What Children are Eating and the Risk of Type 2 Diabetes Mellitus

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Abstract

Type 2 diabetes mellitus (T2DM) is an insidious, intergenerational disease. Although T2DM presents most often in adults, it is starting to present in childhood. In New Zealand (NZ), the overall prevalence of diagnosed T2DM in the adult population (≥15 years) is 5.8% with double the prevalence in the most deprived quintile of society (8.6% vs 4.4%). Prevalence differs by ethnicity: Māori (7.3%), Pacific (12.5%) and Asian (6.8%) compared with European (4.7%). Patterns of eating as well as rate of childhood growth and body size are three closely connected risk factors for T2DM that may be modifiable across the life course.

The key to T2DM prevention is taking into account one's whole diet and combinations of consumed foods, rather than one food item or nutrient. One in nine NZ children aged 2-14 years are obese (a major risk factor for T2DM). The rate of obesity among children living in the most deprived areas (20%) is higher than children living in the least deprived areas (4%).

Diagnosis of diabetes is a common clinical challenge. The measurement of glycated haemoglobin A_1c (HbA_1c) in human blood is an indicator of average blood glucose over the previous 2-3 months. This measurement plus elevated serum uric acid (SUA) concentration - a risk factor for T2DM- can assist in the identification of the profile for risk for T2DM.

The focus of this exploratory PhD was to identify groups of children with distinct eating patterns and investigate the relationships of eating patterns with risk of T2DM, using the blood biomarkers, HbA_1c and SUA concentrations. In addition, information concerning conditions at birth (i.e., maternal age, maternal education, birth weight, and gender) and body size (i.e., current weight, height and waist), were examined as possible cofactors and covariates.

Four connected investigations were undertaken to collect the information from high-risk children living in Auckland. To this aim, two longitudinal birth cohorts were studied: 1) the Pacific Islands Families (PIF) study at age 14 years, and 2) Metformin in Gestational Diabetes the Follow-up (MiGTOFU) study, at age 9 years.

Firstly, a performance and utility investigation of the Afinion^{TM} point of care test (POCT) for HbA_1c was conducted with 94 girls and 96 boys aged 15 years in a nested

subsample of the PIF cohort. In doing so, HbA_1c was measured three times on two different occasions. The first occasion was collecting samples at schools using a capillary finger-prick sample. A year after, the second sample collection was completed, using the same model POCT and finger-prick. Simultaneously, HbA_1c from a venous sample was analysed by boronate affinity chromatography at a certified laboratory. For the same day analysis, the mean difference in capillary and venous measures was 0.54 $mmol.mol^{-1}$ (0.05%) (95% CI mean: 0.25, 0.83, $p < 0.001$) and the ± 1.96SD limits of agreement: 4.48, -3.40 $mmol.mol^{-1}$. There was a moderate to strong correlation between the two POCT measures taken one year apart (r = 0.55, 95% CI [0.44, 0.65], p< 0.001) with a mean difference of 0.14 ± 2.18 (SD) $mmol.mol^{-1}$. The within-day difference between the reference and the POCT was less than the precision of the POCT and was not biologically or clinically meaningful. The Afinion POCT ™ AS100 test provided a valid and biologically reliable measure of HbA_1C and had the potential to identify children at risk of elevated HbA_1c.

The second investigation, studied the association of eating patterns of 931 PIF children at 14 years. This included a cluster analysis of the data from a self-reported dietary habits questionnaire with the concentration of HbA_1c measured with POCT. As a result of this, four eating patterns were derived for 740 children. In addition, the effect of body anthropometric measurements, body composition (i.e., measured by Bioimpedance Analysis) and some early life factors (i.e., maternal age at conception, maternal education, type of baby feeding, birth weight, and number of siblings) were evaluated. The mean of HbA_1c concentration was not different between four derived eating patterns. Waist-to-height ratio and mothers' age were positively (standardised β 0.108 $p = 0.03$) and negatively (standardised β -0.091 $p = 0.012$) related to HbA_1c $R^2 = 0.020$, but not the eating patterns.

In the third investigation, the associations of reported consumption of added-sugar foods and risk of T2DM, was measured among a nested subsample from the PIF cohort with 204 Pacific children aged 15 years old. Findings suggest that boys drank more sugary drinks and ate fewer snacks and sweets than girls. After adjusting for gender 'snacks and sweets' group and 'sugary drinks' group were negatively and positively related to SUA respectively ($R^2 = 0.309$), but no associations were found with HbA_1c concentration. There was a positive and significant association between SUA and all the body size measurements. However, HbA_1c concentration was positively and

significantly related to weight, BMI, waist circumference, waist-to-height ratio, FM and FFM.

For the last investigation data was sourced from the 'Metformin in Gestational Diabetes the Follow-up' study when the offspring (n = 99) of mothers treated for gestational diabetes mellitus were aged 7-9 years. Food consumption, HbA_1body measurements including dual energy x-ray absorptiometry measurements and early life factors (e.g., maternal age at conception, maternal education, mother's type of treatment, type of baby feeding, birth weight, and ethnicity) were included in the analysis. Five eating patterns were derived as a result of cluster analysis. After adjusting for waist-to-height ratio, there was almost a positive significant difference (p = 0.052) between 'refined carbs' and 'not refined carbs' eating patterns for HbA_1c (1.4 $mmol.L^{-1}$) (95% CI [-0.2, 2.8]). Eating patterns were associated with weight z scores, height z scores and visceral abdominal fat.

Evidence presented in this thesis demonstrates that these high-risk groups of children are not meeting the NZ food guidelines and that their eating patterns are weakly associated with T2DM risk factors. Age and relative homogeneity of the children are the probable reasons for not finding associations of eating patterns among these apparently healthy children with a relatively high prevalence of overweight and obesity. Additionally, the methods and methodology used to collect dietary information, methodological difficulties in assessing the diets and the cross-sectional design of the analyses may have contributed. The findings of this thesis support initiatives to inform positive actions such as research, benchmarking, and quality improvement of the food environment. From this point of view, this research would benefit individuals, communities, and policy makers.

Attestation of Authorship

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person (except where explicitly defined in the acknowledgements), nor material which to a substantial extent has been submitted for the award of any other degree or diploma of a university or other institution of higher learning.

Signed:

Date: 12/10/2018

Acknowledgments

"This ocean of being has flooded from the indiscernible

No one has pierced this pearl of research

Everyone has expressed an absurd opinion

Reality, as it is, nobody can describe"

Omar Khayyam

This thesis is the destination of my PhD journey. This journey is challenging, but I have not undertaken it alone. At different stages of this journey, various generous people in one way or another granted me their valuable assistance in facing different challenges and as a result, every day I could pass a new milestone. Every time I stopped to catch my breath, I looked back and realized the once tough challenge transformed into another skill that I could obtain. This is a thank you to all the people who took a walk with me and made my journey of PhD memorable.

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Table of Contents

Abstract i
Attestation of Authorship iv
Acknowledgments v
List of Figures xii
List of Tables xiv
Abbreviations xvi
Chapter 1 Introduction 1
1.1 Preface 4
1.2 Overview of the studies 6
Chapter 2 Literature review: type 2 diabetes mellitus and children 9
2.1 Emergence of type 2 diabetes mellitus in children 9
2.1 Diagnosis and screening for risk of type 2 diabetes mellitus in children 11
2.1.1 Glycated haemoglobin A_1c 12
2.1.2 Point of care and screening diabetes mellitus 13
2.2 Risk factors for type 2 diabetes mellitus 14
2.2.1 Life exposure, food consumption and type 2 diabetes mellitus 15
2.2.2 Growth and type 2 diabetes mellitus 17
2.2.3 Breastfeeding and type 2 diabetes mellitus 19
2.2.4 Maternal education 20
2.3 Eating patterns 21
2.3.1 Eating patterns among New Zealand children 24
2.3.2 Eating patterns, ethnicity and type 2 diabetes mellitus 26
2.4 Growth trajectory, diabetes biomarkers and eating patterns: cohort studies 26
2.5 Sugar-Sweetened Beverages 28
2.6 Glycaemic index and glycaemic load of the diet 29
2.7 Summary 30
Chapter 3 Literature review: identification of eating patterns 32
3.1 Theoretically defined eating patterns (*a priori*) 33
3.2 Empirically derived eating patterns (*a posteriori*) 34
3.3 Cluster analysis 38
3.3.1 Advantages of using cluster analysis for generating eating patterns 41
3.3.2 Limitations of using cluster analysis 42
3.4 Rationale for derivation of eating patterns in this body of work 43
Chapter 4 Point of care test for HbA_1c: performance and utility 44
4.1 Introduction 44
4.2 Methods 46
4.2.1 Participants 46
4.2.2 Laboratory method 46
4.2.3 Point-of-care device method 47

4.2.4 Statistical analysis 47
4.3 Results 48
4.3.1 Validity phase 48
4.3.2 Agreement of POCT measures over time 51
4.4 Discussion 51
4.5 Recommendation 53
4.6 Conclusion 53
Chapter 5 Eating patterns and risk of type 2 diabetes mellitus: Pacific Islands Families main study 54
5.1 Introduction 54
5.2 Design and methods 58
5.2.1 Study participants 59
5.2.2 Ethical considerations 59
5.2.3 Early life information 59
5.2.4 Physical measurements at age 14 years 59
5.2.5 Measurement of Glycated Haemoglobin A_1c (HbA_1c) 60
5.2.6 Anthropometry measurements 60
5.2.7 Body composition measurements 61
5.2.8 Food information 61
5.2.9 Statistical analysis 62
5.3 Results 66
5.3.1 Maternal characteristics, birth and measurements of children at birth and 14 years 66
5.3.2 Associations between HbA_1c and body measurements at 14 years 69
5.3.3 Associations between HbA_1c, gender and early life information 70
5.3.4 Foods consumption reported by the children at 14 years 70
5.3.5 Identification of eating patterns 78
5.3.6 Associations between eating patterns and HbA_1c 80
5.3.7 Associations between eating patterns and body measurements at age 14 years 80
5.3.8 Associations between eating patterns and early life information 81
5.3.9 Regression analysis 81
5.3.10 Summary of main findings 83
5.4 Discussion 85
5.4.1 Food choices and eating patterns among Pacific children 87
5.4.2 Eating patterns, foods consumption and HbA_1c 91
5.4.3 Eating patterns and current body size measurements 93
5.4.4 Current body size measurements and HbA_1c 95
5.4.5 Early life factors and type 2 diabetes mellitus 96
5.4.6 Maternal education, age at conception and type 2 diabetes mellitus 97
5.5 Strengths and limitations 98
5.6 Recommendations 100
5.7 Conclusion 100

Chapter 6 Sweet food consumption and risk for type 2 diabetes mellitus: Pacific Islands Families sub-study 102
6.1 Introduction 102
6.2 Design and methods 106
6.2.1 Study participants 106
6.2.2 Ethical considerations 106
6.2.3 Food information 107
6.2.4 Blood biomarkers 108
6.2.5 Body measurements 109
6.2.6 Carbohydrate content of individual food 109
6.2.7 Statistical analysis 110
6.3 Results 111
6.3.1 Body and blood measurements of children at 15 years 111
6.3.2 Association between body measurements and biomarkers 114
6.3.3 Food consumption reported by the children at 15 years 114
6.3.4 Associations between HbA_1c and food consumption 122
6.3.5 Associations between SUA concentration and food consumption 123
6.3.6 Regression and mediation analysis 123
6.3.7 Summary of main findings 125
6.4 Discussion 126
6.5 Strengths and limitations 131
6.6 Recommendations 131
6.7 Conclusion 132
Chapter 7 9-year Metformin in Gestational Diabetes the Follow-up Study 133
7.1 Introduction 133
7.2 Methods 138
7.2.1 Study participants 139
7.2.2 Ethical considerations 139
7.2.3 Sociodemographic information 139
7.2.4 Body measurements 140
7.2.5 Fasting venous blood sample 141
7.2.6 Food information 141
7.2.7 Glycemic index and glycemic load of individual foods 143
7.2.8 Statistical analysis 144
7.3 Results 148
7.3.1 Characteristics of children 148
7.3.2 Maternal and birth characteristics of children 148
7.3.3 Physical measurements of children at the age of 7-9 years 149
7.3.4 Associations between HbA_1c, gender, ethnicity and early life information 152
7.3.5 Associations between HbA_1c and body measurements 153
7.3.6 Foods consumption of children reported by the mothers 154
7.3.7 Consumption of foods and HbA_1c 158

7.3.8 Consumption of foods based on glycaemic index 159
7.3.9 Identification of eating patterns 159
7.3.10 Eating patterns and HbA_1c 163
7.3.11 Eating patterns and body measurements 164
7.3.12 Eating patterns and early life factors 164
7.3.13 Summary of main findings 165
7.4 Discussion 167
7.4.1 Eating patterns, foods consumption and T2DM 170
7.4.2 Eating patterns and food choices among children 170
7.4.3 Eating patterns and current body size measurements 174
7.4.4 HbA_1c 175
7.4.5 Current body size measurements and HbA_1c concentration 175
7.4.6 Influence of early life factors 176
7.5 Limitations and strengths 178
7.6 Recommendations 179
7.7 Conclusion 180
Chapter 8 Discussion and conclusion 181
8.1 Strengths and limitations 185
8.2 Recommendations for future research to inform policy 186
8.3 Conclusion 190
References 192

Appendix A: Pacific Island Families main study ethics approval 225
Appendix B: Pacific Island Families main study physical measurement form 229
Appendix C: Bioelectrical impedance analysis instruction 232
Appendix D: Pacific Islands Families main study nutrition and health questionnaire 233
Appendix E: Pacific Islands Families sub-study ethics approval 242
Appendix F: Pacific Islands Families sub-study food frequency questionnaire 246
Appendix G: Pacific Islands Families sub-study questionnaire tablet screen shot 264
Appendix H: Metformin in Gestational Diabetes the Follow-up study ethics approval 265
Appendix I: Metformin in Gestational Diabetes the Follow-up study questionnaire .. 266
Appendix J: Children food recommendations suggested by New Zealand Ministry of Health 270

List of Figures

Figure 1.1. Structure and flow of the thesis 7
Figure 1.2. Mediation model underpinning the research questions 8
Figure 2.1. Modifiable risk factors for type 2 diabetes mellitus in children 15
Figure 2.2. A conceptual framework for relationship between maternal education level and the health of children 21
Figure 3.1. Approaches to analysis of eating patterns 33
Figure 3.2. Cluster analysis step-by-step 40
Figure 4.1. Frequency of glycated haemoglobin A_1c (HbA_1c) measured by reference method and point of care 48
Figure 4.2. Bland-Altman plot of point of care and reference HbA_1c measurements 50
Figure 4.3. Bland-Altman plot of point of care and reference HbA_1c measurements without outliers 50
Figure 5.1. Investigation plan and variables considered for analysis 57
Figure 5.2. Frequency of glycated haemoglobin A_1c by gender among Pacific children at 14 years old 69
Figure 5.3. Type of bread chosen most of the time by gender 76
Figure 5.4. Type of milk chosen most of the time by gender 77
Figure 5.5. Scree plots of eigen values of food components 78
Figure 5.6. Height of the children at 14 years by eating patterns 81
Figure 5.7. Factors found with significant associations 85
Figure 6.1. Frequency of glycated haemoglobin A_1c by gender among Pacific children at 15 years old 112
Figure 6.2. Frequency of serum uric acid by gender among Pacific children at 15 years old 113
Figure 6.3. Mediation model including all Pacific children (n = 199) 124
Figure 6.4. Mediation model including Pacific girls (n = 96) 124
Figure 6.5. Mediation model including Pacific boys (n = 103) 124
Figure 6.6. Factors found with significant associations 126
Figure 7.1. Investigation plan and variables considered for analysis 137
Figure 7.2. Step-by step analysis procedure to derive eating patterns 145
Figure 7.3. Frequency of glycated haemoglobin A_1c concentration by gender among children at 7-9 years old 151
Figure 7.4. HbA_1c with waist circumference (n = 93) 153
Figure 7.5. HbA_1c with the waist-to-height ratio (n = 93) 153
Figure 7.6. Scree plots of eigen values of food components 160
Figure 7.7. Percentage of eating patterns in European and non-European children 162
Figure 7.8. Association between HbA_1c and the five derived eating patterns 163

Figure 8.1. Contribution of relative factors to rise in number of diagnosed type 2 diabetes mellitus 183

List of Tables

Table 2.1. The recommendations of the American Diabetes Association consensus statement on testing for type 2 diabetes in children aged 18 years and younger [a] 11

Table 2.2. Eating patterns named by researchers 24

Table 2.3. Studies focused on eating patterns of New Zealand children 25

Table 2.4. Research questions by chapter 31

Table 3.1. Examples of questions concerning eating patterns using *a priori* and *a posteriori* approaches and their associated methods 37

Table 5.1. Summary of variables for PIF main study 58

Table 5.2. Frequency of consumption of food and the applied weighting factor to standardise to a daily rate 64

Table 5.3. Number of servings of consumption of food per day and the applied weighting factor to standardise to a daily rate 64

Table 5.4. Characteristics of Pacific children at birth and maternal baseline information 67

Table 5.5. Physical measurements of Pacific children at age 14 years from the PIF main study 68

Table 5.6. Pacific children classified as thin, normal, overweight and obese based on international obesity task force cut-offs [a] 69

Table 5.7. Eleven dietary habits of 931 Pacific children at 14 years by gender (girls n = 463, boys n = 468) 72

Table 5.8. Daily frequency of consumption of fruit, vegetables, milk and bread of 931 Pacific children at 14 years by gender (girls n = 463, boys n = 468) 74

Table 5.9. Daily frequency of consumed foods by gender. Ranked by descending frequency of consumption 75

Table 5.10. Factor loading matrix derived from food or food groups for 4 major eating patterns [a] 79

Table 5.11. Gender distribution by eating patterns 80

Table 5.12. Point of care HbA_1c at 14 years by eating patterns 80

Table 5.13. Distribution of the children based on eating patterns and early life information 82

Table 5.14. Characteristics of the current study and articles included in the present discussion 86

Table 6.1. Food, biomarkers and body measurements included in the PIF substudy ... 106

Table 6.2. Predefined food groups based on the applied food frequency questionnaire in the PIF main study 108

Table 6.3. Frequency of food consumption and the applied weighting factor to standardise to a daily rate 108

Table 6.4. Physical measurements of Pacific children (n = 204) 111

Table 6.5. Frequency of food consumption by gender (girls = 100, boys = 104) 115

Table 6.6. Daily frequency of consumed foods by gender 119

Table 6.7. Number of times a week the foods with added sugar were consumed by Pacific children at age 15 years. Ranked by descending frequency of consumption ... 121

Table 6.8. Characteristics of the articles included in the present discussion 127

Table 7.1. Summary of variables for 9-year MiGTOFU [a] 138

Table 7.2. Frequency of consumption of food and the applied weighting factor to standardise to a daily rate 142

Table 7.3. Food grouping and the food items in each group..... 143

Table 7.4. Foods grouped based on the glycaemic index (GI)..... 143

Table 7.5. Food groups used for analysis of eating patterns 146

Table 7.6. Characteristics of children (n = 99) followed-up at age 9 years 148

Table 7.7. Characteristics of children at birth and maternal baseline information 149

Table 7.8. Physical measurements at 9 years 150

Table 7.9. Children classified as thin, normal, overweight and obese based on the international obesity task force cut-offs [a] 152

Table 7.10. HbA_1c by ethnic group..... 152

Table 7.11. Comparison of frequency of consumed food with Ministry of Health food guidelines (n = 99) 154

Table 7.12. Comparison of food consumption following Ministry of Health food Guidelines by ethnicity 156

Table 7.13. Frequencies of foods consumed each day overall and by gender for 99 children..... 157

Table 7.14. Frequency of consumption from low, medium and high glycaemic index foods each day by gender 159

Table 7.15. Factor loading matrix derived from food or food groups for 5 major factors [a] 161

Table 7.16. Gender distribution by eating patterns 162

Table 7.17. Characteristics of the articles included in the present discussion 168

Abbreviations

BF%	Percentage body fat
AUT	Auckland University of Technology
BIA	Bioelectrical impedance analysis
BMI	Body mass index
BMI z scores	Body mass index z scores
CI	Confidence intervals
CNS 2002	Children's nutrition survey
DEXA	Dual energy x-ray absorptiometry
DHBs	District Health Boards
DM	Diabetes
EP	Eating pattern
FFM	Fat free mass
FFQ	Food frequency questionnaire
FM	Fat mass
GDM	Gestational diabetes mellitus
HbA_1c	Glycated haemoglobin A_1c
IFG	Impaired fasting glucose
IQR	Interquartile range
MiG	Metformin in Gestational Diabetes
MiGTOFU	Metformin in Gestational Diabetes the Offspring Follow-Up
NCDs	Non-communicable diseases
NZ	New Zealand
OR	Odds ratio
PIF	Pacific Island Families study
POCT	Point of care test
RCT	Randomized controlled trial
SD	Standard deviation
SSBs	Sugar Sweetened Beverages
SUA	Serum uric acid
T1DM	Type 1 diabetes mellitus
T2DM	Type 2 diabetes mellitus

Chapter 1 Introduction

Young people are the future of the country and it is therefore important to optimize their future health. Type 2 diabetes mellitus (T2DM) is an insidious disease, which may have its origin in childhood, but most often presents itself in adulthood. This non-communicable disease significantly decimates society and has a severe and noticeable effect on health care systems (Bergman, Stefanovski, & Kim, 2014; Dasari, Oza-Frank, & Venkat Narayan, 2008). In children, the incidence of T2DM is increasing but in a paediatric population, early onset of diabetes mellitus (hyperglycaemia) may be due to Type 1 mellitus (T1DM), an autoimmune disease or monogenetic defects of pancreatic β cell function.

In NZ, the overall prevalence of diagnosed T2DM in the adult population (≥15 years) is 5.8% (one in 17 adults), which is around 205,000 adults (Ministry of Health, 2013b). Age standardised prevalence of T2DM in the most deprived quintile of society is twice that of the least deprived (8.6% vs 4.4%). Māori (7.3%), Pacific (12.5%), and Asian (6.8%) are more likely to develop T2DM than European (4.7%) (Ministry of Health, 2013b). Aligned with the NZ population health objective “to reduce the incidence and impact of diabetes” (Ministry of Health, 2000, p. 6), it is imperative to prevent future risks of T2DM, especially among Pacific, Māori, and Asian ethnic groups. Among Asian groups, Indians have increasing prevalence of T2DM (Kolt, Schofield, Rush, Oliver, & Chadha, 2007).

Individual-level factors (e.g., physical activity, dietary behaviors, and smoking) have been studied more than other risk factors associated with developing T2DM (Gary-Webb, Suglia, & Tehranifar, 2013). Dietary behaviors, (e.g., eating patterns) can affect the risk of developing T2DM (Gary-Webb et al., 2013), as can a poor diet that includes energy-dense, nutrient-poor foods with inadequate fruit and vegetables (Esposito et al., 2014; Ley, Hamdy, Mohan, & Hu, 2014). Investigations suggest that food choice and interactions between dietary components affect children’s health (Pala et al., 2013). Since food behaviour and choice persist into adulthood (Mikkilä, Räsänen, Raitakari, Pietinen, & Viikari, 2005), it is important to investigate the combinations of the foods that children eat to develop a comprehensive picture of contemporary children’s eating patterns. Here the key to identifying potentially modifiable risk factors for T2DM prevention is determining children’s diet and combinations of foods they eat, rather than

one food or nutrient (Alhazmi, Stojanovski, McEvoy, & Garg, 2014; Shirani, Salehi-Abargouei, & Azadbakht, 2013).

Diagnosis of diabetes is a common clinical challenge (Colagiuri, 2011). To do a traditional diagnostic test (e.g. a two-hour 75-gram oral glucose tolerance test), requires instruction to fast for eight hours before presentation to a laboratory in the morning. The two-hour 75-gram oral glucose tolerance test involves collecting a baseline blood glucose sample, asking the individual to drink 75g of glucose and to wait quietly for two hours, and finally taking another blood sample after two hours. In contrast the measurement of glycated haemoglobin (HbA_1c) in human blood is an indicator of average blood glucose over the previous 2-3 months (Garg et al., 2014). It is both a regular monitoring tool for people with diabetes (Hoelzel et al., 2004), which also assists in the diagnosis of diabetes (Garg et al., 2014). This only requires one blood sample and there is no need for fasting.

Beside the role of HbA_1c in T2DM diagnosis, serum uric acid (SUA) concentration is also showed strong relationship with T2DM as a factor (Xu et al., 2016). Quite recently, high SUA has been recognized as an independent risk factor for T2DM (Dehghan, van Hoek, Sijbrands, Hofman, & Witteman, 2008; Xu et al., 2016).

NZ's second national children health and wellbeing survey (Adolescent Health Research Group, 2008) revealed that since 2001 NZ's young population has grown more ethnically diverse. Currently, more than 20% (9,911,300) of the total population in NZ is aged between 0-14 years (Statistics New Zealand, 2014). However, the results of the NZ Health Survey for Children (Ministry of Health, 2012b) shows that the rate of obesity, which is a major risk factor for T2DM, among 5-14 year olds increased to 11% in 2011/12 from 9% in 2002 and 8% in 2006/07. The results of this survey also show that obesity was more common among Māori and Pacific children and children living in the most deprived areas (Ministry of Health, 2012b).

In 2013, there were 312,000 (21%) children aged 0 to 14 years in multi-ethnic Auckland region population of 1.493M (Statistics New Zealand, 2013). Considering the fact that some children could belong to multiple ethnic groups 57% of them were NZ European, 18% were Māori, 25% were Pacific, 22% were Asian, and 2% of the population was made of Middle Eastern, Latin American, and African ethnicities. In 2013, Counties Manukau District Health Board, one of three district health boards in the Auckland region, reported that 12% of all NZ Māori and 40% of all NZ Pacific peoples living in

NZ, reside in South Auckland (2013). More importantly South Auckland has a relatively young (24% aged ≤ 14years, 14% of NZ's child population), and diverse (38% European and other, 23% Pacific, 16% Māori, and 22% Asian) population (Counties Manukau District Health Board, 2013), with a growth rate of 2 - 3% per year (Counties Manukau District Health Board, 2013). As a result of this ethnic diversity and large numbers of children, additional research among this age-group will provide a firm basis for implementing policies and interventions to promote children's future wellbeing.

In an extensive search of MEDLINE, Web of Science, Science Direct, OVID, EBSCO and PubMed databases, a considerable amount of research has been published on eating patterns of children. In these studies, the association of derived eating patterns were examined against behavioural and nutrition-related diseases such as T2DM, HbA_1c or biomarkers of risk such as SUA concentrations. There are several studies that examined the correlation between eating patterns and obesity (e.g. Ambrosini et al., 2012; Bahreynian, Paknahad, & Maracy, 2013; Cutler, Flood, Hannan, Slavin, & Neumark-Sztainer, 2012; Howe, Black, Wong, Parnell, & Skidmore, 2013; and Pala et al., 2013). There are some studies that examined the relationship between eating patterns against specific diseases or outcomes, such as adiposity, cardiovascular, and also cognitive outcomes (Golley et al., 2013), cardiovascular disease (Perichart-Perera et al., 2010) and/ or atherosclerosis (Räsänen et al., 2002). There are a few studies that have examined eating patterns of children in NZ (e.g., Davison et al., 2017; Y. Liu, 2010; Wall, Thompson, Robinson, & Mitchell, 2013) and a few others on food consumption and diabetes using HbA_1c as the biomarker among children with T1DM (e.g., Lamichhane et al., 2015; and Meissner et al., 2014). Nevertheless, to the knowledge of the researcher there is a lack of research on healthy children.

Generally, it can be said that the majority of studies on children have focused on eating patterns and the risk of obesity in later life (e.g., Ambrosini, 2014; Newby, 2007). Interestingly, the cross-sectional relationship between eating patterns and the risk of T2DM has been studied more extensively among adults (e.g., Dekker et al., 2015; Montonen et al., 2005), and the reviews of associations reported (e.g., Jannasch, Kröger, & Schulze, 2017; and Maghsoudi, Ghiasvand, & Salehi-Abargouei, 2016) more frequently on adult studies compared to children. Generally, it was concluded that the risk of developing T2DM can be reduced by changing eating patterns, particularly by adding more fruit and vegetables to one's diet (Maghsoudi et al., 2016; Montonen et al.,

2005). Examples of diets which have been reported to reduce the incidence of T2DM are Mediterranean diet, Dietary Approaches to Stop Hypertension (DASH) and/ or Alternative Healthy Eating Index (AHEI) (Jannasch et al., 2017). However, to address the risk of T2DM in multi-ethnic populations, ethnic-specific approaches should be developed and encouraged (Dekker et al., 2015).

The pattern of eating among children is characterized by more energy-dense and poor-nutrient dense food, such as fast food and sugary drinks. There is a need to consider the contemporary changes in foods consumed, the growing number of children with T2DM and the relationship between food consumption and risk of T2DM. The unique and timely body of work in this thesis examined the importance of the whole diet and food combinations (eating patterns), rather than one food or nutrient, for future health and prevention of T2DM. This study determined the eating patterns among children and investigated the associations between those eating patterns and risk of T2DM.

The information for this thesis was collected from two birth cohort studies at the Auckland University of Technology (AUT), both of which were looking at growth and development of children born in contemporary Auckland. The two studies are the Pacific Islands Families (PIF) and the Metformin in Gestational Diabetes the Offspring Follow-Up study (MiGTOFU). Eating patterns, HbA_1c and other factors were measured in these studies (2014 to 2016). Both the PIF and MiGTOFU cohorts had information that enhances understanding of children's past and present wellbeing to inform timely development and implementation of interventions and policies to optimise children's health. This is in line with the proposition that "preventing a trigger effect will more dramatically abolish any adverse risk associated with exposures experienced earlier in the chain of events" (Ben-Shlomo & Kuh, 2002, p. 287).

1.1 Preface

This PhD thesis is presented through four projects (Figure 1). The first project in this thesis, evaluated the performance and the use of a point of care testing (POCT) to measure the glycated haemoglobin A_1c (HbA_1c) concentration, as a biomarker for screening for T2DM and validated this against a reference method. This validation was with Pacific children, who participated in both the PIF main (n = 931) and in the nested sub-study (n = 204).

Author's contributions: The author was responsible for managing the process of collecting HbA_1c measures via POCT including all training and the quality control of HbA_1c POCT measurements in the field and at Auckland Hospital. Data cleaning and statistical analysis of the collected HbA_1c at schools (PIF main study) and hospitals (PIF sub-study) were undertaken by the author. The questions in this thesis were not part of the existing PIF research plan.

In the second investigation, the information was collected from Pacific children (n = 931, age = 14 years old) recruited in PIF main study. Assessments were carried out from May 2014 to July 2015. Eating patterns of these children based on the NZ food habit questionnaire were identified. Then the association between eating patterns and HbA_1c (measured by the POCT) were explored. Besides body measurements, early life factors such as birth weight and mothers' age at conception were included as these may influence the relationship. In this study, the focus was on Pacific ethnicity, which has a higher risk of developing T2DM and obesity compared to other ethnicities in NZ.

Author's contributions: The author was involved in training assessors to undertake anthropometric and bioimpedance measurements and measuring HbA_1c via POCT and the quality control of the POCT. In addition, the author was the main supporter of the assessor team interviewing and measuring children at schools. Moreover, the author was responsible for dietary data cleaning and categorisation, exploration and identification of eating patterns, and determining the associations with risk of T2DM among these children. These questions were not asked as part of the Health Research Council (HRC) funded PIF study: understanding growth.

In the third study in this series of investigation, the risk of T2DM and the frequency of consumption of sweet, sugar added and energy-dense foods; and occasional foods were examined. This was tested through a nested sub-study of the PIF main study, (n = 204, age ≈ 15 years old). The PIF sub-study was completed almost one year after PIF main study and assessments were carried out from October 2015 to February 2016. The research questions focused on the relationships between sweet and energy-dense foods and concentration of HbA_1c and SUA. In addition, the association of these as T2DM biomarkers with body measurements, as possible mediators, were proposed and the author executed the statistical analysis.

Author's contributions: The author was involved in training an assessor for measuring HbA_1c via POCT and controlling the quality of the POCT at the hospital. In analysis

process, the author was responsible for dietary data cleaning, and with integration of nutrition knowledge to explore the associations between sweet and energy-dense foods with concentration of SUA and HbA_1c as T2DM biomarkers, as well as body measurements. These questions would not be asked as part of the existing PIF research plan.

In the last study, named 9-year MiGTOFU, the relationship between HbA_1c and eating patterns was evaluated among a group of multi-ethnic children (n = 99, age = 9 years old) whose mothers were diagnosed with gestational diabetes mellitus (GDM) before they were born. These children and their mothers were part of the MiGTOFU study, where the mothers were randomised to treatment for GDM with metformin or insulin. Assessments were carried out from September 2012 to May 2015. Body measurements and some of the early life factors such as birth weight and mothers' age at conception were also considered in this study as these might affect the relationships between eating patterns and concentration of HbA_1c.

Author's contributions: The author observed data collection and entry in the 9-year-olds – specifically the variables that were of interest to this thesis. The work undertaken by the author included data entry and checking, dietary data cleaning and categorisation-identification of eating patterns, diversity of diet among ethnicity (e.g. European and Pacific), and from point of analyses, integration of nutrition knowledge and expertise into the analysis. These questions would not be asked as part of the existing MiGTOFU research plan.

1.2 Overview of the studies

The integration of these studies into this written thesis is illustrated in Figure 1.1.

The focus of this exploratory PhD study was to identify the eating patterns and investigate the relationships among patterns of foods consumed with glycaemic control, using the blood biomarkers, HbA_1c, and SUA concentrations.

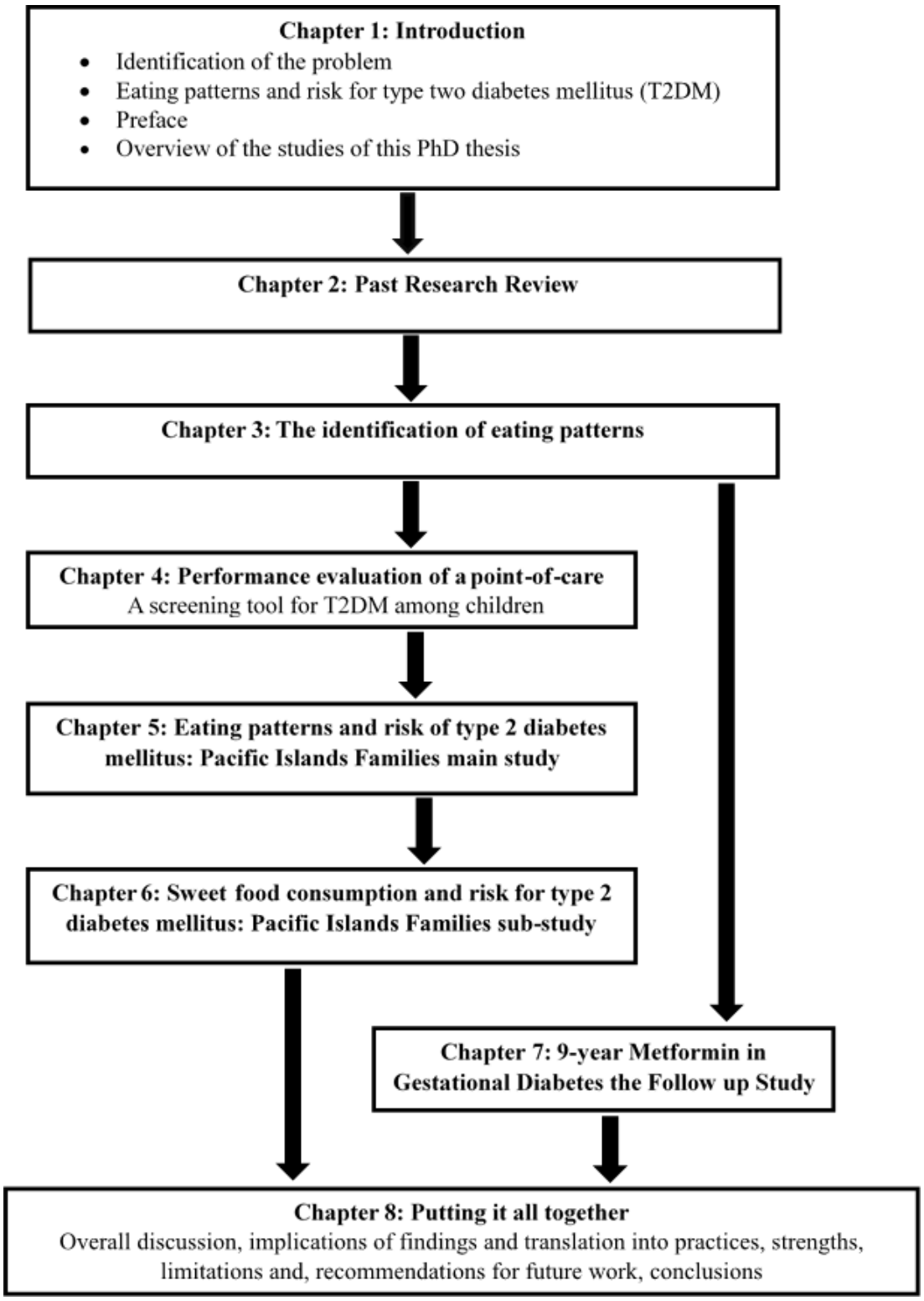


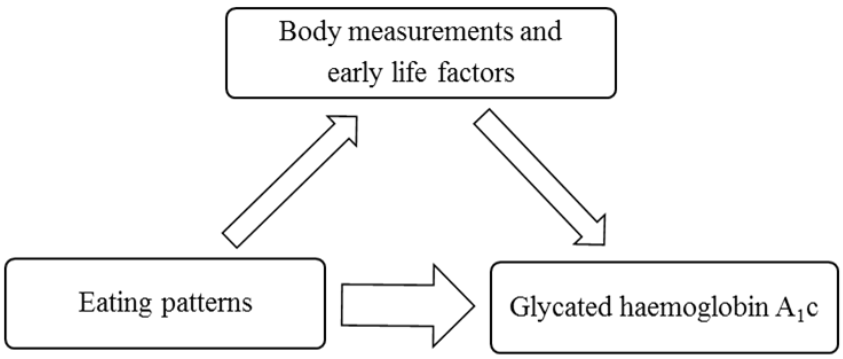
Figure 1.1. Structure and flow of the thesis

Chapters Two and Three present an overview of the literature relevant to this doctoral research study. In particular, three literature review questions were addressed:

1. What is the emergence of T2DM among children and what are its risk factors?
2. What is the advantage of applying POCT for screening for T2DM?
3. How to identify eating patterns?

The first study (performance evaluation of a point-of care analyser to measure HbA_1c), had a cross-sectional design. Children who participated in the PIF sub-study were measured twice with a year apart. The design of the three observational investigations for this thesis; PIF main study (14 years old, n = 931), PIF sub-study (15 years old, n = 204) and MiGTOFU (9 years old, n = 99) was cross-sectional.

A simple mediation model (Wang et al., 2014) (Figure 1.2) was used to conceptualise the possible relationships– by looking at the dependent variable (HbA_1c) in relation to the independent variable (eating patterns). This model included some explanatory factors such as growth (body size), and early life course variables (e.g. maternal age). This model was applied to the two cohort studies – which differed in the ways that diet and covariates were measured.



Participants
1. From Pacific Island Families Study, 14 years old, n= 931
2. From Metformin in Gestational Diabetes the Follow-up Study, 9 years old, n= 99

Figure 1.2. Mediation model underpinning the research questions

The main hypothesis of this PhD study was that a less healthy eating pattern including more foods with poor-nutrient profile and less fruit and vegetables would be associated with higher HbA_1c than a more prudent eating pattern. Eating patterns (e.g., prudent versus imprudent) were identified using, cluster analysis, which is a data driven approach.

Chapter 2 Literature review: type 2 diabetes mellitus and children

There is a rise in prevalence of T2DM among NZ children and youth. One of the modifiable risk factors of T2DM is the diet and more specifically the overall patterns and groupings of foods children and youth habitually consume. Traditionally, advice to address risk factors in the population is based on individual foods such as avoiding sugary drinks and eating more of some food groups such as vegetables rather than considering the whole diet and patterns of eating.

Literature was searched using MEDLINE, Web of Science, Science Direct, OVID, EBSCO and PubMed databases. Search terms included ‘food pattern’, ‘eating pattern’, ‘dietary pattern’, ‘food preference’, ‘food behaviour’, ‘nutrition behaviour’, ‘glycated haemoglobin A_1c’, ‘HbA_1c’, ‘type 2 diabetes mellitus’, ‘obesity’, ‘body mass’, ‘body composition’, ‘fat mass’, ‘children’, ‘youth’, ‘ethnicity’, ‘dietary pattern analysis method’. Search terms were limited by adding ‘cluster analyses’, ‘cohort’, ‘Pacific’, ‘uric acid’, ‘serum uric acid’, ‘urate’ and ‘gestational diabetes’. In addition, truncated links from references listed in research and review articles were followed-up.

2.1 Emergence of type 2 diabetes mellitus in children

One of the most worrying features of the rapid increase of T2DM is the marked and recent increase in the development of T2DM among children and adolescents (International Diabetes Federation, 2013; World Health Organization, 2016). While thirty years ago T2DM was a rare disease among this age group, current research projects predict an increasing trend (Temneanu, Trandafir, & Purcarea, 2016) in early incidence. This is very concerning for future generations (World Health Organization, 2016). The global prevalence of T2DM among adults has risen dramatically from 4.7% in 1980 to 8.5% in 2014, with a parallel increase in the prevalence of obesity (World Health Organization, 2016). Children and adolescents from indigenous populations (particularly in North America and Australia), Pacific Island people, African Americans, Hispanics and some Asian populations have the highest proportion with T2DM (Zimmet, Magliano, Herman, & Shaw, 2014). The prevalence of T2DM in NZ Māori is also higher than European (Ministry of Health, 2014a) and Māori may have a genetic predisposition to obesity and T2DM.

In NZ the prevalence of T2DM in adult Pacific is around three times higher than other New Zealanders (Ministry of Health, 2016a). In a regional paediatric cohort with T2DM in Auckland, NZ, Jefferies and colleagues (2012) reported that the annual incidence of new cases of T2DM in children < 15 years increased fivefold in the Auckland region from 1995 (0.5/ 100,000; 95% confidence interval (CI) 0.0– 2.2) to 2007 (2.5/100 000; 95% CI [1.0, 5.5]). They also reported that an average annual incidence per 100 000 over the 1st January 1995 to 31st December 2007 period was 1.3 (95% CI [1.0, 1.8]) overall, 0.1 (0.0– 0.4) in Europeans, 3.4 in both Māori (2.0– 5.3), Pacific (2.2– 5.0) and 0.6 in Asian/Middle Eastern/Latin American/African (95% CI [0.1, 1.8]), and 57% of children were asymptomatic at presentation (Jefferies et al., 2012).

Diabetes mellitus, which is a metabolic disorder of multiple etiologies, is characterized by chronic hyperglycaemia with disturbances in carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both (Alberti & Zimmet, 1998).

As found in the adult population, T2DM is a “metabolic disorder of fuel homoeostasis” (Nolan, Damm, & Prentki, 2011, p. 169) in which pancreatic islet Beta (β) cells are unable to secrete sufficient insulin, which is related to different degrees of over-nutrition, inactivity, obesity, and insulin resistance (Nolan et al., 2011; Skyler, 2014). This eventually leads to a decline in insulin secretion and a persistent increase in fasting blood glucose (International Diabetes Federation, 2013; Marcovecchio, Mohn, & Chiarelli, 2005; Reinehr, 2013). It is clear that nutritional, behavioural, and environmental factors are directly and indirectly linked with T2DM (Prasad, 2011).

The incidence of T2DM in children has a disrupting effect on their quality of life during a critical period of growth and development and leads to increased healthcare costs (Fazeli Farsani, van der Aa, van der Vorst, Knibbe, & de Boer, 2013). This is related to a variety of severe complications of early onset of T2DM such as nephropathy, retinopathy, neuropathy, dyslipidaemia and cardiovascular disease in children, with an increased risk for co-morbidity and mortality (Fazeli Farsani et al., 2013). Therefore, prevention of T2DM starting early in life and then across the life course should be given high priority.

2.1 Diagnosis and screening for risk of type 2 diabetes mellitus in children

The diagnosis of T2DM can be very complicated as some people might be asymptomatic, whereas others may show evident symptoms (Ang, Thevarajah, Alias, & Khor, 2015; Colagiuri, 2011). Unfortunately, in children and adolescents T2DM is often asymptomatic (Temneanu et al., 2016). As untreated disease may lead to long term complications such as metabolic, microvascular, and macrovascular complications, presence of some criteria (Table 2.1) it should be taken seriously and patients should be sent for screening for T2DM diagnosis and treatment (Bennett, Guo, & Dharmage, 2007; Kester, Hey, & Hannon, 2012). However, the choice of screening methodology among children remains controversial (Kester et al., 2012). Most often, T2DM is diagnosed around the ages of 13-14, with an earlier onset in girls, which might be related to the role of physiological insulin resistance during puberty (Temneanu et al., 2016). Generally, there are two distinct test: plasma-specific tests and the whole blood glycated haemoglobin (HbA_1c) test (Ang et al., 2015).

Table 2.1. The recommendations of the American Diabetes Association consensus statement on testing for type 2 diabetes in children aged 18 years and younger [a]

Criteria	Overweight (BMI> 85th percentile for age and sex, weight for height > 85th percentile, or weight> 120% of ideal weight)
Plus any two of the following risk factors	Family history of T2DM in first or second degree relative
	Race/ ethnicity (Native American, African American, Latino, Asian American, Pacific Islander)
	Signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, polycystic ovary syndrome, or small for gestational age birthweight)
	Maternal history of diabetes or GDM during the child's gestation
Age of initiation:	Age 10 years or at onset of puberty, if puberty occurs at a younger
Frequency	Every 3 years

[a] (American Diabetes Association, 2014, p. S18)

Screening for T2DM in asymptomatic patients is recommended to be done by questionnaires that evaluate the risk or by measuring a fasting plasma glucose or having the patient undergo an oral glucose tolerance test. A random blood glucose test can also be useful. Blood tests require fasting for the previous eight hours (Saudek et al., 2008)

and for confirmation of diagnosis some tests such as FPG need to be repeated at least twice (Bennett et al., 2007). Due to the need to make the diagnosis of T2DM opportunistic, efficient and timely, the International Expert Committee (2009) and American Diabetes Association (2014) advise that HbA_1c should be used as both a biomarker for risk for diabetes and a diagnostic tool - with the added advantage that unlike blood glucose, HbA_1c does not require fasting or a two hour blood test following a glucose challenge.

2.1.1 Glycated haemoglobin A_1c

In healthy individuals aged >1 year, haemoglobin consists of approximately 97% adult haemoglobin (HbA), 2.5% HbA_2 and 0.5% foetal haemoglobin (HbF) (Lenters-Westra, Schindhelm, Bilo, & Slingerland, 2013). Nearly 6% of HbA in a healthy person is glycated and it consists of HbA_1a and HbA_1b (minor components) and HbA_1c (main component) (Lenters-Westra et al., 2013). The formation of HbA_1c mainly depends on the interaction between blood glucose concentration and the life span of red blood cells (in healthy person = 38-59 days, in person with diabetes = 39-56 days) (Lenters-Westra et al., 2013). However, the lifespan of a red blood cell is genetically determined and red cell survival is a continuous variable within a population (Church & Simmons, 2014). Generally, for a population HbA_1c represents a 2–3-month average of blood glucose concentrations (Bennett et al., 2007). However, there are limitations in the measurement of HbA_1c particularly if the rate of red blood cell turnover is compromised (Simmons & Hlaing, 2014) by for example recent blood loss and anaemia. A red blood cell count plus measures of red blood cell size, haemoglobin concentration and haematocrit could also be useful to determine anomalies in red cell turnover.

The measurement of HbA_1c in blood is the gold standard for the long-term control of the glycaemic state of patients with diabetes (Jeppsson et al., 2002; Lenters-Westra et al., 2013). Recently, both World Health Organization (WHO) and the American Diabetes Association (ADA) has advocated HbA_1c as a reliable biomarker for screening for diabetes (American Diabetes Association, 2010). To support this, there are a number of studies including reviews that have pointed out HbA_1c as a robust biomarker for screening T2DM (Bennett et al., 2007; Berhan et al., 2014; Colagiuri, 2011; Florkowski, 2013; Jesudason, Dunstan, Leong, & Wittert, 2003; Lenters-Westra et al., 2013; Shah, Kublaoui, Oden, & White, 2009; Wiener & Roberts, 1998). However, applying HbA_1c as the sole biomarker of diagnosing T2DM is still a matter of debate

(Temneanu et al., 2016), in part due to a lack of consensus on its cut-off points in diagnosis, especially by age groups and different ethnicities.

The main factors in support of applying HbA_1c as a screening and diagnostic biomarker of T2DM are: 1) fasting is not required, and 2) compared to plasma glucose; HbA_1c reflects a long-term glycaemia status. Additionally, there are well standardized and reliable laboratory methods for HbA_1c measurement, and in the infrequent instance of an error in measurement caused by nonglycaemic factors, the plasma glucose (fasting plasma glucose or 2 –hour oral glucose tolerance test) can be measured to support the diagnosis of diabetes (Saudek et al., 2008).

2.1.2 Point of care and screening diabetes mellitus

Point-of-care testing (POCT) using capillary blood obtained from a finger prick, a portable instrument that makes screening for risk of T2DM possible in the general practitioners' office, workplace, or at home (Lenters-Westra & Slingerland, 2014). It is crucial to ensure the reliability and validity of the POCT instrumentation being used; as the interpretation, the sensitivity, and specificity of the testing devise is questionable (Lenters-Westra & Slingerland, 2014). In the last decade, rapid POCT tests for HbA_1c have been adopted by specialty diabetes clinics and urban primary care clinics which have been compared favourably with clinical laboratory testing (Tamborlane et al., 2005). One HbA_1c instrument that meets the analytical performance criteria is the AFINION ™ AS100 Analyser.

Recent validation studies for this analyser have been reported by Dupuy et al. (2014), who stated that Afinion analyser met the acceptance criteria of analytical performance, requires minimal operator interaction, and presents good correlation with the laboratory method ($r > 0.96$, $n = 155$). The authors recognized different regression lines for different batches of reagents as a matter of concern (Dupuy et al., 2014). In another study, the performance of Afinion was evaluated beside the InnovaStar, Cobas B101, DCA Vantage, Quo-Lab, Quo-Test, and B-analyst HbA_1c POCT instrument (Lenters-Westra & Slingerland, 2014). The Afinion, DCAVantage, Cobas B101 and B-analyst instruments met the generally accepted performance criteria for HbA_1c (Lenters-Westra & Slingerland, 2014).

AFINION ™ AS100 Analyzer uses a cartridge method to do multiple analyses such as HbA_1c, albumin/creatinine ratio, and C-Reactive protein. The results of these tests then appear after a very short time (e.g., around 3 minutes for HbA_1c) on the color screen of

the devise (Alere, 2012). The screen is also touch sensitive, makes it even more versatile for users. Other features that made this devise very popular include small amount of sample size (e.g., 1.5 µL for HbA_1c), long shelf life of reagents at room temperature (15-32°c), and an integrated error detection system (Alere, 2012).

2.2 Risk factors for type 2 diabetes mellitus

Risk factors are the attributes, characteristics or exposure of an individual to a substance or condition that increase the probability of developing a disease or injury. To inform screening and prevent T2DM, identifying attributable factors and exposures for its development among asymptomatic population is essential (Jia, Zhang, Kang, & Wu, 2013). Of concern is how the early incidence of NCDs including T2DM at a young age transfers to adulthood considering its accelerated risk of significant comorbidities at a young age (Darnton-Hill, Nishida, & James, 2004). Therefore, efforts have been made to understand T2DM personal risk factors, including family history of diabetes, ethnicity, age, previous gestational diabetes, obesity, physical inactivity, alcohol intake, smoking, serum triglyceride concentration, serum uric acid (SUA) concentration and coronary heart disease and the quality and quantity of foods in the habitual diet (World Health Organization, 2016; Xu et al., 2016).

Some of these risk factors –such as genetics, ethnicity and age – are not modifiable. However, other factors such as excess weight gain, unhealthy diet, and lack of physical activity are habits that can be changed. Examples of beneficial changes that would influence and support the individual child (Figure 2.1) are environmental – and are known to exert their effect from conception as mother's diet and life-style determine her child's nutrition, growth, birth weight and development. Moreover, mother's education and social, economic, food and activity environments determine her and her family's ability to provide for the child. Review papers with the main focus on T2DM in children concluded that the risk factors attributed to development of T2DM in children are related to the development of obesity (Jordan & Jordan, 2012; World Health Organization, 2016), lack of physical activity, and a sedentary lifestyle (Jordan & Jordan, 2012; Marcovecchio et al., 2005), diet (Alhazmi et al., 2014), and family medical history (Jordan & Jordan, 2012; Marcovecchio et al., 2005).

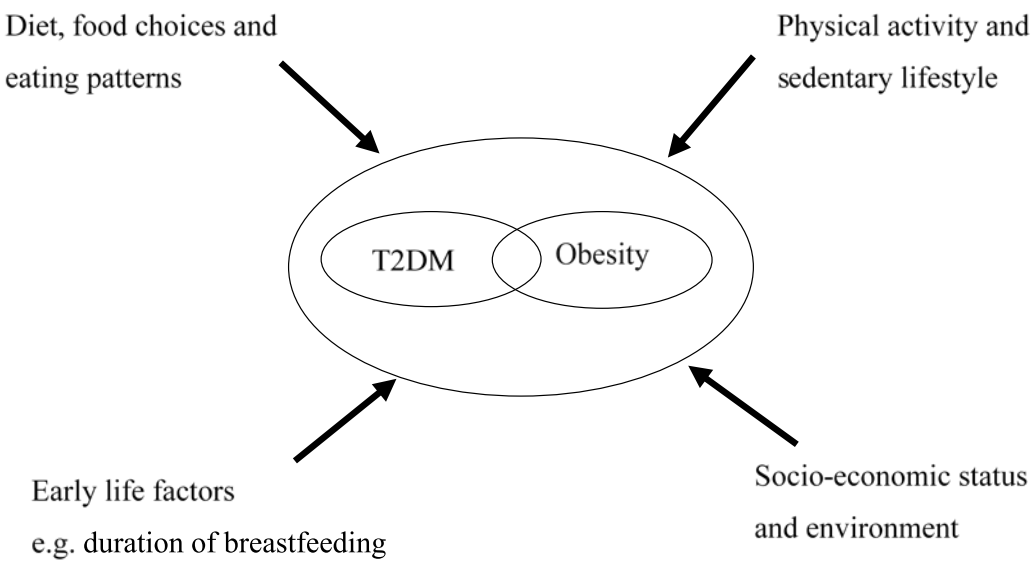


Figure 2.1. Modifiable risk factors for type 2 diabetes mellitus in children

2.2.1 Life exposure, food consumption and type 2 diabetes mellitus

Onset of many adult non-communicable chronic diseases (NCDs) including excess weight and T2DM appear to have their origins in early life (Buyken, Mitchell, Ceriello, & Brand-Miller, 2010) and nutrition in foetal life has been identified as a central programming stimulus (J. Harding, 2001). In the epidemiology for public health, it has been accepted that events *in utero* and during childhood are potentially crucial determinants of adult health and this model of health prevailed since the first half of the 20th century (Ben-Shlomo & Kuh, 2002; Gary-Webb et al., 2013). More recently, early life from conception to late gestation and early postnatal life, the periodicity of the adiposity rebound (~ age 4–6 years, describing the nadir in the BMI growth curve) and adolescence have been examined and are proposed as potentially critical periods for the development of obesity and T2DM later in life (Buyken et al., 2010). It is during the childhood period that an individual's patterns of obesogenic behaviours are shaped, with evidence that overall diet, physical activity and sedentary patterns of behaviour persist or track into different stages of life (Leech, McNaughton, & Timperio, 2014).

Diet, the sum of all foods and drinks consumed in children, is an important and modifiable exposure that has both short and long-term effects on growth and development. For a long time an 'unhealthy diet' has been identified as a major risk factor in developing T2DM (Ley et al., 2014; Prasad, 2011). Defining an unhealthy diet

is difficult due to the complexity, variety, preparation and quantities of foods eaten and drinks consumed. Many “Western” eating patterns, which include high intakes of saturated fat, processed foods, and red meat increase the risk of T2DM, conversely diets high in omega-3 fatty acids, fibre, whole grains, coffee, and alcohol are associated with a lower prevalence of T2DM. This has been reported both in reviews (Gary-Webb et al., 2013; Micha et al., 2017) and cohort studies (Fung, Schulze, Manson, Willett, & Hu, 2004; van Dam, Rimm, Willett, Stampfer, & Hu, 2002).

The recent Alhazmi et al. (2014) review of fifteen prospective cohort studies concluded that eating patterns represented by less healthy food choices (like refined grains, red or processed meats, high - fat dairy and sweets) and higher energy density of the diet were strongly associated with the incidence of T2DM. Such studies among adults highlight the need to understand what children eat and their eating patterns (Ambrosini, 2014; Esposito et al., 2014), rather than one food or nutrient, as a key and potentially modifiable risk factor for T2DM prevention (Alhazmi et al., 2014; Shirani et al., 2013).

A subsequent Malmö Diet and Cancer cohort study by Mandalazi and colleagues (2016), after following 26,868 Swedish participants up for 17 years, when their participants reached 44-74 years old concluded that there was no relationship between the baseline diet quality index (assessed based on current Swedish food guidelines) and the incidence of T2DM (Mandalazi et al., 2016). Although 3,838 cases were diagnosed with T2DM, the authors acknowledged that this finding was the result of the dietary components that were chosen to represent adherence to the recommendations to predict CVD. In other words, they asserted that the dietary recommendations might not applicable to T2DM (Mandalazi et al., 2016). The recommendations relied on the previous report that suggested an association between the recommended diet and reduced risk of CVD and not T2DM, (Mandalazi et al., 2016). The diet quality index used did not include an assessment of protein, meat, processed meats and sugar sweetened beverages.

Eating pattern analysis considers a more comprehensive and holistic overview of the diet, has been used most often in adults, this analysis is needed to be further explored in children populations (Cutler et al., 2012). The importance of eating patterns of children in association with growth, weight status and the obesogenic environment has been identified as an area that requires more research (Cutler, Flood, Hannan, & Neumark-Sztainer, 2011). The relationship between food exposures in childhood and its impacts

on later adult life is complex (Wall et al., 2013) and therefore requires prospective studies.

2.2.2 Growth and type 2 diabetes mellitus

Perhaps the most manifest and researched risk factor of T2DM after potential genetic, epigenetic and environmental risk factors, which may lead to insulin resistance and limited β-cell reserve, is childhood obesity (Jordan & Jordan, 2012; Natale et al., 2013; Skyler, 2014). Obesity is best defined as an excess of body fat but for population surveys surrogate measures of relative weight for height squared– dependent on age and gender up to 18 years, have been used most commonly.

Over the last two decades the prevalence of childhood obesity has increased and this is associated with an increase in the proportion of adolescents with T2DM, especially among minority populations (Gary-Webb et al., 2013). Moreover, treating or preventing T2DM is hard without first addressing its main predisposing risk factor: obesity (The Lancet, 2014).

Obesity and T2DM often go hand in hand and can affect the same people. This suggests similar genes and environments contribute to the development of both disorders (Fadason, Ekblad, Ingram, Schierding, & O'Sullivan, 2017). In addition, Fadason and colleagues (2017) found that many of the regulatory ‘single nucleotide polymorphisms-gene’ (they are regions of the DNA that commonly vary between individuals and that have been linked to a disease) (SNP-gene) connections affected body tissues not usually thought of as driving obesity or T2DM such as breast tissue, brain or the fat that sits just beneath the skin. They pinpointed visceral fat as a big contributor to T2DM, but found few SNP-gene connections (Fadason et al., 2017). They suggested that this “combinatorial action” (Fadason et al., 2017, p. 150) within different tissues reduces the ability of these tissues to contribute to the maintenance of a healthy energy metabolism. Beside genetic relationships between obesity and T2DM, the main contributors to a pandemic of global childhood excess body fatness and rapid growth appeared to be unfavourable changes in food and lifestyle patterns, particularly an increase in nutrient-poor foods and reduction in physical activity. The prevalence of overweight and obesity in children and adults in NZ, defined by international BMI cut-offs, is the 3rd highest among Organisation for Economic Cooperation and Development countries (Organisation for Economic Cooperation and Development, 2014). One in nine children aged 2–14 years is obese (10.8%) (Ministry of Health, 2015a).

