symptoms, diarrhoea and weight loss. Other modes of presentation included anaemia, reduced bone density, screening of first degree relatives, and incidental diagnosis at endoscopy. This study also examined whether presenting symptoms had changed over time. They observed that 73% of those diagnosed before 1993 had diarrhoea compared with 43% diagnosed after 1993 ($P = 0.0001$).

### 6.4 Ongoing symptoms

Most participants (96%) found their health improved to some extent after commencing a GFD, but for many there was a concerning incomplete recovery from each of the different symptoms experienced prior to the diagnosis of CD following initiation of a GF diet. Indeed 2% found their health did not improve at all following commencement of a GF diet. The significance of ongoing symptoms and accidental ingestion of gluten is difficult to assess in terms of health outcomes. It

is not known at what level and at what frequency of gluten intake causes long term damage, and is a highly topical area of research (92). However, the longer a person with CD is exposed to gluten, the more likely long term damage will occur (91, 93). Whether long term damage occurs or not, continuing symptoms impacts on the quality of life for those with CD (81).

The symptom with the highest proportion of participants reporting full recovery was weight loss. 49% of participants reported weight loss at the time of diagnosis, and 76% of these participants reported full recovery with treatment. Weight loss was also the symptom with the highest recovery rate in the Canadian survey (77). 69% of Canadian survey participants reported weight loss prior to diagnosis with 84% of reporting full recovery with a GF diet. For the more common typical CD symptoms (bloating, gas and abdominal pain, extreme weakness or tiredness, and diarrhoea), slightly more than half reported full recovery. Recovery rates were less than half for most of the other more atypical symptoms.

These low recovery rates are surprising, as 92% reported following a strict GF diet. This is similar to that reported in the Canadian survey (90%) (77). The low recovery rates suggest continued consumption of gluten, whether deliberate or inadvertent. Inadvertent consumption of gluten has been identified as a key reason for ongoing symptoms (94). In this survey 93% reported accidentally consuming gluten. Most (76%) were aware that they had inadvertently consumed gluten less frequently than monthly, but at least annually, while 12% were aware of accidental gluten consumption at least monthly and 5% at least weekly. Participants were aware that when eating out there was a high risk of consuming gluten contaminated food, and many avoided these social situations, but other sources of gluten contamination and hidden gluten may be less readily identified. Further, not all people and their families may be aware of all hidden

sources of gluten in the diet, and hence the importance of referral to a dietitian with expertise in CD at diagnois for education and follow up (95).

Although unlikely to be a major reason, the low recovery rates in this study may also be partly explained by a the few participants who reported what symptoms they had at diagnosis, but did not indicate whether they had fully recovered or not. A few more participants may have been diagnosed with CD close to the time of completing the survey and there had been insufficient time for full recovery from symptoms. For a small number of people, there may be other clinical reasons for ongoing symptoms, including co-existent disorders such as irritable bowel syndrome and lactose tolerance (96). In this survey 18% of participants reported co-existing lactose intolerance and only 38% had completely recovered.

### 6.5 Treatment – the Gluten Free Diet and Education

The cornerstone treatment for CD is a GF diet, and adopting such a diet is imperative for cessation of symptoms and decreasing the risk of developing associated conditions such as malignancies and autoimmune disorders. Prompt referral to a dietitian for education following diagnosis is important for the successful adoption of a GF diet, but this does not always occur. In this survey 94% of respondents were told they needed to follow a GF diet for life, but only 82% were referred to a dietitian. Some participants' Gastroenterologist, GP or other health professional may have taken the role of dietary education and follow up. However, this is not ideal as they are not likely to have adequate time, resources and dietary expertise to provide adequate education. Indeed in this survey, the quality of the information provided by health professionals was often perceived to be inadequate. 14% seeking information from a gastroenterologist, 19% from a dietitian, 30% from a GP, and 44% from an alternative medical

professional perceived the information to be fair or poor. In the New Zealand CD survey 91% of participants obtained information about their disease from the national patient support group Coeliac New Zealand Incorporated. However, in some respects this is not surprising as the organisation's membership list was used for this survey. Other common sources of information about CD were dietitian/nutritionist (75%), cookbooks (69%), gastroenterologist (61%), the internet (58%), newspapers and magazines (52%) and other people with CD (51%). The quality of the information sourced was variable with Coeliac New Zealand Incorporated providing the most highly rated information, which was considered to be excellent or very good by 91%. Other highly rated sources of information were other people with CD (72%), regional coeliac groups (71%), gastroenterologists (66%), the internet (66%) and cookbooks (65%).

Like New Zealanders, most people with CD in the US acquire information about their disease from Coeliac support groups, books, the internet, family and friends with only fewer than 20% seeking information from their primary physician or dietitian (79).

In the absence of good accessible professional dietary information, it appears people with CD are relying on alternative sources of information, which may or may not be ideal. Information from questionable sources such as the internet or from a friend, may be false, outdated or conflict with other information (49). In practice this means the person with CD could ingest gluten containing foods unknowingly and restrict more foods than they need to and therefore necessary nutrients and energy (49). Any conflicting information could potentially lead to uncertainty about a good balance diet and add more stress to the already difficult time of changing their lifestyle to accommodate a GF diet.

### 6.6 Dietetic Advice

Dietitians are essential in the management of CD (49, 95, 97, 98). The dietitian is critical for an appropriate nutritional assessment, treatment of nutritional deficiencies, and education –both of the person with CD and their support network (family). Dietitians also rationalise that understanding CD and its consequences will motivate those with CD to manage it correctly, and that a dietitian is essential to provide the tools for them to be able to do this. Dietitians must also be aware of the process and content that needs to be used in the education of CD. Nutritional education alone will not suffice as CD has a large emotional and psychological impact on the person and affects their quality of life (49). Thus strategies for coping must also be included in the education.

In this survey particular dietary issues that were emphasised by participants were difficulty finding good quality GF food, difficulty identifying GF food from the food product label, finding gluten free food in stores, and not being invited out and avoiding restaurants due to gluten free requirements. This suggests that dietitians have a role to address some of these ongoing issues and challenges, particularly advising on quality and sources of GF food and label reading.

While dietetic expertise is necessary for the treatment of CD, dietitians do not necessarily have the appropriate knowledge. This was evident in this New Zealand survey, and has been observed elsewhere. In Lee and Newman's survey, (34) among the 13% who received dietary information from a dietitian, only 21% considered the information to be helpful. A similar US study reported that 53% of those who saw a dietitian thought the sessions were not helpful, and 54% considered the dietitian was not knowledgeable about CD (99).

### 6.7 Issues beyond the home

Avoiding restaurants and travelling were common lifestyle issues in New Zealand as well as Canada. In Canada 81% avoided going to restaurants some or most of the time, 38% avoided travelling some or most of the time and 94% brought GF foods with them when travelling. The New Zealand survey findings were very similar, as are findings from the US. In New York people with CD or gluten sensitivity ate take-out or restaurant food, and at people's houses significantly less often than the general public (100). Most found eating food considered to be 'gluten free' in restaurants was a problem and 63% avoided restaurants due to trying to adhere to a GFD.

While those with CD still want the option of eating out either from food outlets or at private houses, a large level of risk is involved as those who prepare GF food may not understand CD and the requirements of GF food preparation. It is important that food manufacturers, restaurant managers and their staff are appropriately educated regarding the provision of GF foods for those with CD. FSANZ has standards and procedures for producing and monitoring GF food. Those producing GF foods need to be aware of and follow these standards. FSANZ also provides training and resources to educate and promote adherence to these standards. However, there is no regular monitoring of adherence to these standards, and there is reliance on the public making complaints to appropriate authorities. Provision of inappropriately labelled 'gluten-free' food does occur in New Zealand. An Oamaru baker was warned by the Commerce Commission in 2011(101).

### 6.8 Financial

Living with CD and eating a GFD is comparatively more expensive than a normal gluten containing diet (102). Among survey participants many would like GF foods to be cheaper. In New Zealand financial support is available through a PHARMAC special authority number or a disability allowance. A special authority number partially subsidises a small number of GF foods which are traditionally more expensive than the gluten containing counterparts. These are GF flour and pasta. To be eligible an individual must have biopsy proven CD (52). While a child disability allowance is not income tested an adult disability allowance is income tested (52). Half of the respondents had a special authority number, and only a few had a disability allowance. Some people reported that the subsidy obtained through a special authority number was not worth it.

### 6.9 Study Strengths and Limitations

An important strength of this study is the relatively large number of responses (1264), the data from which has provided good information about the health of people with CD in New Zealand. Until this survey there was limited information of this kind in New Zealand and the findings have highlighted some concerns in the diagnosis and treatment of CD. This survey used a validated questionnaire developed in Canada, and was adapted slightly for the New Zealand setting. The key results from this study are similar to those from the Canadian survey (77) and similar to those from surveys in the USA (79), UK (81) and Sweden (83). Thus, the findings of this study can be used with confidence to inform health professional practice, and direct future research in New Zealand.

The response rate was 46.5%, which is less than ideal, but better than the response rate (40%) in a recent survey of members of Coeliac UK. The Canadian Coeliac Health Survey was sent to a membership of 3408 and the response rate was 65%. Efforts to improve the response rate in this study included two email reminders and a chance to win a fifty dollar book voucher was offered for completing the survey. It has been shown response rates increase when an incentive is offered (103). For those who did not have an email address and to whom a postal questionnaire was sent, a follow-up reminder letter was not sent. If this had been done, the response rate may have been slightly better. While it was not possible to ascertain the characteristics of those who did not respond, it was possible to compare the characteristics of the respondents with the characteristics of all those who were sent the survey. The proportion of males and females were very similar. The proportion of males and females who were responded were 22% and 78%, respectively, compared with 24% and 76%, respectively for the entire New Zealand Coeliac Society membership. The age distribution for the membership and the study participants were similar. While the demographics of the membership and the study participants were similar, those who responded may have been different in other ways such as more concerned about their health, and thus introduce a response bias.

The study conveniently surveyed members of Coeliac New Zealand Incorporated. As members may be different in some way from non-members, there may be a selection bias and the results may or may not be representative of the entire population of New Zealanders with CD. Those who belong to this society may be more active in the management of their CD, better able to manage their disease due to the support they receive, better educated regarding CD, and have

been diagnosed with CD for a shorter time than those who don't or no longer belong to Coeliac New Zealand Incorporated. It is also plausible that non-members confidently manage their CD.

The survey was self administered and there were no validation checks of medical details. Self-reporting can introduce recall bias. Also respondents may have over-reported the good (e.g. sticking to a GFD) and under-reported the things they perceive as bad (e.g. voluntary consumption of gluten). However, the effect of this is likely to be minimal as the survey was anonymous and names were only recorded if the participant decided to enter the draw for a book voucher.

The survey had two modes of administration, online and on paper. While this allowed the survey to be administered more easily at a lower cost, and cater for those with and without computers and internet access, the formatting of each of these was slightly different, which may have affected the way respondents answered questions, particularly the online version. The online version had some initial unexpected technical errors (despite testing), which may have introduced errors, as a small number of people had to redo the survey. Data entered for the paper surveys were double checked periodically to minimise data entry errors, but errors may still have occurred.

The Canadian Celiac Health Survey was used and adjusted for a New Zealand audience. While the New Zealand version was pre-tested, this was limited due to the extensive piloting done on the original survey. However, it was apparent that one or two questions were ambiguous resulting in these questions being answered incorrectly by a few. The effect of this on the results is likely to be small.

In conclusion, CD is a common condition in New Zealand, and it appears that the incidence is increasing worldwide, including in New Zealand. The disease is often misdiagnosed with the diagnosis being delayed for up to 12 years despite patients frequently consulting a doctor, and having a reduced quality of life. Once diagnosed not all patients are referred to a dietitian as is recommended internationally, and when referred to a dietitian 19% of patients considered information received to be of fair or poor quality. Despite best efforts, accidental ingestion of gluten appears to be a problem with most people having ongoing symptoms to varying degrees. This may relate to inadequate dietary education, contaminated food products, or co-existing conditions. Given the significant delays in the diagnosis of CD, and the fact that a GFD is the only available treatment for CD, there needs to be greater awareness among health professionals of CD symptoms and its atypical presentation, improved knowledge about CD and its dietary treatment among dietitians, and improved knowledge among those preparing and manufacturing gluten free foods.

## 8.0 RECOMMENDATIONS/ APPLICATION TO DIETETIC PRACTICE

- As the diagnosis of CD increases more people will be referred to dietitians for dietary advice regarding a GFD. There needs to be sufficient appropriately skilled and knowledgeable dietitians to provide a timely service.
- As there are often significant delays, up to many years, in the diagnosis of CD, dietitans treating these patients need to be aware of medical conditions and nutritional deficiencies associated with untreated CD. They also need to take the person's current wellbeing and quality of life into account in regards to motivation and practicality when prescribing a GFD. Understanding and sympathy is critical as many patients will not be enjoying optimal health and wellbeing immediately post diagnosis.
- Iron deficiency anaemia is common and needs to be treated through increasing dietary iron and iron absorption capacity, and in some cases with iron supplementation.
- Symptoms can be ongoing due to a lengthy recovery or accidental ingestion of gluten. If continual accidental consumption of gluten is suspected a dietitian can through education/re-education on label reading, familiarisation with hidden gluten sources, and possibly an elimination diet.
- Coeliac New Zealand Incorporated is an important support and educational resource for people with CD. It is part of the dietitian's role to refer the patient to this support source.
- A dietitan should be aware of the source of financial support available to those with CD and direct the patient to the appropriate channels to apply for this. When giving GF dietary advice, dietitians need to make sure their advice is financial viable to the patient.

This may involve money saving tips, a choice of cheaper GF foods and practical ways to make a GFD more affordable.

