

Copyright is owned by the Author of the thesis. Permission is given for a copy to be downloaded by an individual for the purpose of research and private study only. The thesis may not be reproduced elsewhere without the permission of the Author.

PreAdolescent CardioMetabolic Associations and Correlates: PACMAC

A thesis presented in partial fulfilment of the requirements for the degree of

Doctor of Philosophy

in

School of Sport and Exercise

at Massey University, Wellington,

New Zealand

Nicholas Castro

2018

Abstract

Cardiovascular disease is typically associated with adults; however, atherosclerosis often initiates during preadolescence and has been linked to cardiometabolic risk factors. Preceding cardiometabolic risk factors include lifestyle factors: body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep. No known study has comprehensively assessed simultaneous associations among lifestyle factors with cardiometabolic risk factors in preadolescent children.

A multicentred cross-sectional study design was utilised to investigate lifestyle factor associations with cardiometabolic risk factors in a sample of 392 children aged 8 to 10 years. Participants were recruited from primary schools located in the Wellington, Canterbury, and Otago regions in New Zealand. Data collection was carried out over 5 days between 09:00 a.m. and 12:00 p.m. at each location.

The first objective assessed the associations among physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with body fatness indicators (body fat percentage, fat mass index, body mass index, and waist-to-hip ratio). Results indicated nutrition independently associated with body fat percentage ($p < 0.05$), whereas cardiorespiratory fitness significantly associated with all four body fatness indicators ($p < 0.05$).

The second objective assessed the associations among body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with cardiometabolic factors (blood pressure, cholesterol, vascular, and carbohydrate-metabolic). Results indicated body fat percentage associated with the blood pressure factor ($p < 0.05$); sedentary minutes, social jetlag, and Fruit and Vegetables pattern associated with the cholesterol factor (all $p < 0.05$); sedentary minutes and Processed

Food pattern associated with the vascular factor (both $p < 0.05$); and cardiovascular fitness ($\dot{V}O_2\text{max}$) and handgrip strength associated with the carbohydrate-metabolic factor (both $p < 0.001$). Accordingly, body fatness, physical fitness, nutrition, and sleep all associated with at least one cardiometabolic factor.

Cardiorespiratory fitness associated with cardiometabolic health and was the key finding in Objective 1; therefore, physical fitness may be the most important lifestyle factor. However, as nutrition, sleep, sedentary behaviour, and body fatness also associated with cardiometabolic health, it appears one specific lifestyle factor does not entirely explain cardiometabolic health in preadolescent children, and thus a multimodal approach for health is required for this population.

Preface

The foundation for this research originated from my passion for working with children in afterschool programs and wanting to make a difference in their health and wellbeing. The PreAdolescent CardioMetabolic Associations and Correlates (PACMAC) study design came about due to the decline of children's overall health in New Zealand and worldwide, indicating the need to examine the importance of lifestyle factors with cardiometabolic health. I believe improving the overall health and wellbeing of children will require a multifaceted approach; the PACMAC study represents the preliminary phase.

Conceptualization

Based on my experience working with children in various settings (academic, athletic, and social), along with researching outcomes from previous paediatric studies, it became clear which influential lifestyle factors should be included. In discussion with my supervisory team, we formatted a list of research questions and together determined a study design detailing the most productive, safe, reliable, and noninvasive way to collect data in a school setting. Based on the preferred study design, preadolescent children aged 8 to 10 years seemed to represent the most likely target age for reliable data, yet would be young enough (prepubertal) to minimise sex differences.

Ethics

One of my first PhD responsibilities was to attain ethical approval. I submitted my ethics application to the Massey University Human Ethics Committee. During the risk assessment process, the PACMAC study was categorised as high risk and invasive given that the methods included a standard finger prick procedure to collect biochemical markers and that the study population was preadolescent children. Therefore, I was required to seek approval from the New Zealand Health and Disability Ethics

Committee (HDEC). I received approval from the HDEC on June 26, 2014 (HDEC:14/CEN/83).

Data Collection

For the data collection process, I recruited each principal, teacher, participant, and parent or guardian that participated in this study. Additionally, I designed and formatted standard operating procedure guidelines for each study measurement, set up the data collection stations, and recruited and trained research assistants at each location. Furthermore, during the week of data collection in the schools I led activities that focused on the importance of being active, eating healthy, and getting the proper amount of sleep. School staff were very appreciative of all the health information I provided to the participants and their families in return for participating in the study.

Data Analysis and Management

Regarding the analysis and interpretation, each day I calculated and verified each participant's measurement means (height, weight, waist circumference, hip circumference, blood pressure, augmentation index pressure, and pulse rate), and then transferred the calculated data from the data collection forms to an Excel spreadsheet. Next, I formatted and entered all the data into the SPSS program. For statistical analysis and interpretation, I was assisted by my supervisors, a mentor (Dr. Paula Skidmore), and a certified statistician.

Writing

The writing of this thesis is my work. I received extensive feedback from my supervisors while writing the literature review through multiple editing processes. The statistical analysis, results, and discussion sections I wrote after robust discussion of the results with my supervisors.

Publications

The PACMAC study protocol has been already published and the following papers based on my research have been published or are in progress. My contribution to each paper is itemised in Table 1.

Table 1

Contributions to Publications

Publications	Conceptual-ization	Study Design	Recruit-ment	Data Collection	Data Analysis	Writing
Paper 1	X	N/A	N/A	N/A	N/A	X
Paper 2	X	X	X	X	X	X
Paper 3		X	X	X		
Paper 4		X	X	X		
Paper 5		X	X	X		

1. **Castro, N.**, Lambrick, D. M., Faulkner, J., Lark, S., Williams, M. A., & Stoner, L. (2013). Decreasing the cardiovascular disease burden in Māori children: The interface of pathophysiology and cultural awareness. *Journal of Atherosclerosis and Thrombosis*, *20*, 833-834. doi:10.5551/jat.20198
2. **Castro, N.**, Faulkner, J., Skidmore, P., Williams, M., Lambrick, D. M., Signal, L., . . . Stoner, L. (2014). Pre-adolescent cardio-metabolic associations and correlates: PACMAC methodology and study protocol. *BMJ Open*, *4*(e005815), 1-9. doi:10.1136/bmjopen-2014-005815
3. Stoner, L., **Castro, N.**, Signal, L., Skidmore, P., Faulkner, J., Lark, S., ... Harrex, H. (2018). Sleep and adiposity in preadolescent children: The importance of social jetlag. *Childhood Obesity*, *14*, 158-164. doi:10.1089/chi.2017.0272

4. Stoner, L., Weatherall, M., Skidmore, P., **Castro, N.**, Lark, S., Faulkner, J., & Williams, M. A. (2017). Cardiometabolic risk variables in preadolescent children: A factor analysis. *Journal of the American Heart Association*, *6*(e007071).
doi:10.1161/JAHA.117.007071
5. Stoner, L., **Castro, N.**, Skidmore, P., Lark, S., Faulkner, J., & Williams, M. A. (2018). Fitness and fatness are both associated with cardiometabolic health in 8-10-year-old New Zealand children. Manuscript in preparation.

Acknowledgements

This thesis would not have been possible without the support of many individuals and organizations, which cannot be done justice with a few words, however, I will endeavour to do so. My entire PhD was funded by Education New Zealand and Massey University, and this study would not have been possible without their support.

Thank you to the staff and participants at the primary schools that participated in my study: Scots College, St. Patrick's, Crofton Downs, St. Bernadette's, Green Island, Holy Cross, Springston, Rāwhiti, and Yaldhurst. I really enjoyed data collection.

Dr. Lee Stoner, from the first day I arrived in New Zealand you were there supporting and mentoring me, which has been essential to my growth and development during my PhD journey. So much appreciated, Lee.

Dr. Sally Lark and Dr. James Faulkner, thanks for all the time you devoted to the PACMAC study. Sally, for keeping the team down-to-earth when things got hectic. James, for the time you took to counsel me and for the constructive criticism.

Dr. Michael Hamlin, thanks for the $\dot{V}O_{2max}$ knowledge and for the support you and your family provided when I was collecting data in the Canterbury region.

Dr. Paula Skidmore, thanks for mentoring me, for the support you provided when I was collecting data in the Otago region, and for all the principal component analysis work you did for the PACMAC study.

To all my family and loved ones, I say thank you with all my heart for always believing in me and supporting this small-town boy from Blythe, California.

To my partner Inge, thanks for always being encouraging and my biggest supporter. I could not have completed this PhD without your unconditional love.

Ethical approval for this study was received from the regional HDEC (14/CEN/83).

List of Publications

Castro, N., Faulkner, J., Skidmore, P., Williams, M., Lambrick, D. M., Signal, L., . . .

Stoner, L. (2014). Pre-adolescent cardio-metabolic associations and correlates:

PACMAC methodology and study protocol. *BMJ Open*, *4*(e005815), 1-9.

doi:10.1136/bmjopen-2014-005815

Castro, N., Lambrick, D. M., Faulkner, J., Lark, S., Williams, M. A., & Stoner, L.

(2013). Decreasing the cardiovascular disease burden in Māori children: The interface of pathophysiology and cultural awareness. *Journal of Atherosclerosis and Thrombosis*, *20*, 833-834. doi:10.5551/jat.20198

Stoner, L., **Castro, N.**, Signal, L., Skidmore, P., Faulkner, J., Lark, S., Williams, M. A.,

Muller, D., & Harrex, H. (2018). Sleep and adiposity in preadolescent children:

The importance of social jetlag. *Childhood Obesity*, *14*, 158-164.

doi:10.1089/chi.2017.0272

Stoner, L., Weatherall, M., Skidmore, P., **Castro, N.**, Lark, S., Faulkner, J., & Williams,

M. A. (2017). Cardiometabolic risk variables in preadolescent children: A factor analysis. *Journal of the American Heart Association*, *6*(e007071), 1-10.

doi:10.1161/JAHA.117007071

Manuscript in Preparation

Stoner, L., **Castro, N.**, Skidmore, P., Lark, S., Faulkner, J., & Williams, M. A. (2018).

Fitness and fatness are both associated with cardiometabolic health in 8-10-year-old New Zealand children.

Table of Contents

Abstract	ii
Preface.....	iv
Acknowledgements	viii
List of Publications	ix
List of Tables	xv
List of Figures	xvi
List of Abbreviations	xvii
Definition of Terms.....	xix
CHAPTER 1—INTRODUCTION	1
1.0 Circumstances and Statement of the Problem	1
1.1 Objectives of Research.....	2
1.2 Outline of Thesis	3
CHAPTER 2—LITERATURE REVIEW	4
2.0 Cardiometabolic Health	4
2.0.1 Background	4
2.0.2 Evaluating Cardiometabolic Health	9
Applied cardiometabolic health assessments.....	15
2.1 Body Fatness	15
2.1.1 Background	15
2.1.2 Evaluating Body Fatness.....	21
Applied body fatness assessments	24
2.2 Physical Fitness.....	25
2.2.1 Background	25
2.2.2 Evaluating Physical Fitness	29
Applied physical fitness assessments.....	32
2.3 Physical [In]activity and Sedentary Behaviour.....	32
2.3.1 Background	32
Physical [in]activity physiological pathways to CVD	33
Sedentary behaviour physiological pathways to CVD	35
2.3.2 Evaluating Physical [In]activity and Sedentary Behaviour	37
Objective measures	38

Subjective measures	41
Applied physical [in]activity and sedentary behaviour assessment.....	43
2.4 Nutrition	43
2.4.1 Background	43
Carbohydrate	44
Protein	50
Lipids	52
Fruits and vegetables.....	53
2.4.2 Evaluating Nutrition.....	55
Applied nutritional assessment	56
2.5 Sleep.....	56
2.5.1 Background	56
2.5.2 Evaluating Sleep	58
Objective measures.	58
Self-report measures.	60
Applied sleep assessment.....	61
2.6 Summary	61
2.6.1 Applied Measures Analysis	62
CHAPTER 3—METHODOLOGY	64
Pre-Adolescent Cardiometabolic Associations and Correlates:	
PACMAC Methodology and Study Protocol	64
3.0 PACMAC Study Protocol.....	64
3.0.1 Research Study Objectives.....	66
3.1 Methodology	66
3.1.1 Study Design	66
3.1.2 Study Participants and Recruitment.....	67
3.1.3 Cultural Sensitivity	67
3.2 Data Collection and Analysis.....	68
3.2.1 Demographics	68
3.2.2 Cardiometabolic Health	69
3.2.3 Adiposity and Anthropometrics	69
3.2.4 Physical Fitness.....	69
3.2.5 Physical Activity.....	70
3.2.6 Nutrition.....	70

3.2.7 Sleep.....	70
3.3 Quality Control	71
3.3.1 Development of Protocols.....	71
3.3.2 Training of Research Assistants.....	71
3.3.3 Data Collection	73
3.3.4 Data Cleaning.....	73
3.4 Sample Size Calculations.....	73
3.5 Statistical Analysis.....	74
3.5.1 Principal Component Analysis	74
Food frequency questionnaire principal components analysis	75
Cardiometabolic principal components analysis.....	78
3.5.2 Univariate Analysis.....	80
3.5.3 Multivariate Analysis.....	80
3.6 Dissemination of Findings	81
3.7 Discussion	82
CHAPTER 4—RESEARCH OBJECTIVE 1	83
Associations With Adiposity in Preadolescent Children: Fitness, Physical [In]activity, Sedentary Behaviour, Nutrition, and Sleep	83
4.0 Background	83
4.1 Methodology	84
4.1.1 Recruitment and Participants	85
4.1.2 Study Design	85
4.2 Data Collection and Analysis.....	88
4.2.1 Demographics	88
4.2.2 Adiposity and Anthropometrics	88
4.2.3 Physical Fitness.....	89
4.2.4 Physical [In]activity and Sedentary Behaviour.....	91
4.2.5 Nutrition	92
4.2.6 Sleep.....	93
4.3 Statistical Analysis.....	94
4.4 Results.....	95
4.4.1 Univariate Models.....	95
4.4.2 Multivariate Models.....	95
4.5 Discussion	98

4.5.1 Comparison With Other Studies	99
4.5.2 Limitations and Strengths of This Study	102
4.5.3 Research Implications	103
4.6 Conclusion	104
CHAPTER 5—RESEARCH OBJECTIVE 2	105
Associations with Cardiometabolic Health in Preadolescent Children: Fatness, Fitness, Physical [In]activity, Sedentary Behaviour, Nutrition, and Sleep	105
5.0 Background	105
5.1 Methodology	106
5.1.1 Recruitment and Participants	106
5.1.2 Study Design	108
5.2 Data Collection and Analysis	109
5.2.1 Demographics	109
5.2.2 Cardiometabolic Health	110
5.2.3 Adiposity and Anthropometrics	111
5.2.4 Physical Fitness	113
5.2.5 Physical [In]activity and Sedentary Behaviour	114
5.2.6 Nutrition	115
5.2.7 Sleep	116
5.3 Statistical Analysis	117
5.4 Results	118
5.4.1 Cardiometabolic Factor Correlations and Analysis	118
5.4.2 Univariate Models	119
5.4.3 Multivariate Models	119
5.5 Discussion	124
5.5.1 Comparison With Other Studies	124
5.5.2 Limitations and Strengths of This Study	127
5.5.3 Research Implications	128
5.6 Conclusion	129
CHAPTER 6—GENERAL DISCUSSION AND CONCLUSIONS	130
6.0 Summary of Findings	130
6.0.1 Objective 1	130
6.0.2 Objective 2	134

6.0.3 Implications.....	138
6.0.4 Future Directions, Limitations, and Strengths	139
6.1 Conclusion	140
References.....	141
Appendices	
Appendix A. PACMAC Questionnaire	195
Appendix B. Standard Operating Protocols	220
Appendix C. Study Invitation Letter	229
Appendix D. Participant Paperwork.....	230
Appendix E. Factor Loadings of Food Items Grouped into Three Identified Dietary Patterns in New Zealand Children.....	235

List of Tables

Table 1. <i>Contributions to Publications</i>	vi
Table 2. <i>Sources and Functions of Key Adipokines</i>	18
Table 3. <i>Physiological Benefits of Physical Fitness</i>	27
Table 4. <i>Function of Micronutrients</i>	47
Table 5. <i>Standard Operating Protocol Foundations</i>	72
Table 6. <i>Cardiometabolic Patterns</i>	79
Table 7. <i>Participant Data Description: Categorical Variables</i>	87
Table 8. <i>Participant Data Description: Continuous Variables</i>	91
Table 9. <i>Linear Association Between Body Fatness and Lifestyle Factors</i>	96
Table 10. <i>Participant Data Description: Categorical Variables</i>	108
Table 11. <i>Participant Data Description: Continuous Variables</i>	111
Table 12. <i>Linear Association Between Cardiometabolic Health and Lifestyle</i> <i>Behaviours</i>	121
Table 13. <i>Cardiometabolic Patterns</i>	123

List of Figures

Figure 1. Noncommunicable diseases (NCDs).....	5
Figure 2. The progression of atherosclerosis.. ..	6
Figure 3. The progression of cardiovascular disease (CVD).....	9
Figure 4. The role of inflammation and lipotoxicity on obesity	16
Figure 5. Body fatness assessments.	21
Figure 6. Dietary intake	46
Figure 7. PACMAC measuring methods.	63
Figure 8. Study protocol.	66
Figure 9. Component plots with factor diagrams from principal component analysis (PCA) with varimax orthogonal rotation.	76
Figure 10. Scree plot of eigenvalues of the 21 food groups from the principal component analysis (PCA).	77
Figure 11. Participant recruitment.	86
Figure 12. Scree plot of eigenvalues of the 21 food groups from the principal component analysis (PCA).	93
Figure 13. $\dot{V}O_{2max}$ and body fat percent linear quadratic and cubic analysis.....	98
Figure 14. Participant recruitment.	107
Figure 15. Scree plot of eigenvalues of the 21 food groups from principal component analysis (PCA).	116
Figure 16. Component plots with factor diagrams from principal component analysis (PCA) with varimax orthogonal rotation.	120

List of Abbreviations

- AIx = Augmentation Index Pressure
- BIA = Bioelectrical Impedance Analysis
- BMI = Body Mass Index
- CVD = Cardiovascular Disease
- CDC = Centers for Disease Control and Prevention
- CBP = Central Blood Pressure
- CSHQ = Child Sleep Habits Questionnaire
- DBP = Diastolic Blood Pressure
- FMI = Fat Mass Index
- FMD = Flow-Mediated Dilation
- HbA1c = Glycosylated Haemoglobin
- HR = Heart Rate
- HRM = Heart Rate Monitors
- HDL-C = High Density Lipoproteins
- LDL-C = Low Density Lipoproteins
- 20-MST = Maximal multistage 20-metre shuttle run test
- MOE = Ministry of Education
- MOH = Ministry of Health
- NCD(s) = Noncommunicable disease(s)
- OECD = Organization for Economic Co-operation and Development
- PEDALS FFQ = Physical Activity, Exercise, Diet, And Lifestyle Study Food
Frequency Questionnaire
- PACMAC = PreAdolescent CardioMetabolic Associations and Correlates
- PCA = Principal Components Analysis

PWA = Pulse Wave Analysis

PWV = Pulse Wave Velocity

SBP = Systolic Blood Pressure

TC = Total Cholesterol

TG = Triglycerides

WC = Waist Circumference

WHtR = Waist-to-Height Ratio

WHR = Waist-to-Hip Ratio

WHO = World Health Organization

YPAQ = Youth Physical Activity Questionnaire

Definition of Terms

Cardiometabolic health factors = Blood pressure, cholesterol, vascular, and carbohydrate-metabolic

Cardiometabolic risk factors/cardiometabolic complications = obesity, hypertension, hyperglycaemia, and hyperlipidaemia

Lifestyle factors/modifiable lifestyle factors = Body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep

Physical activity = Active behaviours resulting in an increased energy expenditure

Physical fitness = Cardiorespiratory fitness and muscular fitness

Physical [in]activity = Physical activity and physical inactivity

Physical inactivity = Achieving less than the recommended amount of daily physical activity

Sedentary behaviour = Low energy expenditure for long periods of time

$\dot{V}O_{2\max}$ = Maximal oxygen uptake

CHAPTER 1—INTRODUCTION

1.0 Circumstances and Statement of the Problem

Whereas cardiovascular disease (CVD) is typically associated with middle or old age, the atherosclerotic process often initiates early in childhood, and is occurring at an increasing rate (Bridger, 2009; Cote, Harris, Panagiotopoulos, Sandor, & Devlin, 2013; Dobashi, 2016; Sorensen et al., 1994; Stoner, Lambrick, Faulkner, & Young, 2013). In children, early onset of atherosclerosis has been linked to cardiometabolic risk factors such as obesity, and worldwide obesity statistics have nearly tripled since 1975 (World Health Organization [WHO], 2017c). Additionally, in 2016, over 340 million children and adolescents (aged 5 to 19 years) globally were overweight or obese (WHO, 2017c). Overweight and obese children are at increased risk of developing cardiometabolic complications, which could lead to noncommunicable diseases (NCDs; Anderson et al., 2015; Centers for Disease Control and Prevention [CDC], 2015b; Ministry of Health [MOH], 2015b; Ogden et al., 2016; WHO, 2017b, 2017c), and NCDs kill 40 million people each year (WHO, 2017b). Furthermore, various lifestyle factors have shown associations with cardiometabolic risk factors; however, it is unknown which lifestyle factor(s) correlate(s) most strongly with cardiometabolic health.

The prevalence of childhood obesity is a significant public health concern (S. A. Ali, Ali, Suhail, Tikmani, & Bano, 2016). In New Zealand, childhood obesity increased from 8% in 2006-07 to 12% in 2016-17 (MOH, 2015b), and in 2014, overweight and obesity prevalence in New Zealand children (aged 5 to 17 years) was the third highest worldwide (Anderson et al., 2017; Kelly & Swinburn, 2015; Organisation for Economic Co-Operation and Development [OECD], 2014). Although frequently cited causes of obesity include: lower than ideal physical fitness (Cohen et al., 2011; Czyż et al., 2017;

Stratton et al., 2007), insufficient daily physical activity (O. Ali et al., 2014; Dumuid et al., 2018; WHO, 2017d), sedentary behaviour (Carson, Hunter, et al., 2016; Carson, Tremblay, Chaput, & Chastin, 2016; Griffiths et al., 2016; Healy & Owen, 2010), unhealthy diet (Harrex et al., 2017; WHO, 2015, 2017a), and inadequate sleep (Davison et al., 2017; Sayin & Buyukinan, 2016; Skidmore et al., 2013), no known study has comprehensively assessed the simultaneous associations among these lifestyle factors with cardiometabolic health in preadolescent children. Consequently, uncertainty emerges as to which lifestyle factor(s) associate(s) more strongly with cardiometabolic risk factors, making it difficult to determine the ideal public health intervention and prevention strategy required for improving cardiometabolic health in children.

Most public resources are aimed towards the fight against obesity; however, in children the lifestyle factor(s) that associate(s) more strongly with obesity have not been definitively identified. Therefore, additional research analysing the associations among lifestyle factors (body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep) with cardiometabolic health is essential. Overall, obesity is an outcome of a number of factors and addressing the disease is not the final goal; improving and maintaining cardiometabolic health is the primary objective. Findings from this study will add to the body of knowledge that informs health professionals and health policy writers regarding health and wellness of children and possibly make a positive impact on the deteriorating health of New Zealand children.

1.1 Objectives of Research

The overall aim of this thesis was to examine simultaneously the associations among lifestyle factors (body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep) with cardiometabolic health in New Zealand preadolescent

children aged 8 to 10 years. Accordingly, the study was undertaken in primary schools across three regions in New Zealand (Wellington, Canterbury, and Otago) specifically to examine these questions:

1. What are the associations among physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with body fatness in preadolescent children? (Objective 1, Chapter 4)
2. What are the associations among body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with cardiometabolic health in preadolescent children? (Objective 2, Chapter 5)

1.2 Outline of Thesis

This thesis is divided into six chapters and presented as a hybrid thesis displaying traits of thesis by publication and a traditional monograph approach. This first chapter (Chapter 1) introduces the thesis and presents the study rationale. Chapter 2 presents a review of the literature on cardiometabolic health, body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep. Chapter 3 is my study protocol paper, which was published in the *BMJ Open* under the title, “Pre-Adolescent Cardiometabolic Associations and Correlates: PACMAC Methodology and Study Protocol” (Castro et al., 2014). Chapter 4 (Objective 1) and Chapter 5 (Objective 2) were written as research papers and will be submitted for publication after the completion of my PhD. The final chapter (Chapter 6) summarises the findings, implications, future direction, and general conclusions of this thesis. It should be noted that Chapter 4 and Chapter 5 were written as research papers for publication and are based on similar data sets; therefore, the background and methodological sections of these two chapters will have some duplication.

CHAPTER 2—LITERATURE REVIEW

The following chapter provides an overview of the literature relating to cardiometabolic health and lifestyle factors: body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep. Many lifestyle factors contribute to cardiometabolic complications; however, uncertainty emerges as to which lifestyle factor(s) associate(s) more strongly with cardiometabolic risk factors in children. This literature review provides an overview of each of the listed lifestyle factors and cardiometabolic risk factors in children, followed by a rationale and evidence supporting the measuring methods applied in this study.

2.0 Cardiometabolic Health

2.0.1 Background

Poor cardiometabolic health refers to a group of NCDs that encompass both cardiac and metabolic conditions. Moreover, cardiometabolic risk factors tend to cluster, creating complications that increase the probability of developing an NCD such as CVD (see Figure 1; Ahrens et al., 2014; Bridger, 2009; Trandafir, Frasinariu, Corciovă, Boiculese, & Moscalu, 2017). Of all NCDs, CVD encompasses the highest mortality rates and annually more people die from CVD than any other cause (Frieden & Jaffe, 2018; Trandafir et al., 2017; WHO, 2017a). Additionally, in 2015 it was projected that 17.7 million people would die from CVD, representing 31% of all global deaths (WHO, 2017a). While CVD is typically associated with middle or old age, the atherosclerotic process (see Figure 2) often initiates early in childhood, within the first and second decades of life, and is occurring at an increasing rate (Sorensen et al., 1994; Stoner et al., 2013; Trandafir et al., 2017).

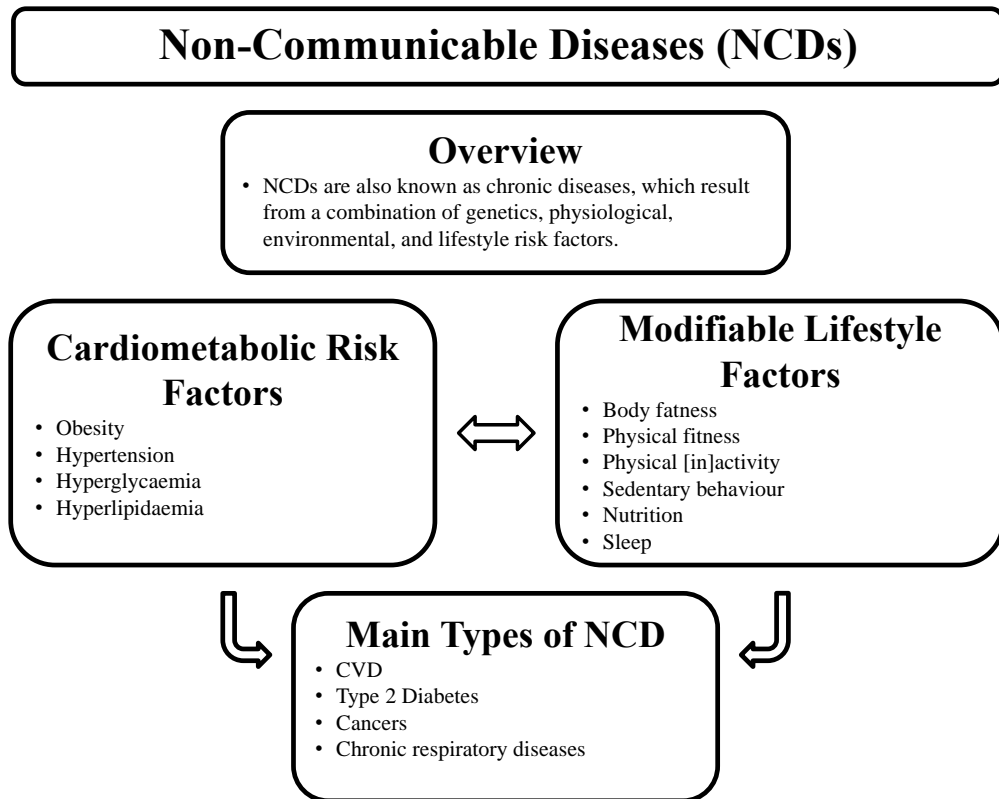
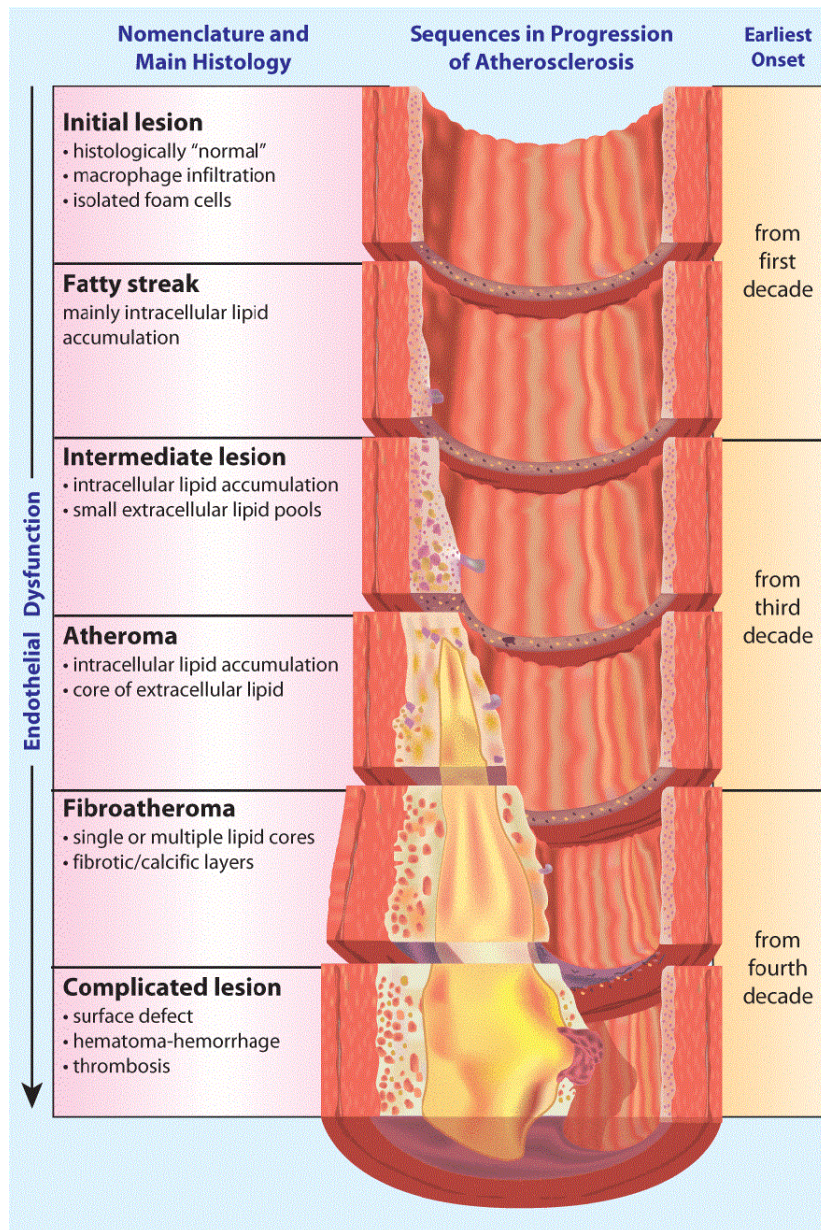


Figure 1. Noncommunicable diseases (NCDs).

Vascular disease is a combination of vascular injury and vascular repair outcomes including: inflammation, oxidative stress, immune dysfunction, vasoconstriction (constriction of blood vessels), increased vascular permeability, and thrombosis, or clotting of the blood (Stein et al., 2008; Vanhoutte, Shimokawa, Tang, & Feletou, 2009). Collectively, vascular disease consists of circulatory system disorders such as endothelial dysfunction, which can lead to atherosclerosis (Stein et al., 2008).

Atherosclerosis is a chronic inflammatory process that initiates from built-up fatty substances inside the artery channels (Beauloye et al., 2007; Kenney, Wilmore, & Costill, 2015). The fatty substances are referred to as plaque or atheroma and are made up of



*Figure 2. The progression of atherosclerosis. Adapted from *Vascular Diseases for the Non-Specialist: An Evidence-Based Guide* by T. P. Navarro, A. Dardik, D. Junqueira, and L. Cisneros (Eds.), 2017, p. 37. Copyright 2017 by Springer.*

calcium, lipoproteins, cholesterol, inflammatory cells, fibrin, and other cellular waste substances, although low density lipoproteins (LDL-C) remain the most important component of atherosclerotic plaque accumulation (Navarro et al., 2017; Ross & Glomset, 1976; Sorensen et al., 1994; Stoner, Lambrick, et al., 2014). The progression of

atherosclerotic plaque buildup begins with LDL-C accumulation in the blood vessel intima (Navarro et al., 2017; Ross & Glomset, 1976; Sorensen et al., 1994; Stoner, Lambrick, et al., 2014; Tedgui & Mallat, 1999). This accumulated LDL-C becomes oxidised by free radicals and through glycation, which in turn initiates the recruitment of monocytes-macrophages (Kenney et al., 2015; Tedgui & Mallat, 1999). Modified LDL-C consumed by macrophage scavenger receptors is transformed into foam cells (Navarro et al., 2017; Tedgui & Mallat, 1999). This process results in the formation of a fibrous cap containing smooth muscle cells, which permits stabilization of the plaque (Kenney et al., 2015; Navarro et al., 2017; Ross & Glomset, 1976; Sorensen et al., 1994; Stoner, Lambrick, et al., 2014; Tedgui & Mallat, 1999). These substances accumulate over time within the artery walls, narrowing the lumen of the artery, which reduces blood flow through the vessel and thus influences/elevates blood pressure (Beauloye et al., 2007; Ross & Glomset, 1976). Collectively, restricted blood flow has a detrimental effect on the functioning of the heart, brain, kidneys, arms, legs, and pelvis (Ahrens et al., 2014; Bridger, 2009; Trandafir et al., 2017).

Endothelial dysfunction precedes atherosclerosis (Bonetti, Lerman, & Lerman, 2003; Davignon & Ganz, 2004). Endothelial cells are a thin layer of cells that line the inside of blood vessels (Bonetti et al., 2003; Davignon & Ganz, 2004). These cells form a smooth protective layer between the blood circulating through the artery in the lumen and the vascular smooth muscle known as the endothelium (Bonetti et al., 2003; Davignon & Ganz, 2004; Kenney et al., 2015). When the endothelium is functioning properly, it produces molecules (nitric oxide, endothelin, and prostaglandin) that provide a protective barrier between toxic substances in the blood and the vessel wall, which in turn helps regulate vascular relaxation, blood clotting, and immune function, so that blood elements

and the vascular smooth muscle remain normal (Bonetti et al., 2015; Davignon & Ganz, 2004; Kenney et al., 2015). However, endothelial dysfunction occurs when endothelial cells become disrupted by oxidised free radicals, causing excessive oxidative stress (Davignon & Ganz, 2004; Galley & Webster, 2004; Sprague, Chesler, & Magness, 2010). The disrupted endothelial cells interfere with the normal release and regulation of nitric oxide (Bonetti et al., 2003; Charakida et al., 2005; Davignon & Ganz, 2004), which obstructs the endothelial cells' ability to dilate properly, causing an imbalance between the narrowing and widening of blood vessels (Bonetti et al., 2003; Charakida et al., 2005; Davignon & Ganz, 2004; Galley & Webster, 2004). In addition, the amount of shear stress stimulus on the intima layer of vessel walls also plays a role in regulating nitric oxide (Davignon & Ganz, 2004; Galley & Webster, 2004; Sprague et al., 2010).

Cardiometabolic risk factors that lead to endothelial dysfunction, then atherosclerosis, and subsequently to CVD include: excessive accumulation and storage of adipose tissue in the body (obesity), elevated levels of LDL-C and plasma homocysteine in the bloodstream (hyperlipidaemia), too much glucose in the bloodstream (hyperglycaemia), and excessive blood pressure on the artery wall (hypertension) exerting shear stress on the vulnerable endothelium (see Figure 3; Kenney et al., 2015; Ross & Glomset, 1976; Sorensen et al., 1994; Widmer & Lerman, 2014; WHO, 2017a, 2017b). Preceding cardiometabolic risk factors are modifiable lifestyle factors including but not limited to: excessive body fatness (Bridger, 2009; Trandafir et al., 2017), lower than ideal physical fitness (Cohen et al., 2011; Czyż et al., 2017; Stratton et al., 2007), insufficient daily physical activity (O. Ali et al., 2014; Dumuid et al., 2018; WHO, 2017d), sedentary behaviour (Carson, Hunter, et al., 2016; Carson, Tremblay, et al., 2016; Griffiths et al., 2016; Healy & Owen, 2010), unhealthy diet (Harrex et al., 2017; WHO, 2015, 2017a), and

inadequate sleep (Davison et al., 2017; Sayin & Buyukinan, 2016; Skidmore et al., 2013). However, no known study has examined simultaneously the associations among the listed lifestyle factors with cardiometabolic risk factors in preadolescent children. Consequently, uncertainty remains as to which lifestyle factor(s) associate more strongly with cardiometabolic risk factors in preadolescent children.

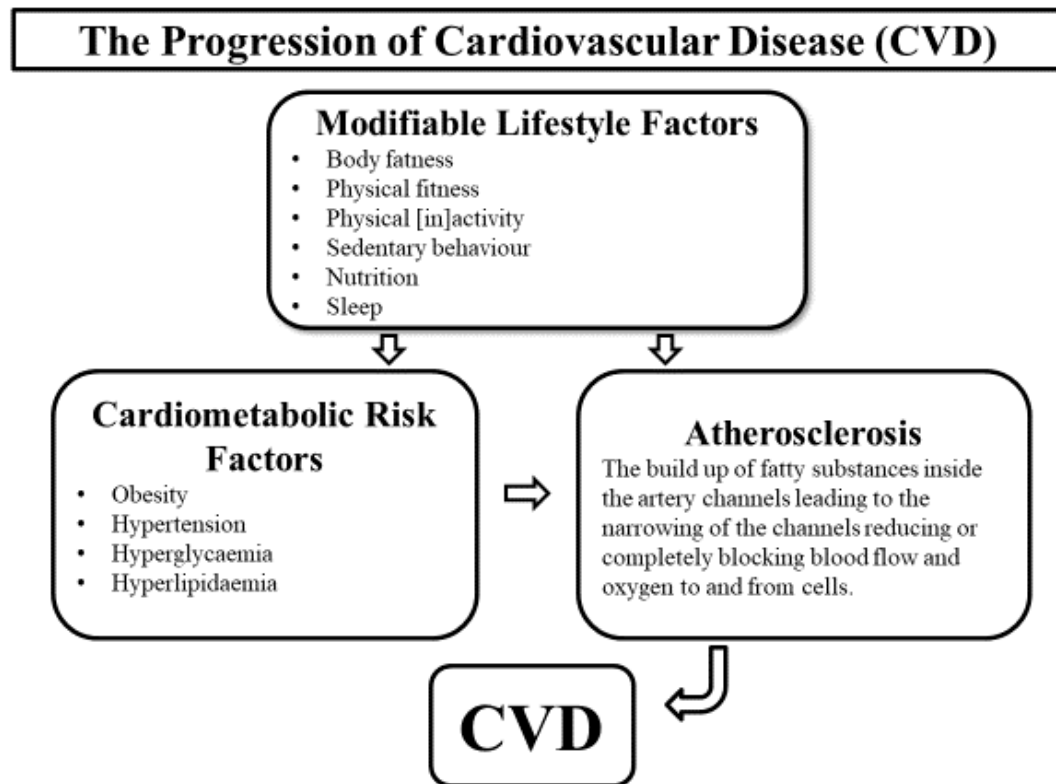


Figure 3. The progression of cardiovascular disease (CVD).

2.0.2 Evaluating Cardiometabolic Health

In preadolescent children, cardiometabolic health can be assessed utilising a collection of approaches including: anthropometric indicators (for further information, see section 2.1.2 on evaluating body fatness), pulse wave analysis (PWA), pulse wave velocity (PWV), flow-mediated dilation (FMD), and cardiometabolic biochemical markers. The following paragraphs elaborate on the indicated cardiometabolic assessment methods and

present evidence supporting the evaluating procedures chosen for this study over other approaches.

Procedures for gauging blood pressure were chosen based on their noninvasive nature and the study's setting. Blood pressure gauged from the upper arm brachial artery is termed peripheral blood pressure (diastolic DBP, systolic SBP), whereas central blood pressure (CBP) is gauged from the aorta, which is the largest artery through which the heart pumps blood (Hirata, Kawakami, & O'Rourke, 2006). Central blood pressure is the resistance the heart encounters while attempting to pump blood throughout the body. When CBP is elevated, the heart must work harder, which could lead to heart failure. As a result, CBP has been shown to predict hypertension and potential cardiometabolic complications (Hirata et al., 2006; Roman et al., 2007). Central blood pressure can be gauged noninvasively by utilising PWA and PWV.

Pulse wave analysis assesses SBP, DBP, CBP, heart rate (HR), and augmentation index pressure (AIx; Stoner, Credeur, Dolbow, & Gater, 2015). The mentioned indicators are estimated by analysing a generated CBP waveform (Stoner et al., 2015). Initially, the pulse pressure wave is formed from the combination of the incident wave (systolic) and waves reflected back from the perimeter (diastolic; J. I. Davies & Struthers, 2003). Then this waveform is transmitted through the arterial wall, generating a corresponding aortic pulse pressure waveform (augmentation pressure), which can be measured and examined on a digital device (J. I. Davies & Struthers, 2003; Stoner et al., 2015). A study carried out on New Zealand children (aged 8 to 10 years; $n = 57$) found that PWA is an appropriate assessment of cardiometabolic risk in children (Stoner, Lambrick, et al., 2014). However, researchers analysed a small sample size in a laboratory setting; therefore, the results do not allow for accurate comparison to a study design with a sample size of 392 preadolescent

children. Yet, PWA is deemed an appropriate assessment method as it investigates the arterial tree mechanisms, including CBP and arterial wave reflection (AIx), and elevated AIx has been identified as a CVD risk predictor (Stoner, Lambrick, et al., 2014). Furthermore, PWA is a simple, noninvasive, valid, and reliable measurement of cardiometabolic risk, particularly in large paediatric epidemiological studies (Hirata et al., 2006; Stoner et al., 2013; Stoner, Lambrick, et al., 2014). However, to my knowledge, there are currently no standard PWA values for assessing a large group of preadolescent children in an uncontrolled field-based environment. Therefore, further paediatric research is necessary in evaluating PWA with other recognised techniques such as PWV and FMD with the intention of establishing reliable standards for applying PWA in preadolescent children (Stoner et al., 2013; Stoner, Lambrick, et al., 2014).

Pulse wave velocity identifies functional changes in the large arteries by analysing the rate at which pressure waves transmit through the vessel (Hirata et al., 2006; McCloskey et al., 2014; Stoner et al., 2015). The velocity of the wave is determined by the time needed for the waveform to pass between two points a measured distance apart (pulse wave distance; McCloskey et al., 2014; Stoner et al., 2015). These readings are frequently obtained on the carotid and femoral arteries, which represent the aortoiliac pathway (Hirata et al., 2006; Reusz et al., 2010). Pulse wave velocity is the most widely accepted method to assess arterial stiffness in children and adolescents by the American Heart Association Atherosclerosis, Hypertension, and Obesity in Youth committee (Thurn et al., 2015; Urbina et al., 2009). A study carried out on German children, adolescents, and young adults (aged 8 to 22 years; $n = 1445$) found that PWV can help identify arterial stiffness and predict cardiometabolic risk in children and adolescents (Elmenhorst et al., 2015). In this study, data were collected in a school setting from a predominantly Caucasian population;

participants only rested for 5 to 10 minutes before blood pressure was assessed. If a measured blood pressure was elevated, the value was excluded ($n = 578$) from the analyses, thus the results from this study do not provide an accurate sample of blood pressure numbers collected in a school setting (Elmenhorst et al., 2015). Another study carried out on American children and adolescents (aged 4 to 18 years, $n = 159$) found that increased PWV was associated with body fatness and hypertension (Kulsum-Meccci et al., 2017). Also in this study, the majority of the participants were Caucasian; therefore, the outcomes from these results may not allow a meaningful conclusion to be drawn for any other racial or ethnic group. Furthermore, it is necessary to point out a few limitations associated with most of the PWV data analysed in children including: the majority of the previous data examined were collected from Caucasian children (Elmenhorst et al., 2015; Kulsum-Meccci et al., 2017; McCloskey et al., 2014; Thurn et al., 2015), various studies utilised dissimilar markers to assess PWV (e.g., some studies assessed carotid artery to radial artery while others assessed carotid artery to femoral artery; J. I. Davies & Struthers, 2003, Elmenhorst et al., 2015; McCloskey et al., 2014; Thurn et al., 2015), and to my knowledge, there are currently no standardised values for assessing children. Consequently, additional research is required to better understand the relationship among PWV and cardiometabolic risk factors in preadolescent children from all backgrounds, with the intention of establishing reliable standards for implementing PWV in paediatric research.