The 2010 New Zealand national survey of physical activity and dietary behaviours with a nationally representative sample of 2,503 participants age 5 to 24 years (Clinical Trials Research Unit, 2010), concluded that healthier eating behaviours (e.g., vegetables and fruit intake 5 or more servings per day) decline with age, and less healthy eating behaviours, such as consumption of foods high in fat, sugar and/or salt, increase with age.

An application of the 2006 WHO growth standard from birth to 4 years to Pacific Island children (n = 659) Rush et al. (Rush, Paterson, Obolonkin, & Puniani, 2008) concluded that although smoking in pregnancy was associated with reduced birth weight, these children were larger at four years compared with children whose mothers never smoked and breastfed for the first six weeks of the child's life. (Rush, Paterson, Obolonkin, et al., 2008). Yet at the age of six years old (n = 722) that there was no difference in relative body weight of children in relation to smoking during pregnancy or early breastfeeding (Rush et al., 2010). However, both boys and girls, who were born heavier, were following a steeper increase in body weight ($r = 0.24$, $p < 0.001$) (Rush et al., 2010).

In another longitudinal study of offspring from pregnancies complicated by gestational diabetes and treated by either metformin or insulin (the Metformin in Gestational Diabetes study- MIG), ethnic and gender differences in body composition were examined at the age of 2 years. This offspring follow-up (MiGTOFU) study used whole body dual energy X-ray absorptiometry to measure total and regional body composition of 48 boys and 56 girls. Children belonged to European (18M, 20F), Indian (12, 8), Polynesian (11, 17) and Other (7, 11) ethnic groups and found that Indian boys had more fat in the central and abdominal regions and less total lean mass than European boys ($p < 0.05$) adjusted for age, weight and height (Rush, Obolonkin, Battin, Wouldes, & Rowan, 2014). In the same children it was reported (Rush, Bristow, Plank, & Rowan, 2013) that the average body size of these children was not different to the WHO, standard but the range of measures was wide and varied by gender and ethnicity. The MiGTOFU and PIF children had higher risk for T2DM later in life which is related to pregnancy, ethnicity and rate of growth.

In addition, Rush (2012) defined that the ethnic comparison is limited due to lack of concurrent measurements of chronic disease risk factors such as blood glucose and lipids that are associated with body mass index (BMI) and fat distribution.

Birth weight is affected by factors e.g. intrauterine exposure, might adversely affect the future health of children. The association of T2DM with birth weight has been reviewed in a meta-analysis of fourteen studies and 132,180 persons aged 6 to 75 years by Harder and colleagues (2007). The expectation was that there would be an inverse linear relation between birth weight and T2DM, which means that a higher birth weight would be associated with decreased risk (Harder et al., 2007). However, results from some studies showed that both low birth weight and high birth weight subjects had high-risk and others that high birth weight but not low birth weight was associated with increased risk of T2DM (Harder et al., 2007). The latter result is aligned with Whincup and his colleagues' (2008) observations that suggested high, rather than low, birth weight could become an increasingly important influence on T2DM risk in the later-life. In 2016, the World Health Organization (WHO) recognised both high and low birth weight as factors that raise the risk of T2DM (World Health Organization, 2016).

In NZ, children of Pacific and Chinese parents have higher weight at birth than European, while Māori and Indian babies weigh less than European (McCowan, Stewart, Francis, & Gardosi, 2004). Therefore, the Pacific Islands Families (PIF) and MiGTOFU cohort studies have the potential to provide an opportunity to study the relationship between birth weight and its possible confounding effect on the association between HbA_1c and eating patterns in later life.

2.2.3 Breastfeeding and type 2 diabetes mellitus

A further confounding factor related to future health is the duration and exclusivity of breastfeeding. While the impact of early food intake and eating behaviours remain a matter of active research, there is expansive literature documenting the impact of pregnancy diet and mode of infant feeding (e.g., breast vs formula) on children's health (Golley et al., 2013). One of the most commonly investigated nutritional interventions is breastfeeding, which is considered to have a long-term protective effect on the prevalence of T2DM (Gruszfeld & Socha, 2013).

Exclusive breastfeeding during the first 6 months of life is recommended by WHO and the American Academy of Paediatrics (AAP) based on scientific evidence of health benefits in breastfed infants (American Academy of Pediatrics, 2012; Ip et al., 2007; World Health Organization, 2003). It is also advised that complementary foods should be added to the diet of infants, if breast milk is insufficient to meet the nutritional needs of them (World Health Organization & Fund, 2000).

The effectiveness of the breastfeeding and decreased risk of later T2DM in children has been exemplified in two reviews. The first review by Taylor et al. (2005) included 12 observational studies published between 1966-2003 and they concluded that all women with or without diabetes should be encouraged to breastfeed as low oestrogen levels in breastfeeding women might play as a protective factor on glucose metabolism and subsequent risk of T2DM. The second review by Horta, Loret de Mola and Victora (2015) included ten studies (with children aged 10-19 years and ≥ 20 years) and reported that the odds ratio of incident T2DM was lower among those subjects who had been breastfed (pooled odds ratio: 0.65 95% CI [0.49, 0.86]) (Horta et al., 2015). In support of significance of infant feeding to long-term health outcomes, there is evidence that breastfeeding may be associated with reductions in blood pressure and total blood cholesterol, and lower risks of obesity and DM in adult life but prospective studies are required that examine not only duration of breastfeeding, but also the quality of the subsequent diet (Robinson & Fall, 2012). There are also other factors such as mother-to-child transmission of HIV (World Health Organization, 2003) that influence whether a mother is able to breastfeed.

2.2.4 Maternal education

Maternal education has used as an indicator for socio-economic status in several studies (such as Cameron et al., 2011; Craig, McNeill, Macdiarmid, Masson, & Holmes, 2010; Huybrechts et al., 2017; Ruijsbroek et al., 2011). A conceptual framework (Ruijsbroek et al., 2011, p. 4) (Figure 2.2) depicts the accumulation of factors that influence children’s health over the first 8 years of their lives. These include maternal education and socio-economic disparities, which are interactive. Higher education usually is associated with improved socioeconomic status. In most studies (including Aranceta, Perez-Rodrigo, Ribas, & Serra-Majem, 2003; Craig et al., 2010; Emmett, Jones, & Northstone, 2015; Huybrechts et al., 2017) higher maternal education was associated with eating patterns that included more fruit and vegetables and less poor nutrient foods such as processed foods or snacks. Higher education is also associated with higher socioeconomic status and less children in the family.

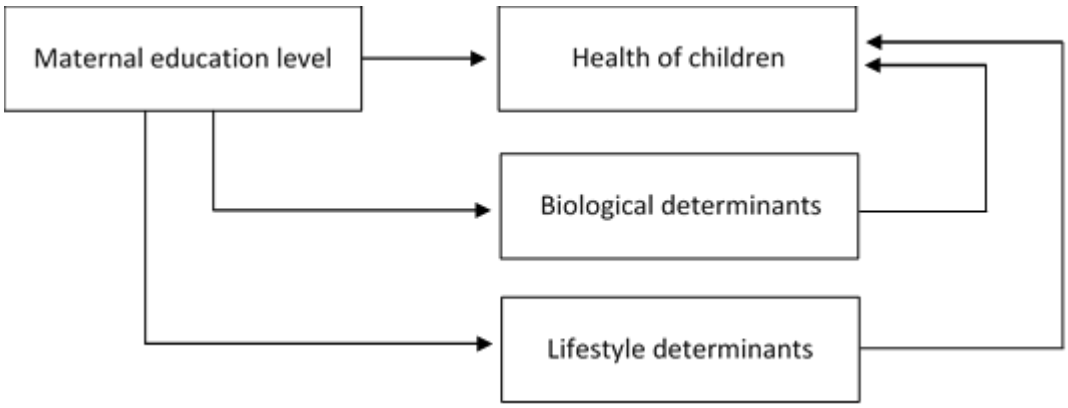


Figure 2.2. A conceptual framework for relationship between maternal education level and the health of children (Ruijsbroek et al., 2011)

2.3 Eating patterns

Over the past two thousand years, common knowledge and understanding of the significance of a healthy diet have been recognized (Michels, 2003). However, it is only in the last few decades that more formal studies of whole diet and health have been explored systematically and statistically and the relationships of overall eating patterns to health and disease started to be investigated (Michels, 2003; Waijers, Feskens, & Ocké, 2007). The basis of this holistic approach was founded on the concept that foods eaten together are as important as examining the role of a single nutrient or food (a method called reductive methodology) (Kant, 2004; Mullie, Clarys, Hulens, & Vansant, 2010).

Diet is a multidimensional exposure variable and takes into account the combination of foods as well as how often, when, and how much of each one is consumed (Hoffmann, Schulze, Schienkiewitz, Nöthlings, & Boeing, 2004). This combination is moderated by a group of genetic, cultural, social, health, environmental, lifestyle, and economic determinants and could provide a picture of which types of foods are consumed (Kant, 2004) and the identification of eating patterns associated with a healthy lifestyle. Methods of identifying eating patterns have been discussed in Chapter 3 .

Studies about diet, health, and development have mainly focused on the role of a specific nutrient or food. The results of such studies cannot explain the clustering of dietary components that might either increase or reduce the risk of health and disease outcomes over time (Golley et al., 2013). Furthermore, dietary components may interact, complicating the search for any relationships between disease and individual dietary factors (Waijers et al., 2007).

People do not eat nutrients or a single food, this means the intake of nutrients and foods are related (Waijers et al., 2007). This can be explained by an example which describes by the association between higher cereal fibre intake and lower risk of T2DM - whole grains count as an important source of cereal fibre, vitamins, minerals, lignans, and other phytochemicals (de Munter, Hu, Spiegelman, Franz, & van Dam, 2007). In addition, the association between some foods or food groups with other foods, can differentiate the potential effects of foods (Togo, Osler, Sørensen, & Heitmann, 2001). For instance, seaweed contains a high proportion of soluble dietary fibre, which may be a barrier to starch digestion, and as a consequence can modify glycaemic response (Goñi, Valdivieso, & Garcia-Alonso, 2000). In a study by Goñi et al. (2000) starch bioavailability in bread was modified by adding Nori alga (a type of seaweed) and the glycaemic response to white bread decreased from 100% to 68% in healthy volunteers. Therefore, to investigate the association between dietary factors and risk of getting a particular disease, considering a variety of approaches is essential (Hu, 2002). One approach is dietary or eating pattern analysis, which cannot replace detailed analysis of nutrients consumed to identify dietary deficits for an individual, but is a complementary approach to the more traditional analyses (Hu, 2002).

The idea of applying statistical methods is to reduce dietary data into something more meaningful than focusing on individual components, was introduced by Schwerin et al. in 1982, when the association of food habits was investigated in relation to nutritional health (Schwerin, Stanton, Smith, Riley, & Brett, 1982). In 1991, Randall et al. (Randall, Marshall, Graham, & Brasure, 1991) suggested that there might be association(s) with eating patterns and other high-risk health behaviours. Then in 1998, Slattery (Slattery, 2008) in their study on foods eaten and colon cancer wondered whether eating patterns characterize the diet- associated disease risk better than any food or nutrient. Since then, this method has been applied globally by other researchers like Terry et al. (2001), Hu and colleagues (Hu et al., 1999; Hu et al., 2000), to a variety of diseases with most of them reaching the same answer to the question: “yes they do” (Slattery, 2008).

“Food patterns can summarize the combined and potentially synergistic effects of a repertoire of foods contributing to usual dietary intake in a defined population.” (Ambrosini, 2014, p. 1). Examples of eating patterns derived by researchers have been summarized in Table 2.2.

In NZ, public health advice is to eat plenty of vegetables and fruit, mostly whole grains and those naturally high in fibre, some milk and milk products and legumes, nuts, seeds, fish, and other seafood poultry and red meat with the fat removed (Ministry of Health, 2015b). In addition foods low in saturated fat, low in sodium (salt), with little or no added sugar and mostly whole and less processed are advised (Ministry of Health, 2015b). This is the pattern agreed by experts that is important for good health and the advice is similar to various international evidence reviews (Ministry of Health, 2015b). In sum, healthier eating pattern identification is informed by the above-stated guidelines.

Identification of different eating patterns within a population assists to point out key, seasonal, affordable and acceptable foods within a food group. This is a viable counterpoint to the reductionist approach that is pragmatic (Ambrosini, 2014). Food-based information considered as an eating pattern might facilitate the translation of data into guidelines for the public (Ambrosini, 2014). Furthermore, studying dietary exposures and effects on these exposures during the life course can play a key role in informing interventions (Wall et al., 2013). On the same note, in the NZ Birth cohort study (Wall et al., 2013) it was found that an eating pattern that includes eating margarine daily was associated with significantly lower IQ scores in children, who were born small for gestational age. In comparison, weekly fish consumption was associated with higher IQ scores. This does not mean that margarine should not be eaten, or fish should be consumed by everyone, but underscores the understanding of the whole diet in the context of other foods frequently eaten Table 2.2).

Table 2.2. Eating patterns named by researchers

Study	Named Eating patterns
Auckland Birthweight cohort (Wall et al., 2013)	Junk (candy bars, hamburgers, soft drinks and chips) Traditional (cauliflower, peas, mixed vegetables, potatoes, pumpkin and beef) Healthy (pineapple, tomatoes, cucumber, 'other' green vegetables, celery, mixed grain bread)
Bogota School children cohort (Shroff et al., 2014)	Snacking (e.g. such as candy, ice cream, packed fried snacks, soda and sugar-sweetened fruit-flavoured drinks) Cheaper Protein Traditional/Starch Animal Protein
The Avon Longitudinal Study of Parents and Children (ALSPAC) (Smith, Emmett, Newby, & Northstone, 2011)	Processed plant-based Plant-based traditional British Traditional British processed
Whitehall cohort study (Brunner et al., 2008)	Unhealthy (white bread, processed meat, fries, and full-cream milk Sweet (white bread, biscuits, cakes, processed meat, and high-fat dairy products) Mediterranean-like (fruit, vegetables, rice, pasta, and wine) Healthy (fruit, vegetables, whole-meal bread, low-fat dairy, and little alcohol)
The Finnish Mobile Clinic Health Examination Cohort Survey (Montonen et al., 2005)	Prudent (higher consumption of fruits and vegetables) Conservative (consumption of butter, potatoes, and whole milk)

2.3.1 Eating patterns among New Zealand children

There are three studies where patterns of eating among NZ children were derived by applying statistical analyses, and associations with factors such as body measurements and socioeconomic background explored (Table 2.3). Despite this, surveys of child nutrition in NZ are scarce (Clinical Trials Research Unit, 2010; Ministry of Health, 2003, 2015a). There are not any studies in NZ context that have looked at eating patterns and the risk for T2DM in children.

Table 2.3. Studies focused on eating patterns of New Zealand children

Author (s)	**Study design**	**Number and age [a]**	**Dietary assessment method**	**EP analysis method**	**Focus of article**	**Derived eating patterns**	**Main outcomes**
Howe et al. (2013)	Cross sectional	681, 15.8	FFQ	PCA	body composition	Treat foods, 'fruits and vegetables', basic foods	Eating patterns were associated to central and total adiposity but not BMI
Wall et al. (2013)	Cohort	550, 3.5 591, 7.5	FFQ	PCA	SEB and obstetric factors among SGA and AGA	Traditional, junk and healthy	SEB, obstetric factors and birth size were associated with EPs, but the associations were not consistent with each eating pattern across the two age groups
Davison et al. (2008)	Cross sectional	401, 9-11	FFQ	PCA	parental diet quality and children EP	'Fruit and vegetables' and snacks	Parents with a poor diet quality were more likely to have children with snacks EP

[a] mean age (years)
Abbreviations: AGA, appropriate for gestational age; EP, eating patterns; FFQ, food frequency questionnaire; PCA, principle components analysis; SD, sedentary behaviours; SEB, socioeconomic background; SGA, small for gestational age

2.3.2 Eating patterns, ethnicity and type 2 diabetes mellitus

There are limited studies of eating patterns in relation to T2DM and ethnicity among both children and adults (e.g., Abu-Saad et al., 2012; Dekker et al., 2015; Lindquist, Gower, & Goran, 2000; McNaughton, Mishra, & Brunner, 2008; Nettleton, Steffen, Ni, Liu, & Jacobs, 2008). Ethnicity was considered a cofactor. All these studies raised the importance of ethnicity-specific approaches in addressing the risk of T2DM in populations. For instance Dekker and colleagues (2015) within five ethnic groups in Netherland including Dutch, South Asian Surinamese, African-Surinamese, Turkish and Moroccan (n = 3776, aged 18-70 years), found two eating patterns named ‘meat and snack’ and ‘vegetable’ patterns. The concentration of HbA_1c and fasting glucose of the Dutch origin participants, as biomarkers of T2DM, were associated with ‘meat and snack’ pattern; nonetheless, they stated that some foods that characterized the biomarker driven eating patterns like roti or couscous were ethnicity-specific foods (Dekker et al., 2015).

2.4 Growth trajectory, diabetes biomarkers and eating patterns: cohort studies

Long-term, gradual damage to health can be the result of environmental or behavioural insults either as a separate exposure or clustered together in socially-patterned ways (Ben-Shlomo & Kuh, 2002). Godfrey et al. (2010) demonstrated that a new global landscape of disease is emerging; where the place each of us is standing is an intersection of genes, environment, and the path we had taken to get here- our growth patterns and development. It is generally accepted that environmental exposures including smoking, behaviour of mothers during pregnancy, gestational weight gain and birth weight, and the duration of breastfeeding play a major role on growth trajectories, and development of children (Rush, Paterson, Obolonkin, et al., 2008).

The life-course epidemiological approach is a useful interdisciplinary framework from which to study the long term effects during of physical or social exposures during critical life periods (e.g., gestation, childhood, adolescence, young adulthood, and later adult life), on later health or disease risk (Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power, 2003).

Throughout the life course, risk factors might add together as a result of ‘chains of risk’ (Ben-Shlomo & Kuh, 2002, p. 287) where one adverse (or beneficial) exposure or experience is likely to have a ripple effect on another (Ben-Shlomo & Kuh, 2002). This

is certainly true in the case of developing the risk of T2DM, where certain factors during early life can increase the risk for emergence of T2DM in adulthood (Kuh & Ben-Shlomo, 2004). Children born to women with gestational diabetes mellitus are more likely to have T2DM later in their life (Barrett et al., 2013). Despite this, the offspring of obese mothers can be either smaller or larger at birth, but such infants are more likely to become overweight and obese and have higher risk of non-communicable disease such as T2DM (Godfrey et al., 2010).

From a literature search, the SEARCH for Diabetes in Youth Study; appears to be the only longitudinal study with children in which associations of HbA_1c and nutritional factors were explored. The focus was on children with T1DM (Lamichhane et al., 2015). It was concluded that HbA_1c and long term glycaemic control were adversely affected by carbohydrate intake and favourably influenced by protein, leucine and omega-3 (n–3) fatty acid intake, such as eicosapentaenoic acid, (Lamichhane et al., 2015). In the Diabetes in Youth Study the focus was on identifying the nutrients rather than eating patterns.

One of the prospective adult studies that has examined the long-term effects of habitual diet on risks of incident diabetes, coronary heart disease, and mortality was undertaken with participants in Whitehall II study in 1985–1988 on 7731 individuals at age 50 years (Brunner et al., 2008). The oral-glucose tolerance test was used to determine when the incidence of T2DM occurred (Brunner et al., 2008). It was found that compared with the unhealthy eating pattern, which was consisted of white bread, processed meat, fries, and full-cream milk, the healthy eating pattern, which was consisted of fruit, vegetables, whole-meal bread, low fat diary and little alcohol, was associated with reduced risk for T2DM and coronary death and the hazard ratios (95% CI) were 0.74 (0.58, 0.94) and 0.71 (0.51, 0.98) respectively (Brunner et al., 2008).

To study the most frequently eaten foods and to identify associations with growth and body among 4-year old children (n = 739) from Pacific Island Family Study a validated 111-item FFQ was used to collect data on food and beverages (frequency and quantity) consumed over a 4-week period (Rush, Paterson, & Obolonkin, 2008). The foods most frequently consumed per day were bread (1.32 times) (77% consumed white bread) and total milk (0.86 times) (85% consumed standard milk), followed by breakfast cereal (0.83 times), and fruits (0.83 times). The proportion who met the guidelines for daily consumption of fruit and vegetables were 60% and 35% respectively (Rush, Paterson, &

Obolonkin, 2008). There was a positive association between the intake of protein based foods, weight and BMI, but there was a negative correlation between weight and BMI, and frequency of consumption of foods high in fat (Rush, Paterson, & Obolonkin, 2008).

2.5 Sugar-Sweetened Beverages

The NZ Beverage Guidance Panel (2014) describes sugar sweetened beverages (SSBs) as any beverage that contains added caloric sweetener (usually sugar). Soft-drinks/fizzy-drinks, sachet mixes, fruit drinks, cordials, flavoured milks, cold teas/ coffees, and energy/sports drinks are the main categories (New Zealand Beverage Guidance Panel, 2014). Consensus from the literature indicates that high (added) sugar intake has been associated with an increased risk of T2DM, dental caries, obesity, cardiovascular disease, gout, and fatty liver disease, some cancers, and hyperactivity (Te Morenga, Mallard, & Mann, 2013).

With data from three large cohorts (Nurses' Health Study, Health Professionals' Follow-up Study and Women's Genome Health Study) using a genetic predisposition score based on 32 obesity genes, Hu (2013) concluded that higher consumption of SSBs was associated with more pronounced genetics effects on elevated BMI and an increased risk of obesity. This excessive adiposity is the strongest risk factor for diabetes (Ley et al., 2014). In addition, these beverages associated with the development of several additional chronic disease like hypertension, dyslipidaemia, inflammation, and coronary heart disease (Hu, 2013).

In recent decades, there is a worldwide constant increase in consumption of sugar-sweetened beverages (SSBs) (Malik, Popkin, Bray, Després, & Hu, 2010). According to the Key results of the 2002 National Children's Nutrition Survey (CNS2002) (Ministry of Health, 2003), the only child nutrition survey in NZ and 16 years ago, beverages (26%) and sugar & sweets (21%) are the main sources of sucrose (the major contributor to total sugars) for NZ children. Meanwhile, consumption of sucrose was higher among Māori children (males 72 g; females 69 g) than NZ European/ Other (66 g; 58 g) and Pacific children (56 g; 55.2 g) (Ministry of Health, 2003).

The first findings of the national survey of physical activity, sedentary behaviours and dietary habits in 5-24 year-olds in NZ showed that 8% of children aged 5-14 years drank regular fizzy or soft drinks at least once a week (Clinical Trials Research Unit,

2010). While many individuals reported multiple servings of SSBs daily, decreasing the intake of these beverages would improve dietary quality and reduce long-term weight gain and risk of developing T2DM (Ambrosini, Johns, Northstone, Emmett, & Jebb, 2016; Hu, 2013; Te Morenga et al., 2013).

Sucrose, a disaccharide of glucose and fructose, does not have a high impact on blood glucose concentrations per se (Lean & Te Morenga, 2016). However, it is known that a diet high in free sugar particularly in the form of sugary drinks, increases overall energy intake and may have long term effects on health, such as increased risk of dental diseases and NCDs (World Health Organization, 2015).

2.6 Glycaemic index and glycaemic load of the diet

The glycaemic index (GI) concept is a mean of classifying different food sources of carbohydrate (CHO) and CHO-rich foods (e.g. potatoes, rice, cereals, where the energy content-80% from CHO) in the diet, according to how quickly and for how long 50g of carbohydrate of the food raises the blood glucose concentration compared to 50g of glucose (Brouns et al., 2005). Foods that are digested and absorbed slowly are classified as low-GI foods (e.g., wholegrain bread), which lead to a low glycaemic response, whereas high-GI foods (e.g., white bread, white rice) are rapidly digested and absorbed and result in a higher glycaemic response (Brouns et al., 2005). A more refined measure that can be applied to the whole diet is glycaemic load (GL) which takes into account both the amount and the quality of all carbohydrate foods consumed over a defined period. It is calculated as the sum of the GI for each food multiplied by the amount of CHO in each food (Mendez, Covas, Marrugat, Vila, & Schroder, 2009; Willett, Manson, & Liu, 2002).

In a meta-analysis study by Rouhani et al. (2013) the evidence for the effect of GI and GL meals on energy intake among children from six randomised clinical trials published between 1999 and 2012 was assessed. The authors inferred that having a low GI diet (not low GL diet) was associated with reductions in energy intake and therefore may be associated with reduced prevalence of obesity in the future (Rouhani et al., 2013).

2.7 Summary

Children are first screened for T2DM in NZ at the age of 10 years, or at the onset of puberty (Temneanu et al., 2016). In NZ, Pacific and Māori have higher risk for developing T2DM and obesity than the other ethnicities. In addition, the prevalence of obesity among NZ children is high. Based on recommendations and scientific evidence, HbA_1c can be a suitable biomarker for screening among children with less barriers such as no need to be fasting, compared to other common screening test (fasting plasma glucose). The existing validation studies for POCT testing apply to adult populations with a high prevalence of T2DM. There is a need to validate the AFINION ™ AS100 Analyser for screening NZ children at high-risk of T2DM, as the existing studies have not examined the sensitivity and specificity in the general adolescent population. This is addressed in proposed "point of care test for HbA_1c: performance and utility" study (Chapter 4).

On the other hand, diet has a key role in preventing or proceeding the initiation of T2DM and obesity and it is modifiable. Eating pattern analysis and consideration of food combinations are considered as more comprehensive and holistic overview of the diet in understanding the risk of diseases such as T2DM compared to individual food or nutrient approaches. Different dietary assessment methods such food frequency questionnaire (FFQ), 24 hour dietary recall or foods records have been the common methods to collect dietary information for identifying eating patterns. However, there is a lack of research on eating patterns among healthy children with the focus on risk for development of T2DM. Part of the methodological challenge is how to collect reliable and accurate dietary data and identify appropriate biomarkers, which are related to later health (Burrows, Martin, & Collins, 2010; Wall et al., 2013).

In addition, results from national health surveys clearly show that in NZ South Asian, Pacific and Māori have a higher prevalence of risk and more NCDs than European, but evidence from longitudinal studies that are not only analyzed by ethnicity, but birth cohorts can provide a better understanding of the developmental origins of health and disease (Rush, 2012). The two examples of Auckland birth cohorts are the PIF and MiGTOFU studies- these studies both include groups of children with a high-risk for nutrition-related NCDs including T2DM.

The aim of this PhD study, was to identify patterns of eating among children and explore associations with a biomarker of T2DM; glycated haemoglobin A_1c (HbA_1c). In

addition, body size and serum uric acid were explored as markers of risk. Specific questions asked in each chapter are listed in Table 2.4. There is a need to answer these questions about markers of risk for T2DM, eating patterns, HbA_1c, body size and SUA and their associations to understand better the early emergence of T2DM in children.

Table 2.4. Research questions by chapter

Chapter	Question
3	How best to analyse the children's eating patterns investigated in this thesis?
4	What are the construct validity and intra-individual variability measurements of HbA_1c applying Afinion ™ analysers among Pacific children?
5	1. What are the eating patterns derived from cluster analysis among Pacific children at the age of 14 years? 2. What are the associations between the derived eating patterns and HbA1c? 3. What are the associations between the derived eating patterns with biomarkers and body size measures? Body size in children is a measure of growth. 4. What are the associations between derived eating patterns and early life factors including birth weight, type of baby's feeding, mothers' education and age at conception? 5. What are the associations between early life factors, derived eating patterns and current HbA1c in this population?
6	1. What are the most frequently consumed foods containing added and simple sugars among Pacific children? 2. What are the associations between these frequently consumed foods and HbA_1c? 3. What are the associations between these frequently consumed foods with SUA concentration? 4. What are the associations between T2DM biomarkers (HbA_1c and SUA concentrations) and body size measurements?
7	1. What are the eating patterns among children born to mothers with GDM at the age of 7-9 years? 2. What are the associations between the derived eating patterns and HbA_1c? 3. What are the associations between the derived eating patterns and current factors including body size measurements? 4. What are the associations between the derived eating patterns and early life factors including birth weight, type of baby's feeding, mothers' education and age at conception? 5. What are the associations between early life factors, derived eating patterns, and current HbA_1c in this population?

Chapter 3 Literature review: identification of eating patterns

Identification of what foods free-living people eat is possible through direct observation, recording foods by asking them, or getting them to complete participant questionnaires such as food records (weighed or with household measures), food diaries, 24-hour food recalls, FFQ, or diet histories and check lists. One simple approach of identifying eating patterns of a population is to look at habitual foods such as fruit and vegetables, and assess the intake of those. An example is the dietary habits questionnaire used in the NZ National Health Nutrition Survey (University of Otago & Ministry of Health, 2011a). The dietary habits questionnaire has an advantage; short dietary questions can assess usual eating patterns and specific food habits associated with major nutrition-related risk factors. Compared with detailed food diary records, these short questions reduce the burden of completion on participants and staff and improve compliance to complete questionnaires. Additionally, summary information can be obtained for a surveyed population. Also of interest from dietary habits questionnaires, is the eating pattern that looks at the whole diet and the combinations of the foods eaten by an individual.

‘Eating pattern analysis’ (also named food pattern or dietary pattern analysis) is a food-based approach which 1) characterizes eating behaviour, and 2) explains the association of the whole diet with health benefits and/or risk for disease such as T2DM. The first reason has a descriptive purpose and the second reason quantitatively tests hypotheses (Moeller et al., 2007) and allows inferences to be drawn from the analysis. Both rely on information about how often foods are eaten.

The question asked of the literature was what would be the best way to analyse the children’s eating patterns in the cohorts to be investigated? The first step was to identify the approaches available (Figure 3.1).

Generally, there are two main and common approaches for deriving eating patterns from dietary data: the *a priori* approach (theoretically defined eating patterns) and the *a posteriori* approach (empirically derived eating patterns) (Devlin, McNulty, Nugent, & Gibney, 2012; Moeller et al., 2007; Newby & Tucker, 2004). Both of these can employ a variety of analytical tools that correspond to a specific path of enquiry (Figure 3.1). The first and most important step in analysing eating patterns is to clarify the purpose for which the information is collected and to demonstrate alignment with any research

question investigated. Although the derived eating patterns from both *a priori* and *a posteriori* approaches are comparable, the choice of the analytical technique depends on the outcome sought and the research question(s) (Devlin et al., 2012; Krebs-Smith, Subar, & Reedy, 2015).

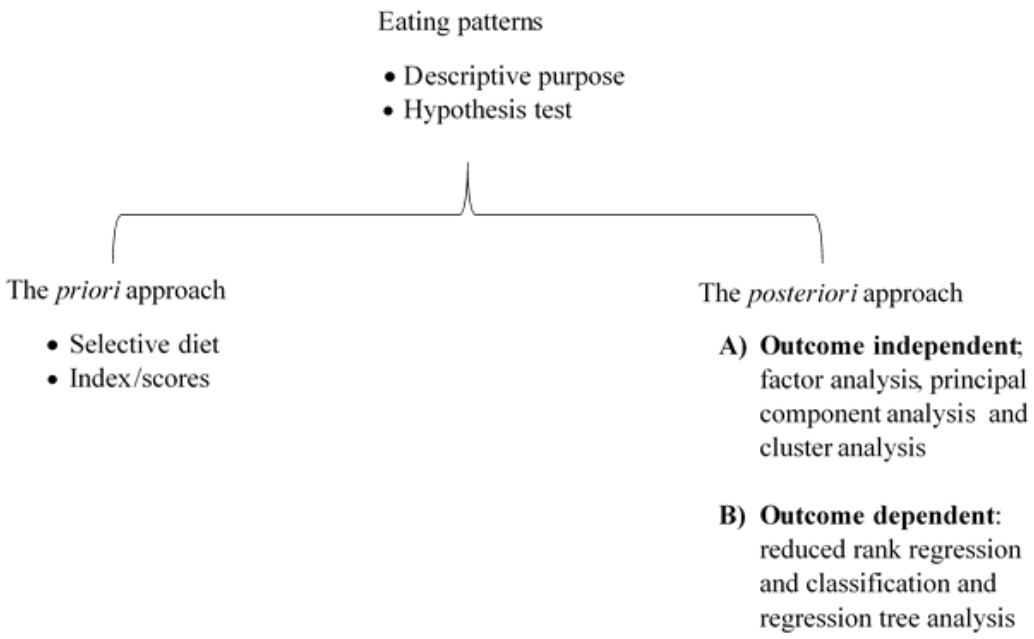


Figure 3.1. Approaches to analysis of eating patterns

3.1 Theoretically defined eating patterns (*a priori*)

Informed by current nutrition knowledge, this approach is known as “diet-quality scores” or “investigator defined” methods, because the characteristics of the eating patterns’ are specified by the researcher *a priori* (Hoffmann et al., 2004; Krebs-Smith et al., 2015; Miller et al., 2010). The compliance with a pre-existing diet quality index, current dietary guidelines, or a specific eating pattern is quantified for each participant, then the researcher will assign diet scores or rank to reflect the level of adherence (Hoffmann et al., 2004; Waijers et al., 2007). The weaknesses of the diet-quality score method are: 1) focusing only on selected aspects and foods in the diet, 2) disregarding the interrelationships between the structures of meals and food and nutrient intakes, and 3) inability of the assigned scores in describing the eating pattern in a practical or useful way (Hoffmann et al., 2004).

3.2 Empirically derived eating patterns (*a posteriori*)

Empirical derivation of eating patterns is also described as a “data driven” approach, utilises different and more objective statistical methods to describe the combinations and frequency of consumption of foods, such as eating patterns empirically i.e. realistically (Miller et al., 2010; Waijers et al., 2007). The statistical tools applied may include principal components analysis (PCA), factor analysis (FA), cluster analysis, and reduced ranked regression (RRR). One of the methodological considerations is the “inherent subjectivity” (Krebs-Smith et al., 2015; Newby & Tucker, 2004). In a review of ninety three studies in which eating patterns were derived from factor and cluster analyses, Newby and Tucker (2004) revealed that subjectivity creates bias both during the analysis and the derivation of the eating patterns. This subjectivity influences a series of decisions that are required. For instance, decisions about grouping of the primary food items into an aggregate variable before entering into analysis can raise questions such as how to group the foods and how to weight the food data. For the latter, decisions could be whether to adjust, for example, for energy intake and/or to transform the variables and standardize them. Some statistical tests are sensitive to outliers and it is common for the distribution of frequencies of foods eaten for a population group to be skewed. Decisions also need to be made about how to treat missing data and foods that are not consumed by an individual. Ongoing decisions include how many eating patterns must be retained to be useful (dependent on sample size) and how those patterns should be named.

Usually there is a need to group food items before applying cluster and FA. But this grouping depends on both research questions and how other studies with the same research design, defined food groups (Newby & Tucker, 2004). For instance, looking at risk for obesity may mean there is a need for grouping the foods with a high energy profiles or high glycaemic indices.

In Newby and Tucker’s (2004) systematic review of eating pattern studies, there were a variety of different criteria for the eating pattern labels utilised. Criteria included rank or order (for example based on the highest factor loading in the FA such as fruit, vegetables and cereals), or according to macronutrient- based descriptions of patterns like high-fat or high-energy density (Newby & Tucker, 2004). However, it is more common to label the eating patterns qualitatively based on specific combinations of foods and/or descriptions of nutrition profile (for example “healthy” pattern when the

high factor loadings are for fruits, vegetables, grains, and low fat dairy) (Newby & Tucker, 2004).

The second step in analysing food patterns is to determine which of the available methods, alone or in combinations, would be the best choice for achieving an analysis that can inform dietary recommendations (Krebs-Smith et al., 2015). Moeller et al. (2007, p. 1237) listed the following questions that assist with guiding which research approach to use.

> "What dietary patterns are shown by the population?
>
> How close is the individual (or population) to meeting dietary recommendations on key aspects of diet?
>
> Who is and who is not following the recommendations?
>
> Do derived patterns relate to biomarkers and measures of health?
>
> Which dietary patterns are most predictive of health outcomes?
>
> What combinations of foods consumed explain the most variation in a specific health outcome?
>
> How does adherence to dietary recommendations relate to health outcomes?" (Moeller et al., 2007, p. 1237)

Each of the listed questions has to be answered through the application of appropriate methods (Moeller et al., 2007). It is common to apply PCA and FA as exploratory methods to derive patterns that depend on dietary habits of the study population (Barbaresko et al., 2014). Both PCA and FA have unique, but different characteristics with derivation of the eating patterns. With PCA the maximum variation in food intake can be explained, so PCA is more likely to capture the real-world eating patterns. However, by applying FA the focus changes to understanding the underlying co-dependencies of explanatory variables (Barbaresko et al., 2014). A concrete example of the use of FA is demonstrated with a publication of a study with 1976 Portuguese children aged between 5 and 10 years, where the researchers aimed to describe derived eating patterns by FA according to gender, parental education, physical activity and obesity (Moreira et al., 2010). Based on eight eating patterns derived from FA the generalized linear models showed that watching ≥ 2 hours/day TV (ß coefficient 0.249, standard error 0.070, 95% CI [0.114, 0.385], $p < 0.001$) and male gender (ß coefficient

0.135, standard error 0.041, 95% CI [0.055, 0.215], $p = 0.001$) were the significant positive predictors for fast food, sugar sweetened beverages, and pastry pattern. While a higher level of maternal education >12 years (ß coefficient 0.107, standard error 0.045, 95% CI [0.012, 0.186], $p = 0.026$) and ≥10 hours/day of sleep (ß coefficient 0.099, standard error 0.053, 95% CI [0.002, 0.211], $p = 0.045$) were positively associated with vegetables, pulses, fruit and olive oil pattern (Moreira et al., 2010).

In FA, smaller numbers of food groupings and food intake interrelationships (covariance) can be examined; this is while PCA takes a much broader overview of the patterns of variance with larger datasets. Critique of both PCA and FA methods includes: 1) researchers subjectivity is required at several analytical steps and 2) there is a poor relationship between the patterns generated and disease risk (Barbaresko et al., 2014) particularly in cross-sectional studies, where the disease may already be present and the diet changed in response to the disease.

Nutritional epidemiologists experience challenges with both collecting and analysing dietary data about foods consumed and presenting this information in a meaningful way (Slattery, 2008). One criticism in much of the literature on the reproducibility and validity of eating patterns is the nature of the diet assessment instrument (e.g., food frequency questionnaire [FFQ], 24-hour recalls, diet-records) and the subjective decisions made by researchers during the analyses or during the development of a food instrument (Moeller et al., 2007).

Table 3.1. Examples of questions concerning eating patterns using *a priori* and *a posteriori* approaches and their associated methods

Approach	Method examples	Research question relevant for the method
a priori	Index/scores	How well is the population meeting a set of food guidelines' recommendations?
	Selective diet	Do persons with a particular eating pattern (such as vegetarian or gluten-free) have a different risk with regard to a health outcome?
a posteriori		
a) Independent outcome	Factor analysis	What combination of correlated foods can explain variation in diets?
	Cluster analysis	Are there any groups of individuals with distinct eating patterns?
b) Dependent outcome	Reduced rank regression	What food groups explains in a set of intermediate health markers? Then, in confirmatory analysis, does that pattern correlate to the outcome of interest?
	Classification and regression tree analysis	Which components of diet can explain the most variation in a health outcome?

Table adopted from Krebs and et al. (2015)

In summary, there is no one analytical approach to deriving eating patterns that is considered better than others. Each approach has its own strengths and limitations and which is really depend on the given purpose of a study (Miller et al., 2010; Moeller et al., 2007).

These differences in generating eating patterns have been explained in two studies. In the Avon Longitudinal Study of Parents and Children (ALSPAC) the eating patterns of 7 year- old children (n = 8279) derived from both cluster and PCA were compared. Both methods generated three eating patterns, very similar in the food items included and sociodemographic characteristics such that authors concluded that both methods were suitable for deriving meaningful eating patterns (Smith et al., 2011). In another nested German case-control study within the European Prospective Investigation into Cancer and Nutrition-Potsdam study 193 cases with incident T2DM and 385 controls were looked at. In this study, the RRR method was applied to extract eating patterns from 49 food groups, specifying four diabetes-related nutrients and nutrient ratios as responses, and the results were compared with PCA (Hoffmann et al., 2004). Authors

found that the four derived factors (eating patterns) by RRR explained 93.1% of response variation, while the first four factors extracted by PCA accounted for only 41.9% (Hoffmann et al., 2004). This highlights RRR as an appropriate statistical method for exploring which eating patterns were associated with the development of diseases by combining prior knowledge gained from previous studies and dietary information from a current study (Hoffmann et al., 2004).

When studying eating patterns as an approach in nutritional epidemiology, an important consideration is that rather than considering the consumption of individual food items, the whole diet of people should be studied. The dietary information can be collected using a dietary assessment method such as FFQ. There are many differences in the profile of nutrient and food content of the generated eating patterns, so this approach cannot identify specific nutrients that are responsible for increasing the risk of getting a disease, nor be able to explain the biological relationships between eating patterns and disease risk (Hu et al., 2000). That means the derived associations between eating patterns and disease risk should be evaluated in light of other evidence from studies on individual nutrients or food consumption (Hu et al., 2000)

Next section discusses cluster analysis as a statistical method using a *posteriori* approach for deriving eating patterns, and the strengths and alignment of this with the research questions in this body of research.

3.3 Cluster analysis

Cluster analysis is also known as an outcome-independent statistical method (Krebs-Smith et al., 2015). This technique is an exploratory analysis that aims at separating individuals into mutually exclusive, non-overlapping clusters (eating patterns), based on similarities among foods and food groupings consumed (Devlin et al., 2012; Emmett et al., 2015). In other words, “cluster analysis aggregates individuals into relatively homogenous clusters with similar diets” (Hu, 2002).

In applying cluster analysis, food variables entered into the analysis can be nutrients, foods or food groups, or a combination of all three (Togo et al., 2001) (Figure 3.2). Each individual can be classified into a distinct cluster according to the frequency of consumed food, the percentage of energy contribution of each food or food group, the average mass of foods eaten, standardized nutrient intakes, or a combination of dietary and biochemical measurements (Hu, 2002). This method represents a specific cluster

(eating pattern), where an individual may belong to only one cluster, and each cluster has a unique food and nutrient composition, and the differences between derived clusters are related to the mean of consumed foods of each individual (Devlin et al., 2012). Clusters can then be used as categorical (nominal) variables in analysis (Moeller et al., 2007).

There are various algorithms that can be applied in the clustering analysis such as K-means, Ward's or Partitioning Around Medoids (PAM) algorithms (Devlin et al., 2012). The aim of all clustering algorithms is to evaluate the "Euclidean distance", which "measures the distance between each food item consumed together by similar individuals" (Devlin et al., 2012, p. 600). The differences between the K-means clustering algorithms, has been comprehensively discussed by Aldenderfer and Blashfield (1984). Ward's method is described as hierarchical, which is able to optimize the minimum variance within clusters by "within-groups sum of squares" or the "error sum of squares". In contrast, K-means is non-hierarchical and iterative, which is also termed as a "nearest centroid sorting pass" (Aldenderfer & Blashfield, 1984, p. 47). K-means attempts to reassign the cases to the cluster with the nearest mean (centroid) and measure a multi-dimensional version of the mean for each of the clusters (Aldenderfer & Blashfield, 1984; Campain et al., 2003). In comparison with K-means algorithm, the PAM algorithm calculates a multi-dimensional version of the median or medoid and since it is less sensitive to outliers, it is considered more robust compared to the K-means (Campain et al., 2003).

For the K-means and PAM algorithms the number of clusters need to be pre-specified, but for Ward's method it does not (Campain et al., 2003; Moeller et al., 2007). Two approaches have been suggested to pre-specify the number of clusters: to calculate variance ratio and/or to generate the scree plots (Devlin et al., 2012) which enables visualisation of the contribution of each factor e.g. food component to the variance explained which is plotted as an Eigen value. The range for the number of derived clusters can be 2 to 8, but it is common to report 5 or 6 clusters (Newby & Tucker, 2004). A smaller number of clusters can be more easily interpreted (Michels & Schulze, 2005). However, there are not any reference methods for identifying the number of clusters and that is finally decided by the researcher who needs to ensure clear, distinct, and nutritionally meaningful clusters (Devlin et al., 2012).

In summary, the two most common cluster analysis methods in deriving eating patterns are K-Means and Ward's Method (Moeller et al., 2007; Newby & Tucker, 2004). Moeller et al. (2007) suggested that utilizing both K-Means and Ward's Method would result in better understanding of the best number of clusters in the data.

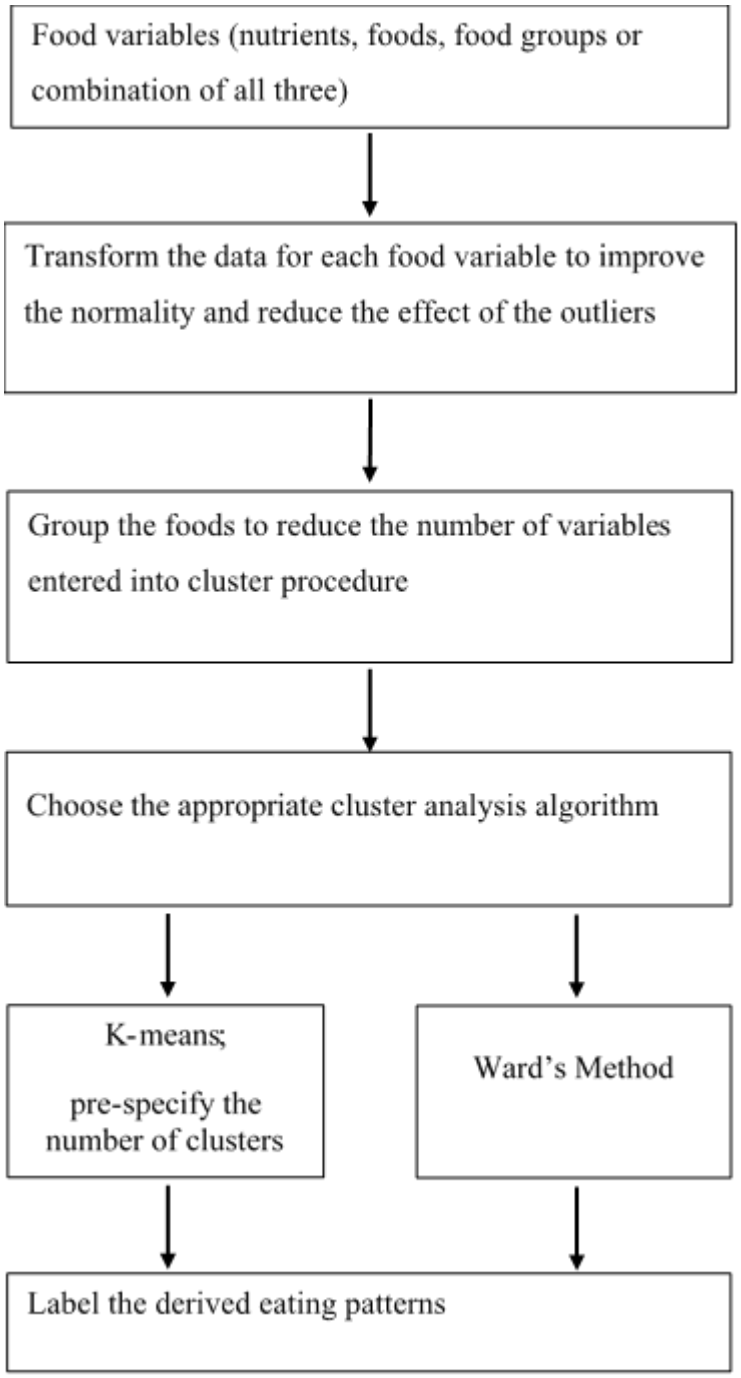


Figure 3.2. Cluster analysis step-by-step

3.3.1 Advantages of using cluster analysis for generating eating patterns

Cluster analysis can be helpful in identifying people in the "healthy population", who may be at nutritional risk. This is done by deriving eating patterns that are lacking or have an excess of specific nutrients based on published recommendations in which the influence of factors such as sex, age, socio-economic status or weight (Devlin et al., 2012) are also considered . For instance, in the Avon Longitudinal Study of parents and children (ALSPAC) at the age of 7 (n = 6837), 10 (n = 6972) and 13 years (n = 5661), k-means cluster analysis was applied (Northstone, Smith, Newby, & Emmett, 2013). Four clusters (eating patterns) were derived at each age: 'processed' (i.e., high in processed foods, chips and soft drinks), 'healthy' (i.e., high in fruit, vegetables, high-fibre bread and water), 'traditional' (i.e., high in meat, potatoes and vegetables) and 'packed lunch' (i.e., high in white bread, sandwich fillings and snacks) (Northstone et al., 2013). Tracking children's eating patterns across ages showed that the children whose mothers had the highest level of education (i.e., attained a qualification in addition to the O level) were nine times more likely to stay in the 'healthy' cluster during all three time points, compared to those with the lowest education (i.e. did not attain O level) (adjusted OR 8·83; 95% CI [4·58, 17·01]). The authors concluded that the changes in eating patterns could be tracked by using cluster analysis over time and this might be an important factor influencing the onset and development of onset of disease later in life (Northstone et al., 2013).

The second advantage of using cluster analysis to obtain eating patterns is that the associations of derived eating patterns with risk for NCDs can be examined (Devlin et al., 2012). In the National Health and Nutrition Examination Survey 2001-2002, LaRowe, Moeller and Adams (2007) assessed the diet quality and body mass index (BMI) in children aged 2 to 5 years (n = 541) and 6 to 11 years (n = 793) by analysing beverage consumption patterns using cluster analysis. Four beverage patterns were derived for children aged 2 to 5 years: 'mix light drinker', 'high-fat milk', 'water' and 'fruit juices'. For children aged 6 to 11 five beverage patterns were derived: 'mix light drinker', 'high-fat milk', 'water', 'sweetened' and 'soda'. It was revealed that only in children aged 6 to 11 years BMI was significantly ($p < 0.05$) higher in the water, sweetened drinks and soda patterns (adjusted mean BMI = 19.9, 18.7, and 18.7 kg/m^2 respectively) compared to mix/light drinker and high-fat milk patterns (adjusted mean BMI = 18.2 and 17.8 kg/m^2 respectively) (LaRowe et al., 2007).

Studying the relationships between eating patterns with nutritional biomarkers such as plasma folate, carotenoids, or energy expenditure, is a third way of understanding the relationships between diet and health. In the Special Turku Coronary Risk Factor Intervention Project a prospective randomized controlled trial from 7 months to 7 years of age the relationship of derived eating patterns of the 7-year old children (n = 1,062) were examined against serum lipids including total cholesterol, high density lipoprotein cholesterol, and serum triglyceride (Räsänen et al., 2002). In addition, daily energy and nutrient intakes were calculated using the Micro-Nutrica program (Räsänen et al., 2002). Four eating patterns were derived (cluster 1 = 1.5% fat milk and butter, cluster 2 = sugar and sweets, cluster 3 = cereal, rice and pasta, cluster 4 = bread, skim milk, and margarine) using K-means cluster analysis. Daily intake of cholesterol was significantly ($p < 0.05$) lower in children who had bread, skim milk, and margarine (mean cholesterol l57 ± 51 mg) compared to children, who had 1.5% fat milk and butter (196 ± 57 mg) but blood lipids were not different. This is while the ratios of polyunsaturated to saturated fat (ideally 1.0 for cardiovascular health) was higher in the bread, skim milk, and margarine eating pattern (0.50 ± 0.13) compared to 1.5% fat milk and butter eating pattern (0.34 ± 0.10) (Räsänen et al., 2002).

3.3.2 Limitations of using cluster analysis

The major limitation in applying cluster analysis is the potential for inherent subjectivity and research bias, which may affect the number and type of derived, reported, and the patterns analysed (Newby & Tucker, 2004). Some examples of the subjective decisions that need to be made by the researcher at different stages of collecting food information, deriving, analysing and reporting eating patterns are:

1. To determine which type of dietary assessment method such as FFQ and/or 24-hour food recall is applied,
2. To decide about transforming food data, because cluster analysis is very sensitive to outliers since variables (i.e., consumed foods) with large variances have a greater effect on the outcome of clusters than those with small variances (Michels & Schulze, 2005; Newby & Tucker, 2004),
3. To decide whether or not to reduce the number of the food items considered by collapsing them into smaller groups before entering into cluster analysis (known as treating data); one reason for this is that the food groups can represent total consumed food, considering any interactions between nutrients within the groups (Devlin et al., 2012) and the proportion of explained variance per factor (here

clusters) decreases with the number of entered variables (Michels & Schulze, 2005),

4. According to the selected cluster method (e.g., K-means or Ward's Method), the number of clusters need to be pre-specified by researchers (Michels & Schulze, 2005; Newby & Tucker, 2004).

These limitations can be partially overcome by exploratory and iterative analyses of the data to ensure that the subjective decisions are statistically robust.

3.4 Rationale for derivation of eating patterns in this body of work

In consideration of the variety of above-mentioned decisions, comparing the results of eating pattern analysis with other studies is challenging. Although the methods of collecting food data are comparable and food group construction is standardized (Krebs-Smith et al., 2015), every population and culture has their own unique eating patterns and pattern of disease risk and biomarkers and which may not be necessarily similar to other populations.

In this PhD study, the research question is whether there are groups of children with distinct eating patterns and if the derived eating patterns have relationship with HbA_1c, as a biomarker for screening children who are at the risk of developing T2DM. According to the above critical review of eating patterns analysis methods, the decision was made by the author to apply cluster analysis with the use of K-means and Wards method algorithms and then choose the most nutritionally-meaningful cluster to answer the research questions in this body of work.

Chapter 4 Point of care test for HbA_1c: performance and utility

4.1 Introduction

The prevalence of type 2 diabetes mellitus (T2DM) among children and adolescents is increasing globally. Within the Auckland region of NZ, the incidence of T2DM in children <15 years increased fivefold (0.5/100,000 to 2.5/100,000) from 1995 to 2007 (Jefferies et al., 2012). Moreover, the prevalence of diabetes in NZ is three times higher among Pacific peoples aged over 15 years than among non-Pacific peoples (Ministry of Health, 2013a). The 2014/15 New Zealand Health Survey found that approximately one third (30%) of Pacific children were obese (a major risk factor for developing T2DM as adults) (Ministry of Health, 2015a). This is three times higher than that for non-Pacific children. Therefore, screening and diagnosis of T2DM at an earlier age in high-risk groups such as Pacific children could lead to timely actions to prevent and reduce future complications.

Since 2000, point-of-care test (POCT) HbA_1c measurements have been used as a routine method in the management of individuals with diabetes (Wood et al., 2012). As a widely used biomarker, HbA_1c reflects a person's average blood glucose over the last two to three months (American Diabetes Association, 2010). The International Expert Committee (2009) and American Diabetes Association (2014) advise that HbA_1c is both a biomarker for risk for diabetes and a diagnostic tool - with the added advantage that unlike blood glucose, fasting and a two hour blood test following a glucose challenge are not required. Measuring HbA_1c with new devices using capillary blood derived from a finger prick instead of the conventional venipuncture requires minimal personnel training and can be easily applied by researchers, physicians, and nurses (Zin, Kamil, Soh, Embong, & Mohamud, 2013). Most clinicians agree that HbA_1c measures are helpful when undertaken at a consultation visit as these may be discussed with patients face to face (Little, 2012; Zin et al., 2013). Moreover, one of the strategic themes of the New Zealand Health Strategy is a "smart system" that focuses on "having reliable, accurate information that is available at the point of care" (Ministry of Health, 2016d, p. 33).

Internationally, laboratory assays for measuring HbA_1c and diagnosing diabetes are highly standardized so that their results can be uniformly applied both across time and populations (American Diabetes Association, 2010; Sacks et al., 2011; World Health

Organization, 2011). However, the National Academy of Clinical Biochemistry and the Evidence Based Laboratory Medicine Committee of the American Association for Clinical Chemistry do not recommend using the POCT HbA_1c assays as the generated results are not reliable enough for the diagnosis of diabetes (Sacks et al., 2011). The quality of evidence for this recommendation is rated as moderate, which means further research is likely to have an important impact on our confidence in the diagnosis of diabetes mellitus by cut-off values. An added proviso is that red blood cell turnover, iron deficiency, haemoglobinopathies, and haemodilution which are associated with pregnancy, should be considered when applying cut-offs. Investigations may change the current estimate and recommendations (Sacks et al., 2011). The limited high quality evidence on accuracy of POCT HbA_1c assays may be the key reason these are not recommended by the American Diabetes Association and National Academy of Clinical Biochemistry for diabetes diagnosis (Little, 2012; Sacks et al., 2011; Sølvik, Røraas, Christensen, & Sandberg, 2013). Therefore, it is crucial to ensure the reliability and validity of the POCT instrumentation, analysis, and assay use; otherwise it can affect the diagnosis of the disease or its treatment plan, with an immense impact on the patient (Lenters-Westra & Slingerland, 2014).

The “Afinion ™ AS100 Analyser" (Alere, AXIS-Shield POCT, Oslo, Norway) is a compact multi-assay analyser for point-of-care testing (Alere, 2012), which was released in 2005. The Afinion POCT for HbA_1c is a fully automated boronate-affinity reflectance assay through which the sample is drawn into an integrated sampling device containing all necessary materials. The principles of the Afinion POCT for HbA_1c have been discussed briefly in some published papers (Criel, Jonckheere, & Langlois, 2016; Wood et al., 2012). Studies have compared the accuracy and precision of the Afinion POCT with other POCT devices or reference methods in samples of healthy adults and adults with diabetes (Criel et al., 2016; Petersen et al., 2010). They reported precision coefficients of variation (CV) of less than 2.5% for HbA_1c (Criel et al., 2016; Petersen et al., 2010). Of the few studies in paediatric and children settings, only one examined the performance of the Afinion included 688 children diagnosed and treated for diabetes. In this study, HbA_1c values were higher than the reference method by 1.5 $mmol.mol^{-1}$ (0.15%) (Wood et al., 2012). The use of the Afinion POCT to screen for elevated HbA_1c has not previously been reported in a paediatric population at high-risk of T2DM.

Young Pacific people are at high-risk for T2DM. The Pacific Islands Families (PIF) study is a prospective longitudinal study tracking the health and development of a birth cohort of Pacific children born in Auckland, NZ in the year 2000. When followed-up at age 14 years, the prevalence of overweight and obesity was more than 70%; this included 30%, who were overweight (Rush, Oliver, et al., 2016). At the same time as the height and weight were measured in schools, a finger prick blood sample was taken and analysed using a POCT with the Afinion ™ AS100 Analyser.

The aim of this study was to test the reliability and validity of a screening HbA_1c test as measured by an Afinion ™ AS100 Analyser with HbA_1c measured at an accredited medical laboratory as a reference method (validity phase). The information was collected from Pacific children (n = 204) approximately one year later in a nested sub-study of the PIF children measured in the field. In addition, the HbA_1c measured in the same children one year apart in different settings by two different Afinion ™ analysers of the same model were utilized to answer the question: what are the construct validity and intra-individual variability (agreement of POCT measures over time) of these measures?

4.2 Methods

4.2.1 Participants

In 2014, 768 Pacific children from the birth cohort of the PIF study had HbA_1c from a finger-prick capillary POCT (Afinion ™ analyser) measured in school setting (Rush, Oliver, et al., 2016). Approximately one year later a nested subsample of children (n = 204) were selected to evenly represent the cohort range of body size, gender, and ethnic profile and take part in a sub-study. This involved with more detailed measurements of body size and composition. Further HbA_1c measured in a fasting state by the same model POCT and from a venipuncture blood sample sent to the hospital laboratory, Lab Plus.