- To promote GFD compliance and avoidance of accidental gluten consumption dietitians should provide education so people with CD can readily identify both obvious and hidden gluten in food products.
- Many patients acquire information from questionable sources such as the internet or from a friend, and the accuracy of this information cannot be guaranteed. This may cause a patient to ingest gluten without knowing or restrict more foods than they need to and therefore nutrients and energy. Conflicting information can result in the patient being confused and stressed. Dietitians need to be aware of the process and content that needs to be used in the education of CD. Strategies for coping must also be included.
- Dietitian can lobby FSANZ for label change legislation, or alternatively, and probably more feasible in the short term, more time and effort could be devoted to label reading education.
- Strategies and methods for eating away from the home should be included in GFD education so people with CD need not suffer by avoiding these situations or through accidental ingestion of gluten. This may include providing lists of restaurants and dishes which are established to be reliably GF, recipes that are easy to prepare, suggestions for making travel easier, and guidelines for foods to look out for and foods to avoid in situations outside the home.

**REFERENCES:**

1. Thompson T, Méndez E. Commercial Assays to Assess Gluten Content of Gluten-Free Foods: Why They Are Not Created Equal. Journal of the American Dietetic Association. 2008;108(10):1682-7.

2. Sapone A, Bai J, Ciacci C, Dolinsek J, Green P, Hadjivassiliou M, et al. Spectrum of gluten-related disorders: consensus on new nomenclature and classification. BMC Medicine. 2012;10(1):13.

3. Holtmeier W, Caspary W. Celiac disease. Orphanet Journal of Rare Diseases. 2006;1(1):1-8.

4. da Silva Neves M, González-Garcia M, Nouws H, Delerue-Matos C, Santos-Silva A, Costa-García A. Celiac disease diagnosis and gluten-free food analytical control. Analytical and Bioanalytical Chemistry. 2010;397(5):1743-53.

5. Shahbazkhani B, Malekzadeh R, Sotoudeh M, Moghadam KF, Farhadi M, Ansari R, et al. High prevalence of coeliac disease in apparently healthy Iranian blood donors. European journal of gastroenterology & hepatology. 2003;15(5):475-8. Epub 2003/04/19.

6. Lohi S, Mustalahti K, Kaukinen K, Laurila K, Collin P, Rissanen H, et al. Increasing prevalence of coeliac disease over time. Alimentary pharmacology & therapeutics. 2007;26(9):1217-25. Epub 2007/10/20.

7. Ussher R, Yeong ML, Stace N. Coeliac disease: incidence and prevalence in Wellington 1985-92. The New Zealand medical journal. 1994;107(978):195-7. Epub 1994/05/25.

8. Mylotte M, Egan-Mitchell B, McCarthy CF, McNicholl B. Incidence of coeliac disease in the West of Ireland. British medical journal. 1973;1(5855):703-5. Epub 1973/03/24.

9. Ertekin V, Selimoglu MA, Kardas F, Aktas E. Prevalence of celiac disease in Turkish children. Journal of clinical gastroenterology. 2005;39(8):689-91. Epub 2005/08/06.

10. Fasano A, Berti I, Gerarduzzi T, Not T, Colletti RB, Drago S, et al. Prevalence of celiac disease in at-risk and not-at-risk groups in the United States: a large multicenter study. Archives of internal medicine. 2003;163(3):286-92. Epub 2003/02/13.

11. Ivarsson A, Persson LA, Nystrom L, Ascher H, Cavell B, Danielsson L, et al. Epidemic of coeliac disease in Swedish children. Acta paediatrica (Oslo, Norway : 1992). 2000;89(2):165-71. Epub 2000/03/10.

12. Cummins AG, Roberts-Thomson IC. Prevalence of celiac disease in the Asia–Pacific region. Journal of Gastroenterology and Hepatology. 2009;24(8):1347-51.

13. Cook B, Oxner R, Chapman B, Whitehead M, Burt M. A thirty-year (1970-1999) study of coeliac disease in the Canterbury region of New Zealand. The New Zealand medical journal. 2004;117(1189):U772. Epub 2004/03/12.

14. Carrington JM, Hewitt CJ, Dowsett LR, Barbezat GO. The prevalence of coeliac disease in Otago. The New Zealand medical journal. 1987;100(828):460-2. Epub 1987/07/22.

15. Cook HB, Burt MJ, Collett JA, Whitehead MR, Frampton CM, Chapman BA. Adult coeliac disease: Prevalence and clinical significance. Journal of Gastroenterology and Hepatology. 2000;15(9):1032-6.

16. Tanpowpong P, Ingham TR, Lampshire PK, Kirchberg FF, Epton MJ, Crane J, et al. Coeliac disease and gluten avoidance in New Zealand children. Archives of disease in childhood. 2012;97(1):12-6. Epub 2011/11/15.

17. Westerbeek E, Mouat S, Wesley A, Chin S. Coeliac disease diagnosed at Starship Children's Hospital: 1999-2002. The New Zealand medical journal. 2005;118(1220):U1613. Epub 2005/09/01.

18. Mee AS, Burke M, Vallon AG, Newman J, Cotton PB. Small bowel biopsy for malabsorption: comparison of the diagnostic adequacy of endoscopic forceps and capsule biopsy specimens. BMJ. 1985;291(6498):769-72.

19. Dickey W, Hughes D. Prevalence of celiac disease and its endoscopic markers among patients having routine upper gastrointestinal endoscopy. The American journal of gastroenterology. 1999;94(8):2182-6. Epub 1999/08/13.

20. Karell K, Louka AS, Moodie SJ, Ascher H, Clot F, Greco L, et al. HLA types in celiac disease patients not carrying the DQA1*05-DQB1*02 (DQ2) heterodimer: results from the European Genetics Cluster on Celiac Disease. Human immunology. 2003;64(4):469-77. Epub 2003/03/26.

21. Megiorni F, Mora B, Bonamico M, Barbato M, Nenna R, Maiella G, et al. HLA-DQ and risk gradient for celiac disease. Human immunology. 2009;70(1):55-9. Epub 2008/11/26.

22. Freeman HJ. Risk factors in familial forms of celiac disease. World journal of gastroenterology : WJG. 2010;16(15):1828-31. Epub 2010/04/17.

23. Dube C, Rostom A, Sy R, Cranney A, Saloojee N, Garritty C, et al. The prevalence of celiac disease in average-risk and at-risk Western European populations: a systematic review. Gastroenterology. 2005;128(4 Suppl 1):S57-67. Epub 2005/04/13.

24. Hill ID, Dirks MH, Liptak GS, Colletti RB, Fasano A, Guandalini S, et al. Guideline for the diagnosis and treatment of celiac disease in children: recommendations of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition. J Pediatr Gastroenterol Nutr. 2005;40(1):1-19.

25. Kagnoff MF. AGA Institute Medical Position Statement on the Diagnosis and Management of Celiac Disease. Gastroenterology. 2006;131(6):1977-80.

26. Green PH. The many faces of celiac disease: clinical presentation of celiac disease in the adult population. Gastroenterology. 2005;128(4 Suppl 1):S74-8. Epub 2005/04/13.

27. Nehra V, Marietta E, Murray J. CELIAC DISEASE. In: Editor-in-Chief: Benjamin C, editor. Encyclopedia of Human Nutrition (Second Edition). Oxford: Elsevier; 2005. p. 407-17.

28. Dickey W. Autoantibodies in Celiac Disease. Autoantibodies Research Progress. 2008:127.

29. Mahan LK, Escott-Stump S. Krause's Food & Nutrition Therapy: Edited by L. Kathleen Mahan and Sylvia Escott-Stump: Elsevier Science Health Science Division; 2008.

30. Green PHR, Cellier C. Celiac disease. New England Journal of Medicine. 2007;357(17):1731-43.

31. Harris LA, Park JY, Voltaggio L, Lam-Himlin D. Celiac disease: clinical, endoscopic, and histopathologic review. Gastrointestinal endoscopy. 2012;76(3):625-40. Epub 2012/08/18.

32. Dewar DH, Ciclitira PJ. Clinical features and diagnosis of celiac disease. Gastroenterology. 2005;128(4):S19-S24.

33. Ciacci C, D'agate C, De Rosa A, Franzese C, Errichiello S, Gasperi V, et al. Self-rated quality of life in celiac disease. Digestive diseases and sciences. 2003;48(11):2216-20.

34. Lee A, Newman JM. Celiac diet: Its impact on quality of life. Journal of the American Dietetic Association. 2003;103(11):1533-5.

35. Casellas F, Rodrigo L, Vivancos JL, Riestra S, Pantiga C, Baudet J, et al. Factors that impact health-related quality of life in adults with celiac disease: a multicenter study. World journal of gastroenterology: WJG. 2008;14(1):46.

36. Fasano A, Catassi C. Current approaches to diagnosis and treatment of celiac disease: an evolving spectrum. Gastroenterology. 2001;120(3):636-51. Epub 2001/02/17.

37. Dieterich W, Ehnis T, Bauer M, Donner P, Volta U, Riecken EO, et al. Identification of tissue transglutaminase as the autoantigen of celiac disease. Nature medicine. 1997;3(7):797-801. Epub 1997/07/01.

38. Kneepkens CM, von Blomberg BM. Clinical practice : coeliac disease. European journal of pediatrics. 2012;171(7):1011-21. Epub 2012/03/17.

39. Lerner A, Kumar V, Iancu TC. Immunological diagnosis of childhood coeliac disease: comparison between antigliadin, antireticulin and antiendomysial antibodies. Clinical and experimental immunology. 1994;95(1):78-82. Epub 1994/01/01.

40. Catassi C, Fasano A. Celiac disease diagnosis: simple rules are better than complicated algorithms. The American journal of medicine. 2010;123(8):691-3. Epub 2010/07/31.

41. McNeish AS, Harms HK, Rey J, Shmerling DH, Visakorpi JK, Walker-Smith JA. The diagnosis of coeliac disease. A commentary on the current practices of members of the European Society for Paediatric Gastroenterology and Nutrition (ESPGAN). Archives of disease in childhood. 1979;54(10):783-6. Epub 1979/10/01.

42. Gislason S. Celiac Disease Immune Mechanisms. West Vancouver, British Columbia, Canada: Environmed Research; [cited 2012 02 December]; Available from: http://alphastore.org/celiac/celiac_mechanisms.htm.

43. Sollid LM, Khosla C. Future therapeutic options for celiac disease. Nature Clinical Practice Gastroenterology & Hepatology. 2005;2(3):140-7.

44. Bayer K. Coeliac disease vaccine being trialled in Christchurch. Otago Daily Times. 2012 October 9, 2012.

45. Adams S. Celiac Disease Alternative Medicine. Celiac.com; 2007 [updated 2 December 2007; cited 2012 13 June]; Available from: http://www.celiac.com/articles/1109/1/Celiac-Disease-Alternative-Medicine/Page1.html.

46. Natural Home Remedies for Celiac Disease. www.tryayurveda.com [updated 2012; cited 2012 13 June]; Available from: http://www.tryayurveda.com/blog/natural-home-remedies-for-celiac-disease/.

47. Cain S. Curing Celiac Disease Using Holistic Naturopathic Techniques. The Health Wyze Report: The Health Wyze Report; 2009 [updated 04 July 2012; cited 2012 13 June];

Available from: http://healthwyze.org/index.php/component/content/article/148-doing-the-impossible-again-naturally-curing-celiac-disease-using-holistic-naturopathic-techniques.html.

48. 2010 Clinical Handbook. 9th ed. Inc DNZ, editor. Wellington: Manor House Press; 2010.

49. Case S. The gluten-free diet: How to provide effective education and resources. Gastroenterology. 2005;128(4, Supplement 1):S128-S34.

50. Kupper C. Dietary guidelines and implementation for celiac disease. Gastroenterology. 2005;128(4, Supplement 1):S121-S7.

51. Coeliac New Zealand. 2012 [cited 2012 20 August ]; Coeliac New Zealand is a not for profit organisation supporting those diagnosed with coeliac disease, dermatitis herpetiformis and those on a gluten free diet.

We assist those who are gluten free, promote and educate, and collaborate with gluten free manufacturers, medical professionals and support research. ]. Available from: http://www.coeliac.org.nz/.

52. Subsidies and allowances. Auckland, New Zeland.

53. Hischenhuber C, Crevel R, Jarry B, Maki M, Moneret-Vautrin DA, Romano A, et al. Review article: safe amounts of gluten for patients with wheat allergy or coeliac disease. Alimentary pharmacology & therapeutics. 2006;23(5):559-75. Epub 2006/02/17.

54. Kaukinen K, Collin P, Holm K, Rantala I, Vuolteenaho N, Reunala T, et al. Wheat starch-containing gluten-free flour products in the treatment of coeliac disease and dermatitis herpetiformis. A long-term follow-up study. Scand J Gastroenterol. 1999;34(2):163-9. Epub 1999/04/07.

55. Lohiniemi S, Maki M, Kaukinen K, Laippala P, Collin P. Gastrointestinal symptoms rating scale in coeliac disease patients on wheat starch-based gluten-free diets. Scand J Gastroenterol. 2000;35(9):947-9. Epub 2000/11/04.

56. Peraaho M, Kaukinen K, Paasikivi K, Sievanen H, Lohiniemi S, Maki M, et al. Wheat-starch-based gluten-free products in the treatment of newly detected coeliac disease: prospective and randomized study. Alimentary pharmacology & therapeutics. 2003;17(4):587-94. Epub 2003/03/08.

57. Ciclitira PJ, Cerio R, Ellis HJ, Maxton D, Nelufer JM, Macartney JM. Evaluation of a gliadin-containing gluten-free product in coeliac patients. Human nutrition Clinical nutrition. 1985;39(4):303-8. Epub 1985/07/01.

58. Report of the Twenty-Seventh Session of the Codex Committee on Methods of Analysis and Sampling. Rome, Italy: Joint Food and Agriculture Organization of the United Nations/World Health Organization Food Standards Program, 2006 3-7 July 2006. Report No.