Flow-mediated dilation is the assessment of blood flow by examining endothelial (dys)function and quantifying the percentage increase in the brachial artery diameter in response to blood flow (Ras, Streppel, Draijer, & Zock, 2013; Stoner et al., 2015). By expression, FMD can be defined as the dilation of an artery following an increase in luminal blood flow and internal wall shear stress (Thijssen et al., 2011). A dysfunctional

endothelium has been proven to precede and predict cardiometabolic complications and subsequently CVD (Ras et al., 2013; Stoner et al., 2015; Thijssen et al., 2011). A systematic review that examined 23 studies including 14,753 subjects (aged 40 to 80 years) across the globe, found that FMD can predict cardiometabolic risk factors and subsequently CVD (Ras et al., 2013). This study review analysed a large sample; however, all the participants were adults. Therefore, the results reported in this systematic review are not comparable to studies with samples of preadolescent children aged 8 to 10 years. A study carried out on obese German children (aged 11 to 16; $n = 96$) found that FMD is a consistent and significant marker of identifying early signs of CVD risk in obese children (Meyer, Kundt, Steiner, Schuff-Werner, & Kienast, 2006). However, in this study, the participants were already obese; therefore, the study participants did not represent a population of children of all body types. Additionally, a study carried out on children (aged 6 to 17 years; $n = 43$) with chronic kidney disease found that children with chronic kidney disease had an increased occurrence of reduced FMD of the brachial artery (A. C. Wilson et al., 2008). In this study, the participants were not free of disease, so do not resemble a population of normative data. Flow-mediated dilation is highly valid and considered the gold standard assessment for endothelial function, however this assessment method is also highly technical, requires a skilled operator (Stoner et al., 2015), and is impractical for large paediatric studies. Additionally, there is limited research available analysing cardiometabolic function in healthy preadolescent children utilising FMD, and to my knowledge, there are currently no standardised values for this age group. Therefore, additional paediatric research is essential to enhance the knowledge surrounding FMD and cardiometabolic health and to establish FMD as a reliable assessment method for endothelial (dys)function in preadolescent children.

Cardiometabolic biochemical markers are utilised to assess cardiometabolic risk factors associated with blood chemistry. Blood is collected utilising a venepuncture or finger prick. The venepuncture method punctures a vein with a needle, usually in the arm, whereas a finger prick pokes the tip of a finger drawing minimal blood. Collection of blood markers by venepuncture is invasive and impractical in preadolescent children, whereas blood marker collection by finger prick is simple, valid, reliable, and practical for large paediatric epidemiological studies. There is abundant paediatric research that validates high blood glucose levels (hyperglycaemia) and high levels of fat in the blood (hyperlipidaemia) as metabolic risk factors that predict cardiometabolic complications, and subsequent CVD into adulthood (Ahrens et al., 2014; Li et al., 2016; Sardinha et al., 2016; Trandafir et al., 2017). A study review carried out on European children (aged 2 to 10 years; $n = 18,745$) found that biochemical markers can be a significant indicator of early signs of metabolic disease in children (Ahrens et al., 2014). Another study carried out on European children (aged 8 to 17 years; $n = 4255$) discovered that biochemical markers provide a more accurate and consistent evaluation of cardiometabolic risk in children when compared to anthropometric indicators (Sardinha et al., 2016). Additionally, a study carried out on obese Romanian children (aged 8 to 14 years; $n = 78$) using biochemical markers found that early identification and treatment of cardiometabolic risk is important to reduce and prevent CVD into adulthood (Trandafir et al., 2017). However in this study, the participants were obese, thus do not provide a standard example of children of all body types.

Cardiometabolic blood markers are a significant aspect of examining and monitoring cardiometabolic health in children. However, additional research is essential to better understand the relationship among lifestyle factors with cardiometabolic biochemical markers in preadolescent children.

Applied cardiometabolic health assessments. In this study, cardiometabolic health was assessed utilising PWA and cardiometabolic biochemical markers: SBP, DBP, CBP, HR, AIX, total cholesterol (TC), high density lipoproteins (HDL-C), LDL-C, triglycerides (TG), serum glucose, and glycosylated haemoglobin (HbA1c). For further information, see section 5.2 in Chapter 5.

2.1 Body Fatness

2.1.1 Background

Childhood obesity continues to escalate as a global epidemic and a significant public health concern (Ajala, Mold, Boughton, Cooke, & Whyte, 2017; S. A. Ali et al., 2016; Czyż et al., 2017; W. H. Dietz, 2004; Sayin & Buyukinan, 2016). Worldwide obesity statistics have nearly tripled since 1975 (WHO, 2017c). In 2016, over 340 million children and adolescents (aged 5 to 19 years) globally were overweight or obese (WHO, 2017c). Furthermore, in New Zealand, childhood obesity increased from 8% in 2006-07 to 12% in 2016-17 (MOH, 2015b), and in 2014, overweight and obesity prevalence in New Zealand children was the third highest worldwide (Anderson et al., 2017; Kelly & Swinburn, 2015; OECD, 2014).

Obesity is a condition characterised by excessive accumulation and storage of adipose tissue in the body (S. A. Ali et al., 2016; WHO, 2017c). This adipose tissue accumulates when energy intake (nutrition) exceeds energy expenditure (physical activity and exercise), resulting in a positive energy balance over time (Romieu et al., 2017). There are two types of adipose tissue in the body: brown and white (DeClercq, Taylor, & Zahradka, 2008; Stoner, Gaffney, Wadsworth, & Page, 2014). Brown adipose tissue is involved in thermogenesis (DeClercq et al., 2008), whereas white adipose tissue is the main energy reservoir storing fat, hormones, and cytokines to be distributed to cells to help

regulate metabolism and insulin effectiveness and to be utilised for energy when necessary (DeClercq et al., 2008; Redinger, 2007). The development and functioning of white adipose tissue is impacted by various cells (vascular cells, fibroblasts, lymphocytes, pre-adipocytes, and macrophages [Ouchi, Parker, Lugus, & Walsh, 2011; Wang & Nakayama, 2010]). As outlined in Figure 4, white adipose tissue is significant to cardiometabolic dysfunction as it initiates the pathological mechanisms associated with obesity, including lipotoxicity and inflammatory responses (DeClercq et al., 2008; Redinger, 2007).

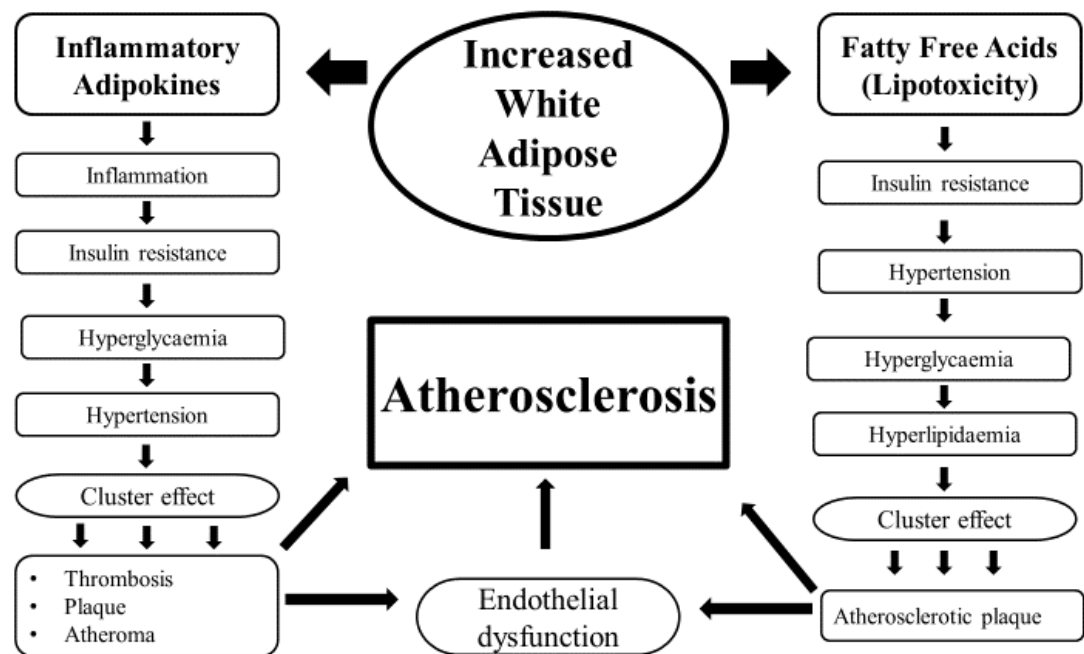


Figure 4. The role of inflammation and lipotoxicity on obesity. Adapted from “The Pathophysiology of Obesity and Its Clinical Manifestations,” by R. N. Redinger, 2007, *Gastroenterology & Hepatology*, 11, p. 856. Copyright 2007 by Gastro-Hep Communications.

White adipose tissue executes several functions and has been identified as a key component of the metabolic system (Trayhurn & Wood, 2004). Along with the pivotal role of lipid storage, its major function is stockpiling various hormones and proteins referred to as adipokines (Ouchi et al., 2011; Trayhurn & Wood, 2004). Adipokines consist of billions of cells that perform pro- and anti-inflammatory actions such as assisting with body fatness regulation and the storage of triacylglycerol in fat deposits as energy reserves (Nakamura, Fuster, & Walsh, 2011; Ouchi et al., 2011; Redinger, 2007). However, abnormal production and/or secretion of adipokines can contribute to the pathogenesis of obesity related complications (Ouchi et al., 2011; Trayhurn & Wood, 2004). This occurs because the expansion of fat cells initiates an undesired molecular and cellular variation of the metabolic process, which triggers secretion of adipokines by white adipose tissue, which is identified as an inflammatory response as shown in Figure 4 (Ouchi et al., 2011; Redinger, 2007; Wang & Nakayama, 2010). Adipocytes, macrophages, fibroblasts, lymphocytes, neutrophils, and precursor, foam, endothelial, and immune cells all contribute to the release of adipokines (Fasshauer & Blüher, 2015; Ouchi et al., 2011; Rabe, Lehrke, Parhofer, & Broedl, 2008). Table 2 provides a summary of the sources and functions of key adipokines (Ouchi et al., 2011; Rabe et al., 2008). The release of adipokines could lead to pathological cardiometabolic complications, which disrupt endothelial cells (thrombosis, plaque, atheroma), result in a dysfunctional endothelium, and ultimately lead to atherosclerotic disease (Ouchi et al., 2011; Redinger, 2007; Wang & Nakayama, 2010). For further information on endothelial dysfunction, see section 2.0.1 for background on cardiometabolic health.

Table 2

Sources and Functions of Key Adipokines

Adipokine	Primary source(s)	Binding partner or receptor	Function
Adiponectin	Adipocytes	Adiponectin receptors 1 and 2, T-cadherin, calreticulin-CD91	Insulin sensitiser Anti-inflammatory
Leptin	Adipocytes	Leptin receptor	Appetite control through the central nervous system
Secreted frizzled-related protein 5	Adipocytes	WNT5a	Suppression of pro-inflammatory WNT signalling
Resistin	Peripheral blood mononuclear cells	Unknown	Promotes insulin resistance and inflammation through interleukin-6 and tumour necrosis factor secretion from macrophages
Retinol binding protein 4	Liver, adipocytes, macrophages	Retinol (vitamin A), transthyretin	Implicated in systemic insulin resistance
Lipocalin 2	Adipocytes, macrophages	Unknown	Promotes insulin resistance and inflammation through tumour necrosis factor secretion from adipocytes
Angiopoietin-like protein 2	Adipocytes, other cells	Unknown	Local and vascular inflammation
Tumour necrosis factor	Stromal vascular fraction cells, adipocytes	Tumour necrosis factor receptor	Inflammation, antagonism of insulin signalling
Interleukin-6	Adipocytes, stromal vascular fraction cells, liver, muscle	Interleukin-6 receptor	Changes with source and target tissue
Interleukin-18	Stromal vascular fraction cells	Interleukin -18 receptor, Interleukin-18 binding protein	Broad-spectrum inflammation
Chemokine ligand 2	Adipocytes, stromal vascular fraction cells	Chemokine ligand 2	Monocyte recruitment
Chemokine ligand 5	Stromal vascular fraction cells (macrophages)	Chemokine ligand 2	Antagonism of insulin signalling through the Janus kinase - signal transducer and activator of transcription pathway
Nicotinamide phosphoribosyl transferase	Adipocytes, macrophages, other cells	Unknown	Monocyte chemotactic activity

Obesity is also associated with lipotoxicity, which is a metabolic disorder resulting from the accumulation of fat in nonadipose tissue (Engin, 2017). Lipotoxicity is the failure of the excessive buildup of fat in cell cytoplasm to combine into lipid droplets, which causes an increase of circulating fatty acids and may result in toxic levels of fatty acids within nonadipose tissue (Engin, 2017). Some previous research has indicated that storing fatty acid as TG inside fat cells protects against fatty acid toxicity. If not stored, free fatty acids would circulate unmonitored within the blood vessels and produce oxidative stress by spreading throughout the body (Engin, 2017). The excessive storage of fat that causes obesity ultimately triggers the release and circulation of fatty acids from lipolysis, which is stimulated by the supportive state of obesity. The discharge of free fatty acids then triggers lipotoxicity, creating oxidative stress to the cytoplasm of cells and mitochondria (Engin, 2017). This excessive accumulation of fat results in an imbalance between the amount of lipids produced and lipids utilised, which is responsible for the processes associated with cellular dysfunction and metabolic disease in many organs (e.g., heart, liver, pancreas) throughout the body (Redinger, 2007; Van Herpen & Schrauwen-Hinderling, 2008). Adipose tissue also stores and releases active compounds such as free fatty acids into the blood circulation; consequently, elevated levels of free fatty acids are frequently associated with obesity (Boden, 2008; M. D. Jensen, 2006). Ordinarily, released free fatty acids travel to cells to be used as energy; however, when introduced in large amounts, they can increase internal glucose production rates and cause insulin resistance (Boden, 2008; M. D. Jensen, 2006).

The free fatty acids released from TG also obstruct lipogenesis, which impedes the clearance of serum triacylglycerol levels that then contributes to the development of hyperlipidaemia (Boden, 2008; Redinger, 2007). Additionally, the secretion of free fatty

acids by endothelial lipoprotein lipase from accumulated serum TG within elevated lipoproteins causes lipotoxicity, resulting in insulin receptor dysfunction (M. D. Jensen, 2006; Redinger, 2007). This subsequently results in insulin resistance, which can lead to hyperglycaemia (M. D. Jensen, 2006; Redinger, 2007). Furthermore, free fatty acids also reduce usage of insulin-stimulated muscle glucose, which can also lead to a hyperglycaemic state. (M. D. Jensen, 2006; Redinger, 2007). Collectively, these circumstances lead to a cascade of cardiometabolic pathophysiological complications such as sustained insulin resistance, hyperglycaemia, hyperlipidaemia, hypertension, and obesity, which can create a cluster effect and increase the risk of developing a dysfunctional endothelium and subsequently atherosclerosis (Galley & Webster, 2004; Redinger, 2007).

Consequently, obesity has been identified as one of the most significant cardiometabolic risk factors in children (P. Dietz, Hoffmann, Lachtermann, & Simon, 2012; Sayin & Buyukinan, 2016). Preceding cardiometabolic risk factors are modifiable lifestyle factors linked to childhood obesity prevalence, which include: lower than ideal physical fitness (Cohen et al., 2011; Czyż et al., 2017; Stratton et al., 2007), insufficient daily physical activity (O. Ali et al., 2014; Dumuid et al., 2018; WHO, 2017d), sedentary behaviour (Carson, Hunter, et al., 2016; Carson, Tremblay, et al., 2016; Griffiths et al., 2016; Healy & Owen, 2010), unhealthy diet (Harrex et al., 2017; WHO, 2015, 2017a), and inadequate sleep (Davison et al., 2017; Sayin & Buyukinan, 2016; Skidmore et al., 2013). However, uncertainty exists as to whether body fatness or another lifestyle factor associates more strongly with cardiometabolic risk factors in preadolescent children.

2.1.2 Evaluating Body Fatness

Body fatness can be evaluated multiple ways; the most common methods include: bioelectrical impedance analysis (BIA), skinfold calliper, hydrostatic weighing, DEXA scan, and anthropometric indicators (see Figure 5). The following paragraphs elaborate on the field-based anthropometric indicators commonly implemented in assessing preadolescent children including: body mass index (BMI), waist-to-hip ratio (WHR), waist-to-height ratio (WHtR), waist circumference (WC), and BIA. Also presented are current circumstances supporting the evaluating procedures applied in this study over other approaches (Gaya et al., 2017).

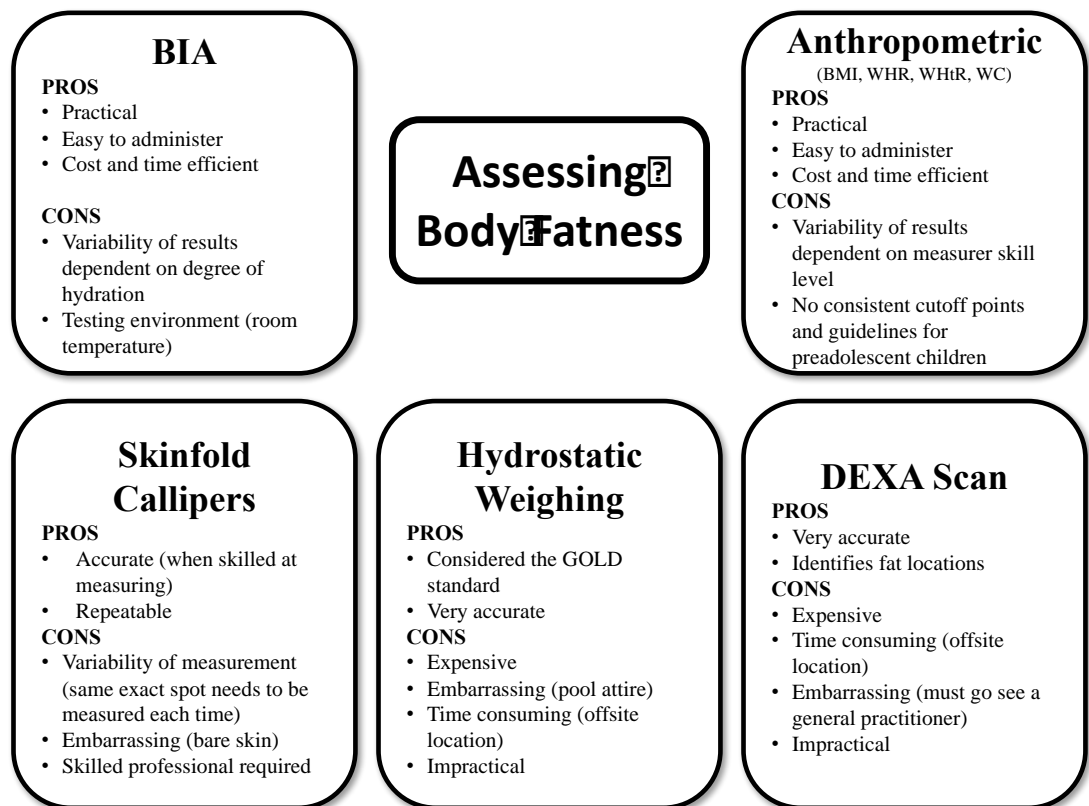


Figure 5. Body fatness assessments.

Anthropometry refers to the study of human body measurements. Thus, anthropometric indicators assess the size, shape, and composition of the human body and are commonly applied to examine healthiness in children (Gaya et al., 2017). Body mass index is calculated as the ratio of body weight in kilograms divided by height squared in metres (kg/m^2) and is the most common anthropometric indicator applied to determine total body composition in children (Ajala et al., 2017; Nambiar, Truby, Abbott, & Davies, 2009; Weber, Leonard, & Zemel, 2012). However, BMI has some limitations as the results are based on body weight and not differentiated by fat mass and muscle mass. Additionally, BMI does not account for fat distribution, whereas alternative anthropometric indicators (WHR, WHtR, WC) do (Javed et al., 2015; Sardinha et al., 2016). Furthermore, there are no consistent validated anthropometric cutoff points and guidelines (CDC, 2015a; International Obesity Task Force, 2015; WHO, 2011) for classifying body size and weight status in preadolescent children (MOH, 2015a). Still, there are advantages associated with anthropometric indicators including their being time efficient, noninvasive, and practical for large paediatric epidemiological studies. Studies carried out on Australian and New Zealand children (aged 5 to 17 years; $n = 3597$; $n = 172$) found that BMI was not an appropriate measurement of body fatness in children and adolescents (Nambiar et al., 2009; Rush, Puniani, Valencia, Davies, & Plank, 2003). However, one of the studies found anthropometric indicators (WHtR, WC) to be dependable gauges of body fatness because they account for body fat distribution (Nambiar et al., 2009). Moreover, various studies have revealed that BMI (Abarca-Gómez et al., 2017; O. Ali et al., 2014; N. S. O. Jensen, Camargo, & Bergamaschi, 2016), WHR (Roy & Sharma, 2016; Seidell, 2010), WHtR (Kahn, Imperatore, & Cheng, 2005; Savva et al., 2000), and WC (N. S. O. Jensen et al., 2016; Savva et al., 2000) have all been shown to be appropriate preliminary indicators of

body fatness in children (Sardinha et al., 2016). Nevertheless, uncertainty still remains as to the significance of BMI when compared to other anthropometric indicators (MOH, 2015a; Sardinha et al., 2016). Overall, BMI is an indicator for body size whereas the other anthropometric indicators (WHR, WHtR, WC) are gauges of body fat distribution (Nambiar, Hughes, & Davies, 2010), and collectively these anthropometric measurements are a good estimate of body fatness in paediatric research (O. Ali et al., 2014; Hara, Saitou, Iwata, Okada, & Harada, 2002; N. S. O. Jensen et al., 2016; Nambiar et al., 2010).

Bioelectrical impedance analysis is utilised to estimate body composition, particularly the amount of body fat in comparison to lean body mass (Rush et al., 2003). Initially, the instrument sends an electric signal through the body, which estimates the amount of fluid and tissue (Kushner, 1992; Kyle et al., 2004a). When compared, lean tissue is mostly water whereas fatty issue has minimal water; therefore, the flow resistance of the electric current is utilised to calculate body fat and lean body mass (Kyle et al., 2004a). A study review carried out for 10 years on Brazilian children (aged 7 to 10 years; $n = 47,726$) found that BIA is a reliable indicator of body fatness when more accurate techniques such as the DEXA scan are not feasible (N. S. O. Jensen et al., 2016). One major limitation in this review was that various BIA devices were utilised across the 27 different studies, therefore, there is no consistency in the results when attempting to determine which BIA device is more reliable when measuring body fat percentage in children (N. S. O. Jensen et al., 2016). An additional study review carried out on German, Austrian, and Swiss children and adolescents (aged 3 to 16 years; $n = 3327$) found that BIA and BMI were similar with regard to association and reliability of cardiometabolic risk factors in overweight children and adolescents; however, this same study also indicated that BIA is the more appropriate fatness indicator for predicting CVD risk in overweight or obese children into their

adolescent years as compared to BMI (Bohn et al., 2015). Conversely, some BIA research has shown inconsistencies with degree of hydration, warmth of testing environment, and reliability in severely obese individuals (Hendel, Gotfredsen, Højgaard, Andersen, & Hilsted, 1996; Kyle et al., 2004b) Ultimately, BIA is an effective assessment of body fatness as it differentiates between body fat and lean body mass. However, BIA does not account for the effects of height and body proportion (Weber et al., 2012), whereas other anthropometric indicators are gauges of body fat distribution (Rush et al., 2003). Therefore, BIA assessment used in conjunction with additional anthropometric measurements provides a more comprehensive estimation of body fatness in paediatric research.

Although implemented less frequently in large paediatric epidemiological studies, clinical methods such as skinfold callipers, DEXA scan, and hydrostatic weighing are the most accurate assessments of body fatness based on reliability and validity when compared to anthropometric indicators (N. S. O. Jensen et al., 2016). However, there are also limitations associated with these clinical methods including that they are costly, time-consuming (offsite location), potentially embarrassing, and require a skilled professional. Additionally, they lack paediatric reference values and are impractical for large paediatric epidemiological field-based studies (Javed et al., 2015). In comparison, field-based assessments like BMI, WHR, WHtR, WC and BIA have shown evidence of effectiveness as pre-screening estimates of body fatness (N. S. O. Jensen et al., 2016; Sardinha et al., 2016), and are practical for large paediatric epidemiological studies.

Applied body fatness assessments. In this study, body fatness was assessed utilising: body fat (%), fat mass (kg), fat mass index (FMI, fat mass/m²), WHR, and BMI (kg/m²). To measure body fat (%) and fat mass (kg), BIA was utilised. To calculate the anthropometric indices (BMI and WHR), height, weight, WC, and hip circumference were

measured using the WHO's 2007 WC and WHR report (WHO, 2011). To calculate BMI, age and sex-specific BMI z scores were calculated using the WHO growth guidelines (De Onis et al., 2007; WHO, 2018a) and BMI values (overweight and nonoverweight) were categorised using the International Obesity Task Force's (2015) sex and age-dependent cutoff points (Cole & Lobstein, 2012). For further information, see the data analysis sections in Chapters 4 and 5, sections 4.2 and 5.2, respectively.

2.2 Physical Fitness

2.2.1 Background

The terms physical fitness, physical exercise, and physical activity are frequently used interchangeably in the field of health and wellness (Caspersen, Powell, & Christenson, 1985). Physical activity is any bodily movement produced by skeletal muscles that leads to energy expenditure (Caspersen et al., 1985; Castillo Garzón, Ortega, & Ruiz, 2005). Physical exercise refers to physical activity or energetic movements that are planned, structured, efficient, and calculated in the sense that improving and/or sustaining physical fitness is the primary objective (Caspersen et al., 1985; Castillo Garzón et al., 2005). For further information on physical activity, see section 2.3.1 for background on physical [in]activity and sedentary behaviour. Physical fitness, on the other hand, is the ability to carry out tasks with vigour and alertness without undue fatigue to full capacity of physiological potential (Caspersen et al., 1985; Castillo Garzón et al., 2005). Physical fitness is reliant on cardiorespiratory and muscular fitness as both are significant components of physical fitness progression and sustainability (Ortega, Ruiz, Castillo, & Sjöström, 2008; Ruiz et al., 2016). Cardiorespiratory fitness is defined as the ability of the circulatory and respiratory systems to supply oxygen to exercising muscles during sustained physical activity (D. Lee, Artero, Sui, & Blair, 2010; Ruiz et al., 2016). Muscular

fitness (strength and endurance) is the ability of the muscle to carry out exertion against resistance and to continue to perform physical movement without fatiguing (Froberg & Andersen, 2005; Ortega et al., 2008; J. J. Smith et al., 2014).

Among adults, elevated levels of physical fitness and consistent engagement in physical activity have been shown to decrease the risk of developing cardiometabolic complications (LaMonte et al., 2005; Press, Freestone, & George, 2003; Schmidt, Magnussen, Rees, Dwyer, & Venn, 2016). In children, higher levels of physical fitness have been associated with healthier cardiometabolic profiles and labelled a significant marker of cardiometabolic functioning into adulthood (Cohen et al., 2011; Hamlin et al., 2014; Howe et al., 2016; Leong et al., 2015; Stratton et al., 2007). Significant improvements in the physiological functioning of the body in response to consistent exercise and improved fitness levels include: increased HDL-C and insulin sensitivity, and reduced body weight, blood pressure, inflammation, and LDL-C levels (Lavie et al., 2015; Myers, 2003; D. L. Smith & Fernhall, 2011). For a complete list of the physiological benefits of physical fitness see Table 3 (Lavie et al., 2015; Myers, 2013). Ultimately, consistent physical activity and physical exercise have been shown to improve and sustain physical fitness levels, which contributes to multiple beneficial outcomes for the cardiovascular system, metabolism, and overall functioning of the body (LaMonte et al., 2005; Press et al., 2003; Schmidt et al., 2016).

Physical exercise produces a greater need to supply oxygen to the active tissues with the aim of maintaining the production of adenosine triphosphate required to support continued muscle contraction (Lavie et al., 2015; Myers, 2003; D. L. Smith & Fernhall, 2011). Simultaneously, there is also the need to release built-up carbon dioxide created as a consequence of intensified cellular respiration (Lavie et al., 2015; Myers, 2003; D. L.

Smith & Fernhall, 2011). In the initial cardiac response, consistent physical exercise increases whole body oxygen consumption in accordance with exercise intensity (Press et al., 2003; D. L. Smith & Fernhall, 2011). This upsurge in oxygen intake subsequently improves myocardial contraction and its electrical consistency, along with increasing stroke volume at rest and during exercise; this leads to a higher maximal cardiac output and oxygen extraction (Lavie et al., 2015; Myers, 2003; Press et al., 2003; D. L. Smith & Fernhall, 2011). These outcomes lead to significant improvements in the physiological functioning of the heart, which is evident both in a lower resting heart rate and at any given level of submaximal cardiac output (Lavie et al., 2015; Press et al., 2003).

Table 3

Physiological Benefits of Physical Fitness

Decreases/Reductions	Increases/Improvements
<ul style="list-style-type: none"> • Reduced blood pressure • Decreased myocardial oxygen demands • Reduced visceral adiposity • Reduced blood and plasma viscosity • Reduced systemic inflammation • Reduction in LDL-C • Reduction in TC • Reduction in glucose levels 	<ul style="list-style-type: none"> • Increased insulin sensitivity • Increased exercise tolerance • Improved heart rate variability • Improved endothelial function • Increased capillary density • Increased mitochondrial density • Improved sleep • Increased HDL-C • Maintenance of lean mass

Regular cardiovascular workouts increase the HR; therefore, force of contraction increases while exercising, which enhances the heart’s ability to supply oxygen-rich blood to the muscles (D. L. Smith & Fernhall, 2011). Cardiac output is a product of stroke volume and HR, and stroke volume increases during exercise, which increases the rate of circulation (Lavie et al., 2015; D. L. Smith & Fernhall, 2011). In the course of exercising,

there is a redistribution of cardiac output with a substantial percentage of output directed to the skin and skeletal muscles (80%) compared with being at rest (20%; Lavie et al., 2015; D. L. Smith & Fernhall, 2011). The percentage of cardiac output supplying the heart is the same; however, the overall amount of blood transported to the heart is greater during exercise compared to being at rest (Lavie et al., 2015; Myers, 2003; Press et al., 2003; D. L. Smith & Fernhall, 2011). As a result, during exercise, blood flow increases to the tissues that are extremely active and decreases to the tissues that are less active (e.g., visceral organs; Lavie et al., 2015; D. L. Smith & Fernhall, 2011). Consequently, this increased blood flow improves shear stress, and enhanced shear stress leads to increased nitric oxide production and bioavailability (Di Francescomarino, Sciartilli, Di Valerio, Di Baldassarre, & Gallina, 2009; Sherman, 2000). Additionally, regular physical activity and exercise lowers inflammatory factors such as plasma fibrinogen concentrations, c-reactive proteins, and white cell count (Di Francescomarino et al., 2009; Press et al., 2003; Sherman, 2000). For further information on the functioning of the endothelium and inflammatory responses, see section 2.0.1 for background on cardiometabolic health and section 2.1.1 on body fatness.

Various metabolic adaptations occur in relation to exercise and post-exercise recovery, such as stimulation of lipid oxidation (Froberg & Andersen, 2005; Press et al., 2003). Additionally, alterations in the transport of blood lipids include a higher ratio of HDL-C to LDL-C transferred and increased lipoprotein lipase activity (Froberg & Andersen, 2005; Press et al., 2003). The increase of this pancreatic enzyme enhances the usage of circulating triacylglycerol as energy, which increases the clearing of unwanted circulating lipids even at rest (Froberg & Andersen, 2005; Press et al., 2003). Furthermore, the activation of this enzyme also speeds up the conversion of the very-low-density protein

to HDL-C (Froberg & Andersen, 2005; Press et al., 2003). For further information on lipoprotein activity, see section 2.4.1 for background on nutrition.

Lastly, consistent exercise enhances sensitivity of the liver, skeletal muscle, and adipose tissue to the actions of insulin (Froberg & Andersen, 2005; Press et al., 2003). Subsequently, there are reductions in fasting insulin levels and the insulin response to glucose, which are associated with increases in the disposal rate for glucose (Froberg & Andersen, 2005; Press et al., 2003). For further information on insulin and glucose, see section 2.1.1 for background on body fatness and section 2.4.1 for background on nutrition.

Physical fitness levels that do not meet the Cooper Institute (2014) FitnessGram[®] cutoff points for children (Howe et al., 2016; Zhu et al., 2010), along with insufficient daily physical activity per MOH (2015c) guidelines, sedentary behaviour (Cohen et al., 2011; Glynn, Emmett, & Rogers, 2005; Hjorth et al., 2013), unhealthy diet (Emmett & Jones, 2015; Harrex et al., 2017; Howe et al., 2016), inadequate sleep (X. Chen, Beydoun, & Wang, 2008; Hjorth et al., 2013; Skidmore et al., 2013), and excessive body fatness (Bridger, 2009; Trandafir et al., 2017) have all been linked to cardiometabolic risk factors (Cohen et al., 2011; Hjorth et al., 2013; Stratton et al., 2007). However, uncertainty exists as to whether physical fitness or another lifestyle factor associates more strongly with cardiometabolic risk factors in preadolescent children.

2.2.2 Evaluating Physical Fitness

In preadolescent children, physical fitness (cardiorespiratory and muscular) is commonly assessed utilising $\dot{V}O_{2\max}$ and muscular strength measuring methods. Frequently, $\dot{V}O_{2\max}$ is estimated utilising the maximal multistage 20-metre shuttle run test (20-MST), whereas muscular strength is commonly assessed utilising a handgrip strength test. The following paragraphs elaborate on the indicated physical fitness assessment methods in

addition to presenting evidence supporting the evaluating procedures applied in this study over other approaches.

The most widely used indicator of cardiorespiratory fitness is maximal oxygen uptake ($\dot{V}O_{2\max}$; Noonan & Dean, 2000). $\dot{V}O_{2\max}$ can be objectively assessed in a laboratory setting or by utilising field-based methods such as the 20-MST (Hamlin et al., 2014; Ramírez-Vélez, Silva-Moreno, et al., 2017). In a laboratory setting, $\dot{V}O_{2\max}$ is assessed utilising a progressive run test or a cycle test to exhaustion (Hamlin et al., 2014; Ramírez-Vélez, Silva-Moreno, et al., 2017). However, in preadolescent children there are several limitations associated with assessing $\dot{V}O_{2\max}$ in a laboratory setting including cost, time, requirement for sophisticated equipment and trained technicians, and impracticality for large paediatric epidemiological studies. Alternatively, field-based tests like the 20-MST can be implemented to measure cardiorespiratory fitness (Hamlin et al., 2014).

In children, the 20-MST is one of the most common field-based tests utilised to assess cardiorespiratory fitness, specifically $\dot{V}O_{2\max}$ (Hamlin et al., 2014; Melo et al., 2011). The 20-MST consists of participants running between two lines set 20-metres apart with a beginning speed of 8.5 km/h⁻¹, which increases by 0.5 km/h⁻¹ at every completed level (Hamlin et al., 2014; Howe et al., 2016; Léger & Lambert, 1982). In accordance with the Cooper Institute (2014) FitnessGram[®] cutoff points, a “healthy cardiorespiratory fitness zone” is reported if girls achieve a $\dot{V}O_{2\max}$ equal to or greater than 39 and boys achieve a $\dot{V}O_{2\max}$ equal to or greater than 42 (Howe et al., 2016; Zhu et al., 2010). A $\dot{V}O_{2\max}$ below those cutoff points is categorised as “needs improvement fitness zone” for both sexes (Cooper Institute, 2014; Howe et al., 2016; Zhu et al., 2010). A study carried out on New Zealand children (aged 8 to 13 years; $n = 53$) determined 20-MST is a consistent field-

based assessment of cardiorespiratory fitness (Hamlin et al., 2014). The most significant limitation associated with this study was all the participants were from one school and similar in age, body size, and socioeconomic status, therefore only representing a limited sample from the population of New Zealand children. Nonetheless, further studies have confirmed the 20-MST as a valid and reliable field-based test for cardiorespiratory fitness in children (Hamlin et al., 2014; Howe et al., 2016; Léger & Lambert, 1982), and the most commonly utilised cardiorespiratory fitness assessment method in large paediatric epidemiological studies (Ortega et al., 2008).

A handgrip dynamometer is utilised to measure handgrip strength, which is an indicator of muscular fitness (Leong et al., 2015). The handgrip strength test involves being seated with shoulders adducted and neutrally rotated, elbow flexed to 90 degrees, and wrist in a neutral position. Participants squeeze the dynamometer handle as hard as possible for three or more seconds, providing a score for each hand. In accordance with the Camry EH101 manual, participants' handgrip strength is characterised as weak, normal, or strong based on the sex-specific cutoff points. A study carried out on Columbian children and adolescents (aged 10 to 17 years; $n = 1950$) utilising a handgrip strength dynamometer found that muscular strength had a direct correlation with cardiometabolic risk (Ramírez-Vélez, Peña-Ibagon, et al., 2017) However, in this study, the majority of the participants were teenagers. Teenagers will have larger hands as compared to preadolescents, and as hand size strongly influences handgrip strength results (Ramírez-Vélez, Peña-Ibagon, et al., 2017), the results from this study are not comparable to a study where the participants are preadolescent children aged 8 to 10 years. Another study carried out on European adolescents (aged 12 to 17 years; $n = 1053$) utilising a handgrip strength dynamometer suggested that enhancing muscular strength could reduce cardiometabolic risk in children;

however, hand size, sex, ethnicity, and maturity must be accounted for when analysing the beneficial effects muscular strength has on reducing cardiometabolic risk in children of different ages (Jiménez-Pavón et al., 2011).

Applied physical fitness assessments. The 20-MST and handgrip strength tests have been shown to be reliable indicators of cardiorespiratory fitness and muscular fitness. Additionally, the 20-MST and the handgrip strength test are easy to administer, time efficient, noninvasive, popular in school settings, and practical for large paediatric epidemiological studies. Therefore, in this study, cardiorespiratory fitness was measured utilising the 20-MST and muscular fitness was measured utilising the handgrip strength test. For further information, see the data analysis sections in Chapters 4 and 5, sections 4.2 and 5.2, respectively.

2.3 Physical [In]activity and Sedentary Behaviour

2.3.1 Background

More than 80% of the world's adolescent population is not sufficiently active (WHO, 2017d, 2018b). Furthermore, 1.6 million deaths annually can be attributed to insufficient physical activity (WHO, 2017d, 2018b). Over the last four decades, accumulated evidence indicates physical inactivity and/or sedentary behaviour in individuals considerably increases the risk of developing coronary heart disease when compared to individuals who have lived an active lifestyle (Da Silva et al., 2018; Ding et al., 2016; I. M. Lee et al., 2012; Liu & Manson, 2001; Pate et al., 1995; Press et al., 2003; Warren et al., 2010). Physical inactivity is one of the major modifiable risk factors of coronary heart disease (WHO, 2017d, 2018b).

In the past, the terms physical inactivity and sedentary behaviour have been used interchangeably. However, being physically inactive signifies not meeting daily physical

activity guidelines, whereas being sedentary refers to very low energy expenditure behaviour for an extended period of time (González, Fuentes, & Márquez, 2017; Van der Ploeg & Hillsdon, 2017). Thus, physical inactivity and sedentary behaviour are dissimilar concepts with different definitions, each with its specific health hazards, and therefore need to be addressed independently (González et al., 2017; Van der Ploeg & Hillsdon, 2017).

Physical [in]activity physiological pathways to CVD. The MOH (2015c) physical activity guidelines for children and young people aged 5 to 17 years state that an accumulation of at least 1 hour a day of moderate-to-vigorous physical activity, with incorporation of vigorous-intensity activities and muscle and bone strengthening activities at least 3 days a week, is the minimal requirement. Physical activity is characterised by the United States' National Institutes of Health (2016) as any bodily movement produced by skeletal muscle resulting in an increased energy expenditure, including aerobic activities (e.g., bicycling and dancing), muscle strengthening activities (e.g., gymnastics and push-ups), bone strengthening activities (e.g., jumping rope and skipping), and stretching (e.g., daily stretching and yoga; Caspersen et al., 1985; González et al., 2017). To determine the intensity of physical activity the metabolic equivalent method can be applied. Therefore, physical activity intensities are categorised as light-intensity (< 3 metabolic equivalents), moderate-intensity (3-6 metabolic equivalents), and vigorous-intensity (> 6 metabolic equivalents; Caspersen et al., 1985; González et al., 2017; Sheldrick, Tyler, Mackintosh, & Stratton, 2018).

In children, moderate-to-vigorous physical activity has been shown to have a positive effect on multiple systems (Faigenbaum & Myer, 2012; Lieberman, 2013; Stoner, Matheson, Hamlin, & Skidmore, 2016). This includes the neuromuscular (promoting skeletal muscle fibre growth), musculoskeletal (straightening and thickening bones), and

cardiovascular systems (the arterial system becoming more elastic; Faigenbaum & Myer, 2012; Lieberman, 2013; Stoner, Matheson, et al., 2016). Moreover, adequate levels of physical activity have been shown to have a positive association with cognition, which is linked to motor skills and locomotor movements such as walking, running, throwing, lifting, skipping, etc. (Chaddock-Heyman et al., 2013; Sibley & Etnier, 2003). Cognitive growth, along with motor skill and movement development, sets the foundation for being active and exercising to become fit and sustain a healthy fitness level into adulthood (Chaddock-Heyman et al., 2013; Sibley & Etnier, 2003). In adults, moderate-to-vigorous physical activity has been shown to improve physical fitness levels and has a direct effect on peak physical fitness levels (Booth, Roberts, & Laye, 2012; Nocon et al., 2008; Press et al., 2003). Furthermore, sustained high levels of physical fitness have multiple positive effects on cardiometabolic conditions such as hypertension, hyperglycaemia, hyperlipidaemia, and obesity (Booth et al., 2012; Nocon et al., 2008; Press et al., 2003). For further information on physiological benefits of physical fitness, see section 2.2.1 for background on physical fitness. Overall, moderate-to-vigorous physical activity is associated with multiple progressive cardiometabolic outcomes (González et al., 2017; Van der Ploeg & Hillsdon, 2017). In contrast, light-intensity physical activity (<3 metabolic equivalents) has not been shown to improve physical fitness levels (González et al., 2017; Van der Ploeg & Hillsdon, 2017).

Physical inactivity is defined as achieving less than the recommended amount of moderate and vigorous activity on a daily basis per physical activity guidelines (MOH, 2015c). There are detrimental consequences for not meeting the daily physical activity recommendations. In children, not meeting the daily moderate-to-vigorous activity requirements could result in detrimental outcomes for biological development of cognitive

functioning and the neuromuscular, musculoskeletal, and cardiovascular systems (Faigenbaum & Myer, 2012; Lieberman, 2013; Sibley & Etnier, 2003). In adults, physical inactivity has been shown to have a direct correlation with type 2 diabetes and obesity (González et al., 2017; Van der Ploeg & Hillsdon, 2017). Obesity also has been shown to have positive correlations with additional cardiometabolic complications such as hypertension, hyperglycaemia, and hyperlipidaemia (Booth et al., 2012; Nocon et al., 2008; Press et al., 2003). For further information on physiological effects of obesity, see section 2.1.1 for background on body fatness. Clearly, physical inactivity is a determinant for overall health and wellness (González et al., 2017; Van der Ploeg & Hillsdon, 2017). Furthermore, recent evidence has shown that both physical inactivity and sedentary behaviour contribute to the global burden of chronic diseases (González et al., 2017; Van der Ploeg & Hillsdon, 2017). Therefore, although physical inactivity and sedentary behaviour are dissimilar concepts, both need to be addressed; however, this should be done independent of the other (González et al., 2017; Van der Ploeg & Hillsdon, 2017).

Sedentary behaviour physiological pathways to CVD. Countries like New Zealand and Canada have characterised sedentary behaviour as a major risk factor of CVD in addition to developing and implementing sedentary lifestyle recommendations (Canadian Society for Exercise Physiology [CSEP], n.d.; MOH 2015a, 2015c, 2017; Tremblay et al., 2011). The recommendations state that school-aged and young people (aged 5 to 17 years) should not have more than 2 hours per day of recreational screen time and should limit sitting for extended periods (CSEP, n.d.; MOH 2015a, 2015c, 2017; Tremblay et al., 2011). Sedentary behaviour is characterised as any waking behaviour characterised by an energy expenditure below or equal to 1.5 metabolic equivalents for long periods of time including: sitting, lying, reading, doing homework, playing video

games, or operating an electronic device while stationary (MOH, 2015a; Van der Ploeg & Hillsdon, 2017). At present in children there is minimal research on the impact of prolonged sitting, thus the physiological outcomes that accompany sustained sedentary behaviour are not completely understood (V. Armstrong, 2017; McManus et al., 2015).

In adults, previous research has indicated that excessive sedentary behaviour has serious public health implications because of the destructive effect prolonged sitting has on vascular function and metabolism (V. Armstrong, 2017; Leitzmann, Jochem, & Schmid, 2017). While the mechanisms are not well understood, one direct mechanism potentially related to prolonged sitting is seated posture (V. Armstrong, 2017; Leitzmann et al., 2017). A seated posture creates bends in major blood vessels in the legs, disturbing and decreasing blood flow patterns, which creates unique changes to leg haemodynamics (Leitzmann et al., 2017; McManus et al., 2015; Restaino, Holwerda, Credeur, Fadel, & Padilla, 2015). This decrease in lower limb blood flow increases pooling of blood in the calf, augments mean arterial pressure, deforms arterial segments, and decreases shear stress (Thosar, Johnson, Johnston, & Wallace, 2012; Tremblay, Colley, Saunders, Healy, & Owen, 2010). Consequently, low mean shear stress in the lower extremities can result in increased oxidative stress and endothelial dysfunction (Thosar et al., 2012; Tremblay et al., 2010). For further information on endothelial dysfunction see section 2.0.1 for background on cardiometabolic health.