4.2.2 Laboratory method

Venous blood samples were collected (ethylenediaminetetraacetic anticoagulant) in the body composition laboratory, University of Auckland and analysed at the International Accreditation New Zealand accredited Lab Plus medical laboratory at Auckland City Hospital by boronate affinity chromatography assay (CLC385, Primus Corp, Kansas City, MO), a high-performance liquid chromatography method (reference method). Notably, from February 2016 LabPlus changed its method to capillary electrophoresis

(Capillarys, Sebia). Therefore, nine of the children measured after this date were excluded from this analysis.

4.2.3 Point-of-care device method

A capillary blood sample from a finger-prick was drawn into the cuvette. The manufacturer's guidelines were followed for collecting and processing the blood sample and quality control checks with standards supplied by the manufacturer were run regularly.

There are no specific HbA_1c cut-off point currently available for diagnosis of T2DM prediabetes among children. This study used the cut-off point recommended by the New Zealand Society for the Study of Diabetes Working Party who classify ≤ 40 $mmol.mol^{-1}$ (5.8%) as normal glucose tolerance, values of 41– 49 $mmol.mol^{-1}$ (5.9– 6.6%) as pre-diabetes or dysglycaemia, and values of ≥ 50 $mmol.mol^{-1}$ (6.7%) as a diagnosis of diabetes (Braatvedt et al., 2012; Saudek et al., 2008).

Generally, a unit change of 5 $mmol.mol^{-1}$ (0.5%) in HbA_1c is considered as a clinically significant difference in glycaemic status (Little, 2012). A meaningful diagnostic difference between the two methods of measurement could also be considered to be 5 $mmol.mol^{-1}$ (0.5%). The laboratory precision expectation of an HbA_1c measurement is a CV of 3.5% (Sacks et al., 2011).

4.2.4 Statistical analysis

Data are summarised as mean ± standard deviation (SD) and 95% confidence intervals (CI) of the mean are reported. Bland Altman analysis (Altman & Bland, 1983) was applied to analyse the agreement between the two different measurements of HbA_1c used the reference method and the POCT. Paired t test, correlation coefficient and linear regression were used to examine the relationships between the measures on the same individual and to determine any differences (bias) between the finger-prick and venous sample drawn and analysed on the same day and the POCT measures from two different POCT analysers (same model) taken one year apart. A p value of less than 0.05 was considered statistically significant. Statistical analyses were performed using SPSS software v22.0 (SPSS Inc., New York, USA).

4.3 Results

The results are presented in two parts: (A) validity phase, in which the POCT and reference laboratory blood for HbA_1c were sampled at the same time, and; (B) agreement of POCT where repeat measures on the same children one year apart are compared.

4.3.1 Validity phase

Of the 204 sub-study participants aged 15 years, 190 children (girls = 94, boys = 96) with an average body mass index of 28.2 ± 6.8 kg/m^2 had HbA_1c determined by both POCT and the reference method. Two children had previously diagnosed T1DM. The mean of measurements was 36.24 ± 4.95 mmol.mol^{-1} (5.4 ± 0.4%) and 36.78 ± 5.29 mmol.mol^{-1} (5.5 ± 0.5%) (4.7 to 9.5%) respectively (Figure 4.1). The majority (92%) had an HbA_1c of less than 40 mmol.mol^{-1} (< 5.8%) (Figure 4.2).

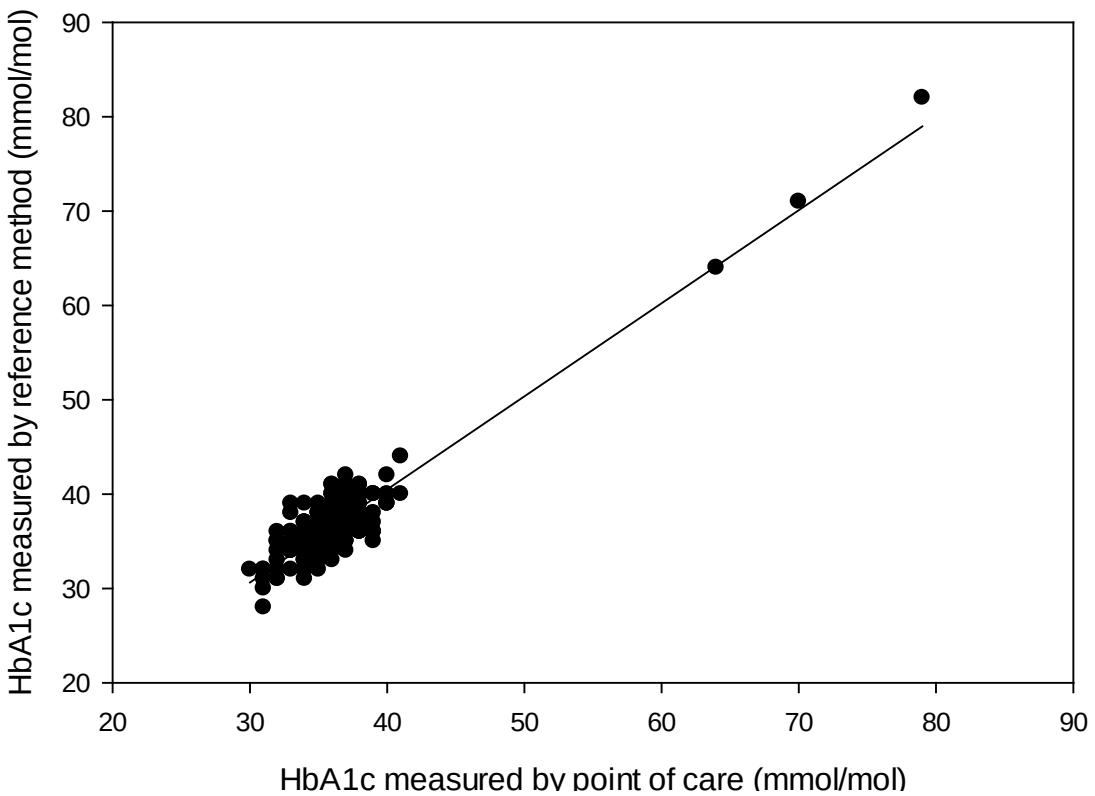


Figure 4.1. Frequency of glycated haemoglobin A_1c (HbA_1c) measured by reference method and point of care

The mean difference between capillary and venous measures was 0.54 mmol.mol^{-1} (0.05%) (95% CI [0.25, 0.83], $p < 0.001$) and the ± 1.96SD limits of agreement: 4.48, -3.40 mmol.mol^{-1} (0.4, -0.3%) (Figure 4.2). This difference is less than the integer value displayed on the instrument screen and not biologically or diagnostically meaningful. The difference between the two measures and the average were weakly correlated (r = 0.169) indicating slight overestimation by POCT at low concentrations of HbA_1c and underestimation at higher concentrations. After exclusion of the three high values the mean difference between capillary and venous measures was 0.53 mmol.mol^{-1} (0.05%) (95% CI [0.24, 0.82]) and the ± 1.96SD limits of agreement: 4.48, -3.42 mmol.mol^{-1} (Figure 4.3).

The 190 measures by the two methods were strongly correlated (r = 0.92, 95% CI [0.90, 0.94], $p< 0.001$) and the equation for HbA_1c from POCT was Reference = 1.002 + 0.987 POCT measure. When only the measures between 28 mmol.mol^{-1} (4.7%) and 45 mmol.mol^{-1} (6.3%) (n = 187) were considered, a strong correlation remained (r = 0.65, 95% CI [0.56, 0.73], $p< 0.001$).

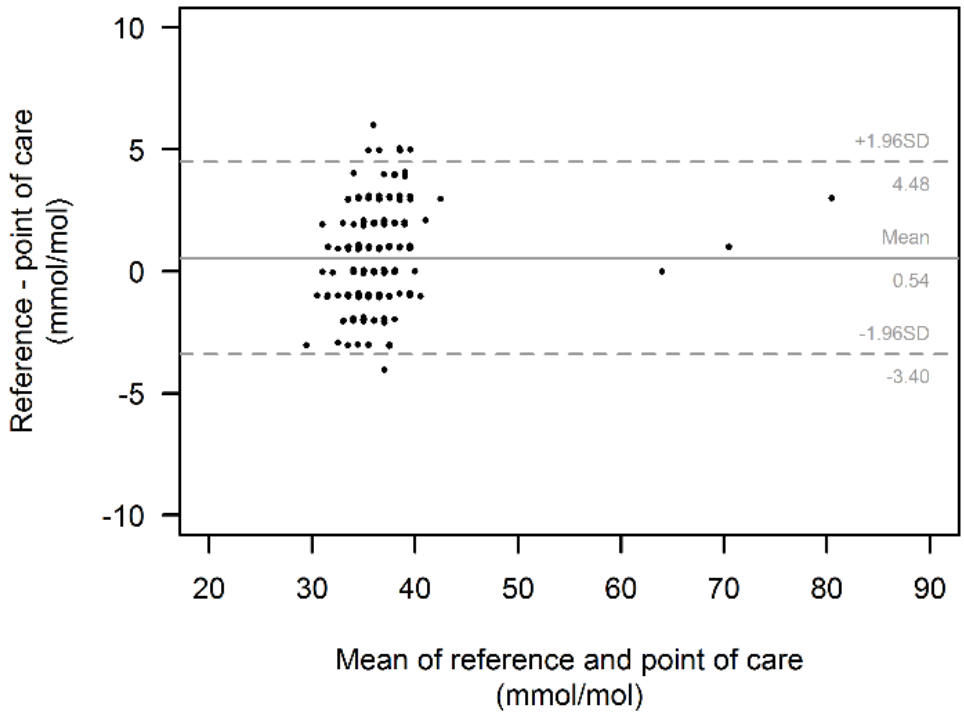


Figure 4.2. Bland-Altman plot of point of care and reference HbA_1c measurements

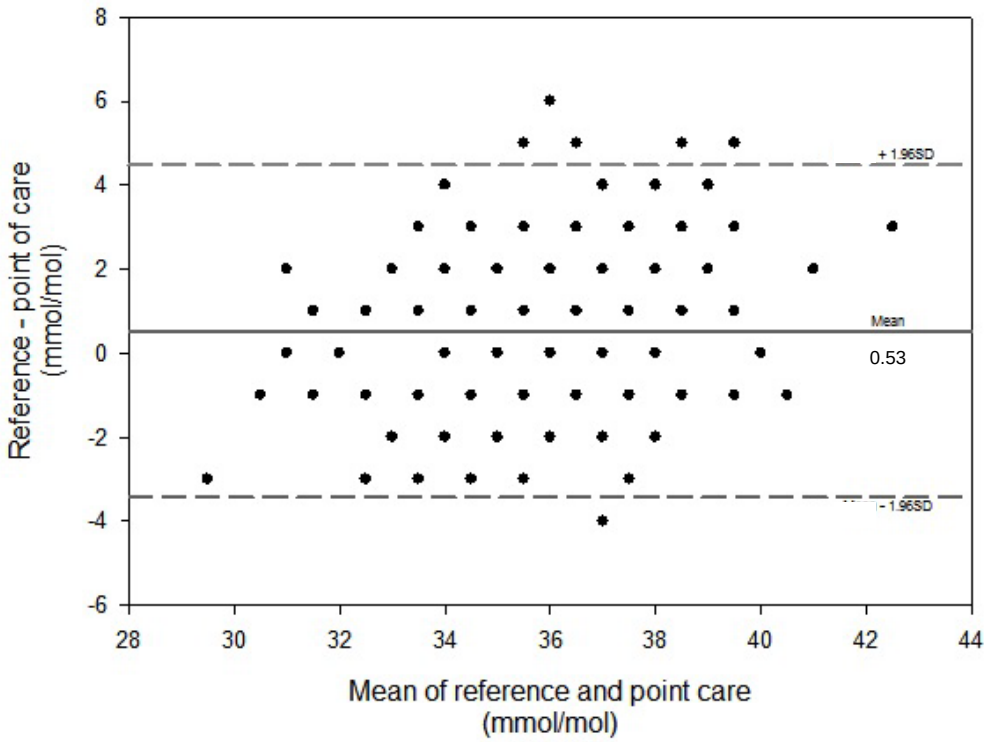


Figure 4.3. Bland-Altman plot of point of care and reference HbA_1c measurements without outliers

4.3.2 Agreement of POCT measures over time

For 32 of the 768 children (4.2%) measured by POCT at school, HbA_1c exceeded 41 $mmol.mol^{-1}$ (> 5.8%) and all were advised to visit their general physicians. Seven with previously elevated HbA_1c were included in the sub-study. Two measurements of HbA_1c by POCT were available for 182 children. Agreement between the two measures one year apart was moderate to strong (r = 0.55, 95% CI [0.44, 0.65], $p < 0.001$) with a mean of difference of 0.14 ± 2.18 $mmol.mol^{-1}$ (0.01 ± 0.2%).

4.4 Discussion

The Afinion ™ POCT performed well when compared with the reference method and also at two time points one year apart. The small difference between the POCT and the reference method (0.5 $mmol.mol^{-1}$, 0.05%) was statistically, but not clinically significant. The present validation with a cohort of children, who had not been screened before, confirms two previous validations with adults with diabetes (Lenters-Westra & Slingerland, 2010; Petersen et al., 2010) and one with children with diabetes (Wood et al., 2012) over the range of 20-103 $mmol.mol^{-1}$ (4 - 11.6%) HbA_1c. Bias of the comparison of Afinion ™ with an electrophoresis reference method ranged from -6 $mmol.mol^{-1}$ (-0.65%) (Petersen et al., 2010), -2 − -1 $mmol.mol^{-1}$ (-0.23 ─ -0.12%) (Lenters-Westra & Slingerland, 2010) and Wood and his colleagues (2010), in the paediatric population found + 1.5 $mmol.mol^{-1}$ (0.15%) difference between HbA_1c measurements.

These small differences compared with reference methods highlight the need for a consistent measurement of HbA_1c in the diagnostic laboratory. Interestingly, when the reference laboratory changed their method - before the completion of this study - a difference to the comparison was observed; we therefore excluded these values. This difference which was the result of using different laboratory methods was also noted by clinicians (i.e., personal communication) and for some patients resulted in a change in their diagnosis, which is usually reliant on a precise cut-off.

Under-or overestimation of HbA_1c using POCT needs to be considered by clinicians in relation to their reference laboratory measures for both treatment regimens and further laboratory blood testing. Moreover, the WHO (2011) recommends the diagnosis of diabetes in asymptomatic patients should be based on additional HbA_1c or plasma glucose test results with a value in the diabetic range, rather than relying on a single abnormal HbA_1c or plasma glucose test. For those with elevated HbA_1c (> 40

mmol.mol^{-1}), which is indicative of “prediabetes” a discussion to optimise prevention strategies should be ensued. Progression rates from prediabetes to diabetes for children are not known; in adults it is in the order of “36 per 1000 person years” (Morris et al., 2013).

The biological construct reliability of this study was supported by a moderate to strong correlation (r = 0.5) between the HbA_1c values measured by two different Afinion POCT analysers, at two different places within a year on the same sample of children. In an extensive search of PubMed and Medline databases, there were not any studies in which the reliability of the Afinion POCT was evaluated at different places or times on the same sample.

In addition to biological variability, other researchers have investigated the impact of changes in cuvette lot number. These researchers have concluded that beyond the analytical performance of this POCT analyser, different regression lines for different lots could be concerning (Dupuy et al., 2014). The Afinion manufacturer (Alere, 2015) reported that they undertook rigorous tests on assay reproducibility for within device precision (CV < 1.2%), lot-to-lot variation of cuvettes (CV < 1.3%) and day-to-day (CV < 0.4%). Additively, this variation is small but it emphasises the need for quality control throughout the supply chain for these measures.

The main limitation of this study was that the distribution of HbA_1c across the range tested was positively skewed – the majority of HbA_1c measurements were less than 45 mmol.mol^{-1} (< 6.3%). However, those with a high HbA_1c were referred for further investigation. The strengths of the study were the feasibility of undertaking this test in a school setting, the adequate biological/construct validity of the measures one year apart, and for some children where venous blood sampling was difficult but the finger-prick happened quickly, easily and generally children participated willingly in the process of sample collection. Therefore, using POCT for screening T2DM as a first step might be more acceptable to be used among this age-range group. Furthermore, the spent in time was less than the laboratory method and material costs were in the order of $NZ12/ test.

The confirmation of the validity and reliability of the POCT HbA_1c is important, because the unique PIF cohort study of children, representative of Pacific children in NZ (Rush et al., 2010) have not previously been screened for risk for diabetes. The Afinion ^{TM} HbA_1c screening test was able to identify Pacific children at risk of hyperglycaemia.

4.5 Recommendation

The dramatic increase in the early life incidence of T2DM emphasizes a need to have robust screening methods such as POCT for HbA_1c. This is especially required if this can be done in a nationally representative sample of young population in NZ. The example of this is the National Diet and Nutrition Survey rolling programme in UK, in which the distribution of HbA_1c and glucose concentrations were collected from a nationally representative sample of the British population (Almoosawi et al., 2014).

In addition, normal ranges for HbA_1c have not been established clearly among children and there is no cut-off for NZ children. Measuring HbA_1c in a sample group of NZ children, who can represent the age and different ethnicities of children population in NZ, would help to provide a national reference for HbA_1c distribution. An example of this type of study is a research undertaken by Saadine and colleagues (2002) among 7,968 American children aged 5-24 years in which racial/ ethnical differences in HbA_1c were considered. The other study in which a cut-off point for HbA_1c was examined to predict T2DM was reported by Mukai and colleagues (2012) in a total of 1982 Japanese participants aged 40-79 years without diabetes. They were followed up for 14 years by annual health examinations and they suggested 37 $mmol.mol^{-1}$ as the cut-off point for predicting T2DM in the Japanese population (Mukai et al., 2012).

The HbA_1c test provides a viable option for future investigations and may actively involve children in conversations about healthier diet and quality physical activity for prevention of T2DM at the time of testing. Cut-off points for action need to be determined and then incorporated into guidelines

4.6 Conclusion

In practical terms, the use of Afinion ^{TM} POCT in this study performed well, with the majority of children reassured that they did not have T2DM. It was also helpful in diagnosing four new cases with HbA_1c higher than the cut-off point, and for two children with T1DM reinforcing that they should be more cautious in managing their condition. However, POCT analysers should routinely be compared to reference methods where and when possible to minimize the risks of underestimated or overestimated measurements with respect to reference methods.

Chapter 5 Eating patterns and risk of type 2 diabetes mellitus: Pacific Islands Families main study

5.1 Introduction

The aetiology of the development of obesity and risk for Non-Communicable Diseases (NCD) is complex and driven by the macro- and micro-environments. Across the lifecycle, eating patterns play a fundamental role in people's wellbeing and health (Katz & Meller, 2014). There is strong research evidence that supports the contribution of suboptimal early life environments, such as maternal environment and epigenetic mechanisms, to adult-onset diseases including obesity and T2DM (Buyken et al., 2010; Godfrey et al., 2010). Additional lifestyle behaviours such as highly processed, refined food consumption and unhealthy eating patterns are other factors that contribute to the development of risk for NCD such as T2DM, obesity, and gout. Moreover, there is evidence that the increase in quantities sugar added to food consumed closely aligns with the dramatic rise in prevalence of obesity, metabolic syndrome, diabetes, and induced hyperuricemia (Johnson et al., 2013; Lean & Te Morenga, 2016).

The number of youngsters in NZ Pacific population is growing; in the 2013 census, 35.7% of Pacific peoples were aged <15 years, compared with only 20.4% of the total population (Ministry of Health, 2014e). The rate of increase in NZ's Pacific population is about three times faster than that for the rest of NZ population (Ministry of Health, 2014c).

Unfortunately, the rate of weight gain in Pacific children from birth is also very rapid (Rush et al., 2010). The number of Pacific obese children aged 2-14 years (27.1%) is more than two times the total NZ obese children (11.1%) (Ministry of Health, 2014c). Furthermore, obesity has a strong, positive relationship with socioeconomic deprivation (Ministry of Health, 2015a). The prevalence of obesity for children (2-14 years), who live in the most deprived areas is five times higher than the children who living in the least deprived neighbourhoods (Ministry of Health, 2015a).

The majority of the Pacific people (65.9%) live in Auckland region, NZ's biggest city (Ministry of Health, 2014e). Of the Auckland Pacific population, 40% live in the Manukau district (Counties Manukau District Health Board, 2015b) followed by 19% in Auckland (Ministry of Health, 2014e). Seventy five percent of the Pacific people, who

live in Counties of Manukau live in the most deprived (deciles 9 and 10) areas (Counties Manukau Health, 2017).

The prevalence of T2DM in adult Pacific is around three times higher than that of other New Zealanders (Ministry of Health, 2016a). Excess weight and childhood obesity are leading contributors to T2DM incidence, but they are potentially modifiable risks to health. The higher prevalence of NCD diseases including T2DM among Pacific people is associated with many factors (Blyth, 2015), but it has been clearly identified that food choices and dietary practices such as feasting affects children's health (Pala et al., 2013) and that these food behaviours and practices persist into adulthood (Mikkilä et al., 2005). The unique birth cohort study of Pacific children (PIF Study), tracked the food consumption frequency of children (n>1000) age 4 to 6 years using a FFQ completed by their caregivers (Savila, Obolonkin, & Rush, 2014). Although eating patterns were not the focus of the previously cited paper, the authors noted that Pacific children consumed the same foods at the same relative frequencies from age 4 through to age 6, which illustrates the consistency of food habits.

Government guidelines for healthy eating for the general public are based on whole foods, and groups of foods, rather than nutrients. Advice is around vegetables and fruits, breads and cereals, milk and milk products and meat and protein sources (Food and Agriculture Organization, 2017). Therefore, it is important to understand which foods are commonly consumed and explore the combinations of foods eaten (patterns) that may have stronger relationship with NCD including T2DM, especially among groups with higher health risks like Pacific people, at younger ages. However, to the author's knowledge there are no, which have investigated the relationship of consumed foods with early risk for T2DM among Pacific people. One reliable biomarker of risk for T2DM suitable for field studies is POCT HbA_1c (Chapter 4). We found that of the 32 children (of the cohort of 741) with $HbA_1c \geq 41$ $mmol.mol^{-1}$ 6 children were overweight and 19 were obese which was not different to the prevalence of overweight and obesity in the full cohort.

This chapter aimed to report foods consumed by the PIF study children at age 14-15 years. This cohort is representative of Pacific children born in the year 2000 and living in South Auckland and in NZ (Rush et al., 2010). The association of eating patterns derived from a dietary habits questionnaire with the objective risk of developing T2DM measured as HbA1c and body size was investigated (Figure 5.1) in order to answer:

1. What are the eating patterns derived from cluster analysis among children at the age of 14 years?
2. What are the associations between the derived eating patterns and HbA_1c?
3. What are the associations between the derived eating patterns with biomarkers and body size measures? Body size in children is a measure of growth.
4. What are the associations between derived eating patterns and early life factors including birth weight, type of baby's feeding, mothers' education and age at conception?
5. What are the associations between early life factors, derived eating patterns and current HbA_1c in this population?

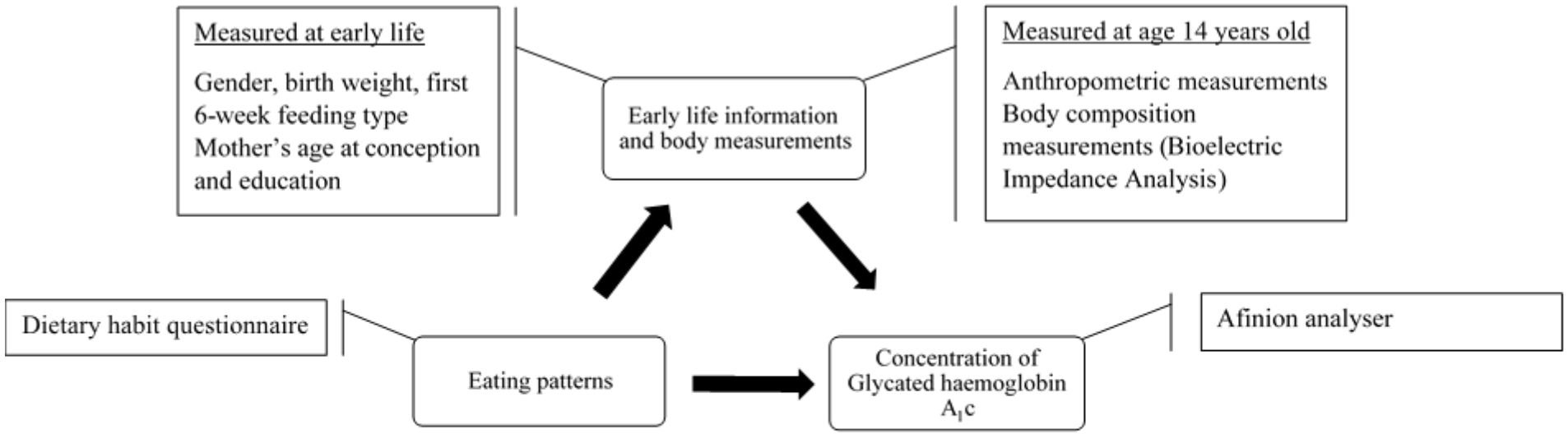


Figure 5.1. Investigation plan and variables considered for analysis

5.2 Design and methods

Selected data from the Pacific Islands Families Study in this thesis were at the age of 14 years. The Pacific Islands Families birth cohort study (PIF) was a large, scientifically and culturally robust longitudinal study that recruited Pacific families (n = 1398) born at Middlemore Hospital (a large tertiary hospital), South Auckland between 15 March and 17 December year 2000 (Paterson et al., 2006). Epidemiological methods were used in the PIF cohort study, which is a prospective, observational design, and quantitative methodology though the use of structured self-report interviews. At six weeks postpartum, the selected mothers were visited in their homes for a one-hour interview and a child assessment was carried out (Paterson et al., 2008). Follow-up interviews/ assessments/ questionnaires with mothers, children, fathers, and the children's teachers were completed at birth, six weeks, 1, 2, 4, 6, 9, 11 and 14 years. At each stage in this birth cohort, the growth and development more than 1000 Pacific children from the year 2000 to 2015 had been followed (Rush, Oliver, et al., 2016). The sample size of the PIF cohort study (n = 1398) enabled generating the findings to the Pacific groups residing in NZ (including Samoan, Tongan, Cook Islands Māori) (Rush, Oliver, et al., 2016).

This section describes the methods used to collect information on consumed foods and to measure concentration of HbA_1c, body composition (i.e., body weight, height and waist circumference, and fat distribution), and life course variables (i.e., birth weight, maternal age at conception, breast feeding and maternal education) of the children, who participated in PIF main study (Table 5.1). The section presents a brief review of the methods used for collecting data, followed by a review of the data processing and statistical analysis that was used to extract results.

Table 5.1. Summary of variables for PIF main study

PIF- main study	Category of the information
Consumed food frequency/ serving size	Food
Finger prick HbA_1c	Biomarker
Body size e.g. BMI z scores, body weight, height and waist, and fat distribution	Body measurements
Maternal age at conception, maternal education, type of baby feeding, birth weight and number of siblings	Early life factors

5.2.1 Study participants

At the age of fourteen years, 931 children from this PIF study (66% retention of the original birth cohort) were followed up (Rush, Oliver, et al., 2016).

5.2.2 Ethical considerations

The ethical approval for the PIF 14-year study was obtained from the Southern Health and Disability Ethics Committee on 4th December 2013 (ref. 13/STH/159) (Appendix A).

Between May 2014 and July 2015 initially, permission to invite the children to participate in the assessment was received from the primary caregivers of children participants. A team of three assessors conducted the group assessments in secondary schools. The children, who could not be assessed at the school, were assessed at their convenient location (including their home or other requested locations including overseas countries like Australia). The details of children participation from the birth cohort have been reviewed by Paterson and her colleagues (2006) and Rush and her colleagues (2016) summarised the recruitment of the children at the age of 14 years. Children who participated were thanked with a gift voucher.

5.2.3 Early life information

At age six weeks quantitative survey covering questions about sociodemographic, cultural, environmental, child development (such as type of feeding), family and household dynamics, childcare, lifestyle, and health issues were asked of children mothers (Paterson et al., 2006). With the mothers' consent, infants' measures of birth weight for gestational age were obtained from Middlemore's Hospital Discharge Summary records (Paterson et al., 2006).

5.2.4 Physical measurements at age 14 years

A group of three trained assessors conducted the physical measurements of consented 14-year-old children during visits to each secondary schools (Rush, Oliver, et al., 2016). A protocol was followed to record the measurements (Appendix B).

5.2.5 Measurement of Glycated Haemoglobin A_1c (HbA_1c)

The concentration of HbA_1c was measured with the Afinion™ AS100 Analyser (AXIS-Shield PoC, Oslo, Norway), using a non-fasting finger prick blood sample of 1.5 µL. The blood sample was drawn into the HbA_1c cuvette, inserted into analyser and the value recorded in $mmol.mol^{-1}$ by a study staff member. The author was responsible for controlling the performance of the assay and the analyser.

This study used the cut-off points recommended by the New Zealand Society for the Study of Diabetes Working Party who classify ≤ 40 $mmol.mol^{-1}$ (5.8%) as normal glucose tolerance, values of 41– 49 $mmol.mol^{-1}$ (5.9– 6.6%) as pre-diabetes or dysglycaemia, and values of ≥ 50 $mmol.mol^{-1}$ (6.7%) as a diagnosis of diabetes (Braatvedt et al., 2012).

5.2.6 Anthropometry measurements

Body size was measured using height, weight, and waist circumference (measurement at narrowest point between lower rib and hip). All measurements were repeated twice and ideally, three readings needed to be recorded if the difference in measures was more than 0.5 cm (height and waist circumference) or 0.1 kg (weight). Then, the average of the two closest measures for the height, weight, waist circumference, and bioelectrical impedance analysis measures were used for analysis.

A portable stadiometer (Seca 213 Hamburg, Germany) and an electronic scale (Tanita BC545, Tokyo) were used to measure height and weight respectively. Waist circumference measurements were made using a non-stretchable tape. BMI was calculated by using the following metric formula:

BMI = (weight in kilograms) divided by (height in meters x height in meters)

The Centres for Disease Control (CDC) z scores (Kuczmarski et al., 2000) were derived from BMI, age and gender. For determining the prevalence of obesity and overweight Cole and Lobstein (2012) reference cut-off values were used as suggested by the Ministry of Health on children (Ministry of Health, 2016c).

5.2.7 Body composition measurements

Body composition of the children was measured using Bioimpedance Analysis (ImpediMed Single Frequency 50 kHz Bioimpedance Analyser, Imp-DF50; Impedimed, Brisbane, Queensland, Australia). The assessor followed the instruction (Appendix C) to measure the children in lying position and on electrodes were connected to the children's right hand and food.

Fat free mass (FFM), fat mass (FM) and percentage fat mass (%FM) were calculated applying the prediction equation validated with Pacific, Māori and European children and the same bioimpedance device (Rush, Puniani, Valencia, Davies, & Plank, 2003). The applied equations are:

FFM (kg) = 0.622 height $(cm)^2$/ resistance + 0.234 weight (kg) + 1.166

FM = weight - FFM

%FM = as 100 x FM/ weight

5.2.8 Food information

Food information was collected by using a validated questionnaire (Rush, Oliver, et al., 2016), which was derived from the NZ Nutrition Survey – dietary habits questionnaire (University of Otago & Ministry of Health, 2011a) (Appendix D). The questions were answered by the children.

A series of questions on dietary habits associated with diet quality and/or nutritional status were asked in the dietary habits questionnaire (University of Otago & Ministry of Health, 2011a). The questionnaire focused on key eating patterns or habits, including the consumption of selected foods such as red meat and food groups such as fruit, servings of some foods, such as vegetables, and breakfast consumption. There were two types of questions about consumed foods: 1) the frequency of consumption such as "how often do you eat red meat?" and 2) the number of consumed servings like "how many servings of fruit- fresh, frozen, canned or stewed do you eat per day?"

5.2.9 Statistical analysis

Food questionnaire, HbA_1c, body measurements, birth and early life information of the children including birth weight, gender, type of feeding during the first 6-week, number of siblings and maternal information (mother's age at conception and maternal education) were entered to a SPSS 24 spreadsheet (SPSS Inc., New York, USA). These variables were analysed for significant differences between groups, based on the groupings below. The significance level was set at 0.05 (5%).

Continuous variables (i.e., food information, HbA_1c, body measurements, birth weight, number of siblings and mother's age at conception) were explored for descriptive data and distribution. Normally distributed variables (just height) are reported as mean ± SD, while non-normally distributed are reported as median with Interquartile range (IQR). To test the differences among derived eating patterns (continuous measures), one-way analysis of variance (ANOVA) was used for normally distributed data and Kruskal-Wallis for non-normally distributed data. Post hoc test comparisons using Tukey HSD were used to determine which groups were significantly different and influenced the test statistic. Pearson's correlation test was applied to evaluate the association between normally distributed data and Spearman correlation test for non-normally distributed data.

Categorical variables (i.e., gender, type of feeding during the first 6-week and maternal education) are reported as frequency and percentage. Chi-square test was applied to determine the difference in proportions between derived eating patterns.

To determine the associations, Chi-square and ANOVA tests were used to determine the differences across quartiles for quantitative and categorical variables, respectively where appropriate. Multiple linear regression analysis (stepwise) was performed for the associations between eating patterns, HbA_1c, body measurements and early life factors.

Step-by- step analysis of eating patterns and justification

For identifying the eating patterns, the researcher used cluster analysis to identify whether groups of children had distinct eating patterns or not. The cluster analysis used the K-means algorithm, which is the most common method in dietary and food studies, especially when there is a large number of input variables included in the analysis (Moeller et al., 2007; Newby & Tucker, 2004). K-means is a non-hierarchical and iterative method that is designed to create the most distance between clusters (eating patterns). It minimizes the sum of squares of differences between each child and the mean of his/her cluster (Northstone et al., 2013). In this algorithm, clusters were derived based on the mean intakes, or centroids of the eaten food item entered (Newby & Tucker, 2004). However, there is a need to pre-specify the number of patterns (clusters) in the analysis (Chapter 3). To select the optimal cluster solution as well as considering the interpretability of the clusters, a scree plot was examined (Cattell, 1966; Devlin et al., 2012; Michels & Schulze, 2005; Newby & Tucker, 2004), the number of clusters was determined, and then statistical tests were applied (Moeller et al., 2007).

From the total number of children (n = 931), 65 children (7% of the total number; girls = 36, boys = 29) were identified as outliers of the 15 food/food groups (highest value = 1 and lowest value = 0) in the analysis. While it was recognised that FA can be sensitive to outliers (Pallant, 2016, p. 188), examination of the outliers identified there were children who had a daily (value = 1) or never (value = 0) consumption of a specific food. From a pragmatic nutrition perspective, it was important to consider these children in the analysis, so they were not excluded.

Questions about food consumption were approached in two ways: either the frequency of consumption (FFQ) or the number of servings (dietary habits) consumed of the food/ food group in a period of time required. Both of these reported measures of food consumption were converted into common estimate of consumption as illustrated below.

The frequency of consumption (frequency or serves) was multiplied by the fraction per day (monthly, weekly or daily) (Table 5.2), so that derived values represented the daily rate of consumption.

Table 5.2. Frequency of consumption of food and the applied weighting factor to standardise to a daily rate

Frequency of consumption	Weighting factor /day
Never or not applicable or I don't know	0
less than once a week	2/30
1-2 times a week	1.5/7
3-4- times a week	3.5/7
5-6 times a week	5.5/7
7 or more times per week	1

In match this, the food/food groups that had been reported based on number of servings were converted to a daily rate (Table 5.3).

Table 5.3. Number of servings of consumption of food per day and the applied weighting factor to standardise to a daily rate

Number of servings per day	Weighting factor /day
Never or not applicable or I don't know	0
Less than one serving per day	0.5
1 serving	1
2 servings	2
3 servings	3
4 or more servings	4

Missing data treatment in cluster analysis

Cluster analysis is a multivariable statistical technique and by using different algorithms, groups can be identified. Like other multivariable statistics, most cluster algorithms ignore all of the data for cases with any missing values, because generally clustering algorithms have no internal way to deal with missing values (Wagstaff, 2004). There are two common solutions to treat the missing data: firstly, filling in the values (imputation), and secondly, ignore the missing data (marginalization) (Wagstaff, 2004). In this study the missing data were ignored and actual observed data were tested. This was to have more reliable than filled-in values (Wagstaff, 2004). There were 191 children (93 girls and 98 boys), who were excluded from analysis of eating patterns analysis due to large blocks of missing responses to the dietary questions. The weight (kg), height (cm), waist circumference (cm), FM (kg), FFM (kg), body FM (%) and

HbA_1c ($mmol.mol^{-1}$) of the excluded children (n = 191) were not statistically different from those included children (370 girls, 370 boys) (*p* values were 0.192, 0.246, 0.088, 0.093, 0.574, 0.083 and 0.127, respectively).

5.3 Results

5.3.1 Maternal characteristics, birth and measurements of children at birth and 14 years

There was no significant difference between girls' and boys' mothers' age at conception (Table 5.4). The minimum girls' mothers' age was 14 years and the youngest boys' mothers were 15 years. However, the highest boys' mothers' age (44 years) was13 years younger than girls' mothers' age (57 years). In terms of schooling, more boys' mothers (38%) had no formal education in comparison with girls' mothers (34%), but this was not significantly different (Table 5.4). Thirty percent of both girls and boys had no biological siblings at the time of birth (Table 5.4). Boys were born slightly heavier (median 3658 g) than girls (3528) (U= 95005.0, Z = -2.784, p = 0.005) (Table 5.4). Forty-nine children (girls = 30, boys = 19) were born below 2500g at birth. Half of the girls (51%) and boys (50%) were breastfed during the first 6 weeks of life (Table 5.4).

Table 5.4. Characteristics of Pacific children at birth and maternal baseline information

	Girls (n = 463)		Boys (n = 468)		*p* Value
Birth weight (g) [a]	3514.1 (± 651.5)		3633.7 (± 592.3)		**0.005**
Infant feeding 6 weeks postpartum [b]					0.358
Breast feeding	235	(51%)	232	(50%)	
Bottle feeding	65	(14%)	54	(12%)	
Both breast and bottle	163	(35%)	182	(39%)	
Mother's age (years) [c]	28.42 (± 6.59)		28.03 (± 5.96)		0.351
Mothers' education [b]					0.208
No formal qualifications	156	(34%)	178	(38%)	
Secondary school qualification	179	(39%)	156	(33%)	
Post school qualification	128	(27%)	134	(29%)	
Number of biological siblings [b]					0.513
None	137	(30%)	143	(31%)	
1	121	(26%)	117	(25%)	
2	84	(18%)	100	(21%)	
3+	121	(26%)	108	(23%)	

Data expressed as mean ± SD or n (%). [a] Mann-Whitney U test. [b] Chi square test.
[c] Independent t test.
Birth weight was missing for 3 girls and 6 boys.

Boys weighed more and were taller than girls; they also had proportionally more FFM and less FM than girls (Table 5.5). There was no difference between mean of girls' HbA_1c (36.26 ± 5.10 $mmol.mol^{-1}$) and boys (35.95 ± 2.87 $mmol.mol^{-1}$) (Figure 5.2). However, there were 32 children (by gender: 15 girls and 17 boys, BMI cut-offs: normal weight = 6, overweight = 6, obese = 19 and missing = 1) whose HbA_1c was ≥ 41 $mmol.mol^{-1}$ and a letter was sent to their parents. Notably, four of these children were previously diagnosed with diabetes (T1DM = 1, T2DM = 3).

Table 5.5. Physical measurements of Pacific children at age 14 years from the PIF main study

	Girls (n = 463)		Boys (n = 468)		
	Mean	**SD**	**Mean**	**SD**	***p* Value** [a]
Age (years)	14.30	0.41	14.27	0.43	0.132
HbA_1c ($mmol.mol^{-1}$) [b]	36.26	5.10	35.95	2.87	0.763
Anthropometry [c]					
Weight (kg)	78.24	19.32	81.93	23.42	**0.039**
Height (cm)	165.07	6.10	171.60	7.56	**0.001**
BMI ($kg.m^{-2}$)	28.61	6.38	27.61	6.87	**0.005**
Weight z scores [d]	1.66	0.77	1.82	1.11	**0.012**
Height z scores [d]	0.64	0.93	0.79	0.94	**0.004**
BMI z scores [d]	1.55	0.71	1.50	0.91	0.937
Waist (cm)	80.66	12.55	87.03	16.13	**0.001**
Waist/ height (cm)	0.48	0.07	0.51	0.09	**0.013**
Body composition [e]					
Fat-free mass (kg)	50.57	9.32	58.86	11.73	**0.001**
Fat mass (kg)	27.83	11.08	23.02	13.43	**0.001**
Fat mass%	34.44	5.86	26.23	8.56	**0.001**

[a] Mann-Whitney U test. Independent t test for height [b] HbA_1c measures missing for 85 girls and 78 boys. [c] Anthropometry measurements of 10 girls and 6 boys were missed. [d] CDC growth charts (Kuczmarski et al., 2000) [e] Single frequency hand to foot 50 kHz Bioelectrical Impedance Analysis (BIA). There were 85 girls and 16 boys who did not have bio impedance measurements.

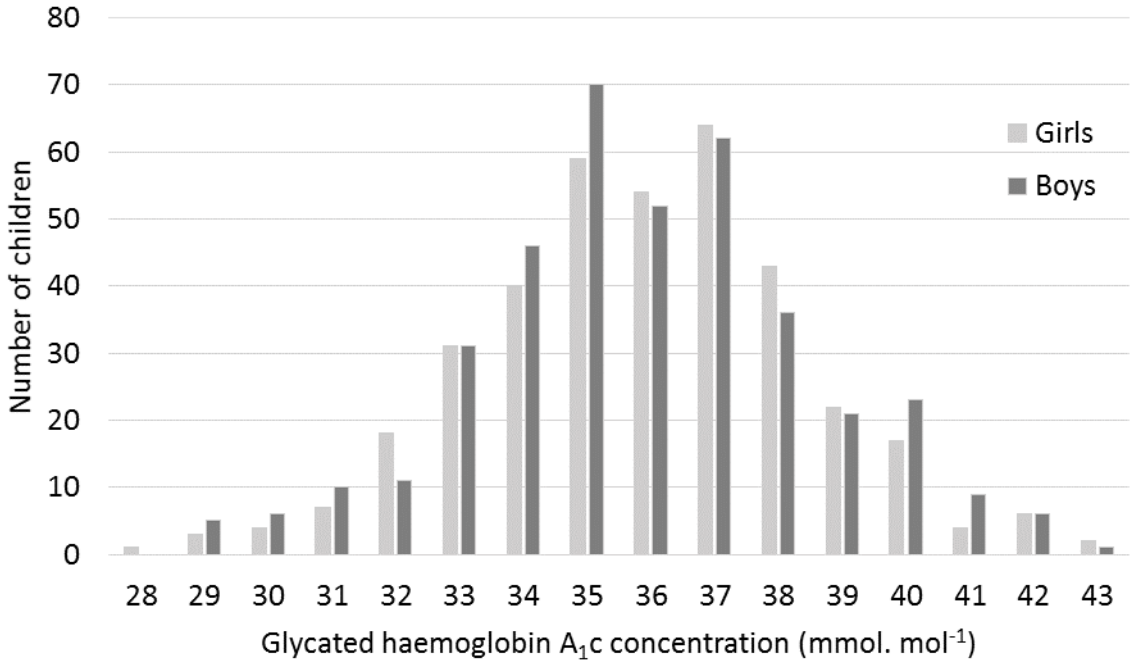


Figure 5.2. Frequency of glycated haemoglobin A_1c by gender among Pacific children at 14 years old Four children with glycated haemoglobin A_1c> 50 mmol.mol-^{1} were excluded from this figure.

Almost two thirds of the children were overweight or obese (Table 5.6) and boys were more likely than girls to not be overweight or obese (χ^2 12.70, p = 0.005). There were more overweight girls than overweight boys (χ^2 8.91, p = 0.002), but there was no significant difference among obese children by gender.

Table 5.6. Pacific children classified as thin, normal, overweight and obese based on international obesity task force cut-offs [a]

	Girls (n = 455) n (%)		**Boys (n = 461) n (%)**		**Total (n = 916) n (%)[b]**	
Thin	0	(0)	4	(1)	4	(0.4)
Normal	98	(22)	130	(28)	228	(25)
Overweight [c]	164	(36)[c]	129	(28)	293	(32)
Obese	193	(42)	198	(43)	391	(43)

[a] Using Cole and Lobstein cut-offs (2012). [b] body mass index measurements were missed for eight girls and seven boys. [c] girls > boys χ^2 8.91, p = 0.002

5.3.2 Associations between HbA_1c and body measurements at 14 years

Data were explored by correlation for the relationships between HbA_1c at 14 years both for gender and then between HbA_1c and successive body measurements.

There was no significant correlation between HbA_1c and height z score (r=-0.020 p = 0.586) or weight z score (r = 0.062 p = 0.082), but there was a positive correlation of

HbA_1c with BMI z score (r = 0.076 *p*= 0.035). When adjusted for gender and age HbA_1c was positively related to waist measurement (r = 0.101 *p* = 0.006), waist-to-height ratio (r = 0.109 *p = 0.003*), FM (r = 0.088 *p* = 0.016), but not FFM (r = 0.065 *p* = 0.075).

5.3.3 Associations between HbA_1c, gender and early life information

There was no significant correlation between HbA_1c with gender, birth weight (adjusted for gender), type of first 6-week feeding and number of siblings. In addition, there was no significant correlation (*p* = 0.121) between HbA_1c and low birth weight (<2500 g) children when controlled for gender. There was a small positive and statistically significant relationship between HbA_1c and mothers' age (r = 0.075 *p* = 0.037). In addition, there was a significant difference between HbA_1c and mothers' education (*p* = 0.018) and children of mothers without formal qualification had 1 $mmol.mol^{-1}$ higher HbA_1c (mean HbA_1c = 36.67 $mmol.mol^{-1}$) compared to children whose mothers had secondary school (mean HbA_1c = 35.84 $mmol.mol^{-1}$) and post school qualifications (mean HbA_1c = 35.76 $mmol.mol^{-1}$).

5.3.4 Foods consumption reported by the children at 14 years

This section reports the patterns of consumption visually observed in the analysis rather than formally testing all the observations. As such it helps identify the frequently consumed and less frequently consumed foods for future work and is descriptive. Statistical differences were not shown as with so many tests there would be Type 1 errors.

Red meat, chicken, and processed meat products were consumed at least 1-2 times per week (girls 72%, 85.5% and 72.6%, boys 79%, 86.1% and 83.5% respectively) (Table 5.7). Overall eleven children (5 girls and 6 boys) were vegetarian. However, one third of both boys and girls never consumed fresh/frozen fish or shellfish, battered or fried fish or shellfish, and canned fish or shellfish (Table 5.7).

Two thirds of both girls and boys ate at least 1-2 times per week from each of the five high energy, fat and sugar food groups: 1) hot chips, french fries, wedges, or kumara chips, 2) fast food/ takeaways, 3) fruit juices and fruit drinks, 4) soft drinks or energy drinks and 5) lollies, sweets, chocolate, and confectionary (Table 5.7).

The top three food/food groups that girls (n = 463) never consumed were fresh/frozen fish or shellfish (n = 202), canned fish or shellfish (n = 191), and battered or fried

fish/shellfish (n = 180). Furthermore, the top three food/food groups that were consumed 7 ≥ per day were lollies, sweets, chocolate, and confectionary (n = 37), fruit juices and fruit drinks (n = 23), and soft drinks/ energy drinks (n = 23) (Table 5.7).

Both girls and boys consumed fruit more frequently than vegetables (Table 5.8); 16% and 23% of children ate fruit and vegetables less than once per day. One-fifth of the children consumed milk less than once per day. More girls than boys (19% vs 14%) consumed bread less than once a day. Twenty-five percent of boys and 16% of girls consumed bread five or more times a day (Table 5.8).

Table 5.7. Eleven dietary habits of 931 Pacific children at 14 years by gender (girls n = 463, boys n = 468)

Frequency	Never [a] n (%)		<1 per week n (%)		1 - 2 per week n (%)		3 - 4 per week n (%)		5 - 6 per week n (%)		7 ≥ per week n (%)		Missing n (%)	
	Girls	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**
Red meat	79 (18.1)	71 (15.2)	47 (10.2)	27 (5.8)	132 (28.5)	134 (28.6)	118 (25.5)	127 (27.1)	37 (8.0)	50 (10.7)	18 (3.9)	27 (5.8)	32 (6.9)	32 (6.8)
Chicken	35 (7.6)	34 (9.4)	32 (6.9)	21 (4.5)	172 (37.1)	170 (36.3)	143 (30.9)	150 (32.1)	35 (7.6)	38 (8.1)	11 (2.4)	17 (3.6)	35 (7.6)	28 (6.0)
Processed meat products	89 (19.2)	51 (10.9)	38 (8.2)	26 (5.6)	168 (36.3)	178 (38.0)	95 (20.5)	127 (27.1)	32 (6.9)	39 (8.3)	8 (1.7)	17 (3.6)	33 (7.1)	30 (6.4)
Fresh/ frozen fish/ shellfish	202 (43.7)	176 (37.6)	99 (21.4)	104 (22.2)	92 (19.9)	96 (20.5)	22 (4.8)	36 (7.7)	6 (1.3)	12 (2.6)	3 (0.6)	5 (1.1)	39 (8.4)	39 (8.3)
Battered/ fried fish/ shellfish	180 (38.9)	169 (36.1)	98 (21.2)	81 (17.3)	109 (23.5)	132 (28.2)	29 (6.3)	36 (7.7)	4 (0.9)	10 (2.1)	2 (0.4)	0 (0.0)	41 (8.9)	40 (8.5)
Canned fish/ shellfish	191 (41.3)	176 (37.6)	98 (21.2)	109 (23.3)	101 (21.8)	96 (20.5)	24 (5.2)	37 (7.9)	8 (1.7)	9 (1.9)	1 (0.2)	3 (0.6)	40 (8.6)	38 (8.1)
Hot chips, french fries, wedges/ kumara chips	30 (6.4)	29 (6.2)	111 (24.0)	101 (21.6)	180 (38.9)	197 (42.1)	91 (19.7)	102 (21.8)	30 (6.5)	18 (3.8)	16 (3.5)	16 (3.4)	5 (1.1)	5 (1.1)

Frequency	Never [a] n (%)		<1 per week n (%)		1 - 2 per week n (%)		3 - 4 per week n (%)		5 - 6 per week n (%)		7 ≥ per week n (%)		Missing n (%)	
	Girls	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**
Fast food/ takeaways	27 (5.8)	31 (6.6)	129 (27.9)	141 (30.1)	196 (42.3)	186 (39.7)	70 (15.1)	83 (17.7)	28 (6.0)	17 (3.6)	9 (1.9)	7 (1.5)	4 (0.9)	3 (0.6)
Fruit juices and fruit drinks	40 (8.7)	54 (11.5)	107 (23.0)	84 (17.9)	156 (33.7)	160 (34.2)	97 (21.0)	110 (23.5)	33 (7.1)	35 (7.5)	23 (5.0)	19 (4.1)	7 (1.5)	6 (1.3)
Soft drinks/ energy drinks	40 (8.7)	54 (11.5)	107 (23.1)	84 (17.9)	156 (33.7)	160 (34.2)	97 (21.0)	110 (23.5)	33 (7.1)	35 (7.5)	23 (5.0)	19 (4.1)	7 (1.5)	6 (1.3)
Lollies, sweets, chocolate and confectionary	16 (3.5)	42 (9.0)	89 (19.2)	106 (22.6)	127 (27.4)	176 (37.6)	143 (30.9)	99 (21.2)	48 (10.4)	21 (4.5)	37 (8.0)	20 (4.3)	3 (0.6)	4 (0.9)

[a] The response including “I don’t know” and “not applicable” given for the frequency of consumption of red meat, chicken, processed meat products, fresh/frozen fish/ shellfish, battered/fried fish/shellfish, canned fish/ shellfish have been considered as ‘never’ for the analysis. The applied dietary habit questionnaire is based on Ministry of health dietary habit questionnaire used at 2008/09 New Zealand adult nutrition survey (University of Otago & Ministry of Health, 2011a).

Table 5.8. Daily frequency of consumption of fruit, vegetables, milk and bread of 931 Pacific children at 14 years by gender (girls n = 463, boys n = 468)

Servings	**Never [a] n (%)**		**<1 per day n (%)**		**1 - 2 per day n (%)**		**3 - 4 per day n (%)**		**5 - 6 per day n (%)**		**7 ≥ per day n (%)**		**Missing n (%)**	
	Girls	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**
Fruit (fresh/ frozen/ canned/ stewed)	40 (8.6)	35 (7.4)	33 (7.1)	47 (10.0)	72 (15.6)	83 (17.7)	144 (31.1)	134 (28.6)	96 (20.7)	87 (18.6)	74 (16.0)	76 (16.2)	4 (0.9)	6 (1.3)
Vegetables (fresh/ frozen/ canned)	55 (11.9)	66 (14.1)	56 (12.1)	40 (8.5)	92 (19.9)	87 (18.6)	110 (23.8)	120 (25.6)	88 (19.0)	97 (20.7)	57 (12.3)	53 (11.3)	5 (1.1)	5 (1.1)
Milk	33 (7.1)	43 (9.2)	62 (13.4)	48 (10.3)	143 (30.9)	110 (23.5)	123 (26.6)	111 (23.7)	57 (12.3)	87 (18.6)	42 (9.1)	62 (13.2)	3 (0.6)	7 (1.5)
Bread/ toast/ bread rolls	40 (8.7)	37 (7.9)	42 (9.1)	28 (6)	151 (32.6)	122 (26.1)	154 (33.3)	160 (34.2)	51 (11)	68 (14.5)	24 (5.2)	50 (10.7)	1 (0.2)	3 (0.6)

[a] The response including “I don’t know” and “not applicable” given for the frequency of consumption of fruit, vegetables, milk and bread/toast/bread rolls have been considered as ‘never’ for the analysis. The applied dietary habit questionnaire is based on Ministry of health dietary habit questionnaire used at 2008/09 New Zealand adult nutrition survey (University of Otago & Ministry of Health, 2011a).

Table 5.9. Daily frequency of consumed foods by gender. Ranked by descending frequency of consumption

	Daily frequency [a] Median (percentile 25th, 75th)			
Food/ Food group	**Total n = 740**	**Girls n = 370**	**Boys n = 370**	***p* value** [b]
Fruit and vegetables	4.00 (2.00, 5.00)	4.00 (2.13, 50)	4.00 (2.00, 5.00)	0.861
Fruit (fresh/ frozen/ canned/ stewed)	2.00 (1.00, 3.00)	2.00 (1.00, 3.00)	2.00 (1.00, 3.00)	0.423
Vegetables (fresh/ frozen/ canned)	2.00 (1.00, 3.00)	2.00 (1.00, 3.00)	2.00 (1.00, 3.00)	0.778
Milk	2.00 (1.00, 3.00)	1.00 (1.00, 2.00)	2.00 (1.00, 3.00)	**0.008**
Bread/ toast/ bread rolls	2.00 (1.00, 2.00)	1.00 (1.00, 2.00)	2.00 (1.00, 3.00)	**0.001**
Chicken	0.21 (0.21, 0.50)	0.21 (0.21, 0.50)	0.21 (0.21, 0.50)	0.453
Processed meat products	0.21 (0.21, 0.50)	0.21 (0.07, 0.50)	0.21 (0.21, 0.50)	**0.001**
Red meat	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.21 (0.21, 0.50)	**0.015**
Hot chips, French fries, wedges/ kumara chips	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.831
Fruit juices and fruit drinks	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.741
Soft drinks/ energy drinks	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.299
Lollies, sweets, chocolate and confectionary	0.21 (0.07, 0.50)	0.21 (0.21, 0.50)	0.21 (0.07, 0.50)	**0.001**
Fast food/ takeaways	0.21 (0.07, 0.21)	0.21 (0.07, 0.21)	0.21 (0.07, 0.21)	0.371
Fresh/ frozen fish/ shellfish	0.07 (0.00, 0.21)	0.07 (0.00, 0.21)	0.07 (0.00, 0.21)	**0.019**
Battered/ fried fish/ shellfish	0.07 (0.00, 0.21)	0.07 (0.00, 0.21)	0.07 (0.00, 0.21)	0.078
Canned fish/ shellfish	0.07 (0.00, 0.21)	0.07 (0.00, 0.21)	0.07 (0.00, 0.21)	0.184

[a] Frequency of consumption was assumed as same as the serving size

[b] Mann-Whitney U test.

Boys more frequently consumed red meat (p = 0.015), processed meat products (p = 0.001), fresh/ frozen fish/shellfish (p = 0.019), milk (p = 0.008), bread/ toast/ bread rolls (p = 0.001) compared to girls (Table 5.9). However, they ate less lollies, sweets, chocolate and confectionary (p = 0.001) (Table 5.9).

The pattern of consumption of types of bread was overall different by gender (χ^2 12.00, p = 0.001) (Figure 5.3). For both girls and boys, the most commonly consumed bread was white (girls 57.7%, boys 57.1%). Girls consumed more fibre (χ^2 37.67, p< 0.0001) and light grain (e.g., Molenberg, Freya's, Ploughmans, MacKenzie High Country) (χ^2 7.60, p = 0.006) bread than boys. The frequency of eating heavier grain breads (e.g., Vogels and Burgen) was slightly higher in girls (7.1%) compared to boys (4.9%), but there was no significant difference.

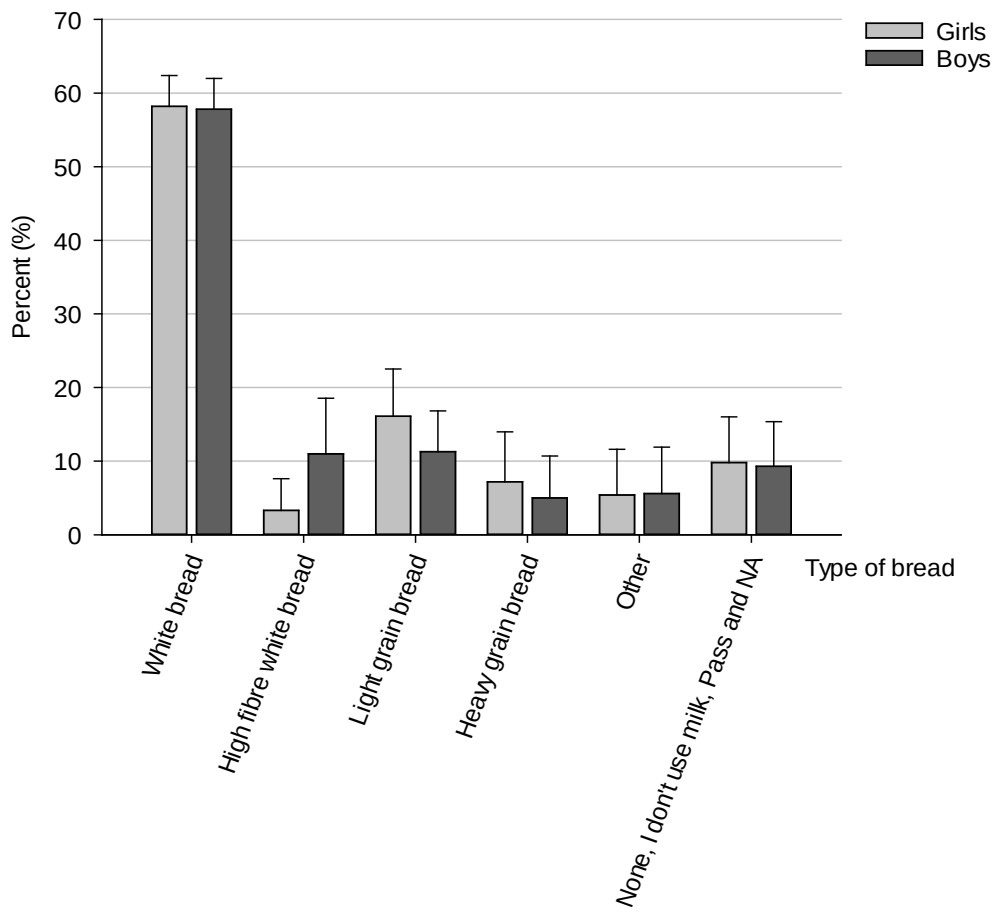


Figure 5.3. Type of bread chosen most of the time by gender

The two most common types of consumed milk were whole or standard milk (dark blue or silver) and reduced fat (light blue) by both girls (66.1%, 18.4% respectively) and boys (61.8%, 17.9%). Boys (13.7%) were less likely to consume milk compared to girls (9.9%). However, there was no significant difference between girls and boys in the type of consumed milk (χ^2 12.00, p = 0.336) (Figure 5.4). Consumption of milk alternatives (i.e., soymilk and other type of milk) were the least type of milk reported by both girls and boys (Figure 5.4).