59. Catassi C, Rossini M, Ratsch IM, Bearzi I, Santinelli A, Castagnani R, et al. Dose dependent effects of protracted ingestion of small amounts of gliadin in coeliac disease children: a clinical and jejunal morphometric study. Gut. 1993;34(11):1515-9. Epub 1993/11/01.

60. Montgomery AM, Goka AK, Kumar PJ, Farthing MJ, Clark ML. Low gluten diet in the treatment of adult coeliac disease: effect on jejunal morphology and serum anti-gluten antibodies. Gut. 1988;29(11):1564-8. Epub 1988/11/01.

61. Ciclitira PJ, Evans DJ, Fagg NL, Lennox ES, Dowling RH. Clinical testing of gliadin fractions in coeliac patients. Clinical science (London, England : 1979). 1984;66(3):357-64. Epub 1984/03/01.

62. Ferguson A, Blackwell JN, Barnetson RS. Effects of additional dietary gluten on the small-intestinal mucosa of volunteers and of patients with dermatitis herpetiformis. Scand J Gastroenterol. 1987;22(5):543-9. Epub 1987/06/01.

63. Rampertab SD, Pooran N, Brar P, Singh P, Green PH. Trends in the presentation of celiac disease. The American journal of medicine. 2006;119(4):355 e9-14. Epub 2006/03/28.

64. Mayer M, Greco L, Troncone R, Auricchio S, Marsh M. Compliance of adolescents with coeliac disease with a gluten free diet. Gut. 1991;32(8):881-5.

65. Laurin P, Wolving M, Falth-Magnusson K. Even small amounts of gluten cause relapse in children with celiac disease. J Pediatr Gastroenterol Nutr. 2002;34(1):26-30. Epub 2001/12/26.

66. Packer SM, Charlton V, Keeling JW, Risdon RA, Ogilvie D, Rowlatt RJ, et al. Gluten challenge in treated coeliac disease. Archives of disease in childhood. 1978;53(6):449-55. Epub 1978/06/01.

67. Mayer M, Greco L, Troncone R, Grimaldi M, Pansa G. Early prediction of relapse during gluten challenge in childhood celiac disease. J Pediatr Gastroenterol Nutr. 1989;8(4):474-9. Epub 1989/05/01.

68. Collin P, Thorell L, Kaukinen K, Maki M. The safe threshold for gluten contamination in gluten-free products. Can trace amounts be accepted in the treatment of coeliac disease? Alimentary pharmacology & therapeutics. 2004;19(12):1277-83. Epub 2004/06/12.

69. Gibert A, Kruizinga AG, Neuhold S, Houben GF, Canela MA, Fasano A, et al. Might gluten traces in wheat substitutes pose a risk in patients with celiac disease? A population-based probabilistic approach to risk estimation. The American journal of clinical nutrition. 2013;97(1):109-16. Epub 2012/11/30.

70. Gibert A, Espadaler M, Angel Canela M, Sanchez A, Vaque C, Rafecas M. Consumption of gluten-free products: should the threshold value for trace amounts of gluten be at 20, 100 or 200 p.p.m.? European journal of gastroenterology & hepatology. 2006;18(11):1187-95. Epub 2006/10/13.

71. Food Standards Australia New Zealand. 2012 [cited 2012 17 September]; Available from: http://www.foodstandards.govt.nz/.

72. Health Canada's Position on Gluten-Free Claims. Health Canada; 2012 [cited 2012 1 December]; Available from: http://www.hc-sc.gc.ca/fn-an/securit/allerg/cel-coe/gluten-position-eng.php.

73. Thompson T, Lee AR, Grace T. Gluten contamination of grains, seeds, and flours in the United States: a pilot study. J Am Diet Assoc. 2010;110(6):937-40. Epub 2010/05/26.

74. Gibert A, Kruizinga AG, Neuhold S, Houben GF, Canela MA, Fasano A, et al. Might gluten traces in wheat substitutes pose a risk in patients with celiac disease? A population-based probabilistic approach to risk estimation. The American journal of clinical nutrition. 2012. Epub 2012/11/30.

75. Thompson T. Gluten contamination of commercial oat products in the United States. The New England journal of medicine. 2004;351(19):2021-2. Epub 2004/11/05.

76. Cranney A, Zarkadas M, Graham I, Switzer C. The Canadian celiac health survey - the Ottawa chapter pilot. BMC Gastroenterology. 2003;3(1):8.

77. Cranney A, Zarkadas M, Graham ID, Butzner JD, Rashid M, Warren R, et al. The Canadian Celiac Health Survey. Digestive diseases and sciences. 2007;52(4):1087-95. Epub 2007/02/24.

78. Riddle DL, Lee KT, Stratford PW. Use of SF-36 and SF-12 health status measures: a quantitative comparison for groups versus individual patients. Medical care. 2001;39(8):867-78. Epub 2001/07/27.

79. Green PHR, Stavropoulos SN, Panagi SG, Goldstein SL, McMahon DJ, Absan H, et al. Characteristics of adult celiac disease in the USA: results of a national survey. The American journal of gastroenterology. 2001;96(1):126-31. Epub 2001/02/24.

80. Thoits PA. Stress, coping, and social support processes: where are we? What next? Journal of health and social behavior. 1995;Spec No:53-79. Epub 1995/01/01.

81. Gray A, Papanicolas I. Impact of symptoms on quality of life before and after diagnosis of coeliac disease: results from a UK population survey. BMC Health Services Research. 2010;10(1):105.

82. Lankisch PG, Martinez Schramm A, Petersen F, Droge M, Lehnick D, Lembcke B. Diagnostic intervals for recognizing celiac disease. Zeitschrift fur Gastroenterologie. 1996;34(8):473-7. Epub 1996/08/01.

83. Norstrom F, Lindholm L, Sandstrom O, Nordyke K, Ivarsson A. Delay to celiac disease diagnosis and its implications for health-related quality of life. BMC Gastroenterology. 2011;11(1):118.

84. Cooper BT. The delayed diagnosis of coeliac disease. The New Zealand medical journal. 1986;99(806):543-5. Epub 1986/07/23.

85. Dickey W, McConnell JB. How many hospital visits does it take before celiac sprue is diagnosed? Journal of clinical gastroenterology. 1996;23(1):21-3. Epub 1996/07/01.

86. HIgher profile needed for celiac disease. JAMA: The Journal of the American Medical Association. 2004;292(8):913-4.

87. Lo W, Sano K, Lebwohl B, Diamond B, Green PH. Changing presentation of adult celiac disease. Digestive diseases and sciences. 2003;48(2):395-8. Epub 2003/03/20.

88. Mustalahti K, Lohiniemi S, Collin P, Vuolteenaho N, Laippala P, Maki M. Gluten-free diet and quality of life in patients with screen-detected celiac disease. Effective clinical practice. 2002;5(3):105-13.

89. Johnston SD, Rodgers C, Watson RGP. Quality of life in screen-detected and typical coeliac disease and the effect of excluding dietary gluten. European journal of gastroenterology & hepatology. 2004;16(12):1281.

90. Ventura A, Magazzu G, Greco L. Duration of exposure to gluten and risk for autoimmune disorders in patients with celiac disease. SIGEP Study Group for Autoimmune Disorders in Celiac Disease. Gastroenterology. 1999;117(2):297-303. Epub 1999/07/27.

91. Green PH, Fleischauer AT, Bhagat G, Goyal R, Jabri B, Neugut AI. Risk of malignancy in patients with celiac disease. The American journal of medicine. 2003;115(3):191-5. Epub 2003/08/26.

92. Akobeng AK, Thomas AG. Systematic review: tolerable amount of gluten for people with coeliac disease. Alimentary pharmacology & therapeutics. 2008;27(11):1044-52. Epub 2008/03/05.

93. McCrae WM, Eastwood MA, Martin MR, Sircus W. Neglected coeliac disease. Lancet. 1975;1(7900):187-90. Epub 1975/01/25.

94. Abdulkarim AS, Burgart LJ, See J, Murray JA. Etiology of nonresponsive celiac disease: results of a systematic approach. The American journal of gastroenterology. 2002;97(8):2016-21. Epub 2002/08/23.

95. Niewinski MM. Advances in celiac disease and gluten-free diet. J Am Diet Assoc. 2008;108(4):661-72. Epub 2008/04/01.

96. Leffler DA, Dennis M, Hyett B, Kelly E, Schuppan D, Kelly CP. Etiologies and Predictors of Diagnosis in Nonresponsive Celiac Disease. Clinical gastroenterology and hepatology : the official clinical practice journal of the American Gastroenterological Association. 2007;5(4):445-50.

97. Niewinski MM. Advances in Celiac Disease and Gluten-Free Diet. Journal of the American Dietetic Association. 2008;108(4):661-72.

98. Simpson S, Thompson T. Nutrition assessment in celiac disease. Gastrointestinal endoscopy clinics of North America. 2012;22(4):797-809. Epub 2012/10/23.

99. Tidwell DK, Bomba AK. Attitudes of people with celiac diseasetowards dietitians and medical nutrition therapy. Journal of the American Dietetic Association. 2001;101(9, Supplement 1):A-29.

100. Simpson S, Lebwohl B, Lewis SK, Tennyson CA, Sanders DS, Green PH. Awareness of gluten-related disorders: A survey of the general public, chefs and patients. European e-Journal of Clinical Nutrition and Metabolism. 2011;6(5):e227-e31.

101. Bruce D. Oamaru baker warned over gluten-free mix-up. Otago Daily Times. 2011 20 December 2011.

102. Stevens L, Rashid M. Gluten-free and regular foods: a cost comparison. Canadian journal of dietetic practice and research : a publication of Dietitians of Canada = Revue canadienne de la

pratique et de la recherche en dietetique : une publication des Dietetistes du Canada. 2008;69(3):147-50. Epub 2008/09/12.

103. Whiteman MK, Langenberg P, Kjerulff K, McCarter R, Flaws JA. A randomized trial of incentives to improve response rates to a mailed women's health questionnaire. Journal of women's health (2002). 2003;12(8):821-8. Epub 2003/11/01.

104. Rossi TM, Albini CH, Kumar V. Incidence of celiac disease identified by the presence of serum endomysial antibodies in children with chronic diarrhea, short stature, or insulin-dependent diabetes mellitus. The Journal of pediatrics. 1993;123(2):262-4. Epub 1993/08/01.

105. Sood A, Midha V, Sood N, Avasthi G, Sehgal A. Prevalence of celiac disease among school children in Punjab, North India. J Gastroenterol Hepatol. 2006;21(10):1622-5. Epub 2006/08/25.

106. Tatar G, Elsurer R, Simsek H, Balaban YH, Hascelik G, Ozcebe OI, et al. Screening of tissue transglutaminase antibody in healthy blood donors for celiac disease screening in the Turkish population. Digestive diseases and sciences. 2004;49(9):1479-84. Epub 2004/10/16.

107. Mehrabani D, Khademolhosseini F, Omrani G, Nejabat M, Saberi-Firouzi M. Prevalence of celiac disease in Shiraz, southern Iran2008 July 1, 2008. 135-8 p.

108. Sher KS, Fraser RC, Wicks AC, Mayberry JF. High risk of coeliac disease in Punjabis. Epidemiological study in the south Asian and European populations of Leicestershire. Digestion. 1993;54(3):178-82. Epub 1993/01/01.

109. Murray JA, Van Dyke C, Plevak MF, Dierkhising RA, Zinsmeister AR, Melton LJ, 3rd. Trends in the identification and clinical features of celiac disease in a North American community, 1950-2001. Clinical gastroenterology and hepatology : the official clinical practice journal of the American Gastroenterological Association. 2003;1(1):19-27. Epub 2004/03/16.

110. Shamir R, Lerner A, Shinar E, Lahat N, Sobel E, Bar-or R, et al. The use of a single serological marker underestimates the prevalence of celiac disease in Israel: a study of blood donors. The American journal of gastroenterology. 2002;97(10):2589-94. Epub 2002/10/19.

111. Hovell CJ, Collett JA, Vautier G, Cheng AJ, Sutanto E, Mallon DF, et al. High prevalence of coeliac disease in a population-based study from Western Australia: a case for screening? The Medical journal of Australia. 2001;175(5):247-50. Epub 2001/10/06.

112. Challar MH JM, Sitzmann FC, Seferian V, Shahin E. Prevalence of asymptomatic celiac disease in a Syrian population sample. JABMS. 2004;6:155–60.

113. Kupfer SS. Making Sense of Marsh. Impact [Internet]. 2009 9 July 2012:[1, 3 pp.].

114. Marsh MN. Gluten, major histocompatibility complex, and the small intestine. A molecular and immunobiologic approach to the spectrum of gluten sensitivity ('celiac sprue'). Gastroenterology. 1992;102(1):330-54. Epub 1992/01/01.

115. Denery-Papini S, Nicolas Y, Popineau Y. Efficiency and Limitations of Immunochemical Assays for the Testing of Gluten-free Foods. Journal of Cereal Science. 1999;30(2):121-31.

116. Israel V, Enrique G, Mercedes L, Enrique M. Innovative approach to low-level gluten determination in foods using a novel sandwich enzyme-linked immunosorbent assay protocol. European journal of gastroenterology & hepatology. 2003;15(5):465-74.

117. ELISA Technologies I. ELISA Technologies. Acceleration.net & WordPress; 2012 [cited 2012 20 September]; Available from: http://www.elisa-tek.com/.

118. Ferre S, García E, Méndez E, editors. Measurement of hydrolysed gliadins by a competitive ELISA based on monoclonal antibody R5: analysis of syrups and beers. Proceedings of the 18th Meeting Working Group on Prolamin Analysis and Toxicity; 2003.

119. Hernando A, García E, Llorente M, Mujico J, Lombardía M, Maki M, et al., editors. Measurements of hydrolysed gliadins in malts, breakfast cereals, heated/hydrolysed foods, whiskies and beers by means of a new competitive R5 ELISA. Proceedings of the 19th Meeting Working Group on Prolamin Analysis and Toxicity, Prague, Czech Republic, Verlag Wissenschaftliche Scripten; 2004.