An additional element to consider is the detrimental outcome sedentarism has on the metabolism. Sedentary behaviour has been shown to affect metabolic functioning by increasing triacylglycerol levels and decreasing HDL-C and insulin sensitivity levels (Demiot et al., 2007; Hamburg et al., 2007; Tremblay et al., 2010). For further information on lipoprotein activity, see section 2.4.1.

Summary. In adults, mounting epidemiological evidence connects sedentary behaviour and physical inactivity to various health and wellness consequences such as cardiovascular and metabolic disease, cancer, and psychosocial problems (Leitzmann et al., 2017; McManus et al., 2015; Tremblay et al., 2010). Relatedly, there is a need for further paediatric research examining the long-term effects of these factors on the physiological mechanisms that trigger cardiometabolic complications (Leitzmann et al., 2017; McManus et al., 2015; Tremblay et al., 2010). As stated in section 2.0.1, cardiometabolic risk factors tend to cluster, and consequently, physical inactivity and sedentary behaviour have been identified as determinants of deteriorating health in children (WHO, 2017a, 2017d). However, uncertainty exists as to whether sedentary behaviour, physical inactivity, or another lifestyle factor associates more strongly with cardiometabolic risk factors in preadolescent children.

2.3.2 Evaluating Physical [In]activity and Sedentary Behaviour

In preadolescent children, physical [in]activity and sedentary behaviour are commonly assessed utilising objective measuring methods (pedometers, accelerometers, and heart rate monitors (HRM), and self-report measuring methods (direct observation, record/diary, survey/questionnaires). In accordance with the New Zealand MOH physical activity guidelines, children aged 5 to 17 years should participate in at least 60 minutes of moderate to vigorous physical activity every day and minimise recreational screen time to no more than 2 hours daily (MOH, 2015a, 2015c, 2017). The following paragraphs elaborate on the mentioned physical [in]activity and sedentary behaviour assessment methods in addition to presenting evidence supporting the evaluating procedures applied in this study over other approaches. For example, although numerous methods to objectively assess physical [in]activity and sedentary behaviour exist, I will concentrate on pedometers,

accelerometers, and HRM as these are the methods most frequently utilised in studies involving children (Loprinzi & Cardinal, 2011; Sallis, 2010).

Objective measures. Pedometers measure distance by counting steps as people walk over a period of time (Hands & Larkin, 2006; Loprinzi & Cardinal, 2011; Sallis, 2010). A study carried out on children (aged 5 to 11 years, $n = 20$) found pedometers to be a reliable indicator of step count when compared to direct observation (Beets, Patton, & Edwards, 2005). However, in this study, there were only 20 participants and the data were collected on a treadmill in a laboratory setting in a controlled environment. Therefore, the data are not comparable to data collected in a field-based study, in an uncontrolled environment, with 392 participants. Another study, carried out on American children (aged 7 to 12 years, $n = 31$), found that pedometers are an accurate indicator of step count when compared to accelerometers (Ramirez-Marrero, Smith, Kirby, Leenders, & Sherman, 2002). This study also reported pedometers to be reliable, yet used solely treadmills to collect data in a laboratory setting. Additionally, this study only examined children of one race (African American). Therefore, the results from this study are not comparable to a field-based study with 392 participants from diverse backgrounds participating in various activities throughout the day (Ramirez-Marrero et al., 2002). Nonetheless, some research suggests that pedometers are the most advanced measure of activeness in younger children when measuring free play activities (Dishman, Washburn, & Schoeller, 2001; Sallis, 2010). Thus, additional research is essential to determine if pedometers are just as effective in a laboratory setting (treadmill) as compared to an uncontrolled environment (free play). Some advantages associated with pedometers include that they are inexpensive, simple to use, and child-friendly (Hands & Larkin, 2006; Loprinzi & Cardinal, 2011; Sallis, 2010). However, there are numerous limitations associated with pedometers including: assessment

accuracy when bicycling or participating in water activities, inability to gauge the intensity of movements, and difficulties determining whether low scores are a result of sedentariness or the device being removed (Hands & Larkin, 2006; Loprinzi & Cardinal, 2011; Sallis, 2010).

Accelerometers provide an objective assessment of movement patterns or rhythms and consist of a small portable device worn on the wrist, ankle, or trunk that identifies and records movement and rest cycles (Hands & Larkin, 2006; Loprinzi & Cardinal, 2011; Sallis, 2010). A study carried out on American children (aged 3 to 6 years, $n = 419$) discovered that accelerometers provide an accurate assessment of movements and sedentary behaviour (Bornstein et al., 2011). However, this study review analysed data from five different studies that each used different devices and dissimilar cutoff points. Moreover, cutoff points cannot be directly compared even when the outcome units are similar (Bornstein et al., 2011). Therefore, future research should attempt to validate accelerometer guidelines and cutoff points so comparisons can be made across studies, resulting in valid and reliable estimates of activity levels in children. Another study carried out on European children (aged 10 to 12 years, $n = 686$) found that accelerometers provided an accurate estimate of activity levels and sedentary time (Verloigne et al., 2012). This study found accelerometers to be accurate when estimating levels of activity and sedentary time. However, three different models of accelerometers were used and as mentioned, cutoff points from model to model cannot be directly compared. Furthermore, one of the accelerometer models used in this study review has been shown to be less suited for monitoring sedentary time and low physical activity levels, so the accuracy of those results is uncertain (Verloigne et al., 2012). Some advantages associated with accelerometers include that the device collects sedentary time data and interprets frequency, duration, and

intensity levels of activeness (Loprinzi & Cardinal, 2011; Rowlands & Eston, 2007; Sallis, 2010). Therefore, accelerometers have become the most accepted and validated physical [in]activity assessment method in adolescents (Hands & Larkin, 2006; Loprinzi & Cardinal, 2011; Sallis, 2010). However, there are some limitations associated with accelerometer assessment including: battery life, cost, susceptibility to damage, participant dependence (i.e., remembering to put it on), absence of universal validated cutoff points and guidelines for children (Bornstein et al., 2011), and inability to accurately assess bicycling or swimming (Hands & Larkin, 2006; Loprinzi & Cardinal, 2011; Sallis, 2010). Furthermore, while accelerometers have been validated in adolescents, the method still lacks reliable and validated instructions and guidelines for wearing, recording, and scoring in preadolescent children (García-Prieto et al., 2017; Hands & Larkin, 2006; Sallis, 2010).

Being physically active triggers an increase in HR; therefore, HRM can be utilised to estimate activeness and sedentariness (Sallis, 2010). Heart rate monitors present an objective estimation of the frequency, intensity, and duration of activeness by monitoring heart beats per minute (Loprinzi & Cardinal, 2011; Sallis, 2010). A study carried out on Spanish children (aged 8 to 10 years, $n = 32$) found that HRM provided more accurate estimates of energy expenditure (intensity) when compared to accelerometers (García-Prieto et al., 2017). Nonetheless, there is no reliable evidence regarding the validity of HRM and accelerometers for estimating the intensity of activities in uncontrolled environments, so results have to be considered with uncertainty (García-Prieto et al., 2017). A study carried out on English children (aged 11 years, $n = 5595$) also found inconsistencies in HRM when differentiating between high and low activity levels, which could pose a problem as most children spend a large percentage of their day sedentary or participating in light activity (Riddoch et al., 2007). Some of the mentioned studies also

found HRM to be susceptible to damage, dependent on participants remembering to put them on, and requiring a substantial amount of data to produce results (Loprinzi & Cardinal, 2011; Sallis, 2010). To the contrary, a few studies have found HRM to be capable of detecting movements in water and able to interpret frequency and duration of movements (Loprinzi & Cardinal, 2011; Sallis, 2010). As a result, HRM have been shown to be useful, but also have demonstrated limitations when tracking movement behaviours, especially intensity levels in children; therefore, further research on tracking movements in children is necessary to determine the effectiveness of these devices in paediatric research.

Subjective measures. Direct observation is when physical [in]activity and sedentary behaviour data are collected by an observer who examines the subjects in their traditional environment. These observations can be made during physical education classes, recess time, participation in sports or activities, or just playing in the park (Hands & Larkin, 2006; Sallis, 2010). A study review carried out on children and adolescents (aged 3 to 18 years, $n = 13$ studies) worldwide found direct observation to be a reliable measurement of sedentary behaviour, specifically for type and duration when compared to self-report and accelerometers; however, when monitoring energy expenditure, accelerometers achieved more accurate and valid results (Lubans et al., 2011). Another study review carried out on children and adolescents (aged 4 to 20 years, $n = 25$ studies) globally, found direct observation to provide the most suitable estimate of physical [in]activity and sedentary behaviour when compared to self-report, accelerometers, and pedometers (McNamara, Hudson, & Taylor, 2010). However, as mentioned, there is no reliable evidence regarding the validity of these approaches for estimating the intensity of activities or sedentariness, thus the results are questionable (García-Prieto et al., 2017; Lubans et al., 2011).

Advantages associated with direct observation include: ability to record numerous

dimensions of physical activities, production of high quality data, adjustable scoring of results, and monitoring of participants in their natural setting (Hands & Larkin, 2006; Sallis, 2010). Limitations associated with direct observation include: cost (e.g., research staff), the need for training observers, exposure to human error, difficulties managing and scoring data (Lubans et al., 2011; Sallis, 2010), and impracticality for a large paediatric epidemiological study like this study.

Self-report measures include documents such as a record/diary or a questionnaire. Self-reports estimate physical [in]activity and sedentary behaviour by participants recalling their daily physical [in]activity and sedentary behaviour in recent days or weeks utilising daily logs, diaries, surveys, or questionnaires (Sallis, 2010). Two previously mentioned review studies also determined that self-report assessment method is the least reliable measurement of physical [in]activity and sedentary behaviour when estimated against direct observation, accelerometers, and pedometers (Lubans et al., 2011; McNamara et al., 2010). However, the self-report measures did provide a well-rounded assessment of sedentary behaviour, measuring all aspects of inactiveness (type, duration, and context), although the alternative measuring methods provided higher quality data overall (Lubans et al., 2011). Another study carried out on children (aged 9 to 12 years, $n = 22$) found self-reports to be reliable and consistent when monitoring physical activity frequency and time when compared to accelerometers; however, the same study found self-reports to be inconsistent when monitoring physical activity intensity levels (Van Hoya, Nicaise, & Sarrazin, 2014). Additional advantages associated with self-reports include that they are noninvasive, inexpensive, simple to use, and able to evaluate large sample sizes (Loprinzi & Cardinal, 2011; Sallis, 2010). Collectively, there are advantages and disadvantages associated with using self-report measures to collect physical [in]activity and sedentary

behaviour data in children. As a final point, for a large paediatric study like this one, it would have been impractical to directly observe 392 participants or to purchase movement devices for each participant, thus self-report measures were the best option.

Applied physical [in]activity and sedentary behaviour assessment. In this study, physical [in]activity and sedentary behaviour were estimated using the Youth Physical Activity Questionnaire (YPAQ; see Appendix A). To determine how many minutes a day each participant was active and sedentary, participants and their caregiver were asked to jointly complete the 47-item YPAQ. The YPAQ assessed the frequency, duration, and type of physical activities and sedentary behaviour the participant took part in 7 days prior to data collection (Brooke, Corder, Griffin, Ekelund, & Van Sluijs, 2013; Corder et al., 2009). For further details, see the data analysis sections in Chapters 4 and 5, sections 4.2 and 5.2, respectively.

2.4 Nutrition

2.4.1 Background

According to the New Zealand MOH (2015a) food and nutrition guidelines, a healthy diet consists of well-proportioned meals and includes a variety of nutrients from the four food groups categories: fruit and vegetables, grains, dairy products, and protein. Listed in Figure 6 and Table 4 are the functions and common dietary sources for each macro and micronutrient. Macronutrients consist of carbohydrates, proteins, and lipids. During digestion, macronutrients are broken down in the gut by digestive enzymes (aided by gastric acid) and separated into subcomponent parts (Kkeveetil, Thomas, & Chander, 2016; Slicker & Vermilyea, 2009; Von Haehling, Doehner, & Anker, 2007). Carbohydrates are broken down into polysaccharides, oligosaccharides, disaccharides, and monosaccharides by amylase and disaccharidase enzymes; proteins are broken down into short chains of

amino acids (peptides) or individual amino acids by protease enzymes; lipids are broken down into glycerol and fatty acids by lipase enzymes (Gropper & Smith, 2012; Kkeveetil et al., 2016; Slicker & Vermilyea, 2009; Von Haehling et al., 2007). The subcomponent parts are absorbed and then stored across the gut wall and taken up into the body's cells to be used (metabolised), or the subcomponent parts attach to proteins in the blood or lymphatic system and are distributed around the body to where required (Gropper & Smith, 2012; Kkeveetil et al., 2016; Tang, Kitai, & Hazen, 2017). Micronutrients consist of vitamins and minerals that are essential for energy metabolism, bone growth, vision, cognitive development, and the functioning of the digestive, immune, and cardiovascular systems (Kkeveetil et al., 2016; Stoner, Matheson, et al., 2016; WHO, 2015).

Carbohydrate. The primary function of macronutrient carbohydrate is to supply energy to all the cells in the body. Energy is released during the breakdown of carbohydrates by the metabolism of glucose (B. I. Campbell & Spano, 2011; Howard & Wylie-Rosett, 2002; Kenney et al., 2015). Carbohydrates are compounds categorised as either complex or simple (see Figure 6). Complex carbohydrates (oligosaccharides and polysaccharides) are chains of monosaccharide units linked together by covalent bonds (B. I. Campbell & Spano, 2011; Gropper & Smith, 2012; Howard & Wylie-Rosett, 2002). They provide the body with vitamins, minerals, and fibre, which are essential to everyday functioning and health (B. I. Campbell & Spano, 2011; Gropper & Smith, 2012; Howard & Wylie-Rosett, 2002). Disaccharides and monosaccharides are simple carbohydrates. Disaccharides consist of two monosaccharide units joined by a covalent bond (Griel, Ruder, & Kris-Etherton, 2006; Gropper & Smith, 2012; Saris et al., 2000). Monosaccharides, however, cannot be reduced to yet smaller components, thus are labelled the simplest form of carbohydrate (B. I. Campbell & Spano, 2011; Gropper & Smith, 2012;

Howard & Wylie-Rosett, 2002). Both forms of simple carbohydrates can be found in foods that provide some nutritional value such as fruits, vegetables, and dairy products (Bessesen, 2001; Griel et al., 2006; Saris et al., 2000). However, disaccharides and monosaccharides are also found in processed and refined sugars such as lollies, granulated sugars, syrups, and fizzy drinks, which are considered empty calorie foods as they provide minimal or no nutritional value (Bessesen, 2001). Both simple and complex carbohydrates are broken down to glucose; however rapidity of digestion and nutritional value are what sets them apart (B. I. Campbell & Spano, 2011; Howard & Wylie-Rosett, 2002; Kenney et al., 2015). Complex carbohydrates contain fibre, some of which slows the movement of intestinal contents and possibly the absorption of some nutrients such as glucose (B. I. Campbell & Spano, 2011; Howard & Wylie-Rosett, 2002; Saris et al., 2000). This in turn elicits a feeling of satiety and from that, the body is not overloaded, which could help stabilise blood sugar levels (B. I. Campbell & Spano, 2011; Howard & Wylie-Rosett, 2002; Saris et al., 2000). Contrariwise, simple carbohydrates are digested quickly, which causes a spike in blood glucose levels (Hotamisligil, 2003; Saltiel & Kahn, 2001; Vaulont, Vasseur-Cognet, & Kahn, 2000). If this spike becomes a chronic occurrence it can lead to impaired glucose tolerance, causing adipose and muscle tissue to become less responsive or resistant to insulin, which then triggers the inflammatory process (Hotamisligil, 2003; Saltiel & Kahn, 2001; Vaulont et al., 2000).

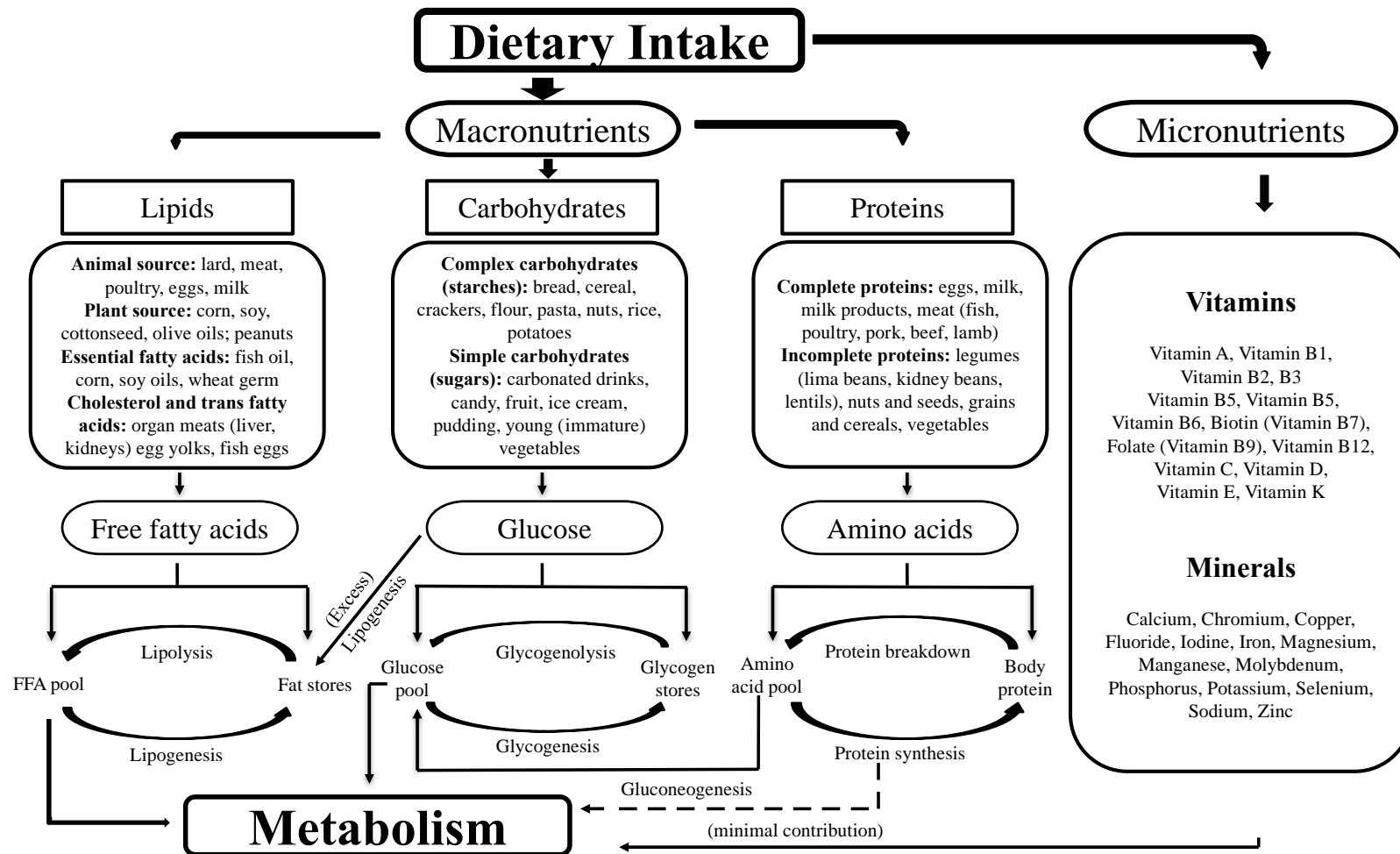


Figure 6. Dietary intake. Adapted from Physiology of Sport and Exercise by W. L. Kenney, J. Wilmore, & D. Costill, 2015, p. 51. Copyright 2012 by Human Kinetics; Human Anatomy and Physiology (8th ed.) by E. N. Marieb, & K. Hoehn, 2009, p. XX. Copyright 2010 Pearson Education.

Table 4

Function of Micronutrients

Micronutrients	Functions	Common dietary sources
Vitamin A ○ <i>Retinol</i> ○ <i>B-Carotene</i>	○ Essential for vision and immune function ○ Needed for cell growth and development	<i>Retinol</i> : Beef liver, eggs, butter, fortified milk <i>β-Carotene</i> : sweet potatoes, pumpkins, carrots, cantaloupes, mangoes, spinach, broccoli, kale, collards
Vitamin B1 ○ <i>Thiamine</i>	○ Helps release energy from carbohydrates and protein	Fortified cereal, bread, pork, enriched white rice, brown rice, peas, macadamia nuts, beans, lentils, cantaloupes
Vitamin B2 ○ <i>Riboflavin</i>	○ Helps release energy from fat, carbohydrates, and protein ○ Helps several antioxidant enzymes	Milk, fortified cereal, bread, eggs, almonds, clams, spinach, chicken, beef, asparagus, salmon, cheese
Vitamin B3 ○ <i>Niacin</i>	○ Helps release energy from fat, carbohydrates, and protein	Fortified cereal, bread, fish, light-meat chicken and turkey, beef, mushrooms, peanuts, avocados
Vitamin B5 ○ <i>Pantothenic Acid</i>	○ Helps release energy from fat, carbohydrates, and protein ○ Helps fat metabolism, and hormone production	Avocados, yogurt, chicken, sweet potatoes, milk, lentils, eggs, peas, mushrooms, fish, broccoli
Vitamin B6	○ Supports variety of metabolic reactions ○ Helps haemoglobin, DNA production	Turkey, chicken, fortified cereal, bread, potatoes (with skin), fish, bananas, hazelnuts, walnuts, pork, beans
Vitamin B7 ○ <i>Biotin</i>	○ Helps release energy from fat, carbohydrates, protein ○ Helps in glucose production	Beef liver, eggs, salmon, avocados, yeast, whole-wheat bread, pork, cheese
Vitamin B9 ○ <i>Folate</i> ○ <i>Folic Acid</i> :	○ Helps red blood cell production ○ Prevents neural tube defects	<i>Folate</i> : beans, lentils, spinach, peas, corn, chicken <i>Folic Acid</i> : enriched rice or products made with enriched flours, such as cereal, pasta, or bread
Vitamin B12	○ Helps the release of energy from fat and protein ○ Helps haemoglobin and red blood cell production	Clams, mussels, crab meat, salmon, beef, rockfish, milk, cheese, eggs, chicken, turkey, fortified cereal
Vitamin C	○ Helps collagen and connective tissue formation ○ Augments functional activity of immune cells ○ Antioxidant in blood and cells	Chili peppers, sweet peppers, guavas, kiwifruits, strawberries, oranges, kale, spinach, broccoli, grapefruit, potatoes, tomatoes
Vitamin D	○ Maintains calcium and phosphorus balance ○ Promotes bone health and immune function ○ Influences cell growth and development	Fish, eggs, fortified soy milk, fortified orange juice, fortified milk, fortified cereal
Vitamin E	○ Supports normal nerve function ○ Augments functional activity of immune cells ○ Antioxidant in cell membranes	Olive oil, safflower oil, sunflower oil, almonds, hazelnuts, peanuts, spinach, carrots, avocados
Vitamin K	○ Helps with blood clotting ○ Modifies certain proteins to allow for calcium binding	Kale, chard, parsley, broccoli, spinach, watercress, leaf lettuce, cashews, peas, soybean oil, canola oil, olive oil, mayonnaise

Calcium	<ul style="list-style-type: none"> ○ Structural component of bones and teeth ○ Helps nerve transmission, muscle contraction, blood vessel constriction, dilation, and reduction of blood pressure 	Milk, yogurt, cheese, tofu (calcium-set), fortified beverages, fortified cereal, rhubarb, spinach, almonds, white beans, bok choy, kale, pinto beans, red beans, broccoli
Chromium	<ul style="list-style-type: none"> ○ Helps insulin action 	Broccoli, grape juice, sweet potatoes, orange juice, beef, turkey, chicken
Copper	<ul style="list-style-type: none"> ○ Helps in energy production and iron utilization ○ Maintains integrity of connective tissue ○ Helps antioxidant enzymes 	Beef liver, oysters, crab meat, clams, sunflower seeds, kale, cashews, lentils, beans, mushrooms, cocoa powder, raisins, peanut butter
Fluoride	<ul style="list-style-type: none"> ○ Structural component of bones and teeth 	Fluoridated water, crab meat, beans, black tea, raisins
Iodine	<ul style="list-style-type: none"> ○ Component of thyroid hormones 	Iodized salt, milk, shrimp, turkey, navy beans, tuna, eggs
Iron	<ul style="list-style-type: none"> ○ Needed for synthesis of haemoglobin ○ Helps antioxidant enzymes ○ Required for synthesis of DNA, amino acids, collagen, neurotransmitters, and certain hormones ○ Critical for normal immune function 	Beef, fortified cereal, beans, oysters, molasses, lentils, firm tofu, kidney beans, cashews, spinach, potatoes (with skin), shrimp, light tuna, eggs, tomatoes, dark meat chicken and turkey, raisins, prunes
Magnesium	<ul style="list-style-type: none"> ○ Helps in hundreds of enzyme reactions involved in the synthesis of DNA and proteins ○ Helps nerve conduction and muscle contraction 	Pumpkin seeds, almonds, cashews, beans, spinach, milk, figs, brown rice, cocoa powder, molasses, peanuts, pineapple, okra, milk, bananas
Manganese	<ul style="list-style-type: none"> ○ Facilitates bone development ○ Helps make and break down proteins and glucose 	Brown rice, oatmeal, spinach, pineapples, almonds, pecans, molasses, whole-wheat bread, sesame seeds
Molybdenum	<ul style="list-style-type: none"> ○ Helps metabolise proteins, DNA, drugs, and toxins 	Beans, lentils, peas, grain, nuts
Phosphorus	<ul style="list-style-type: none"> ○ Structural component of bones and teeth, DNA, and cell membranes ○ Helps in energy production and storage 	Milk, yogurt, salmon, halibut, lentils, beef, peanuts, sunflower seeds, beans, chicken, turkey, almonds, cheese, eggs, whole-wheat bread
Potassium	<ul style="list-style-type: none"> ○ Maintains fluid and electrolyte balance ○ Required for proper nerve conduction and muscle contraction ○ Lowers blood pressure 	Beans, potatoes (with skin), prunes, raisins, acorn squash, bananas, spinach, tomato juice, artichokes, molasses, tomatoes, oranges
Selenium	<ul style="list-style-type: none"> ○ Influences thyroid hormone function ○ Component of antioxidant enzymes 	Brazil nuts, crab meat, salmon, halibut, pasta, pork, shrimp, whole-wheat bread, brown rice, beef, light-meat chicken, milk, black walnuts
Sodium	<ul style="list-style-type: none"> ○ Maintains fluid and electrolyte balance ○ Vital for nerve conduction and muscle contraction ○ Increases blood pressure 	Baked goods, processed meat, restaurant food, pizza, canned soups, table salt
Zinc	<ul style="list-style-type: none"> ○ Helps in hundreds of enzyme reactions, haemoglobin production, and immune function 	Oysters, beef, crab meat, dark-meat chicken and turkey, pork, yogurt, milk, cashews, chickpeas, almonds, peanuts, cheese

Note. Adapted from “Nutrition Facts Label Programs and Materials” by U.S. Food and Drug Administration (USFDA), 2018a, *Food*, Copyright 2018 by Author. Adapted from “Vitamins” by USFDA, 2018, Copyright 2018b by Author.

Newly absorbed glucose then travels through the bloodstream to cells where it is either utilised for energy (glucose pool), stored away as a reserve energy supply (glycogen stores), or converted (excess carbohydrates) into fatty acids via the process of lipogenesis (Jiang & Zhang, 2003; Saltiel & Kahn, 2001). When the glycogen stored in the liver is required by active tissues, initially it must be converted back into glucose (glycogenolysis) before it can be transported by the blood to where it will be metabolised (Hundal et al., 2000; Jiang & Zhang, 2003), whereas glycogenesis is the process for the conversion of glucose to glycogen (Hundal et al., 2000; Jiang & Zhang, 2003; Saltiel & Kahn, 2001). However, elevated levels of blood glucose in the body stimulate the release of insulin, which ensures the uptake of glucose and conversion into glycogen for storage via glycogenesis (Hundal et al., 2000; Saltiel & Kahn, 2001). Consequently, sustained increased blood glucose levels trigger various negative reactions (Hundal et al., 2000; Jiang & Zhang, 2003; Saltiel & Kahn, 2001). For example, increased amounts of sugar overload the liver, and over time the liver begins to convert carbohydrates to fat, resulting in excessive buildup of fat, which may result in fatty liver disease, a contributor to diabetes (American Diabetes Association, 2014; Hundal et al., 2000). Additionally, continual high blood glucose levels can reduce the elasticity of the blood vessels due to irreversible cross-linking of collagen fibres, which increases the likelihood of plaque buildup, reducing blood flow and causing elevated blood pressure and increased resistance in the circulatory system (Conget & Giménez, 2009; Kuipers et al., 2011). Collectively, the mentioned disorders trigger an inflammatory response that attempts to counteract the development of these cardiometabolic complications (Conget & Giménez, 2009). However, when inflammation fails to eliminate the accumulating disorders it remains active and can linger for months or even years (Ouchi et al., 2011; Wang & Nakayama, 2010). There is also the possibility that

inflammation remains active even after the initial threat has been eliminated (Ouchi et al., 2011; Wang & Nakayama, 2010). The immune system then triggers white blood cells to attack healthy tissues and organs, resulting in chronic inflammatory processes which can lead to many diseases, including CVD (Navarro et al., 2017; Sorensen et al., 1994; Tedgui & Mallat, 1999). For further information on the inflammatory process, see section 2.0.1 on cardiometabolic health and section 2.1.1 on body fatness.

Protein. Proteins are large biological molecules made of amino acids joined together by peptide bonds (Damodaran, 2007; Iroyukifujita, Eiichiyokoyama, & Yoshikawa, 2008; Sarmadi & Ismail, 2010). The development of proteins occurs when numerous amino acids come together by dehydration to form peptide bonds, which are the covalent bonds holding amino acids together (Damodaran, 2007; Sarmadi & Ismail, 2010; Wu, Shen & Shiau, 2003). The body utilises proteins for various tasks including: building and repairing tissues, storing energy, acting as enzymes, aiding the immune system, and serving as hormones (see Figure 6; B. I. Campbell & Spano, 2011; Damodaran, 2007). Additionally, protein can be used as an energy source; however, it must first be converted to glucose and this process is called gluconeogenesis (B. I. Campbell & Spano, 2011; Gropper & Smith, 2012). Overall, proteins serve important functions as enzymes, but provide minimal energy for metabolism (B. I. Campbell & Spano, 2011; Cooper, 2000; Gropper & Smith, 2012; Iroyukifujita et al., 2008). Enzymes act as catalysts by changing the rate of chemical reactions in the body (Cooper, 2000; Gropper & Smith, 2012; Iroyukifujita et al., 2008). Enzymes are classified according to the type of reaction they catalyse (Cooper, 2000; Iroyukifujita et al., 2008). For example, ligases connect compounds, hydrolases split compounds, isomerases relocate atoms within a molecule, oxidoreductases transfer electrons, and transferases move functional groups (Cooper, 2000;

Gropper & Smith, 2012; Iroyukifujita et al., 2008). The physiological process of energy production and digestion depends on enzyme function (Cooper, 2000; Gropper & Smith, 2012; Iroyukifujita et al., 2008).

Amino acids are the basic building blocks of proteins, which contain nitrogen; the body must have enough of both nonessential and essential amino acids to produce the wide variety of proteins required (Damodaran, 2007; Trumbo, Schlicker, Yates, & Poos, 2002). Protein is essential for the repair, growth, and maintenance of the cells, so it is vital to obtain both forms of amino acids (Damodaran, 2007; Trumbo et al., 2002; R. P. Wilson, 2003). The body can synthesise nonessential amino acids using available amino acids or through metabolic processes; however, essential amino acids cannot be made by the body, which means they must be obtained from dietary intake (Damodaran, 2007; Trumbo et al., 2002; R. P. Wilson, 2003). Incomplete proteins are plant source proteins that are lower in essential amino acids but provide sufficient intake of nonessential amino acids (Richter, Skulas-Ray, Champagne, & Kris-Etherton, 2015; Trumbo et al., 2002), whereas, complete proteins generally come from animal sources and contain all the amino acids required (Richter et al., 2015; Trumbo et al., 2002). When analysing and comparing plant-based with animal-based proteins, the outcomes on cardiometabolic risk factors are significant (Clifton, 2011; Richter et al., 2015). For example, complete proteins are associated with animal meats high in saturated fat, which can increase levels of LDL-C in the body (Clifton, 2011; Trumbo et al., 2002; Van Herpen & Schrauwen-Hinderling, 2008). Consequently, an accumulation of lipids results in an imbalance between the amount of lipids produced and lipids utilised, which may lead to cell dysfunction, triggering inflammation and significantly increasing the risk of developing cardiometabolic complications (Clifton, 2011; Richter et al., 2015; Trumbo et al., 2002). For further

information on lipids and the inflammatory process, see section 2.0.1 on cardiometabolic health and section 2.1.1 on body fatness.

Lipids. Lipids are hydrocarbon chains composed of carbon, hydrogen, and oxygen; however, their arrangement, number, type, and placement of interconnecting bonds give lipids their various classifications and functions (Gropper & Smith, 2012; Hubler & Kennedy, 2016; Jones & Papamandjaris, 2012). The carbon-carbon bonds within the hydrocarbon chain can be saturated or unsaturated (Gropper & Smith, 2012; Scherfeld, Kahya, & Schwille, 2003). Lipids are hydrophobic, meaning they are incapable of being dissolved in water, yet they are soluble in nonpolar solvents (Gropper & Smith, 2012; Jones & Papamandjaris, 2012; Scherfeld et al., 2003). Lipids are extremely suitable for energy storage because they store a substantial amount of energy for their size and contain over two times as much energy as the same weight of carbohydrates or proteins (Gropper & Smith, 2012; Hubler & Kennedy, 2016; Jones & Papamandjaris, 2012). The majority of lipids stored are triacylglycerol, which consists of a glycerol molecule combined with free fatty acids (unsaturated fats, saturated fats, and trans-fat; see Figure 6; M. D. Jensen, 2006; Jones & Papamandjaris, 2012; Redinger, 2007). Fatty acid is a major component of fats that are used by the body for energy and tissue development (M. D. Jensen, 2006; Jones & Papamandjaris, 2012; Redinger, 2007). Glycerol is a three-carbon substance that serves as the central structural component of a triglyceride and is the only part that can be converted to glucose if needed (Jones & Papamandjaris, 2012; Van Herpen & Schrauwen-Hinderling, 2008).

The body uses various lipoproteins to transport digested lipids through the bloodstream (Hubler & Kennedy, 2016; Jones & Papamandjaris, 2012; Van Herpen & Schrauwen-Hinderling, 2008). Chylomicrons transport dietary lipids from the mucosal cells

of the small intestine to muscle and adipose tissue (De Souza et al., 2015; Hubler & Kennedy, 2016; Jones & Papamandjaris, 2012). High density lipoproteins remove cholesterol from the body and transport it to the liver for disposal; consequently HDL-C is referred to as “good” cholesterol (De Souza et al., 2015; Hubler & Kennedy, 2016; Jones & Papamandjaris, 2012). Low density lipoproteins, however, contain a higher proportion of cholesterol, so they mainly transport cholesterol from the liver to tissues (Hubler & Kennedy, 2016; Jones & Papamandjaris, 2012; Van Herpen & Schrauwen-Hinderling, 2008). However, too much circulating LDL-C leads to the accumulation of these cholesterols in the blood vessel intima (Navarro et al., 2017; Sorensen et al., 1994; Tedgui & Mallat, 1999). This accumulated LDL-C then becomes oxidised by free radicals and by glycation, which then initiates the pathophysiology of atherosclerosis (Kenney et al., 2015; Tedgui & Mallat, 1999). For further information on lipids and the pathophysiology of atherosclerosis, see the background on cardiometabolic health (section 2.0.1) and body fatness (section 2.1.1).

Fruits and vegetables. Fruits and vegetables provide the body energy and nutrients through an assortment of micro and macronutrients (Reddy & Katan, 2004; Slavin & Lloyd, 2012). Fruits and vegetables contain fibre, and consistent fibre intake has been shown to have a positive influence on cardiometabolic complications in multiple ways, including the feeling of satiation; reduced LDL-C levels, blood pressure, and inflammation; and slowed absorption of monosaccharides, which helps to improve glucose levels (Lie et al., 2018; M. A. Pereira & Liu, 2003; Slavin & Lloyd, 2012). Fruits and vegetables also provide the body with minerals and vitamins, some of which are labelled good sources of chemical compounds that function as antioxidants (M. A. Pereira & Liu, 2003; Slavin & Lloyd, 2012). Moreover, a sufficient number of antioxidants is essential to the functioning

of the body as an imbalance between antioxidants and prooxidants (free radicals) can lead to oxidative stress, which triggers inflammation, and ultimately results in endothelial dysfunction, leading to atherosclerosis and subsequent CVD (Davignon & Ganz, 2004; Galley & Webster, 2004; Slavin & Lloyd, 2012). For further information on oxidative stress, inflammation, and free radicals, see the cardiometabolic health background in section 2.0.1. Despite the known importance of fruits and vegetables for health, their consumption continues to decline, and insufficient fruit and vegetable intake is among the top 10 health risk factors for global mortality and responsible for approximately 1.7 million deaths worldwide (Hawkes, 2006; Mozaffarian, 2006; WHO, 2018c).

Collectively, fruit and vegetables, grains, dairy products, and protein provide essential nutrients the body requires to function properly day-to-day. Furthermore, an unhealthy diet (Emmett & Jones, 2015; Harrex et al., 2017; Howe et al., 2016), along with lower than ideal physical fitness levels (Cohen et al., 2011; Howe et al., 2016; Stratton et al., 2007), insufficient daily physical activity (O. Ali et al., 2014; Dumuid et al., 2018; WHO, 2017d), sedentary behaviour (Cohen et al., 2011; Glynn et al., 2005; Hjorth et al., 2013), inadequate sleep (X. Chen et al., 2008; Hjorth et al., 2013; Skidmore et al., 2013), and excessive body fatness (Bridger, 2009; Trandafir et al., 2017) have all been linked to cardiometabolic risk factors. As mentioned in section 2.0.1, cardiometabolic risk factors tend to cluster; therefore, an unhealthy diet has been identified as one of the determinants of deteriorating health in children (WHO, 2015, 2017a). However, uncertainty exists as to whether nutrition or another lifestyle factor associates more strongly with cardiometabolic risk factors in preadolescent children.

2.4.2 Evaluating Nutrition

In preadolescent children, dietary intake is commonly assessed utilising self-report assessment methods including a record of food and food frequency questionnaires (Saeedi, Skeaff, Wong, & Skidmore, 2016). The New Zealand MOH food and nutrition guidelines provide a detailed outline of the four food groups in addition to recommending daily dietary serving sizes for children (MOH, 2015a). The following paragraph elaborates on the indicated nutritional methods as well as presenting evidence supporting the evaluating procedures applied in this study over other approaches.

Dietary intake refers to the daily eating patterns of an individual, including the quantity of calories, nutrients, and specific foods consumed. A record of food is limited and often employed to document dietary intake of the foods and beverages consumed over a few days, capturing only short-term dietary intake (Cade, Thompson, Burley, & Warm, 2002). In comparison, food frequency questionnaires are utilised in a variety of ways and in various study designs with the most common method being to obtain estimates of an individual's food intake over a longer period, usually a 7-day span (Cade et al., 2002). Additionally, food frequency questionnaires are the most commonly used dietary assessment tool in large nutritional epidemiological studies (Cade et al., 2002; Saeedi et al., 2016). A study carried out on New Zealand adolescents (aged 14 to 18 years, $n = 41$), compared a 4-day food record with a dietary quality index obtained from a food frequency questionnaire, and the results indicated that the dietary quality index assessment method was moderately reliable and valid for rating dietary intake in adolescents (Wong, Parnell, Howe, Black, & Skidmore, 2013). In this study, a small-scale sample size was assessed ($n = 41$) and the participants were teenagers, so the results do not provide an accurate comparison to a study design with a sample size of 392 preadolescent children. However,

the food frequency questionnaire used in this example study is very similar to the one applied in my study design, which is essential when comparing food frequency questionnaires results. It is important to highlight that for large-scale epidemiological studies, food frequency questionnaires must be designed for a specific populations, including age appropriate and culturally relevant food items (Kolodziejczyk, Merchant, & Norman, 2012; Saeedi et al., 2016).

Applied nutritional assessment. Therefore, in this study, information on food choice was collected utilising the recently developed Physical Activity, Exercise, Diet, and Lifestyle Study Food Frequency Questionnaire (PEDALS FFQ; see Appendix A), which was specifically designed to estimate dietary intake of preadolescent children and has been validated in this age group (Saeedi et al., 2016). See the data analysis sections in Chapters 4 and 5, sections 4.2 and 5.2, respectively, for further information.

2.5 Sleep

2.5.1 Background

Sleep disturbances may occur in up to 43% of children (Archbold, Pituch, Panahi, & Chervin, 2002), and a good night's rest is significant to how a child functions throughout an entire day (Curcio, Ferrara, & De Gennaro, 2006; Dewald, Meijer, Oort, Kerkhof, & Bögels, 2010; Meijer, Habekothé, & Van Den Wittenboer, 2000). For example, children who go to bed early are more active, excel in school, and have minimal behaviour problems while at school, when compared to children who suffer from sleep disturbances (Archbold et al., 2002; Curcio et al., 2006; Dewald et al., 2010; Meijer et al., 2000; Owens, Spirito, McGuinn, & Nobile, 2000). Sleep-wake is when the nervous system is inactive, which begins a period of rest for the body and mind. During this state of unconsciousness the

mind and body are working to repair, grow, learn, and restore, which makes sleep essential to everyday life (Curcio et al., 2006).

Sleeping, waking, and metabolic cycles are linked at the molecular level, and the functioning of these cycles is controlled by the internal circadian clock (Challet, 2013). In today's world, the rhythm of the circadian clock is often interrupted by social obligations, including school, sports, and other extracurricular activities (Wittmann, Dinich, Mellow, & Roenneberg, 2006). The circadian clock regulates energy homeostasis and its interruption could contribute to weight-related complications (Roenneberg, Allebrandt, Mellow, & Vetter, 2012). Additionally, insufficient sleep changes the levels of the appetite-regulating hormones leptin and ghrelin in the body. Leptin is an appetite suppressant and ghrelin is an appetite stimulant; during sleep deprivation ghrelin levels increase, while leptin levels decrease (Sayin & Buyukinan, 2016; Skidmore et al., 2013). As a result of sleep deprivation, a signal of limited energy supply is sent to the brain, which then triggers hunger. In general, children with poor sleep habits are more likely to have deficient dietary patterns, which could alter glucose and fatty acid breakdown and lead to hyperglycaemia, hyperlipidaemia, undesired weight gain, obesity, and eventually CVD (Chaput et al., 2015; Harrex et al., 2017; Sayin & Buyukinan, 2016; Skidmore et al., 2013).

Consequently, inadequate sleep (X. Chen et al., 2008; Harrex et al., 2017; Hjorth et al., 2013; Skidmore et al., 2013), along with lower than ideal physical fitness levels (Cohen et al., 2011; Howe et al., 2016; Stratton et al., 2007), insufficient daily physical activity (O. Ali et al., 2014; Dumuid et al., 2018; WHO, 2017d), sedentary behaviour (Cohen et al., 2011; Glynn et al., 2005; Hjorth et al., 2013), and unhealthy diet (Emmett & Jones, 2015; Harrex et al., 2017; Howe et al., 2016), have all been linked to cardiometabolic risk factors, including obesity (Cohen et al., 2011; Hjorth et al., 2013; Stratton et al., 2007). As stated in

section 2.0.1, cardiometabolic risk factors tend to cluster; accordingly quality of sleep has been identified as one of the determinants of cardiometabolic risk factors in adolescents (Harrex et al., 2017; Skidmore et al., 2013); however, uncertainty exists as to whether sleep or another lifestyle factor associates more strongly with cardiometabolic risk factors in preadolescent children.

2.5.2 Evaluating Sleep

In preadolescent children, sleep is commonly assessed utilising objective assessment methods (polysomnography, actigraphy) and self-report assessment methods (record/diary, questionnaire). In accordance with the New Zealand MOH *Children and Young People Living Well and Staying Well* report, children aged 5 to 13 years should sleep between 9 to 11 hours on a nightly basis (MOH, 2017). The following paragraphs elaborate on the indicated sleep assessment methods, in addition to presenting evidence supporting the evaluating procedures applied in this study over other approaches.

Objective measures. Polysomnography is an electronic recording of bodily functions and physiological changes that occur while sleeping. This test is administered by placing electrodes on the face, scalp, chest, and limbs. The electrodes send the sleep data recorded through electrical signals to the polysomnography machine. Laboratory-based polysomnography is considered the gold standard measurement for assessing quality of sleep in children and adults (Ancoli-Israel et al., 2003; Meltzer, Biggs, et al., 2012; Van de Water, Holmes, & Hurley, 2011). In children (aged 2 to 18 years), polysomnography is the commonly preferred assessment method when assessing and diagnosing obstructive sleep apnoea, yet polysomnography has not been completely standardised in its functioning or interpretation in paediatric research (Schechter, 2002). Polysomnography assessment has also been associated with additional limitations including: invasiveness, time required,

inconsistencies with setup and criteria, limited time to acclimate with sleep lab study setup, difficulties sleeping in an unfamiliar environment, and cost to administer (Beck & Marcus, 2009; Schechter, 2002). Furthermore, there is limited research available on utilising polysomnography to assess sleep in preadolescent children, and polysomnography testing is impractical for large paediatric epidemiological studies.