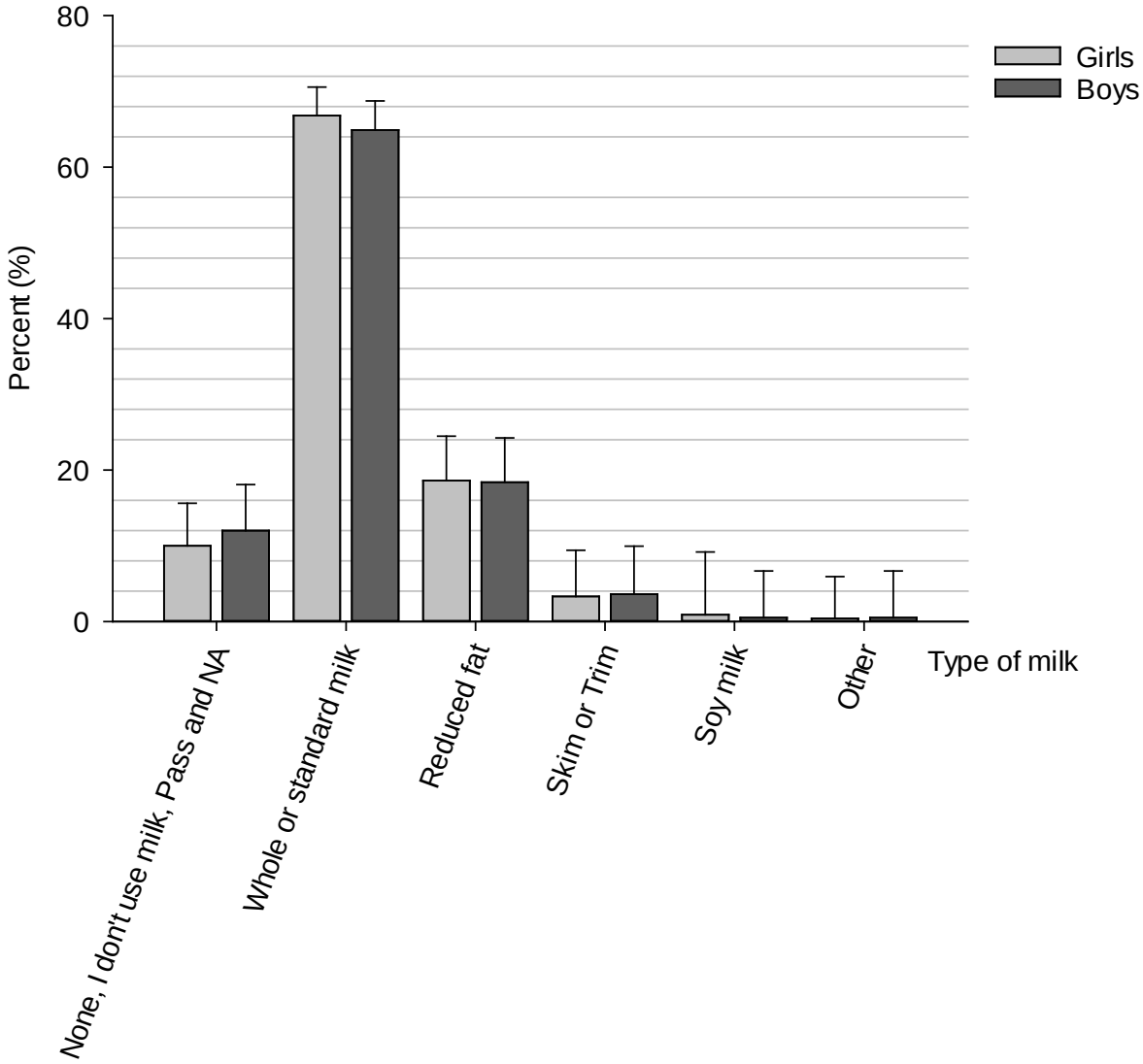


Figure 5.4. Type of milk chosen most of the time by gender

5.3.5 Identification of eating patterns

The frequency of consuming 15 specific food items or food categories (hereafter called "foods") were examined using SPSS factor analysis with Varimax rotation loading, Kaiser Normalization and scree plots (Figure 5.5). The optimal number of four eating patterns (clusters) with a factor loading greater than 0.3 (Table 5.10) were identified. The greater the factor loading for a food, the greater the effect of that food on a specific eating pattern. For example, in eating pattern one, fast food/ takeaways (factor loading = 0.769) had a much higher and positive load than the positive loading of fruit juices and fruit drinks (factor loading 0.493).

Subjective names for each of the four eating patterns were nominally assigned by the researcher to reflect the consumed foods in each cluster. Together the four clusters explained 54% of the total variance in food frequency. The first cluster was called 'occasional' as most of the foods were processed and not wholesome and the second was called 'seafood' as fish and seafood with different styles of cooking were consumed. The third was foods from three of the recommended food groups – fruit and vegetables, and milk and named 'basic and staples'. The fourth because it included red meat, chicken, processed meat products and bread/ toast/ bread rolls was named 'meat and bread'.

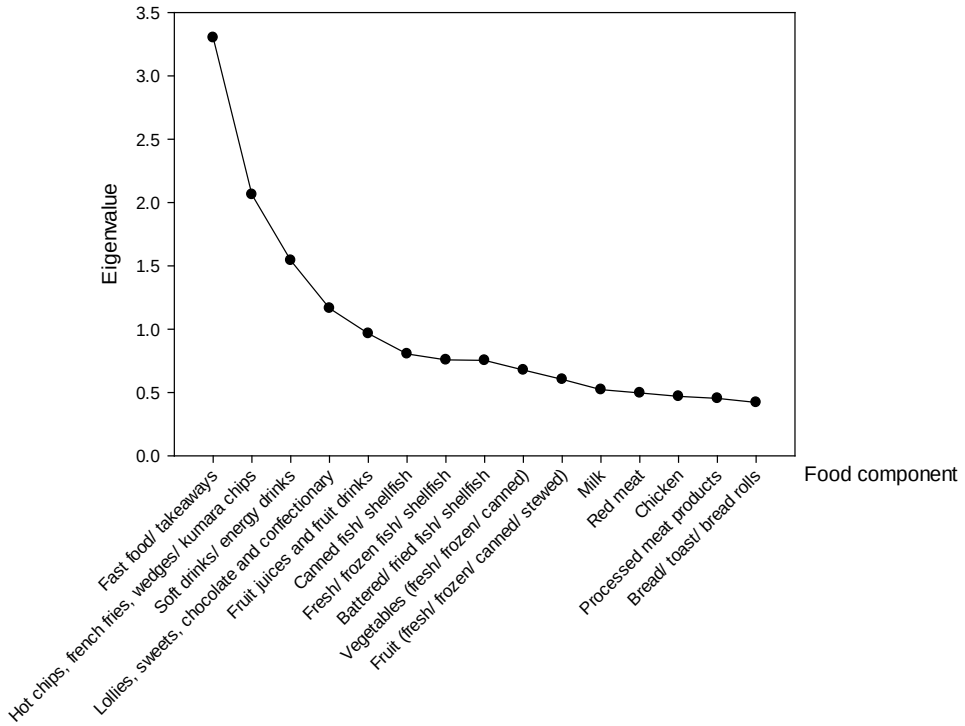


Figure 5.5. Scree plots of eigen values of food components

Table 5.10. Factor loading matrix derived from food or food groups for 4 major eating patterns [a]

Food category	**Eating pattern 1**	**Eating pattern 2**	**Eating pattern 3**	**Eating pattern 4**
Fast food/ takeaways	**0.769**			
Hot chips, French fries, wedges/ kumara chips	**0.733**			
Soft drinks/ energy drinks	**0.732**			
Lollies, sweets, chocolate and confectionary	**0.702**			
Fruit juices and fruit drinks	**0.493**		0.348	
Canned fish/ shellfish		**0.822**		
Fresh/ frozen fish/ shellfish		**0.804**		
Battered/ fried fish/ shellfish		**0.777**		
Vegetables (fresh/ frozen/ canned)			**0.800**	
Fruit (fresh/ frozen/ canned/ stewed)			**0.794**	
Milk			**0.556**	
Red meat				**0.680**
Chicken				**0.680**
Processed meat products				**0.643**
Bread/ toast/ bread rolls				**0.500**
Eigen value	3.30	2.06	1.54	1.16
Variance explained %	22.0	13.7	10.3	7.7
Name of eating pattern	Occasional	Seafood	Basic and staples	Meat and bread

[a] Absolute values <0.30 were excluded from the table for simplicity.

There were more girls than boys within the seafood and basic and staples patterns and less in the occasional and meat and bread patterns (χ^2 8.9, p = 0.03) compared to the boys (Table 5.11). Boys (57%) were more likely to be present in the ‘meat and bread’ pattern compared to the girls (43%) (χ^2 4.3, p = 0.04) (Table 5.11).

Table 5.11. Gender distribution by eating patterns

Gender [a]	**Occasional n (%)**	**Seafood n (%)**	**Basic and staples n (%)**	**Meat and bread n (%)**
Girls (n = 370)	64 (17)	106 (29)	128 (35)	72 (19)
Boys (n= 370)	80 (22)	89 (24)	105 (28)	96 (26)

There were 93 girls and 98 boys with missing data who were excluded from the eating pattern analysis.
[a] Chi-square test (χ^2 8.9, p = 0.03).

5.3.6 Associations between eating patterns and HbA_1c

There was no difference revealed among eating patterns by HbA_1c. In addition, the correlations of frequency of consumption of individual foods with HbA_1c were tested (total and by gender). A small positive correlation between daily consumption of chicken and HbA_1c was found among boys (r = 0.125 p = 0.017).

Table 5.12. Point of care HbA_1c at 14 years by eating patterns

		Occasional n, mean (SD)[a]	**Seafood n, mean (SD)**	**Basic and staples n, mean (SD)**	**Meat and bread n, mean (SD)**
HbA_1c	Girls (n=297)[b]	55, 36.2 (2.1)	86, 36.7 (6.4)	96, 36.4 (7.3)	60, 35.9 (2.7)
	Boys (n=307)[b]	67, 35.7 (2.6)	74, 36.1 (2.7)	88, 35.8 (3.6)	78, 36.0 (2.7)
	Total (n=604)[b]	122, 35.9 (2.4)	160, 36.4 (5.0)	184, 36.1 (5.8)	138, 36.0 (2.7)

[a] Standard deviation. [b] One-way ANOVA (p> 0.7)

5.3.7 Associations between eating patterns and body measurements at age 14 years

Data were explored visually and by correlation for the relationships between derived eating patterns and body measurements including weight (kg), height (cm), waist (cm), waist-to-height ratio (cm), fat free mass (kg), fat mass (kg), fat mass (%) and BMI at 14 years. One-way ANOVA was used to explore the differences between height and eating patterns of the children, and there was a significant difference at the F (3, 724) = 2.701, p = 0.045. Despite reaching statistical significance, the actual difference in mean scores between the groups was small (≈ 2cm) (Figure 5.6). The calculated effect size using eta squared, was 0.01. Post-hoc comparisons using Tukey HSD test indicated that the mean score for ‘occasional’ (mean = 169.4, SD = 8.3 cm) was significantly greater than ‘seafood’ (mean = 167.2, SD = 7.1 cm). The other two eating patterns, ‘basic and

staples' and 'meat and bread' did not differ significantly from either 'occasional' pattern or 'seafood'. After applying Kruskal-Wallis test, no significant differences in other body measurements of the children including weight (kg) (p = 0.592), waist circumference (cm) (p = 0.538), waist-to-height ratio (cm) (p = 0.648), BMI (p = 0.842), fat free mass (g) (p = 0.193), fat mass (kg) (p = 0.898), fat mass (%) (p = 0.740), were seen by eating patterns.

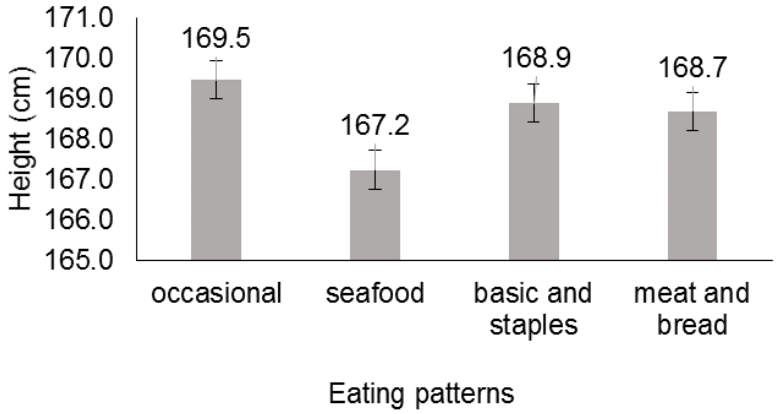


Figure 5.6. Height of the children at 14 years by eating patterns

5.3.8 Associations between eating patterns and early life information

A Kruskal-Wallis test revealed no statistical significance difference in birth weight of the children across the four eating patterns (p = 0.333) (Table 5.13). There was no significant difference (p = 0.295) between types of feeding during the first 6-week of life and eating patterns among the children. However, the majority of children (30%, n = 116), who were breastfed (n = 386), had 'basic and staples' at 14 years and the least number of them had 'occasional' pattern (19%, n = 73).

There were no statistically significant relationships derived between either mothers' age or education, and children's eating patterns (p = 0.762). Moreover, there was no statistically significant difference between the number of biological siblings and derived eating patterns (p = 0.324) (Table 5.13).

5.3.9 Regression analysis

The influence of gender, central fat (waist-to-height ratio), BMI z scores and mothers' education was explored by stepwise regression. Only waist-to-height ratio and mothers' education were positively (standardised β 0.108 p = 0.03) and negatively (standardised

β -0.091 p = 0.012) related to HbA_1c (R^2 0.020) respectively. However, no association was found with the eating patterns.

Table 5.13. Distribution of the children based on eating patterns and early life information

	Occasional n = 144 (19%)	**Seafood** n = 195 (26%)	**Basic and staples** n = 233 (32%)	**Meat and bread** n = 168 (23%)	***p* value**
Birth weight (kg)	3638.2 (646.6)	3517.6 (627.7)	3586 (594.1)	3611.7 (667.0)	[a] 0.333
Infant feeding 6 weeks postpartum					[b] 0.295
Breast feeding	73 (19%)	102 (26%)	116 (30%)	95 (25%)	
Bottle feeding	13 (15%)	31 (35%)	28 (32%)	17 (19%)	
Both breast and bottle	58 (22%)	62 (23%)	89 (34%)	56 (21%)	
Mother's age at conception (years)	28.16 (5.89)	27.95 (7.11)	27.88 (6.45)	28.34 (5.92)	[c] 0.893
Mothers' education					[b] 0.762
No formal qualifications	45 (17%)	72 (27%)	87 (33%)	59 (22%)	
Secondary school qualification	57 (21%)	71 (26%)	76 (28%)	65 (24%)	
Post school qualification	42 (20%)	52 (25%)	70 (34%)	44 (21%)	
Number of biological siblings					[b] 0.324
None	39 (17%)	63 (27%)	82 (35%)	49 (21%)	
1	34 (18%)	44 (23%)	66 (34%)	49 (25%)	
2	31 (23%)	33 (24%)	40 (29%)	32 (23%)	
3+	40 (23%)	55 (31%)	45 (25%)	38 (21%)	

Birth weight and mother's age presented as mean (SD)
Infant feeding 6 weeks postpartum, mother's education and number of siblings presented as number (%)
[a] Kruskal-Wallis test applied to compare the birth weight by eating patterns
[b] Chi-square test applied to compare type of first 6-week feeding, mothers' education and number of siblings by eating patterns
[c] One-way analysis of variance applied to compare mothers' age at pregnancy by eating patterns

5.3.10 Summary of main findings

Food choices and derived eating patterns

- Majority of the children (76%) consumed insufficient daily vegetables and fruit (> 5 times per day).
- Children were identified with four eating patterns through cluster analysis; occasional, seafood, basic and staples, and meat and bread. More girls than boys were within the seafood and basic and staples patterns and less in the occasional and meat and bread patterns (χ^2 8.9, p = 0.03).

Eating patterns, food consumption and HbA_1c

- The mean of HbA_1c was not different by four derived eating patterns.
- The correlations of individual foods with HbA_1c were tested (total and by gender). There was a small correlation among boys between daily consumption of chicken and HbA_1c (r = 0.125 p = 0.017).

Eating patterns and current body size measurements

- Investigating the correlations between derived eating patterns and body measurements showed that there was a significant difference between height (cm) and derived eating patterns (p = 0.045). In particular, children with occasional pattern were about 2cm taller than children with seafood pattern.

Child early life factors and T2DM

- There was no statistical significance difference in birth weight of the children across the four eating patterns (p = 0.333).
- There was no significant difference (p = 0.295) between types of feeding during the first 6-week and eating patterns among the children. However, the majority of the children (30%, n = 116), who were breastfed (n = 386), had ‘basic and staples’ at 14 years and the least number of them had ‘occasional’ pattern (19%, n = 73).
- There were no significant correlation between HbA_1c and gender, birth weight (adjusted for gender), type of first 6-week feeding, and number of siblings. In addition, there was no significant correlation (p = 0.121) between HbA_1c and low birth children controlled by gender.

Maternal education, age at conception and T2DM in children

- There was a significant association between HbA_1c and mothers' age at conception (r = 0.075 *p* = 0.037) also with mothers' education (*p* = 0.018).
- There were no significant correlations between four derived eating patterns and either of factors collected from early life (
- Table 5.13).

Eating patterns, body measurements, mother's age and HbA_1c

- The results of regression models revealed that only waist-to-height ratio and mothers' age were positively (standardised β 0.108 *p* = 0.03) and negatively (standardised β -0.091 *p* = 0.012) related to HbA_1c R^2 = 0.020, but not the eating patterns.

5.4 Discussion

For the first time, this 2014-2015 investigation provides an account of eating patterns in relation to risk for T2DM for a cohort of Pacific children at the age of fourteen. These children, who were born in a relatively deprived area of NZ (Counties Manukau Health, 2017) had on average a body weight of 80kg and for their age and gender a BMIz score of 1.5. In addition, the children followed a relatively homogenous diet with foods high in sugar and energy.

The findings (Figure 5.7) in relation to other evidence (i.e., local and international, concerning eating patterns and risk of T2DM are discussed. However, at the time of writing there were few studies concerning eating patterns and risk for T2DM identified by HbA1c among healthy children so studies with adults are also included (Table 5.14).

Discussion of the findings is followed by discussion of how findings could be disseminated and translated into positive actions, with particular reference to the contribution that epidemiologists, nutritionists and dieticians can make to improve the patterns of eating and health of children. In addition, strengths, and limitations of the study are declared and recommendations put forward for future research on eating patterns and the risk of developing T2DM in children, based on their ethnicity.

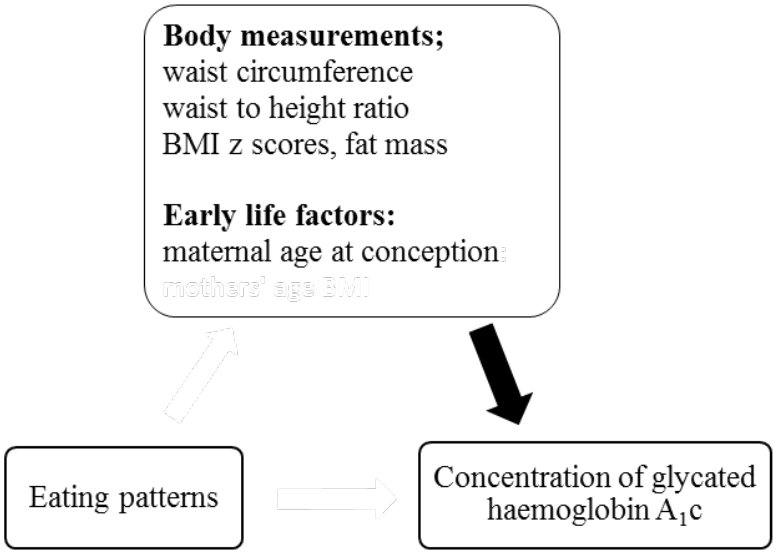


Figure 5.7. Factors found with significant associations

Table 5.14. Characteristics of the current study and articles included in the present discussion

Reference	**Study design**	**Number and age in years of participants (child unless otherwise specified)**		**Dietary assessment method**	**Eating pattern method**	**Risk factors**
Present body of work	Cross-section	931[a],	14	FFQ	CA	Anthropometric and body composition measurements (DEXA), HbA_1c and SEB
Alexy et al. (2004)	Cohort (10 years follow-up)	228,	2-18	3-day weighed dietary record	CA	BMI
Boone-Heinonen (2008)	Prospective Cohort	251,	9	FFQ	CA	PA and SB
Craig (2010)	Cross-section	1233,	5-11, 12-17	FFQ	PCA	SEB, PA and obesity
Howe et al. (2013)	Cross-section	681,	15.8	FFQ	PCA	Body composition (BIA)
Ambrosini et. al (2016)	Cohort	6722,	7, 10 and 13	3-day food diaries	RRR	Adiposity
Aranceta et. al (2003)	Cross-section	3534,	2-24	24-h recall and FFQ	FA	SEB
Schumacher (2014)	Cross-section	332,	13.7	FFQ	Food index	SEB and core foods

Abbreviations: BIA; Bioelectrical Impedance Analysis, BMI, body mass index; CA, cluster analysis; DEXA, dual energy x-ray absorptiometry; FA, factor analysis; FFQ, food frequency questionnaire; HbA_1c, Glycated haemoglobin A_1c ; PCA, principle components analysis; SEB, socioeconomic background; SD, sedentary behaviours, 24-h recall; 24-hour food recall.

[a] There were 93 girls and 98 boys who were excluded from the eating pattern analysis.

5.4.1 Food choices and eating patterns among Pacific children

Overall these children reported a relatively homogeneous diet in that insufficient vegetables (Ministry of Health, 2014b) were consumed and many "occasional" (Ministry of Health, 2007, p. 8), energy dense, nutrient poor foods were consumed daily. However, in the Children's Nutrition Survey (CNS2002) with 3,275 children aged 5 – 14 years measured, the frequency of adequate vegetable intake was higher than fruit; 60% ate vegetables three or more times a day but only 40% ate fruit at least twice a day. In addition, 84% and 71% of Pacific children consumed fizzy drinks and fast food at least once a week respectively (Ministry of Health, 2017a).

In particular, only 35.8% of the children in CNS2002 consumed fruit ≥ 2 servings/ day (Ministry of Health, 2012a) and 56.4% ate vegetables ≥ 3 servings/day (Ministry of Health, 2012a), and only 21% consumed bread ≥ 5-6 servings/ day (Ministry of Health, 2012a). However, in NZ national survey with participants aged 5-24 years (n = 2, 503), findings from the self-reported dietary habit questionnaire showed that most children and young adult met the guideline for fruit consumption, but only 40% of the participants consumed the recommended servings of vegetables per day (Clinical Trials Research Unit, 2010). In general, only one third of the children met the guideline recommendation for total fruit and vegetable intake each day (≥ 5 servings/ day) (Clinical Trials Research Unit, 2010).

In NZ, there is a substantial rise in the prices of fruit, vegetables, meat, poultry, and fish between years ending 30th June 2013 and 30th June 2016, based on monthly and annual food price index reported by Statistics New Zealand (Statistics New Zealand, 2017). One of the major problems for Pacific families is food security, which means "access at all times to enough and nutritionally appropriate food to provide the energy and nutrients needed to maintain an active and healthy life" (Rush, Puniani, Snowling, & Paterson, 2007, p. 448). In a study of 1376 Pacific mothers, as part of PIF study approximately six weeks after the birth of babies, sometimes 39.6% (n = 545) of household ran out of food due to lack of money and a further 3.9% (n = 54) often ran out of food (Rush et al., 2007). Affordability of food is an issue for Pacific households with children and they were more likely to often or sometimes run out of food (53.9%) than those with Māori (37.5%) and NZEO children (13%) (Ministry of Health, 2003). In Pacific families, both affordability of food and the restraints imposed by parental employment factors are adversely influenced food choices (Statistics New Zealand &

Ministry of Pacific Island Affairs, 2011). On the other hand, 76% of Pacific children aged 0-14 years in Counties Manukau live in socioeconomic deprived areas (deprivation index 9 or 10) (Counties Manukau District Health Board, 2015b). Therefore, affordability of the food choices besides personal, family, and cultural preferences can explain 'occasional' pattern as the strongest eating pattern among these children.

The second reason could be adolescents tend to have less structured eating habits with more meals eaten outside (Livingstone, Robson, & Wallace, 2007). On the other hand, from the NZ Children's Nutrition Survey (CNS 2002) it was revealed that boys aged 11-14 years were more likely to buy from a shop/takeaway (27.3%) than girls of the same age (21.7%) (Ministry of Health, 2003). In addition, almost one quarter of Pacific children aged 11-14 years brought most of the food they consumed at school from the canteen or tuckshop and this was the highest for the children from the most deprived areas (Ministry of Health, 2003). Although this study only focused on the frequency of food consumption and not the access to food, it can be inferred from the findings that Pacific children with 'occasion' eating pattern had a higher interest of consuming foods prepared or produced out of home (39% in this study).

Pacific children compared to European and Māori children in NZ had a lower median usual daily intake of dietary fibre in the CNS2002 (Ministry of Health, 2003). The main identified sources of dietary fibre for NZ children were bread (20%), potatoes, kumara, taro and fruit (together 14%), then breakfast cereals and vegetables (both 11%) (Ministry of Health, 2003). Notably, in this study white bread with low amount of fibre (dietary fibre = 2.8g/serving https://www.shop.countdown.co.nz) was the most preferred bread type by both gender (60%) among the five asked types of bread ($p = 0.001$) which was similar to the CNS2002 (79%) (Ministry of Health, 2003). In the current study girls consumed better nutritional quality bread [more fibre ($p = 0.001$) and light grain bread ($p = 0.006$)] than boys, and they ate less bread ($p = 0.001$) than the boys.

Pragmatically four eating patterns were derived, a number that has previously shown meaningful (95% CI, $p < 0.05$) associations with NCDs including T2DM (Newby & Tucker, 2004). Each pattern considered in turn in the paragraphs that follow.

The 'occasional' eating pattern included fast food/ takeaways hot chips, french fries, wedges/ kumara chips, soft drinks/ energy drinks, lollies, sweets, chocolate and confectionary, fruit juices, and fruit drinks. 'Basic and staples' eating pattern consisted of vegetables (fresh/ frozen/ canned), fruit (fresh/ frozen/ canned/ stewed) and milk.

These two eating patterns were almost similar to two of the derived eating patterns (‘junk’ and ‘healthy’) among younger children lived in Auckland, NZ (Wall et al., 2013). In the Wall et al study, overall three eating patterns were derived (‘traditional’, ‘junk’ and ‘healthy’) among NZ European children who participated in a NZ birth cohort study at the age of 3.5 (n = 550) and 7 years (n = 591) (Wall et al., 2013).

‘Occasional’ pattern was the strongest eating pattern, which explained 22% of variance out of the total variance of 53%. This ‘occasional’ eating pattern was seen more among boys than girls ($p = 0.03$). The children who clustered in this pattern had similar frequency of eating of the low quality of nutrition profile foods with high fat (such as fast food/ takeaway and hot chips) and also high in carbohydrates and sugar (such as soft drinks/energy drinks and lollies). Cost of foods was one of the reasons that might be attributed to make ‘occasional’ pattern the strongest eating pattern. In a cohort of Scottish children aged 12-17 years (n = 2800) ‘vegetables’ pattern was associated with lower levels of deprivation and the ‘puddings’ pattern was associated with higher levels of deprivation (Craig et al., 2010). In another study by Schumacher and colleagues (2014) the eating patterns of Australian adolescent girls of low income socioeconomic position (n = 332, mean age = 13.7 ± 0.4 years) were studied. Consumed foods were categorized into core food group or energy-dense, nutrient-poor categories in line with the Australian Guide to Healthy Eating (Schumacher et al., 2014). This study showed poor consumption of core food group with an excessive consumption of energy-dense and nutrient-poor foods among the studied Australian children (Schumacher et al., 2014).

Factor loadings reflects the contribution of each food in each derived eating pattern. Interestingly, in the current study the order factor loadings of the foods with high sugar content in ‘occasional’ eating pattern was very similar to the list of the main sources of sugar for NZ children in CNS2002. Soft drinks/ energy drinks, lollies, sweets, ‘chocolate and confectionary’ and ‘fruit juices and fruit drinks’ in current study vs beverages (26%), sugar and sweets (21%) in CNS2002 (Ministry of Health, 2003) as sources of sucrose. On the other hand, NZ Ministry of health reported that the percent of Pacific children aged 2-14 years old who consumed fizzy drink three or more time per week have not significantly changed since 2006/2007 to 2015/2016 (24.2%, 30.6% respectively) (Ministry of Health, 2016b).

The second derived eating pattern was ‘seafood’ in which there were more girls (n = 106) than boys (n = 89) (p = 0.03). This pattern was based on higher consumption of fish and seafood. In CNS2002, this food group was identified as a source of protein for Pacific children, compared to other ethnicities (Ministry of Health, 2003). Therefore, identifying ‘seafood’ pattern among children in current study is mostly reasonable. Craig and colleagues among Scottish boys aged 5-11 years old (n = 381) identified an eating pattern named ‘fish and sauce’ (Craig et al., 2010). That pattern explained 3.0% variance out of 11.6%, is a similar proportion to the variance of ‘seafood’ pattern in the current study (13.7% out of 53.7%).

The third eating pattern in this study, clustered children who had similar frequency of consumption of vegetables, fruit and milk. This pattern explained 10.3% variance out of 53.7%. In ‘meat and bread’ pattern, the last of the four derived eating patterns, there were more boys (57%) compared to the girls (43%) (χ^2 4.3, p = 0.05) in this study. Based on CNS 2002 survey among 5-14 years old (Ministry of Health, 2003), the mean contribution to daily energy intake from protein (including bread, milk, poultry, fish and seafood and red meat) was higher for boys (14.0%, 88g/day among 11-14 years) than girls (13.5%, 66g/day among 11-14 years). In addition, among Pacific children, this was 13.3% for girls and 13.6% for boys (Ministry of Health, 2003). This may explain the higher presence of boys in ‘meat and bread’ pattern in this study.

Generally, ‘basic and staples’ provided the highest amount of dietary fibre in this study due to fruit and vegetables consumption (included one quarter of children). ‘Meat and bread’ pattern was the second pattern to provide dietary fibre, which was identified in one fifth of the children. Breakfast cereals was not asked by the dietary habit questionnaire in this study, although it is one of the main sources of dietary fibre for NZ children and it was eaten at least once a day by 39% of boys and 28% of girls aged 11-14 years in the CNS2002 (Ministry of Health, 2003). However, in general patterns of eating and food choices in this study reflect quite low intake of dietary fibre among Pacific children. In CNS2002, the median usual daily intake of dietary fibre was 17.9g, and it appeared adequate for NZ children (Ministry of Health, 2003). The benefits of foods with whole grain and fibre, and the effect on reducing the risk of T2DM have confirmed in extensive literature (such as S. Liu, 2002; Ye, Chacko, Chou, Kugizaki, & Liu, 2012) and the importance of daily consumption of foods with high dietary fibre such whole grain has been highlighted by many. Review on adults’ cohort studies with a minimum of three years follow-up (Alhazmi et al., 2014; Esposito et al., 2014)

suggested that eating patterns including more foods with plant-based such as fruit, vegetables, and grains may decrease the risk of T2DM.

Considering the individual consumption of fish in this study including fresh/ frozen fish/ shellfish, battered/ fried fish/ shellfish and canned fish/ shellfish showed that one out of three children in both genders never consumed fish and seafood. On daily basis, the median frequency of consuming fresh/ frozen fish/ shellfish, battered/ fried fish/ shellfish and canned fish/ shellfish was 0.07 times and there was no significant gender difference based on their daily frequency of consumption. This finding was different from the findings of CNS 2002 survey, in which Pacific boys consumed fish and seafood (protein from fish and seafood = 8%) more than the girls (5%) (Ministry of Health, 2003). This could be because the Pacific sample in the CNS was small, and those from PIF study were all from Counties Manukau region.

Individual food consumption of children from the PIF study has been tracked from age 4 (n = 1,066) to 6 (n = 1,019) (Savila et al., 2014), but not the eating patterns. Out of 111 foods asked by FFQ, nine food groups were defined: cereals and breads, meat and alternates, vegetables, fruit, mixed dishes, dairy, drinks, treats and fat (Savila et al., 2014). The method of dietary assessment at age 4 and 6 was different from that undertaken age 14 years (FFQ vs dietary habit questionnaire). However, the two largest food groups that comprised the largest percentage of daily food portions across both 4 and 6 years periods (Savila et al., 2014), was the same as the ‘meat and bread’ pattern among 14-year-old children. Moreover, Savila and colleagues (2014) concluded that the average daily consumption of food groups was very stable among 4 and 6 years (r^2 = 0.96). This highlights the stability of dietary habits and eating behaviours, which has been shaped during childhood and adolescents as the “key periods” (Leech et al., 2014, p. 2). Leech and colleagues in a review of eighteen studies among 5-18 years old children concluded that diet, physical activity and sedentary behaviours might be tracked into adulthood (Leech et al., 2014).

5.4.2 Eating patterns, foods consumption and HbA_1c

In this study the derived eating patterns were not related to HbA_1c, neither in total and nor by gender. Nevertheless, there were quite an extensive number of studies in which eating patterns with more plant-based foods like fruit and vegetables and less processed foods such as takeaways were associated with lower risk of T2DM among adults. For example in two studies on adults cohorts; Nurses’ Health Study (Fung et al., 2004) and

the Health Professionals Follow-Up Study (van Dam, Rimm, et al., 2002) two major eating patterns were derived using FA: ‘prudent’ (higher intakes of fruit, vegetables, whole grains, legumes, fish, and poultry) and ‘western’ (higher consumption of red meat, processed meat, french fries, high-fat dairy products, refined grains, and sweets and desserts). Then, researchers investigated the association of the derived patterns with the risk of T2DM. For T2DM diagnosis the existence of one or more following criteria rather than HbA_1c were considered: manifestation of classic symptoms such as excessive thirst, increased in fasting glucose concentration (7.8 $mmol.L^{-1}$) abnormal glucose tolerance test result (> 200 $mg.dL^{-1}$ 2 hours after glucose load) (Fung et al., 2004; van Dam, Rimm, et al., 2002). They found that the ‘prudent’ eating pattern was significantly associated with a reduced risk of T2DM. In particular Fung and colleagues (2004) after adjusting for potential confounders reported the RR for T2DM of 1.49 (95% CI [1.26, 1.76]), when compared the highest and lowest quintiles of the ‘western’ eating pattern with higher consumption of red and processed meats, sweets and desserts, French fries, and refined grains. Van Dam and colleagues (2002) reported that the RR for T2DM was 1.59 (95%CI 1.32-1.93), when compared the scores of ‘western’ eating pattern which was included similar foods in ‘western’ pattern in the study done by Fung et al. (2004).

Although children with the ‘occasional’ pattern were about 2cm higher (mean = 169.4, SD = 8.3) than children with ‘seafood’ pattern (mean = 167.2, SD = 7.1) (p = 0.045), no other associations were found between eating patterns and body measurements and neither with the HbA_1c. The foods with high sugar such as lollies and fruit juice were included in ‘occasional’ eating pattern. Hence, generally it may be concluded that this eating pattern had the highest carbohydrate and sugar compared to the other ones. Diets with these characteristics tend to have a high GI and glycaemic load and high GI content might contribute to the positive association observed between dietary carbohydrate and T2DM risk (Alhazmi et al., 2014).

One fifth of children in this study had the ‘meat and bread’ eating pattern characterized by red meat, chicken, processed meat products and bread/toast and bread rolls. Evidence from cohort studies suggests that the risk of T2DM may be mediated with eating patterns high in meat (Alhazmi et al., 2014). Multiple mechanisms may explain this effect associated with increased meat consumption. These include a high saturated fat content or the effect of a lower carbohydrate content on elevated glucagon and cortisol hormones, which in turn may increase insulin resistance (Alhazmi et al., 2014).

On the other hand, when considering the correlation of individual foods with HbA_1c, it was found that only among boys there was a small significant association between daily consumption of chicken and HbA_1c (r = 0.125 *p* = 0.017), but not for the red meat or processed meat. Finding no association with the latter food items might be related to higher consumption of poultry as a major source of protein compared to other ethnicities (Ministry of Health, 2003). In an adult cohort of men, the Health Professionals Follow-Up Study at 12 years (n = 42,504, aged 40-75 years) it was found that T2DM had a positive associations with consumption of processed meat (bacon, hot dogs, and other processed meats) (RR 1.46, CI 1.14–1.86 for ≥ 5/week vs. < 1/ month, *p* for trend < 0.0001) but no relation with chicken or red meat consumption was found (van Dam, Willett, Rimm, Stampfer, & Hu, 2002). The findings of a systematic review of 12 cohort studies among adults (minimum age of 26 years) led to the conclusion that high consumption of red meat and processed meat were risk factors for T2DM (Aune, Ursin, & Veierod, 2009).

5.4.3 Eating patterns and current body size measurements

Boys were more likely to have excess weight (being overweight and obese) (χ^2 12.7, *p* = 0.002), weighed more (*p* = 0.039), and were taller than the girls (*p* = 0.001) (Table 5.5). In addition, the body composition measurements showed that boys had proportionally more FFM (*p* = 0.001) and less FM (*p* = 0.001). There was a significant difference between height (cm) and derived eating patterns (*p* = 0.045) and in particular children with ‘occasional’ pattern were about 2cm higher than children, who had seafood pattern. However, there was no significant difference between eating patterns and other physical measurements especially the ones that have key role in addressing the weight status such as FM, FFM, weight, waist-to-height ratio and/ or BMI. Similar to our findings there are four studies among children (Alexy et al., 2004; Craig et al., 2010; Howe et al., 2013; Schumacher et al., 2014) that also failed to find any significant associations among derived eating patterns and BMI.

In Dortmund Nutritional Anthropometric Longitudinally Designed Study 228 German children aged 2-18 were followed between 1985 and 2002 and four distinct fat intake patterns were derived using cluster analysis (Alexy et al., 2004). The findings of this study showed no significant difference between derived patterns and BMI z scores. In another study, in 2005 Food Standards Agency Scotland commissioned the ‘Survey of Sugar Intake among Children in Scotland’ among 2800 children aged 3 to 16 years (Craig et al., 2010). The food frequency information in each age (5-11 and 12-17 years)

and gender group were analysed using PCA to identify eating patterns which in general were named ‘healthier’ with more fruit and vegetables and ‘unhealthy’ patterns named specifically in groups by ‘snacks’ and ‘puddings’, and no association between those eating patterns and BMI were found (Craig et al., 2010).

In the third study, Schumacher and colleagues (2014) similarly concluded that the weight status of adolescent girls (n = 332) did not vary among eating patterns and this was contrary to their hypothesis that patterns with high energy dense, nutrient poor foods were associated with weight of the girls.

In “Add Health” a prospective American cohort study, the eating patterns of 9,251 adolescents aged 11-21 years as well as their PA were analysed by gender applying CA (Boone-Heinonen et al., 2008). For girls, six eating patterns were derived and for boys seven patterns (Boone-Heinonen et al., 2008). It was concluded that among girls compared to ‘school Clubs and Sports’ with average diet (referent cluster), ‘average diet and activity’ pattern with average diet; ‘sedentary behaviours’ with high hours of screen activities and ‘restrictive dieting and smoking’ pattern with low food consumption were significantly associated with an increased prevalence of obesity (BMI> CDC 95th percentile) (adjusted odds ratios 2.02, 2.02 and 2.37 respectively) (Boone-Heinonen et al., 2008). While for boys a ‘sedentary behaviours’ pattern with high hours of screen activities was inversely associated with prevalence of obesity (adjusted odds ratios 1.27), it was not the cause of obesity for boys in ‘school Clubs and Sports’ with average diet (referent cluster) (Boone-Heinonen et al., 2008).

Ambrosini and colleagues (2016) characterised two major eating patterns of 6722 children from the Avon Longitudinal Study of Parents and Children (ALSPAC) by percentage of total energy intake (%E) from free sugars, %E from total fat and dietary energy density and fibre density by using reduced rank regression at 7, 10, and 13 y of age (Ambrosini et al., 2016). Eating pattern 1 was characterized by higher intakes of energy-dense foods, including confectionery and chocolate, cakes and biscuits, SSBs, and low-fibre bread, and lower intakes of fruit, vegetables, and high-fibre bread and cereals and greater %E from sugar, %E from fat, and dietary energy density and lower dietary fibre density than eating pattern 2 (Ambrosini et al., 2016). A 1 SD increase in z score for eating pattern 1 was associated with a mean increase in fat mass index z score of 0.04 SD units (95%CI 0.01,0.07) and greater odds of excess fat (OR 1.12 95% CI 1.0, 1.25). It was concluded that an energy dense eating pattern high in %E from total

fat and free sugars is associated with greater adiposity in children (Ambrosini et al., 2016).

Bogota study of nutrition and health in school-age children from Bogota´ School Children Cohort, is an ongoing longitudinal study of nutrition and health on children aged 5-12 years from Bogota´, Colombia (Shroff et al., 2014). In this study, the follow-up over a median 2.5 years of 961 children showed that the ‘snacking” pattern characterized by candy, ice cream, packed fried snacks, soda and sugar-sweetened fruit-flavored drinks, had a 0.09 $kg.m^2$ change in BMI (as an overall indicator adiposity) but not for the waist circumference as an indicator of truncal adiposity (Shroff et al., 2014).

5.4.4 Current body size measurements and HbA_1c

Thirty two children were diagnosed with $HbA_1c \geq 41$ $mmol.mol^{-1}$. Based on IOTF cut-offs (Cole & Lobstein, 2012) only one in five were normal weight and the rest of them were grouped as overweight (n = 6) or obese (n = 19). Seven out of ten children had excess weight in this study which unfortunately confirms previous study findings on extremely high rate of overweight and obesity in Pacific children. The rate of overweight/ obesity among Pacific children in NZ is 3.6 times more than non-pacific children and since 2006/2007 survey to 2015/2016 survey it had a 7% growth in the proportion (23.1, 29.8) (Ministry of Health, 2016b). In addition, twenty percent of children in the most deprived areas are overweight or obese, compared with four percent living in the least deprived areas (Ministry of Health, 2016b). One of the reasons can be the contribution of lower incomes and poverty that increase stress, nutrition-limited food availability and obesity in women and children (Savila & Rush, 2014).

There was a positive correlation with BMI z-score and HbA_1c. After adjusting for gender and age, the HbA_1c was positively associated to waist, waist-to-height ratio (central fat), and FM , too. On the other hand, excluding the four children who previously diagnosed with diabetes, the other 28 children whose HbA_1c was ≥ 41 $mmol.mol^{-1}$ had more overweight and obese BMI cut-offs (normal weight = 5, overweight = 6, obese = 16 and missing = 1). Considering a large number of Pacific children with excess weight, these findings might confirm that HbA_1c can be a good predictor of T2DM among overweight and obese ones. This has been confirmed in two studies done by Ehelalt and colleagues (2017) (n = 4848, aged 7-17 years) in Germany and Shah and colleagues (2009) (n = 468, >10 years of age) on American obese and overweight children. They suggested that HbA_1c can be a good predictor of T2DM in

children with excess weight (overweight and obese). Moreover, among obese and overweight children a cut-off value of 42 mmol.mol^{-1} (94% sensitivity, 93% specificity) was suggested to screen TDM among obese and overweight children (Ehehalt et al., 2017).

Moreover, the rate of overweight (including obesity) and obesity among the children who were measured in PIF at the age of 10 years was 70% and 50% (Rush, Obolonkin, & Savila, 2013) and it is known that childhood obesity often persists into later life (Leech et al., 2014). An exploratory study including 30 Pacific children aged 16-24 years highlighted that the future health trajectory of Pacific children is poor and related to the obesogenic environment (Tupai-Firestone et al., 2016). Therefore, there will be a large increase in prevalence of T2DM among Pacific children (Savila & Rush, 2014), if overweight and obese children in this study continue the current pattern in rapid weight gain and exposure to the present environment.

5.4.5 Early life factors and type 2 diabetes mellitus

Results from some authors showed that both low birth weight and high birth weight subjects have high-risk or revealed that high birth weight but not low birth weight is followed by an increased risk of T2DM (Harder et al., 2007). However, the birth weight of children in this study was not associated with HbA_1c or high or low birthweight.

This finding is in contrast to findings from 2,164 Japanese with T2DM aged 40-59 years (Anazawa, Atsumi, & Matsuoka, 2003) in which HbA_1c was considered as a biomarker for defining T2DM. Birth weights < 2,500, 2,501–3,699, and > 3,700 g were defined as low, normal, and high, respectively and people with HbA_1c> 48 mmol.mol^{-1} were defined as patients with T2DM (Anazawa et al., 2003). The Japanese results showed that low birth weight was also associated with the development of T2DM in Japanese participants (Anazawa et al., 2003) and the odds ratio of T2DM per 1 kg increase in birth weight was 0.65 (95% CI [0.38, 1.09]) (Whincup et al., 2008). This highlights the importance of continued followed-up studies as the difference may manifest in later life.

In an analysis of two birth cohort studies in Brazil (n = 1984, age = 22-23 years) undertaken by Silva and colleagues (Silva et al., 2012) it was concluded that birth weight might affect insulin sensitivity and secretion in young adults. In both cohorts, birth weight z-scores were negatively associated with insulin sensitivity (log HOMA2-%S) only after adjusting for gender, age, schooling, smoking habit, alcohol consumption, preterm birth and maternal schooling at the time of the child’s birth

(cohort 1: standardized coefficient (95% CI) = 0.04 [-0.01, 0.09] p = 0.050, cohort 2: standardized coefficient (95% CI) = -0.05 (-0.09; -0.01) p = 0.042) (Silva et al., 2012).

The Ministry of Health recommends exclusive breastfeeding until a baby is about six months old, when solid food is introduced. The reported percentage of Māori and Pacific babies fully breastfed at 5-6 weeks was lower than "European and other" babies in NZ (Ministry of Health, 2002a). Half of both Pacific girls and boys in the current study were exclusively breastfed during the first six weeks and this was the same as the percentage of Pacific babies breastfed at 4- 6 weeks (50%) as reported by Counties Manukau District Health in 2014 (Counties Manukau District Health Board, 2015a).

A study with similar findings used data from National Longitudinal Study of Adolescent Health (n = 16,903, average age 15 y) in which breastfeeding was examined as one of fifteen indicators including diabetes of American adolescents well-being (Evenhouse & Reilly, 2005). Only 55% of children were ever breastfed and by 3 months only 12% were breastfed. The indicator for defining diabetes was not explained but juvenile-onset diabetes is referred to throughout the text. The authors highlighted that the using a very small sample size (only 97 cases out of 16,903) was a possible reason in the case of rare outcome for diabetes (Evenhouse & Reilly, 2005).

The SEARCH Case-Control study, an ancillary study to SEARCH for Diabetes in Youth aged < 20 years, 80 patients with T2DM were compared with 167 control subjects (Mayer-Davis et al., 2008). In this study, the duration of breastfeeding was categorized to 'never breastfed', 'breastfed for <6 months' and breastfed for ≥ 6 months and there was a significant difference found between participants and the duration of breastfeeding (p< 0.0001) (Mayer-Davis et al., 2008).

5.4.6 Maternal education, age at conception and type 2 diabetes mellitus

In the current study, there were no significant relationships were found between derived eating patterns with either maternal education levels or maternal age. This is while there is a possibility that children's intention to eat healthily predicted by their mothers' intentions and perceived behavioral control (Sumodhee & Payne, 2016). The level of maternal education as an indicator of socio-economic status of the family has been examined against the derived eating patterns among children in several studies such as AVON longitudinal study (Emmett et al., 2015), three cross-sectional surveys among Spanish children and adolescents (Aranceta et al., 2003), European (HELENA) and Australian (NCNPAS) adolescents (Huybrechts et al., 2017), and the Survey of Sugar

Intake among Children in Scotland (Craig et al., 2010). These studies show that the lower education levels of mother or the main food provider was related to eating patterns that include processed and energy-dense foods.

In ALSPAC study with children aged four (n = 9550) and seven (n = 8286) years old, three derived eating patterns including ‘junk type’, ‘traditional British diet’ (based on meat, potatoes and vegetables) and ‘health-conscious’ pattern (based on meat, potatoes and vegetables) (Northstone & Emmett, 2005). It was the ‘health-conscious’ pattern that was more likely related with increasing the maternal levels of education and age (Northstone & Emmett, 2005).

Interestingly, the HbA_1c was positively correlated to maternal age at conception and also the levels of education. In the SEARCH Case-Control study (Mayer-Davis et al., 2008) the maternal age was categorized to < 35 years and ≥ 35 years and the maternal education levels were ‘less than high school’ and ‘high school graduate and more’. It was found that there was no statistically significant difference between participants with T2DM (n=80) and the control (n=160) and maternal age (p = 0.5194), but more participants with T2DM 13.8% had less than high school education compared with the control where 6.6% had less than high school education (p = 0.0690) (Mayer-Davis et al., 2008).

5.5 Strengths and limitations

One strength of this work is that the number of children surveyed, which provided adequate statistical power (power >80%, p <0.05, 370 girls and 370 boys) for comparisons of boys and girls and to observe associations. That enabled to understand the general eating patterns and risk of T2DM in this young growing ethnic group, which has a higher risk of developing NCDs such as T2DM compared to other ethnic groups in NZ (Ministry of Health, 2016b). Moreover, differences in food-related beliefs, preferences and behaviours are associated to ethnicity (Kumanyika, 2008). In addition, eating patterns are also influenced by other features of an ethnic population (Leech et al., 2014) such as the contributory factors of culture, for example church attendance, secular trends, age of the population, socio-economic status, the built environment, and time available. Thus, the findings of this study can provide a valuable example of patterns of eating among Pacific children but the contributory factors need to be explored further before causality or relative contribution can be determined.

Another strength was that all except one of the assessors for obtaining the consent and collecting data were Pacific and of both gender. This made the process of communication between research team and caregiver of the children and children easier. The rationale behind this was having the opportunity for communicating with participants in Pacific languages if needed and undertaking measurements by an assessor of the same gender.

The dietary habit questionnaire that was used to collect food information in this study, reported the frequency of consumption of only fifteen specific foods and it was able to reflect and compare with the eating habits as measured by the Ministry of Health in surveys with reduced participant burden. Unfortunately, this questionnaire does not include questions concerning the frequency of consumption of some key foods such as breakfast cereals, rice, or noodles. These three foods were in the top twelve consumed foods reported at the ages of 4 and 6 years among PIF children and were shown to have moderate stability in frequency of consumption at age 4 and 6 years ($r^2 = 0.53$) (Savila et al., 2014) and were more commonly eaten by NZ children measured in a 121 item food frequency questionnaire (Ministry of Health, 2003). This omission and the lack of detail in the questionnaire affects the ability to assess the relationship of derived eating patterns with HbA_1c. The shorter dietary habit questionnaire was pragmatically chosen over the longer FFQ to limit the time of each interview.

Moreover, children were asked to complete the online dietary habits questionnaire, using tablets in groups of a maximum ten children. The assessor gave the instruction at the beginning and the completion of the questionnaire, which was self-administrated unless the assessor was asked for help. This might be a reason for missing food information collected through this method for eating pattern analysis. In addition, the design of the used online questionnaire allowed the researcher to check the responses after completion of the data collection. Moreover, it is important to provide a more clear description for children about the answer options such as ‘1-2 times per week’,’ not applicable’ and ‘I do not know’ by providing proper written examples from everyday life and programming to alert the child, when a question left unanswered.

The major strength of this relatively small contribution to the understanding of growth and health in this cohort of Pacific children is that it builds on the information collected at birth, six weeks, 1, 2, 4, 6, 9, 11, and 14 years. Moreover, it adds more data to the

already large (>11000 variables) database that tracks these Pacific families and their children.

5.6 Recommendations

Pacific population in NZ has its own characteristics of food consumption: frequent consumption of certain foods, serving sizes and cooking practices (Metcalf, Scragg, Sundborn, & Jackson, 2014). Applying each dietary assessment methods has its own limitations. In order to capture a more accurate information on consumed foods perhaps using a combination of two methods like food record and FFQ can be useful in deriving eating patterns. An example of this can be found in Avon Study (Emmett et al., 2015) in which food records were reviewed by a trained nutrition fieldworker to clarify any uncertainties such as cooking methods and possible missing items. Moreover, the misreporting energy intake was assessed by an individual method (Emmett et al., 2015).

In addition, supervising children when completing questionnaires, especially when online questionnaires are being used is beneficial. This can decrease the amount of missing data in collected data.

A cohort study with 6002 girls and 4917 boys at the age of 9-16 years concluded that increases in screen time which included television, electronic games, and digital versatile discs, correlated with increased amount of poor nutritious foods and beverages such as sugar sweetened beverages, fast food, sweets and salty snacks (Falbe et al., 2014). The findings of PIF main study showed that the strongest eating pattern was occasional pattern and consumption of poor nutritious foods was frequent. Considering the sedentary behavior of these children and exploring the association of those factors with eating patterns and T2DM biomarkers is greatly recommended.

5.7 Conclusion

The current study found that the overall vegetables consumption by the focused children was insufficient. This study also found that one fifth of the children consumed milk less than once a day. Furthermore, the ‘occasional’ eating pattern with poor nutrient and high-energy food items was the strongest derived eating pattern among PIF main study children. This highlights that Pacific children need encouragement to choose healthy food options wisely (Regan, Parnell, Gray, & Wilson, 2008).

The paradoxical findings on eating patterns and risk of T2DM suggest that, these apparently healthy children, who are at higher risk of developing T2DM compared to other NZ ethnicities in NZ, should be monitored.

There is an urgent need to take on-time actions to address the current state of Pacific health inequalities (Statistics New Zealand & Ministry of Pacific Island Affairs, 2011) and inequities. Actions such as a living wage, improvements of the built environment including community gardens and dry housing would support the Pacific community, especially children and young adults to have healthier food choices, can improve the future health and wellbeing of the Pacific community who have high risk of developing T2DM.

Chapter 6 Sweet food consumption and risk for type 2 diabetes mellitus: Pacific Islands Families sub-study

6.1 Introduction

In the previous study on Pacific children (Chapter 5), four eating patterns were derived, with the 'occasional' eating pattern being the strongest one. That eating pattern clustered children with a similar frequency of consuming foods which were high in sugar and energy content such as fast food, soft drinks and fruit juices.

The World Health Organization (WHO) reports concern at the quantity of 'free sugars' in foods and beverages consumed. Free sugars are defined as "monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and fruit juices" (World Health Organization & Food and Agriculture Organization, 2003, p. 56). Of special concern are sugars in the form of sugar sweetened beverages (SSBs), because of associations with poor dietary quality, obesity, risk of NCDs including T2DM, and dental diseases (World Health Organization, 2015).

Evidence from many studies with adults (e.g. Malik, Popkin, Bray, Després, Willett, et al., 2010; Te Morenga et al., 2013) and children (e.g. Ambrosini et al., 2016; Lin et al., 2016) links the consumption of free sugars including SSBs with the development of NCDs such as obesity and T2DM. However, some reviews report inconsistent associations between SSBs and risk of excess weight and obesity-related diseases. This inconsistency is attributed to: 1) the lack of strong cohort studies with repeated measurements in which confounders were carefully adjusted (Hu, 2013), 2) limitations in adjusting for energy intake from SSB (Trumbo & Rivers, 2014), and/ or small effect size estimate, and 3) equivocal statistical significance (Kaiser, Shikany, Keating, & Allison, 2013).

It has been reported that the leading source of consumed total sugars among NZ children (5-14 years) was from beverages (*Non-alcoholic beverages*: all teas, coffee and substitutes, Milo™, juices, cordial, soft drinks, water, powdered drinks, sports, and energy drinks; *Alcoholic beverages*: wine and spirits), followed by fruit (including all fruit, fresh, canned, cooked and dried) and sugar and sweets (including sugars, syrups, confectionery, chocolate, jam, honey, jelly, sweet toppings and icing, ice blocks, artificial sweeteners) (Ministry of Health, 2003). By ethnicity, like other children in NZ

the main source of sucrose for Pacific children was sourced from beverages, but compared to the proportion of consumption of NZ European and Other children (24%), the proportion of Pacific children (32%) was greater (Ministry of Health, 2003). In addition, children living in socioeconomically deprived areas are more likely to drink three or more fizzy drinks per week (Ministry of Health, 2016b) than those living in less-derived areas.

Based on the nutritional survey on NZ children (Ministry of Health, 2003), the main types of sugar consumed were sucrose (boys 67 g/ day; girls 61.1 g/ day), fructose (19.6 g/ day; 18.5 g/ day), glucose (17.8 g/ day; 16.5 g/ day), lactose (15.5 g/ day; 13.2 g/ day), and maltose (4.1 g/ day; 3.2 g/ day).

The main contributors of sweet-tasting sugars naturally found in foods are lactose in milk, sucrose in fruit, and vegetables. Sucrose or “commercial sugar” (Lean & Te Morenga, 2016, p. 2) or “centrifugal cane sugar” (Rush, Savila, & Obolonkin, 2014, p. 47) is a disaccharide of glucose and fructose. Sweeteners, such as high fructose corn syrup (HFCS), contain free fructose, and free glucose in relatively equal proportions (Malik & Hu, 2015). Different forms of HFCS are used mainly in processed foods such as cereals, baked goods, desserts, fruit flavoured non-carbonated beverages, candies, and many fast food items (Malik & Hu, 2015).

Fructose is 50% of sucrose, a disaccharide frequently added to food and high fructose corn syrup (HFCS) contains up to 60% fructose. It is suggested that consumption of > 50 g/ day of fructose may contribute to the aetiology of metabolic syndrome and T2DM by increasing the intracellular fat in the liver, brain, blood vessels, muscle, kidney and adipose cells (Johnson et al., 2009). It has a pathway in metabolism that leads to the generation of uric acid (Johnson et al., 2013; Johnson et al., 2009; Malik & Hu, 2015) and this may also induce insulin resistance (Johnson et al., 2009).

Johnson and colleagues (2013; 2009) and Malik and Hu (2015) explained the pathway of fructose metabolism and production of serum uric acid (SUA). The potential mechanisms for fructose and SUA-induced insulin is ‘fructose enters cell via a transporter (primarily Glut 5) where it is acted on by fructokinase. As part of this metabolism, ATP depletion may occur, generating uric acid with systemic effects that block insulin-dependent NO-mediated vascular dilation as well as direct cellular effects on the adipocyte. Fructose intake also drives de novo lipogenesis that can leads to accumulation of intracellular triglycerides in the liver, which induces insulin resistance’

(Johnson et al., 2009, p. 101). In particular, elevated SUA concentrations are shown to be associated with incidence of T2DM (Xu et al., 2016), the possible pathways are through hepatic, white adipose tissue, brain, renal, vascular, and/ or pancreatic islet cell changes associated with metabolic function (Johnson et al., 2013; Johnson et al., 2009).

Xu and colleagues (2016) recently evaluated the suggested positive correlation between SUA and T2DM by systematically reviewing sixteen publications of cohort studies (mean age ≥ 37 years). It was concluded that SUA was independently associated with progression of T2DM (in both genders, older and young people) and insulin resistance increased as baseline SUA concentration increased (Xu et al., 2016). Prediction equations indicated that each 1 mg/dl (0.06 $mmol.L^{-1}$) increase in SUA led to a 13.1% increase in the risk of T2DM (Xu et al., 2016). In order to clarify whether this relationship is causal or simply a co-occurrence, there is a need for more evidence of aetiology, mechanisms and especially genetics (Xu et al., 2016).