120. Hernando A, Mujico JR, Mena MC, Lombardia M, Mendez E. Measurement of wheat gluten and barley hordeins in contaminated oats from Europe, the United States and Canada by Sandwich R5 ELISA. European journal of gastroenterology & hepatology. 2008;20(6):545-54. Epub 2008/05/10.

## Appendix 1: Prevalence/ Incidence of CD Summary Table

**Table: Summary of studies assessing the incidence and prevalence of Coeliac Disease in New Zealand and New Zealand**

| Source | Citation | Country | Study Population | Rate |
|---|---|---|---|---|
| **Incidence in Children** | | | | |
| Cook, B., Oxner, R., Chapman, B., Whitehead, M., & Burt, M. (2004). A thirty-year (1970-1999) study of coeliac disease in the Canterbury region of New Zealand. *N Z Med J, 117*(1189), U772. | (13) | Canterbury, New Zealand | Patients with intestinal biopsy proven CD in the Canterbury region from 1970 – 1999, <20 years old | 2.3/100,000 years |
| Ussher, R., Yeong, M. L., & Stace, N. (1994). Coeliac disease: incidence and prevalence in Wellington 1985-92. *N Z Med J, 107*(978), 195-197. | (7) | Wellington, New Zealand | All patients diagnosed with CD at Wellington Hospital from 1985 - 1992 | 0.19 per 100,000 |
| Rossi TM, Albini CH, Kumar V. Incidence of celiac disease identified by the presence of serum endomysial antibodies in children with chronic diarrhoea, short stature or insulin dependent diabetes mellitus. J. Ped. 1993; 123: 262–4. | (104) | Western New York area | Children | 1.29 per 10,000 live births |
| Ivarsson A, Persson LA, Nystrom L et al. Epidemic | (11) | Sweden | Children who were diagnosed with CD | Children <2 years old |

| | | | | |
|---|---|---|---|---|
| of celiac disease in Swedish children. Acta Paediatr. 2000; 89: 165–71. | | | | *1973 to 1984* - 65 per 100 000 person years. *1987 to 1994* - 198 per 100 000 person years. *1995 to 1997* - 51 per 100 000 person years. |
| Mylotte et al. Incidence of coeliac disease in the West of Ireland | (8) | Ireland | Children born 1960-1970 who were diagnosed as having coeliac disease before September 1972, as were adults who were diagnosed between 1969-1972 | 1:597 |
| **Prevalence in Children** | | | | |
| Ussher, R., Yeong, M. L., & Stace, N. (1994). Coeliac disease: incidence and prevalence in Wellington 1985-92. *N Z Med J, 107*(978), 195-197. | (7) | Wellington, New Zealand | All patients diagnosed with CD at Wellington Hospital from 1985 - 1992 | 14 per 100,000. |
| (14) | (14) | Otago, New Zealand | Children and adults who were investigated for malabsorption or steatorrhoea, or who had a small intestinal biopsy, or who had a coded diagnosis of coeliac disease in Otago hospital between 1975 – 1984. | Cumulative prevalence 35 per 100,000. Prevalence 9 per 100,000. |
| Fasano A, Berti I, | (10) | United States of America | Children who were | 1:320 |

| | | | | |
|---|---|---|---|---|
| Gerarduzzi T et al. Prevalence of celiac disease in at-risk and not-at-risk groups in the United States. Arch. Intern. Med. 2003; 163: 286–92. | | | serologically screened by capturing blood donors, and patients seen in outpatient clinics for routine checkups | |
| Sood A, Midha V, Sood N, Avasthi G, Sehgal A. Prevalence of celiac disease among school children in Punjab, North India. J. Gastroenterol. Hepatol. 2006; 21: 1622–5. | (105) | Punjab, North India | 4347 school children (age range 3-17 years). | 1:310 |
| Ertekin V, Selimoglu MA, Kardas F, Aktas E. Prevalence of celiac disease in Turkish children. J. Clin. Gastroenterol. 2005; 39: 689–91. | (9) | Turkey | 1,489 children were randomly selected from healthy school children (6 to 17 years) | 1:158 |
| **Incidence in Adults** | | | | |
| Ussher, R., Yeong, M. L., & Stace, N. (1994). Coeliac disease: incidence and prevalence in Wellington 1985-92. *N Z Med J, 107*(978), 195-197. | (7) | Wellington, New Zealand | All patients diagnosed with CD at Wellington Hospital from 1985 - 1992 | 1.59 per 100,000 |
| Mylotte et al. Incidence of coeliac disease in the West of Ireland | (8) | Ireland | Children born 1960-1970 who were diagnosed as having coeliac disease before | 1:303 |

| | | | | |
|---|---|---|---|---|
| | | | September 1972, as were adults who were diagnosed between 1969-1972 | |
| **Prevalence in Adults** | | | | |
| Cook, H. B., Burt, M. J., Collett, J. A., Whitehead, M. R., Frampton, C. M., & Chapman, B. A. (2000). Adult coeliac disease: Prevalence and clinical significance. *Journal of Gastroenterology and Hepatology, 15*(9), 1032-1036. doi: 10.1046/j.1440-1746.2000.02290.x | (15) | Canterbury New Zealand | 1064 Randomly selected adults from the electoral roll, Christchurch | 1.2% or 1:82 |
| Shahbazkhani B, Malekzadeh R, Sotoudeh M et al. High prevalence of coeliac disease in apparently healthy Iranian blood donors. Eur. J. Gastroenterol. Hepatol. 2003; 15: 475–8. | (5) | Iran | 2000 apparently healthy blood donors (18-65 years) 1998/1999 | Minimum prevalence of gluten sensitivity 1/166 |
| Tatar G, Elsurer R, Simsek H et al. Screening of tissue transglutaminase antibody in healthy blood donors for celiac disease screening in the Turkish population. Dig. Dis. Sci. 2004; 49: 1479–84. | (106) | Turkey | 2000 healthy blood donors (Mean age 33 +/- 9 years) | tTG antibody positivity prevalence in healthy blood donors 1.3% |
| Prevalence of celiac disease in Shiraz, southern | (107) | Shiraz, Iran | 1440 randomly selected subjects (age range 29-83 | 1:180 positive for IgA anti-tTG antibodies, 1:720 Iga- |

| | | | | |
|---|---|---|---|---|
| Iran. Mehdi Saberi-Firouzi, Gholamhossein R Omrani, Marzieh Nejabat, Davood Mehrabani, Farnaz Khademolhosseini | | | years) | class anti-EMA antibodies |
| Lohi et al. Increasing prevalence of coeliac disease over time | (6) | Finland | Finnish adult population in 1978–80 (8000 people) and 2000–01 (8028 people) | 1.05% in 1978-80 and 1.99% in 2000-01 |
| **Incidence in Total Populations** | | | | |
| Cook, B., Oxner, R., Chapman, B., Whitehead, M., & Burt, M. (2004). A thirty-year (1970-1999) study of coeliac disease in the Canterbury region of New Zealand. *N Z Med J, 117*(1189), U772. | (13) | Canterbury, New Zealand | Patients with intestinal biopsy proven CD in the Canterbury region from 1970 - 1999 | 2.2/100,000 years |
| Ussher, R., Yeong, M. L., & Stace, N. (1994). Coeliac disease: incidence and prevalence in Wellington 1985-92. *N Z Med J, 107*(978), 195-197. | (7) | Wellington, New Zealand | All patients diagnosed with CD at Wellington Hospital from 1985 - 1992 | 1.8 per 100,000 |
| Sher KS, Fraser RC, Wicks AC, Mayberry JF. High risk of celiac disease in Punjabis. Epidemiological study in the South Asian and European populations in Leicestershire. Digestion 1993; 54: 178–82. | (108) | Leicestershire, England | Retrospective study of 106 histologically confirmed cases of coeliac disease between 1975 and 1989 | Populations (rates per 100,000 per year) Urban :6/100,000 year Rural: 3.2/100,000/year Europeans: 2.5/100,000/year Gujaratis: 0.9/100,000/year Punjabis: 6.9/100,000/year |

| | | | | |
|---|---|---|---|---|
| Murray et al. Trend in the identification and clinical features of Coeliac disease in a North America community. | (109) | Olmsted County, Minnesota | All county residents diagnosed with celiac disease between 1950 and 2001 | 2.1 per 100,000. (9.1 per 100,000 1999- 2001) |
| **Prevalence in Total Populations** | | | | |
| Ussher, R., Yeong, M. L., & Stace, N. (1994). Coeliac disease: incidence and prevalence in Wellington 1985-92. *N Z Med J, 107*(978), 195-197. | (7) | Wellington, New Zealand | All patients diagnosed with CD at Wellington Hospital from 1985 - 1992 | 70 per 100,000 |
| Shamir R. The use of a single serological marker underestimates the prevalence of celiac disease in Israel: a study of blood donors. Am. J. Gastroenterol. 2002; 97: 2589–94. | (110) | Israel | 1571 healthy blood donors | At least 1:157 in the general population (0.6%) |
| Hovell CJ, Collett JA, Vautier G et al. High prevalence of coeliac disease in a population-based study from Western Australia: a case for screening? MJA 2001; 175: 247–50. | (111) | Western Australia | Retrospective analysis of stored serum samples from 3,011 random subjects | Newly diagnosed 1:430 all AEA-positive and previously diagnosed 1:251 |
| Challar MH, Jouma M, Sitzmann FC, Seferian V, Shahin E. Prevalence of | (112) | Syria | 400 Healthy Syrian subjects 6- 66 years | At least 1.5% |

| | | | | |
|---|---|---|---|---|
| asymptomatic celiac disease in a Syrian population sample. JABMS 2004; 6: 155–60E. | | | | |

## Appendix 2: Marsh Criteria

The diagnosis of CD by intestinal biopsy is made by assessing the autoimmune intestinal damage. The key points in the diagnosis is villous atrophy and crypt hyperplasia, or in simpler terms, flattening of the villi. The characteristic histopathologic features of the mucosa in those with CD are characterised as Marsh type 0, 1, 2, 3 (divided into 3a, 3b and 3c) or 4. This criteria was introduced by M. N. Marsh in 1992 (13). Histopathological features assessed when determining which Marsh criteria a specimen fits include; villi length and crypt length. In a normal functioning intestine the ratio of villi to crypt length should be between 3:1 and 5:1. In addition to assessing the physical state of the lining; the density of lymphocytes in the top layer of the epithelial cells is measured.

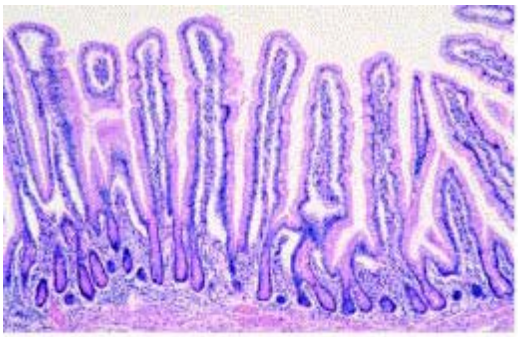
In a normal functioning intestine (Marsh type 0) there should be no more than 30 (-40) lymphocytes per 100 epithelial cells.

Marsh type 1 histopathologic features include normal villi and crypt length but an increase in the number of lymphocytes above the normal level.

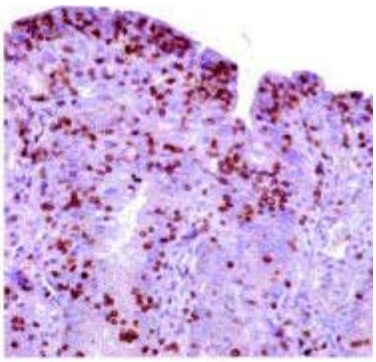
Marsh type 2 (hyperplastic lesion) histopathologic features include an increase in intraepithelial lymphocyte density and crypt hyperplasia, manifested by crypt branching, crypt elongation and increased mitotic activity. Villous height to crypt depth ratio will decrease below 3:1 – 5:1.

Marsh type 3 histopathologic features include increased intraepithelial lymphocyte density, crypt hypertrophy and villous atrophy. Marsh type III a, b and c differ in the extent of these three criteria. Type a refers to partial villous atrophy, where the villi are shorter, type b refers to subtotal villous atrophy where the villi are significantly shortened but still recognisable, type c refers to total villous atrophy where the villi have near disappeared.

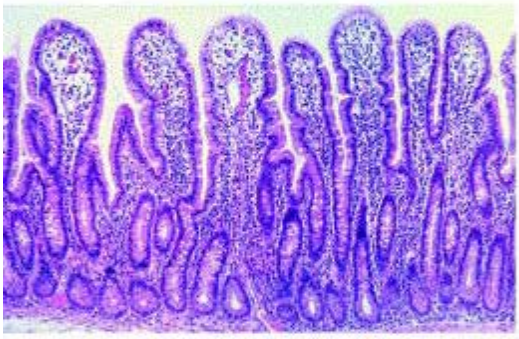
Marsh type 4 histopathologic features include hyperplastic or complete atrophy of the villi. This only occurs in extreme cases of chronic inflammation and it is suspected to have caused irreversible damage. Marsh type 4 is associated with refractory CD and T-cell lymphoma (113) (32).