Actigraphy is a noninvasive measuring method utilised to monitor sleep while individuals remain in their natural environment. Actigraphy testing consists of a small portable device worn on the wrist, ankle, or trunk that identifies and records movement and rest cycles (Meltzer, Montgomery-Downs, Insana, & Walsh, 2012; Meltzer et al., 2016). In the last 20 years, specifically for infants and children, actigraphy has gained popularity in paediatric sleep research due to the ability to measure sleep continuously 24 hours a day and for an extended period (Ancoli-Israel et al., 2003; Beck & Marcus, 2009; Meltzer, Montgomery-Downs, et al., 2012). When compared to self-report measures, actigraphy is more objective and has the ability to continuously record over an extended period (Meltzer et al., 2016). Additionally, actigraphy is cost-efficient and less invasive when compared to overnight polysomnography (Meltzer et al., 2016). However, there are limitations associated with actigraphy assessment including: battery life inconsistencies, susceptibility to damage, participant dependence (e.g., remembering to put on; Meltzer, Montgomery-Downs, et al., 2012), and impracticality for large epidemiological studies. Additionally, in children (aged 5 to 12 years) there is limited research available utilising actigraphy to assess sleep (Ancoli-Israel et al., 2003; Meltzer, Montgomery-Downs, et al., 2012; Meltzer et al., 2016; Sadeh, Hauri, Kripke, & Lavie, 1995); therefore, actigraphy lacks consistency and standardised cutoff points, variables, and preparation guidelines in preadolescent children (Meltzer, Montgomery-Downs, et al., 2012).

Self-report measures. Sleep can be measured utilising self-report documents such as a record/diary or a questionnaire. Sleep self-report measures have been validated among adolescents (Wolfson et al., 2003). Additionally, self-report measures have been utilised to assess the effects of inadequate sleep on poor school performance, lack of daily focus, and below average energy levels (Wolfson et al., 2003). A study carried out on American children (aged 11 to 15 years, $n = 450$) found that the Paediatric Daytime Sleepiness Scale record is appropriate for assessing daytime sleepiness in middle school-aged children (Drake et al., 2003). However, the paediatric daytime sleepiness scale does not account for the amount of sleep an individual gets daily or the time of day the sleep occurs, even though sleep duration and sleep onset delay are two of the eight sleep subscales that associate with sleep complaints relevant to this age group. Another study carried out on American children (aged 11 to 17 years, $n = 411$) found that the Cleveland Adolescent Sleepiness Questionnaire is appropriate for assessing daytime sleepiness in teenagers (Spilsbury, Drotar, Rosen, & Redline, 2007). The majority of the participants in this study were from Caucasian families from higher socioeconomic backgrounds, so the outcomes may not provide an accurate estimation of daytime sleepiness compared to participants from diverse backgrounds living in lower socioeconomic neighbourhoods. Collectively, there are strengths and limitations associated with self-report measures. Strengths include that they are cost-efficient, noninvasive, easy to administer, and practical for large epidemiological studies. Limitations associated with self-report measures include consistency of data collected, incomplete accounting for all data relevant to the age group, and vulnerability to human error. Overall, the participants in this study were preadolescent children aged 8 to 10 years, and although there are limited reliable questionnaires available to measure quality of sleep in younger children, there is evidence that children as young as

8 years of age can provide sufficient information compared to a parent report (Meltzer et al., 2013; Meltzer, Montgomery-Downs, et al., 2012; Paavonen et al., 2000). Additional research is essential before recommendations can be made regarding the validity and reliability of self-report measures as an indicator of quality of sleep in preadolescent children.

Applied sleep assessment. In this study, quality of sleep (sleep duration, social jetlag, and sleep disturbances) was assessed using the Child Sleep Habits Questionnaire (CSHQ; Owens et al., 2000) and a self-report document (see Appendix A). To determine average sleep duration, participants' caregiver(s) were asked to note what time their child usually went to bed and what time they usually got up on both school days and weekend days. Average sleep duration was then calculated using a ratio of 5 weekdays to 2 weekend days. Social jetlag was calculated as the absolute difference between the midpoints of sleep on weekdays versus weekend days (Roenneberg et al., 2012). Sleep disturbances were recorded using the 33-item CSHQ. The CSHQ includes eight subscales that align with the key sleep complaints relevant for this age group: bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, night waking, parasomnias, sleep-disordered breathing, and daytime sleepiness. For further information, see the data analysis sections in Chapters 4 and 5, sections 4.2 and 5.2, respectively.

2.6 Summary

Globally, CVD has been identified as the leading cause of death as more people die from CVD than any other cause annually (WHO, 2017a). Preceding cardiometabolic risk factors are modifiable lifestyle factors including but not limited to: body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep. However, uncertainty remains as to which lifestyle factor(s) associate(s) more strongly with

cardiometabolic risk factors in preadolescent children. This is the first study to examine simultaneously the associations among the listed lifestyle factors with cardiometabolic risk factors in preadolescent children, attempting to identify the ideal public health strategy necessary for improving cardiometabolic health in preadolescent children.

2.6.1 Applied Measures Analysis

After reliability and validity were established, other factors, including those detailed in the previously described studies, determined measurement approaches including: invasiveness, time efficiency, cost, child friendliness, and practicality (see Figure 7). Furthermore, as mentioned throughout the literature review, this study was a large epidemiological study carried out on various primary school grounds across the North and South Island of New Zealand, so portable assessment methods were essential. Additionally, preceding data collection, study funding was absorbed entirely by the cardiometabolic biochemical markers requirements. Therefore, study assistants had to be recruited as volunteers from surrounding universities, thus simple evaluation methods that required minimal training were crucial as there was limited time to instruct new study assistants at each primary school location (see section 3.3 for more information on protocols).

Study Factors	Outcome Measures		Rationale
	Selected Methods	Alternatives	
Cardiometabolic	Finger prick Lipid panel, Serum glucose & HbA1c	Venepuncture	Less invasive, Cost efficient, Age appropriate, Simplicity
Cardiometabolic	PWA (SBP, DBP, CBP, HR AIX)	PWV, FMD	Simplicity, Cost efficient, Multiple measurements analysed simultaneously
Body Fatness	Anthropometry (BMI & WHR)	Skinfold callipers, Ultra sound, WHtR, WC	Less invasive, Timely, Simplicity
Body Fatness	BIA (Body fat percent & FMI)	Hydrostatic weighing, DEXA scan	Practical, Cost efficient, Simplicity
Physical Fitness	Cardiorespiratory fitness (VO_{2max})	Polysomnography, ECG, Motion sensors	Practical, Validated in pre-adolescent children
Physical Fitness	Muscular fitness Hand grip dynamometer	Standing long jump, Sit-ups, Push-ups	Efficiency, Validated in pre-adolescent children
Physical [In]activity, Sedentary Behaviour	Questionnaire (YPAQ)	Movement devices, Direct observation, Additional self-report measures	Practical, Timely, Cost efficient, Simplicity
Nutrition	Questionnaire (PEDALS FFQ)	Record of food diary, Additional self-report measures	Simplicity, Validated in pre-adolescent children
Sleep	Questionnaire (CSHQ) Self-report	Polysomnography, Actigraphy, Sleep diary, Additional self-report measures	Cost efficient, Timely, Simplicity

Figure 7. PACMAC measuring methods.

CHAPTER 3—METHODOLOGY

Pre-Adolescent Cardiometabolic Associations and Correlates:

PACMAC Methodology and Study Protocol¹

3.0 PACMAC Study Protocol

While CVD is typically associated with middle or old age, the atherosclerotic process often initiates early in childhood (Sorensen et al., 1994). The process of atherosclerosis appears to be occurring at an increasing rate, even in preadolescents, and has been linked to the childhood obesity epidemic (Lambrick, Stoner, Faulkner, & Hamlin, 2014; Olshansky et al., 2005). In New Zealand, the prevalence of childhood (aged 2 to 14 years) obesity increased from 8.4% in 2006-2007 to 11.1% in 2012–13 (MOH, 2014). Furthermore, within New Zealand, much higher rates of obesity have been reported among Māori children (18.6%) than among their counterparts of European ancestry (7.5%; MOH, 2014). The prevalence of obesity has taken its toll among the Māori population, placing these cohorts at greater risk for obesity-related cardiometabolic complications, including dyslipidaemia, hypertension, type 2 diabetes mellitus, and subsequent CVD (Stoner, Stoner, Young, & Fryer, 2012).

A child's body composition is determined by interactions between their genes and lifestyle behaviours. The genetic makeup of children has not altered dramatically over the last two decades in which obesity has rapidly increased. It is likely, therefore, that lifestyle

¹ Previously published as an Open Access journal with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license as Castro, N., Faulkner, J., Skidmore, P., Williams, M., Lambrick, D. M., Signal, L., . . . Stoner, L. (2014). Pre-adolescent cardiometabolic associations and correlates: PACMAC methodology and study protocol. *BMJ Open*, 4(e005815), 1-9. doi:10.1136/bmjopen-2014-005815

behaviours and environmental influences are a probable cause of this childhood obesity epidemic (Hill, Wyatt, Reed, & Peters, 2003; Stoner et al., 2012). While commonly cited causes of obesity include declining physical activity and fitness levels (Waters et al., 2011), poor nutrition (Waters et al., 2011) and poor sleep habits (Carter, Taylor, Williams, & Taylor, 2011), no study has comprehensively assessed all of these components together in primary school-aged children. Furthermore, it is unknown whether (a) body fatness (body composition), physical fitness, physical activity, nutritional behaviour, or sleep is the most important correlate (strongest independent predictor) of a child's cardiometabolic health, and (b) whether these relationships are consistent between Māori and Caucasian children. The findings from this study may allow us to develop culturally appropriate interventions for children to promote sustainable obesity-preventing lifestyles and to improve cardiovascular health (Lambrick et al., 2014).

Our aim is to investigate the relationships among obesity, lifestyle behaviours (nutrition, physical activity, physical fitness, sleep behaviour, psychosocial influences) and cardiometabolic health in prepubescent (8- to 10-year-old) children. Furthermore, the research will investigate whether there are differences in the correlates of cardiometabolic health between Māori and Caucasian children. The research outcomes from Pre-Adolescent Cardiometabolic Associations and Correlates (PACMAC) will help inform health professionals, health and physical education curricula, and children health policy guidelines as to the most effective way to make an impact on the deteriorating health of children. The stated research will increase the understanding of CVDs that are affecting New Zealanders, and subsequently through more targeted interventions, enhance the health and well-being of children and consequently adults.

3.0.1 Research Study Objectives

1. Investigate the associations of physical [in]activity, physical fitness, nutrition, sedentary behaviour, and sleep with body fatness.
2. Investigate the association of body fatness, nutrition, physical [in]activity, physical fitness, sedentary behaviour, and sleep with cardiometabolic health.

3.1 Methodology

3.1.1 Study Design

A cross-sectional study design will be used to investigate relationships among obesity, lifestyle behaviours, and cardiometabolic health in a sample of prepubescent children aged 8 to 10 years from three representative sample sites from across New Zealand (Wellington, Canterbury, and Otago). Data collection will begin in October 2014 and will persist for 18 months. To reduce disruption and burden for the children, previously validated broadly applicable research instruments, which have been used in large-scale national and multinational epidemiological studies, will be utilised (see Figure 8).

	Monday - Thursday					Friday		
Location	School: Quiet Room					School Hall	Home	
Outcome	BMI	WHR	Rest	PWA	Taste	Blood	20-MST	Web Survey
Mins:	5	20	5	5	5	10	60	

Figure 8. Study protocol. 20-MST, 20-m multistage shuttle run test; bio-impedance analysis (BIA); body mass index (BMI); pulse wave analysis (PWA); waist-to-hip ratio (WHR).

3.1.2 Study Participants and Recruitment

Four hundred children will be recruited for participation where measures will be largely based on self-report.² In addition to having self-reported measures, objective measures will be taken. Children will be deemed eligible if they are aged 8 to 10 years and are asymptomatic of injury or illness as determined by a standardised health screening questionnaire and a physical activity readiness-questionnaire (PAR-Q), completed by a parent or guardian. Children will be ineligible to participate if they have had an orthopaedic injury or surgery that has prohibited full function within the previous 4 weeks or are prescribed any cardiovascular medications. Written parental or guardian consent and child assent will be obtained prior to participation

Schools (Wellington, Canterbury, and Otago) will be stratified by socioeconomic status and randomly selected. School principals will be contacted initially via email invitation, followed by a face-to-face meeting. Written consent will be sought from both the principal and the classroom teachers of each school before participants in Year 3 to 5 classes are recruited. Subsequently, an information packet will be sent home with the children, and the children will be eligible to participate if they return the parent informed consent and child assent forms signed (see Appendices C and D).

3.1.3 Cultural Sensitivity

Several steps will be included within the design of the study to ensure cultural sensitivity and long-term viability of PACMAC:

² See Appendix A for the full questionnaire.

- Māori academic consultants have been involved in the development of the study and will have an ongoing role in advising on cultural issues to ensure that the study findings are appropriately delivered to Māori communities.
- Guidance will also be provided on acceptable methods of engaging Māori participants, cultural issues that may arise in relation to the study design and measures, and how these might inform the development and success of the study.

3.2 Data Collection and Analysis

The outline is depicted in Figure 8. Monday through Thursday, anthropometric, cardiometabolic, and taste outcomes will be assessed. Students will report for testing between the hours of 9:00 a.m. and 12:00 p.m., having fasted for at least 3 hours, and refrained from exercise for 24 hours (Stoner et al., 2013). Assessments will take place in a quiet, climate controlled room at the school. On the Friday, all children will have their physical fitness assessed in the school hall. A web-based (Lime Survey, open source) questionnaire will be used to collect demographic data, as well as psychosocial, sleep behaviour, physical activity, and nutrition behaviour using previously validated scales. The questionnaire will be web-based and will be completed at the child's home in consultation with a parent or guardian.

3.2.1 Demographics

Demographic data will include home and school address, sex, ethnicity, parent marital status, parent occupation, parent academic qualifications, and household income. Home address will be used to determine urban versus rural residential area, and New Zealand deprivation score as a measure of neighbourhood socioeconomic status. The school address will be used to calculate school decile rating.

3.2.2 Cardiometabolic Health

Biochemical markers TC, HDL-C, LDL-C, TG, serum glucose, and HbA1c will be supplemented with PWA. Biochemical markers will be collected using a well-tolerated finger prick procedure and analysed using portable lipid/glucose (CardioChek PA, PTS Diagnostics, IN, USA) and HbA1c (A1Cnow+, PTS Diagnostics, IN, USA) analysers. Pulse wave analysis will estimate SBP, DBP, CBP, HR, and AIx utilising the BP+ device (Uscom, Sydney, Australia). Pulse wave analysis is a simple to use, noninvasive, valid, and reliable technique that has been widely used in epidemiological studies (Stoner et al., 2013; Stoner, Lambrick, et al., 2014; Vlachopoulos et al., 2010). Each variable will be standardised as follows: $\text{standardised value} = (\text{value} - \text{mean}) / \text{SD}$. The z scores of the individual risk factors will be summed to create a cardiometabolic risk score.

3.2.3 Adiposity and Anthropometrics

Body composition will be estimated using BMI, WC, WHR, WHtR, and body composition (fat mass, fat-free mass) using BIA (BodyStat Quadscan 4000, Isle of Man, UK). The FMI will be calculated by dividing fat mass (kg) by height squared (m) and fat-free mass index (FFMI) by dividing fat-free mass (kg) by height squared (m). Using BMI, children will be classified as normal weight, overweight, or obese according to the International Obesity Task Force's cutoff points (Cole, Bellizzi, Flegal, & Dietz, 2000).

3.2.4 Physical Fitness

Cardiorespiratory fitness will be estimated using the 20-MST, a reliable and valid test that requires limited facilities (Melo et al., 2011). The results are recorded as laps taken until exhaustion.

3.2.5 Physical Activity

Physical activity type and context (e.g., modality), will be determined using the YPAQ. The 47-item YPAQ asks students to quantify the frequency, time, and duration of a range of physical and sedentary activities for both week and weekend days over the past 7 days.³

3.2.6 Nutrition

Food choice will be assessed using a specially modified, short version of New Zealand Adolescent Food Frequency Questionnaire (NZAFFQ), the New Zealand Children's Food Frequency Questionnaire (NZCFFQ), designed for use in this age group. This food frequency questionnaire (FFQ) is based on the Health Behaviour in School Children (HBSC) FFQ, which is suitable for use in this age group. This questionnaire was pretested in a sample of adolescents from Otago before using in a study, and showed good repeatability and relative validity (Wong, Parnell, Black, & Skidmore, 2012). This is a nonquantitative FFQ (i.e., it measures frequency only, not portion size). Frequency of consumption of 15 commonly consumed food items will be recorded by asking the respondent how many times weekly each item is consumed. Dietary patterns will be generated from this FFQ data using principal components analysis (PCA).

3.2.7 Sleep

Increasing evidence has indicated that short sleep duration may be related to cardiometabolic complications, including to obesity (X. Chen et al., 2008), among children. Sleep habits and sleep disturbances will be recorded using the CSHQ (Owens et al., 2000).⁴ A total score and scores on the eight subscales that cover key domains of sleep and sleep

³ See Appendix A.

⁴ See Appendix A.

behaviour (bedtime resistance, sleep onset, sleep duration, anxiety around sleep, night waking, parasomnias, sleep-disordered breathing, and morning waking/daytime sleepiness) will be calculated.

3.3 Quality Control

3.3.1 Development of Protocols

For each study method, standard operating protocols were developed (see Appendix B; Table 5). During data collection, a copy of each protocol was placed at each measuring station (e.g., cardiometabolic health, body fatness, physical fitness). These protocols included an explanation of each measurement method, step-by-step procedures, equipment guidelines, and safety precautions.

3.3.2 Training of Research Assistants

The process of training each research assistant began about a month before data collection was initiated. Research assistants were graduate students from the local universities in each region (Wellington, Canterbury, and Otago) who volunteered to assist with the PACMAC study. For each region, research assistants were contacted by phone, email, or in person. During this time, information was gathered about each research assistant such as their field of study, research background, prior training, and experience working with children. After this information was gathered, research assistants were assigned to a measuring method (e.g., anthropometry, BIA, blood pressure, handgrip strength test, 20-MST) based on their background, knowledge, and experience. They were provided a copy of the standard operating protocol (in person or via email) for their assigned measuring method. Additionally, at each day and every study site, the research assistants carried out the same measuring method for accuracy and reliability purposes.

Table 5

Standard Operating Protocol Foundations

Study Factors	Outcome Measures	Standard Operating Protocol Sources
Cardiometabolic	Finger prick, lipid panel, serum glucose & HbA1c	Barrett, Huffman, & Johnson, 2011; Parikh, Mochari, & Mosca, 2009
Cardiometabolic	PWA (SBP, DBP, CBP, & HR AIX)	Stoner et al., 2013
Body Fatness	Anthropometry (BMI & WHR)	International Obesity Task Force, 2015; WHO, 2011; WHO, 2018a;
Body Fatness	BIA (Body fat percentage & FMI)	Kyle et al., 2004a; 2004b
Physical Fitness	Cardiorespiratory fitness 20-MST ($\dot{V}O_{2max}$)	Cooper Institute (2014) FitnessGram®; Hamlin et al., 2014
Physical Fitness	Muscular fitness, handgrip dynamometer	Leong et al., 2015
Physical [In]activity, Sedentary Behaviour	Questionnaire (YPAQ)	Brooke et al., 2013; Corder et al., 2009
Nutrition	Questionnaire (PEDALS FFQ)	Saeedi et al., 2016
Sleep	Questionnaire (CSHQ), self-report	Owens et al., 2000; Roenneberg et al., 2012; Wolfson et al., 2003

A few weeks prior to data collection, all research assistants received in person practical training on the measuring method they would be implementing. Additionally, there was preparation for scenarios that may arise while collecting data on preadolescent children (e.g., participants have not fasted, are not dressed in the proper attire, or decide

they are not willing to participate). Accordingly, all research assistants were provided a copy of study protocols, extensive data collection guidance and training, and multiple repetitions of the measuring method they would be implementing before they were permitted to collect data for the PACMAC study.

3.3.3 Data Collection

Each day of data collection, the research team arrived at the primary school two hours before data collection was scheduled to begin. This allowed sufficient time to set up and inspect measurement equipment, and for members to carry out a few trial runs on each other. During data collection, the research assistants operated in pairs; therefore, each measuring station had two assistants per one participant. As one assistant took measurements from a participant, the other assistant recorded the results onto the data collection sheet (see Appendix B). This system helped to ensure that consistent data collection procedures were followed day-to-day, ethical standards were upheld, and that reliable data were collected from each participant at each primary school.

3.3.4 Data Cleaning

Each day, the participants' measurements (height, weight, WC, hip circumference, blood pressures, AIX, and HR) were recorded. The means of each variable were calculated and examined for accuracy. Then those means were transferred to an Excel spreadsheet, and the data were double-checked against the original data collection sheet for accuracy.

3.4 Sample Size Calculations

Using magnitude-based inferences to estimate the sample size required to detect the smallest beneficial (or detrimental) effect in a cross-sectional study (in this case a correlation of 0.10 which is set lower than the estimated correlation of 0.20 found in previous research between fitness and fatness [Moschonis et al., 2013], with the maximum

chances of a Type 1 and 2 error set at 5% [i.e., very unlikely]), approximately 272 children are required (Hopkins, Marshall, Batterham, & Hanin, 2009). To allow for an approximate 30% noncompletion rate across all study measures, 400 children will be recruited to the study.

3.5 Statistical Analysis

All variables will be checked for normality of distribution before analyses, and transformations will be applied where necessary. For Objective 1, the dependent variables will be body fatness indicators and the independent/predictor variables will be physical [in]activity, physical fitness, sleep, sedentary behaviour, and nutrition. Initially, partial correlations will be used to examine bivariate correlations of the predictor variables with dependent variables. Subsequently, two separate multiple regression models will be used to examine the association of the predictor variables with dependent variables. Model 1 will include each independent variable separately. Model 2 will adjust for the other predictor variables to test independent associations. The same two models will be used in logistic regression to examine the association of each predictor variable with risk of obesity. For Objective 2, the dependent variable will be composite cardiometabolic risk score and the predictor variables will include body fatness, physical [in]activity, physical fitness, nutrition, sedentary behaviour, and sleep.

3.5.1 Principal Component Analysis

In this study, PCA will be utilised because of the large number of interacting variables. Principal component analysis creates new variables by reducing and reorganising large data samples containing possibly correlated variables into a smaller number of new, combined uncorrelated variables referred to as principal components/patterns (T. Davies & Fearn, 2004). These principal components are a linear combination of the original variables

in which the maximum variance is extracted from these original variables (see Figure 9 for an example).

Food frequency questionnaire principal components analysis. Information on food choice and dietary patterns will be collected using the PEDALS FFQ, which is made up of 28 items (see Appendix A). These 28 food items from the PEDALS FFQ have been aggregated into 21 groups based on similarity in nutritional content (Saeedi et al., 2016). For example, potato chips and hot chips have been combined to form Salty Snacks, and tomato sauce, peanut butter, Nutella, jam, and honey have been combined to form Spreads. Then, PCA will be conducted to identify patterns of the components of these 21 food groups. Principal component analysis with varimax orthogonal rotation will be performed to extract the dietary patterns that maximise the sum of the variance captured. In PCA, the axes of the factors can be rotated within the multidimensional variable plot, while attempting to determine the strongest link among the variables (Corner, 2009). See Figure 9, which shows grouped variables and how they relate having undergone varimax orthogonal rotation. During the rotation, the axes move to a position that accounts for the greatest number of data points; an orthogonal rotation is when the factors are assumed to not be correlated (Corner, 2009). In this study, this will allow dietary patterns to be established from various unrelated variables. The initial PCA will describe most of the variability of the data, while the subsequent principal components will explain the maximum amount of the remaining variability (Corner, 2009; T. Davies & Fearn, 2004).

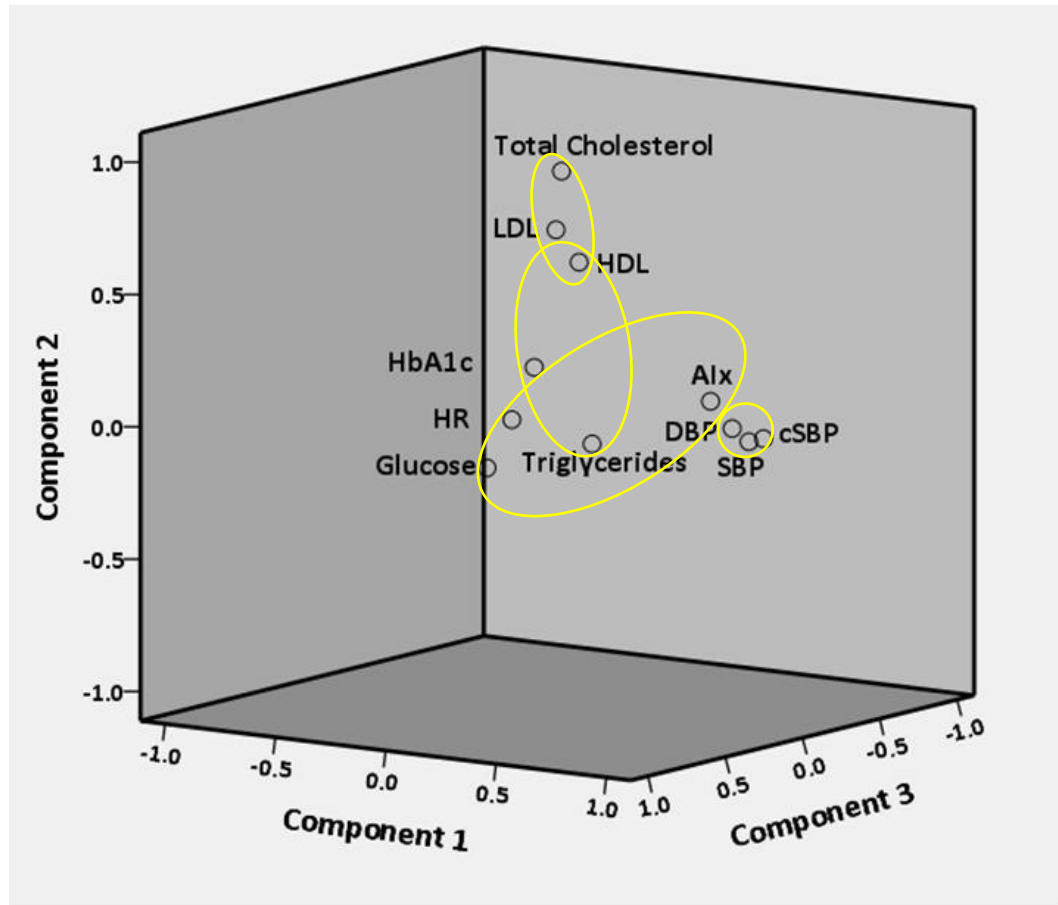


Figure 9. Component plots with factor diagrams from principal component analysis (PCA) with varimax orthogonal rotation. Augmentation index (AIx), central blood pressure (CBP), diastolic blood pressure (DBP), glycosylated haemoglobin (HbA1c), high-density lipoproteins (HDL-C), heart rate (HR), low-density lipoproteins (LDL-C), systolic blood pressure (SBP).

Determining the number of dietary components/patterns to be retained will be based on the eigenvalues > 1 (see Appendix E), identification of the elbow in the scree plot (see Figure 10), and the interpretability of factors within components/patterns, as per expert recommendations (Schulze, Hoffmann, Kroke, & Boeing, 2003). In PCA, the amount of difference accounted for by each factor is referred to as its eigenvalue, and the sum of the eigenvalues for all the factors equals the total variance (Costello & Osborne, 2005; Mulaik, 2009). Therefore, when attempting to explain the variables in terms of a smaller number of

factors, each factor must account for the variance of at least one variable (Costello & Osborne, 2005; Mulaik, 2009). The implication is that if an eigenvalue is less than one, then the derived dimension captures less variability in the data than any single variable (Costello & Osborne, 2005; Mulaik, 2009). Visual inspection of the scree plot is a more accurate method to determine the number of components/patterns to be retained than relying on eigenvalues of more than one (Schulze et al., 2003). Therefore, factor loadings greater than 0.30 will be used to interpret the factor pattern, which is based on previous FFQ PCA (Saeedi et al., 2016). Based on the work of Saeedi et al. (2016), we identified three dietary components/patterns: Processed Food, Fruit and Vegetables, and Breakfast Food (see Figure 10). Collectively, the three factors explained 70% of the variance in the measured variables.

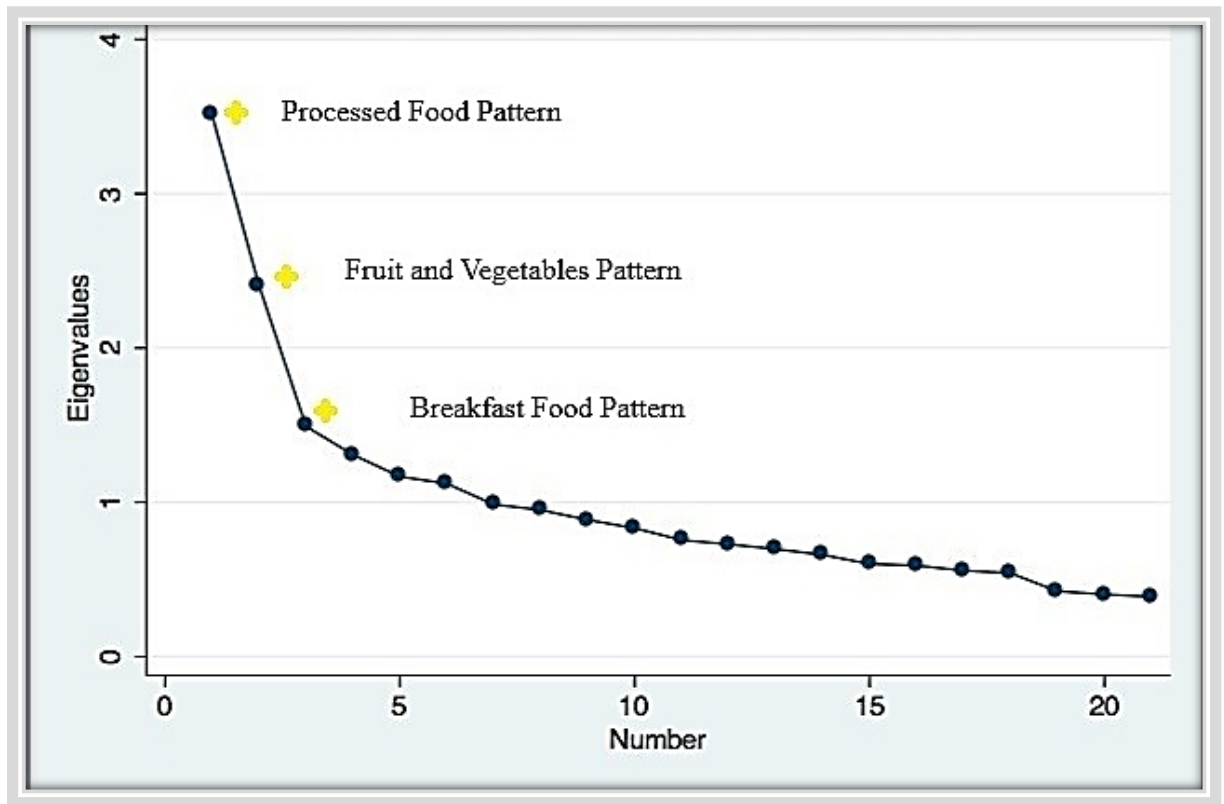


Figure 10. Scree plot of eigenvalues of the 21 food groups from the principal component analysis (PCA).

Cardiometabolic principal components analysis. Cardiometabolic factors will be derived from a PCA of the variables: SBP, DBP, CBP, TC, LDL-C, HDL-C, AIx, HR, glucose, TG concentration, and HbA1c. The PCA results are summarised in Figure 10 and Table 6. Previous research has indicated that it is common for individuals to have elevated concentrations/levels of these factors; however, we do not know the more comprehensive picture (Ahrens et al., 2014; Li et al., 2016; Sardinha et al., 2016; Trandafir et al., 2017). Therefore, these analyses will be purely exploratory, and the first aim of this analysis will be to determine if we can identify patterns using PCA. Determining the number of cardiometabolic factors to be retained will be based on the eigenvalues > 1 . The principal components will then be subjected to varimax orthogonal rotation. Factor loadings, the correlation between the derived factors and the underlying variables, will be used to interpret each factor. We used a loading of greater than 0.40 to interpret the factor pattern. On these terms, we identified four factors that represent blood pressure, cholesterol, vascular, and a carbohydrate-metabolic factor. Collectively, the four factors explained 60% of the variance in the measured variables. The SBP, DBP, and CBP loaded positively onto the blood pressure factor. Augmentation index, glucose, and HR loaded positively onto the vascular factor. Total cholesterol, LDL-C, and HDL-C loaded positively onto the cholesterol factor. In addition, HDL-C loaded positively onto the carbohydrate-metabolic factor along with TG, and HbA1c.

Table 6

Cardiometabolic Patterns

	Factor 1	Factor 2	Factor 3	Factor 4	Communality
	Blood Pressure	Cholesterol	Vascular	Carbohydrate- Metabolic	
Systolic blood pressure (mmHg)	0.90	0.06	0.16	-0.01	0.84
Diastolic Blood Pressure (mmHg)	0.89	-0.01	0.04	-0.05	0.78
Central Blood Pressure (mmHg)	0.88	-0.02	-0.08	0.04	0.80
Total Cholesterol (mmol/L)	0.01	0.92	-0.01	0.07	0.85
LDL-Cholesterol (mmol/L)	0.02	0.70	0.03	0.03	0.50
HDL-Cholesterol (mmol/L)	0.00	0.55	-0.14	-0.45	0.52
Augmentation Index (%)	0.18	-0.04	-0.73	0.02	0.57
Heart Rate (bpm)	0.27	0.10	0.68	0.02	0.54
Fasting Blood Glucose (mmol/L)	0.04	-0.13	0.51	-0.05	0.28
Triglycerides (mmol/L)	0.15	-0.10	-0.02	0.73	0.57
Glycosylated Haemoglobin (%)	-0.17	0.15	-0.10	0.68	0.52
Eigenvalue	2.5	1.7	1.3	1.2	
% Variance Explained	23	16	12	11.0	
% Cumulative Variance	23	39	51	62	
KMO	0.6				
Bartlett's Test	<0.001				

Note. Bold numbers represent variables with a factor loading ≥ 0.4 ; components retained based on an eigenvalue of > 1 .

3.5.2 Univariate Analysis

Univariate descriptive analysis of a single variable has the purpose to summarise, describe, and find patterns in the variable distribution in one sample (Canova, Cortinovis, & Ambrogi, 2017). Initially in the study, univariate analysis will be utilised to examine independently the associations among each independent variable with each dependent variable. Variables that had been previously found to be associated with the study outcomes were included in the initial univariate analysis (Carson, Hunter, et al., 2016; Carson, Tremblay, et al., 2016; Czyż et al., 2017; Moschonis et al., 2013). In utilising this approach, the many variables that fit the criteria will be identified; therefore, subsequent multivariate models will only contain variables with a significant univariate association.

3.5.3 Multivariate Analysis

Multivariate analysis allows exploration of multiple single variables at one time, which represents more of a “real life” situation, rather than looking at variables independently (Everitt & Dunn, 2001; Mengual-Macenile, Marcos, Golpe, & González-Rivas, 2015). Furthermore, multivariate analysis performs an important role in the understanding of multifaceted data while exploring more than one statistical result simultaneously (Everitt & Dunn, 2001; Mengual-Macenile et al., 2015). This analysis strategy presents the methods for both describing and exploring data, aiming to obtain the essential patterns and structure of a data set (Everitt & Dunn, 2001; Mengual-Macenile et al., 2015). Multivariate analysis will be used in this study because multiple variables (i.e., lifestyle factors) will be examined simultaneously in attempting to identify correlations among lifestyle factors and cardiometabolic risk factors.

All variables will be checked for normality of distribution before analyses, and transformations will be applied where necessary. For Objective 1, the dependent variables

will be body fatness indicators and the independent/predictor variables will be physical [in]activity, physical fitness, sleep, sedentary behaviour, and nutrition. Initially, partial correlations will be used to examine bivariate correlations of the predictor variables with dependent variables. Subsequently, two separate multiple regression models will be used to examine the association of the predictor variables with dependent variables. Model 1 will include each independent variable separately. Model 2 will adjust for the other predictor variables to test independent associations. The same two models will be used in logistic regression to examine the association of each predictor variable with risk of obesity.

For Objective 2, the dependent variable will be composite cardiometabolic risk score and the predictor variables will include body fatness, physical [in]activity, physical fitness, nutrition, sedentary behaviour, and sleep.

3.6 Dissemination of Findings

Outcomes will be shared with public health professionals and the public through outlets such as Nutrition Society Newsletters and traditional media outlets, including popular newspapers, magazines, radio and television, including ONE Te Karere. In addition, findings will be shared with the academic community through high impact, open access journal articles, and through international conferences. Most importantly, results from the study will be disseminated to participating schools and relevant Māori health entities. This will be conducted in a culturally appropriate manner and will be supported and guided by the Māori advisory team. This is likely to include a hui (Māori assembly) where cultural processes are adhered to and which promotes discussion format with participants across all ages and facets of the community and held at an appropriate venue (ideally a Marae).

3.7 Discussion

Worldwide, health systems are struggling under the escalating burden of NCDs, of which obesity and subsequent CVD are arguably at the forefront. Obesity, and co-morbid complications, are occurring at an increasingly younger age, prolonging the burden of disease and resulting in lost years of productivity, a decreased quality of life, and a growing economic burden. The research outcomes from this study will help inform health professionals as to the most effective way to make an impact on the deteriorating health of our children. The stated research will increase the understanding of current diseases affecting Māori children as well as all New Zealand children, and subsequently through more targeted interventions, enhance the health and wellbeing of children and consequently adults.

Short-term, the findings from this study will elucidate targets for decreasing obesity and improving cardiometabolic health among preadolescent children in New Zealand. The aim is to ensure an immediate impact by disseminating these findings in an applicable manner via popular media, community outlets, and traditional academic forums. Long-term, this research will facilitate larger-scale prospective and interventional studies, focusing on potential ethnic differences, changes with/across age groups, comparisons between nations, and implications on health care systems.

CHAPTER 4—RESEARCH OBJECTIVE 1

Associations With Adiposity in Preadolescent Children:

Fitness, Physical [In]activity, Sedentary Behaviour, Nutrition, and Sleep¹

4.0 Background

Globally, childhood obesity is a significant paediatric public health concern (S. A. Ali et al., 2016). In New Zealand, childhood obesity increased from 8% in 2006-07 to 12% in 2016-17 (MOH, 2015b), and in 2014, overweight and obesity prevalence in New Zealand children (aged 5 to 17 years) was the third highest worldwide (Anderson et al., 2017; Kelly & Swinburn, 2015; OECD, 2014). Short-term, overweight and obese children are at greater risk of developing physical (Maziak, Ward, & Stockton, 2008; Nishtar, Gluckman, & Armstrong, 2016; Reilly & Kelly, 2011; Sahoo et al., 2015; Stoner, Rowlands, et al., 2016; Waters et al., 2011; Williams et al., 2013), psychosocial (De Niet & Naiman, 2011; Maziak et al., 2008; Reilly & Kelly, 2011; Sahoo et al., 2015; Waters et al., 2011; Williams et al., 2013), and psychological complications (Maziak et al., 2008; Nishtar et al., 2016; Reilly & Kelly, 2011; Sahoo et al., 2015; Stoner, Matheson, et al., 2016; Waters et al., 2011; Williams et al., 2013). Long-term, overweight and obese children are at increased risk of developing cardiometabolic complications, which could lead to NCDs later in life (Anderson et al., 2015; CDC, 2015b; MOH, 2015b; Ogden et al., 2016; WHO,

¹ As described in section 1.2, this thesis is presented as a hybrid thesis. Chapters 4 and 5 each describe a distinct research objective (see section 3.0.1). The description of methodology (sections 4.1 and 5.1), data collection (sections 4.2 and 5.2), and statistical analysis (sections 4.3 and 5.3) will reflect duplication of elements consistent between the two research objectives.

2017c). While frequently cited causes of obesity include lower than ideal physical fitness (Cohen et al., 2011; Czyż et al., 2017; Stratton et al., 2007), insufficient daily physical activity (O. Ali et al., 2014; Dumuid et al., 2018; WHO, 2017d)), sedentary behaviour (Carson, Hunter, et al., 2016; Carson, Tremblay, et al., 2016; Griffiths et al., 2016; Healy & Owen, 2010), unhealthy diet (Harrex et al., 2017; WHO, 2015, 2017a), and inadequate sleep (Davison et al., 2017; Sayin & Buyukinan, 2016; Skidmore et al., 2013), no known study has comprehensively assessed the simultaneous associations among these lifestyle factors with cardiometabolic health in preadolescent children (for further information, see section 2.0.1 in Chapter 2). Consequently, uncertainty remains as to which factor correlates most strongly with body fatness (Carson, Hunter, et al., 2016; Carson, Tremblay, et al., 2016; Kuzik & Carson, 2016; Saunders et al., 2016). Therefore, it is difficult to identify the optimal public health intervention and prevention strategy for addressing childhood obesity.

The purpose of this cross-sectional study was to examine simultaneously the associations among physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with body fatness in preadolescent children. Findings from this study will add to the body of knowledge that informs health professionals and health policy writers regarding health and wellness of children and possibly make an impact on the deteriorating health of New Zealand children.

4.1 Methodology

This observational study was carried out in accordance with STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines (Von Elm et al., 2007). The methodology was prospectively detailed in Castro et al. (2014).

4.1.1 Recruitment and Participants

Children aged 8 to 10 years were recruited from schools in three major cities across New Zealand (Wellington, Christchurch, and Dunedin) between April 2015 and April 2016 (see Appendix C; see Figure 11). Within each sample site, schools from the region were stratified by decile rating and schools from each stratification were randomly invited to participate. Until 2017, New Zealand public-funded schools were classified on a 10-point decile rating based on the predominant socioeconomic status of the attending students. The Ministry of Education (MOE, 2016) website was used to determine school's decile rating. Schools were categorised by decile ratings, ranging from low (Deciles 1 to 5) to high (Deciles 6 to 10; see Table 7; MOE, 2016). Decile 1 schools comprised 10% of schools with the highest proportion of students from low socioeconomic status communities, whereas Decile 10 schools comprised 10% of the schools with the lowest proportion of students from low socioeconomic status communities (MOE, 2016).

At invited schools, all children were eligible to participate unless they had an orthopaedic injury or surgery that had prohibited full function within the previous 4 weeks or were currently prescribed any cardiovascular medications. Parental or guardian consent and child assent were obtained prior to participation in accordance with the requirements of the New Zealand HDEC (Appendix D, HDEC:14/CEN/83).

4.1.2 Study Design

The methodology for this study is fully outlined in the PACMAC study protocol (see Chapter 3). Data for this study were collected as part of a larger cross-sectional study examining PACMAC. The trial was prospectively registered with the Australia and New Zealand Clinical Trial Registry (ACTRN12614000433606), and details of the wider study

have been previously published (Castro et al., 2014). Only study details relevant to this thesis will be presented in this chapter.

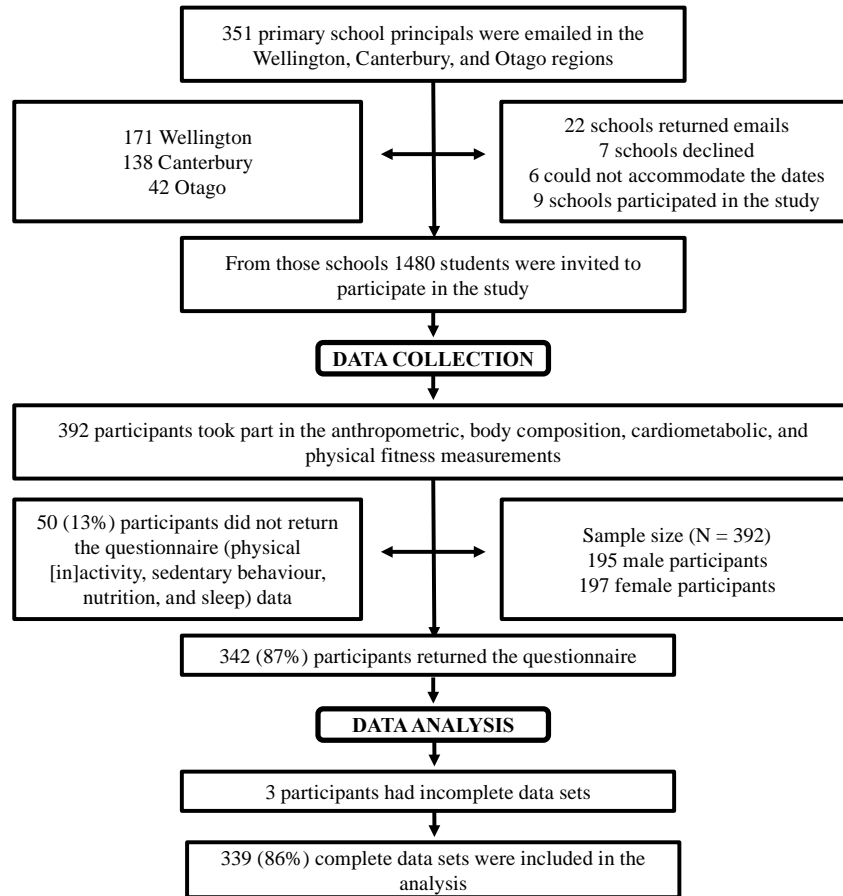


Figure 11. Participant recruitment.