In another systematic literature review (Jia et al., 2013) on twelve cohort studies (mean age of > 24 years), it was concluded that there is a nonlinear and positive relationship ($p < 0.01$) between SUA and incidence of impaired fasting glucose (IFG) and T2DM.

The Rotterdam study is a prospective cohort study ongoing since 1990 with 7,983 participants aged ≥ 55 years in the city of Rotterdam in the Netherlands (Dehghan et al., 2008). During a mean follow-up of 10.1 years of 4,536 healthy adults at baseline to study the association between SUA concentration and risk of T2DM, 462 of participants developed T2DM (Dehghan et al., 2008). The authors concluded that based on the findings in this population, SUA is a “strong and independent” (Dehghan et al., 2008, p. 361) risk factor for T2DM.

From the genetic and metabolism perspective, there are differences in SUA metabolism among Polynesian, when compared with non-Polynesian people (Houghton, 1996). While the specific cause or gene has not been identified, SUA and the prevalence of gout is higher in those of Māori, South Pacific Polynesian, Samoa, the Cook Islands, the Tokelaus, and Tongan origins (Houghton, 1996). Thus, it appears that there is a genetic predisposition that is sensitive to the environment. Hollis-Moffatt and colleagues (2009) studied the genetic basis of gout in NZ sample sets of Māori (n = 56), Pacific (n = 69) and Caucasian (n = 131) patients while considering a control group. They determined the association of SLC2A9 gene with gout in Māori and Pacific patients (Hollis-Moffatt et al., 2009). Moreover, urbanisation and emigration of Pacific Island groups to NZ was

associated with an increase in the prevalence of NCDs such as gout, T2DM and obesity (Prior, 1981).

For the diagnosis of T2DM as well as the official recommendation by an expert committee to use the HbA_1c (The International Expert Committee, 2009), elevated SUA recently has been recognised by epidemiologists as a risk factor for T2DM (Dehghan et al., 2008; Xu et al., 2016). On the other hand, there has been limited studies on healthy children in which SUA reported as a predictor for T2DM (de Miranda et al., 2015; Lin et al., 2016; Sun, Pei, Lue, & Chen, 2015). Therefore, in the current investigation the association of both biomarkers HbA_1c and SUA with the consumption of added-sugar foods was investigated.

The main question was ‘what are the associations of added-sugar foods and risk of T2DM among Pacific children?’. For this, the frequency of added sugar foods (fast food/ takeaways, hot chips, French fries, wedges/ kumara chips, soft drinks/ energy drinks, lollies, sweets, chocolate and confectionary, fruit juices, and fruit drinks) tested against the concentration of HbA_1c and SUA to examine whether the consumptions of the named foods were related to T2DM biomarkers in Pacific children (n = 204, age ≈15 years). These food items included in the ‘occasional’ eating pattern derived from Pacific children participated in the PIF main study (n = 740, age = 14 years) (Chapter 5 Table 5.10).

The sequential questions are:

1. What are the most frequently consumed foods containing added and simple sugars among Pacific children?
2. What are the associations between these frequently consumed foods and HbA_1c?
3. What are the associations between these frequently consumed foods with SUA concentration?
4. What are the associations between T2DM biomarkers (HbA_1c and SUA concentrations) with body size measurements?

6.2 Design and methods

Aligned with the existing PIF child database is the PIF Sub-study, which was undertaken for this doctoral thesis (Table 6.1). This Sub-study named “understanding growth study”, was a cross sectional project, on children of 15 years of age; this time was selected as a critical time during which children experience rapid growth. The aim of the PIF Sub-study was to investigate the influences on growth rates of Pacific children. Information about food consumption and activity patterns, pubertal development, measuring blood biomarkers such HbA_1c and SUA, the family and wider environment factors were collected to identify the relationships with assessed body size, fatness and growth rate. This section presents a brief review of the methods used for collecting data on food, blood biomarkers and body measurements of the children.

Table 6.1. Food, biomarkers and body measurements included in the PIF substudy

PIF sub-study	Category of the information
Frequency of food consumption	Food
HbA_1c	Biomarker
Urate concentration	Biomarker
Body size e.g. BMI z scores, body weight, height and waist, and fat distribution	Body measurements

6.2.1 Study participants

Between October 2014 and February 2016, the PIF sub-study collected the data of a nested subsample (boys = 104, girls = 100), with a random selection from those children who had already been measured in PIF main study in full cohort (n = 931). The randomized selection of children was for10 boys and 10 girls from each decile of body weight at 11 years (Rush, Oliver, et al., 2016) and required that they were resident in Auckland. A Pacific research assistant visited the selected children to deliver the invitation to participate in the sub-study phase.

6.2.2 Ethical considerations

The ethics approval for PIF sub-study was obtained from the Central Health and Disability Ethics Committee on 28 July 2014 (ref. 14/CEN/108) (Appendix E). Informed consent was obtained from parent and the child in advance. For the measurements, based on earlier arrangements with the child and parent, the child in an overnight fasted state was transported to and from the Body Composition Laboratory of

the University of Auckland, Department of Surgery, based at Auckland City Hospital, by the research assistant. At the completion of the assessment, children were provided with breakfast and they were thanked for their participation with a gift voucher.

6.2.3 Food information

The online validated qualitative food frequency questionnaire, based on the MOH FFQ (Ministry of Health, 2003; University of Otago & Ministry of Health, 2011b) with the focus on sugar intake (particularly fructose) was administered at the time of obtaining the consent from the children (Appendix F and Appendix G). It included foods grouped as follows: fruit, cereals, spreads, sauces, convenience meals/snacks, dairy (excluding milk), biscuits/cakes, snacks and sweets, milks, and other (non-milk) drinks. In this questionnaire, the frequency consumption of a food or group of foods was applied to children, who ate a particular food monthly or more often. This included all the children, who ate the food 1–3 times a month, 1–2 times a week, 3–4 times a week, 5–6 times a week, once a day and 2 or more times a day 2 times a week, 3–4 times a week, 5–6 times a week, once a day and 2 or more times a day.

For food analysis and investing the associations with T2DM biomarkers and body measurements the focus was on the frequency of consumed foods which were highlighted in ‘occasional’ eating pattern. The predefined food groups based on the applied FFQ were used (Table 6.2).

Table 6.2. Predefined food groups based on the applied food frequency questionnaire in the PIF main study

Predefined food groups	Food items
Spreads and sauces	included jam or honey, Nutella, Marmite or Vegemite, peanut butter, mayonnaise or salad dressing, tomato sauce or ketchup and other spreads or sauces
Convenience meals/ snacks	included canned spaghetti with tomato sauce, baked beans and other convenience meals/snacks
Biscuits/ cakes	included chocolate coated or cream filled biscuits, biscuits e.g. plain, bars e.g. muesli bar, crackers or crispbreads, cake or slice, doughnuts or croissants, scones, muffins or sweet buns, pancake or pikelets, fruit pie, fruit crumble or tart, pudding, custard or custard puddings and other item of the biscuits/ cakes
Snacks and sweets	included popcorn, chocolate, candy coated chocolate and other sweets
Other drinks [a]	included juice, powdered fruit drink, fruit drink from concentrate or cordial, Coca cola or other cola drinks, Mountain Dew, 'New Age' drinks, soft drinks, sports drinks, Ice blocks, tea, coffee and other item of the 'Other drinks'

[a] Contain free sugar. It is named 'sugary drinks' in the current study.

The frequency of consumption was multiplied by the fraction per day (monthly, weekly, or daily) (Table 6.3), so that derived values represented the daily rate of consumption.

Table 6.3. Frequency of food consumption and the applied weighting factor to standardise to a daily rate

Frequency of consumption	Weighting factor /day
Never or less than once a month	1/200
1-3 times a month	2/30
1-2 times a week	1.5/7
3-4- times a week	3.5/7
5-6 times a week	5.5/7
Once a day	1
2 or more times a day	2

6.2.4 Blood biomarkers

The concentration of HbA_1c was measured by using the Afinion^{TM} AS100 Analyzer (AXIS-Shield PoC, Oslo, Norway), a non-fasting finger prick blood sample of 1.5 µL.

Fasting venous blood sample was collected to assess the concentration of SUA. For this, the sample was sent to the International Accreditation New Zealand (IANZ) accredited Lab Plus medical laboratory at Auckland City Hospital. There SUA was measured by a clinical chemistry automated analyzer (Roche c702), using reagents, calibrators, and standard operating procedures as specified by the manufacturer. The laboratory provided the cut-off points for hyperuricemia of > 0.36 $mmol.L^{-1}$ for girls and > 0.42 $mmol.L^{-1}$ for boys.

6.2.5 Body measurements

For anthropometric measurements, height, weight, and waist circumference (measurement at narrowest point between lower rib and hip) were measured. The measurements were repeated twice with three readings unless the measures were not more than 0.5 cm (height and waist circumference) or 0.5 kg (weight).

A portable stadiometer (Seca 213 Hamburg, Germany) and an electronic scale (Tanita BC545, Tokyo) were used to measure height and weight respectively. Waist circumference measurements were made using a non-stretchable tape. BMI was calculated by using the following metric formula:

BMI = (weight in kilograms) divided by (height in meters x height in meters)

Body composition analysis was assessed by Dual energy x-ray absorptiometry (DXA, model IDXA, GE Lunar, Madison, Wisconsin, USA) to measure the total body and abdominal fatness (Rush, Oliver, et al., 2016). The instruction was explained by hospital staff to the children. Total fat mass and fat free mass were analysed using enCORE software version 13.6. This method is previously described in adults (Kaul et al., 2012).

The Centres for Disease Control (CDC) z scores (Kuczmarski et al., 2000) were derived from BMI, age, and gender. To determine the prevalence of obesity and overweight the International Obesity Task Force cut-offs (Cole & Lobstein, 2012) applied to categorise as thin, normal, overweight, and obese.

6.2.6 Carbohydrate content of individual food

To investigate the amount of average carbohydrate (CHO), sugar, glucose, and fructose, Food Works software (version 7, add more details from the Food) was used. This Australian software provided analysis of components of different food products.

6.2.7 Statistical analysis

Children information including age and gender, food information, HbA_1c and SUA concentrations and body measurements were entered to SPSS 24 spreadsheet (SPSS Inc., New York, USA). These variables were analysed for significant differences between groups, based on the groupings below. The significance level was set at 0.05 (5%).

Continuous variables from the above list were explored for descriptive data and distribution. Normally distributed variables including age, height, weight z scores, height z scores, BMI z scores, FFM, FM% and SUA are reported as mean ± SD, while non-normally distributed are reported as median with IQR. To test for differences by gender independent T-test and Mann Whitney test were used. Pearson and Spearman correlation tests were applied to assess the associations. Multiple linear regression (stepwise) was performed accounting for gender.

6.3 Results

6.3.1 Body and blood measurements of children at 15 years

The children selected for this sub-study were not different to the full cohort in terms of weight, height, and BMI when measured at the time of the full study. Seven months later on average boys were taller (9 cm) but not heavier than girls (Table 6.4) and had less FM (5 kg less) and more FFM (10 kg more) than girls (Table 6.4). Forty-two percent of girls and 39% of boys were obese, a further 34% and 32% were overweight based on International Obesity Task Force BMI cut-off (Cole & Lobstein, 2012).

Table 6.4. Physical measurements of Pacific children (n = 204)

	Girls (n = 100)		**Boys (n = 104)**		
	Mean	**SD**	**Mean**	**SD**	***p* Value** [e]
Age (years)	14.92	0.47	14.88	0.43	0.48 [f]
Anthropometry					
Weight (kg)	81.2	20.6	85.8	25.2	0.24
Height (cm)	166.6	5.6	175.4	7.1	**0.001** [f]
BMI ($kg.m^{-2}$)	29.1	6.5	27.7	7.5	0.07
Waist (cm)	84.5	16.2	88.6	18.8	0.19
Waist/ height (cm)	0.50	0.09	0.50	0.10	0.57
Weight z scores [a]	1.68	0.75	1.79	1.18	0.44 [f]
Height z scores [a]	0.75	0.86	0.81	0.90	0.57 [f]
BMI z scores [a]	1.55	.70	1.42	1.02	0.26 [f]
Body composition [b]					
Fat free mass (kg)	49.7	8.4	60.0	11.3	**0.001** [f]
Fat mass (kg)	31.3	13.0	25.9	15.6	**0.001**
Fat mass%	37.3	6.5	28.0	9.3	**0.001** [f]
Biomarkers					
HbA_1c ($mmol.mol^{-1}$) [c]	36.6	5.9	35.8	3.4	0.304
SUA ($mmol.L^{-1}$) [d]	0.33	0.63	0.43	0.09	**0.001** [f]

[a] CDC growth charts (Kuczmarski et al., 2000). [b] Measured by Dual X-ray absorptiometry (DEXA). [c] HbA_1c; Glycated haemoglobin A_1c. There were two boys missed from HbA_1c finger prick measurement. [d] SUA; serum uric acid. There were four girls and a boy missed from urate measurement. [e] Mann-Whitney U test. [f] Independent t test.

Concentration of HbA_1c ranged 49 $mmol.mol^{-1}$ with five children ≥ 41 $mmol.mol^{-1}$ (Figure 6.1. and was not different by gender (Table 6.4). Fifty-five percent of the boys

and thirty percent of girls had SUA higher than the gender specific cut-off points for hyperuricemia (Figure 6.2.). The concentration of SUA was higher (p = 0.001) in boys (0.43 ± 0.09 mmol.L^{-1}) compared to girls (0.33 ± 0.63 mmol.L^{-1}) (Table 6.4).

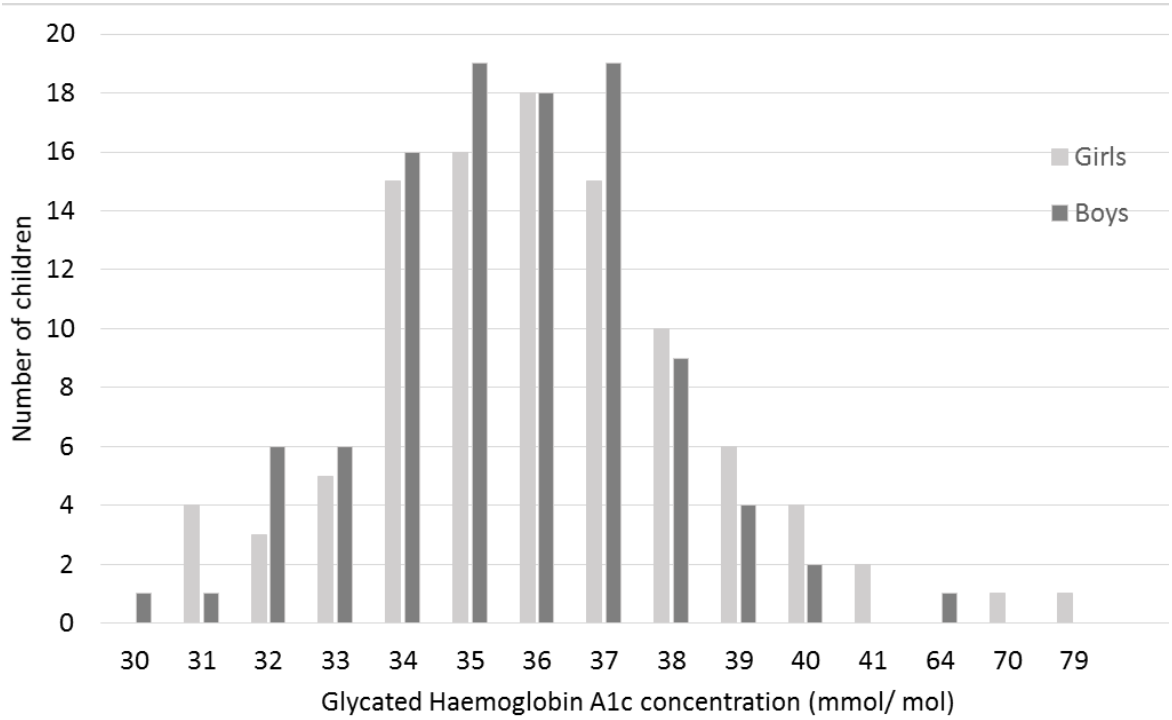


Figure 6.1. Frequency of glycated haemoglobin A_1c by gender among Pacific children at 15 years old

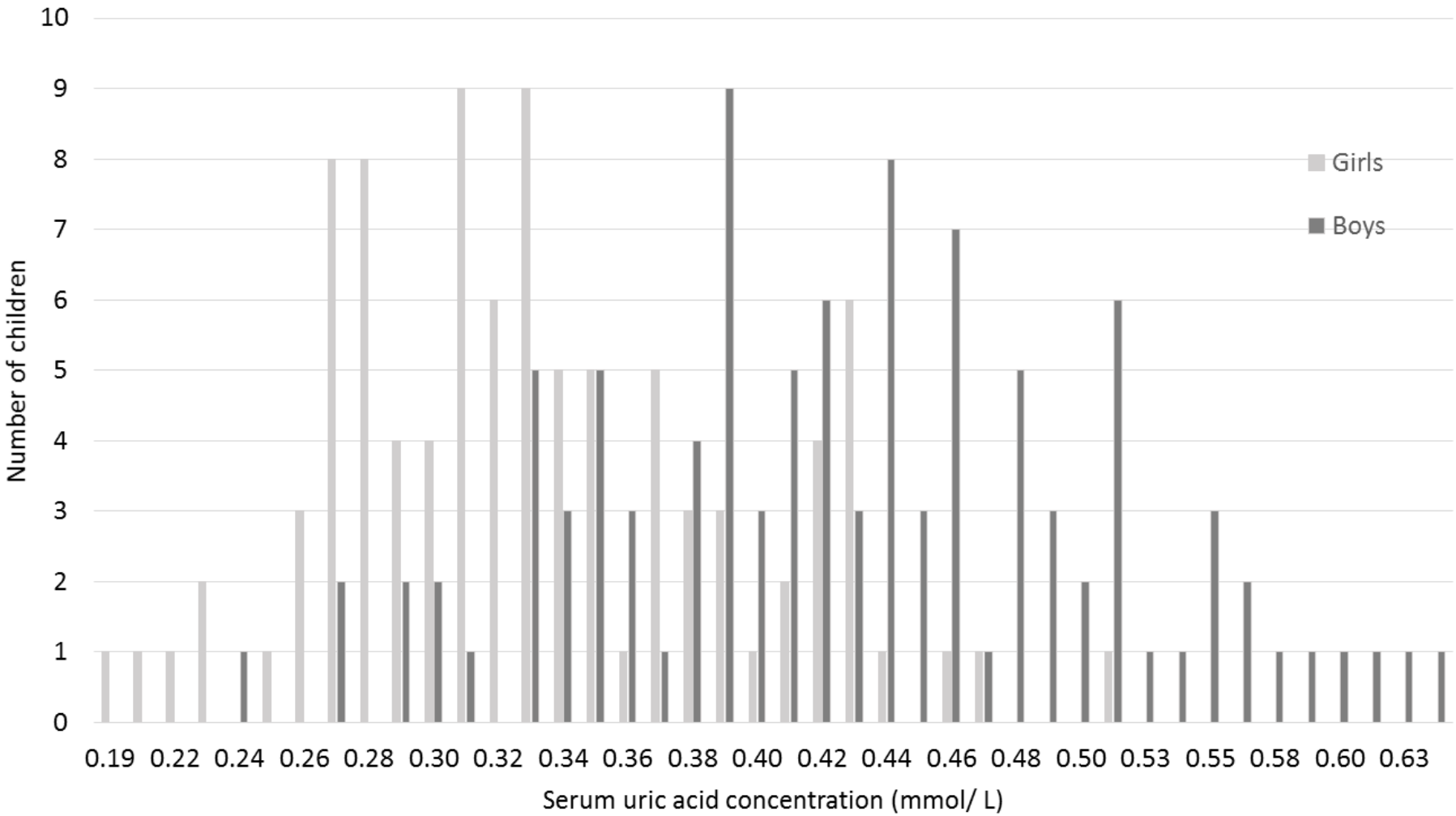


Figure 6.2. Frequency of serum uric acid by gender among Pacific children at 15 years old

6.3.2 Association between body measurements and biomarkers

Adjusted for gender and age HbA_1c was positively related to weight (r = 0.174 *p* = 0.014), BMI (r = 0.155 *p* = 0.029), waist (r = 0.165 *p* = 0.020), waist-to-height ratio (r = 0.150 *p* = 0.034), FM (r = 0.166 *p* = 0.019) and FFM (r = 0.153 *p* = 0.031), but not height or weight z score, height z score and BMI z score.

There was a positive association between SUA concentration and all the body measurements; weight z score (r = 0.453 *p* = 0.001), height z score (r = 0.197 *p* = 0.005) and BMI z score (r = 0.373 *p* = 0.001). Adjusted for gender and age SUA concentration was positively related to weight (r = 0.500 *p* = 0.001), height (r = 0.224 *p* = 0.002), BMI (r = 0.484 *p* = 0.001), waist (r = 0.502 *p* = 0.001), waist-to-height ratio (r = 0.478 *p* = 0.001), FM (r = 0.476 *p* = 0.001) and FFM (r = 0.454 *p* = 0.001).

6.3.3 Food consumption reported by the children at 15 years

More than 50% of the children reported consuming each of tomato sauce, chocolate coated or cream filled biscuits, biscuits, bars, crackers or crispbreads, chocolate, other sweets, juice, powdered fruit drink, Coca Cola or other cola drinks, Mountain Dew and soft drinks once a week or more. In addition 15% reported each of tomato sauce, chocolate-coated or cream filled biscuits, juice, powdered drinks, Coca Cola or other cola drinks, Mountain Dew and soft drinks ≥ 5 times a week (Table 6.5).

Overall 47% of girls and 60% of boys consumed sugary drinks twice or more a day. The mean of BMI z scores of these children (n = 111) was 2.02 (SD = 1.22) and only one quarter (26%) of them had normal weight and almost three quarter of them were overweight and obese (31% and 43 respectively). However, daily consumption of sugary drinks was not associated with either of BMI cut-offs (*p* = 0.403), BMI (*p* = 0.813) or weight (*p* = 0.069), using Chi square test.

In general, children who consumed > 5 times a day from the reported foods (minimum of one food item from each of the five food category) (n = 126), had a mean z score BMI of 2.00 (SD = 1.22) and less than 30% were normal weight (30% overweight and 41% obese).

Table 6.5. Frequency of food consumption by gender (girls = 100, boys = 104)

Frequency	**Never** [a] **n (%)**		**1- 3 a month n (%)**		**1- 2 a week n (%)**		**3- 4 a week n (%)**		**5- 6 a week n (%)**		**Once a day n (%)**		**2 ≥ times a day n (%)**	
	Girls	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**
Spreads and sauces														
Jam or honey	44 (44.0)	51 (49.0)	24 (24.0)	18 (17.3)	18 (18.0)	15 (14.4)	9 (9.0)	11 (10.6)	2 (2.0)	2 (1.9)	2 (2.0)	5 (4.8)	0 (0.0)	2 (1.9)
Nutella	31 (31.0)	29 (27.9)	37 (37.0)	28 (26.9)	19 (19.0)	20 (19.2)	6 (6.0)	10 (9.6)	2 (2.0)	7 (6.7)	1 (1.0)	7 (6.9)	4 (4.0)	3 (2.9)
Marmite or Vegemite	80 (80.0)	78 (75.0)	10 (10.0)	13 (12.5)	5 (5.0)	4 (3.8)	2 (2.0)	3 (2.9)	1 (1.0)	3 (2.9)	0 (0.0)	3 (2.9)	2 (2.0)	0 (0.0)
Peanut butter	55 (55.0)	42 (40.4)	18 (18.0)	24 (23.1)	13 (13.0)	28 (28.0)	9 (9.0)	10 (9.6)	0 (0.0)	3 (2.9)	4 (4.0)	7 (6.7)	1 (1.0)	1 (1.0)
Mayonnaise or salad dressing	16 (16.0)	24 (23.1)	33 (33.0)	32 (30.8)	28 (28)	23 (22.1)	11 (11.0)	8 (7.7)	7 (7.0)	10 (9.6)	5 (5.0)	5 (4.8)	0 (0.0)	2 (1.9)
Tomato sauce or ketchup	10 (10.0)	10 (9.6)	13 (13.0)	24 (23.1)	24 (24.0)	23 (22.1)	33 (33.0)	19 (18.3)	9 (9.0)	17 (16.3)	7 (7.0)	6 (5.8)	4 (4.0)	**5 (4.8)**
Other spreads or sauces [b]	1 (1.0)	0 (0.0)	1 (1.0)	4 (4.0)	5 (5.0)	1 (1.0)	2 (2.0)	1 (1.0)	1 (1.0)	3 (3.0)	4 (4.0)	2 (2.0)	0 (0.0)	3 (3.0)
Convenience meals/ snacks														
Canned spaghetti with tomato sauce	30 (30.0)	37 (35.6)	34 (34.0)	27 (26.0)	22 (22.0)	22 (21.2)	9 (9.0)	11 (10.6)	2 (2.0)	6 (5.8)	2 (2.0)	1 (1.0)	1 (1.0)	
Baked beans	66 (66.0)	70 (67.3)	22 (22.0)	20 (19.2)	7 (7.0)	7 (6.7)	1 (1.0)	3 (2.9)	4 (4.0)	2 (1.9)	0 (0.0)	2 (1.9)	0 (0.0)	

Frequency	Never [a] n (%)		1- 3 a month n (%)		1- 2 a week n (%)		3- 4 a week n (%)		5- 6 a week n (%)		Once a day n (%)		2 ≥ times a day n (%)	
	Girls	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**
Other Convenience meals/snacks [c]	4 (4.0)	1 (1.0)	10 (10.0)	17 (16.3)	14 (14.0)	16 (15.4)	18 (18.0)	18 (17.3)	6 (6.0)	10 (9.6)	5 (5.0)	4 (3.8)	2 (2.0)	6 (5.8)
Biscuits/ cakes														
Chocolate coated or cream filled biscuits	9 (9.0)	15 (14.4)	30 (30.0)	27 (26.0)	27 (27.0)	23 (22.1)	16 (16.0)	20 (19.2)	9 (9.0)	10 (9.6)	6 (6.0)	4 (3.8)	3 (3.0)	4 (3.8)
Biscuits e.g. plain	10 (10.0)	21 (20.2)	33 (33.0)	31 (29.8)	30 (30.0)	24 (23.1)	16 (16.0)	13 (12.5)	6 (6.0)	4 (3.8)	3 (3.0)	8 (7.7)	1 (1.0)	1 (1.9)
Bars e.g. muesli	26 (26.0)	24 (23.1)	26 (26.0)	22 (21.2)	20 (20.0)	19 (18.3)	15 (15.0)	20 (19.2)	5 (5.0)	9 (8.7)	5 (5.0)	4 (3.8)	2 (2.0)	5 (4.8)
Crackers or crispbreads	16 (16.0)	25 (24.0)	24 (24.0)	23 (22.1)	32 (32.0)	24 (23.1)	20 (20.0)	15 (14.4)	3 (3.0)	7 (6.7)	2 (2.0)	3 (2.9)	3 (3.0)	6 (5.8)
Cake or slice	20 (20.0)	28 (26.9)	53 (53.0)	45 (43.3)	14 (14.0)	17 (16.3)	10 (10.0)	6 (5.8)	1 (1.0)	3 (2.9)	2 (2.0)	3 (2.9)	0 (0.0)	1 (1.0)
Doughnuts or croissants	35 (35.0)	44 (42.3)	47 (47.0)	35 (33.7)	10 (10.0)	11 (10.6)	5 (5.0)	7 (6.7)	2 (2.0)	2 (1.9)	1 (1.0)	4 (3.8)	0 (0.0)	1 (1.0)
Scones, muffins or sweet buns	27 (27)	44 (42.3)	41 (41)	37 (35.6)	22 (22)	15 (14.4)	8 (8)	3 (2.9)	1 (1)	0 (0.0)	1 (1)	1 (1)	0 (0.0)	4 (3.8)
Pancake or pikelets	34 (34)	33 (31.7)	39 (39.0)	43 (41.3)	15 (15.0)	11 (10.6)	8 (8.0)	10 (9.6)	2 (2.0)	4 (3.8)	1 (1.0)	3 (2.9)	0 (0.0)	0 (0.0)
Fruit pie, fruit crumble or tart	84 (84.0)	76 (73.1)	10 (10.0)	21 (20.2)	4 (4.0)	3 (2.9)	0 (0.0)	1 (1.0)	1 (1.0)	0 (0.0)	1 (1.0)	3 (2.9)	0 (0.0)	0 (0.0)

Frequency	Never [a] n (%)		1- 3 a month n (%)		1- 2 a week n (%)		3- 4 a week n (%)		5- 6 a week n (%)		Once a day n (%)		2 ≥ times a day n (%)	
	Girls	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**
Pudding	70 (70.0)	65 (62.5)	17 (17.0)	28 (26.9)	10 (10.0)	3 (2.9)	0 (0.0)	0 (0.0)	2 (2.0)	1 (1.0)	1 (1.0)	3 (2.9)	0 (0.0)	2 (1.9)
Custard or custard puddings	73 (73.0)	73 (70.2)	19 (19.0)	22 (21.2)	6 (6.0)	3 (2.9)	1 (1.0)	2 (1.9)	0 (0.0)	1 (1.0)	1 (1.0)	1 (1.0)	0 (0.0)	1 (1.0)
Other item of the biscuits/ cakes [d]	2 (2.0)	1 (1.0)	3 (3.0)	3 (2.9)	1 (1.0)	3 (2.9)	0 (0.0)	2 (1.9)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Snacks and sweets														
Popcorn	26 (26)	36 (34.6)	47 (47)	41 (39.4)	16 (16)	16 (15.4)	5 (5)	4 (3.8)	1 (1)	6 (5.8)	3 (3)	1 (1)	2 (2)	0 (0.0)
Chocolate	12 (12)	20 (19.2)	32 (32)	29 (27.9)	28 (28)	27 (26)	13 (13)	18 (17.3)	4 (4)	5 (4.8)	5 (5)	4 (3.8)	6 (6)	1 (1)
Candy coated chocolate	13 (13)	28 (26.9)	36 (36)	31 (29.8)	28 (28)	24 (23.1)	9 (9)	12 (11.5)	5 (5)	5 (4.8)	3 (3)	3 (2.9)	5 (5)	1 (1)
Other sweets [e]	15 (15)	31 (29.8)	24 (24)	27 (26)	28 (28)	21 (20.2)	18 (18)	13 (12.5)	9 (9)	8 (7.7)	3 (3)	2 (1.9)	2 (2)	1 (1)
Sugary drinks														
Juice	15 (15)	15 (14.4)	35 (35)	24 (23.1)	22 (22)	23 (22.1)	17 (17)	20 (19.2)	6 (6)	12 (11.5)	4 (4)	9 (8.7)	1 (1)	1 (1)
Powdered fruit drink	20 (20)	19 (18.3)	29 (29)	26 (25)	20 (20)	19 (18.3)	17 (17)	19 (18.3)	8 (8)	6 (5.8)	5 (5)	8 (7.7)	1 (1)	6 (5.8)

Frequency	**Never [a] n (%)**		**1- 3 a month n (%)**		**1- 2 a week n (%)**		**3- 4 a week n (%)**		**5- 6 a week n (%)**		**Once a day n (%)**		**2 ≥ times a day n (%)**	
	Girls	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**
Fruit drink from concentrate or cordial	26 (26)	28 (26.9)	35 (35)	32 (30.8)	24 (24)	16 (15.4)	8 (8)	10 (9.6)	4 (4)	8 (7.7)	1 (1)	9 (8.7)	1 (1)	1 (1)
Coca cola or other cola drinks	27 (27)	17 (16.3)	24 (24)	25 (24)	21 (21)	25 (24)	12 (12)	17 (16.3)	6 (6)	11 (10.6)	6 (6)	5 (4.8)	4 (4)	4 (3.8)
Mountain Dew	27 (27)	24 (23.1)	33 (33)	18 (17.3)	19 (19)	28 (26.9)	7 (7)	13 (12.5)	6 (6)	13 (12.5)	5 (5)	4 (3.8)	3 (3)	3 (2.9)
‘New Age’ drinks	76 (76)	67 (64.4)	14 (14)	16 (15.4)	7 (7)	13 (12.5)	1 (1)	5 (4.8)	0 (0.0)	2 (1.9)	0 (0.0)	0 (0.0)	2 (2)	1 (1)
Soft drinks	13 (13)	10 (9.6)	25 (25)	28 (26.9)	33 (33)	30 (28.8)	16 (16)	15 (14.4)	8 (8)	9 (8.7)	3 (3)	7 (6.7)	2 (2)	5 (4.8)
Sports drinks	36 (36)	22 (21.2)	31 (31)	35 (33.7)	20 (20)	25 (24)	8 (8)	9 (8.7)	0 (0.0)	5 (4.8)	1 (1)	0 (0.0)	4 (4)	3 (2.9)
Ice blocks	27 (27)	38 (36.5)	36 (36)	33 (31.7)	20 (20)	15 (14.4)	10 (10)	9 (8.7)	2 (2)	2 (1.9)	5 (5)	6 (5.8)	0 (0.0)	1 (1)
Tea	37 (37)	46 (44.2)	22 (22)	9 (8.7)	15 (15)	20 (19.2)	14 (14)	11 (10.6)	8 (8)	5 (4.8)	3 (3)	9 (8.7)	1 (1)	3 (2.9)
Coffee	59 (59)	69 (66.3)	16 (16)	13 (12.5)	8 (8)	8 (7.7)	7 (7)	6 (5.8)	3 (3)	3 (2.9)	3 (3)	2 (1.9)	4 (4)	2 (1.9)
Other item of the ‘Other drinks’ [f]	1 (1)	3 (2.9)	4 (4)	7 (6.7)	8 (8)	5 (4.8)	7 (7)	4 (3.8)	4 (4)	6 (5.8)	2 (2)	3 (2.9)	2 (2)	0 (0.0)

[a] The responds including “less than a month”. [b] Eighty six girls and ninety boys were missed. [c] Forty one girls and thirty two boys were missed. [d] Ninety four girls and ninety five boys were missed. [e] One girl and one boy were missed. [f] Seventy two girls and seventy six boys were missed.

There were no significant differences in the frequency of food consumption between genders except for peanut butter, 'New age' drink, sports drink, scones, muffins or sweet buns, other sweet and 'sugary drinks' group. Boys consumed more peanut butter, 'New age' drink, sports drink and 'sugary drinks' group than the girls (Table 6.6). However, the consumption of scones, muffins, or sweet buns, other sweets and were less among the boys compared to the girls (Table 6.6).

Table 6.6. Daily frequency of consumed foods by gender

	Daily frequency [a] Median (percentile 25th, 75th)			
	Total n = 204	**Girls n = 100**	**Boys n =104**	***p* Value** [b]
Spreads and sauces				
Jam or honey	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.871
Nutella	0.01 (0.01, 0.01)	0.07 (0.01, 0.21)	0.07 (0.01, 0.50)	0.083
Marmite or Vegemite	0.07 (0.01, 0.21)	0.01 (0.01, 0.01)	0.01 (0.01, 0.05)	0.382
Peanut butter	0.07 (0.07, 0.21)	0.01 (0.01, 0.21)	0.07 (0.01, 0.21)	**0.040**
Mayonnaise or salad dressing	0.21 (0.07, 0.50)	0.21 (0.07, 0.21)	0.07 (0.07, 0.21)	0.531
Tomato sauce or ketchup	0.00 (0.00, 0.00)	0.50 (0.21, 0.50)	0.21 (0.07, 0.79)	0.528
Other spreads or sauces	0.07 (0.01, 0.21)	0.00 (0.00, 0.00)	0.00 (0.00, 0.00)	0.959
Total	1.07 (0.64, 2.05)	1.00 (0.63, 1.75)	1.08 (0.63, 2.40)	0.392
Convenience meals/snacks				
Canned spaghetti with tomato sauce	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.889
Baked beans	0.01 (0.01, 0.07)	0.01 (0.01, 0.07)	0.01 (0.01, 0.07)	0.939
Other Convenience meals/snacks	0.07 (0.00, 0.50)	0.07 (0.00, 0.50)	0.21 (0.00, 0.50)	0.129
Total	0.28 (0.08, 0.79)	0.22 (0.07, 0.64)	0.43 (0.07, 0.98)	0.271
Biscuits/ cakes				
Chocolate coated or cream filled biscuits	0.21 (0.07, 0.50)	0.21 (0.07, 0.5)	0.21 (0.07, 0.50)	0.694
Biscuits e.g. plain	0.21 (0.07, 0.50)	0.21 (0.07, 0.5)	0.07 (0.07, 0.50)	0.343
Bars e.g. muesli	0.21 (0.01, 0.50)	0.07 (0.01, 0.5)	0.21 (0.07, 0.50)	0.212
Crackers or crispbreads	0.21 (0.07, 0.50)	0.21 (0.07, 0.5)	0.21 (0.02, 0.50)	0.546

	Daily frequency [a] Median (percentile 25th, 75th)			
	Total n = 204	**Girls n = 100**	**Boys n =104**	***p* Value** [b]
Cake or slice	0.07 (0.07, 0.21)	0.07 (0.07, 0.21)	0.07 (0.01, 0.21)	0.566
Doughnuts or croissants	0.07 (0.01, 0.07)	0.07 (0.01, 0.07)	0.07 (0.01, 0.07)	0.859
Scones, muffins or sweet buns	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.07 (0.01, 0.07)	**0.027**
Pancake or pikelets	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.524
Fruit pie, fruit crumble or tart	0.01 (0.01, 0.01)	0.01 (0.01, 0.01)	0.01 (0.01, 0.07)	0.071
Pudding	0.01 (0.01, 0.07)	0.01 (0.01, 0.07)	0.01 (0.01, 0.07)	0.491
Custard or custard puddings	0.01 (0.01, 0.07)	0.01 (0.01, 0.07)	0.01 (0.01, 0.07)	0.632
Other item of the biscuits/ cakes	0.00 (0.00, 0.00)	0.00 (0.00, 0.00)	0.00 (0.00, 0.00)	0.429
Total	1.38 (0.84, 2.52)	1.35 (0.92, 2.22)	1.43 (0.64, 2.94)	0.987
Snacks and sweets				
Popcorn	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.342
Chocolate	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.340
Candy coated chocolate	0.07 (0.07, 0.21)	0.14 (0.07, 0.21)	0.07 (0.01, 0.21)	0.113
Other sweets	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.07 (0.01, 0.21)	**0.012**
Total	0.57 (0.26, 1.28)	0.67 (0.36, 1.20)	0.49 (0.23, 1.41)	0.065
Sugary drinks				
Juice	0.21 (0.07, 0.50)	0.14 (0.07, 0.50)	0.21 (0.07, 0.50)	0.058
Powdered fruit drink	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.346
Fruit drink from concentrate or cordial	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.07 (0.01, 0.50)	0.266
Coca cola or other cola drinks	0.21 (0.07, 0.50)	0.07 (0.01, 0.50)	0.21 (0.07, 0.50)	0.099
Mountain Dew	0.07 (0.01, 0.50)	0.07 (0.01, 0.21)	0.21 (0.07, 0.50)	0.066
‘New Age’ drinks	0.01 (0.01, 0.07)	0.01 (0.01, 0.01)	0.01 (0.01, 0.07)	**0.048**
Soft drinks	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.21 (0.07, 0.50)	0.344
Sports drinks	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.07 (0.07, 0.21)	**0.015**
Ice blocks	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.07 (0.01, 0.21)	0.275
Tea	0.07 (0.01, 0.50)	0.07 (0.01, 0.50)	0.07 (0.01, 0.50)	0.882
Coffee	0.01 (0.01, 0.07)	0.01 (0.01, 0.18)	0.01 (0.01, 0.07)	0.186

	Daily frequency [a] Median (percentile 25th, 75th)			
	Total n = 204	**Girls n = 100**	**Boys n =104**	***p* Value** [b]
Other item of the 'Other drinks'	0.00 (0.00, 0.07)	0.00 (0.00, 0.07)	0.00(0.00, 0.01)	0.746
Total	2.12 (1.13, 3.64)	1.89 (0.99, 3.03)	2.35 (1.44, 4.22)	**0.034**

[a] Frequency of foods consumed was assumed as frequency of consumption of a serving of that food
[b] Mann-Whitney U test.

Of all the listed thirty two food items in Table 6.7, only tea and coffee were sweetened with added sugar. The weekly median consumption of tomato sauce or ketchup, chocolate-coated or cream-filled biscuits, biscuits, bars, crackers or crispbreads, chocolate, juice, powdered fruit drink, Coca Cola or other cola drinks and soft drinks were ≥ 1 (Table 6.7).

Table 6.7. Number of times a week the foods with added sugar were consumed by Pacific children at age 15 years. Ranked by descending frequency of consumption

Food item	**Average number of times a week**	**Median (25th and 75th percentiles)**	**Portion size**	**g added sugars [a]/ serve**
Tomato sauce or ketchup	3.1	1.5 (0.5, 3.5)	65 g	4
Chocolate coated or cream filled biscuits	2.5	1.5 (0.5, 3.5)	4 biscuits	18
Biscuits	1.9	1.5 (0.5, 3.5)	4 arrowroot biscuits	8
Bars	2.2	1.5 (0.1, 3.5)	1 bar	5
Crackers or crispbreads	2.2	1.5 (0.5, 3.5)	1 cracker	3
Chocolate	2.2	1.5 (0.5, 3.5)	1 bar	41
Juice	2.2	1.5 (0.5, 3.5)	150 ml	11
Powdered fruit drink	2.4	1.5 (0.5, 3.5)	200 ml with water	30
Coca cola or other cola drinks	2.4	1.5 (0.5, 3.5)	1 can (355ml)	40
Soft drink	2.4	1.5 (0.5, 3.5)	1 cup	28
Nutella	1.8	0.5 (0.1, 1.5)	3 tsp [b]	9
Peanut butter	1.3	0.5 (0.1, 1.5)	3 tsp [b]	1
Mayonnaise or salad dressing	1.8	0.5 (0.5, 1.5)	1 tbs [c]	15

Food item	Average number of times a week	Median (25th and 75th percentiles)	Portion size	g added sugars [a]/ serve
Canned spaghetti with tomato sauce	1.3	0.5 (0.1, 1.5)	1 cup	14
Cake	1.1	0.5 (0.5, 1.5)	1 slice (90 g)	32
Doughnuts or croissants	1	0.5 (0.1, 0.5)	1 small doughnut	6
Scones, muffins or sweet buns	1.1	0.5 (0.1, 1.5)	1	1
Pancake or pikelets	1	0.5 (0.1, 1.5)	1	1
Popcorn	1.1	0.5 (0.1, 1.5)	1 cup	5
Candy coated chocolate	1.8	0.5 (0.5, 1.5)	10 pieces	6
Fruit drink from concentrate or cordial	1.6	0.5 (0.1, 1.5)	38 g	13
Mountain Dew	2.1	0.5 (0.1, 3.5)	240 g	30
Sports drink	1.7	0.5 (0.1, 1.5)	500 ml	34
Ice- block	1.4	0.5 (0.1, 1.5)	1 ice- block	14
Tea	1.9	0.5 (0.1, 3.5)	1 cup	0
Jam/ honey	1.2	0.5 (0.1, 0.5)	3 tsp [b]	16
Marmite or Vegemite	0.6	0.1 (0.1, 0.1)	1 tsp [b]	1
Baked beans	0.6	0.1 (0.1, 0.5)	1 cup	10
Fruit pie, fruit crumble or tart	0.4	0.1 (0.1, 0.1)	1 whole tart (60 g)	21
Pudding	0.6	0.1 (0.1, 0.5)	90 g	17
Custard or custard puddings	0.5	0.1 (0.1, 0.5)	170 g	18
New Age drink	0.7	0.1 (0.1, 0.5)	1 can Red Bull	27
Coffee	1.2	0.1 (0.1, 0.5)	1 cup	6

[a] Will include in addition to the added sugar, natural sugars for items that include fruit or milk. [b] tsp; teaspoon, [c] tbs; table spoon.

6.3.4 Associations between HbA_1c and food consumption

Data were explored visually by scatter plot and by correlation for relationships between either individual food items or predefined food groups and HbA_1c. Except a consumption positive association between HbA_1c and coffee ($r = 0.215$ $p = 0.002$) and ‘other drink’ ($r = 0.139$ $p = 0.049$) no other significant associations were found.

6.3.5 Associations between SUA concentration and food consumption

There was a positive association between SUA and consumption of Mountain Dew (r = 0.184 *p* = 0.009), New Age drink (r = 0.207 *p* = 0.003) and 'sugary drink' group (r = 0.169 *p* = 0.017), and negative association with 'other sweets' (r = -0.166 *p* = 0.019) and scones, muffins or sweet buns (r = -0.149 *p* = 0.036).

6.3.6 Regression and mediation analysis

The influence of consumption of food groups on HbA_1c and SUA concertation was explored by step wise regression. Only 'snacks and sweets' group and 'sugary drinks' group were negatively (standardised β -0.331 *p* = 0.001) and positively (standardised β 0.438 *p* = 0.001) related to SUA respectively (R^2 0.309) after adjusting for gender:

SUA concentration = 0.234 + (0.093 x Gender) – (0.009 x Frequency of daily consumption of snacks and sweets) + (0.006 x Frequency of daily consumption of 'sugary drinks' group)

SEE = 0.076
Adjusted R^2 = 0.299
Gender: 1 = female, 2 = male

However, no association was found with the HbA_1c.

Uric acid was positively related to body mass, in particular FFM (lean). The relationship between the exposure (dietary intake) and the outcome (biomarker of risk) and the mediator (FFM) was explored for all children and by gender (Figure 6.3, Figure 6.4 and Figure 6.5). Fat free mass met the criteria to be a mediator of the relationship between sugary drinks and SUA concentration for all three analyses. In other words, a change of intake of sugary drinks was associated with change in FFM.

Fat free mass was also associated with SUA concentration. However, the effect of the intake on sugary drinks was reduced when FFM is considered in the relationships.

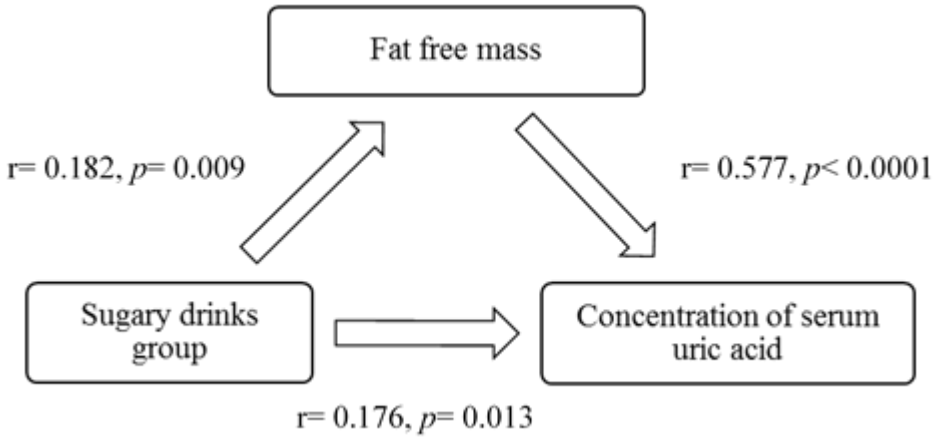


Figure 6.3. Mediation model including all Pacific children (n = 199)

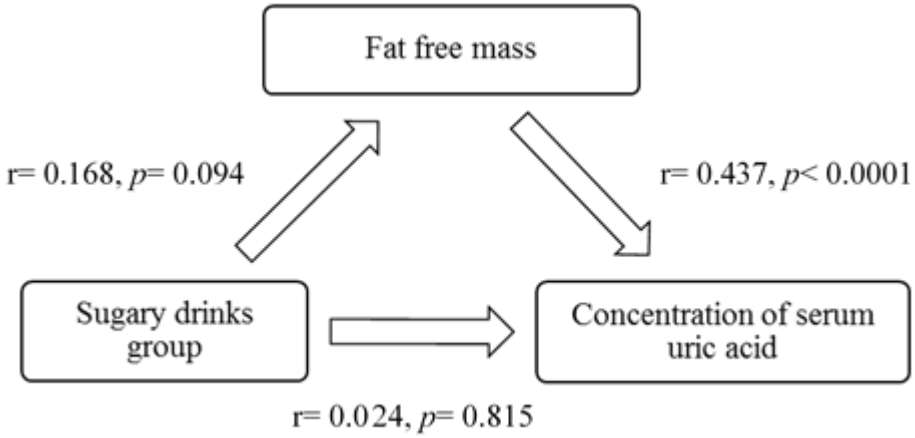


Figure 6.4. Mediation model including Pacific girls (n = 96)

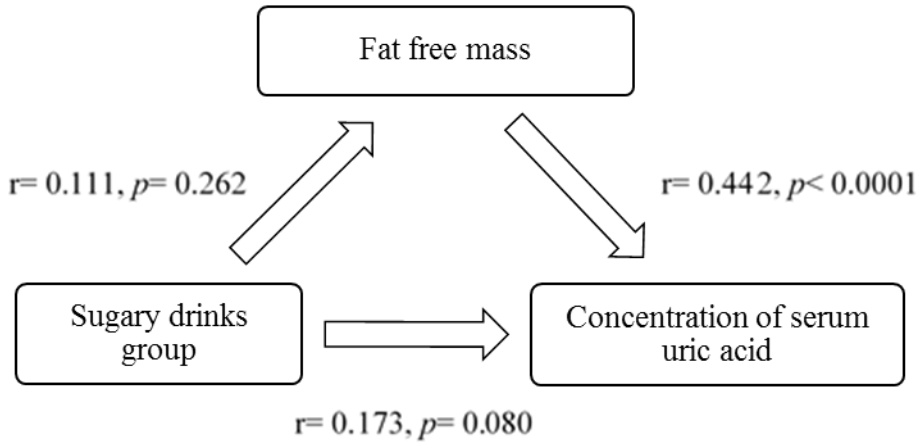


Figure 6.5. Mediation model including Pacific boys (n = 103)

6.3.7 Summary of main findings

Daily frequency of foods

- Boys significantly consumed more peanut butter, 'New age' drinks, sports drinks and 'sugary drinks' group than the girls. However, the consumption of 'scones, muffins or sweet buns' and other sweets was less among the boys compared to the girls.
- Tomato sauce or ketchup, chocolate-coated or cream-filled biscuits, soft drink, powdered fruit drink, Coca Cola or other cola drinks, bars, crackers or crispbreads, juice, chocolate and Mountain Dew were the eight most frequently consumed foods out of the thirty three foods with added sugar that were considered. The top 8 were each consumed on average of 2 or more times a week.

Food items and type 2 diabetes mellitus biomarkers

- After adjusting for gender only 'snacks and sweets' group and 'sugary drinks' group were negatively and positively related to SUA respectively ($R^2 = 0.309$), but no association was found with HbA_1c.

Body measurements and T2DM biomarkers

- On average boys weighed slightly more than the girls and had less FM (5kg) and more FFM (10 kg) than girls.
- The concentration of SUA was higher in boys compared to girls ($p = 0.001$).
- There was a positive and significant association between SUA and all the body measurements. However, HbA_1c was positively and significantly related to weight, BMI, waist circumference, waist-to-height ratio, FM, and FFM.

6.4 Discussion

For the first time, this 2015-2016 investigation has provided an account of added sugar and processed foods in relation to HbA_1c and SUA concentrations as T2DM biomarkers for a nested sample of a cohort of 15-year-old Pacific children. These children had a high rate of excess weight; two third were overweight and obese, and in the context of relatively high intake of daily free sugar where 47% of girls and 60% of boys consumed sugary drinks such as free sugars, twice or more a day. Relatively small but statistically significant associations of HbA_1c were found with body anthropometric and body composition (FM and FFM) measurements. No association of the frequency of daily consumption of sugary drink with HbA_1c was found. The frequency of consumption of the 'sugary drinks' group of drinks was positively related to the concentration of SUA, but when FFM is considered in the relationships as a mediator, the effect of the intake on sugary drinks is reduced.

The main focus of this investigation was foods with added sugar and risk of T2DM, so findings (Figure 6.6) will be discussed in relation to existing evidence, local and international, on the association sugary drinks consumption and SUA concentration. However, due to lack of studies with healthy children (n = 2) in which HbA_1c and SUA concentrations were considered as biomarkers of T2DM, findings from two studies on adults are included in the critique (Table 6.8).

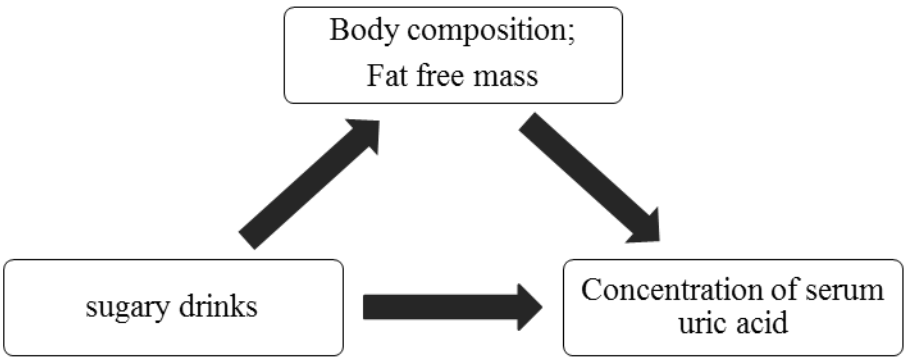


Figure 6.6. Factors found with significant associations

Table 6.8. Characteristics of the articles included in the present discussion

Reference	**Study design**	**Number and age in years of participants (child unless otherwise specified)**		**Measured biomarkers**	**Body measurements**	**Dietary assessment method**	**Food**
Present body of work	Cross sectional	204,	15	HbA_1c, SUA	Wt, Ht, Wc, and body composition by DEXA	FFQ	Sugar added foods
Gao et al. (2007)	Cross sectional	4073,	>18	SUA	Wt, Ht, BMI, BP	24-hour recall	Added sugar foods
Krishnan et al. (2012)	Prospective cohort	5012,	18-30	SUA, fasting glucose, biomarkers of insulin resistance, LDL	Wt, Ht, BMI, Wc, BP	-	-
Lin et al. (2016)	Cross sectional	1454,	12-16	Fasting plasma glucose, SUA, biomarkers of insulin resistance	Wt, Ht, Wc, Hc and body fat by BIA	FFQ	Sugar added drinks
de Miranda et al. (2015)	Cross sectional	245,	8-18	SUA, cholesterol, HDL, LDL, Triglycerides, glucose and insulin	Wt, Ht, Wc BP	-	-
Andersen et al. (2005)	Cross sectional	1650,	8-13	-	Wt, Ht, BMI	Food diary	Sugar sweetened soft drinks
Malik et al. (2006)	Review	children and adults		-	BMI		Sugar sweetened beverages including milk and juice
Rush et al. (2014)	Cross sectional	707,	4	-	-	FFQ	Added sugar foods including fruit and vegetables

Abbreviations: BIA; Bioelectrical Impedance Analysis; BMI, body mass index; BP, blood pressure; DEXA, dual X-ray absorptiometry; FFQ, food frequency questionnaire; Hc, hip circumference; HDL, high-density lipoprotein; Ht, height; LDL, low density lipoprotein. SEB, socioeconomic background; SD, sedentary behaviours; SUA, serum uric acid; Wc, waist circumference; Wt, weight; 24-h recall, 24 hour food recall.

The statistical relationship between sugary drinks and SUA concentration was present in total but not by gender. The findings of the National Health and Nutrition Examination Survey 2001–2002 (Gao et al., 2007) with US 2,085 women and 1,988 men aged >18 years, showed that men in highest intake quartile of estimated consumption of added sugars or sugar sweetened drinks had higher SUA concentration than those in the lowest intake quartiles (*P* for interaction <0.01). This association was not significant among women (Gao et al., 2007). They argued that sex hormones might moderate the metabolic response induced by fructose (Gao et al., 2007). Therefore, Gao and colleagues (2007) examined possible interactions of foods with added sugar as an ingredient such as bread, cake, soft drinks, jam, and ice cream (Cleveland, Cook, Krebs-Smith, & Friday, 1997), as well as those eaten separately with gender in relation to SUA. Foods with natural sugar such as fruit and milk were excluded (Cleveland et al., 1997). In this survey body composition, (FFM and FM) measurements were not considered.

In a study with 1,454 children aged 12-16 years in Taiwan, the association between SSBs including sweetened teas, soft drinks and fruit drinks consumptions with biomarkers of insulin resistance and fasting plasma glucose, within considering the effect of adiposity and SUA were determined (Lin et al., 2016). Lin and colleagues (2016) reported that the SSB consumption (>750 $mL.d^{-1}$) in children was associated with elevated insulin resistance and this relationship might be partially mediated by waist circumference (visceral adiposity), but not subcutaneous fat (body fat). The model they examined was adjusted for different cofactors including age, gender, ethnicity, physical activity, total calories and the consumption of meat and fruit. Based on their findings it was suggested that to a certain extent adverse consequences of fructose intake from SSB consumption might mediated by visceral adiposity (Lin et al., 2016). However, in the current study with Pacific children after adjusting for gender no association was found between any foods consumed, including sugary drinks, and HbA_1c.

Boys had more FFM and higher SUA concentration compared to girls. After adjusting for age and gender there was a positive significant association between SUA concentration with FM, FFM and anthropometric measurements including weight, height, BMI, waist and waist-to-height ratio. On the other hand, more than two third of the both girls and boys were overweight and obese. Krishnan and colleagues (2012) analysed the data on fifteen-year follow-up of 5,012 Americans aged 18-30 years at

enrolment to examine the utility of hyperuricemia as a biomarker for T2DM and IFG (prediabetes). At baseline (1986) the prevalence of obesity was 12% (Krishnan et al., 2012). During the follow-up period, people with greater SUA (0.42 $mmol.L^{-1}$) had higher incidence rates of T2DM (hazard ratios 1.87, 95% CI [1.33, 2.62]), insulin resistance (hazard ratios 1.36, 95% CI [1.23, 1.51]) and IFG (hazard ratios 1.25, 95% CI [1.04, 1.52]) (Krishnan et al., 2012). The authors concluded that hyperuricemia during mid-twenties can be an independent predicting factor for T2DM and prediabetes. In the current study, the prevalence of obesity among 14 year old Pacific children was higher (40% cf 12%) than the prevalence at 18-30 years reported by Krishnan et. al (2012). Moreover, 30% of girls and 55% of boys had SUA higher than the gender specific cut-off. This may increase the risk of developing T2DM among these children.

In another study, the association between SUA and insulin resistance was examined among children with obesity (mean BMI = 27.0 ± 4.5) (de Miranda et al., 2015). In this study all the measurements of obese children (n = 134) was compared with a control group (n = 111). The prevalence of insulin resistance was 26.9% and obese children had significantly ($p< 0.001$) higher anthropometric variables (BMI, waist circumference), systolic and diastolic blood pressure and biomarkers including total cholesterol, low and high density lipoproteins, triglycerides, glucose, insulin, homeostatic model assessment for insulin resistance and SUA (de Miranda et al., 2015). It was concluded that one uric acid unit increases by 91% the likelihood of insulin resistance (Odds ratio of uric acid was 1.91, $p< 0.001$, 95% CI [1.40, 2.62]) (de Miranda et al., 2015).

In the current study, there was no relationship between consumption of sugary drinks with BMI or weight or measures of overweight and obesity. Similarly, the results of a study with 3,139 Norwegian children aged 8-14 years measured between 1993 and 2000 showed no association between consumption of sweetened soft drinks measured by an 18-page pre-coded food diary and being overweight (based on Cole et al 2000) (Andersen et al., 2005). Notably, weight and height in Andersen et al.'s survey was self-reported and there was 18-19% missing data on weight and height of 2000 survey and 4% in 1993 survey.

However, findings from a review of thirteen cross- sectional studies in children showed a positive significant relationship between SSBs and excess weight gain in children (Malik et al., 2006) and the results of a meta-analysis of twelve studies showed that there was a significant positive association between SSBs intake and weight gain (kg) in

children (0.08; 95% CI [0.03, 0.13] (Malik, Willett, & Hu, 2009). Association is not a measure of causality and there could be other foods associated with the intake of SSBs that add to increased caloric intake and therefore more rapid weight gain in children.