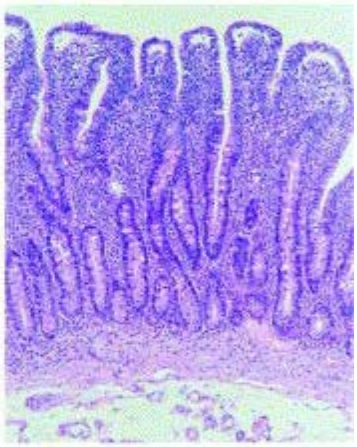
Marsh I: lymphocytic enteritis



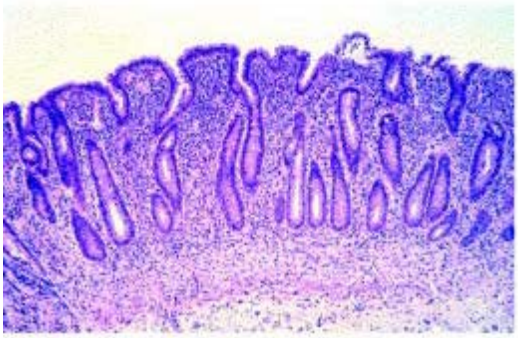
Intraepithelial lymphocytosis
HE CD3 IEL



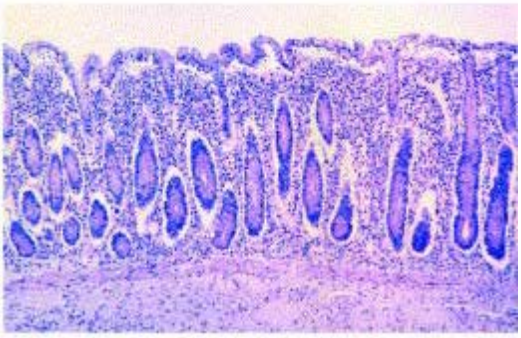
Marsh II: lymphocytic enteritis with crypthyperplasia



Marsh III A: partial villous atrophy



Marsh III B: subtotal villous atrophy



Marsh III C: total villous atrophy

**Figure: Original Marsh description of histology spectrum in Coeliac Disease *(114)***

## Appendix 3: ELISA and R5 ELISA Tests

**ELISA**

This method was developed by Skerritt and Hill in 1991. It uses a 40% ethanol extraction and measures monoclonal antibodies to ω-gliadin. The ω-gliadin fraction of wheat gliadin is not denatured by heat and so withstands cooking and processing. However only measuring ω-gliadin fraction can introduce inaccuracy as this is only one of the three gliadin fractions found in wheat. α-, β- and γ-gliadin are not measured, and the proportion of ω-gliadin in total gliadin has been shown to vary greatly from 6% to 20% (115). Further, ELISA does not accurately detect or quantify barley prolamins, hordein (116), and cannot accurately quantify hydrolysed gluten. The official AOAC method can quantify gluten $\geqslant$150ppm (117), however but more sensitive versions are available.

**R5 ELISA**

The R5 ELISA was developed by Mendez (58). It is based on the R5 monoclonal antibody that detects and quantifies gluten (to 3ppm) in wheat, barley and rye. (1). Advantages of the R5 ELISA compared with ELISA is that it can detect and quantify gluten from all grains at a much lower level. R5 ELISA can detect all fractions of wheat gliadin (not just ω-gliadin) as well as hordeins in barley and secalins in rye. Gluten can also be detected and measured in heated and unheated foods.

Two aspects of the R5 ELISA that contribute to problems in measuring gluten is that it overestimates barley contamination and like the ELISA it currently does not accurately measure hydrolysed prolamins, new methods are being developed to correct this (118, 119). The sandwich R5 Prolamin Working Group gliadin standard overestimates gluten from barley by a factor of 2.4-2.6 (120). This is because the assay measures α/β, γ-gliadin and ω-gliadin from wheat but the polymeric high and low molecular weight glutenin fractions are not extracted and therefore not measured. The extracted to non-extracted fractions have a relative ratio of 65:35, to correct this the amount extracted is multiplied by 2 (rounding to a 50:50 ratio). With barley γ-hordeins, C-hordeins and B-hordeins are all extracted. B-hordeins are rich in the QQPFP epitope, overestimating gluten from barley by 2.4-2.6 times. This is only a problem in foods contaminated with barley. This problem can be corrected by using a hordein standard for the sandwich R5 ELISA. This When the hordein content is extracted this amount is not multiplied by 2 as with the gliadin standard (19).

## Appendix 4: Study Protocol

# Study Protocol: The health of people with Coeliac Disease in New Zealand and the impact of a gluten free diet

## List of Abbreviations:

CD: Coeliac Disease
GF: Gluten-Free
GFD: Gluten-Free Diet
NZ: New Zealand

## Principal Investigator, Research Team, and Study Site:

Principal investigator: Kiri Sharp (Masters of Dietetic student)

Co-Investigators and Supervisors: Dr. Kirsten Coppell (Senior Research Fellow), Heather Walker (Statistician)

Research team and contact Information:

**Kiri Sharp**
Ph: +64 6 354 3635
Email: shaki801@student.otago.ac.nz
Post: Coeliac Health Survey Study
6 Greenwood Place
Fitzherbert
Palmerston North

**Dr Kirsten Coppell**
Ph: +64 3 474 7774
Email: kirsten.coppell@otago.ac.nz
Post: Coeliac Health Survey Study
Edgar National Centre for Diabetes and Obesity Research
Department of Medicine
PO Box 913
Dunedin

**Heather Walker**
Email: heather.walker@otago.ac.n
Post: LINZ Unit

PO Box 56
Dunedin

Study site: Palmerston North & Dunedin, New Zealand

## Research Synopsis

**Study Title**

The health of people with Coeliac Disease and the impact of a gluten free diet

**Study Population**

The survey study population is individuals with Coeliac Disease resident in New Zealand who are members of the Coeliac New Zealand Incorporated. The study population will be further described through demographic information acquired through the survey.

**Study Design**

This study consists of a survey which will be administered via email or a hard copy sent in the post to members of the Coeliac New Zealand Incorporated.

**Sample Size**

The total sample size available for this study is 2,700. Around 500 are not able to be contacted by email and so a hard copy of the survey will be mailed out.

**Study Duration**

This study commenced on the 23rd of February 2012 and will be completed by the 20th of December 2012, with duration of 10 months.

**Primary Objective**

The primary objective of this study is to describe the health of people with Coeliac Disease.

**Secondary Objectives**

The secondary objective of this study is to determine the extent of on-going symptoms due to 'accidental' ingestion of gluten through contaminated food products.

## Study design/methods:

This study is a cross-sectional observation study. A questionnaire will be sent to members of the Coeliac New Zealand Incorporated (for which permission has been obtained) via email or a link will be emailed where the participants can complete the survey online. For participants who do not have an email address a hard copy of the questionnaire will be mailed to them via the Coeliac New Zealand Incorporated to maintain anonymity.

The survey questionnaire will be based on the Canadian Coeliac Health Survey (for which permission for use has been obtained). This is a comprehensive questionnaire with 10 sections including demographics, diagnosis of Coeliac Disease, diet, coeliac quality of life issues, bone disease, reproduction, Coeliac Disease among family members (optional) and personal recommendations. This questionnaire was extensively pretested by the Canadian group and has since been used successfully in a number of countries and studies. To encourage participation in this study a prize draw will be offered to those who complete and return the survey. The survey contains open and closed questions providing quantitative and qualitative data.

## Study Population:

The survey study population is individuals with Coeliac Disease resident in New Zealand who are members of the Coeliac Society of New Zealand. The study population will be further described through demographic information acquired through the survey.

**Inclusion /Exclusion Criteria**

Participants must have Coeliac Disease and be a New Zealand resident.

## Study Duration/ Study Timeline:

Stage 1 (1 April 2012 - 8 May 2012): Contact Dr. Mohsin Rashid to request permission to use the Canadian Celiac Health survey for this study. Contact Coeliac New Zealand Incorporated to ask whether they would be willing to distribute the survey to their members.
Stage 2 (May 2012): Ethics application
Stage 3 (1 June 2012): Distribute questionnaires to all participants via the Coeliac New Zealand Incorporated either as an online link, attachment to an email or a hard copy in the post.
Stage 4 (by 30 June 2012): Collect questionnaires. These should be posted back in self-addressed envelopes or emailed back or completed online.
Stage 5 (1 July 2012 - 31 July 2012): Data entry and data checks
Stage 6 (1 August 2012 - 31 August 2012): Perform statistical analysis
Stage 7 (1 September 2012 - 20 December 2012): Interpretation of the data and writing up of the study.

## Statistical Analysis Plan:

Means and standard deviations or percentages were calculated for variables of interest.

## Informed Consent Process:

An information sheet will be attached to the survey so participants are fully informed of what is involved, benefits and risks before making a decision to participate.

***Privacy and confidentiality:***

Confidentiality will be maintained about any information relating to participants. Name and mailing information will be kept confidential to the Coeliac New Zealand Incorporated. Returned completed questionnaires will be viewed by only two of the investigators (Kiri Sharp and Dr Kirsten Coppell). No reports arising from this study will include identifiable information.

## Risk/Benefit:

***Risk to participants:***

There are no anticipated risks. All completed questionnaires will remain confidential and no identifying information will be retained.

***Benefits to Participants***

Participating in this research will provide a good overall description of coeliac disease in New Zealand, as well as information about the needs of people and families with coeliac disease. The results will inform food production and food handling practices, food labelling processes, and dietetic practice.

## Data Safety Monitoring:

All completed questionnaires will remain confidential and no identifying information will be retained.

## Conflict of Interest:

There is no known conflict of interest between any parties involved in this study.

## Publication and Presentation Plans:

A Masters of Dietetics thesis will be submitted. A summary of the results will be published in the Coeliac New Zealand Incorporated quarterly magazine. Study results will be published in appropriate peer reviewed journals and presented at relevant fora and conferences.

## References:

1. Cook HB, Burt MJ, Collett JA, Whitehead MR, Frampton CM, Chapman BA. Adult celiac disease: prevalence and clinical significance. J Gastroenterol Hepatol 2000; 15: 1032-36.
2. Cook B, Oxner R, Chapman B, Whitehead M, Burt M. A thirty-year (1970-1999) study of coeliac disease in the Canterbury region of New Zealand. NZ Med J 2004 Feb 20;117(1189):U772.
3. Gillet PM, Gillet R, Israel DM, Metzger DL, Stewart L, Chanoine J-P, Freeman HJ. High prevalence of celiac disease in patients with type 1 diabetes detected by antibodies to endomysium and tissue transglutaminase. Can J Gastroenterol 2001; 15: 297-301.
4. Abdulkarim AS, Burgart LJ, See J, Murray JA. Etiology of nonresponsive celiac disease: results of a systematic approach. Am J Gastroenterol. 2002; 97: 2016-21.
5. Rubio-Tapia A, Murray JA. Classification and management of refractory coeliac disease. Gut. 2010; 59: 547-57.
6. Leffler DA, Dennis M, Hyett B, Kelly E, Schuppan D, Kelly SP. Etiologies and predictors of diagnosis in nonresponsive celiac disease. Clin Gastroenterol Hepatol 2007; 5: 445-450.
7. Gelinas P, McKinnon CM, Mena MC, Mendez. Gluten contamination of cereal foods in Canada. Int J Food Sci Tech 2008; 43:1243-1252.
8. Thompson T. Gluten contamination of grains, seeds, and flours in the United States: A pilot study. J Am Diet Assoc 2010; 110: 937-940.

## Appendix 5: Ethics Approval letter

**Multi-region Ethics Committee**
c/- Ministry of HealthPO Box 5013
1 the Terrace
Wellington
Phone: (04) 816 2403
Email: multiregion_ethicscommittee@moh.govt.nz

6 June 2012

Ms Kiri Sharp
University of Otago
6 Greenwood Place
Fitzherbert
Palmerston North

Dear Ms Sharp

Ethics ref: **MEC/12/EXP/072** (please quote in all correspondence)

Study title: The health of people with Coeliac disease in New Zealand and the impact of a gluten free diet.

This study was given expedited ethical approval by the Chair of the Multi-region Ethics Committee on 5 June 2012.

Approved Documents
- Expedited application form
- Study protocol
- New Zealand Coeliac Health Survey, dated May 2012
- Information sheet, Version 1, dated May 2012

This approval is valid until 31 October 2012, provided that Annual Progress Reports are submitted (see below).

Amendments and Protocol Deviations
All significant amendments to this proposal must receive prior approval from the Committee. Significant amendments include (but are not limited to) changes to:
— the researcher responsible for the conduct of the study at a study site
— the addition of an extra study site
— the design or duration of the study
— the method of recruitment
— information sheets and informed consent procedures.
Significant deviations from the approved protocol must be reported to the Committee as soon as possible.
Annual Progress Reports and Final Reports
The first Annual Progress Report for this study is due to the Committee by 5 June 2013.

The Annual Report Form that should be used is available at www.ethicscommittees.health.govt.nz.
Please note that if you do not provide a progress report by this date, ethical approval may be withdrawn.
A Final Report is also required at the conclusion of the study. The Final Report Form is also available at www.ethicscommittees.health.govt.nz.
Statement of compliance
The committee is constituted in accordance with its Terms of Reference. It complies with the *Operational Standard for Ethics Committees* and the principles of international good clinical practice.
The committee is approved by the Health Research Council's Ethics Committee for the purposes of section 25(1)(c) of the Health Research Council Act 1990.

We wish you all the best with your study.

Yours sincerely

**Sarah Delgado**
Administrator
Multi-region Ethics Committee

## Appendix 6: New Zealand Coeliac Health Survey

# NEW ZEALAND COELIAC HEALTH SURVEY

**June 2012**

**CONFIDENTIALITY**

**This questionnaire is not coded in any way to maintain strict confidentiality of the information provided, and to assure the anonymity of the respondents.**

**Developed by the**

***Canadian Celiac Association***

**in collaboration with the**

***Faculty of Medicine* of the *University of Ottawa***

***Canada***

**REPRODUCED with modifications for the New Zealand setting WITH PERMISSION from the Canadian Celiac Association, 2012.**

**© 2002 Canadian Celiac Association. All rights reserved.**

**This questionnaire may not be reproduced, in whole or in part, in any form or by any means, without permission in writing from the Canadian Celiac Association.**

***Canadian Celiac Association*** **Tel: (905) 507-6208 Fax: (905) 507-4673**

**E-mail:** celiac@look.ca

# NEW ZEALAND COELIAC HEALTH SURVEY

INSTRUCTIONS

1. Please complete this questionnaire for EACH FAMILY MEMBER in the household with coeliac disease.
2. Please answer ALL QUESTIONS as completely and accurately as possible.
3. Please return the completed questionnaire BY 3 AUGUST 2012, in the enclosed postage-paid envelope.