At each school, data collection began on Monday and concluded on the following Friday, with each testing session taking between 30 to 45 minutes per participant. Up to 12 participants were measured each day. During the study week, adiposity and physical fitness (for further information, see sections 2.1.2 and 2.2.2) were assessed between the hours of 09:00 a.m. and 12:00 p.m. with participants having (a) refrained from exercise for the previous 24 hours, (b) fasted for at least 3 hours, and (c) ensured adequate hydration. Within 7 days of the assessments described above, physical [in]activity, dietary patterns,

sleep habits, and demographic data were collected using a questionnaire (see Appendix A). The questionnaires were completed at home jointly by the primary caregiver and participant using an online survey (Lime Survey, open source) where available. If this was not possible, a paper copy was provided, which was subsequently recorded into Lime Survey by the researcher. This study format was replicated at each school that participated in the study. Only participants with complete data sets were included in the data analyses (see section 3.3.4 for details on data cleaning).

Table 7

Participant Data Description: Categorical Variables

	All	Female	Male
Categorical Variables	<i>n</i> = 392 (%)	<i>n</i> = 197 (%)	<i>n</i> = 195 (%)
Ethnicity			
New Zealand European	279 (71)	135 (69)	144 (74)
Māori	37 (9)	21 (11)	16 (8)
Pacifics	22 (6)	12 (6)	10 (5)
Not Recorded	54 (14)	29 (15)	25 (13)
School Year			
4	82 (21)	45 (23)	37 (19)
5	114 (29)	55 (28)	59 (30)
6	127 (32)	62 (31)	65 (33)
7	69 (18)	35 (18)	34 (17)
School Decile			
Low (< 5)	211 (54)	106 (54)	105 (54)
High (> 5)	181 (46)	91 (46)	90 (46)
Obesity Status			
Overweight	122 (31)	58 (30)	64 (33)
Nonoverweight	270 (69)	139 (70)	131 (67)
Cardiorespiratory Fitness (ml/kg/min)			
$\dot{V}O_{2max}$ Low	111 (28)	46 (23)	65 (33)
$\dot{V}O_{2max}$ High	277 (71)	150 (76)	127 (66)
Not Recorded	4 (1)	1 (1)	3 (1)

4.2 Data Collection and Analysis

4.2.1 Demographics

Demographic information was self-reported by the participant's caregiver (see Tables 7 and 8, Appendix A). Demographic data collected included participants' date of birth, age, sex, ethnicity, and school address. The MOE (2016) website was used to determine school decile rating, as described in section 4.1.1. The ethnicity data were applied to categorise participants into four classifications: (a) New Zealand European and Others (NZE0), (b) Māori, (c) Pacifics, and (d) not specified. These classifications were determined by using the New Zealand in Profile 2015 prioritised ethnicity protocol (Statistics New Zealand, 2015).

4.2.2 Adiposity and Anthropometrics

Five dependent variables were recorded (see Table 8): body fat (%), fat mass (kg), FMI (kg/m^2), WHR, and BMI (kg/m^2). Multifrequency BIA (BodyStat Quadscan 4000, Isle of Man, UK) was used to measure body fat (%) and fat mass (kg; see section 3.3 and Appendix B for further information on measurement protocols). The instrument was calibrated in accordance with the manufacturer's instructions, and measurements were conducted according to standardised procedures (Kyle et al., 2004b). In accordance with manufacturer guidelines, participants drank 100 ml of water and removed any jewellery prior to the assessment. Measurements were undertaken with participants in the supine position on a nonconductive surface, with arms and legs abducted at a 30° to 45° angle from the trunk to avoid medial body contact by upper and lower extremities. Fat mass index was calculated by dividing fat mass (kg) by height squared (m^2 ; Peltz, Aguirre, Sanderson, & Fadden, 2010).

To calculate the anthropometric indices (BMI and WHR), height, weight, WC, and hip circumference were measured (see Table 8). Height was measured to the nearest 0.1 cm with shoes and socks removed and head in the Frankfort plane, using a calibrated portable stadiometer (Seca 213, Hamburg, Germany). Weight was measured to the nearest 0.1 kg (with shoes and socks removed) using a calibrated portable scale (Seca 813, Hamburg, Germany). Using nonelastic tape (Seca 203, Hamburg, Germany), WC and hip circumference were measured to the nearest 0.1 cm. Waist circumference was measured during midexpiration at the midpoint between the lower costal margin and the level of the anterior superior iliac crest, and hip circumference was measured around the widest portion of the buttocks (WHO, 2011). For each assessment, participants were measured twice, and the average was recorded (unless the two measurements were more than 0.5 cm apart; then a third measurement was taken and the average of the three was recorded). Age and sex-specific BMI z scores were calculated using the 2007 WHO growth guidelines (De Onis et al., 2007; WHO, 2018a), and BMI values (overweight and nonoverweight) were categorised using the International Obesity Task Force's sex and age-dependent cutoff points (Cole & Lobstein, 2012; International Obesity Task Force, 2015).

4.2.3 Physical Fitness

Cardiorespiratory and muscular fitness were recorded using the 20-MST and handgrip strength test (see Table 8). The 20-MST has been found to be valid and noninvasive, requiring limited space and no special facilities, and is popular in school settings as many students can be tested simultaneously (Boreham, Paliczka, & Nichols, 1990; Hamlin et al., 2014; Melo et al., 2011). The 20-MST took place at 10:00 a.m. on a Friday, in similar conditions as possible based on resources across schools. After a warm-up, stretch, and practice run, participants were asked to run in groups of 10 between two

lines set 20-metres apart (Léger & Lambert, 1982). The participants started running at a speed of 8.5 km/h⁻¹, which increased by 0.5 km/h⁻¹ every completed level; the pace was set by the audio signal emitted from a speaker (Hamlin et al., 2014). Participants were warned the first time they did not reach the line in time with the audio signal and were subsequently removed from the test if they did not reach the line for two successive shuttles, or if the participant stopped voluntarily (Hamlin et al., 2014). We estimated $\dot{V}O_{2\max}$ using the regression equation established by Hamlin et al. (2014), where total distance covered during the test, participant's body fat percentage, and age were used in the formula:

$$[\dot{V}O_{2\max} \text{ (ml/kg)} = 42.18 + (0.009 \times \text{Beep test distance in metres}) + (-0.1762 \times \text{body fat\%}) + (-0.4091 \times \text{age})].$$

The described formula has been previously validated on New Zealand children (Hamlin et al., 2014). In accordance with the Cooper Institute (2014) FitnessGram[®] cutoff points, a “healthy cardiorespiratory fitness zone” (high) was reported if girls achieved a $\dot{V}O_{2\max}$ equal to or greater than 39 and if boys achieved equal to or greater than 42 (Howe et al., 2016; Zhu et al., 2010). A $\dot{V}O_{2\max}$ below those cutoff points was categorised as “needs improvement fitness zone” (low) for both sexes (Zhu et al., 2010; see Table 7).

A handgrip dynamometer (Camry, EH101) was used to assess each participant's muscular strength (Leong et al., 2015). This method is rapid, noninvasive, simple to use, inexpensive, and of minimal risk (Leong et al., 2015), which are important factors when assessing a large group of children. The participants were seated with shoulders adducted and neutrally rotated, elbow flexed to 90 degrees, and their wrist in a neutral position (between 0 and 30 degrees extension, and between 0 and 15 degrees ulnar deviation). Then,

the participants placed their fingers around the dynamometer handle, counted down from three, and squeezed the dynamometer handle as hard as they could for 3 or more seconds. Each participant was given three attempts with each hand, alternating hands, and with a minute recovery time between each attempt. Isometric handgrip strength was measured in kilograms, and the best score for each hand was recorded for analysis.

Table 8

Participant Data Description: Continuous Variables

Continuous Variables	Mean (SD)		
	All <i>n</i> = 392	Female <i>n</i> = 197	Male <i>n</i> = 195
Age (years)	9.5 (1.1)	9.5 (1.2)	9.6 (1.0)
Body Fatness			
Weight (kg)	34.7 (9.2)	34.7 (9.2)	34.7 (9.3)
Body Fat (%)	20.2 (9.4)	22.0 (9.4)	18.6 (9.1)
Fat Mass Index (fat mass/m ²)	3.6 (2.4)	3.9 (2.3)	3.3 (2.4)
Body Mass Index (kg/m ²)	17.9 (3.3)	17.9 (3.1)	17.8 (3.4)
Waist-to-Hip Ratio (w/h)	.84 (.06)	.83 (.06)	.84 (.05)
Physical Activity & Sedentary Behaviour			
Physically Active (minutes)	164 (135)	152 (130)	175 (140)
Sedentary Behaviour (minutes)	284 (209)	286 (219)	282 (199)
Sleep			
Average Sleep Duration (hours)	10.1 (.8)	10.1 (.9)	10.1 (.8)
Social Jetlag (hours)	.7 (.6)	.8 (.5)	.7 (.6)
Sleep Disturbances	40.4 (6.0)	40.8 (6.5)	40.0 (5.4)

4.2.4 Physical [In]activity and Sedentary Behaviour

Physical [in]activity and sedentary behaviour were estimated using the YPAQ. To determine how many minutes a day each participant was active and sedentary, participants and their caregiver were asked to jointly complete the 47-item YPAQ (see Appendix A). The YPAQ assessed the frequency, duration, and type of physical activities and sedentary behaviour the participant took part in 7 days prior to data collection (Brooke et al., 2013;

Corder et al., 2009). Frequency and duration were used to calculate the total number of active and sedentary minutes on a day-to-day basis, giving each participant a daily average and weekly total of active and sedentary minutes (see Table 8). Type of activities was utilised to classify actions as active movements or sedentary behaviour. For example, playing rugby, walking to school, or skipping were considered being active, whereas reading, watching television, and doing homework were considered being sedentary.

4.2.5 Nutrition

Information on food choice was collected using the PEDALS FFQ, which is made up of 28 items (see Appendix A). The PEDALS-FFQ has been validated in this age group, and shows acceptable reliability and validity (Saeedi et al., 2016). In this study, these 28 items were aggregated into 21 groups, and PCA was conducted to identify components/patterns from these 21 food groups (Davison et al., 2017; Saeedi et al., 2016). A statistical data reduction method, PCA restructures large data samples into new combined variables called principal components (T. Davies & Fearn, 2004). The principal components account for variation in the sample, enabling the dietary data to be captured with fewer variables (see section 3.5.1 for additional information). Determining the number of components/patterns to be retained was based on the eigenvalues > 1 (see Appendix E), identification of the elbow in the scree plot (see Figure 12), and the interpretability of factors within components/patterns (Schulze et al., 2003). Visual inspection of the scree plot is a more accurate method to determine the number of components/patterns to be retained than relying on eigenvalues of more than one (Schulze et al., 2003). As Figure 12 shows, three dietary components/patterns are identifiable: Processed Food, Fruit and Vegetables, and Breakfast Food.

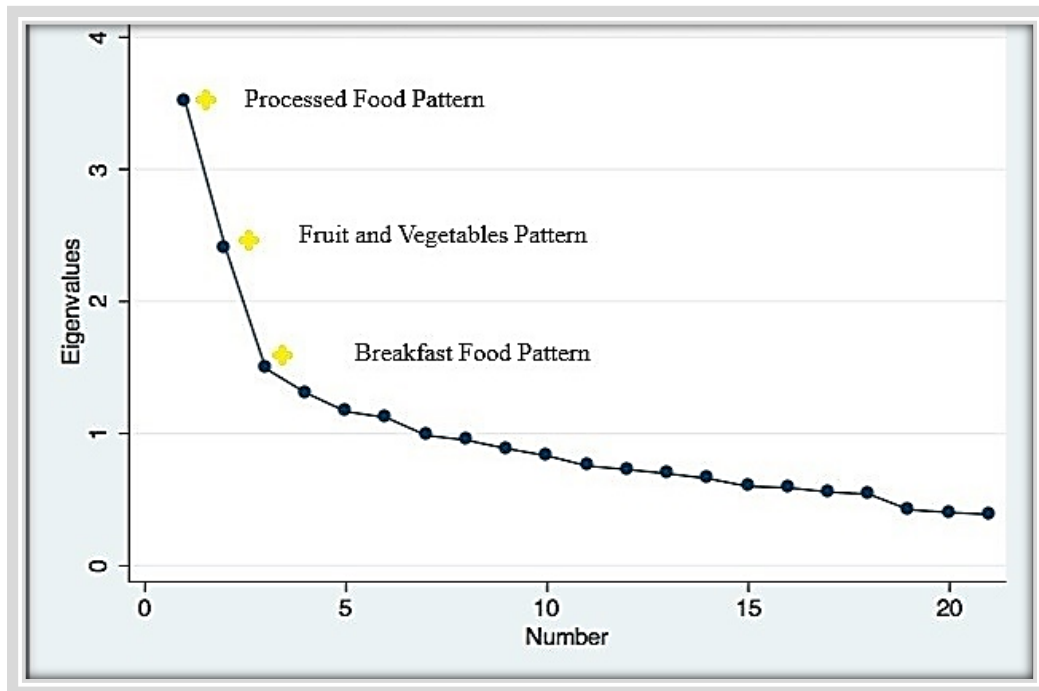


Figure 12. Scree plot of eigenvalues of the 21 food groups from the principal component analysis (PCA).

4.2.6 Sleep

Sleep duration, social jetlag, and sleep disturbances were recorded to analyse quality of sleep. To determine average sleep duration, the participant’s caregiver(s) was asked to note what time their child usually went to bed and what time they usually got up on both school days and weekend days. Single items of habitual school/weekday sleep show reasonable concurrent validity with actigraphy and diary data (Wolfson et al., 2003). Average sleep duration was calculated using a ratio of 5 weekdays to 2 weekend days. Social jetlag was calculated as the absolute difference between the midpoints of sleep on weekdays versus weekend days (Roenneberg et al., 2012). Social jetlag measures the difference between the circadian and social clocks, which could lead to sleep deprivation. Additionally, the circadian clock also regulates energy homeostasis and its interruption (in relation to social jetlag) could contribute to weight-related complications (Roenneberg et

al., 2012). Sleep disturbances were recorded using the 33-item CSHQ, which demonstrates adequate internal consistency, acceptable test-retest reliability, and discriminant validity (see Appendix A; Owens et al., 2000). The 33 questions were answered on a 7-point Likert scale from 7 (*always*) to 0 (*never*), with higher scores indicative of greater sleep disturbance. The CSHQ includes eight subscales that align with the key sleep complaints relevant for this age group: bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, night waking, parasomnias, sleep-disordered breathing, and daytime sleepiness. A Total Sleep Disturbances score was calculated as the sum of all CSHQ scored questions, with a potential range of 33 to 99. A Total Sleep Disturbances score > 41 was used to indicate a paediatric sleep disorder, as this cutoff point has been shown to accurately identify 80% of children with a clinically diagnosed sleep disorder. For this study, only the Total Sleep Disturbances score was analysed (Owens et al., 2000).

4.3 Statistical Analysis

Statistical analyses were performed using Statistical Package for Social Sciences Version 22 (SPSS). Regression analyses were performed using Gaussian family generalised estimating equations with robust standard errors to allow for the clustering of data among students attending the same schools (Goldstein, 2010) and to ensure results are representative of the population in the sampled area. Three separate regression models were used (see Table 9). Model 1 included univariate analyses that used one independent variable (lifestyle factors: $\dot{V}O_{2max}$, sleep duration, sleep disturbances, social jetlag, Processed Food pattern, Fruit and Vegetables pattern, and Breakfast Food pattern) and one dependent variable (body fatness indicators: body fat percentage, FMI, BMI, WHR). Model 2 included all independent variables and one dependent variable. Model 3 comprised Model 2 with further adjustment for sex, ethnicity, age, and school decile. All regression

models were assessed by examination of the model residuals plotted against their normal scores.

4.4 Results

Of the 392 participants who took part in the study, only 339 participants (see data description in Tables 7 and 8) had complete data sets, and only complete data sets were included in the analyses. Each model was performed as a linear quadratic and cubic analysis attempting to identify linear status among measuring variables. As demonstrated in Figure 13, $\dot{V}O_{2\max}$ has a nonlinear relationship with body fat percentage.

4.4.1 Univariate Models

The univariate outcomes (Model 1) are shown in Table 9. Initially, each independent lifestyle factor was analysed for association with each body fatness indicator. Those results indicated physical activity, sedentary behaviour, and handgrip strength did not associate strongly with each body fatness indicator. As a result, those factors were excluded from Table 9 and from multivariate analysis as only independent factors that univariately associated with body fatness indicators were included.

4.4.2 Multivariate Models

Multivariate analyses (Models 2 and 3) are also shown in Table 9. Multivariate analysis adjusted for school decile in Model 2, and age, sex, ethnicity, and school decile in Model 3. When adjusted for potential confounders in Model 3: (a) $\dot{V}O_{2\max}$, Fruit and Vegetables pattern and Breakfast Food pattern associated with body fat percentage ($\beta = -11.34$, $.44$ and $-.58$, respectively; all $p < 0.05$), and (b) $\dot{V}O_{2\max}$ was the only factor associated with FMI ($\beta = -2.45$; $p < .001$), BMI ($\beta = -3.54$; $p < .001$) and WHR ($\beta = .03$; $p \leq 0.01$).

Table 9

Linear Association Between Body Fatness and Lifestyle Factors

	Multivariate											
	Model 1 (Univariate)				Model 2				Model 3			
	β	LCI	UCI	<i>p</i>	β	LCI	UCI	<i>p</i>	β	LCI	UCI	<i>p</i>
Body Fat (%)												
$\dot{V}O_{2\max}$ (ml/kg/min)	-1.599	-1.746	-1.452	<0.001	-11.34	-13.22	-9.462	0.000	-11.34	-13.85	-8.838	0.000
$\dot{V}O_{2\max}$ Poly (ml/kg/min)	-0.018	-0.020	-0.016	0.000	0.112	0.089	0.135	0.000	0.112	0.083	0.141	0.000
Sleep Duration (hours)	-0.538	-1.730	0.654	0.375	0.351	-0.181	0.883	0.196	0.351	-0.368	1.070	0.339
Sleep Disturbances	0.185	0.018	0.352	0.030	0.039	-0.037	0.114	0.314	0.039	-0.062	0.140	0.452
Social Jetlag (hours)	3.123	1.443	4.802	0.000	0.217	-0.261	0.695	0.374	0.217	-0.950	1.384	0.716
Processed Food	0.616	0.077	1.154	0.025	0.175	-0.031	0.380	0.096	0.175	-0.107	0.456	0.224
Fruit & Vegetables	-0.780	-1.416	-0.144	0.016	0.440	0.124	0.756	0.006	0.440	0.058	0.822	0.024
Breakfast Food	-1.220	-2.018	-0.422	0.003	-0.581	-0.890	-0.271	0.000	-0.581	-1.108	-0.053	0.031
Fat Mass Index (fat mass/m²)												
$\dot{V}O_{2\max}$ (ml/kg/min)	-0.390	-0.429	-0.351	0.000	-3.590	-4.315	-2.866	0.000	-3.590	-4.252	-2.929	0.000
$\dot{V}O_{2\max}$ Poly (ml/kg/min)	-0.004	-0.005	-0.004	0.000	0.037	0.029	0.045	0.000	0.037	0.029	0.045	0.000
Sleep Duration (hours)	-0.262	-0.561	0.038	0.086	-0.012	-0.168	0.144	0.882	-0.012	-0.199	0.175	0.901
Sleep Disturbances	0.056	0.014	0.098	0.009	0.017	-0.007	0.041	0.162	0.017	-0.010	0.043	0.210
Social Jetlag (hours)	0.816	0.393	1.230	0.000	0.033	-0.101	0.168	0.626	0.033	-0.238	0.305	0.809
Processed Food	0.178	0.042	0.313	0.010	0.050	0.049	0.149	0.323	0.050	-0.032	0.132	0.231
Fruit & Vegetables	-0.238	-0.397	-0.078	0.004	0.066	-0.016	0.145	0.116	0.066	-0.015	0.148	0.110
Breakfast Food	-0.274	-0.475	-0.072	0.008	-0.113	-0.185	-0.041	0.002	-0.113	-0.249	0.023	0.103

Note. Model 1: Univariate model containing one dependent and one independent variable; Model 2: Model containing $\dot{V}O_{2\max}$, sleep duration, sleep disturbances, social jetlag, Processed Food pattern, Fruit and Vegetables pattern, Breakfast Food pattern, and one dependent variable; Model 3: Model 2 with further adjustments for sex, ethnicity, age, and decile.

	Multivariate											
	Model 1 (univariate)				Model 2				Model 3			
	β	LCI	UCI	<i>p</i>	β	LCI	UCI	<i>p</i>	β	LCI	UCI	<i>p</i>
Body Mass Index (kg/m²)												
$\dot{V}O_{2\max}$ (ml/kg/min)	-0.352	-0.418	-0.285	0.000	-3.538	-4.828	-2.249	0.000	-3.538	-5.152	-1.924	0.000
$\dot{V}O_{2\max}$ Poly (ml/kg/min)	-0.004	-0.005	-0.003	0.000	0.037	0.023	0.051	0.000	0.037	0.019	0.055	0.000
Sleep Duration (hours)	-0.488	-0.892	-0.085	0.018	-0.209	-0.583	0.166	0.274	-0.209	-0.589	0.172	0.282
Sleep Disturbance	0.067	0.011	0.124	0.019	0.026	-0.024	0.076	0.315	0.026	-0.019	0.070	0.262
Social Jetlag (hours)	0.972	0.398	1.547	0.001	0.250	-0.218	0.717	0.295	0.250	-0.305	0.805	0.378
Processed Food	0.164	-0.019	0.348	0.079	0.020	-0.251	0.290	0.887	0.020	-0.235	0.274	0.881
Fruit & Vegetables	-0.327	-0.542	-0.111	0.003	-0.032	-0.151	0.087	0.599	-0.032	-0.203	0.139	0.714
Breakfast Food	-0.188	-0.463	0.086	0.179	-0.050	-0.274	0.174	0.661	-0.050	-0.298	0.198	0.692
Waist-To-Hip Ratio												
$\dot{V}O_{2\max}$ (ml/kg/min)	-0.003	-0.004	-0.002	0.000	-0.033	-0.054	-0.012	0.002	-0.033	-0.059	-0.007	0.013
$\dot{V}O_{2\max}$ Poly (ml/kg/min)	0.000	0.000	0.000	0.000	0.000	0.000	0.001	0.003	0.000	0.000	0.001	0.016
Sleep Duration (hours)	-0.008	-0.015	-0.001	0.025	-0.006	-0.013	0.000	0.060	-0.006	-0.013	0.001	0.072
Sleep Disturbances	0.000	-0.001	0.001	0.654	0.000	-0.001	0.000	0.467	0.000	-0.001	0.001	0.605
Social Jetlag (hours)	0.013	0.004	0.023	0.008	0.008	0.002	0.015	0.008	0.008	-0.003	0.019	0.135
Processed Food	0.001	-0.002	0.004	0.556	0.000	-0.004	0.003	0.796	0.000	-0.004	0.003	0.777
Fruit & Vegetables	-0.002	-0.006	0.001	0.204	0.000	-0.004	0.004	0.947	0.000	-0.004	0.004	0.947
Breakfast Food	-0.002	-0.006	0.003	0.519	-0.001	-0.007	0.006	0.864	-0.001	-0.006	0.005	0.842

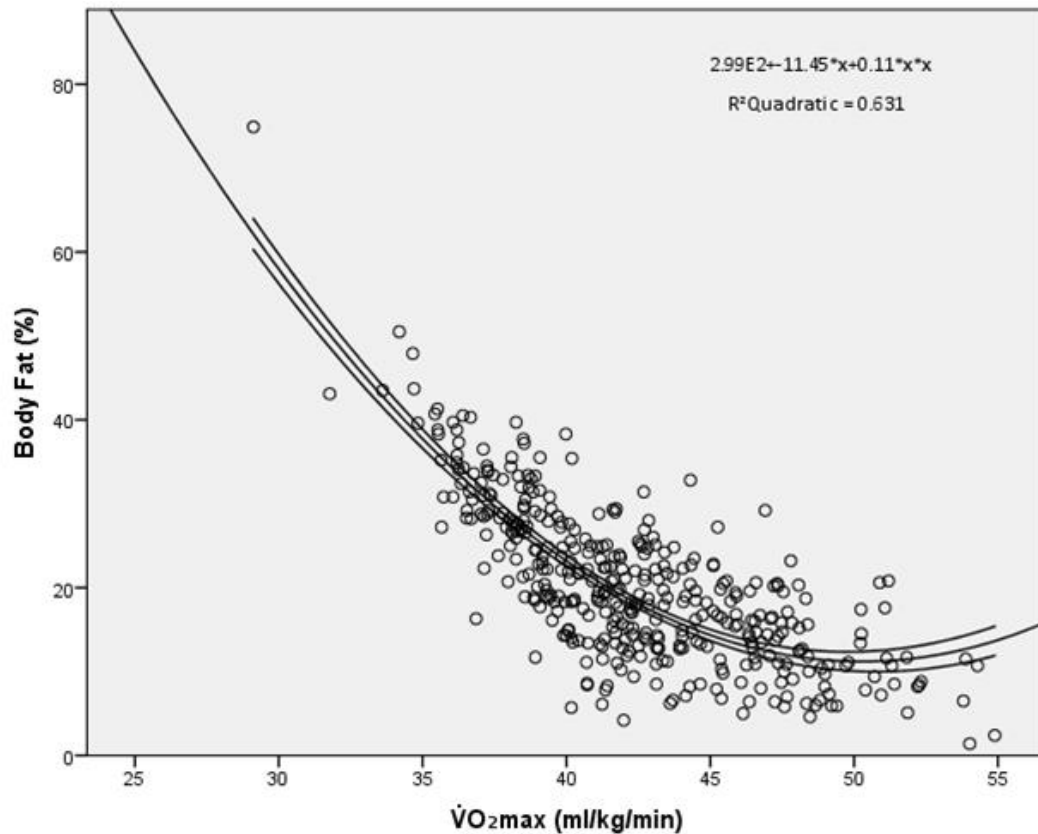


Figure 13. $\dot{V}O_{2\max}$ and body fat percentage linear quadratic and cubic analysis.

4.5 Discussion

The purpose of this study was to investigate the associations among physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with body fatness in preadolescent children, and to the best of my knowledge, no known study has assessed these associations simultaneously in this population. Following adjustments for potential confounders, Fruit and Vegetables pattern and Breakfast Food pattern independently associated with body fat percentage, while $\dot{V}O_{2\max}$ was associated with all four body fatness indicators (see Table 9, Model 3). These findings indicated that cardiorespiratory fitness is likely most significant to body fatness, while dietary intake also plays a role. Collectively,

both lifestyle factors could be imperative health policy targets to focus on childhood obesity in preadolescent children.

4.5.1 Comparison With Other Studies

Of the lifestyle factors measured, regression analysis revealed only $\dot{V}O_{2\max}$ associated solely with all four estimates of body fatness (body fat percentage, FMI, BMI, and WHR), establishing cardiorespiratory fitness as the strongest predictor of obesity in this study (see Table 9, Model 3). A one-unit (ml/kg/min) decrease in $\dot{V}O_{2\max}$ was associated with an 11.34% increase in body fat, a 3.59 (kg/m²) increase in FMI, a 3.54 (kg/m²) increase in BMI, and a 0.03 (ratio) increase in WHR. However, it should be recognised that $\dot{V}O_{2\max}$ was nonlinearly associated with body fat percentage; beyond ~42 ml/kg/min, an increase in $\dot{V}O_{2\max}$ does not correspond with change in body fat percentage (see Figure 13). Collectively, children and adolescents with low levels of cardiorespiratory fitness are at greater risk of myocardial infarction, CVD, and sustaining lower than average physical fitness levels in adulthood (Högstrom, Nördstrom, & Nordström, 2014; Ruiz et al., 2016). This suggests that it may be particularly important to focus on improving cardiorespiratory fitness in children with a $\dot{V}O_{2\max}$ below 42 ml/kg/min.

An individual's peak cardiorespiratory fitness level exhibits the complete capacity of the cardiovascular and respiratory systems (Ortega et al., 2008; Ruiz et al., 2016). While studies in children are limited, findings for adults suggest that cardiorespiratory fitness is possibly a more significant predictor of death compared to other major risk factors including smoking, obesity, hypertension, hyperlipidaemia, and hyperglycaemia (Ross et al., 2016). Additionally, among adults, cardiorespiratory fitness has been shown to reduce the risk of morbidity and mortality among slightly overweight, overweight, and obese

individuals (LaMonte et al., 2005; Schmidt et al., 2016). High levels of fitness increase HR; therefore, force of contraction increases while exercising (D. L. Smith & Fernhall, 2011). This enhanced cardiac output improves the rate of circulation and increased blood flow improves shear stress; enhanced shear stress leads to increased nitric oxide production and bioavailability, which help fend off the inflammatory process (Di Francescomarino et al., 2009; Sherman, 2000). Additionally, consistent exercise and improved fitness enhance sensitivity of the liver, skeletal muscle, and adipose tissue to the actions of insulin (Froberg & Andersen, 2005; Press et al., 2003). This is significant because when these tissues become less responsive or resistant to insulin, the progression of inflammation is triggered, and an inflammatory response is a pathological mechanism associated with obesity and additional cardiometabolic complications (Hotamisligil, 2003; Saltiel & Kahn, 2001; Vaulont et al., 2000). In children, there is some evidence of a correlation between lower cardiorespiratory fitness and cardiometabolic risk factors such as obesity (Ortega et al., 2008). However, further study is warranted to continue investigating cardiorespiratory fitness in preadolescent children.

In this study, multivariate analysis demonstrated no association of dietary patterns with BMI, FMI, and WHR. However, there was an association of dietary intake (Fruit and Vegetables pattern and Breakfast Food pattern) with body fat percentage in preadolescent children (see Table 9, Model 3). Dietary intake provides the body with the nutrients required to function properly; however, when there is an imbalance and/or deficiency among the nutrients consumed, the outcome could have detrimental effects on the cardiovascular system (Kkeveetil et al., 2016; WHO, 2015). Carbohydrates are the primary provider of energy to the body; however, consuming too many simple carbohydrates can cause glucose levels to increase drastically, which could lead to impaired glucose tolerance

(Hotamisligil, 2003; Saltiel & Kahn, 2001; Vaulont et al., 2000). Continual high levels of blood glucose can reduce the elasticity of the blood vessels and ultimately trigger inflammation (Conget & Giménez, 2009; Kuipers et al., 2011). Proteins function as energy sources, enzymes, hormones, immune system support, and repairer of tissues (see Figure 6; B. I. Campbell & Spano, 2011; Damodaran, 2007). However, intake of too much complete protein increases the levels of LDL-C in the body, affecting the balance of lipids created and lipids used, which could trigger the inflammatory process (Clifton, 2011; Richter et al., 2015; Trumbo et al., 2002). The body uses lipids for various tasks (e.g., as messengers, storers, transporters, and insulators), although too much circulating LDL-C leads to an accumulation in the blood vessel intima, which initiates the progression of atherosclerosis (Navarro et al., 2017; Ross & Glomset, 1976; Sorensen et al., 1994; Stoner, Lambrick, et al., 2014; Tedgui & Mallat, 1999). Fruits and vegetables contain an assortment of micro and macronutrients that provide the body with energy and nutrients such as fibre (Reddy & Katan, 2004; Slavin & Lloyd, 2012). Additionally, many breakfast foods such as muesli, Wheat-Bix, oats, bran muffin, and whole grain cereal contain fibre. Regular fibre intake has been shown to have beneficial effects on cardiometabolic risk, such as improving glucose levels and reducing LDL-C levels, blood pressure, and inflammation (Lie et al., 2018; M. A. Pereira & Liu, 2003; Slavin & Lloyd, 2012). Additionally, fruits and vegetables provide the body with vitamins and minerals and some are good sources of antioxidants (M. A. Pereira & Liu, 2003; Slavin & Lloyd, 2012). An adequate amount of antioxidants in the body is essential for the balance with prooxidants because an imbalance can lead to oxidative stress and trigger inflammation (Davignon & Ganz, 2004; Galley & Webster, 2004; Slavin & Lloyd, 2012).

Although univariate analysis revealed that all three sleep variables (sleep duration, sleep disturbances, and social jetlag) showed a relationship with at least one of the body fatness measures (body fat percentage, FMI, BMI, and WHR; see Table 9, Model 1), multivariate analysis revealed that none of the sleep indicators associated with body fatness (see Table 9, Model 3). However, this does not suggest that quality of sleep is insignificant to body fatness as it may be linked to other lifestyle behaviours, including nutritional behaviour. Finally, the analysis revealed none of the body fat measures associated strongly with muscular fitness, physical [in]activity, or sedentary behaviour.

4.5.2 Limitations and Strengths of This Study

This study had several potential methodological and physiological limitations, thus existing results were explained based on these considerations. First, this was a cross-sectional study, making it difficult to determine causality; further longitudinal research is required to better determine the associations among physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with body fatness. This initial stage is necessary prior to allotting time and resources into costly trials. Second, data collection was conducted at primary schools in group settings; therefore, the noise, facility limitations (e.g., secluded space, tinted windows, and private room), distractions, interruptions, and weather could not be controlled or measured (Gortmaker et al., 1999). Consequently, those uncontrollable factors could have influenced the blood pressure, HR, body temperature, focus, and attention span of the participants (Gortmaker et al., 1999). Lastly, this study investigated New Zealand-based preadolescents, which may not simulate preadolescents in other nations. For instance, dietary intake (Mediterranean diet vs. Western diet), types of physical activities (rugby vs. American football), and social ecological factors may affect a child's physiology differently, so further research is required to determine if these findings

would be consistently associated with preadolescent outcomes globally. A considerable strength of this study was the large and diverse group of New Zealand-based preadolescents included in the research from various parts of the country. Similar studies have shown comparable results to this study, but most of those studies analysed each factor independently with body fatness (Duncan, Schofield, Duncan, & Rush, 2008; Kuzik & Carson, 2016; Moschonis et al., 2013; Skidmore et al., 2013; Stratton et al., 2007; Williams et al., 2013).

4.5.3 Research Implications

The current findings identified cardiorespiratory fitness as the strongest indicator of body fatness in preadolescent children. All four body fatness measures associated with cardiorespiratory fitness. In adults, physical activity explains about 30% of $\dot{V}O_{2max}$, compared to genetics controlling 70% of $\dot{V}O_{2max}$ (Bouchard, Rankinen, & Timmons, 2011). In comparison, in children, physical activity only explains a moderate amount of $\dot{V}O_{2max}$, meaning the exclusive benefits of being physically active on cardiorespiratory fitness are less certain in preadolescents (N. Armstrong, 2013). Overall, genetics, maturation stage, activeness, and inactiveness all should be considered when examining $\dot{V}O_{2max}$. Therefore, preventive strategies that target cardiorespiratory fitness should be at the forefront of paediatric health guidelines. Further study is warranted to continue investigating cardiorespiratory fitness, body fatness, and other modifiable lifestyle factors in preadolescent children.

4.6 Conclusion

To the best of my knowledge, this was the first study to simultaneously investigate the associations among physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with body fatness in preadolescent children. The preliminary findings suggest that cardiorespiratory fitness correlated most strongly with body fatness (all four body fatness estimators); therefore, high-intensity activities and sports should be at the forefront of paediatric health guidelines. Devoting resources towards prevention and intervention strategies that target improving and maintaining cardiorespiratory fitness could be one of the essential components for addressing the childhood obesity epidemic, and subsequently making an impact on the deteriorating health and wellness of preadolescent children.

CHAPTER 5—RESEARCH OBJECTIVE 2

Associations With Cardiometabolic Health in Preadolescent Children: Fatness, Fitness, Physical [In]activity, Sedentary Behaviour, Nutrition, and Sleep

5.0 Background

Even though CVD is typically associated with middle or old age, the atherosclerotic process often initiates early in childhood and is occurring at an increasing rate (Stoner, Lambrick, et al., 2014; Trandafir et al., 2017). The early onset of atherosclerosis has been linked to cardiometabolic risk factors. Preceding cardiometabolic risk factors are modifiable lifestyle factors, including but not limited to: excessive body fatness (Bridger, 2009; Trandafir et al., 2017), lower than ideal physical fitness (Cohen et al., 2011; Czyż et al., 2017; Stratton et al., 2007), insufficient daily physical activity (O. Ali et al., 2014; Dumuid et al., 2018; WHO, 2017d), sedentary behaviour (Carson, Hunter, et al., 2016; Carson, Tremblay, et al., 2016; Griffiths et al., 2016; Healy & Owen, 2010), unhealthy diet (Harrex et al., 2017; WHO, 2015, 2017a), and inadequate sleep (Davison et al., 2017; Sayin & Buyukinan, 2016; Skidmore et al., 2013), which all have been linked to cardiometabolic complications such as obesity, hypertension, dyslipidaemia, and insulin resistance (Trandafir et al., 2017; WHO, 2017a, 2017b; for further information, see section 2.0.1). These cardiometabolic complications are risk factors that may lead to NCDs such as CVD (WHO, 2017a, 2017b). To date, no known study of preadolescent children has undertaken a comprehensive assessment of the simultaneous associations between these lifestyle factors with cardiometabolic health. Because uncertainty remains as to which lifestyle factor(s) associate more strongly with cardiometabolic risk factors in children, it is difficult to

determine the ideal public health intervention and prevention strategy needed to improve cardiometabolic health in children.

Therefore, the purpose of this cross-sectional study was to examine simultaneously the associations among body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with cardiometabolic health in preadolescent children. Findings from this study will contribute to the body of knowledge informing health professionals and health policy writers regarding children's health and wellness, potentially making a positive impact on the deteriorating health of New Zealand children.

5.1 Methodology

This observational study was carried out in accordance with STROBE guidelines (Von Elm et al., 2014). The methodology was prospectively detailed in Castro et al. (2014) and was carried out simultaneous to investigation of association among physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with body fatness as described in section 4.1.

5.1.1 Recruitment and Participants

Children aged 8 to 10 years were recruited from schools in three major cities across New Zealand (Wellington, Christchurch, and Dunedin) between April 2015 and April 2016 (see Figure 14). Within each sample site, schools from the region were stratified by decile rating and schools from each stratification were randomly invited to participate. Until 2017, New Zealand public-funded schools were classified on a 10-point decile rating based on the predominant socioeconomic status of the attending students. The MOE (2016) website was used to determine schools' decile rating. Schools were categorised by decile ratings ranging from low (Deciles 1 to 5) to high (Deciles 6 to 10; see Table 10; MOE, 2016). Decile 1 schools comprised 10% of schools with the highest proportion of students from low

socioeconomic status communities whereas Decile 10 schools comprised 10% of the schools with the lowest proportion of students from low socioeconomic status communities (MOE, 2016).

At invited schools, all children were eligible to participate unless they had an orthopaedic injury or surgery that had prohibited full function within the previous 4 weeks or were currently prescribed any cardiovascular medications. Parental or guardian consent and child assent were obtained prior to participation, in accordance with the requirements of the New Zealand HDEC (HDEC:14/CEN/83).

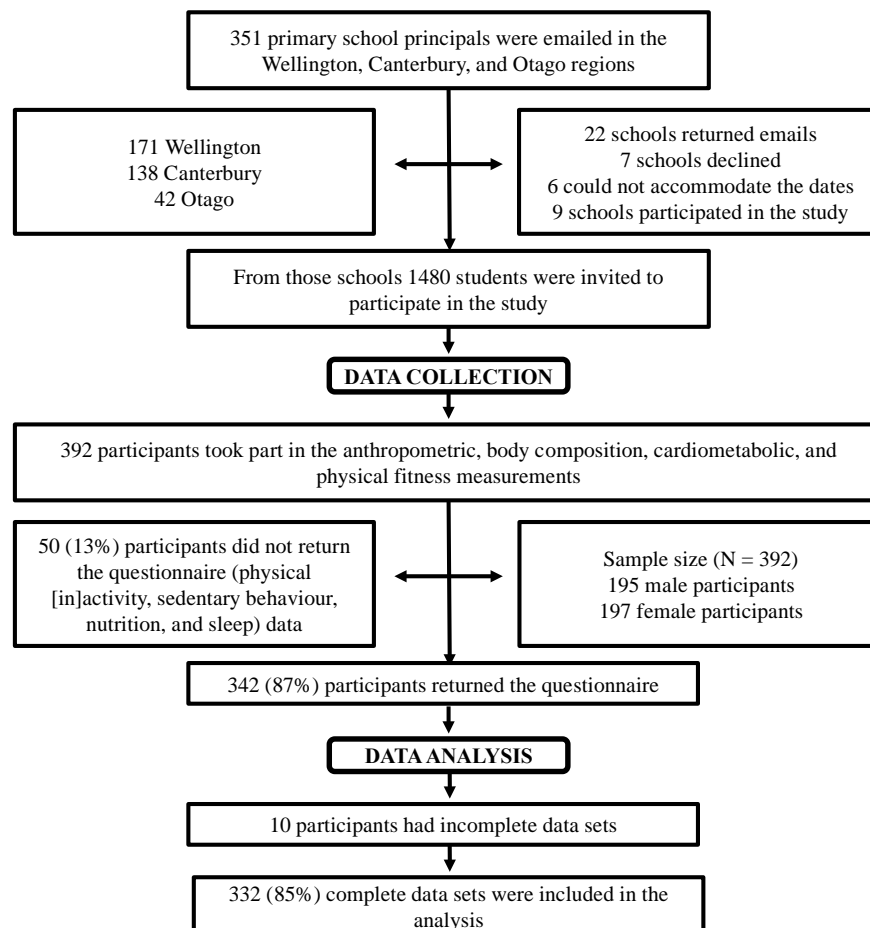


Figure 14. Participant recruitment.

Table 10

Participant Data Description: Categorical Variables

Categorical Variables	All	Female	Male
	<i>n</i> / 392 (%)	<i>n</i> / 197 (%)	<i>n</i> / 195 (%)
Ethnicity			
New Zealand European	279 (71)	135 (69)	144 (74)
Māori	37 (9)	21 (11)	16 (8)
Pacifics	22 (6)	12 (6)	10 (5)
Not Recorded	54 (14)	29 (15)	25 (13)
School Year			
4	82 (21)	45 (23)	37 (19)
5	114 (29)	55 (28)	59 (30)
6	127 (32)	62 (31)	65 (33)
7	69 (18)	35 (18)	34 (17)
School Decile			
Low (< 5)	211 (54)	106 (54)	105 (54)
High (> 5)	181 (46)	91 (46)	90 (46)
Obesity Status			
Overweight	122 (31)	58 (30)	64 (33)
Nonoverweight	270 (69)	139 (70)	131 (67)
Cardiorespiratory Fitness (ml/kg/min)			
$\dot{V}O_{2\max}$ Low	111 (28)	46 (23)	65 (33)
$\dot{V}O_{2\max}$ High	277 (71)	150 (76)	127 (66)
Not Recorded	4 (1)	1 (1)	3 (1)

5.1.2 Study Design

The methodology for this study is fully outlined in the PACMAC study protocol (see Chapter 3) and was simultaneously used to investigate the research objective described in Chapter 4. Data for this study were collected as part of a larger cross-sectional study examining PACMAC. The trial was prospectively registered with the Australia and New Zealand Clinical Trial Registry (ACTRN12614000433606), and details of the wider study have been previously published (Castro et al., 2014). Only study details relevant to this thesis will be presented.

At each school, data collection began on Monday and concluded on the following Friday with each individual testing session taking between 30-45 minutes per participant; up to 12 participants were measured each day. During the study week, cardiometabolic health, adiposity, and physical fitness (for further information, see Chapter 2 sections 2.02, 2.1.2, and 2.2.2) were assessed between the hours of 09:00 a.m. and 12:00 p.m., with participants having (a) refrained from exercise for the previous 24 hours, (b) fasted for at least 3 hours, and (c) ensured adequate hydration. Within 7 days of the assessments described above, physical [in]activity, dietary patterns, sleep habits, and demographic data were collected using a questionnaire (see Appendix A). The questionnaires were completed at home jointly by the primary caregiver and participant using an online survey (Lime Survey, open source) where available. If this was not possible, a paper copy was provided, which was subsequently entered into Lime Survey by the researcher. This study format was replicated at each school that participated in the study. Only participants with complete data sets were included in the data analyses (see section 3.3.4 for details on data cleaning).

5.2 Data Collection and Analysis

5.2.1 Demographics

Demographic information was self-reported by the participant's caregiver (see Tables 10 and 11, Appendix A). Demographic data collected included participants' date of birth, age, sex, ethnicity, and school address. The MOE (2016) website was used to determine school decile rating, as described in section 5.1.1. The ethnicity data were applied to categorise participants into four classification groups: (a) New Zealand European and Others (NZEO), (b) Māori, (c) Pacifics, and (d) not specified. These classifications were determined by using the New Zealand in Profile 2015 prioritised ethnicity protocol (Statistics New Zealand, 2015).

5.2.2 Cardiometabolic Health

Eleven dependent variables were recorded to estimate cardiometabolic health utilising PWA and cardiometabolic biochemical markers: SBP, DBP, CBP, HR, AIx, TC, HDL-C, LDL-C, TG, serum glucose, and HbA1c (see section 3.3 and Appendix B for further information on protocols).