Intake excessive amounts of sugars (predominantly sucrose) is a recognised risk factor of NCDs and through experimental studies it has been shown that high intakes of fructose (over 100g/ d) can reduce insulin sensitivity; although somewhat its high intake might also effect a raise in serum triglycerides (Macdonald, 2016). All the reported foods in this investigation had sugar added, except tea and coffee which adding sugar was optional. In addition, these sugar-added foods tend to be highly processed and low in protein, fibre, fruit and vegetable content, and therefore mostly energy-dense with poor nutrient profile.

The WHO recommends that free sugars should not provide more than 10% and ideally less than 5% of total energy intake in both adults and children (World Health Organization, 2015). Although the total energy intake of the children in the present study was not measured, the median number of times each day that sugar added foods were consumed was 5.9 (IQR: 3.8, 10.2). The NZ eating and activity guidelines include the statement ‘choose and/ or prepare foods and drinks with little or no added sugar’ and ‘that are mostly whole and less processed’ (Ministry of Health, 2015b, p. 6). The rationale is that consuming foods with added sugar easily provides more energy than required as they have high palatability and low satiety (Bellisle, Drewnowski, Anderson, Westerterp-Plantenga, & Martin, 2012). In addition, sugar intake is associated with dental caries (Moynihan & Kelly, 2014).

Based on the CNS 2002 (Ministry of Health, 2003) the daily median intake of sucrose among girls and boys aged 5 to 14 years were 61.1 g and 67.0 g respectively. However, in these children -who completed a 24-hour food recall- generally the average intake of sucrose was 69.3 g/ day. More than one-quarter of the daily sucrose in CNS 2002 came from beverages (including all teas, coffee and substitutes, Milo, juices, cordial, soft drinks, water, powdered drinks, sports, and energy drinks) and one-fifth from ‘sugar and sweets’ (sugars, syrups, confectionery, chocolate, jam, honey, jelly, sweet toppings and icing, ice-blocks, artificial sweeteners) (Ministry of Health, 2003). In contrast, Pacific children in current study had more sucrose from solid foods listed in Table 6.7 such as biscuits, chocolate, jam or ice-blocks than from beverages (38.0 g/ day, 55%) such as sports drinks, juices or soft drinks, than beverages (31.3 g/ day, 45%).

Rush and colleagues (2014) reported the average number of times per week of consumption of added sugar foods among PIF children at the age of four. Consumption of the majority of the foods at these ages were the same except for bread and foods with milk (e.g., flavored milk and ice-cream). The daily median number of times foods containing sugar were consumed was 5.8 (IQR: 4.0, 8.6) (Rush, Savila, et al., 2014). For the current analysis among fourteen years old Pacific children the consumption of some of the foods (e.g. flavored milk and ice-cream) was not included as it was when the PIF children were four years old. Thus, with considering those foods the daily median of sugary food consumption will be raised to 7.4 (IQR: 5.1, 12.2). Many sugar containing food items would not be classified as everyday foods, because of their nutrition profile and added sugar (Heart Foundation, 2018). Moreover, most of these foods were sourced from outside home (shop, diaries, takeaways, tuckshops and canteens) and Pacific children aged 11-14 years sourced a greater portion of their food from outside the home than younger Pacific children (Ministry of Health, 2003). Therefore, Pacific children should be supported in making wise food choices. On the hand, attention must be paid to providing access to healthy choices within school environment.

6.5 Strengths and limitations

For the first time the SUA concentration of Pacific children beside HbA_1c was measured to identify the risk of T2DM in an ethnicity with high rate of SUA and T2DM. The focus of the used FFQ was on NZ foods with added sugar.

Due to the used dietary method assessment (FFQ), the portions of consumed food items were not recorded; as a result, the total energy intake could not be evaluated. This could be used in adjusting the regression models to give more precious. However, FFQ could capture more variety of foods consumed compared to a single 24-hour recall.

Sugary drinks in this analysis included fruit juice, and not sweetened milk drinks or dairy products. These were consumed by most less than once a week, which may be related to the higher costs of these foods.

The results of this study cannot be conclusive as it was a cross sectional investigation. Longer and retrospective follow-up of these children would provide a stronger outcome.

6.6 Recommendations

Based on the findings of this study the consumption of sugary drinks was related to SUA concentration and among boys this association was mediated by FFM. Further

investigations are required to confirm causality of the found associations and the observed difference by gender.

In addition, more sedentary activities and leisure time mean that less energy is required yet energy intake increases because of sugar added to foods without adding much to nutrient density of the diet (Rush, Savila, et al., 2014). Therefore, the first suggestion is to encourage children to drink more water than SSB including fruit juice and replace the sugar added foods with the ones with less amount of sugar. Secondly, including more physical activities in everyday life will contribute to a more balanced energy intake.

In addition, the high intake of sugary drinks is of concern – not just at a critical period of growth, but at an age when it is a transition to adulthood. This is because at this age children experience variety of changes such as changes in social, home and school/ work environments, which can influence their diet and eating behaviours (Winpenny, Penney, Corder, White, & van Sluijs, 2017). Therefore, it is important to improve food security for communities and look at marketing, access, and supply of sugary drinks to vulnerable children. One action that has been started in Auckland is the provision of drinking fountains in public places and ensuring to locate them at every kilometre along popular walkway circuits (Auckland Council, 2018).

In addition, gender differences in eating patterns and food literacy (e.g., the greater consumption of sugary drinks by boys compared with girls) within cultural contexts need further exploration.

6.7 Conclusion

Recognition of high SUA as a biomarker for T2DM has been a matter of debate for some time and the consensus on its mechanism and aetiology, developmental pathway is still unresolved. The current study, however added earlier-in-life evidence to 1) the time course of relatively high SUA concentrations in Pacific, 2) the positive relationship with FFM and body mass, and 3) also the relationship with free sugar.

Chapter 7 9-year Metformin in Gestational Diabetes the Follow-up Study

7.1 Introduction

The current rise in the prevalence of T2DM among all ages reveals that positive family history is not the only reason for the incidence of this disease (Vrachnis et al., 2012). Its risk is a combination of early prenatal (e.g., gestational diabetes mellitus) and postnatal factors (e.g., obesity, poor physical activity and inappropriate food intake), that affect the epigenetic influences on growth and development of organs such as the pancreas (Vrachnis et al., 2012). Early epigenetic effects, such as exposure to excess blood glucose in the intrauterine environment, play a key role in both development of the disease and its course in the offspring (Darnton-Hill et al., 2004; Vrachnis et al., 2012) but the evidence mainly comes from observational and animal studies and the evidence from randomized controlled trial (RCT) studies is weak.

Gestational diabetes mellitus (GDM) or diabetes with the onset occurring during pregnancy (Ministry of Health, 2014d) means that the foetus is exposed to hyperglycaemia (Battista, Hivert, Duval, & Baillargeon, 2011). The diagnosis of GDM is staged. Firstly, if the HbA_1c of the pregnant women (ideally tested before 20 weeks' gestation), is ≥ 50 $mmol.mol^{-1}$ then immediate referral to a diabetes in pregnancy service with specialist physicians should occur. If the HbA_1c values are in the range of 41– 49 $mmol.mol^{-1}$, the diagnostic oral glucose tolerance test at 24–28 weeks will be offered, because these women are at an increased risk of GDM (Ministry of Health, 2014d). At 24-28 weeks all other women should be offered screening for GDM using the one hour, 50g oral glucose tolerance (challenge) test. If one hour glucose is ≥ 7.8 – 11.0 mol. L^{-1} then a 75g two hour oral glucose tolerance test should be undertaken. If fasting glucose is ≥5.5 $mol.L^{-1}$ or 2 hour value is > 9.0 $mmol.L^{-1}$ then referral for GDM also occurs (Ministry of Health, 2014d).

In NZ, approximately 8-9% of pregnant women are diagnosed with GDM (Ministry of Health, 2014d). In terms of ethnicity, in 2012, the percentages of women diagnosed with GDM in 2012 were: European 3%, Asian 8.1%, Pacific 7.2%, and Middle Eastern, Latin American and African 7.5% (Ministry of Health, 2014d). However, for Māori women there were less diagnosed cases (median 3.3%), which may be due to fewer pregnant Māori women screened rather than a lower GDM prevalence in Māori people

(Ministry of Health, 2014d). It should be noted that even when testing is undertaken the prevalence of GDM depends on the screening and diagnostic criteria applied.

Gestational diabetes is not only associated with increased future risk for the mother (such as T2DM, metabolic syndrome and cardiovascular disorders) (Damm, 2009; Damm et al., 2016; Harreiter, Dovjak, & Kautzky-Willer, 2014; Malcolm, 2012), but it also affects foetal growth and the long-term growth, development and health of the offspring by increasing the risk for developing a variety of diseases such as respiratory distress (Hay, 2012), T2DM, and excess weight gain (Damm et al., 2016; Malcolm, 2012). In a systematic review and meta-analysis of 20 follow-up studies by Bellamy, Casas, Hingorani and William (2009) it was reported that women with GDM are seven times more likely (95% CI [4.79, 11.51]) and children four times more likely to develop T2DM in later life compared to those who had normoglycaemic pregnancy.

Catalano and colleagues (2003) measured the body composition of 195 infants of women with GDM and 220 infants of women with normal glucose tolerance by anthropometry and total body electrical conductivity. They concluded that offspring of women with GDM, who were even average weight for gestational age, had greater (p = 0.002) fat mass (436 ± 206 g) compared with infants of women with normal glucose tolerance (362 ± 198 g). Therefore, offspring of women with GDM have a higher risk of childhood obesity than those were born to women without GDM (Catalano et al., 2003).

Early life body composition and size have implications for the development of later-life obesity as well as insulin resistance and the development of type 2 diabetes. It has been reported that Danish children have an 8-fold risk of diabetes or prediabetes at the age of 19-27 years (Damm, 2009) if their mothers had GDM. In addition is the concern for those women who are not diagnosed and therefore cannot be treated. Two generations are affected by GDM and it is a part of a positive loop, which increases the risk of diabetes in subsequent generations.

Moreover, since 30 years ago there is an accepted concept proposed by Freinkel that the inter-uterine environment has consequences for later life (Malcolm, 2012). Therefore, offspring who were exposed to a diabetic intrauterine environment have a higher prevalence of developing diabetes in future (almost 40%) compared to the ones with a genetic predisposition, but not the diabetic environment (Vrachnis et al., 2012). Besides, animal studies (e.g., Aerts & Van Assche, 2006; Harder et al., 2001), there are two studies (Gillman et al., 2010; Landon et al., 2015) that are following up children aged

from four to ten years, who were born to mothers with GDM (treated or untreated). In both studies, BMI was used as a primary outcome to assess obesity among these children and regardless of whether maternal GDM was treated or untreated, no significant difference was found in BMI of the offspring. The Danish National Birth Cohort was the other study in which the BMI of children was an indicator of childhood obesity. This study examined the children with intrauterine exposure to maternal hyperglycaemia. In this cohort 101,042 pregnancies were included and 1379 of them were identified with GDM (Zhu et al., 2016). Children's health and development including height and weight at 6 months, 18 months and 7 years were followed through health records and questionnaires sent to the parents (Zhu et al., 2016). It was shown that after adjusting for maternal pregnancy BMI, among women with GDM maternal fasting plasma glucose was associated in 661 offspring with birth weight (ß = 0.46; 95% CI [0.14, 0.78 per 1 $mmol.L^{-1}$ increase) and overweight including obesity at the age of 7 years (RR = 1.21; 95% CI [1.01, 1.50]) (Zhu et al., 2016). There were no measurements of the diet of mothers or their children in this follow-up study.

On the other hand, there is both common and intergenerational knowledge about the importance of diet for health (Michels, 2003), and that having nutrient-poor and, energy-rich diets is a major contributor to the development of obesity and T2DM (Ley et al., 2014). Evidence from prospective observational studies and randomised controlled trails goes back over the past twenty years (Ley et al., 2014). Food consumption and diet is a modifiable risk factor for T2DM. However, when it comes to the diet of children, the shaped eating behaviours during early life may persist into adulthood (Leech et al., 2014). There are examples of studies such as the NZ birth cohort (Wall et al., 2013) that derived eating patterns of children at two different periods (at 3.5 years and 7.5 years) and found only moderate correlations (r~ 0.35) eating pattern scores between the two time periods. The Pacific Islands Families study reported that quantities of specific foods such as fruit and vegetables consumed by Pacific children at ages four and six were moderately similar (r~ 0.7) (Savila et al., 2014) but reduced between 4 and 6 years.

The Metformin in Gestational Diabetes the Offspring Follow-Up study (MiGTOFU) that provided data for the present study, follows up at 9 years of age a birth cohort randomised trial; Metformin in Gestational Diabetes (MiG). Women (436 from Auckland, NZ and 181 from Adelaide, South Australia) diagnosed with GDM were randomised to receive either metformin or insulin as a treatment. Perinatal and maternal

outcomes (Rowan, Hague, Gao, Battin, & Moore, 2008) and maternal and neonatal circulating markers of metabolic and cardiovascular risk (Barrett et al., 2013) have been reported with very little difference between the treatments of metformin and insulin. The children were followed up with measures of body composition by anthropometry and DEXA (Rowan et al., 2011), food consumption by FFQ and 24-hour food recall and physical activity recall (Bristow, 2010) at age 2 years and minimal differences were found by treatment. More than 25% of the two-year-olds did not consume the minimum of 2 servings of either fruit or vegetables daily and treat foods and sweet drinks were consumed by two thirds of the children at least once a day.

In the current study, named “9-year MiGTOFU” the aim was to examine information on food consumption, HbA_1c, body measurements, and early life factors (e.g., birth weight) of these children were examined (Figure 7.1) to answer the questions below:

1. What are the eating patterns among children at the age of 7-9 years?
2. What are the associations between the derived eating patterns and HbA_1c?
3. What are the associations between the derived eating patterns and current factors including body size measurements?
4. What are the associations between the derived eating patterns and early life factors including birth weight, type of baby’s feeding, mothers’ education and age at conception?
5. What are the associations between early life factors, derived eating patterns, and current HbA_1c in this population?

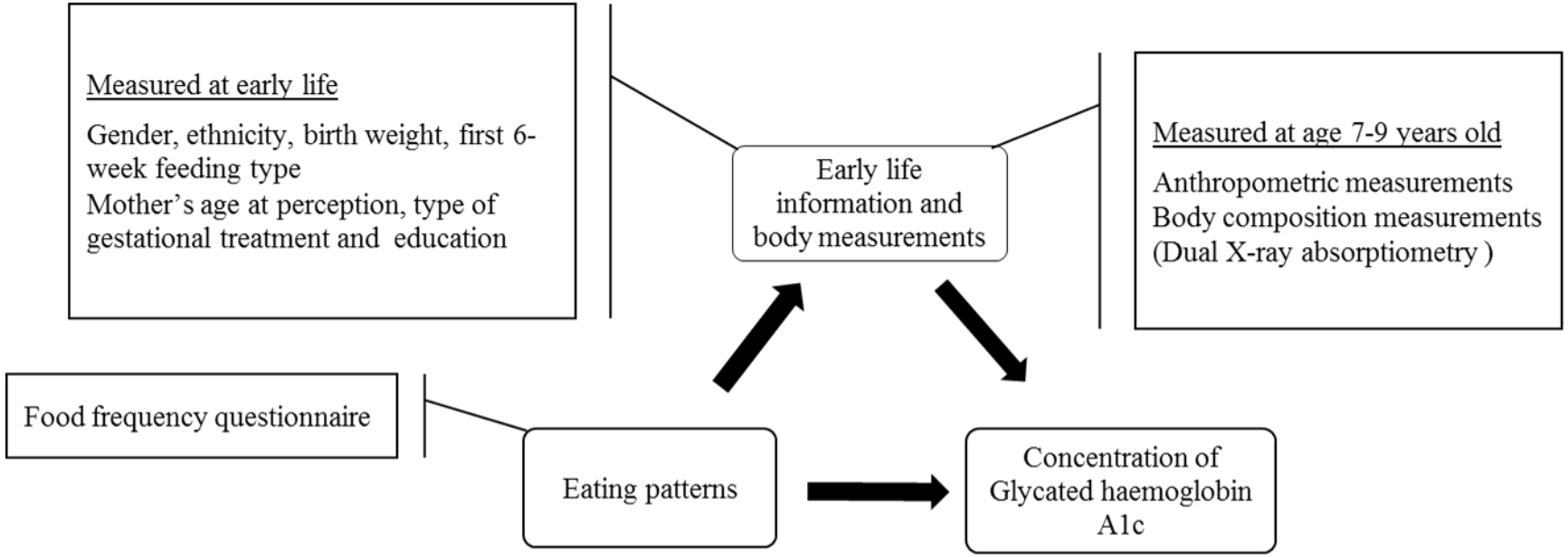


Figure 7.1. Investigation plan and variables considered for analysis

7.2 Methods

This chapter presents a brief overview of the 9-year MiGTOFU design and methods used to collect information on food consumption, concentration of HbA_1c, body measurements of the 9-year-old children and the early life factors including birth weight, type of babies' feeding, maternal age at conception and maternal education from the birth dataset (Table 7.1). This is followed by a description of data derivation grouping, transformation, and statistical analysis.

Table 7.1. Summary of variables for 9-year MiGTOFU [a]

Variables	**Measured at birth**	**Measured at 2 years**	**Measured at 9 years**
Consumed foods			X
HbA_1c			X
Covariates			
Sociodemographic information:			
Maternal age at conception	X		
Maternal education	X		
Breast feeding		X	
Birth weight	X		
Ethnicity	X		
Body measurements:			
Weight	X		X
Height	X		X
Waist circumference			X
Fat distribution (fat mass and fat free mass)			X

[a] Metformin in Gestational Diabetes the Follow-up study

7.2.1 Study participants

Three hundred and ninety six Auckland women were recruited to the MiG trial during pregnancy and the offspring of that pregnancy. It was budgeted to see 100 offspring at the age of 9 years at Auckland City Hospital for further follow-up measures. This follow-up study was powered to show a significance difference related to the effects of the treatment of metformin versus insulin on body composition. It was determined that 74 children were required. This study of eating patterns was exploratory, opportunistic and descriptive and would provide data to determine the number of participants needed in future studies.

7.2.2 Ethical considerations

The Offspring Follow-up (TOFU) study funded by the Auckland Medical Research Foundation is registered with the Australasian Clinical Trials Registry (ACTRN12605000311651) and the Auckland arm has local ethics approval (Auckland AKX/04/08/228/AM04) (Appendix H). When the child was 7-9 years old the information sheet and consent form regarding participation in MiGTOFU was sent to women who agreed when contacted by telephone for a further follow-up. When the child (fasting) and parents reported to Auckland City Hospital the study was verbally explained and written informed consent obtained from the mother/guardian of the child. Children then provided the researchers with verbal consent before each procedure including the blood sampling. After obtaining the consent from parents, a number of the investigations performed at the MiGTOFU two-year assessment were repeated in the 9-year-MiGTOFU on ninety-nine children at the body composition unit, Department of Surgery, University of Auckland. The visits were arranged on three or four mornings a month, planning to see six or seven children were measured each month. The mother completed, assisted as necessary, the same food frequency questionnaire questions as at age two years.

7.2.3 Sociodemographic information

Sociodemographic information (e.g. mothers' education, ethnicity and type of feeding the baby) were recorded at birth and two years through questionnaire. Child ethnicity information was provided by the mother.

7.2.4 Body measurements

Body measurements were performed at the University of Auckland's Body Composition Laboratory at Auckland City Hospital. The method for each measurement was based on those used in two-year-old follow-up phase (Rowan et al., 2011), and based on NZ Children's Nutrition Survey (CNS 2002) (Ministry of Health, 2003). In addition, the personnel were trained by the same person (Professor Elaine Rush), who trained the researchers for the MiG and MiGTOFU 2 year old studies. A standard protocol was followed as prescribed in the study manual.

Anthropometry measurements

Anthropometry measurements of children included weight recorded to the nearest 0.1kg, height to 0.1cm, and waist circumferences to 0.1 cm. All measurements were repeated at least twice within a given tolerance and the average calculated. A further measurement was made if the difference in measures was more than 0.5 cm (height and waist circumference) or 0.5 kg (weight). In this case, the average of the two closest measures was calculated and used in the analysis.

The BMI of each child was calculated using the following metric formula:

BMI = (weight in kilograms) divided by (height in meters x height in meters).

The Centres for Disease Control (CDC) z scores (Kuczmarski et al., 2000) were derived from BMI, age, and gender and the International Obesity Task Force cut-off criteria (Cole & Lobstein, 2012) applied to categorise as thin, normal, overweight, and obese.

Weight was measured with standing SECA scale (model 703 1321008, Germany) and height (Seca 206, Hamburg, Germany). An Anthropometric Measuring Tape was used to measure the waist circumference.

Body composition measurements

Body composition was measured using dual energy x-ray absorptiometry (DEXA) for body fatness. Whole body scan of the child was performed using a GE-Lunar iDXA (software version 15).

As well as total fat, lean soft tissue, and bone mineral content, visceral abdominal fat was analysed for regional fat content using enCORE software version 13.6. This method was previously used in adults (Kaul et al., 2012). In addition, total fat free mass

was calculated from total body mass less fat mass. The DEXA scan was performed and interpreted by a trained person.

7.2.5 Fasting venous blood sample

The children had a blood sample taken by a trained phlebotomist to examine biomarkers of risk for chronic disease including HbA_1c. Some of the samples were centrifuged and the plasma stored. On the same day, the whole blood sample was analysed by LabPlus, an accredited medical laboratory at Auckland City Hospital, for HbA_1c. The concentration of HbA_1c was measured using the boronate-affinity HPLC assay on a Trinity Biotech Analyser Model CLC385 (Primus Corp., Kansas City, MO). If a child had eaten, investigations were performed and the phrase “non-fasting status” was documented on the form.

This investigation used the cut-off points recommended by the New Zealand Society for the Study of Diabetes Working Party for adults. They classify ≤ 40 $mmol.mol^{-1}$ (5.8%) as normal glucose tolerance, values of 41–49 $mmol.mol^{-1}$ (5.9–6.6%) as pre-diabetes or dysglycaemia, and values of ≥ 50 $mmol.mol^{-1}$ (6.7%) as a diagnosis of diabetes (Braatvedt et al., 2012).

7.2.6 Food information

The semi-abbreviated food frequency questionnaire (FFQ) which was also used at age 2, had been developed with the assistance of a paediatric dietician and was based on the results of the child nutrition survey pilot study (Bristow, 2010). This FFQ was based on the most commonly consumed foods with account taken of ethnic foods rather than to quantify total energy and nutrients intake from all foods eaten (Bristow, 2010), so it appeared as an acceptable tool for collecting key food information of the children at 9 years (Appendix I).

In the FFQ at the age of nine years, the frequency of consuming thirty eight food items over a period of month was asked (Appendix I). Among common foods (e.g., milk, bread, juice, and etc.), some ethnic-specific foods were included, such as chapatti (or roti, a popular Indian staple made of whole wheat flour), taro (a starchy root crop which is consumed more by people from Pacific Islands), kumara (also called sweet potato brought to NZ by Māori people) and dumplings (a dish that consists of small pieces of dough made from a variety of starch sources and often wrapped around a filling that is eaten by Chinese. They might be cooked by boiling, frying, simmering, or steaming.

The trained research staff assisted the mothers to complete the questionnaires accurately when the mother and child attended for the assessments of body composition. For each food item (38 food items) in the questionnaire it has been asked “how often does the child eat or drink?” and “how many times?”. For each item in the list, if it was eaten/drunk never or less than once a month, the response was “never”. If it was eaten/drunk 1-3 times a month, the possible response was “monthly”, 1-6 times/week, “weekly” and at least once a day, was “daily”, respectively. Then, in the next column the respondent circled how many times the item was eaten/drunk each month/week or day, and the possible reply ranged from 1-7.

For the consumed food, ‘frequency per day’ rather than the ‘serving per day’ are presented, because the amount of food was not quantified. Foods consumed were recorded based on the child’s current (average) diet and how often foods were consumed monthly, weekly or daily. The frequency of consumption was multiplied by the fraction per day (Table 7.2) and reported as times/ day to allow a consistency in analysis.

Table 7.2. Frequency of consumption of food and the applied weighting factor to standardise to a daily rate

Frequency of consumption	**Weighting factor /day**
Monthly	1/30 x (how many times)
Weekly	1/7 x (how many times)
Daily	1 x (how many times)

To compare the food consumption of the children in this study with the Ministry of Health food and nutrition guidelines for children aged 2 to 18 years, (Ministry of Health, 2012a) (Appendix J) the thirty six food items, water and tea/ coffee were excluded, were grouped into five food groups; fruit, vegetables, milk and dairy products, meat and other protein, and bread and cereals by summing the total daily frequency of intake of each food by the children (

Table 7.3). In addition, the author created ‘sugary drink’ and ‘high energy food’ groups to determine the amount of high-fat and high-sugar foods children were consuming.

Table 7.3. Food grouping and the food items in each group

Food group	Food items
Fruit	Fresh fruit and tinned/frozen fruit
Vegetables	Fresh vegetables, tinned/ frozen vegetables, taro, kumara and mashed/boiled/ jacket potatoes
Breads and cereals	White breads, other types of breads, sweetened cereals [a], plain cereals [a], dumplings, pasta, noodles, rice and chapatti/roti
Milk and dairy products	Milk, cheese and yogurt
Meat and other protein	Red meat, chicken/ pork, sausage/ bacon, fish, eggs, and lentils/ kidney beans/ soy beans
Sugary drinks	Juice, fizzy/ energy drink, cordial/ powdered fruit drink and food drink
High energy	Roasted/ chips potatoes, chippies/ crisps, crackers, sweet biscuits, lollies/ sweeties, chocolate and takeaways

[a] breakfast cereals

7.2.7 Glycemic index and glycemic load of individual foods

The glycemic index (GI) of individual foods from the FFQ (n = 38) was checked through the GI database provided by the University of Sydney (http://www.glycemicindex.com/foodSearch.php) (Table 7.4). Foods containing little or no carbohydrate (such as meat, fish, eggs, and most vegetables) cannot have a GI value. There are three ratings for GI: low = GI value ≤ 55 , medium = GI value of 56 – 69 inclusive and high = GI ≥ 70 (Glycemic Index Foundation, 2016).

Table 7.4. Foods grouped based on the glycaemic index (GI)

GI level	Food Items
Low GI food	water, juice, tea/ coffee, food drink, milk, cheese, yogurt , chippies/ crisps, dumplings, noodles, lollies/ sweeties, chocolate, takeaways, fresh fruit, tinned/ frozen fruit, fresh vegetables/ salad, tinned/ frozen vegetables, chicken/ pork, red meat, sausage/ bacon, fish, eggs, lentils/ kidney beans/ soy beans
Medium GI food	fizzy drink/ energy drink, cordial/ fruit drink, potatoes roasted/ chips, taro, pasta, chapatti/ roti, crackers, sweet biscuits, takeaways
High GI food	white bread, other types of bread, sweetened cereals [a], cereals [a], mashed/ boiled/ jacket potatoes, kumara and rice

[a] breakfast cereals

The limitation of classifying foods by GI is that it does not take into account the effect of quantity of the food eaten or for example, the foods that are eaten together and that effect on glycaemia. It does however have a relationship to glycaemia and was considered as part of the holistic assessment of foods.

7.2.8 Statistical analysis

Food questionnaire, $HbA_{1}c$, body measurements, birth and early life information of the children including birth weight, type of feeding, number of siblings, and maternal information (mother's age at conception and maternal education) were entered into an SPSS 24 spreadsheet (SPSS Inc., New York, USA). Statistical significance was set at < 0.05 (5%).

Continuous variables from the above list were explored for descriptive data and distribution. Normally distributed variables (only height) are described as mean ± SD, while non-normally distributed are described as median with IQR and range.

Categorical variables including gender, ethnicity, infant feeding 6-8 weeks postpartum, Mother's type of treatment, eating patterns are reported as frequency and percentage. Chi-square test was applied to determine the difference in proportions of children between derived eating patterns. Stepwise linear regression explored relationships among eating patterns and related factors with a p of >0.10 for exclusion of the factor.

Step-by- step analysis of eating patterns and adjustments

Similarly to the investigation of the PIF children at age 14 years (Chapter 5) cluster analysis and the K-means algorithms were applied to identify groups of children with distinct eating patterns (Figure 7.2). Factor analysis and scree plots were applied to identify the optimal number of patterns (clusters).

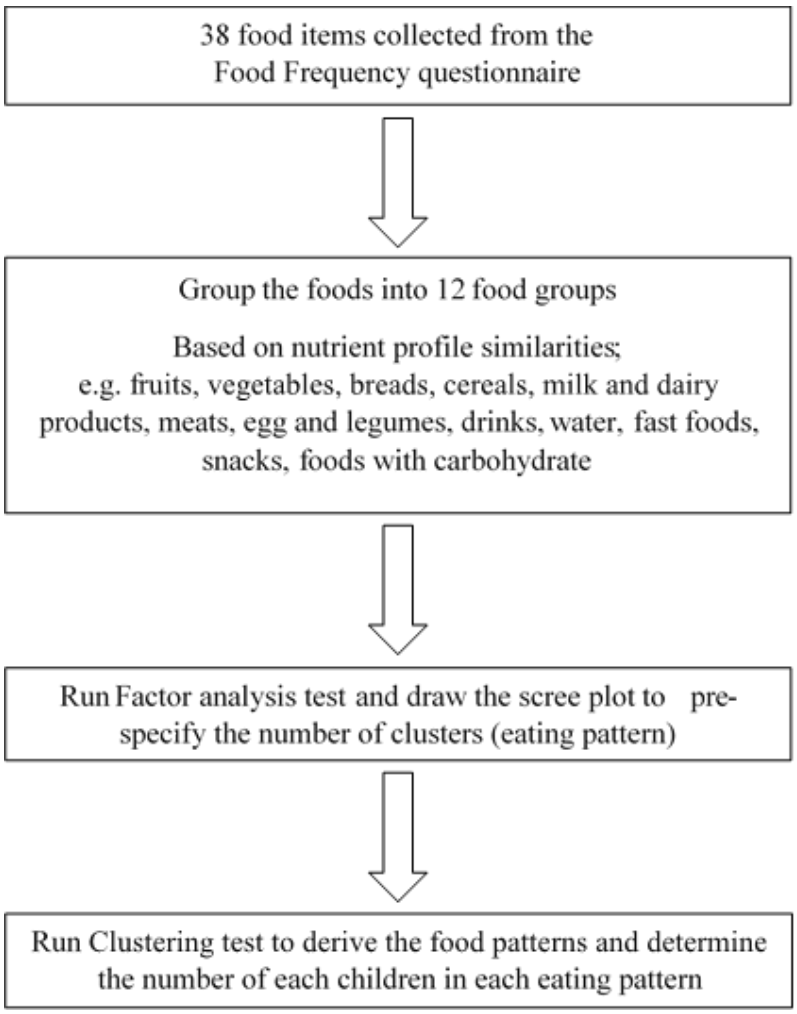


Figure 7.2. Step-by step analysis procedure to derive eating patterns

Notably, it is recognized that FA (Pallant, 2016), and also cluster analysis, can be sensitive to outliers, and can affect the cluster solution in that variables with larger values that will outweigh those with smaller values (Newby & Tucker, 2004). The frequency of food consumption in the questionnaire did not have normal distribution and many foods were reported as not eaten by some children (i.e., zero value). Two limitations related to the distribution of the food frequency data were apparent. Firstly transformation to include the zero values can dilute differences in the derived patterns (Moeller et al., 2007) and the ideal number of participants for running a FA is >150 (Pallant, 2016) and the number of children in this study was 99. Therefore, the author decided to perform a transformation to standardize the data rather than removing the outliers. Box cox transformation was used to standardize the food data. It is defined as: $T(Y) = (Y\lambda-1)/\lambda$, where Y is the response variable and λ is the transformation parameter. For $\lambda = 0$, the natural log of the data is taken instead of using a formula (http://www.itl.nist.gov/div898/handbook/eda/section3/eda336.htm). This method is not

among the list of SPSS tests, so the transformation was performed with R software by a statistical advisor, and then the transformed food data was copied to the SPSS software to perform the cluster analysis.

After transforming the food data for each of the relatively large number of food items (n = 38), the researcher classified each food item (i.e., transformed value), into eleven food groups based on similarity in their nutrient content and profile (

Table 7.5). Water is a beverage, but based on its nutrient profile, it was challenging to group it with other consumed beverages such as juice or soft drinks. Therefore, the researcher repeated the analysis (including deriving the eating patterns) with water as a beverage with other types of beverages consumed by children, but this changed the foods patterns remarkably. As a result, water was then considered as an individual food item.

Table 7.5. Food groups used for analysis of eating patterns

Food Group	**Food Item**
Fruits	fresh fruit and tinned/ frozen fruit
Vegetables	fresh vegetables, tinned/ frozen vegetables, taro, kumara and mashed/boiled/ jacket potatoes
Breads	white breads and other types of breads
Cereals	sweetened cereals and plain cereals
Milk and dairy products	milk, cheese and yogurt
Meats	red meat, chicken/ pork and fish
Egg and legumes	eggs and lentils/ kidney beans/ soy beans
Foods with carbohydrate	dumplings, pasta, noodles, rice and chapatti/ roti
Fast foods	sausage/ bacon, roasted/ chips potatoes and takeaways
Snacks	chippies/ crisps, crackers, sweet biscuits, lollies/ sweeties and chocolate
Beverages	juice, fizzy/ energy drink, cordial/ powdered fruit drink, food drink and tea/coffee
Water	Water

In order to pre-specify the number of eating patterns FA test and scree plot was applied. The analysis was done with Varimax rotation of the eleven food groups. To determine the number of eating patterns Eigenvalues >1.0 and where the scree plot reached a plateau (Michels & Schulze, 2005), and the interpretability of the eating patterns were considered (Cattell, 1966; Newby & Tucker, 2004).

Then, for the purpose of identifying one of the derived eating pattern for each child K-means clustering with consideration of the number of eating patterns derived from the FA was applied. The K-means method groups individuals into clusters (eating patterns) where the distance between the centre of each cluster is maximum from the others, though the distance between any single individual and the centre of their closest cluster is the minimum (Tucker, 2010). This was to cluster each child under one unique eating pattern (Devlin et al., 2012; Newby & Tucker, 2004).

Differences in derived eating patterns means were tested in normally distributed data by one-way analysis of variance (ANOVA) and for non-normally distributed data Kruskal-Wallis test was applied. Post hoc tests comparisons using Tukey HSD were used to determine which groups were significantly different and influenced the test statistic.

7.3 Results

7.3.1 Characteristics of children

More than half of children (n = 57/ 99, 58%) were boys (Table 7.6) and represented 25.1% of the original Auckland birth cohort (34.0% from National Women's Health and 2.6% from Middlemore Hospital) (personal communication with J. Rowan).

In this multi-ethnic birth cohort study, the children were categorised into five ethnic groups from the ethnic groups identified by the mother at birth: Polynesian (includes Māori, Pacific), European, Indian, Chinese and other. As only one Māori child was measured, this child was included with Pacific. Almost half of the children were European (Table 7.6).

Table 7.6. Characteristics of children (n = 99) followed-up at age 9 years

Characteristic	n	%
Gender		
Girls	43	43
Boys	56	57
Ethnicity		
European/ Caucasian	46	47
Polynesian	13	13
Māori	1	
Pacific Island	12	
Indian	23	23
Chinese	6	6
Other	11	11

7.3.2 Maternal and birth characteristics of children

Boys were born slightly heavier (3324 ± 572 g) than girls (3174 ± 511 g), but this was not a statistically significant difference (Table 7.7). More than half of the girls 53% and boys 57 % had been fully breastfed during the 6-8 weeks postpartum (Table 7.7). Boys' mothers were about two years younger than the girls' mothers' when they got pregnant (p = 0.050) (Table 7.7). The majority of mothers' had tertiary education (Table 7.7) and half of the mothers of both boys and girls had been treated with insulin and the rest had been treated with metformin (Table 7.7).

Table 7.7. Characteristics of children at birth and maternal baseline information

Characteristic	Girls (n = 43)		Boys (n = 56)		*p* Value
Birth weight (g)	3174.4	(511.1)	3323.7	(572.2)	0.181 [a]
Infant feeding 6-8 weeks postpartum					0.957 [b]
Breast feeding	23	(53%)	32	(57%)	
Bottle feeding	6	(14%)	9	(16)	
Both breast and bottle	12	(28%)	15	(27%)	
Not seen	2	(5%)	0	(0%)	
Mother's age (years)	35.72	(5.32)	33.77	(4.41)	**0.050** [a]
Mothers' tertiary education	26	(60%)	34	(61%)	0.980 [b]
Mother's type of treatment					0.300 [b]
Insulin	26	(60%)	28	(50%)	
Metformin	17	(40%)	28	(50%)	

Data expressed as mean ± SD or n (%).[a] Independent t test. [b] Chi square test, it is empty due to the small number of participants.

7.3.3 Physical measurements of children at the age of 7-9 years

Boys and girls were not different by weight, height, BMI or measures of weight, height, and CDC BMI z score (Kuczmarski et al., 2000) by age and gender. However, girls had proportionally more fat mass than boys (12.81 kg vs 10. 20 kg, 33.5% vs 29.0%) and visceral abdominal fat (782.90 g vs 552.89 g) (Table 7.8).

Table 7.8. Physical measurements at 9 years

	Girls (n = 43)		Boys (n = 56)		
	Mean (median)	**SD (25th/ 75th)**	**Mean (median)**	**SD (25th/ 75th)**	***p* Value**
Age (years)	8.88	0.41	8.94	0.45	0.425 [b]
	8.76	(8.6, 9.01)	8.75	(8.65, 9.12)	
HbA_1c ($mmol.mol^{-1}$) [c]	34.71	2.63	34.83	2.39	0.719 [b]
	35.00	(33.00, 36.00)	35.00	(33.25, 36)	
Anthropometry					
Weight (kg)	35.98	12.40	33.58	8.43	0.646 [b]
	32.925	(27.65, 40.55)	32.8	(27.34, 38.04)	
Height (cm)	135.97	8.71	136.24	6.17	0.857 [a]
	136.35	(131.25, 141.4)	137.83	(131.51, 140.66)	
BMI ($kg.m^{-2}$)	19.07	4.45	17.88	3.30	0.201 [b]
	17.7017	(16.03, 20.54)	17.13	(15.21, 19.36)	
Weight z scores [d]	0.74	1.17	0.58	1.26	0.501 [a]
	0.69	(-0.14, 1.57)	0.86	(-0.61, 1.52)	
Height z scores [d]	0.54	1.22	0.50	1.06	0.847 [a]
	0.58	(-0.06, 1.31)	0.56	(-0.27, 1.29)	
BMI z scores [d]	0.67	1.06	0.41	1.21	0.255 [a]
	0.64	(-0.15, 1.48)	0.58	(-0.54, 1.34)	
Waist (cm)	66.50	11.02	66.65	10.67	0.966 [b]
	62.95	(58.95, 70.45)	63.97	(58.15, 72.45)	
Waist/ Height (cm)	0.49	0.09	0.49	0.08	0.799 [b]
	0.4529	(0.43, 0.54)	0.47	(0.42, 0.53)	
DEXA [e]					
Fat free mass (kg)	23.37	6.12	23.37	3.78	0.996 [a]
	22.54	(20.53, 25.73)	23.10	(20.53, 25.72)	
Fat mass (kg)	12.81	6.65	10.20	5.27	**0.015** [b]
	10.61	(6.23, 12.85)	7.98	(6.23, 12.85)	
Fat mass%	33.96	6.32	28.85	7.66	**0.001** [a]
	32.71	(22.34, 34.17)	27.31	(22.34, 34.17)	
Visceral abdominal fat (g)	782.90	644.49	552.89	471.37	**0.041** [b]
	925.50	(373.25, 1223)	607.00	(373.25, 1223)	

DEXA, dual X-ray absorptiometry. [a] Independent t test. [b] Mann-Whitney U test. [c] HbA_1c measures missing for four boys and 2 girls. [d] CDC growth charts (Kuczmarski et al., 2000). [e] There was one girl who did not have DEXA measurements

There were three (2 girls and a boy) (Figure 7.3) out of 93 children, who had $HbA_1c \geq 41$ mmol.mol^{-1} (a girl with 'Other' ethnicity and a European/ Caucasian boy with $HbA_1c = 41$ mmol.mol^{-1} and a Pacific girl with $HbA_1c = 43$ mmol.mol^{-1}). The parents were notified and advised to contact their general practitioner for further tests.

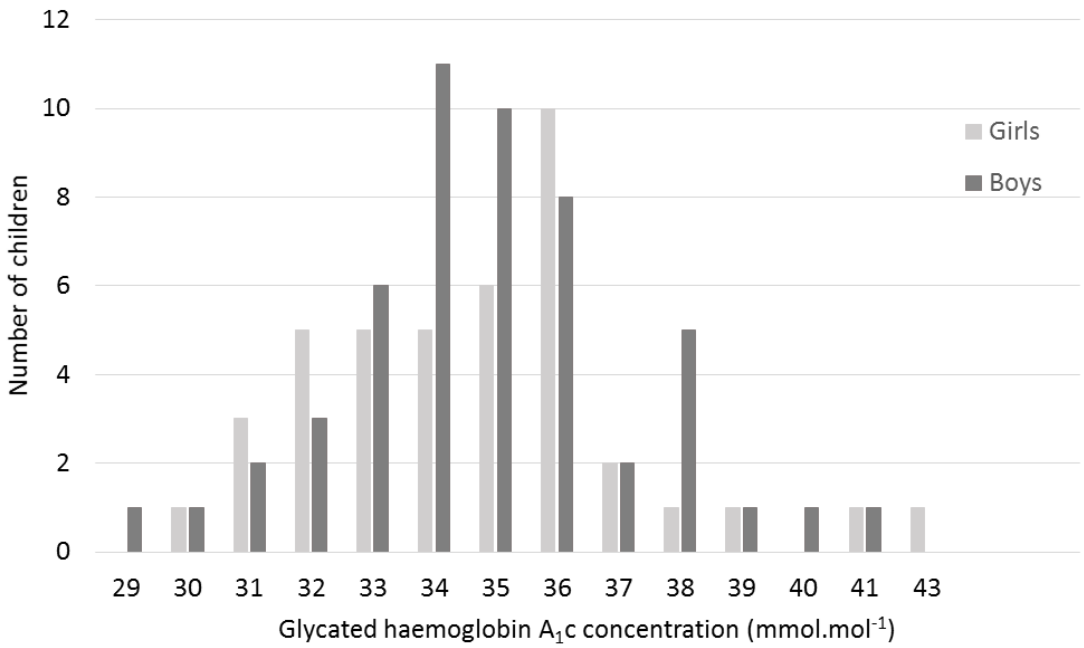


Figure 7.3. Frequency of glycated haemoglobin A_1c concentration by gender among children at 7-9 years old

Based on the international obesity task force (IOTF) BMI cut-offs, which account for age and gender (Cole & Lobstein, 2012) children were classified as thin, normal weight, overweight, and obese. Of the nine children classified as thin, five were Indian, three Chinese and one European. Five in ten (50%) children were classified as normal weight (26 girls, 30 boys, and 56 overall). While, with the same manner one in five was overweight and one in seven children obese (Table 7.9) The ethnic groups of the children classified as obese were Polynesian (5), European (6), Indian (2) and 'Other' ethnicities (1).

Table 7.9. Children classified as thin, normal, overweight and obese based on the international obesity task force cut-offs [a]

	Girls (n = 43) n (%)		Boys (n = 56) n (%)		Total (n = 99) n (%)	
Thin	2	(5)	7	(13)	9	(9)
Normal	25	(58)	31	(55)	56	(57)
Overweight	9	(21)	11	(20)	20	(20)
Obese	7	(16)	7	(12)	14	(14)

[a] Using Cole and Lobstein cut-offs (2012). Chi-square test applied.

7.3.4 Associations between HbA_1c, gender, ethnicity and early life information

Data were explored visually and by correlation for relationships between HbA_1c at 9 years and age, gender, ethnicity, birth weight, type of infant feeding, ethnicity as well as maternal age at conception, maternal education and type of GDM treatment. No statistically significant correlations ($p < 0.05$) were found. There was no significant difference (U = 1020.00, Z = -0.36, $p = 0.72$, r = 0.04) in HbA_1c of girls' (median = 35, n = 41), and boys' (median = 35, n = 52). A Kruskal- Wallis analysis of variance indicated no significant difference [χ^2 (4, n = 93) = 2.24, $p = 0.691$] among children by ethnicity (Table 7.10).

Table 7.10. HbA_1c by ethnic group

Ethnic group	**HbA_1c (mmol.mol^{-1})**		
	Minimum	**Maximum**	**Mean**
European/Caucasian (n = 43)	29	41	34
Polynesian (n = 13)	31	43	35
Indian (n = 22)	30	40	35
Chinese (n = 5)	33	39	36
Other (n = 10)	31	41	35

Missing = 7.

7.3.5 Associations between HbA_1c and body measurements

There was a small negative correlation between HbA_1c with both waist and waist-to-height ratio (r = -0.245 $p < 0.018$ and r = -0.286 $p < 0.005$ respectively) (Figure 7.4 and Figure 7.5). There were no correlations of HbA_1c with any of the other body measurements.

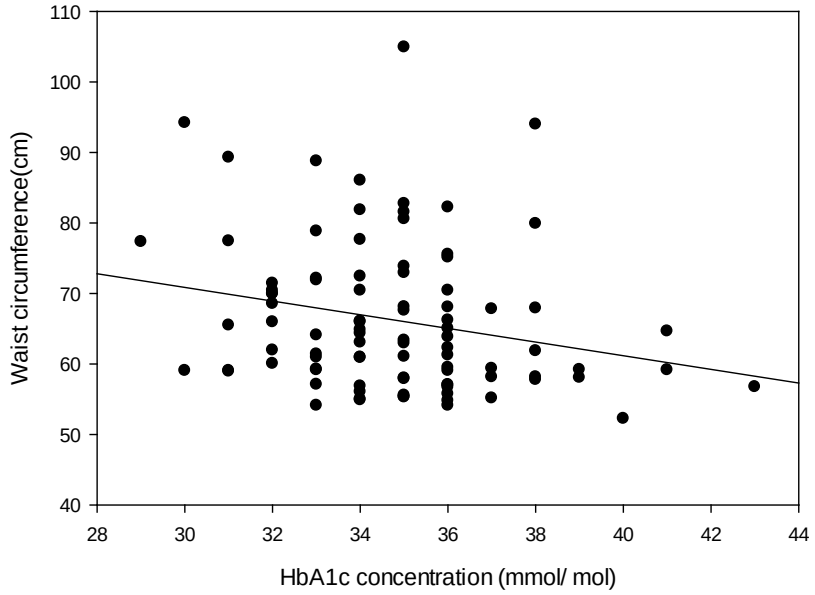


Figure 7.4. HbA_1c with waist circumference (n = 93)

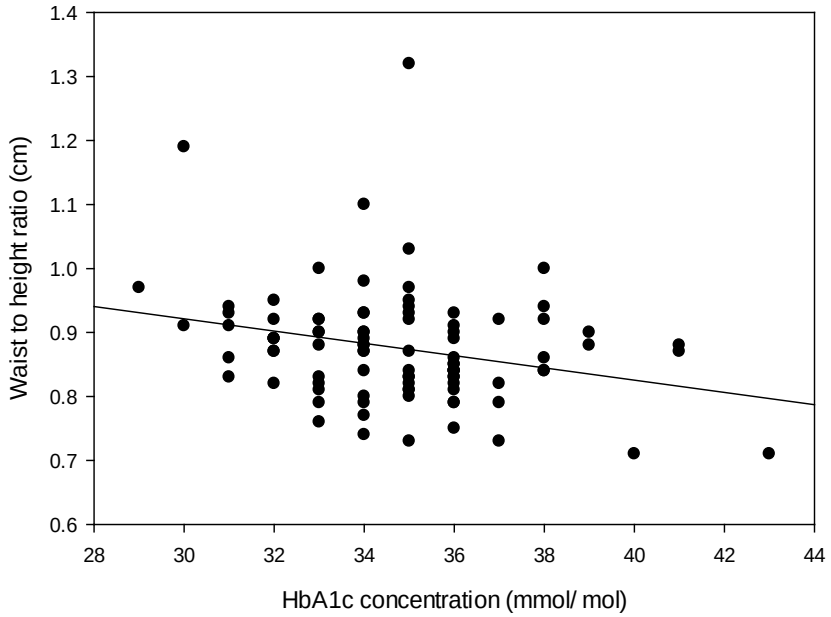


Figure 7.5. HbA_1c with the waist-to-height ratio (n = 93)

7.3.6 Foods consumption of children reported by the mothers

Children consumed a median of 25.05 (IQR: 18.56 and 30.92) servings of any food per day. There were two Indian boys with vegetarian diet (no consumption of meat, chicken, fish, and sausage/ bacon). The proportion of children who met the Ministry of Health (Ministry of Health, 2012a) recommended number of servings each day for the five food groups (fruit and vegetables are separate groups) was lowest for vegetable (37.4%) (Table 7.11) and highest for fruit (61%).

Table 7.11. Comparison of frequency of consumed food with Ministry of Health food guidelines (n = 99)

Food guideline	**Guideline servings/ day** [a]	**Children achieving guideline (times/day) n (%)**	
Fruit (fresh and tinned/ frozen fruit)	2 or more	61	(61.6)
Vegetables (fresh and tinned/ frozen vegetables, mashed potatoes, kumara and taro)	3 or more	37	(37.4)
Milk and dairy products (milk, cheese and yogurt)	at least 2-3	41	(41.4)
Meat and other protein (meat, chicken, fish, egg, legumes and sausage or bacon)	at least 1-2	43	(43.4)
Bread and cereals (bread white, bread other, cereal sweetened, cereal, dumplings, pasta, noodles, rice and chapatti)	at least 5	40	(40.4)
Children meeting guidelines	0 guideline	9	(9.1)
	Only 1 guideline	26	(26.3)
	Only 2 guidelines	21	(21.2)
	Only 3 guidelines	25	(25.3)
	Only 4 guidelines	11	(11.1)
	All 5 guidelines	7	(7.1)

[a] (Ministry of Health, 2012a)

In general, more than half of the children (62%, girls = 65%, boys 61%) consumed ≥ 5 times/day from fruit and vegetables. Of the forty children, meeting the daily breads and cereals guideline the number who had at least one serving that was wholegrain or did not have added sugar were thirty-two and twenty-four respectively. Of the ninety-nine

children, twenty-four consumed only white bread and thirty-eight children consumed white rice at least once a day.

The daily consumption of foods by thirteen children, who did not report eating any breads or cereals, from the most common to the least were from high energy foods (once a day, n = 9), meat and other protein group (1-2/ day, n = 8), fruit group (at least twice a day, n = 8), vegetables group (at least 3/ day, n = 8), sugary drinks (at least once a day, n = 7) and milk and dairy products (at least 2-3/ day, n = 2).

Most of the children ate at least three times a week from the high energy food group (take- away, roasted potato, chippies crackers, biscuits, lollies, and chocolate) and sugary drinks (juice, fizzy drink, cordial, and food drink) (86% and 71% respectively).

The majority of children 85/99 consumed high energy food group foods at least three times a week. There was no statistically significant difference by gender, but there was a significant difference by ethnicity (χ^2 21.125, p = 0.001) with European children consuming most often (n = 45/46), followed by the Indian (n = 20/23), Polynesian (n = 10/ 13), Chinese (n = 5/6), and ‘Other’ (n = 5/11).

Out of 70/99 children who consumed sugary drinks at least three times a week, in descending proportion 12/13 Polynesian, 19/23 Indian, 7/11 ‘Other’, and 3/6 Chinese and 29/43 were European. However, there was no difference found by gender or ethnicity.

The pattern of compliance with the guidelines varied by ethnicity (Table 7.12). In particular, only one third of Chinese, Indian, and Other consumed fruit at the frequency required to meet the guidelines. Although consumption from food groups was not statistically different among children by ethnicity, Chinese and ‘Other’ reported less frequent eating of fruit and vegetables than the other ethnic groups (Table 7.12).

Table 7.12. Comparison of food consumption following Ministry of Health food Guidelines by ethnicity

	Children achieving guideline [a] (times/ day) n (%)									
	Fruit (≥ 2 servings/ day)		**Vegetables** (≥ 3 servings/ day)		**Milk and dairy products** (at least 2-3 servings/ day)		**Meat and other protein** (at least 1-2 servings/ day)		**Bread and cereals** (at least 5 servings/day)	
European/Caucasian	30	(65)	32	(70)	31	(67)	10	(77)	38	(83)
Polynesian	11	(85)	9	(69)	10	(77)	31	(67)	11	(85)
Indian	14	(61)	12	(52)	13	(57)	13	(57)	23	(100)
Chinese	2	(33)	1	(17)	5	(83)	5	(83)	4	(66.7)
Other	4	(36)	2	(18)	9	(82)	9	(82)	10	(91)

[a] (Ministry of Health, 2012a)

Girls more frequently consumed tinned/ frozen fruit (Mann-Whitney U test U = 902.00, Z = -2.246, *p* = 0.025, r = 0.23), kumara (U = 884.50, Z = -2.401, *p* = 0.016, r = 0.32), noodles (U = 709.00, Z = -3.542, *p* = 0.001, r = 0.36), high energy food group (U = 811.50, Z = -2.729, *p* = 0.006, r = 0.27), roasted potatoes (U = 771.00, Z = -3.127, *p* = 0.002, r = 0.42), chippies (U = 886.50, Z = -2.258, *p* = 0.024, r = .030), lollies (U = 841.00, Z = -2.597, *p* = 0.009, r = 0.35) and snacks group (U = 826.50, Z = -2.623, *p* = 0.009, r = 0.26) than boys (

Table 7.13). Overall, water, fresh fruit, fresh vegetables, milk, other types of bread, white bread, yogurt, cereals and crackers were the foods most frequently reported by the group. Each of these foods were consumed ≥ 0.43 times in a day (

Table 7.13).

Table 7.13. Frequencies of foods consumed each day overall and by gender for 99 children

Food groups	Frequency/ day Median (percentile 25th, 75th)						*p* Value [a]
	Girls n = 43		Boys n = 56		Total n = 99		
Fruit group							
Fresh	2.00	(1.00, 4.00)	2.00	(1.00, 5.00)	2.00	(1.00, 4.00)	0.705
Tinned/Frozen	**0.06**	**(0.00, 0.28)**	**0.00**	**(0.00, 0.06)**	**0.03**	**(0.00, 0.14)**	**0.025**
Total	2.14	(1.14, 4.00)	2.05	(1.00, 5.00)	2.14	(1.00, 4.29)	0.930
Vegetable group							
Fresh	2.00	(0.57, 3.00)	1.00	(0.714, 3)	1.00	(0.57, 3.00)	0.918
Tinned/ frozen	0.14	(0.00, 0.43)	0.14	(0.00, 0.53)	0.14	(0.00, 0.43)	0.696
Kumara	**0.06**	**(0.00, 0.14)**	**0.00**	**(0.00, 0.06)**	**0.03**	**(0.00, 0.13)**	**0.016**
Taro	0.00	(0.00, 0.00)	0.00	(0.00, 0.00)	0.00	(0.00, 0.00)	0.316
Mashed potatoes	0.28	(0.14, 0.43)	0.23	(0.13, 0.43)	0.29	(0.13, 0.43)	0.636
Total	2.43	(1.20, 4.43)	2.3	(1.18, 4.97)	2.35	(1.2, 4.57)	0.753
Milk and milk products							
Milk	1.00	(0.57, 2.00)	1.00	(0.11, 2.00)	1.00	(0.43, 2.00)	0.282
Cheese	0.30	(0.09, 0.57)	0.43	(0.14, 0.57)	0.29	(0.14, 0.57)	0.225
Yoghurt	0.43	(0.09, 1.00)	0.28	(0.06, 0.71)	0.43	(0.07, 0.71)	0.148
Total	2.28	(1.38, 3.57)	1.78	(0.81, 3.58)	2.10	(1.00, 3.57)	0.332
Meat and other protein							
Chicken	0.28	(0.28, 0.57)	0.28	(0.28, 0.57)	0.29	(0.29, 0.57)	0.856
Red meat	0.28	(0.28, 0.43)	0.28	(0.15, 0.43)	0.29	(0.29, 0.43)	0.530
Fish	0.14	(0.06, 0.14)	0.14	(0.03, 0.14)	0.14	(0.03, 0.14)	0.577
Eggs	0.28	(0.06, 0.57)	0.14	(0.09, 0.42)	0.29	(0.10, 0.43)	0.284
Legumes	0.03	(0.00, 0.14)	0.00	(0.00, 0.14)	0.00	(0.00, 0.14)	0.702
Total	0.85	(0.60, 1.42)	0.78	(0.60, 1.14)	1.39	(1.07, 1.91)	0.257
Breads and cereals							
White bread	0.57	(0.00, 2.00)	0.36	(0.00, 1.75)	0.43	(0.00, 2.00)	0.353
Other types of bread	0.57	(0.00, 2.00)	0.50	(0.00, 2.00)	0.57	(0.00, 2.00)	0.605
Sweetened cereals	0.13	(0.00, 0.43)	0.14	(0.00, 0.93)	0.13	(0.00, 0.57)	0.659
Cereals	0.28	(0.00, 0.57)	0.43	(0.00, 1.00)	0.43	(0.00, 1.00)	0.364
Dumplings	0.00	(0.00, 0.00)	0.00	(0.00, 0.03)	0.00	(0.00, 0.03)	0.187

Food groups	Frequency/ day Median (percentile 25th, 75th)						*p* Value [a]
	Girls n = 43		Boys n = 56		Total n = 99		
Pasta	0.14	(0.06, 0.42)	0.14	(0.03, 0.28)	0.14	(0.07, 0.29)	0.313
Noodles	**0.28**	**(0.14, 0.42)**	**0.11**	**(0.03, 0.14)**	**0.14**	**(0.07, 0.29)**	**0.001**
Rice	0.28	(0.14, 0.71)	0.28	(0.14, 0.57)	0.29	(0.14, 0.57)	0.673
Chapatti	0.00	(0.00, 0.42)	0.00	(0.00, 0.09)	0.00	(0.00, 0.14)	0.552
Total	2.00	(0.85, 4.00)	1.14	(0.96, 4.00)	4.24	(2.32, 6.96)	0.663
Sugary drinks							
Fizzy drink	0.14	(0.03, 0.42)	0.09	(0.03, 0.28)	0.14	(0.03, 0.29)	0.206
Cordial drink	0.00	(0.00, 0.28)	0.00	(0.00, 0.06)	0.00	(0.00, 0.14)	0.057
Juice	0.42	(0.13, 1.00)	0.28	(0.07, 0.57)	0.29	(0.1, 0.71)	0.157
Food drink	0.14	(0.00, 0.42)	0.14	(0.03, 0.42)	0.14	(0.03, 0.43)	0.828
Total	1.30	(0.60, 2.91)	0.74	(0.34, 1.43)	1.03	(0.46, 2.26)	0.053
High energy foods							
Take-away	0.14	(0.06, 0.14)	0.14	(0.06, 0.14)	0.14	(0.07, 0.14)	0.349
Roasted potatoes	**0.28**	**(0.14, 0.42)**	**0.14**	**(0.06, 0.24)**	**0.14**	**(0.07, 0.29)**	**0.002**
Sausage/bacon	0.14	(0.06, 0.28)	0.14	(0.06, 0.28)	0.14	(0.07, 0.29)	0.370
Total	**2.78**	**(1.93, 3.80)**	**1.88**	**(1.34, 2.80)**	**2.13**	**(1.57, 3.14)**	**0.006**
Snacks							
Chippies	**0.28**	**(0.14, 1.00)**	**0.28**	**(0.06, 0.42)**	**0.29**	**(0.1, 0.57)**	**0.024**
Crackers	0.42	(0.14, 0.85)	0.28	(0.09, 0.92)	0.43	(0.13, 0.86)	0.482
Biscuits	0.28	(0.14, 0.71)	0.28	(0.14, 0.57)	0.29	(0.14, 0.57)	0.767
Lollies	**0.28**	**(0.14, 0.42)**	**0.14**	**(0.06, 0.28)**	**0.29**	**(0.1, 0.43)**	**0.009**
Chocolate	0.14	(0.06, 0.28)	0.14	(0.06, 0.28)	0.14	(0.07, 0.29)	0.498
Total	**2.78**	**(1.50, 3.00)**	**1.96**	**(1.12, 2.50)**	**2.31**	**(1.27, 2.71)**	**0.009**
Water [b]	4.00	(3.00, 5.00)	4.00	(3.00, 6.50)	4.00	(3.00, 6.00)	0.869
Tea	0.00	(0.00, 0.09)	0.00	(0.00, 0.00)	0.00	(0.00, 0.00)	0.508

[a] Mann-Whitney U test. [b] The water consumption of three boys was missed.