**** Please note that in this questionnaire "coeliac disease" includes dermatitis herpetiformis.***

**IF YOU ARE COMPLETING THIS QUESTIONNAIRE FOR A CHILD, please indicate how you are related to the child (e.g., mother).**

**Your relationship _____**

SECTION 1: GENERAL INFORMATION

1. Date of Birth: Day:__ __ Month:__ __Year:__ __ __ __

2. Sex: M ____ F ____

3. Region where you live in New Zealand _____

4. Ethnicity: (Please tick all that apply)

☐ NZ European ☐ NZ Maori ☐ NZ Pacific Islander ☐ Other please state_____

## *SECTION 2: DIAGNOSIS OF COELIAC DISEASE*

5. Please indicate if a doctor told you that you have coeliac disease and/or dermatitis herpetiformis*.
**PLEASE TICK ANSWER.**

a) Coeliac disease ☐ **YES** ☐ **NO**

b) Dermatitis herpetiformis* ☐ **YES** ☐ **NO** ☐ **DON'T KNOW**

6. What year did a doctor tell you that you had coeliac disease? _____ Year

7. Was your diagnosis confirmed by any of the following tests? **PLEASE TICK ANSWERS**

a) Intestinal biopsy ☐ **YES** ☐ **NO** ☐ **DON'T KNOW**

b) Antibody test ☐ **YES** ☐ **NO** ☐ **DON'T KNOW**

c) Skin biopsy ☐ **YES** ☐ **NO** ☐ **DON'T KNOW**

d) **If you answered NO to all of the above**, please indicate why you believe that you have coeliac disease. **PLEASE TICK ALL THAT APPLY.**

i) You were diagnosed before biopsy procedures were being performed. ☐

ii) You had a dramatic improvement in your condition with a gluten-free diet. ☐

iii) You diagnosed the condition yourself. ☐

iv) Your skin condition cleared when you followed a gluten-free diet. ☐

8. a) Were you FIRST diagnosed as a child ? ☐ **YES** ☐ **NO** ☐ **DON'T KNOW**

b) **If YES,** did the symptoms disappear, and then reappear when you were an adult?
☐ **YES** ☐ **NO**

9, Before your diagnosis of coeliac disease, did a doctor ever tell you that your coeliac symptoms were due to any of the following? **PLEASE TICK ALL THAT APPLY**.

a) Acid reflux (GERD) ☐

b) Anaemia ☐

c) Chronic fatigue syndrome ☐

d) Diverticulitis ☐

e) Food allergy ☐

f) Gall bladder ☐

g) Hiatus hernia ☐

h) Irritable bowel syndrome ☐

i) Menstrual problems ☐

j) Stomach or duodenal ulcers ☐

k) Stress/nerves/depression ☐

l) Vitamin deficiencies ☐

10. Please indicate whether you had any of the following conditions **WITHIN SIX MONTHS PRIOR** to the onset of your coeliac symptoms. **PLEASE TICK ALL THAT APPLY.**

a) Severe influenza ☐

b) Major surgery ☐

c) Pregnancy ☐

d) Severe stress (e.g., family death, divorce) ☐

e) Severe intestinal infection ☐

11. **How long** did you have your coeliac symptoms before you were diagnosed? _____ Years

12. **PLEASE TICK WHETHER OR NOT YOU HAD THE FOLLOWING CONDITIONS** before your diagnosis of coeliac disease, **and WHETHER OR NOT YOU HAVE FULLY RECOVERED** from these conditions.

| CONDITION | YES | NO | FULLY RECOVERED | NOT FULLY RECOVERED |
|---|---|---|---|---|
| a) Extreme weakness or tiredness | ☐ | ☐ | ☐ | ☐ |
| b) Anemia | ☐ | ☐ | ☐ | ☐ |
| c) Weight loss | ☐ | ☐ | ☐ | ☐ |
| d) Diarrhoea | ☐ | ☐ | ☐ | ☐ |
| e) Constipation | ☐ | ☐ | ☐ | ☐ |
| f) Bloating, gas, abdominal pain | ☐ | ☐ | ☐ | ☐ |
| g) Large, pale, foul-smelling stools | ☐ | ☐ | ☐ | ☐ |
| h) Nausea or vomiting | ☐ | ☐ | ☐ | ☐ |
| i) Easy bruising of skin | ☐ | ☐ | ☐ | ☐ |
| j) Migraine headaches | ☐ | ☐ | ☐ | ☐ |
| k) Swollen hands/feet/ankles | ☐ | ☐ | ☐ | ☐ |
| l) Bone/joint pain | ☐ | ☐ | ☐ | ☐ |
| m) Muscle cramps | ☐ | ☐ | ☐ | ☐ |
| n) Mouth ulcers | ☐ | ☐ | ☐ | ☐ |
| o) Lactose intolerance | ☐ | ☐ | ☐ | ☐ |
| p) Eczema | ☐ | ☐ | ☐ | ☐ |
| q) Itchy skin | ☐ | ☐ | ☐ | ☐ |
| r) Mood swings/depression | ☐ | ☐ | ☐ | ☐ |
| s) Hypoglycemia | ☐ | ☐ | ☐ | ☐ |
| t) Poor growth | ☐ | ☐ | ☐ | ☐ |

13. If you have **NOT FULLY RECOVERED** from the conditions you checked in question 12**, PLEASE TICK WHETHER YOU FEEL THAT ANY OF YOUR CONTINUING SYMPTOMS MAY BE DUE TO:**

a) problems with adhering to a gluten-free diet ☐

b) unaware of hidden sources of gluten ☐

c) some other food allergy ☐

d) unknown cause ☐

14. How many of the following types of doctors did you consult about your coeliac symptoms before your diagnosis?

| TYPE OF DOCTOR | HOW MANY? | TYPE OF DOCTOR | HOW MANY? |
|---|---|---|---|
| a) General practitioner | | i) Allergist | |
| b) Paediatrician | | j) Haematologist | |
| c) Gastroenterologist | | k) Psychiatrist/psychologist | |
| d) Physician | | l) Neurologist | |
| e) Dermatologist | | m) Endocrinologist | |
| f) Gynaecologist | | n) Dentist | |
| g) Rheumatologist | | o) Oncologist | |
| h) Orthopaedic surgeon | | p) Alternative medical practitioner | |

15. What type of doctor made your final diagnosis of coeliac disease? _____

SECTION 3: DIET

**PLEASE TICKTHE BEST ANSWERS**

16. a) When you were diagnosed with coeliac disease, were you told that you needed to follow a gluten-free diet for life? ☐ **YES** ☐ **NO**

b) **If YES,** were you referred to a nutritionist/dietitian? ☐ **YES** ☐ **NO**

17. a) Did you follow a gluten-free diet? ☐ **YES** ☐ **SOMETIMES** ☐ **NO**

b) **If YES**, how much did your health improve when you went onto a gluten-free diet? ☐ **A LOT** ☐ **MODERATELY** ☐ **A LITTLE** ☐ **NOT AT ALL**

18. How difficult has it been for you to follow a gluten-free diet? ☐ **VERY DIFFICULT** ☐ **MODERATELY DIFFICULT** ☐ **A LITTLE DIFFICULT** ☐ **NOT DIFFICULT**

Why? (Please state)

_____

_____

_____

_____

19. How would you describe your present diet?

☐ **STRICTLY GLUTEN-FREE** ☐ **PARTIALLY GLUTEN-FREE** ☐ **NOT GLUTEN-FREE**

Please provide any additional comments regarding your current diet?

_____
_____
_____

_____

_____

20. a) Do you have a reaction if you accidentally consume gluten-containing foods? If you indicate "not sure" or "no" please go to question 22.

☐ **YES** ☐ **NOT SURE** ☐ **NO**

b) **If YES, PLEASE TICK <u>ALL</u> SYMPTOMS YOU EXPERIENCE**

i) Diarrhoea ☐

ii) Constipation ☐

iii) Bloating/gas ☐

iv) Stomach/abdominal discomfort

□

v) Nausea/vomiting □

vi) Fatigue □

vii) Mouth ulcers □

viii) Headache □

ix) Insomnia □

x) Itchy skin □

c) **If you have checked any of the above**, how soon does the first symptom appear?

**_____ HOURS** **_____ DAYS** □ **DON'T KNOW**

21. How often do you accidentally consume gluten?

i) At least weekly

□

ii) At least monthly □

iii) Occasionally (less frequently than monthly, but at least annually) □

iv) Rarely □

22. Please indicate: **WHETHER YOU HAVE RECEIVED ANY INFORMATION** about coeliac disease and its treatment from following sources, and the **QUALITY** of information received. **PLEASE TICK THE BEST ANSWER FOR EACH SOURCE.**

| | **RECEIVED** | | **QUALITY OF INFORMATION** | | | | |
|---|---|---|---|---|---|---|---|
| | **YES** | **NO** | **EXCEL-LENT** | **VERY GOOD** | **GOOD** | **FAIR** | **POOR** |
| a) Family doctor | □ | □ | □ | □ | □ | □ | □ |
| b) Gastroenterologist | □ | □ | □ | □ | □ | □ | □ |
| c) Dietitian/nutritionist | □ | □ | □ | □ | □ | □ | □ |
| d) Coeliac New Zealand Incorporated | □ | □ | □ | □ | □ | □ | □ |
| e) Regional coeliac group | □ | □ | □ | □ | □ | □ | □ |
| f) Alternative medical professional | □ | □ | □ | □ | □ | □ | □ |
| g) Another person with celiac disease | □ | □ | □ | □ | □ | □ | □ |
| h) Newspapers/magazines | □ | □ | □ | □ | □ | □ | □ |
| i) Internet | □ | □ | □ | □ | □ | □ | □ |
| j) Medical books | □ | □ | □ | □ | □ | □ | □ |
| k) Cook books | □ | □ | □ | □ | □ | □ | □ |

SECTION 4: SPECIAL COELIAC QUALITY OF LIFE ISSUES

The following statements are about how coeliac disease has affected your daily life**. PLEASE TICK EACH STATEMENT** that best describes **YOUR** situation **DURING THE PAST YEAR**, e.g., **I** avoided restaurants because of coeliac disease.

**IF YOU ARE COMPLETING THIS QUESTIONNAIRE FOR A CHILD, PLEASE TICK EACH STATEMENT** that best describes **THE FAMILY'S** situation **DURING THE PAST YEAR**, e.g., **We** avoided restaurants because of coeliac disease.

| STATEMENT | ALL OF THE TIME | MOST OF THE TIME | SOME OF THE TIME | NEVER |
|---|---|---|---|---|
| 23. I (we) **avoided restaurants** because of coeliac disease. | ☐ | ☐ | ☐ | ☐ |
| 24. I (we) **avoided travelling** because of coeliac disease. | ☐ | ☐ | ☐ | ☐ |
| 25. I (we) **brought gluten-free food** when I (we) travelled. | ☐ | ☐ | ☐ | ☐ |
| 26. I was (we were**) not invited out for meals** because of coeliac disease. | ☐ | ☐ | ☐ | ☐ |
| 27. I (we) worried about **staying in hospital** because of coeliac disease. | ☐ | ☐ | ☐ | ☐ |

| | | | | |
|---|---|---|---|---|
| 28. I (we) found it **difficult to find gluten-free food** in the stores. | ☐ | ☐ | ☐ | ☐ |
| 29. I (we) found it difficult to find **good quality** gluten-free food. | ☐ | ☐ | ☐ | ☐ |
| 30. I ate gluten-containing food because I (we) **could not afford gluten-free food.** | ☐ | ☐ | ☐ | ☐ |
| 31. I (we) found it **difficult to determine if foods were gluten-free** from reading their labels. | ☐ | ☐ | ☐ | ☐ |

ADULTS **(18 years of age and over),** please go to question 38.

QUESTIONS FOR CHILDREN **(under 18 years of age)**

**PLEASE TICK EACH STATEMENT** that best describes **YOUR** situation **DURING THE PAST YEAR.**

If you are completing this questionnaire on behalf of a child under 14 years of age, **PLEASE READ THE FOLLOWING STATEMENTS TO THE CHILD AND RECORD THE CHILD'S RESPONSE**.

| STATEMENT | ALL THE TIME | MOST OF THE TIME | SOME OF THE TIME | NEVER |
|---|---|---|---|---|
| 32. **I felt left out of activities** at school or friends' homes because of coeliac disease. | ☐ | ☐ | ☐ | ☐ |
| 33. **I felt different from other kids** because of coeliac disease. | ☐ | ☐ | ☐ | ☐ |
| 34. **I felt embarrassed** because I had to bring gluten-free foods to parties at school or at friends' homes. | ☐ | ☐ | ☐ | ☐ |
| 35. **I felt angry** about having to follow a special diet. | ☐ | ☐ | ☐ | ☐ |
| 36. **I felt my teachers and friends did not understand** why I couldn't eat foods with gluten in them. | ☐ | ☐ | ☐ | ☐ |

37. **I felt that I could be healthy** without following a special diet. ☐ ☐ ☐ ☐

**SECTION 5: BONE DISEASE**

38.a) Have you **EVER** broken any bones ? ☐ **YES** ☐ **NO**

b) **If YES**, **PLEASE TICK ALL THAT APPLY**. Please indicate whether you broke the right or the left bone, and if you broke a bone more than once, how many times.

| | **YES** | **If YES, I broke the** | | **If YES, how many times I broke the same bone** | |
|---|---|---|---|---|---|
| | | **right** | **left** | **right** | **left** |
| i) Wrist(s) | ☐ | ☐ | ☐ | _____ | _____ |
| ii) Hip(s) | ☐ | ☐ | ☐ | _____ | _____ |
| iii) Rib(s) | ☐ | ☐ | ☐ | _____ | _____ |
| iv) Foot (feet) | ☐ | ☐ | ☐ | _____ | _____ |
| v) Ankle(s) | ☐ | ☐ | ☐ | _____ | _____ |
| vi) Shoulder(s) | ☐ | ☐ | ☐ | _____ | _____ |
| vii) Pelvis | ☐ | ☐ | ☐ | _____ | _____ |
| viii) Vertebrae (spine) | ☐ | ☐ | ☐ | _____ | _____ |

39. a) Have you had your bone density measured ? ☐ **YES** ☐ **NO**

b) **If YES,** how was your bone density? ☐ **NORMAL** ☐ **BELOW NORMAL** ☐ **DON'T KNOW**

40. a) Have you been diagnosed with

i) osteopaenia ? ☐**YES** ☐**NO** ☐ **DON'T KNOW**

ii) osteoporosis ? ☐**YES** ☐**NO** ☐ **DON'T KNOW**

b) **If YES** to **either** osteopaenia or osteoporosis, please indicate how old you were when you were diagnosed with either of these conditions.