Peripheral blood pressures, CBP, HR, and AIx were recorded utilising the BP+ device (Uscom, Sydney, Australia). Following 20 minutes of undisturbed rest, oscillometric pressure waveforms were recorded by a single operator on the left upper arm, following standard manufacturer guidelines (Stoner et al., 2013). Each measurement cycle lasted approximately 40 seconds, consisting of a brachial blood pressure recording and then a 10 second supra-systolic recording (Stoner et al., 2017). A corresponding aortic pressure waveform was generated using a validated transfer function, from which CBP was estimated (Butlin, Qasem, & Avolio, 2012). Augmentation pressure was calculated from the suprasystolic waveform using the formula: $AIx = (P_3 - P_0) / (P_1 - P_0)$, where P_0 denotes the pressure at the onset of the pulse, P_1 the peak pressure of the incident wave, and P_3 the peak pressure of the reflective wave. This index describes the relative height of the reflected pressure wave when compared to the incident waveform. Only recordings with a high signal quality were accepted (signal to noise > 3dB), and two high signal quality measurements were taken within a 5-minute interval. A third recording was taken, and the closest two recordings were averaged if blood pressures differed by > 5 mmHg or AIx > 4% (Stoner, Lambrick, et al., 2014; see Table 11).

Cardiometabolic biochemical markers were recorded using a standard finger prick procedure (see Table 11). Standard operating protocol for finger prick blood samples were followed to extract capillary blood and assess fasting TC, HDL-C, LDL-C, TG, serum

glucose (CardioChek PA, PTS Diagnostics, IN, USA; Parikh et al., 2009) and HbA1c (A1CNow+, PTS Diagnostics, IN, USA; Barrett et al., 2011).

Table 11

Participant Data Description: Continuous Variables

Continuous Variables	Mean (SD)		
	All <i>n</i> = 392	Female <i>n</i> = 197	Male <i>n</i> = 195
Age (years)	9.5 (1.1)	9.5 (1.2)	9.6 (1.0)
Body Fatness			
Weight (kg)	34.7 (9.2)	34.7 (9.2)	34.7 (9.3)
Body Fat (%)	20.2 (9.4)	22.0 (9.4)	18.6 (9.1)
Fat Mass Index (fat mass/m ²)	2.7 (1.7)	2.9 (1.6)	2.5 (1.7)
Body Mass Index (kg/m ²)	17.9 (3.3)	17.9 (3.1)	17.8 (3.4)
Waist-to-Hip Ratio (w/h)	.84 (.06)	.83 (.06)	.84 (.05)
Physical Activity & Sedentary Behaviour			
Physical Activity (minutes)	164 (135)	152 (130)	175 (140)
Sedentary Behaviour (minutes)	284 (209)	286 (219)	282 (199)
Sleep			
Average Sleep Duration (hours)	10.1 (.8)	10.1 (.9)	10.1 (.8)
Social Jetlag (hours)	.7 (.6)	.8 (.5)	.7 (.6)
Sleep Disturbances	40.4 (6.0)	40.8 (6.5)	40.0 (5.4)
Cardiometabolic health			
Systolic Blood Pressure (mmHg)	100.8 (7.9)	101.1 (8.2)	100.6 (7.6)
Diastolic Blood Pressure (mmHg)	61.7 (6.3)	62.0 (6.5)	61.4 (6.1)
Central Blood Pressure (mmHg)	93.2 (9.0)	93.2 (10.6)	93.3 (7.1)
Augmentation Index (%)	55.9 (15.9)	56.5 (16.7)	55.5(15.2)
Heart Rate (bpm)	74.8 (11.4)	77.5 (12.1)	72.0 (9.9)
Fasting Blood Glucose (mmol/L)	5.0 (0.4)	5.0 (0.4)	5.1 (0.4)
Glycosylated Haemoglobin (%)	5.1 (0.3)	5.1 (0.3)	5.1 (0.3)
Total Cholesterol (mmol/L)	3.6 (0.6)	3.6 (0.6)	3.5 (0.6)
HDL Cholesterol (mmol/L)	1.5 (0.4)	1.4 (0.3)	1.5 (0.4)
LDL Cholesterol (mmol/L)	1.9 (0.5)	1.9 (0.5)	1.8 (0.5)
Triglycerides (mmol/L)	0.9 (0.4)	0.9 (0.4)	0.8 (0.5)

5.2.3 Adiposity and Anthropometrics

Five dependent variables were recorded: body fat (%), fat mass (kg), FMI (fat mass/m²), WHR, and BMI (kg/m²). Multifrequency BIA (BodyStat Quadscan 4000, Isle of Man, UK) was used to measure body fat (%) and fat mass (kg). The instrument was

calibrated in accordance with the manufacturer's instructions, and measurements were conducted according to standardised procedures (Kyle et al., 2004b). In accordance with manufacturer guidelines, participants drank 100 ml of water and removed any jewellery prior to the assessment. Measurements were undertaken with participants in the supine position on a nonconductive surface, with arms and legs abducted at a 30°–45° angle from the trunk to avoid medial body contact by upper and lower extremities. The FMI was calculated by dividing fat mass (kg) by height squared (m^2 ; Peltz et al., 2010; see Table 11).

To calculate the anthropometric indices (BMI and WHR), height, weight, WC, and hip circumference were measured. Height was measured to the nearest 0.1 cm with shoes and socks removed and head in the Frankfort plane using a calibrated portable stadiometer (Seca 213, Hamburg, Germany). Weight was measured to the nearest 0.1 kg (with shoes and socks removed) using a calibrated portable scale (Seca 813, Hamburg, Germany). Using nonelastic tape (Seca 203, Hamburg, Germany), WC and hip circumference were measured (also to the nearest 0.1 cm). Waist circumference was measured during mid-expiration at the midpoint between the lower costal margin and the level of the anterior superior iliac crest, and hip circumference was measured around the widest portion of the buttocks (WHO, 2011). For each assessment, participants were measured twice, and the highest value was recorded (unless the two measurements were more than 0.5 cm apart; then a third measurement was taken and the average of the three was recorded). Age- and sex-specific BMI z scores were calculated using the 2007 WHO growth guidelines (De Onis et al., 2007; WHO, 2018a), and BMI values (overweight and nonoverweight) were categorised using the International Obesity Task Force's sex- and age-dependent cutoff points (Cole & Lobstein, 2012; International Obesity Task Force, 2015; see Table 10).

5.2.4 Physical Fitness

Cardiorespiratory and muscular fitness were recorded using the 20-MST and handgrip strength test. The 20-MST has been found to be valid and noninvasive, requiring limited space and no special facilities, and is popular in school settings as many students can be tested simultaneously (Boreham et al., 1990; Hamlin et al., 2014; Melo et al., 2011). The 20-MST took place at 10:00 a.m. on a Friday in similar conditions as possible based on resources across schools. After a warmup, stretch, and practice run, participants were asked to run in groups of 10 between two lines set 20 metres apart (Léger & Lambert, 1982). The participants started running at a speed of 8.5 km/h⁻¹, which increased by 0.5 km/h⁻¹ every completed level; the pace was set by the audio signal emitted from a speaker (Hamlin et al., 2014). Participants were warned the first time they did not reach the line in time with the audio signal and were subsequently removed from the test if they did not reach the line for two successive shuttles or if the participant stopped voluntarily (Hamlin et al., 2014). To estimate $\dot{V}O_{2\max}$ we used the regression equation established by Hamlin (Hamlin et al., 2014), where total distance covered during the test, participant's body fat percentage, and age were used in the regression formula:

$$[\dot{V}O_{2\max} \text{ (ml/kg)} = 42.18 + (0.009 \times \text{Beep test distance in metres}) + (-0.1762 \times \text{body fat\%}) + (-0.4091 \times \text{age})]$$

The described formula has been previously validated on New Zealand children (Hamlin et al., 2014). In accordance with the Cooper Institute (2014) FitnessGram[®] cutoff points, a “healthy cardiorespiratory fitness zone” was reported if girls achieved a $\dot{V}O_{2\max}$ equal to or greater than 39 and if boys achieved equal to or greater than 42 (Howe et al., 2016; Zhu et al., 2010). A $\dot{V}O_{2\max}$ below those cutoff points was categorised as “needs

improvement fitness zone” for both sexes (Zhu et al., 2010; see Table 10). A handgrip dynamometer (Camry, EH101) was used to assess each participant’s muscular strength (Leong et al., 2015). The method is rapid, noninvasive, simple to use, inexpensive, and of minimal risk (Leong et al., 2015), which are key factors when assessing a large group of children. The participants were seated with shoulders adducted and neutrally rotated, elbow flexed to 90 degrees, and their wrist in a neutral position (between 0 and 30 degrees extension, and between 0 and 15 degrees ulnar deviation). Then the participants placed their fingers around the dynamometer handle, counted down from three, and squeezed the dynamometer handle as hard as they could for three or more seconds. Each participant was given three attempts with each hand, alternating hands, and a minute recovery time between each attempt. Isometric handgrip strength was measured in kilograms, and the best score for each hand was recorded for analysis.

5.2.5 Physical [In]activity and Sedentary Behaviour

Physical [in]activity and sedentary behaviour were estimated using the YPAQ. To determine how many minutes a day each participant was active and sedentary, participants and their caregiver were asked to jointly complete the 47-item YPAQ (see Appendix A). The YPAQ assessed the frequency, duration, and type of physical activities and sedentary behaviour the participant took part in 7 days prior to data collection (Brooke et al., 2013; Corder et al., 2009). Frequency and duration were used to calculate the total number of active and sedentary minutes on a day-to-day basis, giving each participant a daily average and weekly total of active and sedentary minutes. Type of activities was utilised to classify actions as active movements or sedentary behaviour. For example, playing rugby, walking to school, or skipping were considered being active, whereas reading, watching television, and doing homework, were considered being sedentary.

5.2.6 Nutrition

Information on food choice was collected using the PEDALS FFQ, which is made up of 28 items (see Appendix A). The PEDALS FFQ has been validated in this age group, and shows acceptable reliability and validity (Saeedi et al., 2016). In this study, these 28 items were aggregated into 21 groups, and PCA was conducted to identify components/patterns from these 21 food groups. A statistical data reduction method, PCA restructures large data samples into new combined variables called principal components (T. Davies & Fearn, 2004). The principal components account for variation in the sample, enabling the dietary data to be captured with fewer variables. Determining the number of components/patterns to be retained was based on the eigenvalues > 1 (see Appendix E), identification of the elbow in the scree plot (see Figure 15), and the interpretability of factors within components/patterns (Schulze et al., 2003). Visual inspection of the scree plot is a more accurate method to determine the number of components/patterns to be retained than relying on eigenvalues of more than one (Schulze et al., 2003). As shown in Figure 15, three dietary components/patterns are identifiable: Processed Food, Fruit and Vegetables, and Breakfast Food.

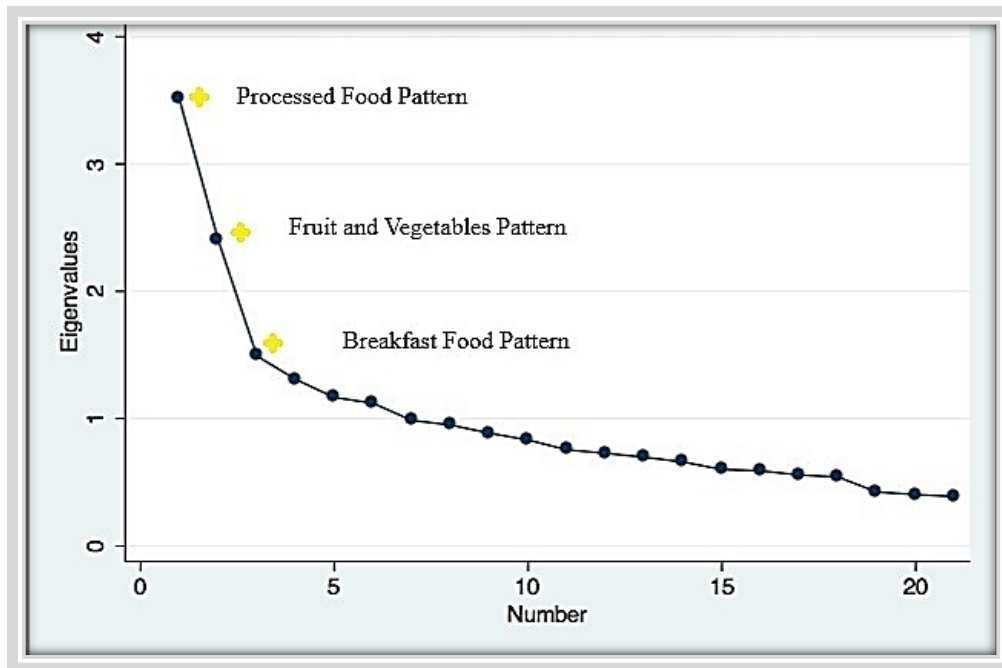


Figure 15. Scree plot of eigenvalues of the 21 food groups from principal component analysis (PCA).

5.2.7 Sleep

Three independent variables were recorded: sleep duration, social jetlag, and sleep disturbances. To determine average sleep duration, the participant’s caregiver(s) was asked to note what time their child usually went to bed and what time they usually got up on both school days and weekend days. Single items of habitual school/weekday sleep show reasonable concurrent validity with actigraphy and diary data (Wolfson et al., 2003). Average sleep duration was calculated using a ratio of 5 weekdays to 2 weekend days. Social jetlag was calculated as the absolute difference between the midpoints of sleep on weekdays versus weekend days (Roenneberg et al., 2012). Sleep disturbances were recorded using the 33-item CSHQ, which demonstrates adequate internal consistency, acceptable test–retest reliability, and discriminant validity (see Appendix A; Owens et al., 2000). The 33 questions were answered on a 7-point Likert scale from 7 (*always*) to 0

(*never*), with higher scores indicative of greater sleep disturbance. The CSHQ includes eight subscales that align with the key sleep complaints relevant for this age group: bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, night waking, parasomnias, sleep-disordered breathing, and daytime sleepiness. A Total Sleep Disturbances score was calculated as the sum of all CSHQ scored questions, with a potential range of 33 to 99. A Total Sleep Disturbances score > 41 was used to indicate a paediatric sleep disorder, as this cutoff point has been shown to accurately identify 80% of children with a clinically diagnosed sleep disorder. For this study, only the Total Sleep Disturbances score was analysed (Owens et al., 2000).

5.3 Statistical Analysis

Statistical analyses were performed using SPSS 22. Regression analyses were performed using Gaussian family generalised estimating equations with robust standard errors to allow for the clustering in data among students attending the same schools (Goldstein, 2010) and to ensure results are representative of the population in the sampled area. Three separate regression models were used (see Table 12). Model 1 included univariate analyses that used one independent variable (lifestyle factors: WHR, body fat%, $\dot{V}O_{2max}$, handgrip strength, sedentary minutes, social jetlag, Processed Food pattern, and Fruit and Vegetables pattern) and one dependent variable (cardiometabolic factors: blood pressure, cholesterol, vascular, and carbohydrate-metabolic). Model 2 included all independent variables and one dependent variable. Model 3 comprised Model 2 with further adjustment for sex, ethnicity, age, and school decile. All regression models were assessed by examination of the model residuals plotted against their normal scores.

Principal component analysis was applied to identify cardiometabolic factors from the cardiometabolic variables: SBP, DBP, CBP, TC, LDL-C, HDL-C, A1x, HR, glucose,

TG concentration, and HbA1c. Determining the number of cardiometabolic factors to be retained was based on the eigenvalues > 1 (see Table 13), the implication being that if an eigenvalue is less than one the derived dimension captures less variability in the data than any single variable. The principal components were then subjected to orthogonal varimax rotation. Factor loadings, the correlation between the derived factors and the underlying variables, were used to interpret each factor. A loading of greater than 0.40 was used to interpret the factor pattern. On these terms (described in the Results section), four factors were identified that represent blood pressure, cholesterol, vascular, and a carbohydrate-metabolic factor.

5.4 Results

Participants' data description is presented in Tables 10 and 11. Of the 392 participants who took part in the study, only 332 participants had complete data sets, and only complete data sets were included in the analyses (see section 3.3.4 for further information about data cleaning).

5.4.1 Cardiometabolic Factor Correlations and Analysis

The factor analysis is summarised in Table 13 and Figure 16. Using the minimum eigenvalue principle of greater than 1, four dimensions were retained in the factor analysis: blood pressure, cholesterol, vascular, and carbohydrate-metabolic. Collectively, the four factors explained 60% of the variance in the measured variables. The SBP, DBP, and CBP loaded positively onto the blood pressure factor. Augmentation index, glucose, and HR loaded positively onto the vascular factor. Total cholesterol, LDL-C, and HDL-C loaded positively onto the cholesterol factor. In addition, HDL-C loaded positively onto the carbohydrate-metabolic factor along with TG and HbA1c.

5.4.2 Univariate Models

The univariate outcomes (Model 1) are shown in Table 12. Initially, each independent lifestyle factor was analysed for association with each cardiometabolic factor. Those results indicated physical activity, BMI, FMI, average sleep duration, sleep disturbances, and Breakfast Food pattern did not associate strongly with cardiometabolic factors. As a result, those factors were excluded from Table 12 and from multivariate analysis as only independent factors that univariately associated with cardiometabolic factors were included.

5.4.3 Multivariate Models

Multivariate analyses (Models 2 and 3) are also shown in Table 12. Multivariate analysis adjusted for school decile in Model 2, and age, sex, ethnicity, and school decile in Model 3. When adjusted for potential confounders in Model 3: (a) body fat percentage associated with blood pressure factor ($\beta = .02$, respectively; $p < 0.05$), (b) sedentary minutes, social jetlag, and Fruit and Vegetables pattern associated with cholesterol factor ($\beta = .001$, $-.20$ and $-.08$, respectively; all $p < 0.05$), (c) sedentary minutes and Processed Food pattern associated with vascular factor ($\beta = .001$ and $.08$, respectively; both $p < 0.05$), and (d) $\dot{V}O_{2max}$, and handgrip strength associated with carbohydrate-metabolic factor ($\beta = -.08$ and $.07$, respectively; both $p < 0.001$).

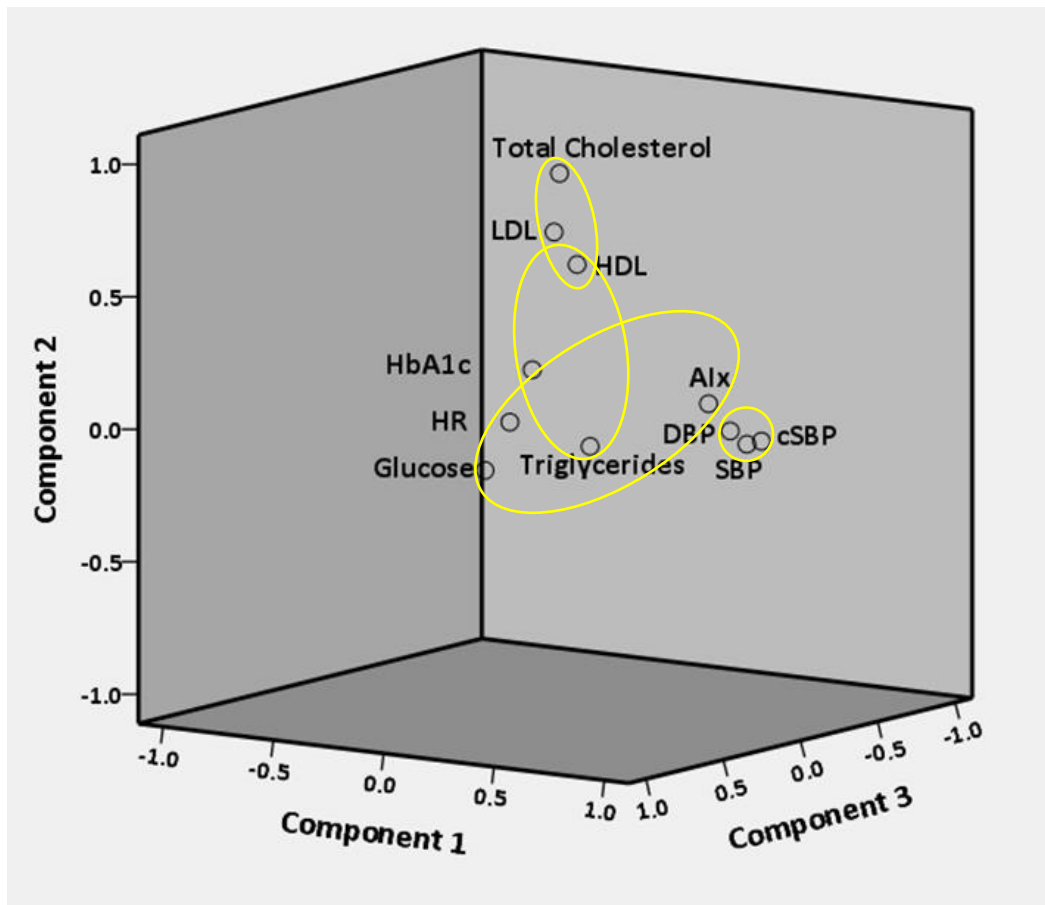


Figure 16. Component plots with factor diagrams from principal component analysis (PCA) with varimax orthogonal rotation. Augmentation index (AIx), central blood pressure (CBP), diastolic blood pressure (DBP), glycosylated haemoglobin (HbA1c), high-density lipoproteins (HDL-C), heart rate (HR), low-density lipoproteins (LDL-C), systolic blood pressure (SBP).

Table 12

Linear Association Between Cardiometabolic Health and Lifestyle Behaviours

	Multivariate											
	Model 1 (Univariate)				Model 2				Model 3			
	β	LCI	UCI	<i>p</i>	β	LCI	UCI	<i>p</i>	β	LCI	UCI	<i>p</i>
Factor 1: Blood Pressure												
Waist-To-Hip Ratio (w/h)	1.869	0.162	3.576	0.032	0.623	-1.440	2.686	0.554	0.623	-1.422	2.668	0.550
Body Fat (%)	0.024	0.014	0.035	0.000	0.023	0.008	0.039	0.003	0.023	0.004	0.042	0.016
$\dot{V}O_{2\max}$ (ml/kg/min)	-0.034	-0.057	-0.011	0.004	0.011	-0.009	0.032	0.275	0.011	-0.026	0.049	0.552
Handgrip Strength (kg)	0.021	-0.004	0.046	0.095	0.015	-0.005	0.035	0.140	0.015	-0.011	0.041	0.257
Sedentary (minutes)	0.000	0.000	0.001	0.407	0.000	0.000	0.001	0.470	0.000	0.000	0.001	0.500
Social Jetlag (hours)	0.163	-0.020	0.346	0.081	0.077	-0.101	0.254	0.396	0.077	-0.093	0.246	0.374
Processed Food	0.015	-0.043	0.073	0.616	-0.007	-0.095	0.081	0.879	-0.007	-0.073	0.059	0.839
Fruit & Vegetables	-0.052	-0.121	0.018	0.143	-0.037	-0.125	0.050	0.404	-0.037	-0.112	0.037	0.324
Factor 2: Cholesterol												
Waist-To-Hip Ratio (w/h)	-0.200	-1.918	1.517	0.819	-0.026	-2.630	2.578	0.984	-0.026	-1.937	1.885	0.979
Body Fat (%)	-0.010	-0.021	0.001	0.062	0.002	-0.008	0.012	0.692	0.002	-0.014	0.018	0.812
$\dot{V}O_{2\max}$ (ml/kg/min)	0.018	-0.005	0.042	0.119	0.026	0.005	0.047	0.016	0.026	-0.011	0.063	0.170
Handgrip Strength (kg)	-0.021	-0.046	0.004	0.099	-0.015	-0.031	0.001	0.073	-0.015	-0.040	0.010	0.249
Sedentary (minutes)	0.000	0.000	0.001	0.106	0.001	0.000	0.001	0.014	0.001	0.000	0.001	0.033
Social Jetlag (hours)	-0.236	-0.418	-0.054	0.011	-0.202	-0.514	0.111	0.206	-0.202	-0.389	-0.014	0.035
Processed Food	-0.070	-0.127	-0.012	0.017	-0.054	-0.096	-0.012	0.012	-0.054	-0.110	0.002	0.060
Fruit & Vegetables	-0.061	-0.130	0.008	0.081	-0.075	-0.143	-0.007	0.030	-0.075	-0.139	-0.011	0.021

Note. Model 1: Univariate model containing one dependent and one independent variable; Model 2: Model containing waist-to-hip ratio, body fat %, $\dot{V}O_{2\max}$, handgrip strength, sedentary, social jetlag, Processed Food pattern, Fruit and Vegetables pattern, and one dependent variable; Model 3: Model 2 with further adjustments for sex, ethnicity, age, and decile.

	Multivariate											
	Model 1 (Univariate)				Model 2				Model 3			
	β	LCI	UCI	<i>p</i>	β	LCI	UCI	<i>p</i>	β	LCI	UCI	<i>p</i>
Factor 3: Vascular												
Waist-To-Hip Ratio (w/h)	2.106	0.401	3.810	0.016	0.327	-1.285	1.939	0.691	0.327	-1.721	2.375	0.755
Body Fat (%)	0.016	0.006	0.027	0.003	0.018	0.001	0.036	0.044	0.018	-0.001	0.038	0.062
$\dot{V}O_{2\max}$ (ml/kg/min)	-0.026	-0.049	-0.003	0.028	0.011	-0.016	0.038	0.416	0.011	-0.025	0.047	0.546
Handgrip Strength (kg)	0.020	-0.006	0.045	0.125	0.016	-0.007	0.039	0.175	0.016	-0.011	0.043	0.248
Sedentary (minutes)	0.001	0.000	0.001	0.001	0.001	0.000	0.001	0.000	0.001	0.000	0.001	0.002
Social Jetlag (hours)	0.254	0.074	0.434	0.006	0.108	-0.082	0.297	0.266	0.108	-0.071	0.286	0.237
Processed Food	0.099	0.042	0.155	0.001	0.074	0.003	0.145	0.042	0.074	0.014	0.133	0.015
Fruit & Vegetables	-0.068	-0.136	0.001	0.052	-0.062	-0.145	0.022	0.147	-0.062	-0.141	0.018	0.128
Factor 4: Carbohydrate-Metabolic												
Waist-To-Hip Ratio (w/h)	1.142	-0.572	2.855	0.191	0.014	-1.479	1.507	0.985	0.014	-1.971	1.999	0.989
Body Fat (%)	0.017	0.006	0.027	0.002	-0.009	-0.024	0.005	0.220	-0.009	-0.025	0.007	0.273
$\dot{V}O_{2\max}$ (ml/kg/min)	-0.056	-0.079	-0.034	0.000	-0.075	-0.103	-0.047	0.000	-0.075	-0.108	-0.042	0.000
Handgrip Strength (kg)	0.047	0.022	0.072	0.000	0.065	0.048	0.083	0.000	0.065	0.040	0.091	0.000
Sedentary (minutes)	0.000	0.000	0.001	0.175	0.000	0.000	0.001	0.154	0.000	0.000	0.001	0.344
Social Jetlag (hours)	0.180	-0.003	0.363	0.053	0.092	-0.025	0.209	0.123	0.092	-0.079	0.263	0.291
Processed Food	-0.018	-0.076	0.040	0.548	-0.044	-0.071	-0.016	0.002	-0.044	-0.098	0.011	0.115
Fruit & Vegetables	-0.005	-0.075	0.064	0.883	0.041	-0.027	0.109	0.237	0.041	-0.020	0.102	0.185
Factor 5: Cumulative												
Waist-To-Hip Ratio (w/h)	4.916	1.517	8.315	0.005	0.938	-1.282	3.157	0.408	0.938	-2.827	4.702	0.625
Body Fat (%)	0.047	0.026	0.068	0.000	0.035	0.010	0.060	0.007	0.035	0.004	0.066	0.029
$\dot{V}O_{2\max}$ (ml/kg/min)	-0.098	-0.144	-0.052	0.000	-0.026	-0.061	0.008	0.132	-0.026	-0.100	0.047	0.480
Handgrip Strength (kg)	0.067	0.017	0.117	0.009	0.082	0.054	0.109	0.000	0.082	0.024	0.139	0.005
Sedentary (minutes)	0.002	0.001	0.003	0.000	0.002	0.001	0.002	0.000	0.002	0.001	0.003	0.001
Social Jetlag (hours)	0.362	-0.006	0.729	0.054	0.075	-0.322	0.471	0.712	0.075	-0.269	0.418	0.670
Processed Food	0.026	-0.091	0.142	0.663	-0.031	-0.163	0.102	0.651	-0.031	-0.157	0.095	0.634
Fruit & Vegetables	-0.186	-0.324	-0.048	0.008	-0.133	-0.250	-0.016	0.025	-0.133	-0.264	-0.002	0.047

Table 13

Cardiometabolic Patterns

	Factor 1 Blood Pressure	Factor 2 Cholesterol	Factor 3 Vascular	Factor 4 Carbohydrate-Metabolic	Communality
Systolic Blood Pressure (mmHg)	0.90	0.06	0.16	-0.01	0.84
Diastolic Blood Pressure (mmHg)	0.89	-0.01	0.04	-0.05	0.78
Central Blood Pressure (mmHg)	0.88	-0.02	-0.08	0.04	0.80
Total Cholesterol (mmol/L)	0.01	0.92	-0.01	0.07	0.85
LDL-Cholesterol (mmol/L)	0.02	0.70	0.03	0.03	0.50
HDL-Cholesterol (mmol/L)	0.00	0.55	-0.14	-0.45	0.52
Augmentation Index (%)	0.18	-0.04	-0.73	0.02	0.57
Heart Rate (bpm)	0.27	0.10	0.68	0.02	0.54
Fasting Blood Glucose (mmol/L)	0.04	-0.13	0.51	-0.05	0.28
Triglycerides (mmol/L)	0.15	-0.10	-0.02	0.73	0.57
Glycosylated Haemoglobin (%)	-0.17	0.15	-0.10	0.68	0.52
Eigenvalue	2.5	1.7	1.3	1.2	
% Variance Explained	23	16	12	11.0	
% Cumulative Variance	23	39	51	62	
KMO	0.6				
Bartlett's Test	< 0.001				

Note. Bold numbers represent variables with a factor loading ≥ 0.4 ; components retained based on an eigenvalue of 1.

5.5 Discussion

The purpose of this study was to investigate the associations among body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with cardiometabolic health in preadolescent children. Following adjustments for potential confounders (Model 3), sedentary behaviour was the lone lifestyle factor to associate with more than one cardiometabolic health indicator, while body fatness, physical fitness, nutrition, and sleep all associated with only one indicator. Therefore, one single lifestyle factor does not entirely explain cardiometabolic health in preadolescent children.

5.5.1 Comparison With Other Studies

Previous paediatric research has examined the associations among body fatness (Bridger, 2009), physical fitness (Czyż et al., 2017), sedentary behaviour (Griffiths et al., 2016), nutrition (Harrex et al., 2017), and sleep (Skidmore et al., 2013) with cardiometabolic health in preadolescent children, yet none of those studies observed all the factors simultaneously like in this study. For example, several studies investigated the effects of sleep duration on obesity with the results indicating that children with shorter sleep durations (< 9 hours) had a 58% higher risk of overweight and obesity when compared to children getting adequate sleep (X. Chen et al., 2008; Locard et al., 1992; Sekine et al., 2002;). Furthermore, 9 to 11 hours of sleep is the nightly requirement, and anything less has been identified as one of the determinants of cardiometabolic risk factors in children (Harrex et al., 2017; Skidmore et al., 2013). Insufficient sleep alters the levels of appetite-regulating hormones leptin and ghrelin in the body, which could lead to unhealthy dietary habits affecting the functioning of the metabolism, and result in undesired weight gain then triggering inflammation (Chaput et al., 2015; Harrex et al., 2017; Sayin & Buyukinan, 2016; Skidmore et al., 2013).

In addition, several studies have investigated the effects of dietary fibre intake on cardiovascular disease with the results indicating that consuming an additional 7 grams of total fibre above the recommended amount (25 grams per day) significantly reduced the risk of coronary heart disease and CVD from 20% to 9% (Buyken et al., 2010; JACC Study Group, 2010; Pietinen et al., 1996; Streppel, Ocké, Boshuizen, Kok, & Kromhour, 2008; Threapleton et al., 2013). In adults, some studies have investigated the effects of cardiorespiratory fitness on body fatness with the outcomes indicating that increased cardiorespiratory fitness levels could reduce cardiometabolic risk regardless of BMI, and in children, higher levels of physical fitness have been associated with healthier cardiometabolic profiles into adulthood (Church et al., 2004; L. Sandvik et al., 1993; Stevens, Cai, Evenson, & Thomas, 2002).

Body fat is a key component for metabolic functioning and performs numerous functions such as storing lipids and adipokines; however, abnormal production of adipokines and lipids can contribute to obesity-related complications, triggering inflammation and lipotoxicity, which disrupt endothelial cells (Ouchi et al., 2011; Trayhurn & Wood, 2004). A deficiency and/or imbalance of nutrients consumed could have a destructive effect on the physiological process of energy production, which may lead to metabolism and biological disorders that can also trigger an inflammatory response (B. I. Campbell & Spano, 2011; Gropper & Smith, 2012). Consistent physical exercise increases HR, and as force of contraction increases while exercising, this improves physical fitness (D. L. Smith & Fernhall, 2011). This enhanced cardiac output in turn increases the rate of circulation, improving shear stress and resulting in increased nitric oxide production and bioavailability, which may forestall the inflammatory process (Di Francescomarino et al., 2009; Sherman, 2000). Collectively, each of the mentioned lifestyle factors cross over

biologically as the outcomes of the behaviours lead to the same result, either triggering or preventing inflammation, which either initiates or counteracts the pathophysiology to atherosclerosis.

Several recent studies carried out on children and adolescents have examined the effects of sedentary behaviour on body composition, physical fitness, and cardiometabolic risks (Carson, Hunter, et al., 2016; Carson, Tremblay, et al., 2016). The results indicated that: (a) screen time (Hjorth, Chaput, Damsgaard, et al., 2014; Hjorth, Chaput, Ritz, et al., 2014), television viewing (Creighton, Goldman, Teruel, & Rubalcava, 2011; Drenowatz et al., 2013), and computer use (Magee, Caputi, & Iverson, 2013) were significantly associated with unfavourable body composition; (b) higher duration of television viewing was associated with elevated blood pressure (Gopinath et al., 2012; Grøntved et al., 2014); (c) and combined sedentary time and video game usage was associated with lower cardiorespiratory fitness (Dowda, Pfeiffer, Lobelo, Porter, & Pate, 2012; Ruiz et al., 2010), with higher duration of screen time being also associated with lower muscular strength (Grøntved et al., 2013). In adults, increased sedentary behaviour, unhealthy body composition, elevated blood pressure, and lower than ideal fitness levels all have been shown to increase the cardiometabolic risk. Currently, there is minimal research on the long-term impact of prolonged sitting on children; therefore, the effects from sedentary behaviour on cardiometabolic health are not completely understood in this age group. Further study is warranted to continue investigating sedentary behaviour in preadolescent children.

Whereas child-specific data are unavailable, in adults sedentary behaviour is an emerging health risk behaviour for the development of chronic diseases. Recent independent studies of sedentary behaviour and diabetes, obesity, CVD, and mortality have

reported overwhelming results that sedentary behaviour has a detrimental effect on each of these outcomes (P. T. Campbell, Patel, Newton, Jacobs, & Gapstur, 2013; De Heer, Wilkinson, Strong, Bondy, & Koehly, 2012; Frydenlund, Jørgensen, Toft, Pisinger, & Aadahl, 2012; George, Rosenkranz, & Kolt, 2013; S. M. P. Pereira, Ki, & Power, 2012; Pulsford, Stamatakis, Britton, Brunner, & Hillsdon, 2013; Van der Ploeg, Chey, Korda, Banks, & Bauman, 2012; Yates et al., 2012). Prolonged sitting is significantly associated with fasting insulin in both men and women (George et al., 2013; Yates et al., 2012). Additionally, sedentary time is associated with total energy expenditure, which is in turn associated with obesity, diabetes, and hypertension (De Heer et al., 2012; Pulsford et al., 2013). Furthermore, associations among sedentary screen time and CVD biomarkers (LDL-C, TG, and elevated blood pressure) have been significant (Frydenlund et al., 2012; S. M. P. Pereira et al., 2012). Moreover, adults could gain at least 2 years of life expectancy by reducing prolonged sitting and screen time to less than an hour per session (P. T. Campbell et al., 2013; Van der Ploeg et al., 2012). Sedentarism has a destructive effect on vascular function and metabolism. While child-specific data are unavailable, in adults prolonged sitting decreases lower limb blood flow, which decreases shear stress among other effects; low mean shear stress can lead to a dysfunctional endothelium (Thosar et al., 2012; Tremblay et al., 2010). Additionally, sedentary behaviour has been shown to increase triacylglycerol levels while decreasing HDL-C and insulin sensitivity levels, which may also trigger an inflammatory response (Demiot et al., 2007; Hamburg et al., 2007; Tremblay et al., 2010).

5.5.2 Limitations and Strengths of This Study

Existing results were explained based on consideration of potential methodological and physiological limitations of this study. As this was a cross-sectional study, it was

difficult to determine causality; further research with longitudinal designs is required to better understand the associations among body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with cardiometabolic health. This initial cross-sectional study is necessary prior to dedicating time and resources into costly longitudinal trials. Also, as data collection took place at primary schools in group settings, factors such as noise, facility limitations (e.g., private space, tinted windows), distractions, interruptions, and weather could not be controlled or measured (Gortmaker et al., 1999). One limitation is that those factors could have influenced data collection of blood pressure, HR, and body temperature, and also the focus and attention span of the participants (Gortmaker et al., 1999). Lastly, this study's sample was limited to New Zealand based preadolescents, which may not simulate preadolescents in other places. For instance, dietary intake (Mediterranean diet vs. Western diet), types of physical activities (rugby vs. American football), and social ecological factors may affect a child's physiology differently and vary by location, so further research is required to assume these findings would be consistently associated with preadolescent outcomes globally. Notable strengths of this study were that the participants represented a large and diverse group of preadolescents from various parts of New Zealand, and that a comprehensive and simultaneous assessment of cardiometabolic risk factors was undertaken as these determinants tend to be clustered when investigating cardiometabolic syndrome in children.

5.5.3 Research Implications

Globally and in New Zealand, health care systems and the health and wellness of children are deteriorating. Cardiometabolic risk factors lead to NCDs and often initiate early in childhood. The current findings indicate that paediatric health and wellness prevention programs are more likely to be effective if they address multiple lifestyle factors

simultaneously; however, sedentary behaviour requires particular attention. To address the sedentary behaviour health risk in children we must start in the schools by implementing the guideline of no more than 2 hours per day of recreational screen time that has been put into place by the MOH (2015c). This can be accomplished by making sure every student is moving and/or participating in physical activity every hour, which would result in sitting less, moving more, and breaking up sitting times. The MOH has written up these guidelines. Now it is time to make sure each school is implementing these guidelines every day. Additional lifestyle factors, such as (a) reducing body fatness, (b) improving quality of sleep, (c) refining dietary patterns, (d) minimizing sedentary behaviour, and (e) enhancing cardiorespiratory and muscular fitness, are also significant to balance out overall health and wellness during the preadolescent years. These necessary steps may enable initial identification of cardiometabolic risk behaviours and subsequently assist future research in producing further evidence on the associations among lifestyle factors, cardiometabolic risk factors, and health of preadolescent children.

5.6 Conclusion

To my knowledge, this was the first study to simultaneously investigate the associations among body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with cardiometabolic health in preadolescent children. This study provides evidence of an association between each lifestyle factor and cardiometabolic health, indicating a single lifestyle factor does not entirely explain cardiometabolic health in preadolescent children. These findings suggest subsequent preventive measures should aim to improve and sustain multiple lifestyle factors in preadolescent children. This step could be the missing component required to reduce cardiometabolic risk factors, and possibly make an impact on the deteriorating health and wellness of preadolescent children.

CHAPTER 6—GENERAL DISCUSSION AND CONCLUSIONS

During preadolescence, poor lifestyle factors could have a negative impact on health by increasing the likelihood of developing cardiometabolic complications (obesity, hypertension, hyperlipidaemia, hyperglycaemia), and subsequent morbidity and mortality in adulthood. This drawn-out burden may place a significant strain on healthcare systems (overpopulation, financial burden, and mass number of surgical procedures; Latimer-Cheung et al., 2016)). Prior research has examined independently the associations between lifestyle factors with cardiometabolic risk factors; however, uncertainty still remains as to which lifestyle factor(s) associate more strongly with cardiometabolic health in preadolescent children. To present, body fatness has been the focal point of most cardiometabolic research in children. Accordingly, obesity has been identified as one of the determinants of cardiometabolic risk; however, it has not been determined conclusively that body fatness is the most significant determinant of cardiometabolic health. Thus, initially in this study, body fatness and lifestyle factors were examined to contextualise the importance of obesity; then, body fatness and lifestyle factors were examined among cardiometabolic risk factors. The aims of this research were to examine simultaneously the associations among lifestyle factors (body fatness, physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep) with body composition markers (Objective 1) and cardiometabolic health indicators (Objective 2).

6.0 Summary of Findings

6.0.1 Objective 1

Currently, considerable time and resources are being exhausted to improve the health and wellness of children with the goal of overcoming the global childhood obesity epidemic (DeClercq et al., 2008; Nishtar et al., 2016). However, it has not been determined

unequivocally that body fatness is the most significant lifestyle factor to cardiometabolic risk. Therefore, the purpose of Objective 1 was to determine the most significant lifestyle factor by investigating the associations among physical fitness, physical [in]activity, sedentary behaviour, nutrition, and sleep with body fatness indicators (body fat percentage, FMI, BMI, WHR) in preadolescent children. The results indicated that nutrition (Fruit and Vegetables pattern and Breakfast Foods pattern) independently associated with body fat percentage, while cardiorespiratory fitness associated with all four body fatness indicators. Cardiorespiratory fitness may be the most significant childhood obesity health policy target in preadolescent children (Ross et al., 2016).

Adipose tissue performs various significant functions such as storing lipids and accumulating hormones and proteins, which are labelled adipokines (Ouchi et al., 2011; Trayhurn & Wood, 2004). These adipokines carry out pro and anti-inflammatory tasks that involve helping to regulate adiposity and the storage of triacylglycerol in fat deposits as energy reserves (Nakamura et al., 2011; Ouchi et al., 2011; Redinger, 2007). However, abnormal production of adipokines can initiate an undesired molecular and cellular variation of the metabolic process, triggering adipose tissue to secrete adipokines (inflammatory response), which could lead to cardiometabolic complications and disrupt the endothelial cells (Ouchi et al., 2011; Redinger, 2007; Wang & Nakayama, 2010). Lifestyle factors such as nutrition and cardiorespiratory fitness precede the accumulation of adipose tissue. Dietary intake and exercise to improve and sustain high levels of fitness involve energy balance, production, and usage; obesity is a direct result of energy consumed exceeding energy utilised (Campbell & Spano, 2011; Gropper & Smith, 2012; Romieu et al., 2017; Ruiz et al., 2016). Therefore, dietary intake and level of cardiorespiratory fitness could have beneficial or detrimental influences on body fatness

during preadolescence, and collectively these lifestyle factors influence the physiological path to cardiometabolic risk, such as obesity in adulthood (Howe et al., 2014; Stoner, Rowlands, et al., 2016).

Dietary intake could be a major contributor to energy imbalance, and an imbalance and/or lack of quality in nutrients consumed could alter the process of energy production (Campbell & Spano, 2011; Gropper & Smith, 2012). This change in energy production could have detrimental effects on the metabolism, which is a significant factor in obesity related complications, and could lead to diet-related chronic disease risk such as CVD and type 2 diabetes (Collins, Watson, & Burroughs, 2010; Potischman, Cohen, Picciano, 2006; World Cancer Research Fund & American Institute for Cancer Research, 2007). In preadolescent children, there is some evidence that nutrition is associated with body composition; however in the relationship between dietary intake and body fatness specifically, the role of obesity is neither clear cut nor completely understood (Howe et al., 2013; Williams et al., 2013; Wong et al., 2012). One reason for this lack of clarity may be because most previous paediatric research investigating obesity and nutrition focused on food groups or single food associations and not dietary patterns (Howe et al., 2013; Williams et al., 2013; Wong et al., 2012). This study examined food choice and dietary patterns with obesity and the results indicated that there is an association between nutrition and body fat percentage. This is significant because we have identified that foods within the Fruit and Vegetables pattern and Breakfast Food pattern have an effect on body composition. However, since associations can be translated as either positive or negative, the outcome is uncertain. Do these results indicate that consuming foods within these patterns increases or decreases body fat percentage? For example, does eating foods within the Breakfast Food pattern cause a child's body fat percentage to decrease? If so, is it

because of the food within the pattern or the timing of the consumption? Breakfast has always been considered the most important meal of the day as it kickstarts the metabolism (Kobayashi et al., 2014). Therefore, further study that investigates dietary intake and body fatness is warranted to better understand this relationship in preadolescent children.

Cardiorespiratory fitness indicates physical functional capability, which is dependent on the integration of biological systems. This physiological systematic approach includes: external and internal respiration, right and left ventricle functioning of the heart, the capability of the blood vessels to transport blood from the heart to match oxygen requirements, and the effectiveness of the muscle cells to then receive and use the oxygen and nutrients delivered by the blood vessels (Ortega et al., 2008; Ruiz et al., 2016). In regard to obesity, when individuals exercise to improve cardiorespiratory fitness levels, the body burns up large amounts of energy as fuel in attempting to achieve maximal physical exertion; therefore, exercising and being active to improve and sustain fitness helps reduce the positive energy balance that leads to an obese state (Laskowski, 2012; I. M. Lee, Djoussé, Sesso, Wang, & Buring, 2010). Moreover, exercise and elevated levels of cardiorespiratory fitness have been shown to improve body composition in all weight groups in children (Khanna, Majumdar, Saha, & Mandal, 1998; Stoner, Rowlands, et al., 2016) and adults (Clark, Fonarow, & Horwich, 2015; McAuley et al., 2012; Pandey et al., 2017). However, with the minimal data available in children, further studies investigating the effects of cardiorespiratory fitness on obesity are warranted.