7.3.7 Consumption of foods and HbA_1c

A comparison of Spearman's correlations showed that there was no relationship between the HbA_1c and either of the consumed foods among children. However, investigating the latter correlation by gender it was revealed that there was a relationship between frequency of daily consumption of food drinks with HbA_1c.

Among girls there was a medium positive correlation (n = 41, r = 0.397 *p* = 0.010), while the consumption of food drinks among boys showed a medium negative correlation with HbA_1c (n = 52, r = -0.297, *p* = 0.033).

7.3.8 Consumption of foods based on glycaemic index

Girls consumed more medium GI foods than boys (Table 7.14). These foods include fizzy drink/ energy drink, cordial/ fruit drink, potatoes roasted/ chips, taro, pasta, chapatti/ roti, crackers, sweet biscuits, takeaways; girls (median = 2.6/ day, n = 43) and boys (median = 1.7/ day, n = 56) (Mann-Whitney U test, U = 704.50, Z = -3.53, *p* = 0.001, r = 0.35 n = 99) (Table 7.14).

Foods from the low GI group such as fruit and vegetables were overall consumed most frequently by both girls (median = 13.9) and boys (median = 13.5) when compared with medium (girls median = 2.5, boys median = 1.7) and high GI foods (girls median = 3.7, boys median = 4.3). However, 52 children consumed ≥ 5 times high GI foods in a day. Out of these children, 11 were overweight, 9 obese, 28 normal weight and five underweight. This is similar to overall proportions of the sample.

Table 7.14. Frequency of consumption from low, medium and high glycaemic index foods each day by gender

Gender	**Frequency of consumption of low GI foods/ day**		**Frequency of consumption of medium GI foods/ day**		**Frequency of consumption of high GI foods/ day**	
	Median (percentile 25th, 75th)		Median (percentile 25th, 75th)		Median (percentile 25th, 75th)	
Girls (n = 43)	13.9	(11.4, 17.9)	2.6	(1.7, 4.3) [a]	3.7	(2.4, 6.4)
Boys (n = 56)	13.5	(10.7, 18.4)	1.7	(1.0, 2.1)	4.3	(2.3, 7.0)

[a] Mann Whitney U test. GI glycaemic index (*p* = 0.001).

7.3.9 Identification of eating patterns

Scree plot (Figure 7.6) and FA with varimax rotation loading were applied to the food frequency data to be able to specify the optimal number of eating patterns (clusters) with a factor loading greater than 0.3 (Table 7.15). For this analysis, eleven food groups which summed the frequency of consumption of foods with similar nutrient profiles and a twelfth food group (water) were entered. The greater the factor loading for a specific food group or food item, the greater the effect of that food group or food item on a specific factor. For example, in factor one, fast foods (factor loading = 0.817) had a higher load than the beverages group (factor loading -0.567).

Five factors were identified (Table 7.15). Names for each factor/eating pattern were determined subjectively and together the five patterns explained 65% of the total variance. The first pattern was named ‘energy dense and bread’ as most of the foods were processed i.e. not wholesome. The second was named ‘healthy’ as foods were from the three recommended food groups – fruit, vegetables and meats. The third included refined carbohydrates, eggs and legumes, and it was named ‘refined carbs, eggs and legumes’. The fourth eating pattern included breakfast cereals and dairy, so it was named ‘breakfast’ and the last was named ‘survival’ as water was the only item identified in this group.

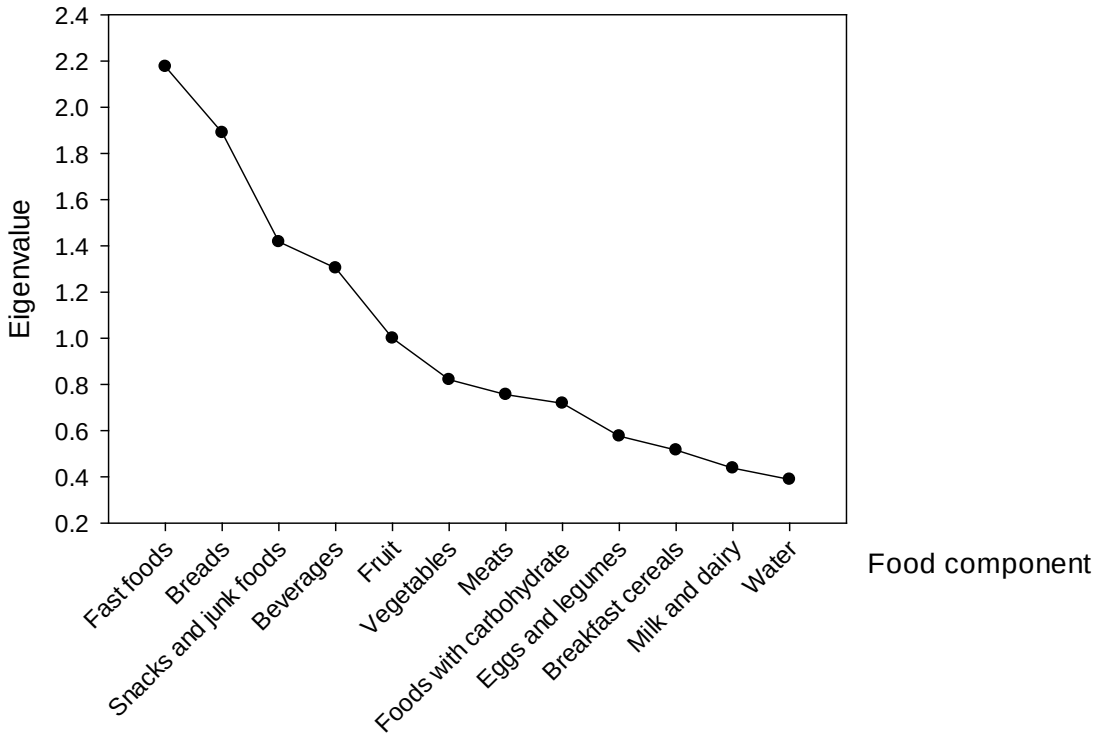


Figure 7.6. Scree plots of eigen values of food components

Table 7.15. Factor loading matrix derived from food or food groups for 5 major factors [a]

Food or food group	**Eating pattern 1**	**Eating pattern 2**	**Eating pattern 3**	**Eating pattern 4**	**Eating pattern 5**
Fast foods (take-away, roasted potatoes and sausage or bacon)	**0.817**				
Breads (white bread and other types of bread)	**0.669**			0.320	
Snacks and junk foods (chippies, crackers, biscuits, lollies and chocolate)	**0.589**				-0.420
Beverages (fizzy drink, cordial drink, juice and food drink)	**0.567**			0.313	-0.306
Fruits (fresh and tinned/frozen fruit)		**0.787**			
Vegetables (fresh and tinned/frozen vegetables, mashed potatoes, kumara and taro)		**0.643**			
Meats (meat, chicken and fish)		**0.504**		-0.387	0.444
Foods with carbohydrate (dumplings, pasta, noodles, rice and chapatti)			**0.805**		
Eggs and legumes (egg and legumes)			**0.727**		
Breakfast cereals (sweetened cereals and cereals)				**0.855**	
Milk and dairy products (milk, cheese and yogurt)	0.444	0.329		**0.522**	
Water					**0.830**
Eigin value	2.18	1.89	1.41	1.30	1.00
Variance explained %	18.1	15.7	11.8	10.9	8.3
Characteristic of the factor	Energy dense and bread	Healthy	Refined carbs, eggs and legumes	Breakfast	Survival

[a] Absolute values < 0.30 were excluded from this table for simplicity.

The number of girls within each eating pattern was not significantly different (p = 0.33) from the number of boys (Table 7.16). However, there was a significant difference in

eating patterns between European and non-European children (χ^2 15.00, p = 0.005) (Figure 7.7). In particular, European children compared to non-European were under represented in the 'energy dense and bread' (χ^2 6.022, p = 0.014) group and more often in the 'survival' (χ^2 8.146 p = 0.004) patterns.

Table 7.16. Gender distribution by eating patterns

Gender [a]	**Energy dense and bread n (%)**	**Healthy n (%)**	**Refined carbs, eggs and legumes n (%)**	**Breakfast n (%)**	**Survival n (%)**
Girls (n = 43)	12 (28)	8 (19)	4 (9)	9 (21)	10 (23)
Boys (n = 56)	22 (39)	5 (9)	10 (18)	9 (16)	10 (18)

[a] Chi-square test. There was no significant difference.

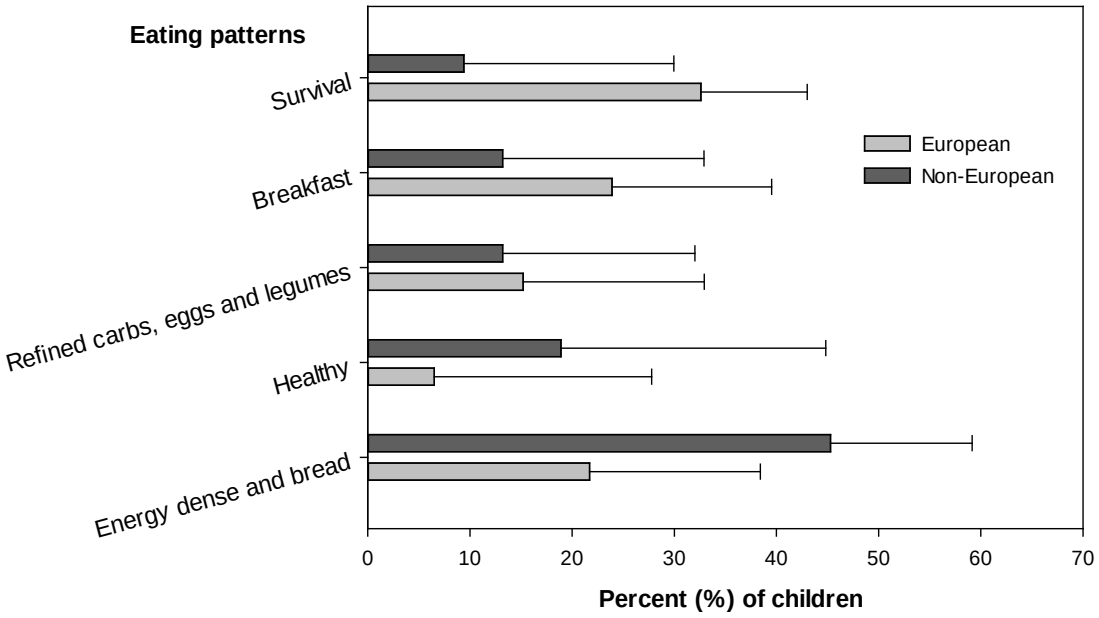


Figure 7.7. Percentage of eating patterns in European and non-European children

7.3.10 Eating patterns and HbA_1c

Eating patterns were explored visually and by correlation for relationships with HbA_1c at 9 years. There were no statistically significant differences among the five eating patterns for HbA_1c (Figure 7.8).

However, the 'refined carbs, eggs and legumes' eating pattern appeared to be higher, so all other eating patterns were compared to this eating pattern. Therefore, recoding children into 'refined carbs' and 'not refined carbs' and after adjusting for central fat (waist-to-height ratio which was correlated with HbA_1c) analysis of variance showed a borderline significant difference (p = 0.052) for HbA_1c (1.4 $mmol.mol^{-1}$) (95% CI [-0.2, 2.8]).

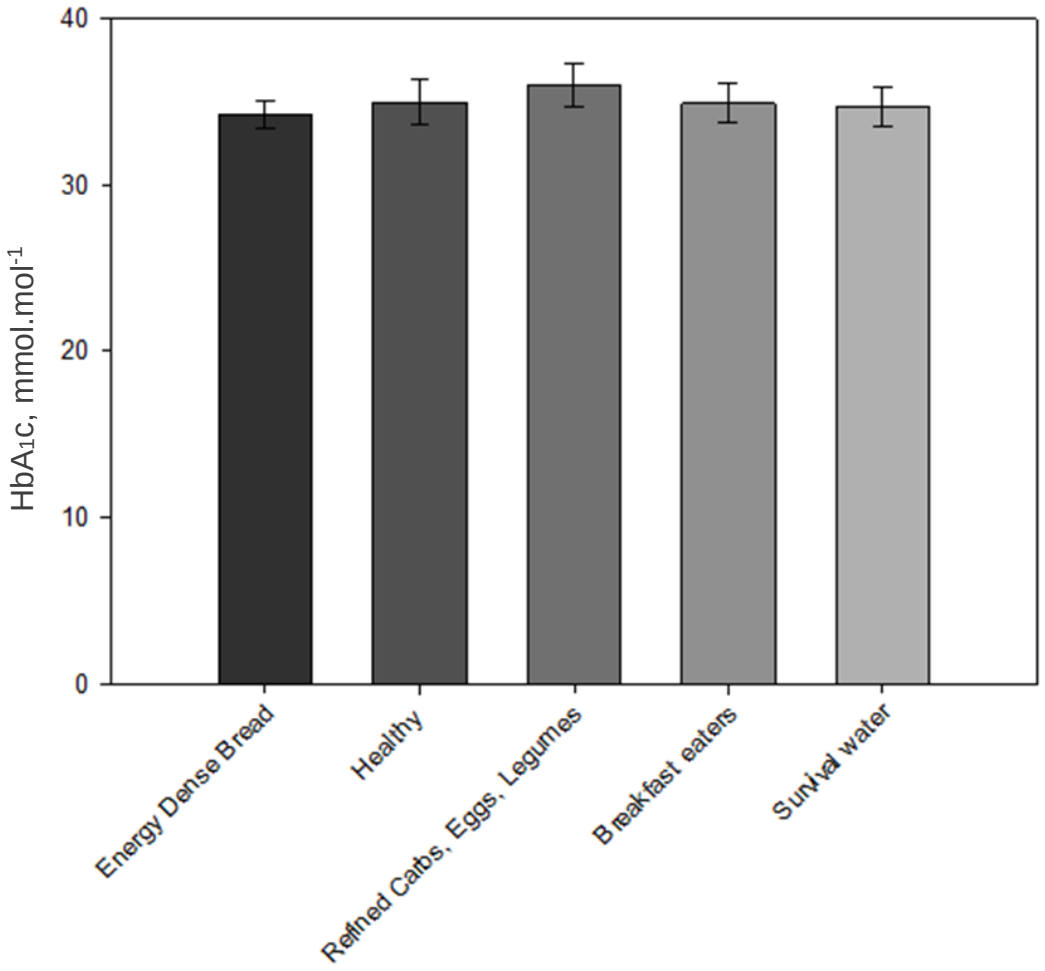


Figure 7.8. Association between HbA_1c and the five derived eating patterns

7.3.11 Eating patterns and body measurements

The only body measurements that showed differences among the five eating patterns using one-way ANOVA were the weight z scores (p = 0.041) and the height z scores (p = 0.018). Post-hoc comparisons using the Tukey HSD test showed that the mean z score for ‘breakfast’ for weight was 1.42 (SD = 0.93), and for height 1.22 (SD = 0.93), which were higher for both measures than the other four eating patterns. They were not different to each other. In particular, the weight z scores of children with ‘breakfast’ pattern were 0.848 higher than children with ‘energy dense and bread’ pattern (p = 0.016), 1.179 z scores higher than ‘Healthy’ (p = 0.0007), 1.037 z scores higher than refined carbs (p = 0.016) and 0.923 z scores higher than survival (p = 0.018). In addition, the height z scores of children with ‘breakfast’ was 0.794 z scores higher than children with ‘energy dense and bread’ pattern (p = 0.013), 1.348 z scores higher than ‘Healthy’ (p = 0.001), 0.818 z scores higher than refined carbs (p = 0.053) and 0.718 z scores higher than survival (p = 0.020). The BMI z scores were not different, and this was because the heavier children were also taller.

The visceral abdominal fat of the children in the breakfast group was also higher (not normally distributed so the Kruskal Wallis test was applied p = 0.031). Children with ‘breakfast’ pattern had the highest mean of visceral fat (885 g), followed by ‘survival’ (663 g), ‘healthy’ (626 g), ‘energy dense and bread’ (570 g) and ‘refined carbs, eggs and legumes’ (553 g) patterns.

7.3.12 Eating patterns and early life factors

Children who had ‘healthy’ eating pattern were born with the lowest birth weight (n = 13, mean ± SD = 3061 ± 466 g) and children with ‘breakfast’ eating pattern were born as the heaviest (n =18, mean ± SD = 3394 ± 56 g) children. However, there was no significant difference between the eating patterns and the birth weight of children (p = 0.289) neither between gender nor birth weight of children (p = 0.720).

Investigating the distribution of children in each eating pattern based on their mothers’ age showed that the children with the ‘breakfast’ eating pattern had younger mothers (n = 18, mean ± SD = 33.53 ± 4.59 years) and children who were following ‘refined carbs, eggs and legumes’ had more senior mothers (n = 14, mean ± SD = 36.12 ± 4.09 years). However, there was no significant difference by age of mothers (p = 0.680).

No significant differences were found between eating patterns and how the baby was fed in the first six months ($p = 0.269$), maternal education ($p = 0.225$) and type of GDM treatment ($p = 0.443$), using Chi square test.

Stepwise linear regression found no predictors of HbA_1c with gender, eating patterns, body size and early life and maternal factors.

7.3.13 Summary of main findings

Eating patterns

- The five derived eating patterns were named ‘energy dense and bread’, ‘healthy’, ‘refined carbs, eggs and legumes’, ‘breakfast’ and ‘survival’.
- There were no significant differences in eating patterns between boys and girls.
- European compared to non-European children clustered more in the ‘survival’ pattern and less in the ‘energy dense and bread’ pattern.

Food and food group consumption

- Only 7 out of 99 children met the food guidelines for the five food groups.
- More children met the fruit recommendation (61%) made by MOH than the vegetables recommendation (37.4%).
- One third of Chinese, Indian and Other children met the fruit consumption guideline.
- Overall, 62% of the children consumed ≥ 5 times a day from fruit and vegetables groups.
- Overall, 86% and 71% of children consumed foods from the high energy and the sugary drinks groups respectively at least three times a week.

HbA_1c

- There were three children (3%) with HbA_1c more than ≥ 41 $mmol.mol^{-1}$.
- There were no differences in concentrations of HbA_1c by gender or ethnicity.
- There was a negative correlation between HbA_1c and both waist circumference and waist-to-height ratio.

Eating patterns and HbA_1c

- HbA_1c was different by gender for some of the individual foods. Among girls there was a significant correlation between food drinks and HbA_1c, but among boys a negative correlation was found with food drink.
- There was a difference in HbA_1c (1.4 $mmol.l^{-1}$) (95% CI [-0.2, 2.8]) between 'refined carbs' and 'not refined carbs' eating pattern after adjusting for waist-to-height ratio of borderline significance (p = 0.052).

Eating patterns and body measurements

- There was a difference between eating patterns and weight z scores, height z scores, and visceral abdominal fat. The mean z score for 'breakfast' for weight was 1.42 (SD = 0.93) and for height 1.22 (SD = 0.93), which were higher for both measures than the other four eating patterns. They were not different to each other.

Eating patterns and early life factors

- No significant difference was found between eating patterns and either birth weight, and how babies were fed in the first six months.
- No significant difference was found between eating patterns and maternal age at conception, maternal education level, and type of maternal GDM treatment.

7.4 Discussion

This body of work examines the relationship between foods consumed and patterns of eating with HbA_1c with consideration, body measurements and early life factors influences in a cohort of 9-year-old boys and girls from five principal ethnic groups in NZ. The mothers' during the early life (i.e., pregnancy) of these children were treated for GDM, which means these children were also in a high-risk group for future T2DM. This is the first time, to the knowledge of the author, that this combination of factors has been explored.

The main finding is that HbA_1c were not significantly associated with the derived eating patterns. However, the HbA_1c of this cohort of children was relatively high, 35 $mmol.mol^{-1}$, compared with other surveys such as the Third National Health and Nutrition Examination Survey (NHANES III), and Child Heart and Health Study in England (CHASE Study). In NHANES III the mean of HbA_1c, for children aged 5 to 24 years (i.e., no increase) was 31 $mmol.mol^{-1}$ (Saaddine et al., 2002). In the CHASE Study, the mean of HbA_1c among 4,796 multi-ethnic children aged 9-10 years was 33 $mmol.mol^{-1}$ (Whincup et al., 2010). Methods for measuring of HbA_1c varied across this and the cited studies, so some of the differences in HbA_1c could be related to that. However, these are children born to mothers with diagnosed GDM, so it was expected in this cohort of high-risk children that HbA_1c would be higher. Three (3%) of the children had an HbA_1c 41 $mmol.mol^{-1}$ or higher.

Studies with children who were born to mothers without GDM and the studies with adult participants are included in this discussion of the results. Firstly, because there are no other similar studies for comparison, and secondly, because comparing the eating patterns between studies is somewhat difficult due to differences in dietary assessment and the statistical techniques for deriving eating patterns (Table 7.17).

Following the discussion, the strengths of the study will be highlighted and acknowledging the limitations of the study. Recommendations are made for future research on eating patterns and the risk of developing T2DM in children.

Table 7.17. Characteristics of the articles included in the present discussion

Reference	**Study design**	**Number and age (yr) of participants (child unless otherwise specified)**		**Diet assessment method**	**EP analysis method**	**Focus of article**
Present body of work	Cross-section	99,	9	FFQ	CA	EP and T2DM in children
Alexy et al. (2004)	Cohort	228,	2-18	3-day weighed dietary record	CA	EP and BMI
Boone-Heinonen et al. (2008)	Cohort	9,251,	11-21	FFQ	CA	EP, PA and SB
Smith et al. (2011)	Cross-section	8279,	6.75	FFQ	CA	Comparison of EP derived by CA and PCA
Wall et al. (2013)	Case-control	550, 591,	3-4 7-8	FFQ	PCA	EP and SEB and obstetric factors among SGA and AGA
Craig et al. (2010)	Cross-section	1,233,	5-11, 12-17	FFQ	PCA	EP, SEB, PA and obesity
Howe et al. (2013)	Cross-section	681,	15.8	FFQ	PCA	EP and body composition
Aranceta et al. (2003)	Cross-section	3,534,	2-24	24-h recall and FFQ	FA	EP and SEB
Moreira et al. (2010)	Cross-section	1976,	5-10	FFQ	FA	EP, SEB, PA, sleeping and obesity
Fung et al. (2004)	Cohort	69554,	38-63	FFQ	FA	EP and T2DM in adult women
van Dam et al. (2002)	Cohort	42504,	40-75	FFQ	FA	EP and T2DM in adult men
Schumacher et al (2014)	Cross-section	332,	13	FFQ	Food score	EP among girls in low income communities
Ambrosini et al. (2016)	Cohort	6722,	7, 10 and 13	3-day food diaries	RRR	EP and adiposity
Alhazmi et al. (2014)	Review	> 20		Varied	Varied	EP and T2DM in adults
Esposito et al. (2014)	Review	>20		Varied	Varied	EP and T2DM in adults

Reference	Study design	Number and age (yr) of participants (child unless otherwise specified)	Diet assessment method	EP analysis method	Focus of article
Bristow (2010)	Cross-section	147, 2	FFQ	-	Food, activity and body composition in children
Savila et al. (2014)	Cohort	646, 4 and 6	FFQ	-	Tracking of food frequency among 4 and 6 yr Pacific children
Donin et al (2014).	Cross-section	2017, 9-10	24-h recall	-	Energy intake in children and T2DM
Anderson et al. (2016)	Cross-section	239, 5-17	24-h recall	-	dietary intake and eating behaviours of obese children and adolescents considered indigenous versus non-indigenous children
Bacha et al. (2003)	Cross-section	50, 13	-	-	Ethnicity and T2DM in obese children
Gillman et al. (2010)	RCT	199, 4-5	-	-	GDM treatment effect on offspring obesity
Landon et al. (2015)	RCT	905, 5-10	-	-	GDM treatment effect on offspring health and obesity
Saadine et al. (2002)	Cross-section	7968, 5-24	-	-	HbA_1c for children and young adults
He et al. (2015)	Cohort	421, 16.87 ± 2.19	-	-	Abdominal obesity and metabolic syndrome
Tayyaba et al. (2015)	RCT	500 pregnant women aged 32.14 ± 6.13	-	-	To compare the efficacy of metformin with insulin in GDM in terms of foetal and maternal outcome
Whincup et al. (2010)	Cross-section	4796, 9-10	-	-	T2DM precursors including HbA_1c and ethnic differences
Zhu et al. (2016)	Cohort	661, 7	-	-	Offspring growth and obesity and exposure to maternal hyperglycemia

Abbreviations: AGA, appropriate for gestational age; BMI, body mass index; CA, cluster analysis; EP, eating pattern; FFQ, food frequency questionnaire; HbA_1c, glycated haemoglobin A_1c; PCA, principle components analysis; SD, sedentary behaviours, SEB, socioeconomic background; SGA, small for gestational age; RCT, randomized controlled trial; yr, years old

7.4.1 Eating patterns, foods consumption and T2DM

In the current study, generally the ‘energy dense and bread’ eating pattern was the strongest derived eating pattern. It is likely that this eating pattern would provides a higher energy intake and high glucose load (white bread) compared to the other three derived eating patterns. After adjusting for waist-to-height ratio, the concentration of HbA_1c showed a clinical (1.4 $mmol.mol^{-1}$) but not statistically significant difference, between ‘refined carbs’ and ‘not refined carbs’ eating pattern. This could be explored further in future studies.

In adult studies, there is a relationship between consumption of foods with whole grain and fibre (S. Liu, 2002; Ye et al., 2012) and more plant based foods (Alhazmi et al., 2014; Esposito et al., 2014), with reduced risk of T2DM. In addition, from adults cohort studies (Nurses’ Health Study and the Health Professionals Follow-Up study), it was found that eating patterns with more plant based foods including fruit and vegetables and less processed and fast foods, were associated with lower risk of T2DM (Fung et al., 2004; van Dam, Rimm, et al., 2002). However, in these studies risk of T2DM was not measured by HbA_1c and the definitions of T2DM were complex and varied. The diagnosis of T2DM was either self-reported (Ye et al., 2012) or participants met one or more criteria based on the classic symptoms of diabetes (e.g., excessive thirst, polyuria, weight loss, and hunger) and had an elevated fasting glucose concentration (> 7.8 $mmol.L^{-1}$), or elevated non-fasting level (>11.1 $mmol.L^{-1}$. In addition if participants were asymptomatic, but plasma glucose concentrations was elevated on at least 2 different occasions (as defined earlier) or abnormal oral glucose tolerance test result (>200 $mg.dL^{-1}$ 2 hours after glucose load); and receiving hypoglycemic treatment for diabetes (Fung et al., 2004; van Dam, Rimm, et al., 2002).

The identification of eating patterns by clustering considered the whole diet rather than nutrients, single foods or food groups or eating occasions, the assessment of diet is complex and should be examined from many angles. For the purpose of identifying the five eating patterns in the present study, food choices were looked at and Ministry guidelines were adhered to. The number of children in the present analysis was relatively small making differences by gender, ethnicity, and diversity underpowered.

7.4.2 Eating patterns and food choices among children

There are similarities between the foods and subjective names of the derived eating patterns in the current study and two other studies on eating patterns of children within

almost the same age range (Craig et al., 2010; Wall et al., 2013). In both studies the eating patterns with more fruit and vegetables were named ‘healthy’ and eating patterns with less healthy food options like candy bars, soft drinks, and snacks were named ‘snacks’ and/ or ‘puddings’ (Craig et al., 2010), and ‘junk’ (Wall et al., 2013). Among 9-year MiGTOFU children, two characteristics of the two strongest eating patterns: ‘energy dense and bread’ and ‘healthy’ that explained 34% of total variance, were similar to the derived eating patterns from the NZ birth cohort. The ‘energy dense and bread’ pattern, which was identified by foods high in energy, fat, and sugar and the ‘healthy’ pattern, which was characterized with fruit, vegetables, and meat were very similar to the ‘junk’ and ‘healthy’ patterns out of the three derived eating patterns in NZ birth cohort study (Wall et al., 2013). The difference between the ‘junk’ pattern in the NZ birth cohort and the ‘energy dense and bread’ pattern in the current study was that children who had similar pattern of consuming bread (white or other types) were clustered within the ‘energy dense and bread’ pattern, while in NZ birth cohort this was part of a ‘healthy’ pattern. This demonstrates the subjectivity of naming of the eating patterns. In the NZ birth cohort only European children were followed up due to the poor response rate of other ethnic groups (Wall et al., 2013).

The strongest eating pattern, energy dense and bread, explained 18% of the 65% of variance explained. This eating pattern included 34 of the 99 children, the majority of boys and non-Europeans frequently consumed fast foods and beverages. However, based on the daily frequency of consumptions of food items for all the children water, fresh fruit and vegetables, milk, other types of bread, white bread, yogurt, cereals and crackers were the most common consumed food items ($\geq$ 0.43 times). This highlights the difference when considering the whole diet of a child rather than averaging the intake of individual foods of a study group.

The second derived eating pattern was ‘healthy’ in which 13 children with similar frequency of consumption of fruit, vegetables, and meat were clustered. Although this eating pattern was appeared as the second one in this study, the explained variance compared with ‘energy dense and bread’ pattern was almost similar (15.7% compared to 18.1%).

The third eating pattern was characterized by similar frequencies of consumption of refined carbohydrate foods like dumplings, noodle, eggs, and legumes. The reported types of legumes in this study are traditionally more often consumed in Asian, Latin

American, African, India, Middle Eastern countries, and ethnic groups (de Almeida Costa, da Silva Queiroz-Monici, Pissini Machado Reis, & de Oliveira, 2006). On the other hand, meal dishes from the named countries are usually served with rice, noodle or foods, which had rice as the main ingredient like dumplings. Although the number of children by ethnicity in this eating pattern is not powered to reveal differences by ethnic group, this eating pattern was still similar to CNS 2002 findings (Ministry of Health, 2003) in which mixed dishes with pasta and noodles were eaten weekly by about half of NZ children.

Breakfast was identified as the fourth eating pattern with higher consumption of milk and dairy products, and breakfast cereals in the current study. Forty percent of NZ children ate breakfast cereals at least once a day and thirty eight percent reported to drink milk every day (Ministry of Health, 2003). Moreover, boys' daily consumption of breakfast cereals at the age of 7-10 year was higher than the girls' (51% vs 32%), but age or sex was unrelated to milk consumption (Ministry of Health, 2003). In the current study 56 and 67 of children had foods more than once a day from cereals and milk (except dairy products) and this can explain the identified 'breakfast' pattern.

The last eating pattern, 'survival' included only water and children who were just similar in daily frequency of drinking water were clustered together. This eating pattern was more frequent among European children than non-European ($p = 0.004$).

Comparing these five derived eating patterns with the three derived eating patterns in the Avon Longitudinal Study of Parents and Children (ALSPAC) showed that the food items in 'refined carbs, eggs, and legumes', 'breakfast' and 'survival' eating patterns among 9-year MiGTOFU children were clustered all together under the name of the 'processed' eating pattern in ALSPAC study (Smith et al., 2011). The greater number of eating patterns compared to ALSPAC study may be due to the large number of participants in that study (n = 8279) (Smith et al., 2011) compared to the number of children in this study (n = 99), and their greater number of food groups (57 from 94 item FFQ) compared to 12 food groups (38 item FFQ) in this study.

Overall, these MiGTOFU children reported an omnivorous mixed diet. Except for the fruit food group, the other food groups including vegetables, milk and milk products, meat and other protein, and bread and cereals were not consumed in adequate quantities for health based on the NZ food guidelines for children (Ministry of Health, 2014b). In contrast mothers' reported that almost nine in ten children consumed from "occasional"

as defined by the MOH (Ministry of Health, 2007, p. 8), energy dense and nutrient poor foods at least three times per week. Overall, more than half of the 9-year MiGTOFU children consumed fruit and vegetables ≥ 5 times per day, but only seven children followed the NZ evidence-based food guidelines for all the five food groups per day (Ministry of Health, 2014b). There is convincing research-based evidence that fruits, vegetables, beans/legumes, nuts/seeds, whole grains, fish, yogurt, fiber, seafood omega-3s, polyunsaturated fats, and potassium are protective; and of unprocessed red meats, processed meats, sugar-sweetened beverages, glycemic load, *trans*-fats, and sodium harmful with causal cardiometabolic effects (Micha et al., 2017).

It could be argued that poor food choices could be driven by food insecurity or limited time. On the other hand, the most frequently consumed foods, including takeaways, may reflect that the cost of the foods may not be an issue for this study group. It might be more related to the significant changes of household expenditures in the last three years to 30 June 2016, that the average weekly expenditure on restaurant meals and ready-to-eat food compared to fruit and vegetables had doubled (28% compared to 12%) (Statistics New Zealand, 2017). Cultural attitudes and habitual practices will also influence the willingness to provide healthful and nutritious foods (Kumanyika, 2008). Differences in cultural beliefs and practices related to food also apply to eating patterns of adults and therefore, how adults feed their children and the chose foods go intergeneration (Kumanyika, 2008). In addition, young children have their earliest experience with food and eating within home environment, which is shaped by parents (Zarnowiecki, Sinn, Petkov, & Dollman, 2012). Furthermore, not only mothers' beliefs and intentions might be involved in shaping children's beliefs and intentions into adulthood (Sumodhee & Payne, 2016), but also mothers' energy intake is a significant predictor of children's energy intake (Gluck et al., 2009). Meanwhile, the 9-year MiGTOFU children were born to mothers with GDM, who had higher risk of developing T2DM themselves and were among highly recommended group of the community to follow healthy balanced diet.

Considering the GI of consumed foods in this study and therefore the glycaemic load of the diet, generally both girls and boys consumed low GI group, foods such as fruit and vegetables more frequently than medium and high GI food groups. Foods with low GI promote fat oxidation and increase post-prandial satiety (Brand-Miller, Holt, Pawlak, & McMillan, 2002). Girls significantly (p = 0.001) had higher consumption of foods with medium GI compared to boys. This is while 52 of children consumed high GI foods

more than five times a day and half of them had a “normal” BMI. Notably, the quantity of foods consumed was not collected in this study. Therefore, only generally it can be concluded that these children are apparently healthy. But as the childhood eating behaviours may persist into adulthood (Leech et al., 2014), if this pattern of eating continue to older ages, it might affect their health and increase the risk of T2DM.

The main sources of dietary fibre for NZ children (70%) were bread, potatoes, kumara and taro, fruit, breakfast cereals and vegetables(Ministry of Health, 2003). Among the five derived eating patterns except ‘survival’ and ‘refined carbs, eggs and legumes’ pattern, the other three eating patterns included at least one of the named dietary sources of fibre for NZ children. However, legumes (lentils, kidney beans, and soybeans) characterized in the ‘refined carbs, eggs, and legumes’ eating pattern can be considered as a source of dietary fibre. Generally, among 9-year MiGTOFU children based on the strength of the derived patterns and their overall nutrition profiles, ‘healthy’ eating pattern could be considered as an eating pattern, which provided more dietary fibre as a marker of whole/unrefined foods.

7.4.3 Eating patterns and current body size measurements

There was a significant and substantial difference between the breakfast eating pattern and the other four patterns, which were different for both weight z scores and height z scores. These differences in body measurements could be due to the higher number of overweight and obese children in this pattern (n = 11) with higher physical measurements compared to smaller number in the other patterns (‘energy dense and bread’ pattern n =10, ‘healthy’ pattern n = 4, ‘refined carbs, eggs and legumes’ pattern n = 3 and ‘survival’ pattern n = 6). On the other hand, no associations were found between eating patterns and other body measurements including BMI, which has been applied as an indicator of excess weight in previous studies on adolescents and similarly no association was not found between BMI and eating patterns (Alexy et al., 2004; Craig et al., 2010; Schumacher et al., 2014). These paradoxical findings could be related to cross sectional measurements, ethnic differences in body composition and growth patterns. This highlight the need for further follow-up of 9-year MiGTOFU children.

A significant difference between eating patterns and visceral abdominal fat (p = 0.031) was found among children with the ‘breakfast’ eating pattern. Similar to adults, children’s visceral abdominal fat distribution compared to whole body fat appeared more strongly related to the risk for T2DM and CVD (Bacha et al., 2003; Weiss et al.,

2003) and is the most important determinant of glucose metabolism (Marcovecchio et al., 2005). A follow-up examination of 5-12 year-old adolescents (n = 421) in the Penn State Children Cohort to investigate the relationship between abdominal obesity and metabolic syndrome, showed that abdominal obesity and insulin resistance had the strongest association compared to lipid-based or blood pressure-based components (He et al., 2015). Based on the findings, it can be suggested that children with the 'breakfast' pattern characterized by high GI foods and the highest visceral abdominal fat may be at higher risk of developing T2DM compared to the children in the other four eating patterns.

7.4.4 HbA_1c

In current study, out of a total of 99 children, two girls with 'other' and 'Pacific' ethnicities and a European/ Caucasian boy were diagnosed with HbA_1c concentration ≥ 41 $mmol.mol^{-1}$. No relationship was found between ethnicity and HbA_1c concentration, which was maybe due to limited number of children by ethnicities or that all children had been exposed to hyperglycaemia during pregnancy and therefore all were high-risk.

In contrast to the current study, the measurement of HbA_1c concentration of children in the CHASE Study (n = 4,796, aged 9–10 years old) showed that the ethnic group differences in T2DM markers mostly followed adult patterns and were present in apparently healthy UK children at the end of the first decade (Whincup et al., 2010). CHASE Study is a school-based investigation of the health of British children, living in London, Leicester, and Birmingham (Whincup et al., 2010). The HbA_1c concentration of children (mean = 33 $mmol.mol^{-1}$) was reported based on their parental ethnicities: white European, South Asian, other Asian, black African-Caribbean and other (Whincup et al., 2010). Some other biomarkers like insulin and glucose and anthropometric measurements were assessed as well (Whincup et al., 2010). It was reported that in age-adjusted comparison with white Europeans (n = 1,153 mean HbA_1c concentration = 33 $mmol.mol^{-1}$), South Asian children (n = 1,306, mean HbA_1c concentration = 34 $mmol.mol^{-1}$) had higher concentration of HbA_1c with 2.1% difference (95% CI [1.6, 2.7]) (Whincup et al., 2010).

7.4.5 Current body size measurements and HbA_1c concentration

Central fat mass (waist-to-height ratio) may reduce glucose disposal and increase insulin resistance. In the current study, however, there was a small negative correlation between HbA_1c concentration and both waist and waist-to-height ratio, but not with

other body measurements including IOTF cut-offs. In NHANES III study, the mean of HbA_1c concentration was higher in overweight children than the non-overweight ones ($p < 0.01$) (Saaddine et al., 2002). Saaddine and colleagues (2002) interpreted that differences in HbA_1c concentration, which were modest and within a normal range, this might be related to genetic or other factors related to HbA_1c metabolism. This also suggest that it might reflect higher average glycaemia over the past 2-3 months or it may be related to some level of relative insulin resistance.

7.4.6 Influence of early life factors

Positive associations between lower maternal education and eating patterns that includes poor nutrients foods such as junk foods has been reported in previous studies (such as Aranceta et al., 2003; Moreira et al., 2010), though no association was found in 9-year MiGTOFU study.

The food consumption of 147 of the Auckland MiGTOFU children at the age of two was investigated by Bristow (2010). Similar to her findings for the two-year-old children, more than half of the nine-year-old children in 9-year-old MiGTOFU consumed fruit at least twice a day. The consumption of vegetables at the age of two was higher (76% ate vegetables twice per day compared with 65% at nine years) (Bristow, 2010). This may be related to the relative ease to feed small children a variety of vegetables and fruit more often as the portion size is smaller and therefore the cost is less. A national survey commissioned by the MOH (Clinical Trials Research Unit, 2010) reported for 2,503 NZ children and young people aged 5-25 years (weighted to match the NZ population), that responses to the dietary habit questionnaire showed that more children met the guidelines for fruit consumption (68.6%) than vegetables (39.7%). The proportional difference for fruit and vegetables consumption of the 9-year MiGTOFU children was similar.

However, at the age of two years almost half of the children from the three named ethnicities had sufficient fruit consumption (≥ 2 per day) (Indian 43 %, Chinese, 67% and ‘Other’ was not reported due to small number of participants, n = 4) (Bristow, 2010). In the present study, one third of Indian, Chinese and ‘Other’ children consumed fruit at least twice a day suggesting a possible decline in fruit and vegetable consumption in these older children.

At the age of two years, insufficient breads and cereals (36% consumed breads and cereals at least four times a day) were consumed (Bristow, 2010) compared to the nine

years (63% ≥ 4 times/day). Rice is in the top ten foods of the household that were still bought when money was limited (Rush et al., 2007) and Pacific children were more likely to eat rice weekly than Māori and NZ European and Others (Ministry of Health, 2003). Among the 9-year-old children in this study, the type of rice (white or brown) was not recorded. However, the findings from the two-year-old children showed that white rice was more preferred than the brown rice (70% vs < 5%, consumed at least twice a week) and by ethnicity more Asian (Chinese and Other Asian) and Indian children compared to European ones consumed rice at least twice a week (Bristow, 2010). Forty of the children at the age of nine years, consumed rice at least once a day, which compared to the frequency of consumption at the age of two years, shows an increase in rice consumption. Notably, in the 9-year-old MiGTOFU study, consumption of rice was grouped with bread and cereals food group.

More evidence of food choices tracked through childhood shows that white bread was the most commonly consumed bread (at least twice a week) at the age of two years (60%) (Bristow, 2010; Rowan et al., 2008), and at nine years a similar proportion of children in the 9-year-old MiGTOFU reported the same rate of consumption. At the age of 9 years, the overall consumption of high energy group foods (i.e., roasted/ chips potatoes, chippies/ crisps, crackers, sweet biscuits, lollies/ sweeties, chocolate, and takeaways) was at least three times per week by 86% of the children. At the age of two, the consumption of treat foods (biscuits, crisps, lollies, muesli bars, and chocolate bars) was at least once a day by 77% and takeaways by 73% at least twice per week, and fries by 23% at least twice per week (Bristow, 2010). This suggests a decrease in consumption of high energy foods at age nine years. This decrease between age two and nine years was in contrast with the key findings in 2008 national survey (Clinical Trials Research Unit, 2010) in which consumption of foods high in fat, sugar, and/or salt (such as takeaways, meat pies or sausage rolls, and confectioneries), increased with age. This finding was also different from the findings of Pacific children, who had similar frequent consumptions of snack foods such as crisps and noodle, food drinks, and powdered fruit drinks at the age of four and six years (Savila et al., 2014).

At the age of nine years 62% of children had sugary drinks at least once a day, while this was 36% at the age of two years (Bristow, 2010). It can be concluded that overall the consumption of sugary drinks among these children increased and this is similar to the increase in consumption of soft drinks with age (Clinical Trials Research Unit, 2010).

7.5 Limitations and strengths

The study findings by ethnicity, gender, and GDM treatment of the mothers are limited because of the relatively small number of children in the overall sample. While a number of other covariates and cofactors could have been included in the analysis, the small sample size means that the possibility of Type 1 and Type 2 statistical errors increases with the data partitioning. In addition physical activity was not objectively measured and the effect of activity could change the glycaemic profile.

Dietary tools are the best way to find out what people are eating with minimum participant burden, but tools including the FFQ have a possibility of missing and/ or under- or over-reporting of the foods that children eat. In addition, the conversion of foods eaten into the number of times/ day was with integral rather than continuous data, so precision was lost. On the other hand, in this study, the food consumption information relied on parental estimates, which can be an accurate measure of children's food consumption at home, but it might be less accurate when parents reported food consumed away from home like school.

Consumption of bread based on its type (white and other types) showed that both girls and boys had a higher consumption of other types of bread (total median = 0.57/day) compared to white bread (total median = 0.43/day), but this difference was not statistically significant either by type of consumed breads or by gender. Furthermore, children with similar frequency of bread (white and other types of bread) consumption were clustered in 'energy dense and bread' eating pattern in which fast foods were included as well. The FFQ used in the 9-year MiGTOFU study, 'other types of bread' was not explained clearly by providing examples such as flatbread and/ or mixed grain. This study was done among a multi-ethnic group, so the ingredients of 'other type of breads' might be mainly from white flour with higher GI compared to 'whole meal' and 'mixed grain bread' which are considered healthier types of bread.

The analysis to compare the FFQ of the same children at age two with that collected at age 7-9 years was not done. The tracking of food patterns through the life course needs to be better understood and could be a future investigation.

Findings only related to 7-9 year-old children born to mothers treated for GDM in Auckland, so these findings may not be applicable to other groups and future children. The strengths of the study include the measurement of HbA_1c concentration in young

children using a venous sample which was analysed in the same day by an accredited laboratory and the eating patterns were identified which accounted for 65% of the variance.

7.6 Recommendations

Future work should explore other factors related to risk for T2DM such as insulin and insulin resistance which are more sensitive measures. These were measured in this study but were available at the time of writing the thesis. Others have shown that the rate of absorption of glucose after consumption of high GI foods like refined breads and rice is fast and that may induce appetite and weight gain (Ludwig et al., 1999). In children, higher total energy intake assessed by 24-hour food recall is associated with increased levels of insulin resistance measured by homeostasis model assessment (HOMA) (Donin, Nightingale, Owen, Rudnicka, Jebb, et al., 2014). Donin and colleagues (2014) demonstrated this relationship in a large multi-ethnic cross-sectional study among 2,017 children at the age of 9 to 10 years in the CHASE Study, where body composition, fasting blood glucose, HbA_1c, serum insulin, and homeostasis model assessment (HOMA) were reported (Donin, Nightingale, Owen, Rudnicka, Jebb, et al., 2014). No association was found between total energy intake with HbA_1c, and the mean of HbA_1c in this study was relatively low at 34 $mmol.mol^{-1}$ (Donin, Nightingale, Owen, Rudnicka, Jebb, et al., 2014). Differences by ethnicity were not reported. The same study (Donin, Nightingale, Owen, Rudnicka, Perkin, et al., 2014) reported that those eating breakfast, everyday particularly one with a high fibre content, had a lower insulin resistance than those who did not eat breakfast every day.

Children’s food choice can be influenced by early intervention and guidance (Reinehr, 2013), which lead them to a healthier diet or increase the interest for choosing unhealthier options such as noodles and/or fruit juices. To this aim, the education system can play a significant role in promoting a healthy eating culture at schools. Following a diet including more plant foods, and less high calorie, processed foods, can reduce the risk of obesity and T2DM.

The findings from animal studies (including Aerts & Van Assche, 2006; Harder et al., 2001) suggest that normalization of maternal blood glucose concentration during pregnancy can reduce the negative effects on offspring, including the induced risk of developing diabetes and obesity. Therefore, encouraging pregnant mothers to have routine appointments with health providers like DHBs or Plunket to follow the routine

clinical check-up and consider the received consultations on controlling the blood glucose concentration, especially if it is not in the range for pregnant women, can reduce future health risks in their children.

7.7 Conclusion

The concentration of HbA_1c showed a clinical difference (1.4 $mmol.L^{-1}$) albeit not statistically significant between ‘refined carbs’ and ‘not refined carbs’ eating patterns after adjusting for waist-to-height ratio. Key foods of less healthy eating patterns that could targeted include white bread, rice, added-sugar foods, high energy, and poor nutrient foods. This could include reformulation by industry, sugar tax and education about healthier food swaps. Generally, more follow-up of these children who were born from mothers with GDM is required.

Chapter 8 Discussion and conclusion

Using cluster analysis, this study aimed to identify eating patterns among children and exploring the associations of these with T2DM screening biomarkers, taking into consideration, body size. To address this aim, eating patterns and risk factors in children from two birth cohorts; the MiGTOFU and PIF main and nested sub-studies, were investigated.

Firstly, it was found that the percentage of children who were diagnosed with $HbA_1c \geq 41$ mmol.mol^{-1} in 9-year MiGTOFU and PIF main studies were 3% and 4.2% respectively. This finding shows that the cohort of children of 9-year MiGTOFU study who were six years younger than the Pacific children had a higher proportion of children with elevated HbA_1c concentration. It is expected that as this high-risk cohort ages the proportion will continue as the prevalence of diabetes increases with age e.g., in the NZ population of 24 to 34 year old in 2006/07 was 0.2% and in 2016/2017 was 0.8 (Ministry of Health, 2017a). In the PIF sub-study, SUA was measured as well as HbA_1c. This revealed that 55% of the boys and 30% of the girls had SUA higher than the gender specific cut-off points for hyperuricemia. In addition, the prevalence of overweight and obesity among children who participated in both PIF main study and 9-year MiGTOFU study was more than 70% and 30%, respectively. This confirms the rapid growth and weight gain among children who are still growing and developing. This study has shown that increased body mass is considered one of the main risk factors for developing T2DM across life course.

Secondly, in both MiGTOFU and PIF main studies, the strongest patterns of eating represented clusters of children who consumed energy dense and high in fat and sugar foods. This can be explained by the fact that ‘occasional’ and ‘energy dense and bread’ eating patterns in PIF main and 9-year MiGTOFU are usually cheaper than healthier options such as fruit and vegetables for children living in Auckland. On the other hand, the findings from PIF sub-study have showed SUA concentration was significantly associated with both FFM and frequency of daily consumption of sugary drinks.

The prevalence of food insecurity, which is an indicator of socio-economic hardship, is increasing for NZ adolescents. It is reported that 40% of secondary school students in NZ (i.e., two out of three Pacific and half of the Māori adolescents) (Utter, Izumi, Denny, Fleming, & Clark, 2017) are food insecure. NZ children’s limited access to

healthy and nutritious food choices can potentially affect their well-being. Although NZ is one of the 25 countries with high income in Organisation for Economic Co-operation and Development, unfortunately fast food consumption is an independent and positive predictor of the mean BMI of adults New Zealanders (De Vogli, Kouvonen, & Gimeno, 2014). This highlights the need to provide environments that are more supportive of "healthy" eating and food choices. The education system can play a significant role in promoting a healthy eating culture at schools. The Health Promoting Schools (HPS) approach is an example of how to promote this aim. It was developed by the WHO in 1980s with the aim of improving the capacity of the schools as a healthy setting for living, learning and working (Ministry of Health, 2012c). Since 1991 many schools in NZ joined HPS, to address all aspects of hauora- physical, mental, emotional, social and spiritual wellbeing (Ministry of Health, 2012c). However, due to lack of infrastructure (i.e., long-term strategic plan), and robust evaluation, HPS has been limited (Ministry of Health, 2012c). In addition, Project Energize takes an HPS approach. The programme has been supported by the Waikato District Health Board in all primary schools in Waikato region since 2009 and the Northland and Capital Coast District Health Boards also fund Energize services so that over 70000 children receive the Energize treatment (Rush, Cairncross, et al., 2016). Therefore, more support of such frameworks at schools is needed.

Household budget management and food selection workshops and short courses for parents may build individual awareness, which may lead to healthier eating patterns for the family (De Vogli et al., 2014; Rush et al., 2007). In addition, government could support and regulate the nutritional quality of fast-foods and sugary drinks; major sources of food and defined eating patterns for the high-risk children investigated in this thesis. Specific current actions include ways of frying chips to reduce fat content (The Chip group) to reductions in the sale of sugary drinks in district health board and council premises (Healthy Together Auckland). Such policies might mitigate food security, obesity, and T2DM epidemic across life course.

Moreover, non-modifiable demographic factors such as ethnicity, age, and obesity account nearly one third of the overall rise in diagnosed T2DM. This is while other factors such as health care were found responsible for the other two thirds (Ministry of Health, 2002b) (Figure 8.1). Nonetheless, children's wellbeing and growth can be improved by experiencing healthy, hygienic and safe housing environment (e.g., dry and less crowded), provision of health and education services, optimal nutrition,

employment opportunities, and improved socioeconomic status (Simpson et al., 2014). This is reinforced by the United Nations and the sustainable development goals, which recognize the need for environmental change (https://www.un.org/sustainabledevelopment/sustainable-development-goals/) to provide a health promoting envirnment.

These “big picture” factors were not specifically addressed in this thesis, but particularly for the PIF study it is known that these children, mainly living in more deprived areas in South Auckland have other factors, other than knowledge, that affect their health and ability to access healthier food.

Currently about 260,000 children in NZ are affected by poverty compared, this number was much smaller 2-3 decades ago (St. John, O'Brien, & Dalem, 2014). Poverty is one of the leading factors contributing to childhood poor health and disease and causes death in NZ (St. John et al., 2014). The life course approach suggests that the development of risk factors (and also protective factors) occurs during childhood (Godfrey et al., 2010) but these factors may not become manifest until late in life.

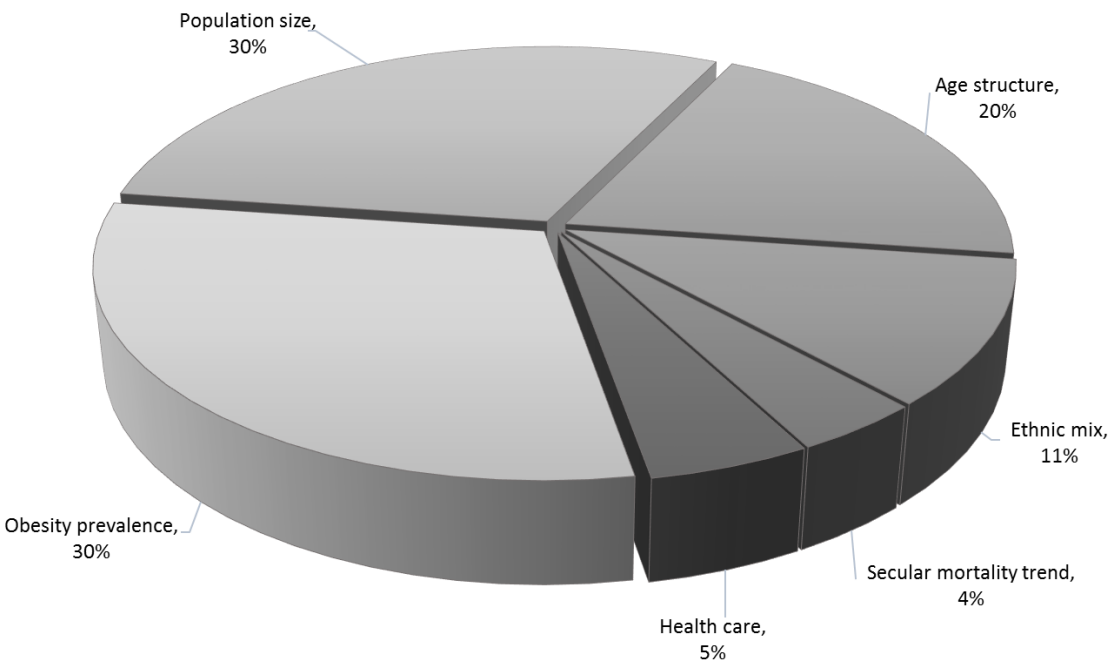


Figure 8.1. Contribution of relative factors to rise in number of diagnosed type 2 diabetes mellitus (Adopted from Ministry of Health, 2002b, p. 8)

Therefore, the Health Select Committee has recommended that the NZ government to continue to progress policies to enhance children's wellbeing and move quickly to reprioritise investment towards practices such as reproductive health and life course nutrition and target vulnerable groups such as Māori and Pacific children (Simpson et al., 2014). The evidence provided in this thesis particularly of the prevalence of obesity, poor eating patterns, elevated HbA_1c, and SUA supports the targeting of Pacific children and children of mothers with GDM for earlier prevention and screening for treatment, and for action at the community level to improve food security.

On the other hand, the annual cost of global healthcare expenditure dedicated to diabetes treatment and related complications has grown 8% since 2015 and reached USD 727 billion (e.g., in the western Pacific including NZ, USD120.3 billion was spent on people with diabetes) (International Diabetes Federation, 2017). Research in the University of Auckland estimated that health care and lost productivity annual costs attributed to overweight and obesity in NZ were between NZD 722 million to NZD 849 million (Faculty of Medical and Health Sciences, 2012).

Mitigating these problems is not possible unless timely diagnosis and appropriate treatment of T2DM, especially among young population are being established (International Diabetes Federation, 2017). In July 2016, the 'Childhood Obesity Plan' was initiated in NZ for the purpose of preventing and managing obesity in children aged ≤ 18 years (Ministry of Health, 2017b). The initiative in this plan that are pertinent to this study are 1) identifying obese children in the national 'Before School Check' programme, 2) subsequent referral to a health professionals for further clinical assessments those children who present ≥ the 90th percentile for weight, and 3) promoting family based 'physical activity, food and nutrition, lifestyle' interventions. School-based interventions, such as Project Energize- which started as a RCT with NZ children living at Waikato district in 2004 (Rush et al., 2012)- or APPLE project- a 'Pilot Program for Lifestyle and Exercise' among NZ children aged 5-12 years (McAuley et al., 2010)- have helped to control the excess weight gain of children and may reduce the risk of future NCDs including T2DM. This is an example of attending to the causes of the causes by changing school eating patterns, activity environments, and increasing the understanding of how to change (e.g., healthy school lunches, water and milk as the best drinks), which would diffuse into the community, and is shared with families.

8.1 Strengths and limitations

This research on NZ children examined for the first time, the relationship between derived eating patterns against biomarkers and measurements for increased risk for T2DM. In particular, children in the PIF main and sub-study were Pacific, which is the third largest ethnic group in Auckland, the NZ's biggest city (Statistics New Zealand, 2013) and are at the highest risk of developing NCDs including T2DM and obesity (Ministry of Health, 2016b). In addition, for the first time the concentration of SUA was measured to assess the correlation between this biomarker and T2DM in Pacific children. Pacific and Māori people, who are both from Polynesia, have a high prevalence of gout (Winnard et al., 2012) compared to other NZ ethnic groups.

Finally, in the 9-year MiGTOFU study the risk of developing T2DM was examined in a multi-ethnic group of children, who has a higher risk of developing T2DM. This was because since they were born to mothers with GDM.

In diet-disease investigations, it is argued that study eating patterns and their associations with the outcome may provide a stronger association compared to identifying associations for single food items with the outcome (Hodge & Bassett, 2016). This study could not find any clear associations of risk with eating patterns, but it was possible to suggest associations. The following list elaborates on the probable reasons for not finding associations between eating patterns and concentration of HbA_1c among healthy children with a relatively high prevalence of overweight and obesity:

1. The age of children in this body of work (9 and 14 years) might be too early to see the associations of the observed factors with the HbA_1c concentration. The children who participated in this study were pre-pubertal. In puberty, children experience increased resistance to the action of insulin and also increased secretion of growth hormone (Kaufman, 2002; Reinehr, 2013). After puberty, insulin often declines to its basal status (Reinehr, 2013). Therefore, puberty as a factor should be considered in future investigations.
2. Children's food information collected through questionnaires. Questionnaires have limited validity in a sense that the patterns are derived based on the data collected. The food questionnaire itself or the subjectivity in the responses entered may not accurately capture the food items or groups of foods that are truly important in determining children's T2DM risk. Moreover, there is a possibility that diet is not the primary driver of risk for T2DM among children at

targeted ages. To verify this assumption children's early life diet should be studied and the findings should be compared with the findings of this current study. It can also beneficial to study the patterns of physical activities among children.