_____ Age

c) **If YES** to **either** osteopaenia or osteoporosis, **PLEASE TICK ALL** of the following treatments you are presently taking

i) Calcitonin (Miacalcic) ☐

ii) Alendronate (Fosamax) ☐

iii) Etidronate (Didronel) ☐

iv) Hormone Replacement Therapy ☐

v) Testosterone ☐

vi) Raloxifene (Evista) ☐

vii) Calcium ☐

viii) Vitamin D ☐

ix) Other - please list _____

**SECTION 6: REPRODUCTION**

**MEN: Please go to question 49. CHILDREN** (under 18): **Please go to question 53.**

**QUESTIONS FOR WOMEN**

41. What age were you when your menstrual periods began? _____years

42. Did you ever have long absences (three months or more) of your periods? ☐ **YES** ☐ **NO**

43. Have you had trouble conceiving children? ☐ **YES** ☐ **NO** ☐ **NOT APPLICABLE**

44. Have you ever been treated for infertility? ☐ **YES** ☐ **NO**

45. How many full term pregnancies have you had? _____

46. a) Have you ever had a miscarriage? ☐ **YES** ☐ **NO**

b) **If YES**, please indicate how many miscarriages _____

47. a) Have you had a child (or children) with spina bifida? ☐ **YES** ☐ **NO**

b**) If YES**, please indicate how many _____

48. If you are post-menopausal, at what age did your periods stop? _____years

**WOMEN: Please go to question 53.**

**QUESTIONS FOR MEN**

49. Has your spouse had trouble conceiving children? ☐ **YES** ☐ **NO** ☐ **NOT APPLICABLE**

50. Have you ever had a decreased sex drive? ☐ **YES** ☐ **NO**

51. a) Have you had a test for sperm count? ☐ **YES** ☐ **NO**

b) **If YES**, please indicate if it was**:** ☐ **NORMAL** ☐ **BELOW NORMAL**

52. a) Have you had other tests for fertility? ☐ **YES** ☐ **NO**

b) **If YES**, please list what tests you have had.

_____

***Sections 7 & 8 are optional. WE WOULD PREFER THAT YOU COMPLETED SECTIONS 7 & 8. If you chose not to complete sections 7 & 8, please go to Section 9, page 14.***

SECTION 7: COELIAC DISEASE AMONG FAMILY MEMBERS

53 a) On the following chart**, PLEASE LIST *ALL* OF YOUR BIOLOGICAL FIRST-DEGREE FAMILY MEMBERS (both alive and deceased).** This includes your mother, father, sisters, brothers, daughters, and sons. Please list them by their relationship to you, e.g., sister 1, sister 2, son 1, son 2, etc. **This list will be used again in questions 53 - 64.**

**Please TICK** whether any of these listed 1st-degree relatives have had a **biopsy-confirmed diagnosis of coeliac disease.**

| **LIST OF 1st-DEGREE RELATIVES** . | **BIOPSY-CONFIRMED COELIAC DISEASE** |
|---|---|
| 1. Mother | ☐ **Yes** |
| 2. Father | ☐ **Yes** |
| 3. | ☐ **Yes** |
| 4. | ☐ **Yes** |
| 5. | ☐ **Yes** |
| 6. | ☐ **Yes** |
| 7. | ☐ **Yes** |
| 8. | ☐ **Yes** |
| 9. | ☐ **Yes** |
| 10. | ☐ **Yes** |

b) In the chart below **please TICK whether any of your 1st - and 2nd degree relatives** (who **HAVE NOT** been diagnosed with coeliac disease), **have symptoms** that lead you to suspect that they might have coeliac disease, and number **how many of these relatives** have coeliac symptoms.

| YOUR 1st-DEGREE RELATIVES | WHO HAS COELIAC SYMPTOMS? | HOW MANY RELATIVES? | YOUR 2nd-DEGREE RELATIVES | WHO HAS COELIAC SYMPTOMS? | HOW MANY RELATIVES? |
|---|---|---|---|---|---|
| Mother | ☐ | ___ | Grandmother(s) | ☐ | |
| Father | ☐ | ___ | Grandfather(s) | ☐ | |
| Sister(s) | ☐ | | Granddaughter(s) | ☐ | |
| Brother(s) | ☐ | | Grandson(s) | ☐ | |
| Daughter(s) | ☐ | | | | |
| Son(s) | ☐ | | | | |

c) **If any,** how many of your undiagnosed relatives have ever gone onto a gluten-free diet? _____

**SECTION 8. OTHER DISEASES AMONG YOU AND YOUR FIRST-DEGREE RELATIVES**

The following section asks about other diseases that you and your first-degree relatives have. For statistical reasons it is important to know if any of your first-degree relatives that you listed in 53.a) will be completing this questionnaire.

54. a) Are any of your first-degree relatives members of Coeliac New Zealand Incorporated

.

□ **YES** □ **NO** □ **DON'T KNOW**

b) **If YES,** how many of these relatives will be completing this questionnaire? _____

**SECTION 8 (cont'd).** Using the list of your first-degree relatives from **question 53. a), please write the relatives** across the top of this chart. **PLEASE TICK ALL DISEASES WITH WHICH YOU, AND THESE RELATIVES, HAVE BEEN DIAGNOSED.**

**You and your relatives listed in Question 53.a)**

| Medical Condition | Myself | 1 Mother | 2 Father | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|
| **55. Gastrointestinal and urinary** | | | | | | | | | | |
| a) Colitis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| b) Crohn's disease | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| c) Primary biliary cirrhosis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| d) Collagenous colitis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| e) Irritable bowel syndrome | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| f) Stomach or duodenal ulcers | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| g) Lactose intolerance | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **56. Cancer** | | | | | | | | | | |
| a) Mouth | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| b) Oesophagus | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| c) Stomach | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| d) Small intestine | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| Medical Condition | Myself | 1 Mother | 2 Father | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|
| e) Colon | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| f) Lymphoma | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| g) Skin | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| h) Melanoma | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| i) Lung | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| j) Breast | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **57. Benign tumor of gastrointestinal tract** | | | | | | | | | | |
| a) Colon polyps | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **58. Immune system** | | | | | | | | | | |
| a) Auto-immune hepatitis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| b) IgA deficiency | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| c) Multiple sclerosis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| d) Lupus | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| e) Refractory coeliac disease | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| f) Sjogren's syndrome | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| g) Food allergies | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **59. Blood** | | | | | | | | | | |
| a) Iron deficiency anemia | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| Medical Condition | Myself | 1 Mother | 2 Father | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|
| b) Folate deficiency anemia | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| c) Vit. $B_{12}$ deficiency anemia | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| d) Hyposplenism | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **60. Endocrine** | | | | | | | | | | |
| a) Addison's disease | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| b) Type 1 diabetes (insulin dependent ) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| c) Type 2 diabetes (non-insulin dependent) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| d) Hypothyroidism | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| e) Thyroiditis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| f) Turner's syndrome | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **61. Nervous system** | | | | | | | | | | |
| a) Migraine headaches | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| b) Peripheral neuropathy | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| c) Epilepsy | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| d) Depression | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| e) Alzheimer's disease | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| f) Attention deficit disorder (ADD/ADHD) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| Medical Condition | Myself | 1 Mother | 2 Father | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|
| g) Multiple sclerosis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| h) Anxiety attacks | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| i) Schizophrenia | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| j) Autism | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| k) Down's syndrome | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| l) Ataxia | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

**62. Musculoskeletal**

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| a) Fibromyalgia | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| b) Sarcoidosis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| c) Osteoarthritis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| d) Rheumatoid arthritis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| e) Osteopaenia | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| f) Osteoporosis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| g) Raynaud's syndrome | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| h) Sacroileitus | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| i) Scleroderma | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| Medical Condition | Myself | 1 Mother | 2 Father | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|
| **63. Pediatric** | | | | | | | | | | |
| a) Failure to thrive | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| b) Short stature | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| c) Dental enamel defects | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| d) Delayed puberty | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **64. Other** | | | | | | | | | | |
| a) Dermatitis herpetiformis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| b) Alopecia areata (patchy hair loss) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| c) Eczema | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| d) Psoriasis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| e) Pneumonia | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| f) Asthma | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| g) Cystic fibrosis | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| h) High cholesterol | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| i) High blood pressure | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| j) Loss of vision -please give details | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

_____

**SECTION 9. OTHER INFORMATION**

**65. PLEASE TICK** the highest level of education you (or your child) have obtained

a) Not started school yet ☐

b) Year 8 or less ☐

c) Some high school ☐

d) High school completed ☐

e) Vocational school or some university/college ☐

f) University degree ☐

g) Post Graduate degree ☐

66. Do you (or does your child) have a Pharmac Special Authority number? ☐ **YES** ☐ **NOT SURE** ☐ **NO**

67. Do you (or your child) receive a Disability Allowance? ☐ **YES** ☐ **NOT SURE** ☐ **NO**

68. Are you a member of Coeliac New Zealand Incorporated? ☐ **YES** ☐ **NOT SURE** ☐ **NO**

**SECTION 10: YOUR RECOMMENDATIONS**

69. From the following list, **PLEASE TICK the TWO ITEMS** that you believe would contribute the most to **IMPROVING THE LIVES OF INDIVIDUALS WITH COELIAC DISEASE.**

**PLEASE TICK TWO**

a) Earlier diagnosis of coeliac disease ☐

b) Better labelling of gluten-containing ingredients in foods ☐

c) Gluten-free choices on restaurant menus ☐

d) Better dietary counselling, especially for newly diagnosed coeliac patients ☐

e) More gluten-free foods in the supermarket ☐

f) Other - please specify ☐

_____
___

70. Do you have any comments/ information/ suggestions regarding any of the above topics? (Please state):

_____
_____
_____
_____
_____
_____
_____
____

_____
_____

_____
_____

_____
_____

71. Would you like to go into the draw to win a $50 book voucher?

☐ **YES** ☐ **NO**

72. If yes, please write your name and postal address in the space below.

_____
_____
_____
_____
_____
_____

_____
_____
_____

**THANK YOU FOR YOUR TIME,**

**YOUR PARTICIPATION IN THIS SURVEY IS GREATLY APPRECIATED.**

## Appendix 7: Cover letter – Email

**Sent:** Sunday, July 01, 2012 10:11 AM
**Subject:** University of Otago Research Study Invite

Coeliac New Zealand

Dear Participant,

**University of Otago Research Study**
Further to our email last week, here is your invitation to take part in the University of Otago survey.

**Simply take part and you can enter the draw to win a $50 book voucher.**

The study aims to describe the health of individuals with coeliac disease in New Zealand which also covers the extent of on-going symptoms due to ingestion of gluten through contaminated gluten-free food products.

To take the survey click on this link https://www.surveymonkey.com/s/57276CN and follow the instructions. Completion of the survey should take no longer than 15 minutes and is available online until the **3 August 2012.**

If you don't wish to take the survey, simply delete this email. If you would prefer to fill out a paper copy of this survey, please email your name and postal address to kirisharp@gmail.com and one will be sent to you to complete.

You are welcome to pass this email onto others with coeliac disease who may be interested in taking the survey.

Thank you for your time and participation, please do not hesitate to contact University of Otago if you have any questions. An information sheet with details about the study as well as contact details can be downloaded here

UNIVERSITY of OTAGO
Te Whare Wānanga o Otāgo

Kiri Sharp
Masters of Dietetics student
Principe Investigator
University of Otago

Dr Kirsten Coppell
Senior Research Fellow
Research Supervisor
University of Otago

**Coeliac New Zealand Incorporated**
PO Box 35724, Browns Bay, Auckland 0753

Visit our website | Contact Us | Unsubscribe

**www.coeliac.org.nz**

## Appendix 8: Cover letter – Postal

June 2012

Dear Participant

We are inviting people who are members of Coeliac New Zealand Incorporated to participate in a study about the health of individuals with Coeliac Disease in New Zealand by taking part in a survey.

The study aims to describe the health of individuals with Coeliac Disease in New Zealand and to determine the extent of on-going symptoms due to ingestion of gluten through contaminated gluten-free food products.

Please find enclosed an information sheet with details about the study, contact details if you have any questions about the study and the study survey to fill out if you choose to participate.

Please return completed survey to Kiri Sharp by posting in the self-addressed envelope enclosed.

Please do not hesitate to contact us if you have any questions.

Dr Kirsten Coppell
Masters of Dietetics student Senior Research Fellow

Kiri Sharp
Principle Investigator Research Supervisor

## Appendix 9: Study Invitation – Email

**Sent:** Thursday, June 21, 2012 6:03 PM

**Subject:** June Newsletter

Coeliac New Zealand

# June Newsletter

**View in your browser**

Dear Participant

Magazine vouchers

Don't forget to use the vouchers in your Winter issue of Coeliac Link before they expire! There is a $1 off any gluten free cereals from Hubbards, 10% off Orgran pastas from IE Produce, a buy-one-get-one-free from Muffin Break and the free try-it voucher from Purebread!