Exercising to improve and sustain cardiorespiratory fitness levels requires the body to operate at a high level; however, excessive body fatness negatively affects the functioning of the cardiovascular system. Excessive adipose tissue in the body requires blood vessels to circulate more blood to the fat tissue, which increases the heart's workload

and puts more pressure on the artery walls (Nakamura et al., 2011; Ouchi et al., 2011; Redinger, 2007). Obesity can also raise heart rate and narrow arteries, which restricts the body's ability to transport blood through vessels efficiently (Nakamura et al., 2011; Ouchi et al., 2011; Redinger, 2007). Furthermore, carrying excessive amounts of weight places a strain on the knees and hips, which could lead to musculoskeletal impairments and limit the ability to be active and exercise at a high level (Laskowski, 2012; Wearing, Hennig, Byrne, Steele, & Hills, 2006). Excessive weight has also been shown to cause respiratory problems as added weight of the chest wall causes restricted breathing, which interrupts sleep patterns, and can lead to daytime sleepiness and low levels of energy, which may also affect exercise intensity (Gozal, Capdevilla, & Kheirandish-Gozal, 2008). Exercise is one of the major actions that help moderate body fatness and all these bodily malfunctions are triggered by obesity, which affect one's ability to exercise and be active at a high level. Also, as previously mentioned, dietary intake provides the body with the nutrients required to produce energy, which is required while being active or exercising to improve and sustain cardiorespiratory fitness levels. Collectively, cardiorespiratory fitness, dietary intake, and body fatness overlap physiologically, and the outcomes could have beneficial or detrimental effects on the cardiovascular system. However, research has not determined beyond question if obesity, cardiorespiratory fitness, dietary intake, or any other lifestyle factor is most significant factor to cardiometabolic risk in preadolescent children.

6.0.2 Objective 2

The purpose of Objective 2 was to consider the results from Objective 1 and further investigate the associations among cardiorespiratory fitness, body fatness, dietary intake, and additional lifestyle factors (muscular strength, physical [in]activity, sedentary behaviour, and sleep) with added cardiometabolic health indicators in attempting to identify

which factors associate more strongly in preadolescent children. Cardiometabolic health indicators were determined by PCA (for further information, see statistical analysis sections 3.5 and 5.3 in Chapters 3 and 5, respectively), and labelled blood pressure factor, cholesterol factor, vascular factor, and carbohydrate-metabolic factor. The results indicated that body fat percentage was associated with blood pressure factor; sedentary minutes, social jetlag; Fruit and Vegetables pattern was associated with cholesterol factor; sedentary minutes and Processed Food pattern were associated with vascular factor; and cardiorespiratory fitness and handgrip strength were associated with carbohydrate-metabolic factor. Similar to Objective 1, body fat percentage, cardiorespiratory fitness, and nutrition again associated with cardiometabolic risk; however in Objective 2 additional lifestyle factors also showed a correlation. Consequently, several lifestyle factors associated with cardiometabolic risk, and thus one specific lifestyle factor does not entirely explain cardiometabolic health in preadolescent children.

Previous research has revealed that lifestyle factors associate independently with cardiometabolic health indicators. For instance, increased body fat percentage and high levels of body mass appear to be significant risk factors for the progression of hypertension (Al-Sendi, Shetty, Musaiger, & Myatt, 2003; Mushengezi & Chillo, 2014). Sedentary behaviour (V. Armstrong, 2017; Leitzmann et al., 2017), quality of sleep (Harrex et al., 2017; Skidmore et al., 2013), and dietary intake (Conget & Giménez, 2009; Kuipers et al., 2011) can negatively or positively affect metabolism independently. Sedentary behaviour (V. Armstrong, 2017; Leitzmann et al., 2017) and dietary intake (Conget & Giménez, 2009; Kuipers et al., 2011) could independently have an impact on the functioning of the vascular system, while exercise and high levels of fitness can have a positive influence on metabolic outcomes (Lavie et al., 2015; Press et al., 2003). Among these lifestyle factors, the

independent associations and biological outcomes that lead to cardiometabolic complications have been widely studied and analysed; however, the overlapping and clustering of the mechanisms is still novel research.

In children, the consequences from the overlapping and clustering of detrimental lifestyle factors that may lead to cardiometabolic risk are less certain. In adults, we do know that harmful lifestyle factors affect each other as the physiological pathways that may lead to cardiometabolic complications cross over biologically, however the foundation is still being explored. As described in Chapter 5 (see section 5.5.1) in discussing comparisons with other studies, each of the mentioned lifestyle factors either has detrimental or beneficial effects on the physiological functioning of the body. Body fat is an important factor to metabolic functioning, yet too much fat could lead to obesity-linked complications, triggering inflammation (Ouchi et al., 2011; Trayhurn & Wood, 2004). Lack of sleep changes the levels of appetite-regulating hormones leptin and ghrelin in the body, which can lead to unhealthy dietary choices that also affect metabolism, which could again initiate the process of inflammation (Chaput et al., 2015; Harrex et al., 2017; Sayin & Buyukinan, 2016; Skidmore et al., 2013). Unhealthy dietary intake, such as insufficient fruit and vegetable intake and excessive consumption of processed and fast-food, could have detrimental outcomes on the biological process of energy production, negatively affecting the functioning of a healthy metabolism, which may result in an inflammatory response (Campbell & Spano, 2011; Gropper & Smith, 2012). A sedentary lifestyle could decrease blood flow and increase triacylglycerol levels, which has a destructive effect on vascular function and metabolism, and may also trigger an inflammatory response (Demiot et al., 2007; Hamburg et al., 2007; Tremblay et al., 2010). In contrast, enhanced physical activity and improved fitness result in increased circulation, which improves shear stress

and increases nitric oxide production and bioavailability, helping to attenuate inflammation (Di Francescomarino et al., 2009; Sherman, 2000). Collectively, each of the mentioned individual lifestyle factors has beneficial or detrimental consequences on the cardiovascular system and metabolism that may trigger or help prevent inflammation onset, which is an important part of the pathophysiology to CVD.

The associations for body fatness and cardiometabolic health are not singular and for children there is a further complication in the social context as children's environmental surroundings may impact their short- and long-term health and wellness. At the preadolescent age, children are given limited choices and decisions regarding sleep, diet, and activity, which are typically determined by teachers, parent(s), or guardians (Hodges, 2003; Savage, Fisher, & Birch, 2007). However, when children are allowed to make their own decision, their immediate environment strongly influences their selections (Dagkas & Stathi, 2007; C. Sandvik et al., 2005; K. R. Smith, Corvalán, Kjellström, 1999). For instance, with technological devices readily available to all ages, sleep duration can be altered and result in sleep deprivation (X. Chen et al., 2008; Owens et al., 2000). There is research available now on the negative effects of blue light from electronics (e.g., computers, tablets, and gaming devices) on melatonin levels, REM sleep, and the circadian clock, which can contribute to weight-related complications (Chang, Aeschbach, Duffy, & Czeisler, 2015; Figueiro, Wood, Plitnick, & Rea, 2011; Fossum, Nordnes, Storemark, Bjorvatn, & Pallesen, 2014; Hale & Guan, 2015). Furthermore, advertisement that promotes fast-food and junk foods as compared to fruits and vegetables sends the wrong nutritional message to children, and having fast-food and convenience stores near schools has been found to have a negative effect on older children's food choices (Davis & Carpenter, 2009; Powell, Auld, Chaloupka, O'Malley, & Johnston, 2007). While outside

the scope of this study, it is clear a multitude of environmental factors influences body fatness, physical fitness, sedentary behaviour, nutrition, and sleep and ultimately cardiometabolic health. Initially, to address these environmental influences, the importance of living a healthy lifestyle could be taught to children within the scope of health education classes, and to parents and guardians in the evenings and on weekends through adult health education programmes.

6.0.3 Implications

This research does highlight the importance of educational guidelines that focus on teaching sustainable lifestyle factors to offset the detrimental influences children are exposed to within their daily environmental surroundings. These guidelines could be implemented during physical education and health education classes in primary schools.

Cardiorespiratory fitness is clearly the lifestyle factor that associates most strongly with body fatness, and obesity has been identified in this research as one of the major determinants of cardiometabolic complications. Physical education classes could incorporate activities focused on improving and sustaining cardiorespiratory fitness levels in children; increased and sustained fitness levels have been shown to have beneficial outcomes on body fatness (Khanna et al., 1998; Stoner, Rowlands, et al., 2016).

Cardiorespiratory fitness is moderated by physical activity, which is strongly influenced by motor skills, and motor skills are instilled during childhood when preadolescent children spend a great deal of their day in school (Chaddock-Heyman et al., 2013; Sibley & Etnier, 2003). Therefore, primary school physical education classes are an ideal place to focus on improving and sustaining fitness in preadolescent children. As part of the physical education curricula, children would participate in energetic activities every day for at least 60 minutes (MOH, 2015a, 2015c, 2017). Periodically, cardiorespiratory fitness levels could

be tested and tracked utilising the 20-MST. Subsequently, the children who test in the “needs improvement fitness zone” category could be identified and provided extra support to improve and maintain fitness levels before their overall healthiness becomes problematic and unmanageable.

Ideally, the importance of living a healthy lifestyle could be taught to children, parents, and guardians through health education curricula. The core curriculum could be implemented daily in health education classes for children and offered in the evenings and on weekends for parents and guardians. The short and long-term consequences from deficient lifestyle factors, including and not limited to excessive body fatness, lower than ideal physical fitness, insufficient daily physical activity, unhealthy diet, and inadequate sleep, could be at the forefront of the health education curricula. Numerous everyday life choices and surrounding influences contribute to different aspects of a child’s overall health and wellness; therefore, health education curricula could focus on teaching sustainable lifestyle factors, the importance of making healthy choices, and the consequences of making unhealthy choices in everyday life.

6.0.4 Future Directions, Limitations, and Strengths

The major limitations associated with this study include that this research only evaluated one age group, this was a cross-sectional study, and association is not causality. However, this initial stage was essential prior to investing the necessary time and resources required in large, pragmatic research trials. The major strengths associated with this research included a customised and superior model of cardiometabolic risk that was utilised to determine associations among lifestyle factors; this is the first time all these lifestyle factors have been examined simultaneously with cardiometabolic risk factors in preadolescent children. Consequently, this important study provides the appropriate

scientific underpinning necessary for longitudinal trials examining lifestyle factors with cardiometabolic health. Longitudinal trials could examine causes to the effects subsequent to lifestyle factors and cardiometabolic health indicators in preadolescent children through their adolescent years over an extended period.

6.1 Conclusion

Findings from this thesis demonstrated that body fatness, physical fitness, sedentary behaviour, nutrition, and sleep all associated with various cardiometabolic risk factors in preadolescent children; therefore, a single lifestyle factor does not entirely explain cardiometabolic health in preadolescent children. Furthermore, analysis conducted in this research provides a customised and superior model for simultaneously examining cardiometabolic risk in preadolescent children. Findings from this thesis could have implications for public health policies targeting cardiometabolic risk in children, health and physical education school curricula, and community-based before school and after school programmes attempting to address the deteriorating health and wellness of preadolescent children.

References

- Abarca-Gómez, L., Abdeen, Z. A., Hamid, Z. A., Abu-Rmeileh, N. M., Acosta-Cazares, B., Acuin, C., . . . Ezzati, M. (2017). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: A pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *The Lancet*, *390*, 2627-2642. doi:10.1016/S0140-6736(17)32129-3
- Acar Tek, N., Yildiran, H., Akbulut, G., Bilici, S., Koksall, E., Gezmen Karadag, M., & Sanlier, N. (2011). Evaluation of dietary quality of adolescents using Healthy Eating Index. *Nutrition Research and Practice*, *5*, 322-328. doi:10.4162/nrp.2011.5.4.322
- Ahrens, W., Moreno, L. A., Mårild, S., Molnár, D., Siani, A., De Henauw, S., . . . Pigeot, I. (2014). Metabolic syndrome in young children: Definitions and results of the IDEFICS study. *International Journal of Obesity*, *38*, S4-S14. doi:10.1038/ijo.2014.130
- Ajala, O., Mold, F., Boughton, C., Cooke, D., & Whyte, M. (2017). Childhood predictors of cardiovascular disease in adulthood. A systematic review and meta-analysis. *Obesity Reviews*, *18*, 1061-1070. doi:10.1111/obr.12561
- Al-Sendi, A. M., Shetty, P., Musaiger, A. O., & Myatt, M. (2003). Relationship between body composition and blood pressure in Bahraini adolescents. *British Journal of Nutrition*, *90*, 837-844. doi:10.1079/BJN2003963

- Ali, O., Cerjak, D., Kent, J. W., James, R., Blangero, J., & Zhang, Y. (2014). Obesity, central adiposity and cardiometabolic risk factors in children and adolescents: A family-based study. *Pediatric Obesity*, 9(3), e58-e62. doi:10.1111/j.2047-6310.2014.218.x
- Ali, S. A., Ali, S., A., Suhail, N, Tikmani, S. S., & Bano, G. (2016). Childhood obesity, a public health crisis: Narrative review of public health interventions for childhood obesity. *Journal of Universal Surgery*, 4(2), 1-6. Retrieved from https://ecommons.aku.edu/pakistan_fhs_mc_chs_chs/367
- American Diabetes Association. (2014). Diagnosis and classification of diabetes mellitus. *Diabetes Care*, 37(Suppl. 1), S81-S90. doi:10.2337/dc14-S081
- Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W., & Pollak, C. P. (2003). The role of actigraphy in the study of sleep and circadian rhythms. *Sleep*, 26, 342-392. doi:10.1093/sleep/26.3.342
- Andersen, L. B., Sardinha, L. B., Froberg, K., Riddoch, C. J., Page, A. S., & Andersen, S. A. (2008). Fitness, fatness and clustering of cardiovascular risk factors in children from Denmark, Estonia and Portugal: The European Youth Heart Study. *International Journal of Pediatric Obesity*, 3(S1), 58-66. doi:10.1080/17477160801896366
- Anderson, Y. C., Cave, T. L., Cunningham, V. J., Pereira, N. M., Woolerton, D. M., Grant, C. C., . . . Hofman, P. L. (2015). Effectiveness of current interventions in obese New Zealand children and adolescents. *New Zealand Medical Journal*, 128(1417), 8-15. Retrieved from <https://www.nzma.org.nz/journal>

- Anderson, Y. C., Wynter, L. E., Grant, C. C., Stewart, J. M., Cave, T. L., Wild, C. E. K., . . . Hofman, P. L. (2017). Physical activity is low in obese New Zealand children and adolescents. *Scientific Reports*, 7(41822), 1-7. doi:10.1038/srep41822
- Archbold, K. H., Pituch, K. J., Panahi, P., & Chervin, R. D. (2002). Symptoms of sleep disturbances among children at two general pediatric clinics. *Journal of Pediatrics*, 140, 97-102. doi:10.1067/mpd.2002.119990
- Armstrong, N. (2013). Aerobic fitness and physical activity in children. *Pediatric Exercise Science*, 25, 548-560. doi:10.1123/pes.25.4.548
- Armstrong, V. (2017). *The impact of shear rate and prolonged sitting on endothelial function in children* (Master's thesis). Retrieved from <https://open.library.ubc.ca/cIRcle/collections/ubctheses/24/items/1.0357351>
- Barrett, S. C., Huffman, F. G., & Johnson, P. (2011). Validation of finger-prick testing of fasting blood glucose, total cholesterol, and HbA1c in adolescents. *Point of Care: Journal of Near-Patient Testing & Technology*, 10(2), 51-58. doi:10.1097/POC.0b013e31821bd65e
- Barry, V. W., Baruth, M., Beets, M. W., Durstine, J. L., Liu, J., & Blair, S. N. (2014). Fitness vs. fatness on all-cause mortality: A meta-analysis. *Progress in Cardiovascular Diseases*, 56, 382-390. doi:10.1016/j.pcad.2013.09.002
- Beauloye, V., Zech, F., Tran Thi Mong, H., Clapuyt, P., Maes, M., & Brichard, S. M. (2007). Determinants of early atherosclerosis in obese children and adolescents. *The Journal of Clinical Endocrinology & Metabolism*, 92, 3025-3032. doi:10.1210/jc.2007-0619
- Beck, S. E., & Marcus, C. L. (2009). Pediatric polysomnography. *Sleep Medicine Clinics*, 4, 393-406. doi:10.1016/j.jsmc.2009.04.007

- Beets, M. W., Patton, M. M., & Edwards, S. (2005). The accuracy of pedometer steps and time during walking in children. *Medicine and Science in Sports and Exercise*, *37*, 513-520. doi:10.1249/01.MSS.0000155395.49960.31
- Bessesen, D. H. (2001). The role of carbohydrates in insulin resistance. *Journal of Nutrition*, *131*(10), 2782S-2786S. doi:10.1093/jn/131.10.2782S
- Boden, G. (2008). Obesity and free fatty acids. *Endocrinology and Metabolism Clinics of North America*, *37*, 635-646. doi:10.1016/j.ecl.2008.06.007
- Bohn, B., Muller, M. J., Simic-Schleicher, G., Kiess, W., Siegfried, W., Oelert, M., . . . Holl, R. W. (2015). BMI or BIA: Is body mass index or body fat mass a better predictor of cardiovascular risk in overweight or obese children and adolescents? A German/Austrian/Swiss Multicenter APV Analysis of 3,327 children and adolescents. *Obesity Facts*, *8*, 156-165. doi:10.1159/000381227
- Bonetti, P. O., Lerman, L. O., & Lerman, A. (2003). Endothelial dysfunction: A marker of atherosclerotic risk. *Arteriosclerosis, Thrombosis, and Vascular Biology*, *23*, 168-175. doi:10.1161/01.ATV.0000051384.43104.FC
- Booth, F. W., Roberts, C. K., & Laye, M. J. (2012). Lack of exercise is a major cause of chronic diseases. *Comprehensive Physiology*, *2*, 1143-1211. doi:10.1002/cphy.c110025
- Boreham, C. A., Paliczka, V. J., & Nichols, A. K. (1990). A comparison of the PWC170 and 20-MST tests of aerobic fitness in adolescent schoolchildren. *Journal of Sports Medicine and Physical Fitness*, *30*(1), 19-23.

- Bornstein, D. B., Beets, M. W., Byun, W., Welk, G., Bottai, M., Dowda, M., & Pate, R. (2011). Equating accelerometer estimates of moderate-to-vigorous physical activity: In search of the Rosetta Stone. *Journal of Science and Medicine in Sport, 14*, 404-410. doi:10.1016/j.jsams.2011.03.013
- Bouchard, C., Rankinen, T., & Timmons, J. A. (2011). Genomics and genetics in the biology of adaptation to exercise. *Comprehensive Physiology, 1*, 1603-1648. doi:10.1002/cphy.c100059
- Brage, S., Wedderkopp, N., Ekelund, U., Franks, P. W., Wareham, N. J., Andersen, L. B., & Froberg, K. (2004). Features of the metabolic syndrome are associated with objectively measured physical activity and fitness in Danish children. *Diabetes Care, 27*, 2141-2148. doi:10.1337/diacare.27.9.2141
- Bridger, T. (2009). Childhood obesity and cardiovascular disease. *Paediatrics & Child Health, 14*, 177-182. doi:10.1093/pch/14.3.177
- Brooke, H. L., Corder, K., Griffin, S. J., Ekelund, U., & Van Sluijs, E. M. (2013). More of the same or a change of scenery: An observational study of variety and frequency of physical activity in British children. *BMC Public Health, 13*(761), 1-19. doi:10.1186/1471-2458-13-761
- Butlin, M., Qasem, A., & Avolio, A. P. (2012). Estimation of central aortic pressure waveform features derived from the brachial cuff volume displacement waveform. *Conference Proceedings Institute of Electrical and Electronics Engineers (IEEE) Engineering in Medicine and Biology Society, 2012*, 2591-2594. doi:10.1109/EMBC.2012.6346494

- Buyken, A. E., Flood, V., Empson, M., Roachtchina, E., Barclay, A. W., Brand-Miller, J., & Mitchell, P. (2010). Carbohydrate nutrition and inflammatory disease mortality in older adults. *American Journal of Clinical Nutrition*, *92*, 634-643.
doi:10.3945/ajcn.2010.29390
- Cade, J., Thompson, R., Burley, V., & Warm, D. (2002). Development, validation and utilisation of food-frequency questionnaires—A review. *Public Health Nutrition*, *5*, 567-587. doi:10.1079/PHN2001318
- Campbell, B. I., & Spano, M. A. (Eds.). (2011). *NSCA's guide to sport and exercise nutrition*. Champaign, IL: Human Kinetics.
- Campbell, P. T., Patel, A. V., Newton, C. C., Jacobs, E. J., & Gapstur, S. M. (2013). Associations of recreational physical activity and leisure time spent sitting with colorectal cancer survival. *Journal of Clinical Oncology*, *31*, 876-885.
doi:10.1200/JCO.2012.45.9735
- Canadian Society for Exercise Physiology. (n.d.). *24-hour movement guidelines for children and youth*. Retrieved from <http://www.csep.ca/>
- Canova, S., Cortinovis, D. L., & Ambrogi, F. (2017). How to describe univariate data. *Journal of Thoracic Disease*, *9*, 1741-1743. doi:10.21037/jtd.2017.05.80
- Carson, V., Hunter, S., Kuzik, N., Gray, C. E., Poitras, V. J., Chaput, J.-P., . . . Tremblay, M. S. (2016). Systematic review of sedentary behaviour and health indicators in school-aged children and youth: An update. *Applied Physiology, Nutrition, and Metabolism*, *41*, S240-S265. doi:10.1139/apnm-2015-0630

- Carson, V., Tremblay, M. S., Chaput, J-P., & Chastin, S. F. M. (2016). Associations between sleep duration, sedentary time, physical activity, and health indicators among Canadian children and youth using compositional analyses. *Applied Physiology, Nutrition, and Metabolism*, *41*, S294-S302. doi:10.1139/apnm-2016-0026
- Carter, P. J., Taylor, B. J., Williams, S. M., & Taylor, R. W. (2011). Longitudinal analysis of sleep in relation to BMI and body fat in children: The FLAME Study. *BMJ*, *342*(d2712), 1-7. doi:10.1136/bmj.d2712
- Caspersen, C. J., Powell, K. E., & Christenson, G. M. (1985). Physical activity, exercise, and physical fitness: Definitions and distinctions for health-related research. *Public Health Reports*, *100*, 126–131. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1424733/>
- Castillo Garzón, M. J., Ortega Porcel, F. B., & Ruiz Ruiz, J. (2005). Improvement of physical fitness as anti-aging intervention. *Medicina Clinica*, *124*, 146-155. doi:10.1157/13071011
- Castro, N., Faulkner, J., Skidmore, P., Williams, M., Lambrick, D. M., Signal, L., . . . Stoner, L. (2014). Pre-adolescent cardio-metabolic associations and correlates: PACMAC methodology and study protocol. *BMJ Open*, *4*(e005815), 1-9. doi:10.1136/bmjopen-2014-005815
- Centers for Disease Control and Prevention (CDC). (2015a). *About child & teen BMI*. Retrieved from https://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html

Centers for Disease Control and Prevention (CDC). (2015b). *Childhood obesity facts*.

Retrieved from <http://www.cdc.gov/obesity/data/childhood.html>

Chaddock-Heyman, L., Erickson, K. I., Voss, M. W., Knecht, A. M., Pontifex, M. B.,

Castelli, D. M., . . . Kramer, A. F. (2013). The effects of physical activity on

functional MRI activation associated with cognitive control in children: A

randomized controlled intervention. *Frontiers in Human Neuroscience*, *7*, 1-72.

doi:10.3389/fnhum.2013.00072

Challet, E. (2013). Circadian clocks, food intake, and metabolism. *Progress in Molecular*

Biology and Translational Science, *119*, 105-135. doi:10.1016/B978-0-12-396971-

2.00005-1

Chang, A.-M., Aeschbach, D., Duffy, J. F., & Czeisler, C. A. (2015). Evening use of light-

emitting eReaders negatively affects sleep, circadian timing, and next-morning

alertness. *Proceedings of the National Academy of Sciences*, *112*, 1232-1237.

doi:10.1073/pnas.1418490112

Chaput, J.-P., Katzmarzyk, P. T., LeBlanc, A. G., Tremblay, M. S., Barreira, T. V.,

Broyles, S. T., . . . Olds, T. (2015). Associations between sleep patterns and lifestyle

behaviors in children: An international comparison. *International Journal of*

Obesity Supplements, *5*, S59-S65. doi:10.1038/ijosup.2015.21

Charakida, M., Donald, A. E., Terese, M., Leary, S., Halcox, J. P., Ness, A., . . . Deanfield,

J. E. (2005). Endothelial dysfunction in childhood infection. *Circulation*, *111*, 1660-

1665. doi:10.1161/01.cir.0000160365.18879.1c

- Chen, W., Srinivasan, S. R., Elkasabany, A., & Berenson, G. S. (1999). Cardiovascular risk factors clustering features of insulin resistance syndrome (Syndrome X) in a biracial (Black-White) population of children, adolescents, and young adults: The Bogalusa Heart Study. *American Journal of Epidemiology*, *150*, 667-674. doi:10.1093/oxfordjournals.aje.a010069
- Chen, X., Beydoun, M. A., & Wang, Y. (2008). Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. *Obesity*, *16*, 265-274. doi:10.1038/oby.2007.63
- Church, T. S., Cheng, Y. J., Earnest, C. P., Barlow, C. E., Gibbons, L. W., Priest, E. L., & Blair, S. N. (2004). Exercise capacity and body composition as predictors of mortality among men with diabetes. *Diabetes Care*, *27*, 83-88. doi:10.2337/diacare.27.1.83
- Clark, A. L., Fonarow, G. C., & Horwich, T. B. (2015). Impact of cardiorespiratory fitness on the obesity paradox in patients with systolic heart failure. *American Journal of Cardiology*, *115*, 209-213. doi:10.1016/j.amjcard.2014.10.023
- Clifton, P. M. (2011). Protein and coronary heart disease: The role of different protein sources. *Current Atherosclerosis Reports*, *13*, 493-498. doi:10.1007/s11883-011-0208-x
- Cohen, D. D., Voss, C., Taylor, M. J., Delestrat, A., Ogunleye, A. A., & Sandercock, G. R. (2011). Ten-year secular changes in muscular fitness in English children. *Acta Paediatrica*, *100*, e175-177. doi:10.1111/j.1651-2227.2011.02318.x
- Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a standard definition for child overweight and obesity worldwide: International survey. *BMJ*, *320*, 1240-1243. doi:10.1136/bmj.320.7244.1240

- Cole, T. J., & Lobstein, T. (2012). Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. *Pediatric Obesity*, 7, 284-294. doi:10.1111/j.2047-6310.2012.00064.x
- Collins, C. E., Watson, J., & Burrows, T. (2010). Measuring dietary intake in children and adolescents in the context of overweight and obesity. *International Journal of Obesity*, 34, 1103-1115. doi:10.1038/ijo.2009.241
- Conget, I., & Giménez, M. (2009). Glucose control and cardiovascular disease: Is it important? No. *Diabetes Care*, 32(Suppl. 2), S334-S336. doi:10.2337/dc09-S334
- Cooper, G. M. (2000). The chemistry of cells: The central role of enzymes as biological catalysts. In G. M. Cooper (Ed.), *The cell: A molecular approach* (2nd ed., pp. 145-146). Sunderland, MA: Sinauer Associates.
- Cooper Institute. (2014). *Healthy Fitness Zone® Standards*. Retrieved from <http://www.cooperinstitute.org/fitnessgram/standards>
- Corder, K., Van Sluijs, E. M., Wright, A., Whincup, P., Wareham, N. J., & Ekelund, U. (2009). Is it possible to assess free-living physical activity and energy expenditure in young people by self-report? *American Journal of Clinical Nutrition*, 89, 862-870. doi:10.3945/ajcn.2008.26739
- Corner, S. (2009). Choosing the right type of rotation in PCA and EFA. *JALT Testing & Evaluation SIG Newsletter*, 13(3), 20-25. Retrieved from <https://hosted.jalt.org/test/PDF/Brown31.pdf>
- Costello, A. B., & Osborne, J. W. (2005). Best practices in exploratory factor analysis: Four recommendations for getting the most from your analysis. *Practical Assessment, Research & Evaluation*, 10(7), 1-9. Retrieved from <https://www.pareonline.net/pdf/v10n7.pdf>

- Cote, A. T., Harris, K. C., Panagiotopoulos, C., Sandor, G. G., & Devlin, A. M. (2013). Childhood obesity and cardiovascular dysfunction. *Journal of the American College of Cardiology*, *62*, 1309-1319. doi:10.1016/j.jacc.2013.07.042
- Creighton, M. J., Goldman, N., Teruel, G., & Rubalcava, L. (2011). Migrant networks and pathways to child obesity in Mexico. *Social Science & Medicine*, *72*, 685-693. doi:10.1016/j.socscimed.2010.12.006
- Curcio, G., Ferrara, M., & De Gennaro, L. (2006). Sleep loss, learning capacity and academic performance. *Sleep Medicine Reviews*, *10*, 323-337. doi:10.1016/j.smrv.2005.11.001
- Czyż, S. H., Toriola, A. L., Starościak, W., Lewandowski, M., Paul, Y., & Oyeyemi, A. L. (2017). Physical fitness, physical activity, sedentary behavior, or diet—What are the correlates of obesity in Polish school children? *International Journal of Environmental Research and Public Health*, *14*, 1-21. doi:10.3390/ijerph14060664
- Da Silva, J. L., De Oliveira, R. F., Bertin, L. D., De Oliveira, P. D., Beltrão Pires, F., Spadaccini, A. F., . . . Almeida Pires-Oliveira, D. A. (2018). Physical inactivity and associated factors in adults of a basic health unit: An observational cross-sectional study. *Manual Therapy, Posturology & Rehabilitation Journal*, *16*(476), 1-6. doi:10.17784/mtprehabjournal.2018.16.476
- Dagkas, S., & Stathi, A. (2007). Exploring social and environmental factors affecting adolescents' participation in physical activity. *European Physical Education Review*, *13*, 369-384. doi:10.1177/1356336X07081800
- Damodaran, S. (2007). Amino acids, peptides and proteins. In S. Damodaran, K. L. Parkin, & O. R. Fennema (Eds.), *Fennema's food chemistry* (4th ed., pp. 425-439). Boca Raton, FL: CRC Press.

- Davies, J. I., & Struthers, A. D. (2003). Pulse wave analysis and pulse wave velocity: A critical review of their strengths and weaknesses. *Journal of Hypertension, 21*, 463-472. Retrieved from http://journals.lww.com/jhypertension/Abstract/2003/03000/Pulse_wave_analysis_and_pulse_wave_velocity___a.4.aspx
- Davies, T., & Fearn, T. (2004). Back to basics: The principles of principal component analysis. *Spectroscopy Europe, 16*(6), 20-23. Retrieved from <http://www.montefiore.ulg.ac.be/~kvansteen/GBIO0009-1/ac20082009/Principles%20of%20PCA.pdf>
- Davignon, J., & Ganz, P. (2004). Role of endothelial dysfunction in atherosclerosis. *Circulation, 109*(23 Suppl. 1), III-27-III-32. doi:10.1161/01.CIR.0000131515.03336.f8
- Davis, B., & Carpenter, C. (2009). Proximity of fast-food restaurants to schools and adolescent obesity. *American Journal of Public Health, 99*, 505-510. doi:10.2105/AJPH.2008.137638
- Davison, B., Saeedi, P., Black, K., Harrex, H., Haszard, J., Meredith-Jones, K., . . . Skidmore, P. (2017). The association between parent diet quality and child dietary patterns in nine- to eleven-year-old children from Dunedin, New Zealand. *Nutrients, 9*(5), 1-11. doi:10.3390/nu9050483
- DeClercq, V., Taylor, C., & Zahradka, P. (2008). Adipose tissue: The link between obesity and cardiovascular disease. *Cardiovascular & Haematological Disorders-Drug Targets, 8*, 228-237. doi:10.2174/187152908785849080
- De Heer H. D., Wilkinson, A. V., Strong, L.L., Bondy, M. L., Koehly, L. M. (2012). Sitting time and health outcomes among Mexican origin adults: Obesity as a mediator. *BMC Public Health, 12*(896) 1-9. doi:10.1186/1471-2458-12-896

- De Niet, J. E., & Naiman, D. I. (2011). Psychosocial aspects of childhood obesity. *Minerva Pediatrica*, 63, 491-505. Retrieved from hdl.handle.net/1765/88291
- De Onis, M., Onyango, A. W., Borghi, E., Siyam, A., Nishida, C., & Siekmann, J. (2007). Development of a WHO growth reference for school-aged children and adolescents. *Bulletin of the World Health Organization*, 85, 660-667. Retrieved from http://www.who.int/growthref/growthref_who_bull/en/
- De Souza, R. J., Mente, A., Maroleanu, A., Cozma, A. I., Ha, V., Kishibe, T., . . . Anand, S. S. (2015). Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: Systematic review and meta-analysis of observational studies. *BMJ*, 351(h3978), 1-16. doi:10.1136/bmj.h3978
- Demiot, C., Dignat-George, F., Fortrat, J. O., Sabatier, F., Gharib, C., Larina, I., . . . Custaud, M. A. (2007). WISE 2005: Chronic bed rest impairs microcirculatory endothelium in women. *American Journal of Physiology-Heart and Circulatory Physiology*, 293(5), H3159-H3164. doi:10.1152/ajpheart.00591.2007
- Dewald, J. F., Meijer, A. M., Oort, F. J., Kerkhof, G. A., & Bögels, S. M. (2010). The influence of sleep quality, sleep duration and sleepiness on school performance in children and adolescents: A meta-analytic review. *Sleep Medicine Reviews*, 14, 179-189. doi:10.1016/j.smrv.2009.10.004
- Dietz, P., Hoffmann, S., Lachtermann, E., & Simon, P. (2012). Influence of exclusive resistance training on body composition and cardiovascular risk factors in overweight or obese children: A systematic review. *Obesity Facts*, 5, 546-560. doi:10.1159/000341560
- Dietz, W. H. (2004). Overweight in childhood and adolescence. *New England Journal of Medicine*, 350, 855-857. doi:10.1056/NEJMp048008

- Di Francescomarino, S., Sciartilli, A., Di Valerio, V., Di Baldassarre, A., & Gallina, S. (2009). The effect of physical exercise on endothelial function. *Sports Medicine*, 39, 797-812. doi:10.2165/11317750-000000000-00000
- Ding, D., Lawson, K. D., Kolbe-Alexander, T. L., Finkelstein, E. A., Katzmarzyk, P. T., Van Mechelen, W., . . . Lancet Physical Activity Series 2 Executive Committee. (2016). The economic burden of physical inactivity: A global analysis of major non-communicable diseases. *The Lancet*, 388, 1311-1324. doi:10.1016/S0140-6736(16)30383-X
- Dishman, R. K., Washburn, R. A., & Schoeller, D. A. (2001). Measurement of physical activity. *Quest*, 53, 295-309. doi:10.1080/00336297.2001.10491746
- Dobashi, K. (2016). Evaluation of obesity in school-age children. *Journal of Atherosclerosis and Thrombosis*, 23, 32-38. doi:10.5551/jat.29397
- Dowda, M., Pfeiffer, K. A., Lobelo, F., Porter, D. E., & Pate, R. R. (2012). Cardiorespiratory fitness and proximity to commercial physical activity facilities among 12th grade girls. *Journal of Adolescent Health*, 50, 497-502. doi:10.1016/j.jadohealth.2011.09.018
- Drake, C., Nickel, C., Burduvali, E., Roth, T., Jefferson, C., & Badia, P. (2003). The Pediatric Daytime Sleepiness Scale (PDSS): Sleep habits and school outcomes in middle-school children. *Sleep*, 26, 455-458. doi:10.1093/sleep/26.4.455
- Drenowatz, C., Kobel, S., Kettner, S., Keszyüs, D., Wirt, T., Dreyhaupt, J., & Steinacker, J. M. (2013). Correlates of weight gain in German children attending elementary school. *Preventive Medicine*, 57, 310-314. doi:10.1016/j.ypmed.2013.06.004

- Dumuid, D., Olds, T., Lewis, L. K., Martin-Fernández, J. A., Barreira, T., Broyles, S., . . . Maher, C. (2018). The adiposity of children is associated with their lifestyle behaviours: A cluster analysis of school-aged children from 12 nations. *Pediatric Obesity, 13*, 111-119. doi:10.1111/ijpo.12196
- Duncan, J. S., Schofield, G., Duncan, E. K., & Rush, E. C. (2008). Risk factors for excess body fatness in New Zealand children. *Asia Pacific Journal of Clinical Nutrition, 17*, 138-147. doi:10.6133/apjcn.2008.17.1.21
- Elmenhorst, J., Hulpke-Wette, M., Barta, C., Dalla Pozza, R., Springer, S., & Oberhoffer, R. (2015). Percentiles for central blood pressure and pulse wave velocity in children and adolescents recorded with an oscillometric device. *Atherosclerosis, 238*, 9-16. doi:10.1016/j.atherosclerosis.2014.11.005
- Emmett, P. M., & Jones, L. R. (2015). Diet, growth, and obesity development throughout childhood in the Avon longitudinal study of parents and children. *Nutrition Reviews, 73*, 175-206. doi:10.1093/nutrit/nuv054
- Engin, A. B. (2017). What is lipotoxicity? In A. B. Engin & A. Engin (Eds.), *Obesity and lipotoxicity* (pp. 197-220). Cham, Switzerland: Springer. doi:10.1007/978-3-319-48382-5_8
- Everitt, B. S., & Dunn, G. (2001). *Applied multivariate data analysis* (2nd ed.). London, England: Wiley.
- Faigenbaum, A. D., & Myer, G. D. (2012). Exercise deficit disorder in youth: Play now or pay later. *Current Sports Medicine Reports, 11*, 196–200. doi:10.1249/JSR.0b013e31825da961

- Fairchild, T. J., Klakk, H., Heidemann, M., Andersen, L. B., & Wedderkopp, N. (2016). Exploring the relationship between adiposity and fitness in young children. *Medicine & Science in Sports & Exercise*, 48, 1708-1714. doi:10.1249/MSS.0000000000000958
- Fasshauer, M., & Blüher, M. (2015). Adipokines in health and disease. *Trends in Pharmacological Sciences*, 36, 461-470. doi:10.1016/j.tips.2015.04.014
- Figueiro, M. G., Wood, B., Plitnick, B., & Rea, M. S. (2011). The impact of light from computer monitors on the melatonin levels in college students. *Biogenic Amines*, 25, 106-116.
- Fossum, I. N., Nordnes, L. T., Storemark, S. S., Bjorvatn, B., & Pallesen, S. (2014). The association between use of electronic media in bed before going to sleep and insomnia symptoms, daytime sleepiness, morningness, and chronotype. *Behavioral Sleep Medicine*, 12, 343-357. doi:10.1080/15402002.2013.819468
- Frieden, T. R., & Jaffe, M. G. (2018). Saving 100 million lives by improving global treatment of hypertension and reducing cardiovascular disease risk factors. *Journal of Clinical Hypertension*, 20, 208-211. doi:10.1111/jch.13195
- Froberg, K., & Andersen, L. B. (2005). Mini review: Physical activity and fitness and its relations to cardiovascular disease risk factors in children. *International Journal of Obesity*, 29(S2), S34-S39. Retrieved from <https://www.nature.com/articles/0803096>
- Frydenlund, G., Jørgensen, T., Toft, U., Pisinger, C., & Aadahl, M. (2012). Sedentary leisure time behavior, snacking habits and cardiovascular biomarkers: The Inter99 Study. *European Journal of Preventive Cardiology*, 19, 1111–1119. doi:10.1177/1741826711419999

- Galley, H. F., & Webster, N. R. (2004). Physiology of the endothelium. *British Journal of Anaesthesia*, 93, 105-113. doi:10.1093/bja/ae1163
- García-Prieto, J. C., Martínez-Vizcaino, V., García-Hermoso, A., Sánchez-López, M., Arias-Palencia, N., Fonseca, J. F. O., & Mora-Rodríguez, R. (2017). Energy expenditure in playground games in primary school children measured by accelerometer and heart rate monitors. *International Journal of Sport Nutrition and Exercise Metabolism*, 27(5), 1-20. doi:10.1123/ijsnem.2016-0122
- Gaya, A. R., Brand, C., Dias, A. F., Gaya, A. C. A., Lemes, V. B., & Mota, J. (2017). Obesity anthropometric indicators associated with cardiometabolic risk in Portuguese children and adolescents. *Preventive Medicine Reports*, 8, 158-162. doi:10.1016/j.pmedr.2017.10.002
- George, E. S., Rosenkranz, R. R., & Kolt, G. S. (2013). Chronic disease and sitting time in middle-aged Australian males: Findings from the 45 and Up Study. *International Journal of Behavioral Nutrition and Physical Activity*, 10(20), 1-8. doi:10.1186/1479-5868-10-20
- Glynn, L., Emmett, P., & Rogers, I. S. (2005). Food and nutrient intakes of a population sample of 7-year-old children in the south-west of England in 1999/2000—What difference does gender make? *Journal of Human Nutrition and Dietetics*, 18, 7-19. doi:10.1111/j.1365-277X.2004.00582.x
- Goel, K., Thomas, R. J., Squires, R. W., Coutinho, T., Trejo-Gutierrez, J. F., Somers, V. K., . . . Lopez-Jimenez, F. (2011). Combined effect of cardiorespiratory fitness and adiposity on mortality in patients with coronary artery disease. *American Heart Journal*, 161, 590-597. doi:10.1016/j.ahj.2010.12.012
- Goldstein, H. (2010). *Multilevel statistical models* (4th ed.). Hoboken, NJ: Wiley.