3. Methodological difficulties in assessing diets of children must be considered. Using available dietary assessment methods like FFQ or 24-hour recall indicated that there may be intra-individual errors in self-reporting the food consumption with over or under-reporting of specific foods and complexity of the diet. This is a common research barrier, especially in children who are overweight and obese (Alexy et al., 2004; Craig et al., 2010). It also has been raised in large studies like 2008/2009 NZ adult nutrition survey (Gemming, Jiang, Swinburn, Utter, & Mhurchu, 2014). This leads to total energy misreporting which is a limitation of dietary surveys. Unfortunately, unlike biomarkers, which can be measured and the effects of the interference (such as body size and gender), food responses always include a percentage of missing foods. NZ national health survey report confirms that collecting accurate dietary information from children aged 10 to 14 years is daunting as children of this age are significantly less likely to report 'not buying takeaways' or 'limiting fat' than the young people aged 15 to 19 years (Clinical Trials Research Unit, 2010).
4. The lack of associations between eating patterns with T2DM might be due to cross-sectional data collection in studies included in this body of work.

8.2 Recommendations for future research to inform policy

Type 2 diabetes mellitus has a multifactorial aetiology including strong contributions from obesity and genetics as well as epigenetics. Therefore, considering and following up a combination of variables may contribute to a better understanding of the influence of individual and/ or combinations of factors for developing or increasing the risk of T2DM among children. These variables can include related biomarkers, information about diet and food consumption, physical activity and sedentary behaviours, and body measurements.

Whilst limited to the associations between eating patterns and risk for T2DM, the evidence presented in this thesis has shown that the measurement of HbA_1c via the POCT to screen for T2DM is an easy and viable option that can be used for screening children at school. The findings can then allow early action and understanding by caregivers, parents, the child and health professionals. Actions such as reducing excess

weight, increasing exercise and applying changes in eating patterns can help to prevent or delay the disease. This strategy might be more cost effective and beneficial than medical treatment. Historically, treating children with T2DM is challenging due to the high rate of dropout from the medical care system (Reinehr, 2013) and having limited medicine (only metformin and insulin) licences for pharmacological treating of under 18 years old children with T2DM (treatment goal for HbA_1c concentration < 53 $mmol.mol^{-1}$) (Reinehr, 2013). Measurement of HbA_1c via the POCT can help with early diagnosis of T2DM in Auckland region of NZ which accounts for about 10% of all new cases of diabetes among children (Jefferies et al., 2012).

Longitudinal research studies have the capacity to demonstrate associations between eating patterns, and health outcome measures over time (Leech et al., 2014). In support of the need for longitudinal studies, Almoosawi et al. (2014), highlighted the differences in diet and eating patterns between men and women, and the implication of variations of dietary trajectories on age-related changes in HbA_1c and fasting glucose. There is one NZ prospective cohort study (Wall et al., 2013), of children aged 3.5 and 7 years who were born small for their gestational age. The Wall et. al study focused on associations of eating patterns with socio-demographic and obstetric factors, but HbA_1c was not measured in this study. Wall et al. (2013) suggested that further, longitudinal, investigation should continue to examine at regular intervals the impact of childhood diet on growth and health outcomes, and identify the influence of other factors such as maternal socio-demography, obstetric factors and birth size. Therefore, longitudinal/ birth cohort such as PIF and MiGTOFU birth cohorts are positioned to add to the understanding of how eating patterns track and explore how clusters of patterns may influence HbA_1c and risk of future T2DM.

The reliability of the dietary assessment methods in relevant studies can be improved through feasibility studies and the use of cameras or food capture electronic applications to validate food choices in the demographic of interest. In addition, supervising participants when answering questionnaires by signalling incomplete questions can decrease the percentage of missing information such food consumption.

In the studies of eating patterns utilizing cluster analysis, it has been suggested to consider the differences in gender, age, geographical area, weight status and socio-economic status considered (Devlin et al., 2012). While, in the two studies on children, PIF main study and 9-yr MiGTOFU, the majority of the identified factors were

considered, the findings were in contrast to the study hypothesis. Including the patterns of children's physical activities as a confounder for adjustments especially in regression models, along with common cofounders such as gender and age can be beneficial. In both presented studies, PIF main study and 9-yr MiGTOFU, the number of children were inadequate to allow adjustment by gender. The longitudinal nature of the research made it also impossible to recruit more children to be studied. However, in Project EAT (Eating Among Teens), which investigated the association between eating patterns and weight status in adolescents participated (n = 4,746) the identified models were adjusted for socio-economic status and physical activity (Cutler et al., 2012). Eating patterns may not be the main determinant of weight status at this age, but at later ages previous behaviours may have more of an impact.

In this study, the eating patterns and their associations had two main limitations: firstly, the analysis was considered for data collection at a specific time (cross-sectional), and secondly the children from both MiGTOFU and PIF study were rapidly growing and changing. Tracking the patterns of eating and investigating their relationships with risks of incident T2DM could provide a more accurate research output. Other than this, it could make it possible to examine the stability of childhood eating patterns into adulthood. An example of this study design can be found at Whitehall II study among 7731 adults with the mean age of 50 years (Brunner et al., 2008). In this study the prospective association of eating patterns derived from cluster analysis were examined with the long-term effect on the risk of coronary heart disease and T2DM (Brunner et al., 2008). Another example is the cohort of 4,304 healthy Finnish adults aged 40- 69 who were followed up for 23 years. In this study, the risk of incidence of T2DM was examined against eating patterns (Montonen et al., 2005). A recent example of identification of patterns of eating and investigation of their relationship with the incidence of chronic diseases is the Dunedin study (Saeedi, 2017). The aim of this study was exploring the associations between eating patterns and markers of cardiovascular health including fitness and arterial health (i.e., pulse wave velocity and augmentation). Saeedi (2017) concluded that there were no significant associations found between eating patterns and markers of cardiovascular health at age 9- 11 years.

There is literature that compares the results of different analysis methods to identify eating patterns (such as Hearty & Gibney, 2013; Manios et al., 2010; Smith et al., 2011). The aim of such studies was mostly comparing the eating patterns identified by the target analysis methods, for instance cluster analysis vs factor analysis. While the

question addressed by each method are different, for example applying cluster analysis addresses whether there are groups of people with similar eating patterns and factor analysis addresses whether there are food groups correlate in explaining variations in diets (Krebs-Smith et al., 2015; Michels & Schulze, 2005). Moreover, there is a lack of review studies in literature that explored the eating patterns derived by cluster analysis method to identify the distinct groups of children focusing particularly on reproducibility, validity and the effect of energy misreporting.

Longitudinal birth cohort studies provide a unique opportunity to understand better the contributors, drivers, context, and reality of the growth and development of children. This thesis is a snapshot at 9-year MiGTOFU and 14-year PIF and future investigations need to look back, outward, and forward to future generations to further develop our understandings of risks for health. Looking back would include the influence of diet during pregnancy, breastfeeding and weaning foods as early life factors that can influence the health of mother and child. In NZ, 9% (95% CI [7.1, 11.4]) of children were exclusively breastfed until at least they are six months old (Ministry of Health, 2017a). Pacific children with 11.6% (95% CI [7.1, 11.4], girls 16.3% and boys 7.4%) had the highest proportion of exclusive breastfeeding to at least six months, then Asian with 10.0% (95% CI [6.0, 16.1], girls 11.2%and boys 8.2%), European/ Other with 8.7% (95% CI [6.3, 11.8], girls 7.3% and boys 9.9%), and Māori 8.7% (95% CI [6.1, 12.1], girls 9.6% and boys 7.8%) (Ministry of Health, 2017a). It is suggested to support breastfeeding, particularly among lower socioeconomic groups, who are at highest risk of developing T2DM (Nolan et al., 2011) and other NCDs. Interventions to encourage breastfeeding for longer time (i.e., more than first six months) can help children health in adulthood.

Other factors to be considered include the proximal (home) and distal (neighbourhood) environment as conceptualised in the Foresight project. Researchers in the PIF study considered other factors such as the influence of parenting styles, the build environment, physical activity measured with accelerometers, metabolic markers, feelings and resilience, use of technology and media, and school life and friends. The eating pattern data can be added to these and future analyses to create a more vivid picture of the health, development and behaviours of this cohort.

8.3 Conclusion

This investigation has shown that the patterns of eating in high-risk groups of children do not meet the NZ food guidelines and that the patterns are associated albeit weakly with the risk factors for T2DM and obesity. The NZ eating and activity guidelines are based on the best current evidence for health, but are not followed by the majority of the population, and the children examined in this thesis. This emphasises the need to support initiatives that improve access to food across the life course, but in particular for children. Therefore, their life course potential is maximised. At all life stages there is potential to reduce risk for and delay onset of T2DM and at younger ages this is likely to have a larger effect. Food is one focus for prevention. Strategies that promote healthier early life eating patterns could orientate children, pregnant women, and their families to healthier patterns that could persist into adolescence and adulthood. To this aim, an intergenerational approach is needed.

There is a need to better promote guidelines for healthy food shopping and choose foods more wisely based on their ingredients (e.g., sugar content) rather than just the price, especially for families living in crowded homes in low socioeconomic areas. In addition, to prepare healthier meals for the family, children could be taught basic cooking skills and be involved in food preparation. This would encourage children from early age to prepare homemade food rather than ready-to-eat food. In addition, more control of food and drink options should be kept at schools. Multidisciplinary inputs with contributions of primary health practitioners, epidemiologists, nutritionists and dieticians can improve the patterns of eating and health of children in the community. There is a need to establish more rigorous policies to limit the access to and availability of less healthy food options. These policies may include taxing sugary drinks, imposing more control of food-chain fast foods and their serving size and the amount of energy and nutrients of their foods. On the other hand, price reduction policies and controlling and monitoring the prices of fruit and vegetables in food markets can encourage families, particularly families living in lower socioeconomic areas to visit these sites more often.

Finally, Auckland has a relatively metropolitan and diverse multi-ethnic population and food preparation and consumption more or less is under the effect of culture and the built environment. In promoting healthy eating, nutrition advice, food guidelines and policies, cultural and environmental differences should be considered in such a way that those messages translate to the majority of people in the community.

This thesis has provided some insights and understanding of the foods eaten by children and on whether or not eating patterns have relationship with biomarkers of risk for T2DM and rapid growth in children with a high prevalence of overweight and obesity.

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Appendix A: Pacific Island Families main study ethics approval

Health and Disability Ethics Committees

Health and Disability Ethics Committees
C/- MEDSAFE, Level 6, Deloitte House
10 Brandon Street
PO Box 5013
Wellington

0800 4 ETHICS
hdecs@moh.govt.nz

04 December 2013

Professor Janis Paterson
HY - Akoranga Campus
Auckland University of Techonology
Private Bag 92006
Auckland 1142

Dear Professor Paterson

Re:	**Ethics ref:**	**13/STH/159**
	Study title:	Pacific Islands Families Study: Transitioning Through Adolescence

I am pleased to advise that this application has been *approved* by the Southern Health and Disability Ethics Committee. This decision was made through the HDEC-Full Review pathway.

Conditions of HDEC approval

HDEC approval for this study is subject to the following conditions being met prior to the commencement of the study in New Zealand. It is your responsibility, and that of the study's sponsor, to ensure that these conditions are met. No further review by the Southern Health and Disability Ethics Committee is required.

Standard conditions:

1. Before the study commences at *any* locality in New Zealand, all relevant regulatory approvals must be obtained.

2. Before the study commences at *a given* locality in New Zealand, it must be authorised by that locality in Online Forms. Locality authorisation confirms that the locality is suitable for the safe and effective conduct of the study, and that local research governance issues have been addressed.

After HDEC review

Please refer to the *Standard Operating Procedures for Health and Disability Ethics Committees* (available on www.ethics.health.govt.nz) for HDEC requirements relating to amendments and other post-approval processes.

Your next progress report is due by 4 December 2014.

Participant access to ACC

The Southern Health and Disability Ethics Committee is satisfied that your study is not a clinical trial that is to be conducted principally for the benefit of the manufacturer or distributor of the medicine or item being trialled. Participants injured as a result of

treatment received as part of your study may therefore be eligible for publicly-funded compensation through the Accident Compensation Corporation (ACC).

Please don't hesitate to contact the HDEC secretariat for further information. We wish you all the best for your study.

Yours sincerely

Ms Raewyn Idoine
Chairperson
Southern Health and Disability Ethics Committee

Encl: appendix A: documents submitted
appendix B: statement of compliance and list of members

Documents submitted

Document	*Version*	*Date*
CV for CI: CV for Professor Janis Paterson (CI)	1	25 October 2013
Evidence of scientific review: Peer review by the Ministry of Science and Innovation, the Ministry of Business, Innovation, and Employment, and the Health Research Council.	1	28 October 2013
PIS/CF: Primary parent participant full information sheet and consent form; and adolescent youth participant information sheet and assent form.	1	28 October 2013
PIS/CF for persons interested in welfare of non-consenting participant: Parental consent for youth assessment - information sheet and consent form.	1	28 October 2013
Survey/questionnaire: Primary caregiver survey.	1	29 October 2013
Survey/questionnaire: Secondary caregiver questionnaire.	1	29 October 2013
Protocol: PIF:TTA Study Protocol.	1	29 October 2013
Survey/questionnaire: Youth Assessment Protocol.	1	29 October 2013
Application		
Response to Request for Further Information		26 November 2013

Statement of compliance and list of members

Statement of compliance

The Southern Health and Disability Ethics Committee:

- is constituted in accordance with its Terms of Reference
- operates in accordance with the *Standard Operating Procedures for Health and Disability Ethics Committees*, and with the principles of international good clinical practice (GCP)
- is approved by the Health Research Council of New Zealand's Ethics Committee for the purposes of section 25(1)(c) of the Health Research Council Act 1990
- is registered (number 00008713) with the US Department of Health and Human Services' Office for Human Research Protection (OHRP).

List of members

Name	*Category*	*Appointed*	*Term Expires*
Ms Raewyn Idoine	Lay (consumer/community perspectives)	01/07/2012	01/07/2015
Mrs Angelika Frank-Alexander	Lay (consumer/community perspectives)	01/07/2012	01/07/2014
Dr Sarah Gunningham	Non-lay (intervention studies)	01/07/2012	01/07/2015
Ms Gwen Neave	Lay (consumer/community perspectives)	01/07/2012	01/07/2014
Dr Nicola Swain	Non-lay (observational studies)	01/07/2012	01/07/2014
Dr Martin Than	Non-lay (intervention studies)	01/07/2012	01/07/2014
Dr Devonie Waaka	Non-lay (intervention studies)	01/07/2013	01/07/2016
Dr Mathew Zacharias	Non-lay (health/disability service provision)	01/07/2012	01/07/2015

http://www.ethics.health.govt.nz

Appendix B: Pacific Island Families main study physical measurement form

PIF Child ID #: _____

PACIFIC ISLANDS FAMILIES STUDY

The Pacific Islands Families Study: Transitioning Through Adolescence

AUT UNIVERSITY

14-Year Youth Physical Measurements Form

*[**INT:** Measurements to be done three times as indicated below].*

Part 1

1. Grip Strength

a. Dominant hand used — Yes or No — Right / Left

b. Grip strength	(1st)	☐☐ kg		77
	(2nd)	☐☐ kg	± 5 kg	77
	(3rd)	☐☐ kg	± 5 kg	77

2. Body Composition (Bioimpedance Analysis)

*[**INT:** Impedance measurements must be greater than resistance measurements. Resistance measurements must be greater than reactance measurements.]*

a. Impedance	(1st)	☐☐☐.☐ ohms		777.7
	(2nd)	☐☐☐.☐ ohms	± 5.0 ohms	777.7
	(3rd)	☐☐☐.☐ ohms	± 5.0 ohms	777.7
b. Phase	(1st)	☐☐.☐ degrees		77.7
	(2nd)	☐☐.☐ degrees	± 1.0 degree	77.7
	(3rd)	☐☐.☐ degrees	± 1.0 degree	77.7
c. Resistance	(1st)	☐☐☐.☐ ohms		777.7
	(2nd)	☐☐☐.☐ ohms	± 5.0 ohms	777.7
	(3rd)	☐☐☐.☐ ohms	± 5.0 ohms	777.7
d. Reactance	(1st)	☐☐☐.☐ ohms		777.7
	(2nd)	☐☐☐.☐ ohms	± 5.0 ohms	777.7
	(3rd)	☐☐☐.☐ ohms	± 5.0 ohms	777.7

PIF Child ID #: _____

3. Anthropometry

a. Height	(1st)	___ . ___	cm		777.7
	(2nd)	___ . ___	cm	*± 0.5 cm*	777.7
	(3rd)	___ . ___	cm	*± 0.5 cm*	777.7
b. Weight	(1st)	___ . ___	kg		777.7
	(2nd)	___ . ___	kg	*± 0.1 kg*	777.7
	(3rd)	___ . ___	kg	*± 0.1 kg*	777.7

*[**INT:** Alternate waist and hip circumference measurements.]*

c. Waist circumference	(1st)	___ . ___	cm		777.7
	(2nd)	___ . ___	cm	*± 0.5 cm*	777.7
	(3rd)	___ . ___	cm	*± 0.5 cm*	777.7
d. Hip circumference	(1st)	___ . ___	cm		777.7
	(2nd)	___ . ___	cm	*± 0.5 cm*	777.7
	(3rd)	___ . ___	cm	*± 0.5 cm*	777.7

Part 2

1. Blood Pressure Measurement **[Refer if systolic >130 mmHg]**

*[**INT:** Child should be seated for at least 5 minutes before blood pressure measurements are taken.]*

a. Systolic	(1st)	___	mmHg		777
	(2nd)	___	mmHg	*± 10 mmHg*	777
	(3rd)	___	mmHg	*± 10 mmHg*	777
b. Diastolic	(1st)	___	mmHg		777
	(2nd)	___	mmHg	*± 10 mmHg*	777
	(3rd)	___	mmHg	*± 10 mmHg*	777
c. Pulse	(1st)	___	bpm		777
	(2nd)	___	bpm	*± 10 bpm*	777
	(3rd)	___	bpm	*± 10 bpm*	777

2. Blood Sugar Control **[Refer if HbA1c >40 mmol/mol]**

a. Glycated haemoglobin (HbA1c) level	___	mmol/mol	777

PIF Child ID #: _____

3. Anthropometry

a. Height	(1st)	___ . ___	cm		777.7
	(2nd)	___ . ___	cm	± 0.5 cm	777.7
	(3rd)	___ . ___	cm	± 0.5 cm	777.7
b. Weight	(1st)	___ . ___	kg		777.7
	(2nd)	___ . ___	kg	± 0.1 kg	777.7
	(3rd)	___ . ___	kg	± 0.1 kg	777.7

*[**INT:** Alternate waist and hip circumference measurements.]*

c. Waist circumference	(1st)	___ . ___	cm		777.7
	(2nd)	___ . ___	cm	± 0.5 cm	777.7
	(3rd)	___ . ___	cm	± 0.5 cm	777.7
d. Hip circumference	(1st)	___ . ___	cm		777.7
	(2nd)	___ . ___	cm	± 0.5 cm	777.7
	(3rd)	___ . ___	cm	± 0.5 cm	777.7

Part 2

1. Blood Pressure Measurement

[Refer if systolic >130 mmHg]

*[**INT:** Child should be seated for at least 5 minutes before blood pressure measurements are taken.]*

a. Systolic	(1st)	___	mmHg		777
	(2nd)	___	mmHg	± 10 mmHg	777
	(3rd)	___	mmHg	± 10 mmHg	777
b. Diastolic	(1st)	___	mmHg		777
	(2nd)	___	mmHg	± 10 mmHg	777
	(3rd)	___	mmHg	± 10 mmHg	777
c. Pulse	(1st)	___	bpm		777
	(2nd)	___	bpm	± 10 bpm	777
	(3rd)	___	bpm	± 10 bpm	777

2. Blood Sugar Control

[Refer if HbA1c >40 mmol/mol]

a. Glycated haemoglobin (HbA1c) level	___	mmol/mol	777

Appendix C: Bioelectrical impedance analysis instruction

BIOIMPEDANCE

What is Bioelectrical Impedance Analysis?

Bioelectrical impedance analysis (BIA) measures the impedance or opposition to the flow of an electric current through the body fluids contained mainly in the lean tissue. Impedance is low in lean tissue, where intracellular fluid and electrolytes are primarily contained, but high in fat tissue. Impedance is thus proportional to total body water **(TBW)** and therefore body fatness. More muscle, more water!

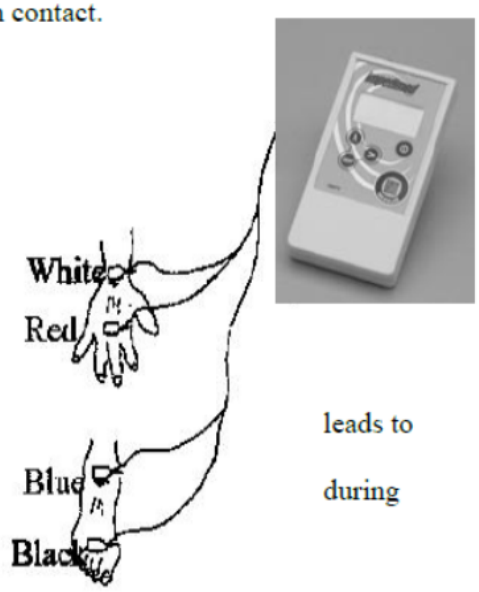
Pre-testing conditions

- The participant should not have eaten for 4-5 hours prior to testing.
- The participant should empty their bladder immediately before testing.

Measurement of bioimpedance

1. The participant should be lying face-up with legs slightly apart and hands resting next to the body palms down. Hands should not be touching any part of the body. The patient's inner thighs should not be in skin to skin contact.
2. The participant should remove the right shoe and sock (generally the measurements are completed on the right side of the body).

- **White or yellow**– placed on an imaginary line bisecting the ulna head (bone on little finger side of right wrist)
- **Red** – first joint of the middle finger, 3rd metatarsal head dorsum on the right hand.
- **Blue** – placed on an imaginary line bisecting the medial malleolus (bone on big toe side of ankle)
- **Black** – placed on the base of the second toe, 2nd metatarsal head dorsum on the right foot. Attach the electrodes.



leads to

during

3. The participant should lie quietly and still the entire test.

Operating Instructions

4. Press On key (top right hand side) to switch on machine.
5. Battery check icons will be displayed – if all rectangles are empty the batteries should be changed
6. Select the direct measurement using the ^ key and press Enter
7. The instruction to place electrodes on patient will appear
8. Press Measure (the largest circle) and record the two values displayed **Impedance Z and Phase P**
9. Press the ^ key and record values. **Resistance R and Reactance Xc Record values.**
9. To repeat measurements, press **Enter**.

Appendix D: Pacific Islands Families main study nutrition and health questionnaire

Book Number: _____

PIF ID | | | | | . | |

Pacific Islands Families Study

14-Year Phase

YOUTH ASSESSMENT PROTOCOL

Thank you for agreeing to complete the following questionnaire as part of the 14-year phase of the Pacific Islands Families (PIF) Study. This is the largest and longest running study of Pacific children in the world, so we are really grateful once again for your participation!

We will be asking you some questions about: your aspirations, identity, peer relations, social and digital media use, physical activity, nutrition, gambling and other issues related to adolescence.

We would just like to reassure you that everything will be kept completely private and you are free to stop at any time.

Most of the questions are straightforward, but if you do come to a question that you don't know the answer to or you REALLY feel uncomfortable about answering: **circle the number 7 (or 7's) in the grey box.**

Like this: (7) 8

If the question is not relevant to you: **circle the number 8 (or 8's) in the grey box.**

It is important to **circle the number, not the word.** For example:

In the **past four weeks** have you eaten red meat – such as beef, pork, mutton, lamb and goat – at all?

No (0) Yes (1) 7

On average, how many hours per day do you spend online?

Less than 1 hour	(1)
1 – 2 hours	(2)
2 – 4 hours	(3)
4 – 6 hours	(4)
6 – 8 hours	(5)
8 or more hours	(6)

7 8

There are a lot of questions that you can skip past depending on how you answer earlier questions - so please pay close attention to these - they will help you get through the survey faster.

Please note: do not enter anything in the boxes in the right hand side of the margin. This is for office use only.

SECTION G: NUTRITION AND HEALTH

[2008/09 NZANS]

Nutrition

I am going to ask you some questions about your usual eating patterns. When answering these questions please think back over the **past four weeks**. Remember to think about all meals (that is breakfast, lunch and dinner) as well as snacks and times when you eat both at home and away from home.

G1. How many days in an **average week** do you have something to eat for breakfast? You may have eaten at home, in a car, or in a café.

NOTE:
- Include both weekends and weekdays
- Include breakfast drinks such as smoothies and shakes
- Breakfast is usually the first meal of the day, eaten within 2 hours of getting up

# of days:	00	01	02	03	04	05	06	07	77	G1

G2. On average, how many slices of bread/toast OR bread rolls do you eat per day?

None, I don't eat bread or toast	(0)
Less than one per day	(1)
1 – 2 per day	(2)
3 – 4 per day	(3)
5 – 6 per day	(4)
7 or more per day	(5)
	7

G2

G3. What type of bread, rolls or toast do you eat most of?

White	(1)
High fibre white	(2)
Light grain bread (e.g. Molenberg, Freya's, Ploughmans, MacKenzie High Country)	(3)
Heavy grain bread (e.g. Vogels and Burgen)	(4)
Other	(5)
	7 8

G3

G4a. In the **past four weeks** have you eaten red meat – such as beef, pork, mutton, lamb and goat – at all?

No (0) Yes (1) 7 ☐ G4

G4b. If **yes to G4a**, how often do you eat red meat?

Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7 8

☐ G5

G5a. In the **past four weeks** have you eaten chicken – such as chicken breast, drumsticks, or whole chickens, but not chicken nuggets or chicken roll – at all?

No (0) Yes (1) 7 ☐ G6

G5b. If **yes to G5a**, how often do you eat chicken?

Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7 8

☐ G7

G6a. In the **past four weeks** have you eaten processed meats – such as ham, bacon, sausages, luncheon, canned corned beef, pastrami, and salami – at all?

No (0) Yes (1) 7 ☐ G8

G6b. If **yes to G6a**, how often do you eat processed meat?

Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7 8

☐ G9

G7a. In the **past four weeks** have you eaten seafood – such as fish or shellfish (including battered, deep fried, and canned) – at all?

No (0) Yes (1) 7 ☐ G10

G7b. If **yes to G7a**, how often do you eat fresh or frozen fish or shellfish? Do not include battered / fried or canned fish or shellfish.

Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7 8

☐ G11

G7c. If **yes to G7a**, how often do you eat battered or fried fish or shellfish? This may include fish bought from the 'Fish and Chip' shop.

Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7 8

☐ G12

G7d. If **yes to G7a**, how often do you eat canned fish or shellfish? Canned fish includes products such as tuna, salmon, and sardines.

Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7 8

☐ G13

G8. On average how many servings of fruit – fresh, frozen, canned or stewed – do you eat per day? Do not include fruit juice or dried fruit.

A serving is the same as a medium piece of fruit such as an apple or two small pieces of fruit such as two apricots, or half a cup of stewed fruit.

Never, I don't eat fruit	(0)
Less than one serving per day	(1)
1 serving	(2)
2 servings	(3)
3 servings	(4)
4 or more servings	(5)
	7

☐ G14

G9. On average how many servings of vegetables – fresh, frozen or canned – do you eat per day? Do not include vegetable juices.

A serving is the same as one potato/kumara, half a cup of peas or a cup of salad. For example, 2 medium potatoes + ½ cup of peas = 3 servings

Never, I don't eat vegetables	(0)
Less than one serving per day	(1)
1 serving	(2)
2 servings	(3)
3 servings	(4)
4 or more servings	(5)
	7

☐ G15

G10. On average how many servings of milk do you drink per day? Include milk used with food such as milk poured on cereal or in pudding.

A serving is the same as one cup (250ml) of milk.

Never, I don't drink/use milk	(0)
Less than one serving per day	(1)
1 serving	(2)
2 servings	(3)
3 servings	(4)
4 or more servings	(5)
	7

☐ G16

G11. What type of milk do you use the most of?

None, I don't use milk	(0)
Whole or standard milk (Dark blue or silver)	(1)
Reduced fat (Light blue)	(2)
Skim or Trim (Green or yellow)	(3)
Soy milk	(4)
Other (such as rice, goats milk)	(5)
	7 8

G17

G12. How often do you eat hot chips, French fries, wedges, or kumara chips? Think about lunch and dinner as well as snacks.

Never	(0)
Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7

G18

G13. How often do you eat fast food or takeaways from places like McDonalds, Burger King, Pizza shops, or fish and chip shops? Think about breakfast, lunch, dinner and snacks. Do not include times when you have only purchased a drink/beverage.

Never	(0)
Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7

G19

G14. How often do you drink fruit juices and fruit drinks? Do not include diet drinks, soft drinks/fizzy drinks, energy drinks, flavoured waters (e.g. H2Go) and sports waters (e.g. Charlies Sports water, Mizone and Aqua-shot).
Fruit juices and drinks include freshly squeezed varieties, and brands such as Just Juice, Fresh-up, Keri, Golden Circle, Ribena, Thextons, McCoy and Charlie's.

Never	(0)
Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7

☐ G20

G15. How often do you drink soft drinks or energy drinks? Soft drinks are often carbonated or 'fizzy' and includes Coca-Cola, Pepsi, lemonade, ginger beer, energy drinks ('V', Red Bull, Lift plus), Powerade, E2, G-force. Do not include diet varieties, flavoured waters (e.g. H2Go), or sports waters (e.g. Charlies Sports water, Mizone and Aqua-shot).

Never	(0)
Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7

☐ G21

G16. How often do you eat lollies, sweets, chocolate, and confectionary?

Never	(0)
Less than once per week	(1)
1 – 2 times per week	(2)
3 – 4 times per week	(3)
5 – 6 times per week	(4)
7 or more times per week	(5)
	7

☐ G22

Health

[Youth Connectedness Survey]

The next section asks questions regarding your health.

G17. How healthy are you?

Very unhealthy	Unhealthy	Average	Healthy	Very healthy	
1	2	3	4	5	7

☐ G23

G18. Thinking about your weight, would you say you are:

Underweight	A little Underweight	About right	A little Overweight	Overweight	
1	2	3	4	5	7

☐ G24

G19. How much of the time during the LAST MONTH did you feel full of energy?

None of the time	A little of the time	Some of the time	Most of the time	All the time	
1	2	3	4	5	7

☐ G25

G20. In the LAST WEEK, on how many nights did you get at least 8 hours of sleep?

# of nights:	00	01	02	03	04	05	06	07	77

☐☐ G26

Appendix E: Pacific Islands Families sub-study ethics approval

Health and Disability Ethics Committees

Health and Disability Ethics Committees
1 The Terrace
C/- MEDSAFE, Level 6, Deloitte House
10 Brandon Street
PO Box 5013
Wellington
6011

+6448196860
hdecs@moh.govt.nz

28 July 2014

Professor Elaine Rush
MB - Manukau Campus
Auckland University of Technology
Private Bag 92006
Auckland 1142

Dear Professor Rush

Re:	**Ethics ref:**	**14/CEN/108**
	Study title:	Pacific Islands Families: Understanding growth from birth to fourteen years

I am pleased to advise that this application has been *approved* by the Central Health and Disability Ethics Committee. This decision was made through the HDEC-Full Review pathway.

The main issues considered by the HDEC in giving approval were as follows.

- The Committee noted that the tissue will be stored to enable further analysis using new panel assays. These samples will be retained for 2-3 years and will only be used in relation to the current research project on these children.
- The Committee commended the research team for specifically recognising that some children will have mixed Maori/ PI parentage.

Conditions of HDEC approval

HDEC approval for this study is subject to the following conditions being met prior to the commencement of the study in New Zealand. It is your responsibility, and that of the study's sponsor, to ensure that these conditions are met. No further review by the Central Health and Disability Ethics Committee is required.

Standard conditions:

1. Before the study commences at *any* locality in New Zealand, all relevant regulatory approvals must be obtained.

2. Before the study commences at *a given* locality in New Zealand, it must be authorised by that locality in Online Forms. Locality authorisation confirms that the locality is suitable for the safe and effective conduct of the study, and that local research governance issues have been addressed.

Non-standard conditions:

3. Please clearly label the PIS/Assent form for children and PIS/CF for caregivers.
4. Please remove reference on page 1 of the PIS to being "specially chosen".
5. Please add the header "Optional" to the PIS for the substudy.
6. Please include a confidentiality statement in the child assent form.
7. Please include reference to the Central HDEC in approval statement.

Non-Standard conditions must be completed before commencing your study. Non-standard conditions do not need to be submitted to HDEC before commencing your study.

If you would like to submit your Non-standard conditions please email Non-standard conditions to HDECS@moh.govt.nz. Do not submit Non-standard conditions as a Post Approval form.

After HDEC review

Please refer to the *Standard Operating Procedures for Health and Disability Ethics Committees* (available on www.ethics.health.govt.nz) for HDEC requirements relating to amendments and other post-approval processes.

Your next progress report is due by 22 July 2015.

Participant access to ACC

The Central Health and Disability Ethics Committee is satisfied that your study is not a clinical trial that is to be conducted principally for the benefit of the manufacturer or distributor of the medicine or item being trialled. Participants injured as a result of treatment received as part of your study may therefore be eligible for publicly-funded compensation through the Accident Compensation Corporation (ACC).

Please don't hesitate to contact the HDEC secretariat for further information. We wish you all the best for your study.

Yours sincerely,

Mrs Helen Walker
Chairperson
Central Health and Disability Ethics Committee

Encl: appendix A: documents submitted
appendix B: statement of compliance and list of members

Documents submitted

Document	*Version*	*Date*
CV for CI: CV for Elaine Rush.	1	25 June 2014
Evidence of scientific review: HRC assessing committee reviews and applicant response.	1	25 June 2014
Protocol: Study protocol	1	08 July 2014
Parent/caregiver consent form	1	08 July 2014
Information sheet	1	08 July 2014
Child information sheet & assent form	1	08 July 2014
Survey/questionnaire: Food frequency & sleeping time questionnaire	1	08 July 2014
Application	1	11 July 2014

Statement of compliance and list of members

Statement of compliance

The Central Health and Disability Ethics Committee:

- is constituted in accordance with its Terms of Reference
- operates in accordance with the *Standard Operating Procedures for Health and Disability Ethics Committees*, and with the principles of international good clinical practice (GCP)
- is approved by the Health Research Council of New Zealand's Ethics Committee for the purposes of section 25(1)(c) of the Health Research Council Act 1990
- is registered (number 00008712) with the US Department of Health and Human Services' Office for Human Research Protection (OHRP).

List of members

Name	*Category*	*Appointed*	*Term Expires*	*Present on 22/07/2014?*	*Declaration of interest?*
Mrs Helen Walker	Lay (consumer/community perspectives)	01/07/2012	01/07/2015	Yes	No
Mr Paul Barnett	Lay (the law)	01/07/2012	01/07/2015	Yes	No
Dr Kay de Vries	Non-lay (observational studies)	19/05/2014	19/05/2017	Yes	No
Mrs Gael Donoghue	Non-lay (health/disability service provision)	01/07/2012	01/07/2015	Yes	No
Mrs Sandy Gill	Lay (consumer/community perspectives)	01/07/2012	01/07/2015	Yes	No
Dr Patries Herst	Non-lay (intervention studies)	01/07/2012	01/07/2015	No	No
Dr Dean Quinn	Non-lay (intervention studies)	01/07/2012	01/07/2015	Yes	No
Dr Cordelia Thomas	Lay (ethical/moral reasoning)	19/05/2014	19/05/2017	No	No

http://www.ethics.health.govt.nz

Appendix F: Pacific Islands Families sub-study food frequency questionnaire

Office use only

Date completed:

Name:

Date of birth:

Food Questionnaire

Different eating patterns may affect people's health. To help us understand these eating patterns, we would like you to **think back over the past 4 weeks** and answer the following questions about the foods you usually eat.

Put a tick in the box which best tells **HOW OFTEN** you usually eat the foods.

Example

If you eat apples on 3 or 4 days each week, put a tick in the '3-4 times a week' box.

2. Apples or pears

If you never or rarely eat a food, tick in the box 'never or less than once a month' and go to the next question.

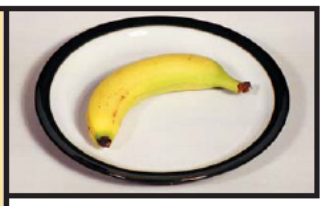
It may be helpful to ask the person who does the cooking and shopping in your household to help you fill in the questions.

PLEASE DO NOT SKIP ANY FOODS

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

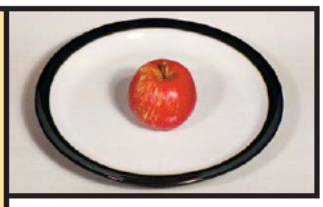
Fruit

1. Banana, raw



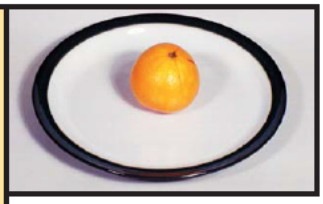
Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

2. Apples or pears



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

3. Oranges or mandarins

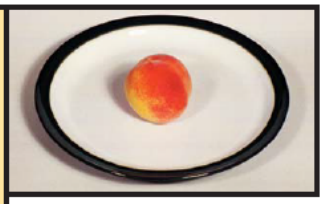


Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

4. Kiwifruit

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

5. Nectarines, peaches, plums or apricots



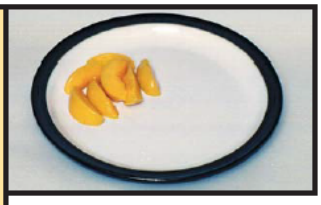
Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

6. Strawberries or other berries

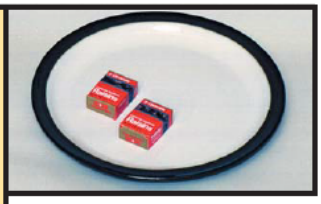
Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

7. Canned or cooked fruit, eg. canned peaches



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

8. Dried fruit, eg. raisins



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

9. Other Fruit (1) If you often have another fruit, not listed - give the name and tick a box to show how often you eat it _____

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

10. Other Fruit (2) If you often have another fruit, not listed - give the name and tick a box to show how often you eat it _____

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

63. Breakfast cereal

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

63a. What type of cereal do you usually have? *(tick up to 3 boxes)*

- ☐ Weetbix type
- ☐ Cornflakes type
- ☐ Rice bubbles
- ☐ Cocopops
- ☐ Muesli
- ☐ Multi-grain type
- ☐ Porridge
- ☐ Other (Please give name) _____

63b. What kind of milk was usually added to your cereal?

- ☐ None
- ☐ Standard milk/dark blue
- ☐ Other (Please give name) _____
- ☐ Light blue
- ☐ Trim (green)
- ☐ Extra calcium
- ☐ Soy milk

63c. Was sugar, honey or syrup added to your cereal?

- ☐ Yes
- ☐ No

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

Spreads, sauces

66. Jam or honey

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

67. Nutella

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

68. Marmite or Vegemite

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

69. Peanut butter

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

70. Mayonnaise or salad dressing

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

71. Tomato sauce or ketchup

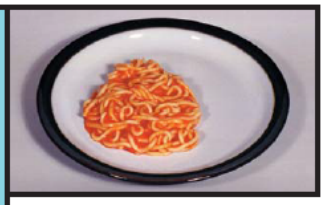
Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

73. Other item of the 'Spreads, sauces' group If you often have another item from this group, not listed - give the name and tick a box to show how often you eat it _____

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

77. Canned spaghetti with tomato sauce

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐



Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

78. Baked beans

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

79. Other item of the 'Convenience meals/snacks' group If you often have another item from this group, not listed - give the name and tick a box to show how often you eat it _____

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

Dairy

80. Ice cream

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

82. Yoghurt or Dairy food (all types)

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

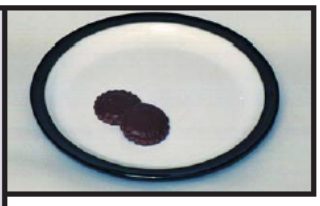
Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

84. **Other item of the 'Dairy' group (not milk drinks)** If you often have another item from this group, not listed - give the name and tick a box to show how often you eat it _____

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

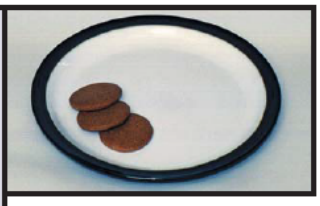
Biscuits/cakes

85. **Chocolate coated or cream filled biscuits**



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

86. **Biscuits,** eg. plain, chocolate chip, semi-sweet, ginger nut, shortbread



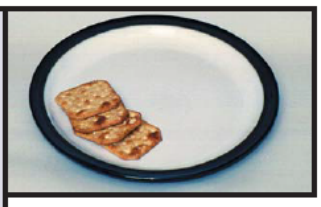
Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

87. **Bars,** eg. muesli

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

88. Crackers or crispbreads



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

89. Cake or slice

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

90. Doughnuts or croissants

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

91. Scones, muffins or sweet buns

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

92. Pancake or pikelets

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

93. **Fruit pie, fruit crumble or tart**

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

94. **Pudding,** eg. sponge pudding or steamed pudding

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

95. **Custard or custard puddings**

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

96. **Other item of the 'Biscuits/cake' group** If you often have another item from this group, not listed - give the name and tick a box to show how often you eat it ☐

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

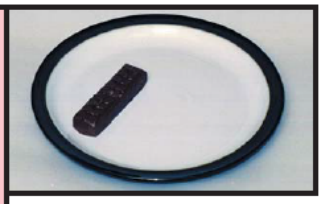
Snacks and sweets

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

98. Popcorn

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

99. Chocolate, eg. Moro bar



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

100. Candy coated chocolate, eg. pebbles

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

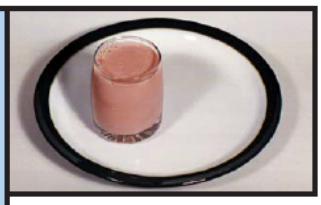
101. Other sweets

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

Milks

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

103. Flavoured milk



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

104. Milk shake

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

105. Food drink, eg. Milo powder, Nesquik

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

105a. With this drink did you use?

☐ All milk

☐ 1/2 milk

☐ 1/4 or less milk

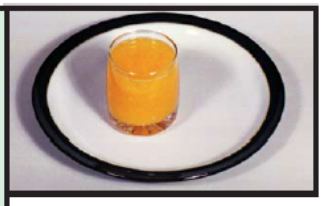
Was **sugar** added?

☐ Yes ☐ No

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

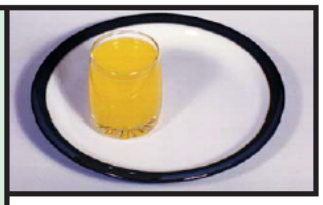
Other drinks

106. Juice, eg. fresh orange juice, juices such as McCoys, Robinson's, Keri



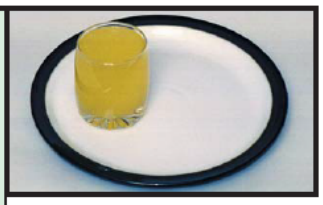
Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

107. Powdered fruit drink, eg. Refresh, Raro



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

108. Fruit drink from concentrate or cordial, eg. Just Juice, Ribena



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

109. Coca cola or other cola drinks

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

110. Mountain Dew

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

111. **'New Age' drinks,** eg. V, E_2, Red Bull

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

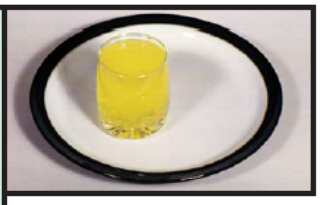
111a. If you have **'New Age' drinks**, which **type** do you usually have? (tick one box)

- ☐ V
- ☐ E2
- ☐ Lift
- ☐ Red Bull
- ☐ Liquid B
- ☐ Ikon
- ☐ Bullrush
- ☐ Other (please name) _____

112. **Soft drinks,** eg. lemonade, orange

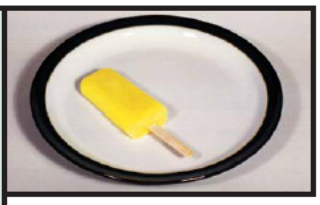
Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

113. **Sports drinks,** eg. Gatorade, Powerade



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

114. **Ice blocks**



Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

Put a tick ☑ in the box which best tells HOW OFTEN you eat the food.

115. Tea

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

115a. Was milk added to your tea?

☐ Yes ☐ No

Was **sugar** added?

☐ Yes ☐ No

116. Coffee

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

116a. Was milk added to your coffee?

☐ Yes ☐ No

Was **sugar** added?

☐ Yes ☐ No

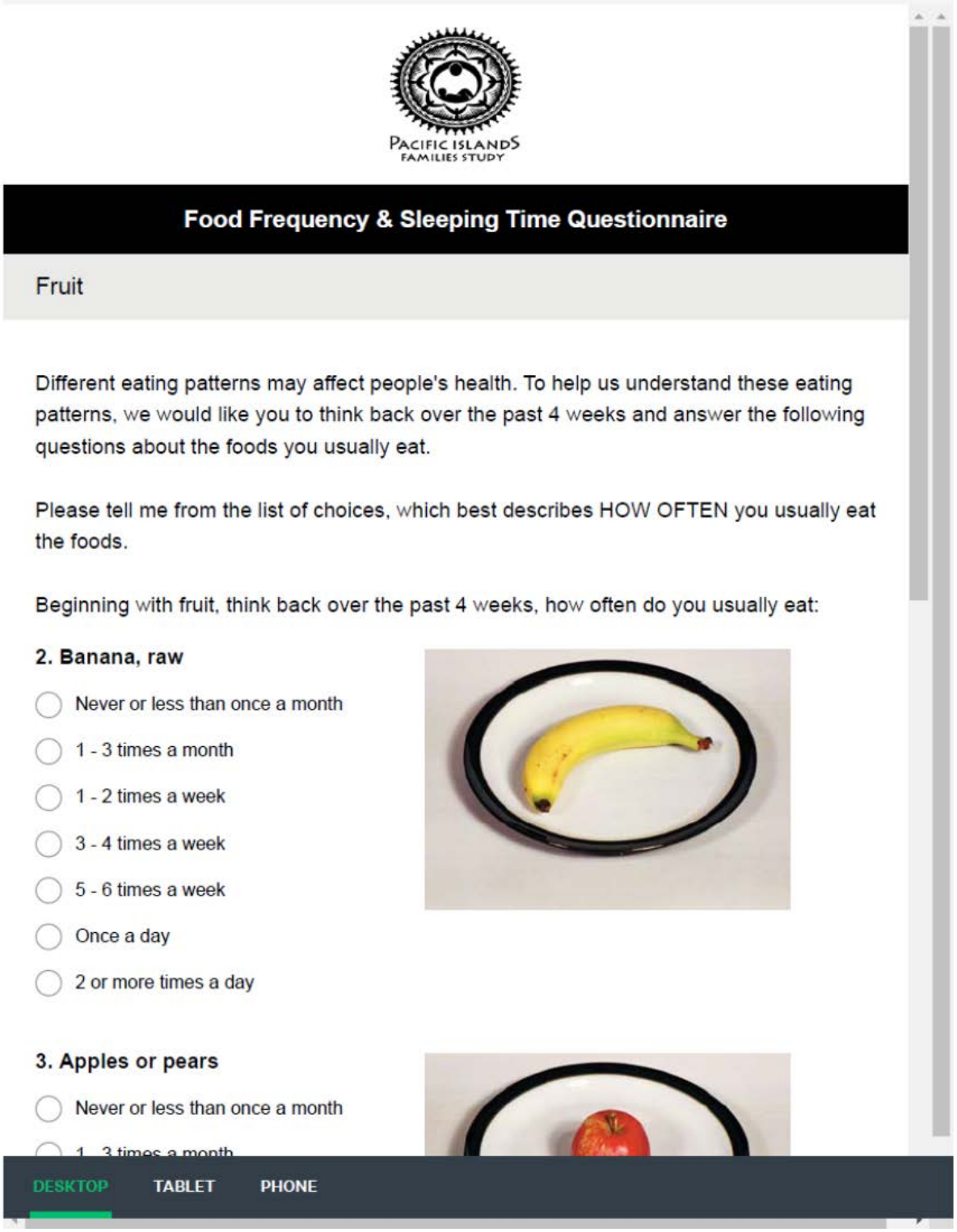
117. Other item of the 'Other drinks' group If you often have another item from this group, not listed - give the name and tick a box to show how often you eat it _____

Never or less than once a month	1-3 times a month	1-2 times a week	3-4 times a week	5-6 times a week	Once a day	2 or more times a day
☐	☐	☐	☐	☐	☐	☐

Thank you very much for filling out this questionnaire.

Please take a moment to fill in any questions you have skipped.

Appendix G: Pacific Islands Families sub-study questionnaire tablet screen shot



PACIFIC ISLANDS FAMILIES STUDY

Food Frequency & Sleeping Time Questionnaire

Fruit

Different eating patterns may affect people's health. To help us understand these eating patterns, we would like you to think back over the past 4 weeks and answer the following questions about the foods you usually eat.

Please tell me from the list of choices, which best describes HOW OFTEN you usually eat the foods.

Beginning with fruit, think back over the past 4 weeks, how often do you usually eat:

2. Banana, raw

- Never or less than once a month
- 1 - 3 times a month
- 1 - 2 times a week
- 3 - 4 times a week
- 5 - 6 times a week
- Once a day
- 2 or more times a day

3. Apples or pears

- Never or less than once a month
- 1 - 3 times a month

DESKTOP TABLET PHONE

Appendix H: Metformin in Gestational Diabetes the Follow-up study ethics approval

Health and Disability Ethics Committees

Northern X Regional Ethics Committee
Private Bag 92522
Wellesley Street
Auckland 1141
Phone: (09) 580 9105
Fax (09) 580 9001
Email: northernx_ethicscommittee@moh.govt.nz

27 June 2012

Dr Janet Rowan
National Women's Health
Lvl 9, Auckland City Hospital
Private Bag 92 024
Auckland 1142

Dear Janet

Re:	Ethics ref:	**AKX/04/08/228** (please quote in all correspondence)
	Study title:	Gestational diabetes: treatment with metformin compared with insulin - the offspring follow-up study. PIS/Cons V#4, 08/08. Substudy: Metformin in Gestational Diabetes: The Offspring follow up at 7-9 years. PIS/Cons V#1, 14/06/12
	Investigators:	Dr Janet Rowan, Associate Professor Elaine Rush, Associate Professor Malcolm Battin, Professor Tim Cundy, Dr Jun Lu, A/Prof Lindsay Plan

We are in receipt of your e-mail requesting ethical approval for an addition to the above study.

The documentation has been reviewed by the Chairperson of the Northern X Regional Ethics Committee under delegated authority.

Ethical approval is granted to:

- Sub study application including Parts 5 and 8
- Protocol number [version received 21/06/12]
- Information sheet/Consent Form for parents version [1 dated 14 June 2012]
- Information sheet/Consent Form for child version [1 dated 14 June 2012]
- Background Questionnaire version [1 dated 16 Jun 2012]
- Food and Activity Questionnaire version [1 dated 14 Jun 2012]

It should be noted that Ethics Committee ethical approval does not imply any resource commitment or administrative facilitation by any healthcare provider, within whose facility the research is to be carried out. Where applicable, authority for this must be obtained separately from the appropriate manager within the organisation.

Yours sincerely

Cheh Chua
Administrator
Northern X Regional Ethics Committee

cc: ADHB Research Office

Appendix I: Metformin in Gestational Diabetes the Follow-up study questionnaire

Site number
Study number
Study Initials

MiG:TOFU Food and Activity Questionnaire – at 7-9 years

Date of assessment: _____

Instructions for food and activity questionnaire
This is for the child to answer with help from parents/guardians as required.
A trained researcher will also go over these questions with you.

Circle the best answer for each question

This questionnaire is in two main sections.

- The first part is about the food you eat
- The second part is about how active you are

FOOD

Do you take any vitamin or mineral supplements? YES/NO

What foods do you NOT eat? E.g. meat, red meat, chicken, pork, fish, milk, peanuts

How many days in an average week do you usually have something to eat for breakfast?
(This includes breakfast eaten at home, in a car, at school/work or in a café.) 0,1,2,3,4,5,6,7

Over the past 5 school days, how often did you bring your lunch to school from home? 0,1,2,3,4,5

Instructions for the following table:

- For each item in the list, if it is eaten/drunk never or less than once a month, circle, N/A (not applicable) and go to the next item.
- If it is eaten/drunk 1-3 times a month, select "monthly", if eaten/drunk 1-6 times/week, select "weekly" and if eaten/drunk at least once a day, select "daily".
- Then, in the next column circle how many times each month/week or day.
  Examples:
  - If you have a drink of water twice a day, circle "daily" then 2.
  - If you eat a takeaway twice a week, circle "weekly" then 2.
  - If you never have tea/coffee, circle N/A and go to next item

How often do you eat or drink the following?

	Never or less than monthly (N/A)	Monthly/ weekly/daily (circle correct one)	How many times? (circle number)
Water	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Juice	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7

Fizzy/Energy drinks (egCoke,Fanta, 'V"etc)	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Cordial/ powdered fruit drink	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Tea/coffee	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Food drink e.g. Milo	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Milk	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Type of milk (circle best answer)	Skim or trim (green or yellow), whole or standard (dark blue or silver), reduced fat (light blue) Other _____		
Cheese	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Yoghurt	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Bread-white (one slice)	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Bread- other (one slice)	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
			1 2 3 4 5 6 7
Cereal-sweetened	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Cereal – plain (eg porridge/weetbix/ plain muesli/no sugar)	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
			1 2 3 4 5 6 7
Potatoes: mashed, boiled, jacket	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Potatoes: roast, chips	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Chippies/crisps	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
			1 2 3 4 5 6 7
Kumara	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Taro	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Dumplings	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
			1 2 3 4 5 6 7
Pasta	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Noodles	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Rice	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Chappati/Roti	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
			1 2 3 4 5 6 7
Crackers	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Sweet biscuits	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Lollies/sweeties	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Chocolate	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Takeaways	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
			1 2 3 4 5 6 7
Fresh fruit	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Tinned/frozen other fruit	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
			1 2 3 4 5 6 7
Fresh vegetables/salad	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Tinned/frozen vegetables	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
			1 2 3 4 5 6 7
Chicken/pork	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Red meat (lamb or beef)	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Sausage/bacon	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Fish	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7

Eggs	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7
Lentils/ kidney beans/soy beans	N/A	Monthly/Weekly/Daily	1 2 3 4 5 6 7

For this question, you will need to ask the person who usually does the food shopping in your family. A number of families feel they are unable to eat properly because some foods are very expensive. Does your family think they can afford to eat properly?

a) Always
b) Sometimes
c) Never
d) Don't Know

ACTIVITY

For the following questions, write correct answer or circle appropriate answer.

	Weekday	Weekend
What time do you usually get out of bed in the morning?	_____	_____
What time do you usually go to bed at night?	_____	_____

How do you normally go to and from school?

1. Walk
2. Bike
3. Skate or other physical activity
4. Car
5. School bus
6. Public transport
7. Other _____
8. N/A as home school
9. Not answered

How many hours do you normally watch TV/DVDs from Monday to Friday night? ____ hrs
(This could be anywhere, not just in your home, and includes DVDs/videos but does not include games. Add up each night and put the total number of hours eg if one hour on four evenings, put 4 hours as answer).

How many hours do you watch TV/DVDs from Saturday morning to Sunday night? ____hrs
(Again, this could be anywhere, not just in your home and includes DVDs/videos but does not include games.)

Do you do any of the following sports/activities for more than 20 minutes at a time? If yes, place a tick in the column which best shows how often you do the activity

	Never	<1/month	1-3/month	1-2/wk	3-6/wk	Once/day	2+/day
Bike/scooter							
Skateboard							
Swim							

Rollerblade/ice-skate							
Gymnastics: bars, beam, tumbling etc.							
Basketball / Miniball							
Baseball/softball/dodgeball							
Netball							
Rugby/League/Touch							
Volleyball							
Soccer							
Hockey							
Cricket							
Racket sport e.g. Tennis							
Judo, Karate, Boxing etc.							
Dance: eg ballet/tap/hip hop							
Kapa Haka							
Horse Riding							
Athletics							
Other							

When you are playing at home, how often would you do activities that make you short of breath (huff and puff)?

a. Less than once a month
b. 1-3 times a month
c. 1-2/week
d. 3-6/week
e. Once a day
f. More than once a day

Last week, how many times did you go to a playground, park, swimming pool, studio or other place for physical activity? (E.g. rugby, dance, kapahaka, martial arts, adventure playground, walking, active family games)

1,2,3,4,5,6,7, more than 7

Are you more active or less active at the weekends compared to weekdays?

a) More active
b) Less active
c) No difference

How active do you think you are compared with other children the same age as you?

a) More active
b) Just as active
c) Less active
d) Much less active
e) Not sure/don't know

Appendix J: Children food recommendations suggested by New Zealand Ministry of Health

1.3 Summary of food groups, serving sizes and recommended intakes

Table 1 shows the four food groups, specific foods included in each group, the minimum number of servings of each group recommended for healthy children and young people, and examples of standard serving sizes.

Table 1: Food groups, specific foods in each group, advice and serving size examples

Food group	Specific foods included	Recommendation (per day)	Serving size examples
Vegetables and fruit	All vegetables and fruit, including potatoes, kūmara and taro Vegetables and fruit – fresh, frozen or canned If consumed, only one serving of no-sugar-added fruit juice or dried fruit can count as contributing a serving to the recommended dietary intake[1]	**Preschoolers:** at least 2 servings of vegetables and at least 2 servings of fruit **Children:** at least 3 servings of vegetables and at least 2 servings of fruit **Young people:** at least 3 servings of vegetables and at least 2 servings of fruit	1 medium potato or kūmara (135 g) ½ cup cooked vegetables (eg, broccoli, peas, corn, spinach, pūhā) (50–80 g) 1 carrot (75 g) ½ cup salad (60 g) 1 tomato (80 g) ½ avocado (80 g) 1 apple, pear, banana or orange (130 g) 2 small apricots or plums (100 g) ½ cup fresh fruit salad (120 g) ½ cup stewed or tinned fruit (135 g) 1 cup no-added-sugar fruit juice (250 ml)^
Breads and cereals	All breads, cereals, rice and pasta (increasing wholegrain options as children age)	**Preschoolers:** at least 4 servings **Children:** at least 5 servings **Young people:** at least 6 servings	1 medium slice of bread (26 g) 1 roll (50 g) 1 pita pocket or tortilla (50–80 g) 2 breakfast wheat biscuits (34 g) ½ cup muesli (55 g) ½ cup porridge (130 g) 1 cup cornflakes (30 g) 1 cup cooked pasta or rice (150 g) 4 grainy crackers (40 g) 2 plain sweet biscuits (14 g) 1 cup plain popcorn
Milk and milk products	Milk (includes calcium-fortified milk alternatives), cheese and yoghurt (choose low-fat options)	**Preschoolers and children:** at least 2–3 servings **Young people:** at least 3 servings	Glass of milk or calcium-fortified milk alternative (250 ml) Pottle of yoghurt (150 g) 2 slices of cheese (40 g)
Lean meat, poultry, seafood, eggs, legumes, nuts and seeds*	Lean meat, poultry, seafood, eggs, legumes (eg, peas, beans, lentils), nuts and seeds* (Limit processed meats)	**Preschoolers and children:** at least 1–2 servings **Young people:** at least 2 servings **Vegetarians:** **Preschoolers (2–5 years):** at least 1–2 servings **School children (5–12 years):** at least 2 servings **Young people (13–18 years):** At least 3 servings	2 slices of cooked meat (100 g) ¾ cup of mince or casserole (195 g) 1 medium fish fillet (100 g) 1 chicken leg or 2 drumsticks (110 g) 1 medium pāua or kina (100–120 g) 1 egg (50 g) ½ can tuna or salmon (90 g) ¾ cup dried cooked beans, peas or lentils (135 g) 1/3 cup nuts or seeds* (50 g)

Notes:

^ The Ministry of Health recommends choosing vegetables and fruit that are fresh, frozen or tinned. If vegetable/fruit juice or dried fruit is consumed, it contributes a maximum of only one serving of the total recommended number of servings for this food group. Servings of fresh, frozen and canned vegetables and fruit are still required to meet the recommendations.

* Do not give small, hard foods such as whole nuts and large seeds until children are at least 5 years old to reduce the risk of choking.