Survey Invitation

## Dates for the Diary...

**20 August -** *Coeliac Link* spring due out

**25-26 August -** Wellington Gluten Free Food & Allergy Show

**27-29 October -** Christchurch Gluten Free Food & Allergy Show

Win with Post-it® Flags and Healthy Food Guide

# University of Otago Research Study:

## Investigating the health of those with coeliac disease

The University of Otago is conducting a study that will inve
the health of people with coeliac disease in NZ which also
the extent of on-going symptoms due to ingestion of gluter
contaminated gluten-free food products.

They invite you to participate in an anonymous online surv
your coeliac disease: it should take less than 15 minutes. T
will be sent out via our e-newsletter early next week.

**Win...**
Complete the survey and go in the draw to win a $50 book

Participation will help provide a good overall description of
disease, as well as information about the needs of people a
lies with coeliac disease. A summary of the results will be p
in *Coeliac Link* once available.

Background information on the survey will be supplied wit
e-newsletter invite next week.

UNIVERSITY of OTAGO
Te Whare Wānanga o Otāgo
NEW ZEALAND

Enter the draw to win one of two **Kitchen-Aid® Artisan Stand Mixers KSM150** worth $1000 each, or one of 200 runner-up prizes of handy heart-shaped Post-it® Pop-up Note dispensers, which all come with a pack of Post-it® Pop-up Notes – perfect for the home-office or kitchen.

Enter now! Terms and conditions apply.Competition closes Sunday 22 July 2012.

### Contact Us

General Enquiries
T: 09 820 5157 (Helpline)
F: 09 414 7468
E:admin@coeliac.org.nz

### Useful Links

**Announcements**
**Shopping Guide**
**Members Login**

**Follow us on Facebook**

**Coeliac New Zealand Incorporated**
PO Box 35724, Browns Bay, Auckland 0753
T: 09 820 5157 F: 09 414 7468
E: admin@coeliac.org.nz W: www.coeliac.org.nz

## Appendix 10: Information Sheet – Email

# Information Sheet for The health of people with Coeliac Disease in New Zealand and the impact of a gluten free diet and gluten contaminated food products study.

This study will survey members of the Coeliac New Zealand Incorporated to seek to describe the health of people with Coeliac Disease in New Zealand and the impact of a gluten free diet. On-going symptoms of Coeliac Disease can occur through 'non-accidental' ingestion of gluten containing products and 'accidental' ingestion of gluten contaminated gluten-free products once a gluten free diet has been implemented. The latter is thought to contribute to on-going symptoms for an unknown proportion of people with coeliac disease. The results of this study will inform food production, food handling, labelling processes, and dietitians. The findings of the study will be published in the Coeliac New Zealand Incorporated magazine.
Please read this information sheet carefully before deciding whether you want to participate.

**Investigator:** Kiri Sharp

**Investigators and Supervisors:** Dr Kirsten Coppell
Heather Walker

**Study Aim**
The aim of this study is describe the health of people with Coeliac Disease in New Zealand and the impact of a gluten free diet and gluten contaminated food products.

**Who can participate?**
Individuals with Coeliac Disease resident in New Zealand.

**What is involved?**
We would like you to complete a survey about your coeliac disease. The link to this survey is attached in this email. Please forward this link onto any other people you know who have coeliac disease who you think will be willing to participate in this survey. If you prefer to fill out a paper copy of this survey, please email your name and postal address in reply to this email and one will be sent to you to complete. If you complete the paper copy of the questionnaire, please return it in the self-addressed stamped envelope. This survey consists of 10 sections, 2 of which are optional to complete. Completion of the questionnaire should take Edgar National Centre for Diabetes and Obesity Research Department of Medicine, PO Box 913, Dunedin 9054, New Zealand. Tel +64 3 470 3805 • Fax +64 3 474 7641 • Emai l *diabetes.research@otago.ac.nz*
*www.otago.ac.nz/diabetes*

no longer than 15 minutes. By completing this survey you have the chance to enter a draw for a $50 book voucher.

**What are the benefits?**
Participating in this research will provide a good overall description of coeliac disease in New Zealand, as well as information about the needs of people and families with coeliac disease. The results will also inform food production, food labelling and food handling processes.

**Possible risks**
There are no anticipated risks. All completed questionnaires will remain confidential and no identifying information will be retained.

**Confidentiality**
Confidentiality will be maintained about any information relating to you in this study. This means that your name and mailing information will be kept confidential to the investigators. Returned questionnaires will be viewed by only two of the investigators (Kiri Sharp and Dr Kirsten Coppell). Following receipt of questionnaires names and mailing addresses will be deleted. No reports arising from this study will include information which could identify you personally.

**General**
If you have any queries of concerns regarding your rights as a participant in this study you may wish to contact a Health and Disability Advocate, phone: (03) 479-0265 or 0800-377- 766, email: advocacy.services@xtra.co.nz, post: Advocacy Services South Island Trust, PO Box 1307, Christchurch.

**Statement of Approval**
This study has received ethical approval from the Multi-regional Ethics Committee.

**Where can I get more information?**
For more information, in the first instance, please contact:
Kiri Sharp, Master of Dietitics Student
Ph: 06 354 3635Edgar National Centre for Diabetes and Obesity Research Department of Medicine, PO Box 913, Dunedin 9054, New Zealand. Tel +64 3 470 3805 • Fax +64 3 474 7641 • Emai l *diabetes.research@otago.ac.nz www.otago.ac.nz/diabetes*

Email: shaki801@student.otago.ac.nz
Post: Coeliac Health Survey Study
6 Greenwood Place
Fitzherbert
Palmerston North
Dr Kirsten Coppell
Ph: +64 3 474 7774
Email: kirsten.coppell@otago.ac.nz
Post: Coeliac Health Survey Study
Edgar National Centre for Diabetes and Obesity Research
Department of Medicine
PO Box 913
Dunedin

Thank you for your time and participation.

## Appendix 11: Information Sheet – Postal

# Information Sheet for The health of people with Coeliac Disease in New Zealand and the impact of a gluten free diet and gluten contaminated food products study.

This study will survey members of the Coeliac New Zealand Incorporated to seek to describe the health of people with Coeliac Disease in New Zealand and the impact of a gluten free diet. On-going symptoms of Coeliac Disease can occur through 'non-accidental' ingestion of gluten containing products and 'accidental' ingestion of gluten contaminated gluten-free products once a gluten free diet has been implemented. The latter is thought to contribute to on-going symptoms for an unknown proportion of people with coeliac disease. The results of this study will inform food production, food handling, labelling processes, and dietitians. The findings of the study will be published in the Coeliac New Zealand Incorporated magazine.
Please read this information sheet carefully before deciding whether you want to participate.

**Investigator:** Kiri Sharp
**Investigators and Supervisors:** Dr Kirsten Coppell
Heather Walker

**Study Aim**
The aim of this study is describe the health of people with Coeliac Disease in New Zealand and the impact of a gluten free diet and gluten contaminated food products.

**Who can participate?**
Individuals with Coeliac Disease resident in New Zealand.

**What is involved?**
We would like you to complete a survey about your coeliac disease. A copy of this survey is enclosed with this letter. Please return the survey in the self-addressed stamped envelope. This survey consists of 10 sections, 2 of which are optional to complete. Completion of the questionnaire should take no longer than 15 minutes. By completing this survey you have the chance to enter a draw for a $50 book voucher.Edgar National Centre for Diabetes and Obesity Research Department of Medicine, PO Box 913, Dunedin 9054, New Zealand. Tel +64 3 470 3805 • Fax +64 3 474 7641 • Emai l *diabetes.research@otago.ac.nz www.otago.ac.nz/diabetes*

**What are the benefits?**
Participating in this research will provide a good overall description of coeliac disease in New Zealand, as well as information about the needs of people and families with coeliac disease. The results will also inform food production, food labelling and food handling processes.

**Possible risks**
There are no anticipated risks. All completed questionnaires will remain confidential and no identifying information will be retained.

**Confidentiality**
Confidentiality will be maintained about any information relating to you in this study. This means that your name and mailing information will be kept confidential to the investigators. Returned questionnaires will be viewed by only two of the investigators (Kiri Sharp and Dr Kirsten Coppell). Following receipt of questionnaires names and mailing addresses will be deleted. No reports arising from this study will include information which could identify you personally.

**General**
If you have any queries of concerns regarding your rights as a participant in this study you may wish to contact a Health and Disability Advocate, phone: (03) 479-0265 or 0800-377-766, email: advocacy.services@xtra.co.nz, post: Advocacy Services South Island Trust, PO Box 1307, Christchurch.

**Statement of Approval**
This study has received ethical approval from the Multi-regional Ethics Committee.

**Where can I get more information?**
For more information, in the first instance, please contact:
Kiri Sharp, Master of Dietitics Student
Ph: 06 354 3635
Email: shaki801@student.otago.ac.nz
Post: Coeliac Health Survey Study
6 Greenwood Place
FitzherbertEdgar National Centre for Diabetes and Obesity Research Department of Medicine, PO Box 913, Dunedin 9054, New Zealand. Tel +64 3 470 3805 • Fax +64 3 474 7641 • Emai l *diabetes.research@otago.ac.nz www.otago.ac.nz/diabetes*

Palmerston North
Dr Kirsten Coppell
Ph: +64 3 474 7774
Email: kirsten.coppell@otago.ac.nz
Post: Coeliac Health Survey Study
Edgar National Centre for Diabetes and Obesity Research
Department of Medicine
PO Box 913
Dunedin

Thank you for your time and participation.

## Appendix 12: First and Second Reminder Letters – Email

**Sent:** Thursday, July 19, 2012 6:14 PM
**Subject:** July Newsletter

Dear Participant,

**Reminder: University of Otago Research Study**
Recently you received an invitation to participate in the University of Otago Research Study. Thank you to all those who have already completed the survey - we have had a great response.
If you have not already completed the survey and would like to, the **survey closes 5 August 2012**. You can take the survey online at https://www.surveymonkey.com/s/57276CN and it should take no longer than 15 minutes. **WIN:** Complete the survey to enter the draw to win a $50 book voucher.

**Sent:** Thursday, August 02, 2012 6:05 PM
**Subject:** Reminder: University of Otago Research Study

Dear Participant,

**Final Reminder: University of Otago Research Study**

If you have not already completed this survey and would like to do so, the survey closing date has been extended until **Sunday 12 August 2012.** You can take the survey online at https://www.surveymonkey.com/s/57276CN and it should take no longer than 15 minutes.

The study aims to describe the health of individuals with coeliac disease in New Zealand which also covers the extent of on-going symptoms due to ingestion of gluten through contaminated gluten-free food products.

Thank you to all those who have already completed the survey.

**WIN: Complete the survey and enter the draw to win a $50 book voucher.**

## Appendix 13: Thank you to Coeliac New Zealand Members

**Sent:** Thursday, September 13, 2012 6:09 PM
**Subject:** September Newsletter

**A note of thanks from the University of Otago on the recent Research Study**
*"A huge thank you to all those people who participated in the New Zealand Coeliac Health Survey and encouraged others to participate also. Your time and effort was very much appreciated. The survey garnered valuable data regarding the health of people with coeliac disease in New Zealand. We are in the process of analysing and interpreting the results, when this is complete a summary will be published in Coeliac Link. Thank you again for your contribution - Kiri Sharp & Kirsten Coppell"*

## Appendix 14: Release of the Canadian Celiac Health Survey questionnaire + Terms and Conditions

Date: 06, April 2012

Dear Dr. Coppell,

Re: Release of the Canadian Celiac Health Survey questionnaire

Thank you for requesting the questionnaire of the Canadian Celiac Health Survey. The Canadian Celiac Association is pleased to release the questionnaire to you free of charge on the following terms and conditions:

(1). This questionnaire is only to be used for your study. It should not be shared with any other research group without the permission of the Canadian Celiac Association.

(2). The Canadian Celiac Association must be acknowledged in any abstract(s) or publication(s) that may arise from any research involving the questionnaire.

(3). The Canadian Celiac Association will be provided with a copy of any abstract(s) or publication(s) that may arise from the research using the questionnaire. The copy should be mailed to the Association's office.

The generic quality of life in the Canadian Celiac Health Survey was evaluated using the SF-12™ health survey. The Canadian Celiac Association does not have the copyright to SF-12™ and had obtained special permission from the owners for its use. Therefore, that quality of life section (Section 4, questions 21 to 32) from the Canadian Celiac Health Survey has been removed. You will have to contact the copyright owners directly for permission to use the SF-12™ health survey. (The reference is: Riddle DL, Lee MS, Stratford PW. Use of SF-36 and SF-12 health status measures a quantitative comparison for groups versus individual patients. *Med Care* 30:867-78, 2001.)

If you agree to the above terms, please let me know and also send a signed letter of acceptance of terms to the Canadian Celiac Association. Also, kindly send me your mailing address, phone and fax number. The questionnaire will be e-mailed to you as a MS Word file. You may modify the document to suit your needs.

With best wishes.

Dr. Mohsin Rashid
for
Professional Advisory Board and Board of Directors
Canadian Celiac Association
Mississauga, Ontario
Canada
PH: (905) 507-6208
FAX: (905) 507-4673

*THE CANADIAN CELIAC ASSOCIATION is a national organization dedicated to providing services and support to persons with celiac disease and dermatitis herpetiformis through programs of awareness, advocacy, education and research. (www.celiac.ca)*

---

Dr. Mohsin Rashid MBBS, MEd, FRCP(C)
Division of Gastroenterology & Nutrition
Department of Paediatrics, Dalhousie University

Mailing address:
IWK Health Centre
5850/5980 University Avenue
P.O. Box 9700
Halifax, Nova Scotia
Canada B3K 6R8

Ph: (902) 470-8746
FAX: (902) 470-7249
Email: mohsin.rashid@iwk.nshealth.ca