- González, K., Fuentes, J., & Márquez, J. L. (2017). Physical inactivity, sedentary behavior and chronic diseases. *Korean Journal of Family Medicine*, *38*, 111-115.
doi:10.4082/kjfm.2017.38.3.111
- Goodman, E., Li, C., Tu, Y.-K., Ford, E., Sun, S. S., & Huang, T. T.-K. (2009). Stability of the factor structure of the metabolic syndrome across pubertal development: Confirmatory factor analyses of three alternative models. *Journal of Pediatrics*, *155*, e1-e8. doi:10.1016/j.jpeds.2009.04.045
- Gopinath, B., Baur, L. A., Hardy, L. L., Kifley, A., Rose, K. A., Wong, T. Y., & Mitchell, P. (2012). Relationship between a range of sedentary behaviours and blood pressure during early adolescence. *Journal of Human Hypertension*, *26*, 350–356.
doi:10.1038/jhh.2011.40
- Gortmaker, S. L., Cheung, L. Y., Peterson, K. E., Chomitz, G., Cradle., J. H., Dart, H., . . . Laird, N. (1999). Impact of a school-based interdisciplinary intervention on diet and physical activity among urban primary school children: Eat well and keep moving. *Archives of Pediatrics & Adolescent Medicine*, *153*, 975-983. doi:10.1001/archpedi.153.9.975
- Gozal, D., Capdevila, O. S., & Kheirandish-Gozal, L. (2008). Metabolic alterations and systemic inflammation in obstructive sleep apnea among nonobese and obese prepubertal children. *American Journal of Respiratory and Critical Care Medicine*, *177*, 1142-1149. doi:10.1164/rccm.200711-1670OC
- Griel, A. E., Ruder, E. H., & Kris-Etherton, P. M. (2006). The changing roles of dietary carbohydrates: From simple to complex. *Arteriosclerosis, Thrombosis, and Vascular Biology*, *26*, 1958-1965. doi:10.1161/01.ATV.0000233384.97125.bd

- Griffiths, L. J., Sera, F., Cortina-Borja, M., Law, C., Ness, A., & Dezaux, C. (2016). Objectively measured physical activity and sedentary time: Cross-sectional and prospective associations with adiposity in the Millennium Cohort Study. *BMJ Open*, 6, 1-13. doi:10.1136/bmjopen-2015-010366
- Grøntved, A., Ried-Larsen, M., Froberg, K., Wedderkopp, N., Kristensen, P. L., Andersen, L. B., & Møller, N. C. (2013). Screen time viewing behaviors and isometric trunk muscle strength in youth. *Medicine & Science in Sports & Exercise*, 45, 1975-1980. Retrieved from <http://hdl.handle.net/11250/225245>
- Grøntved, A., Ried-Larsen, M., Møller, N. C., Kristensen, P. L., Wedderkopp, N., Froberg, K., . . . Andersen, L. B. (2014). Youth screen-time behaviour is associated with cardiovascular risk in young adulthood: The European Youth Heart Study. *European Journal of Preventive Cardiology*, 21, 49-56. doi:10.1177/2047487312454760
- Gropper, S. S., & Smith, J. L. (2012). *Advanced nutrition and human metabolism*. Belmont, CA: Cengage Learning.
- Hale, L., & Guan, S. (2015). Screen time and sleep among school-aged children and adolescents: A systematic literature review. *Sleep Medicine Reviews*, 21, 50-58. doi:10.1016/j.smrv.2014.07.007
- Hamburg, N. M., McMackin, C. J., Huang, A. L., Shenouda, S. M., Widlansky, M. E., Schulz, E., . . . Vita, J. A. (2007). Physical inactivity rapidly induces insulin resistance and microvascular dysfunction in healthy volunteers. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 27, 2650-2656. doi:10.1161/ATVBAHA.107.153288

- Hamlin, M. J., Fraser, M., Lizamore, C. A., Draper, N., Shearman, J. P., & Kimber, N. E. (2014). Measurement of cardiorespiratory fitness in children from two commonly used field tests after accounting for body fatness and maturity. *Journal of Human Kinetics, 40*, 83-92. doi:10.2478/hukin-2014-0010
- Hands, B., & Larkin, D. (2006). Physical activity measurement methods for young children: A comparative study. *Measurement in Physical Education and Exercise Science, 10*, 203-214. doi:10.1207/s15327841mpee1003_5
- Hara, M., Saitou, E., Iwata, F., Okada, T., & Harada, K. (2002). Waist-to-height ratio is the best predictor of cardiovascular disease risk factors in Japanese schoolchildren. *Journal of Atherosclerosis and Thrombosis, 9*, 127-132. doi:10.5551/jat.9.127
- Harrex, H. A., Skeaff, S. A., Black, K. E., Davison, B. K., Haszard, J. J., Meredith-Jones, K., . . . Skidmore, P.M. L. (2017). Sleep timing is associated with diet and physical activity levels in 9-11-year-old children from Dunedin, New Zealand: The PEDALS study. *Journal of Sleep Research, 27*(e12601). doi:10.1111/jsr.12634
- Hawkes, C. (2006). Uneven dietary development: Linking the policies and processes of globalization with the nutrition transition, obesity and diet-related chronic diseases. *Globalization and Health, 2*(4), 1-18. doi:10.1186/1744-8603-2-4
- Healy, G. N., & Owen, N. (2010). Sedentary behaviour and biomarkers of cardiometabolic health risk in adolescents: An emerging scientific and public health issue. *Revista Española de Cardiología (English Version), 63*, 261-264. doi:10.1016/S1885-5857(10)70057-8

- Hendel, H. W., Gotfredsen, A., Højgaard, L., Andersen, T., & Hilsted, J. (1996). Change in fat-free mass assessed by bioelectrical impedance, total body potassium and dual energy X-ray absorptiometry during prolonged weight loss. *Scandinavian Journal of Clinical and Laboratory Investigation*, *56*, 671-679. doi:10.3109/00365519609088814
- Hill, J. O., Wyatt, H. R., Reed, G. W., & Peters, J. C. (2003). Obesity and the environment: Where do we go from here? *Science*, *299*, 853-855. doi:10.1126/science.1079857
- Hirata, K., Kawakami, M., & O'Rourke, M. F. (2006). Pulse wave analysis and pulse wave velocity. *Circulation Journal*, *70*, 1231-1239. doi:10.1253/circj.70.1231
- Hjorth, M. F., Chaput, J. P., Damsgaard, C. T., Dalskov, S. M., Andersen, R., Astrup, A., . . . Sjödin, A. (2014). Low physical activity level and short sleep duration are associated with an increased cardio-metabolic risk profile: A longitudinal study in 8-11 year old Danish children. *PLOS ONE*, *9*(e104677). doi:10.1371/journal.pone.0104677
- Hjorth, M. F., Chaput, J.-P., Michaelsen, K., Astrup, A., Tetens, I., & Sjödin, A. (2013). Seasonal variation in objectively measured physical activity, sedentary time, cardio-respiratory fitness and sleep duration among 8-11 year-old Danish children: A repeated-measures study. *BMC Public Health*, *13*(808), 1-10. doi:10.1186/1471-2458-13-808
- Hjorth, M. F., Chaput, J. P., Ritz, C., Dalskov, S. M., Andersen, R., Astrup, A., . . . Sjödin, A. (2014). Fatness predicts decreased physical activity and increased sedentary time, but not vice versa: Support from a longitudinal study in 8- to 11-year-old children. *International Journal of Obesity*, *38*, 959-965. doi:10.1038/ijo.2013.229

- Hodges, E. A. (2003). A primer on early childhood obesity and parental influence. *Pediatric Nursing*, 29(1), 13-16.
- Hopkins, W. G., Marshall, S. W., Batterham, A. M., & Hanin, J. (2009). Progressive statistics for studies in sports medicine and exercise science. *Medicine and Science in Sports and Exercise*, 41(1), 3-13. doi:10.1249/MSS.0b013e31818cb278
- Hotamisligil, G. S. (2003). Inflammatory pathways and insulin action. *International Journal of Obesity*, 27, S53-S55. doi:10.1038/sj.ijo.0802502
- Howard, B. V., & Wylie-Rosett, J. (2002). Sugar and cardiovascular disease: A statement for healthcare professionals from the Committee on Nutrition of the Council on Nutrition, Physical Activity, and Metabolism of the American Heart Association. *Circulation*, 106, 523-527. doi:10.1161/01.CIR.0000019552.77778.04
- Howe, A. S., Black, K. E., Wong, J. E., Parnell, W. R., & Skidmore, P. M. (2013). Dieting status influences associations between dietary patterns and body composition in adolescents: A cross-sectional study. *Nutrition Journal*, 12, 1-10. doi:10.1186/1475-2891-12-51
- Howe, A. S., Skidmore, P. M. L., Parnell, W. R., Wong, J. E., Lubransky, A. C., & Black, K. E. (2016). Cardiorespiratory fitness is positively associated with a healthy dietary pattern in New Zealand adolescents. *Public Health Nutrition*, 19, 1279-1287. doi:10.1017/S1368980015002566
- Hubler, M. J., & Kennedy, A. J. (2016). Role of lipids in the metabolism and activation of immune cells. *Journal of Nutritional Biochemistry*, 34, 1-7. doi:10.1016/j.jnutbio.2015.11.002

- Hundal, R. S., Krssak, M., Dufour, S., Laurent, D., Lebon, V., Chandramouli, V., . . . Shulman, G. I. (2000). Mechanism by which metformin reduces glucose production in type 2 diabetes. *Diabetes*, *49*, 2063-2069. doi:10.2337/diabetes.49.12.2063
- International Obesity Task Force. (2015). *Child body mass index cut-offs*. Retrieved from <http://www.worldobesity.org/resources/child-obesity/newchildcutoffs/>
- Iroyukifujita, H., Eiichiyokoyama, K., & Yoshikawa, M. (2008). Classification and antihypertensive activity of angiotensin I-converting enzyme inhibitory peptides derived from food proteins. *Journal of Food Science*, *65*, 564-569. doi:10.1111/j.1365-2621.2000.tb16049.x
- JACC Study Group. (2010). Dietary fiber intake is associated with reduced risk of mortality from cardiovascular disease among Japanese men and women. *Journal of Nutrition*, *140*, 1445-1453. doi:10.3945/jn.110.122358
- Javed, A., Jumean, M., Murad, M. H., Okorodudu, D., Kumar, S., Somers, V. K., . . . Lopez-Jimenez, F. (2015). Diagnostic performance of body mass index to identify obesity as defined by body adiposity in children and adolescents: A systematic review and meta-analysis. *Pediatric Obesity*, *10*, 234-244. doi:10.1111/ijpo.242
- Jensen, M. D. (2006). Adipose tissue as an endocrine organ: Implications of its distribution on free fatty acid metabolism. *European Heart Journal Supplements*, *8*(Suppl. B), B13-B19. doi:10.1093/eurheartj/sul003
- Jensen, N. S. O., Camargo, T. F. B., & Bergamaschi, D. P. (2016). Comparison of methods to measure body fat in 7-to-10-year-old children: A systematic review. *Public Health*, *133*, 3-13. doi:10.1016/j.puhe.2015.11.025

- Jiang, G., & Zhang, B. B. (2003). Glucagon and regulation of glucose metabolism. *American Journal of Physiology-Endocrinology and Metabolism*, 284(4), E671-E678. doi:10.1152/ajpendo.00492.2002
- Jiménez- Pavón, D., Castillo, M. J., Moreno, L. A., Kafatos, A., Manios, Y., Kondaki, K., . . . Ruiz, J. R. (2011). Fitness and fatness are independently associated with markers of insulin resistance in European adolescents: The HELENA study. *Pediatric Obesity*, 6, 253-260. doi:10.3109/17477166.2011.575158
- Jones, P. J., & Papamandjaris, A. A. (2012). Lipids: Cellular metabolism. In J. W. Erdman, I. A. Macdonald, & S. H. Zeisel (Eds.), *Present knowledge in nutrition* (10th ed., pp. 132-148).
- Kahn, H. S., Imperatore, G., & Cheng, Y. J. (2005). A population-based comparison of BMI percentiles and waist-to-height ratio for identifying cardiovascular risk in youth. *Journal of Pediatrics*, 146, 482-488. doi:10.1016/j.jpeds.2004.12.028
- Kelly, S., & Swinburn, B. (2015). Childhood obesity in New Zealand. *New Zealand Medical Journal*, 128, 6-7. Retrieved from <https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2015/vol-128-no-1417-3-july-2015/6572>
- Kenney, W. L., Wilmore, J., & Costill, D. (2015). *Physiology of sport and exercise* (6th ed.). Champaign, IL: Human Kinetics.
- Khanna, G. L., Majumdar, P., Saha, M., & Mandal, M. (1998). Cardiorespiratory fitness and body composition in Indian children of 10-16 years. In J. Parizková & A. P. Hills (Eds.), *Physical fitness and nutrition during growth* (pp. 132-144). Basel, Switzerland: Karger. doi:10.1159/000061751

- Kkeveetil, C. V., Thomas, G., & Chander, S. J. U. (2016). Role of micronutrients in congestive heart failure: A systematic review of randomized controlled trials. *Tzu Chi Medical Journal*, 28, 143-150. doi:10.1016/j.tcmj.2016.08.001
- Kobayashi, F., Ogata, H., Omi, N., Nagasaka, S., Yamaguchi, S., Hibi, M., & Tokuyama, K. (2014). Effect of breakfast skipping on diurnal variation of energy metabolism and blood glucose. *Obesity Research & Clinical Practice*, 8, e249-e257. doi:10.1016/j.orcp.2013.01.001
- Kolodziejczyk, J. K., Merchant, G., & Norman, G. J. (2012). Reliability and validity of child/adolescent food frequency questionnaires that assess foods and/or food groups. *Journal of Pediatric Gastroenterology and Nutrition*, 55, 4-13. doi:10.1097/MPG.0b013e318251550e
- Kuipers, R. S., De Graaf, D. J., Luxwolda, M. F., Muskiet, M. H. A., Dijck-Brouwer, D. A. J., & Muskiet, F. A. J. (2011). Saturated fat, carbohydrates and cardiovascular disease. *The Netherlands Journal of Medicine*, 69, 372-378. Retrieved from <http://www.njmonline.nl/>
- Kulsum-Meccì, N., Goss, C., Kozel, B. A., Garbutt, J. M., Schechtman, K. B., & Dharnidharka, V. R. (2017). Effects of obesity and hypertension on pulse wave velocity in children. *Journal of Clinical Hypertension*, 19, 221-226. doi:10.1111/jch.12892
- Kushner, R. F. (1992). Bioelectrical impedance analysis: A review of principles and applications. *Journal of the American College of Nutrition*, 11, 199-209. Retrieved from <http://www.americancollegeofnutrition.org/content/the-journal>

- Kuzik, N., & Carson, V. (2016). The association between physical activity, sedentary behavior, sleep, and body mass index z-scores in different settings among toddlers and preschoolers. *BMC Pediatrics*, *16*, 1-8. doi:10.1186/s12887-016-0642-6
- Kyle, U. G., Bosaeus, I., De Lorenzo, A. D., Deurenberg, P., Elia, M., Gómez, J. M., . . . Pichard, C. (2004a). Bioelectrical impedance analysis—Part I: Review of principles and methods. *Clinical Nutrition*, *23*, 1226-1243. doi:10.1016/j.clnu.2004.06.004
- Kyle, U. G., Bosaeus, I., De Lorenzo, A. D., Deurenberg, P., Elia, M., Gómez, J. M., . . . Pichard, C. (2004b). Bioelectrical impedance analysis—Part II: Utilization in clinical practice. *Clinical Nutrition*, *23*, 1430-1453. doi:10.1016/j.clnu.2004.09.012
- Lambert, M., Paradis, G., O'Loughlin, J., Delvin, E., E. Hanley, J. A., & Levy, E. (2004). Insulin resistance syndrome in a representative sample of children and adolescents from Quebec, Canada. *International Journal of Obesity*, *28*, 833-841. doi:10.1038/s.j.ijo.0802694
- Lambrick, D. M., Stoner, L., Faulkner, J., & Hamlin, M. J. (2014). Preventive medicine needs to begin with our children. *International Journal of Preventive Medicine*, *5*, 129-131. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3915467/>
- LaMonte, M. J., Barlow, C. E., Jurca, R., Kampert, J. B., Church, T. S., & Blair, S. N. (2005). Cardiorespiratory fitness is inversely associated with the incidence of metabolic syndrome: A prospective study of men and women. *Circulation*, *112*, 505-512. doi:10.1161/CIRCULATIONAHA.104.503805
- Laskowski, E. R. (2012). The role of exercise in the treatment of obesity. *PM&R*, *4*, 840-844. doi:10.1016/j.pmrj.2012.09.576

- Latimer-Cheung, A. E., Copeland, J. L., Fowles, J., Zehr, L., Duggan, M., & Tremblay, M. S. (2016). The Canadian 24-hour movement guidelines for children and youth: Implications for practitioners, professionals, and organizations. *Applied Physiology, Nutrition, and Metabolism, 41*, S328-S335. doi:10.1139/apnm-2016-0086
- Lavie, C. J., Arena, R., Swift, D. L., Johannsen, N. M., Sui, X., Lee, D. C., . . . Blair, S. N. (2015). Exercise and the cardiovascular system: Clinical science and cardiovascular outcomes. *Circulation Research, 117*, 207-219. doi:10.1161/CIRCRESAHA.117.305205
- Lee, D-C., Artero, E. G., Sui, X., & Blair, S. N. (2010). Mortality trends in the general population: The importance of cardiorespiratory fitness. *Journal of Psychopharmacology, 24*(Suppl. 4), 27-35. doi:10.1177/1359786810382057
- Lee, I.-M., Djoussé, L., Sesso, H. D., Wang, L., & Buring, J. E. (2010). Physical activity and weight gain prevention. *Journal of the American Medical Association, 303*, 1173-1179. doi:10.1001/jama.2010.312
- Lee, I.-M., Shiroma, E. J., Lobelo, F., Puska, P., Blair, S. N., & Katzmarzyk, P. T. (2012). Effect of physical inactivity on major non-communicable diseases worldwide: An analysis of burden of disease and life expectancy. *The Lancet, 380*(9838), 219-229. doi:10.1016/S0140-6736(12)61031-9
- Léger, L. A., & Lambert, J. (1982). A maximal multistage 20-m shuttle run test to predict $\dot{V}O_2$ max. *European Journal of Applied Physiology and Occupational Physiology, 49*, 1-12. doi:10.1007/bf00428958
- Leitzmann, M. F., Jochem, C., & Schmid, D. (Eds.). (2017). *Sedentary behaviour epidemiology*. Cham, Switzerland: Springer. doi:10.1007/978-3-319-61552-3

- Leong, D. P., Teo, K. K., Rangarajan, S., Lopez-Jaramillo, P., Avezum, A., Jr., Orlandini, A., . . . Yusuf, S. (2015). Prognostic value of grip strength: Findings from the Prospective Urban Rural Epidemiology (PURE) study. *The Lancet*, *386*, 266-273. doi:10.1016/s0140-6736(14)62000-6
- Li, L., Pérez, A., Wu, L.-T., Ranjit, N., Brown, H. S., & Kelder, S. H. (2016). Cardiometabolic risk factors among severely obese children and adolescents in the United States, 1999–2012. *Childhood Obesity*, *12*, 12-19. doi:10.1089/chi.2015.0136
- Lie, L., Brown, L., Forrester, T. E., Plange-Rhule, J., Bovet, P., Lambert, E. V., . . . Dugas, L. R. (2018). The association of dietary fiber intake with cardiometabolic risk in four countries across the epidemiologic transition. *Nutrients*, *10*(5), 1-12. doi:10.3390/nu10050628
- Lieberman, D. (2013). *The story of the human body: Evolution, health, and disease*. New York, NY: Pantheon.
- Liu, S., & Manson, J. E. (2001). Dietary carbohydrates, physical inactivity, obesity, and the 'metabolic syndrome' as predictors of coronary heart disease. *Current Opinion in Lipidology*, *12*, 395-404. Retrieved from <https://journals.lww.com/co-lipidology/>
- Locard, E., Mamelle, N., Billette, A., Miginiac, M., Munoz, F., & Rey, S. (1992). Risk factors of obesity in a five year old population: Parental versus environmental factors. *International Journal of Obesity and Related Metabolic Disorders*, *16*, 721-729. Retrieved from <https://www.nature.com/ijo/>
- Loprinzi, P. D., & Cardinal, B. J. (2011). Measuring children's physical activity and sedentary behaviors. *Journal of Exercise Science & Fitness*, *9*(1), 15-23. doi:10.1016/S1728-869X(11)600002-6

- Lubans, D. R., Hesketh, K., Cliff, D. P., Barnett, L M., Salmon, J., Dollman, J., . . . Hardy, L. L. (2011). A systematic review of the validity and reliability of sedentary behaviour measures used with children and adolescents. *Obesity Reviews*, *12*, 781-799. doi:10.1111/j.1467-789X.2011.00896x
- Magee, C. A., Caputi, P., & Iverson, D. C. (2013). Patterns of health behaviours predict obesity in Australian children. *Journal of Paediatrics and Child Health*, *49*, 291-296. doi:10.1111/jpc.12163
- Marieb, E. N., & Hoehn, K. N. (2009). *Human anatomy and physiology* (8th ed.). San Francisco, CA: Benjamin Cummings.
- Maziak, W., Ward, K. D., & Stockton, M. B. (2008). Childhood obesity: Are we missing the big picture? *Obesity Reviews*, *35-42*. doi:10.1111/j.1467-789X.2007.00376.x
- McAuley, P. A., Artero, E. G., Sui, X., Lee, D. C., Church, T. S., Lavie, C. J., . . . Blair, S. N. (2012). The obesity paradox, cardiorespiratory fitness, and coronary heart disease. *Mayo Clinic Proceedings*, *87*, 443-451. doi:10.1016/j.mayocp.2012.01.013
- McCloskey, K., Sun, C., Pezic, A., Cochrane, J., Morley, R., Vuillermin, P., . . . Ponsonby, A.-L. (2014). The effect of known cardiovascular risk factors on carotid-femoral pulse wave velocity in school-aged children: A population based twin study. *Journal of Developmental Origins of Health and Disease*, *5*, 307-313. doi:10.1017/S2040174414000282
- McManus, A. M., Ainslie, P. N., Green, D. J., Simair, R. G., Smith, K., & Lewis, N. (2015). Impact of prolonged sitting on vascular function in young girls. *Experimental Physiology*, *100*, 1379-1387. doi:10.1113/EP085355

- McNamara, E., Hudson, Z., & Taylor, S. J. C. (2010). Measuring activity levels of young people: The validity of pedometers. *British Medical Bulletin*, *95*, 121-137.
doi:10.1093/bmb/ldq016
- Meijer, A. M., Habekothé, H. T., & Van Den Wittenboer, G. L. H. (2000). Time in bed, quality of sleep and school functioning of children. *Journal of Sleep Research*, *9*, 145-153. doi:10.1046/j.1365-2869.2000.00198.x
- Melo, X., Santa-Clara, H., Almeida, J. P., Carnero, E. A., Sardinha, L. B., Bruno, P. M., & Fernhall, B. (2011). Comparing several equations that predict peak VO₂ using the 20-m multistage-shuttle run-test in 8–10-year-old children. *European Journal of Applied Physiology*, *111*, 839-849. doi:10.1007/s00421-010-1708-z
- Meltzer, L. J., Avis, K. T., Biggs, S., Reynolds, A. C., Crabtree, V. M., & Bevens, K. B. (2013). The Children's Report of Sleep Patterns (CRSP): A self-report measure of sleep for school-aged children. *Journal of Clinical Sleep Medicine*, *9*, 235-245.
doi:10.5664/jcsm.2486
- Meltzer, L. J., Biggs, S., Reynolds, A., Avis, K. T., Crabtree, V. M., & Bevens, K. B. (2012). The Children's Report of Sleep Patterns–Sleepiness Scale: A self-report measure for school-aged children. *Sleep Medicine*, *13*, 385-389. doi:10.1016/j.sleep.2011.12.004
- Meltzer, L. J., Montgomery-Downs, H. E., Insana, S. P., & Walsh, C. M. (2012). Use of actigraphy for assessment in pediatric sleep research. *Sleep Medicine Reviews*, *16*, 463-475. doi:10.1016/j.smr.2011.10.002
- Meltzer, L. J., Wong, P., Biggs, S. N., Traylor, J., Kim, J. Y., Bhattacharjee, R., . . . Marcus, C. L. (2016). Validation of actigraphy in middle childhood. *Sleep*, *39*, 1219-1224. doi:10.5665/sleep.5836

- Mengual-Macenlle, N., Marcos, P. J., Golpe, R., & González-Rivas, D. (2015).
Multivariate analysis in thoracic research. *Journal of Thoracic Disease*, 7(3), E2-E6. doi:10.3978/j.issn.2072-1439.2015.01.43
- Meyer, A. A., Kundt, G., Steiner, M., Schuff-Werner, P., & Kienast, W. (2006). Impaired flow-mediated vasodilation, carotid artery intima-media thickening, and elevated endothelial plasma markers in obese children: the impact of cardiovascular risk factors. *Pediatrics*, 117, 1560-1567. doi:10.1542/peds.2005-2140
- Ministry of Education. (2016). *School deciles*. Retrieved from <http://www.education.govt.nz/school/running-a-school/resourcing/operational-funding/school-decile-ratings/>
- Ministry of Health. (2014). *New Zealand health survey: Annual update of key findings 2012/13*. Retrieved from <http://www.health.govt.nz/publication/new-zealand-health-survey-annual-update-key-findings-2012-13>
- Ministry of Health. (2015a). *Food and nutrition guidelines for healthy children and young people (aged 2-18 years): A background paper*. Retrieved from <http://www.health.govt.nz/publication/food-and-nutrition-guidelines-healthy-children-and-young-people-aged-2-18-years-background-paper>
- Ministry of Health. (2015b). *Obesity statistics*. Retrieved from <http://www.health.govt.nz/nz-health-statistics/health-statistics-and-data-sets/obesity-data-and-stats>
- Ministry of Health. (2015c). *Physical activity*. Retrieved from <http://www.health.govt.nz/our-work/preventative-health-wellness/physical-activity>

- Ministry of Health. (2017). *Children and young people living well and staying well: New Zealand childhood obesity programme baseline report*. Retrieved from <http://www.health.govt.nz/publication/children-and-young-people-living-well-and-staying-well-new-zealand-childhood-obesity-programme>
- Moschonis, G., Mougios, V., Papandreou, C., Lionis, C., Chrousos, G. P., Malandraki, E., & Manios, Y. (2013). “Leaner and less fit” children have a better cardiometabolic profile than their “heavier and more fit” peers: The Healthy Growth Study. *Nutrition, Metabolism and Cardiovascular Diseases*, 23, 1058-1065. doi:10.1016/j.numecd.2012.11.010
- Mozaffarian, D. (2016). Dietary and policy priorities for cardiovascular disease, diabetes, and obesity. *Circulation*, 133, 187-225. doi:10.1161/CIRCULATIONAHA.115.018585
- Mulaik, S. A. (2009). *Foundations of factor analysis* (2nd ed.). Boca Raton, FL: CRC Press.
- Mushengezi, B., & Chillo, P. (2014). Association between body fat composition and blood pressure level among secondary school adolescents in Dar es Salaam, Tanzania. *Pan African Medical Journal*, 19, 327339. doi:10.11604/pamj.2014.19.327.5222
- Myers, J. (2003). Exercise and cardiovascular health. *Circulation*, 107(1), e2-e5. doi:10.1161/01.CIR.0000048890.59383.8D
- Nakamura, K., Fuster, J. J., & Walsh, K. (2014). Adipokines: A link between obesity and cardiovascular disease. *Journal of Cardiology*, 63, 250-259. doi:10.1016/j.jjcc.2013.11.006

- Nambiar, S., Hughes, I. & Davies, P. S. W. (2010). Developing waist-to-height ratio cut-offs to define overweight and obesity in children and adolescents. *Public Health Nutrition*, 13, 1566-74. doi:10.1017/S1368980009993053
- Nambiar, S., Truby, H., Abbott, R. A., & Davies, P. S. (2009). Validating the waist-height ratio and developing centiles for use amongst children and adolescents. *Acta Paediatrica*, 98, 148-152. doi:10.1111/j.1651-2227.2008.01050.x
- National Institutes of Health. (2016). *Types of physical activity*. Retrieved from <https://www.nhlbi.nih.gov/health/health-topics/topics/phys/types>
- Navarro, T. P., Dardik, A., Junqueira, D., & Cisneros, L. (Eds.). (2017). *Vascular diseases for the non-specialist: An evidence-based guide*. Cham, Switzerland: Springer.
- Nishtar, S., Gluckman, P., & Armstrong, T. (2016). Ending childhood obesity: A time for action. *The Lancet*, 387(10021), 825-827. doi:10.1016/S0140-6736(16)00140-9
- Nocon, M., Hiemann, T., Müller-Riemenschneider, F., Thalau, F., Roll, S., & Willich, S. N. (2008). Association of physical activity with all-cause and cardiovascular mortality: A systematic review and meta-analysis. *European Journal of Preventive Cardiology*, 15, 239-246. doi:10.1097/hjr.0b013e3282f55e09
- Noonan, V., & Dean, E. (2000). Submaximal exercise testing: Clinical application and interpretation. *Physical Therapy*, 80, 782-807. doi:10.1093/ptj/80.8.782
- Ogden, C. L., Carroll, M. D., Lawman, H. G., Fryar, C. D., Kruszon-Moran, D., Kit, B. K., & Flegal, K. M. (2016). Trends in obesity prevalence among children and adolescents in the United States, 1988-1994 through 2013-2014. *Journal of the American Medical Association*, 315, 2292-2299. doi:10.1001/jama.2016.6361

- Olshansky, S. J., Passaro, D. J., Hershov, R. C., Layden, J., Carnes, B. A., Brody, J., . . . Ludwig, D. S. (2005). A potential decline in life expectancy in the United States in the 21st century. *New England Journal of Medicine*, *352*, 1138-1145. doi:10.1056/NEJMSr043743
- Organisation for Economic Co-Operation and Development. (2014). *Obesity update*. Retrieved from <http://www.oecd.org/health/Obesity-Update-2014.pdf>
- Ortega, F. B., Ruiz, J. R., Castillo, M. J., & Sjörström, M. (2008). Physical fitness in childhood and adolescence: A powerful marker of health. *International Journal of Obesity*, *32*, 1-11. doi:10.1038/sj.ijo.0803774
- Ouchi, N., Parker, J. L., Lugus, J. J., & Walsh, K. (2011). Adipokines in inflammation and metabolic disease. *Nature Reviews Immunology*, *11*, 85-97. doi:10.1038/nri2921
- Owens, J. A., Spirito, A., McGuinn, M., & Nobile, C. (2000). Sleep habits and sleep disturbance in elementary school-aged children. *Journal of Developmental & Behavioral Pediatrics*, *21*, 27-36. doi:10.1097/00004703-200002000-00004
- Paavonen, E. J., Aronen, E. T., Moilanen, I., Piha, J., Räsänen, E., Tamminen, T., & Almqvist, F. (2000). Sleep problems of school-aged children: A complementary view. *Acta Paediatrica*, *89*, 223-228. doi:10.1111/j.1651-2227.2000.tb01220.x
- Pandey, A., Cornwell, W. K., Willis, B., Neeland, I. J., Gao, A., Leonard, D., . . . Berry, J. D. (2017). Body mass index and cardiorespiratory fitness in mid-life and risk of heart failure hospitalization in older age. *JACC Heart Failure*, *5*, 367-374. doi:10.1016/j.jchf.2016.12.021

- Parikh, P., Mochari, H., & Mosca, L. (2009). Clinical utility of a fingerstick technology to identify individuals with abnormal blood lipids and high-sensitivity C-reactive protein levels. *American Journal of Health Promotion, 23*, 279-282. doi:10.4278/ajhp.071221140
- Pate, R. R., Pratt, M., Blair, S. N., Haskell, W. L., Macera, C. A. Bouchard, C., . . . Wilmore, J. H. (1995). Physical activity and public health: A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *Journal of the American Medical Association, 273*, 402-407. doi:10.1001/jama.1995.03520290054029
- Peltz, G., Aguirre, M. T., Sanderson, M., & Fadden, M. K. (2010). The role of fat mass index in determining obesity. *American Journal of Human Biology, 22*, 639-647. doi:10.1002/ajhb.21056
- Pereira, M. A., & Liu, S. (2003). Types of carbohydrates and risk of cardiovascular disease. *Journal of Women's Health, 12*, 115-122. doi:10.1089/154099903321576501
- Pereira, S. M. P., Ki, M., & Power, C. (2012). Sedentary behaviour and biomarkers for cardiovascular disease and diabetes in mid-life: The role of television-viewing and sitting at work. *PLOS ONE, 7*(e31132), 2-9. doi:10.1371/journal.pone.0031132
- Pietinen, P., Rimm, E. B., Korhonen, P., Hartman, A. M., Willett, W. C., Albanes, D., & Virtamo, J. (1996). Intake of dietary fiber and risk of coronary heart disease in a cohort of Finnish men: The Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study. *Circulation, 94*, 2720-2727. doi:10.1161/01.CIR.94.11.2720

- Potischman, N., Cohen, B. E., & Picciano, M. F. (2006). Dietary recommendations and identified research needs for The National Children's Study. *Journal of Nutrition*, *136*, 686-689. doi:10.1093/jn/136.3.686
- Powell, L. M., Auld, M. C., Chaloupka, F. J., O'Malley, P. M., & Johnston, L. D. (2007). Associations between access to food stores and adolescent body mass index. *American Journal of Preventive Medicine*, *33*(4), S301-S307. doi:10.1016/j.amepre.2007.07.007
- Press, V., Freestone, I., & George, C. F. (2003). Physical activity: The evidence of benefit in the prevention of coronary heart disease. *QJM: An International Journal of Medicine*, *96*, 245-251. doi:10.1093/qjmed/hcg041
- Pulsford, R. M., Stamatakis, E., Britton, A. R., Brunner, E. J., & Hillsdon, M. M. (2013). Sitting behavior and obesity: Evidence from the Whitehall II study. *American Journal of Preventive Medicine*, *44*, 132-138. doi:10.1016/j.amepre.2012.10.009
- Rabe, K., Lehrke, M., Parhofer, K. G., & Broedl, U. C. (2008). Adipokines and insulin resistance. *Molecular Medicine*, *14*, 741. doi:10.2119/2008-00058.Rabe
- Ramírez-Marrero, F. A., Smith, B. A., Kirby, T. E., Leenders, N., & Sherman, W. M. (2002). Evaluation of a step-counter during treadmill walking in 7-12 year old African-American children. *Journal of National Black Nurses' Association*, *13*(1), 1-5. Retrieved from <https://www.nbna.org/nbna%20journal>
- Ramírez-Vélez, R., Peña-Ibagon, J. C., Martínez-Torres, J., Tordecilla-Sanders, A., Correa-Bautista, J. E., Lobelo, F., & García-Hermoso, A. (2017). Handgrip strength cutoff for cardiometabolic risk index among Colombian children and adolescents: The FUPRECOL Study. *Scientific Reports*, *7*(42622), 1-7. doi:10.1038/srep42622

- Ramírez-Vélez, R., Silva-Moreno, C., Correa-Bautista, J. E., González-Ruíz, K., Prieto-Benavides, D. H., Villa-González, E., & García-Hermoso, A. (2017). Self-rated health status and cardiorespiratory fitness in a sample of schoolchildren from Bogotá, Colombia. The FUPRECOL Study. *International Journal of Environmental Research and Public Health*, *14*, 1-14. doi:10.3390/ijerph14090952
- Ras, R. T., Streppel, M. T., Draijer, R., & Zock, P. L. (2013). Flow-mediated dilation and cardiovascular risk prediction: A systematic review with meta-analysis. *International Journal of Cardiology*, *168*, 344-351. doi:10.1016/j.ijcard.2012.09.047
- Reddy, K. S., & Katan, M. B. (2004). Diet, nutrition and the prevention of hypertension and cardiovascular diseases. *Public Health Nutrition*, *7*, 167-186. doi:10.1079/PHN2003587
- Redinger, R. N. (2007). The pathophysiology of obesity and its clinical manifestations. *Gastroenterology & Hepatology*, *3*, 856-863. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3104148/>
- Reilly, J. J., & Kelly, J. (2011). Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: Systematic review. *International Journal of Obesity*, *35*, 891-898. doi:10.1038/ijo.2010.222
- Restaino, R. M., Holwerda, S. W., Credeur, D. P., Fadel, P. J., & Padilla, J. (2015). Impact of prolonged sitting on lower and upper limb micro-and macrovascular dilator function. *Experimental Physiology*, *100*, 829-838. doi:10.1113/EP085238
- Reusz, G. S., Cseprekal, O., Temmar, M., Kis, É., Cherif, A. B., Thaleb, A., . . . Salvi, P. (2010). Reference values of pulse wave velocity in healthy children and teenagers. *Hypertension*, *56*, 217-224. doi:10.1161/hypertensionaha.110.152686

- Richter, C. K., Skulas-Ray, A. C., Champagne, C. M., & Kris-Etherton, P. M. (2015). Plant protein and animal proteins: Do they differentially affect cardiovascular disease risk? *Advances in Nutrition*, 6, 712-728. doi:10.3945/an.115.009654
- Riddoch, C. J., Mattocks, C., Deere, K., Saunders, J., Kirkby, J., Tilling, K., . . . Ness, A. R. (2007). Objective measurement of levels and patterns of physical activity. *Archives of Disease in Childhood*, 92, 963-969. doi:10.1136/adc.2006.112136
- Roenneberg, T., Allebrandt, K. V., Merrow, M., & Vetter, C. (2012). Social jetlag and obesity. *Current Biology*, 22, 939-943. doi:10.1016/j.cub.2012.03.038
- Roman, M. J., Devereux, R. B., Kizer, J. R., Lee, E. T., Galloway, J. M., Ali, T., . . . Howard, B. V. (2007). Central pressure more strongly relates to vascular disease and outcome than does brachial pressure. *Hypertension*, 50, 197-203. doi:10.1161/hypertensionaha.107.089078
- Romieu, I., Dossus, L., Barquera, S., Blotière, H. M., Franks, P. W., Gunter, M., . . . Willett, W. C. (2017). Energy balance and obesity: What are the main drivers? *Cancer Causes & Control*, 28, 247-258. doi:10.1007/s10552-017-0869-z
- Ross, R., Blair, S. N., Arena, R., Church, T. S., Després, J.-P., Franklin, B. A., . . . Wisloff, U. (2016). Importance of assessing cardiorespiratory fitness in clinical practice: A case for fitness as a clinical vital sign: A scientific statement from the American Heart Association. *Circulation*, 136, e1-e48. doi:10.1161/CIR.0000000000000461
- Ross, R., & Glomset, J. A. (1976). The pathogenesis of atherosclerosis. *New England Journal of Medicine*, 295, 369-377. doi:10.1056/NEJM197608122950707
- Rowlands, A. V., & Eston, R. G. (2007). The measurement and interpretation of children's physical activity. *Journal of Sports Science & Medicine*, 6, 270-276. Retrieved from <https://jssm.org/vol6/n3/1/v6n3-1pdf.pdf>

- Roy, N., & Sharma, R. (2016). Relationship between height, weight, BMI, waist circumference, hip circumferences, waist-to-hip ratio and waist-to-height ratio among male school children. *International Journal of Physical Education, Sports and Health*, 3, 614-617. Retrieved from <http://www.kheljournal.com/archives/2016/vol3issue3/PartG/3-3-74-334.pdf>
- Ruiz, J. R., Cavero-Redondo, I., Ortega, F. B., Welk, G. J., Andersen, L. B., & Martinez-Vizcaino, V. (2016). Cardiorespiratory fitness cut points to avoid cardiovascular disease risk in children and adolescents; What level of fitness should raise a red flag? A systematic review and meta-analysis. *British Journal of Sports Medicine*, 50, 1451-1458. doi:10.1136/bjsports-2015-095903
- Ruiz, J. R., Ortega, F. B., Castillo, R., Martín-Matillas, M., Kwak, L., Vicente-Rodríguez, G., . . . Moreno, L. A. (2010). Physical activity, fitness, weight status, and cognitive performance in adolescents. *Journal of Pediatrics*, 157, 917-922. doi:10.1016/j.jpeds.2010.06.026
- Rush, E. C., Puniani, K., Valencia, M. E., Davies, P. S. W., & Plank, L. D. (2003). Estimation of body fatness from body mass index and bioelectrical impedance: Comparison of New Zealand European, Maori and Pacific Island children. *European Journal of Clinical Nutrition*, 57, 1394-1401. doi:10.1038/sj.ejcn.1601701
- Sadeh, A., Hauri, P. J., Kripke, D. F., & Lavie, P. (1995). The role of actigraphy in the evaluation of sleep disorders. *Sleep*, 18, 288-302. doi:10.1093/sleep/18.4.288
- Saeedi, P., Skeaff, S. A., Wong, J. E., & Skidmore, P. M. L. (2016). Reproducibility and relative validity of a short food frequency questionnaire in 9-10 year-old children. *Nutrients*, 8(5), 1-13. doi:10.3390/nu8050271

- Sahoo, K., Sahoo, B., Choudhury, A. K., Sofi, N. Y., Kumar, R., & Bhadoria, A. S. (2015). Childhood obesity: Causes and consequences. *Journal of Family Medicine and Primary Care*, 4, 187-192. doi:10.4103/2249-4863.154628
- Sallis, J. F. (2010). Measuring physical activity: Practical approaches for program evaluation in Native American communities. *Journal of Public Health Management and Practice*, 16, 404-410. doi:10.1097/PHH.0b013e3181d52804
- Saltiel, A. R., & Kahn, C. R. (2001). Insulin signalling and the regulation of glucose and lipid metabolism. *Nature*, 414, 799-806. doi:10.1038/414799a
- Sandvik, C., De Bourdeaudhuij, I., Due, P., Brug, J., Wind, M., Bere, E., . . . Klepp, K.-I. (2005). Personal, social and environmental factors regarding fruit and vegetable intake among schoolchildren in nine European countries. *Annals of Nutrition and Metabolism*, 49, 255-266. doi:10.1159/000087332
- Sandvik, L., Erikssen, J., Thaulow, E., Erikssen, G., Mundal, R., & Rodahl, K. (1993). Physical fitness as a predictor of mortality among healthy, middle-aged Norwegian men. *New England Journal of Medicine*, 328, 533-537. doi:10.1056/NEJM199302253280803
- Sardinha, L. B., Santos, D. A., Silva, A. M., Grøntved, A., Andersen, L. B., & Ekelund, U. (2016). A comparison between BMI, waist circumference, and waist-to-height ratio for identifying cardio-metabolic risk in children and adolescents. *PLOS ONE*, 11(e0149351), 1-14. doi:10.1371/journal.pone.0149351

- Saris, W. H. M., Astrup, A., Prentice, A. M., Zunft, H. J. F., Formiguera, X., Verboeket-Van De Venne, W. P. H. G., . . . Keogh, G. F. (2000). Randomized controlled trial of changes in dietary carbohydrate/fat ratio and simple vs complex carbohydrates on body weight and blood lipids: The CARMEN study. *International Journal of Obesity*, *24*, 1310-1318. doi:10.1038/sj.ijo.0801451
- Sarmadi, B. H., & Ismail, A. (2010). Antioxidative peptides from food proteins: A review. *Peptides*, *31*, 1949-1956. doi:10.1016/j.peptides.2010.06.020
- Saunders, T. J., Gray, C. E., Poitras, V. J., Chaput, J.-P., Janssen, I., Katzmarzyk, P. T., . . . Carson, V. (2016). Combinations of physical activity, sedentary behaviour and sleep: Relationships with health indicators in school-aged children and youth. *Applied Physiology, Nutrition, and Metabolism*, *41*, S283-S293. doi:10.1139/apnm-2015-0626
- Savage, J. S., Fisher, J. O., & Birch, L. L. (2007). Parental influence on eating behavior: Conception to adolescence. *Journal of Law, Medicine & Ethics*, *35*, 22-34. doi:10.1111/j.1748-720X.2007.00111.x
- Savva, S. C., Tornaritis, M., Savva, M. E., Kourides, Y., Panagi, A., Silikiotou, N., . . . Kafatos, A. (2000). Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. *International Journal of Obesity*, *24*, 1453-1458. doi:10.1038/sj.ijo.0801401
- Sayin, F. K., & Buyukinan, M. (2016). Sleep duration and media time have a major impact on insulin resistance and metabolic risk factors in obese children and adolescents. *Childhood Obesity*, *12*, 272-278. doi:10.1089/chi.2015.0126

- Schechter, M. S. (2002). Technical report: Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics*, *109*(4), 1-20. Retrieved from <http://pediatrics.aappublications.org/content/109/4/e69.full>
- Scherfeld, D., Kahya, N., & Schwille, P. (2003). Lipid dynamics and domain formation in model membranes composed of ternary mixtures of unsaturated and saturated phosphatidylcholines and cholesterol. *Biophysical Journal*, *85*, 3758-3768. doi:10.1016/S0006-3495(03)74791-2
- Schmidt, M. D., Magnussen, C. G., Rees, E., Dwyer, T., & Venn, A. J. (2016). Childhood fitness reduces the long-term cardiometabolic risks associated with childhood obesity. *International Journal of Obesity*, *40*, 1134-1140. doi:10.1038/ijo.2016.61
- Schulze, M. B., Hoffmann, K., Kroke, A., & Boeing, H. (2003). An approach to construct simplified measures of dietary patterns from exploratory factor analysis. *British Journal of Nutrition*, *89*, 409-419. doi:10.1079/bjn2002778
- Seidell, J. C. (2010). Waist circumference and waist/hip ratio in relation to all-cause mortality, cancer and sleep apnea. *European Journal of Clinical Nutrition*, *64*, 35-41. doi:10.1038/ejcn.2009.71
- Sekine, M., Yamagami, T., Handa, K., Saito, T., Nanri, S., Kawaminami, K., . . . Kagamimori, S. (2002). A dose–response relationship between short sleeping hours and childhood obesity: Results of the Toyama Birth Cohort Study. *Child: Care, Health and Development*, *28*, 163-170. doi:10.1046/j.1365-2214.2002.00260.x
- Sheldrick, M. P., Tyler, R., Mackintosh, K. A., & Stratton, G. (2018). Relationship between sedentary time, physical activity and multiple lifestyle factors in children. *Journal of Functional Morphology and Kinesiology*, *3*(15), 1-16. doi:10.3390/jfmk3010015

- Sherman, D. L. (2000). Exercise and endothelial function. *Coronary Artery Disease, 11*, 117-122. doi:10.1097/00019501-200003000-00005
- Sibley, B. A., & Etnier, J. L. (2003). The relationship between physical activity and cognition in children: A meta-analysis. *Pediatric Exercise Science, 15*, 243-256. doi:10.1123/pes.15.3.243
- Skidmore, P. M., Howe, A. S., Polak, M. A., Wong, J. E., Lubransky, A., Williams, S. M., & Black, K. E. (2013). Sleep duration and adiposity in older adolescents from Otago, New Zealand: Relationships differ between boys and girls and are independent of food choice. *Nutrition Journal, 12*, 1-10. doi:10.1186/1475-2891-12-128
- Slavin, J. L., & Lloyd, B. (2012). Health benefits of fruits and vegetables. *Advances in Nutrition, 3*, 506-516. doi:10.3945/an.112.002154
- Slicker, J., & Vermilyea, S. (2009). Pediatric parenteral nutrition: Putting the microscope on macronutrients and micronutrients. *Nutrition in Clinical Practice, 24*, 481-486. doi:10.1177/0884533609339073
- Smith, K. R., Corvalán, C. F., & Kjellström, T. (1999). How much global ill health is attributable to environmental factors? *Epidemiology, 10*, 573-584. Retrieved from <https://www.jstor.org/stable/3703353>
- Smith, J. J., Eather, N., Morgan, P. J., Plotnikoff, R. C., Faigenbaum, A. D., & Lubans, D. R. (2014). The health benefits of muscular fitness for children and adolescents: A systematic review and meta-analysis. *Sports Medicine, 44*, 1209-1223. doi:10.1007/s40279-014-0196-4
- Smith, D. L., & Fernhall, B. (2011). *Advanced cardiovascular exercise physiology*. Champaign, IL: Human Kinetics.

- Sorensen, K. E., Celermajer, D. S., Georgakopoulos, D., Hatcher, G., Betteridge, D. J., & Deanfield, J. E. (1994). Impairment of endothelium-dependent dilation is an early event in children with familial hypercholesterolemia and is related to the lipoprotein(a) level. *Journal of Clinical Investigation*, *93*, 50-55. doi:10.1172/jci116983
- Spilsbury, J. C., Drotar, D., Rosen, C. L., & Redline, S. (2007). The Cleveland Adolescent Sleepiness Questionnaire: A new measure to assess excessive daytime sleepiness in adolescents. *Journal of Clinical Sleep Medicine*, *3*, 603-612. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2045721/pdf/jcsm.3.6.603.pdf>
- Sprague, B., Chesler, N. C., & Magness, R. R. (2010). Shear stress regulation of nitric oxide production in uterine and placental artery endothelial cells: Experimental studies and hemodynamic models of shear stress forces on endothelial cells. *International Journal of Developmental Biology*, *54*, 331-339. doi:10.1387/ijdb.082832bs
- Statistics New Zealand. (2015). *New Zealand in profile: 2015*. Retrieved from http://www.stats.govt.nz/browse_for_stats/snapshots-of-nz/nz-in-profile-2015.aspx
- Stein, J. H., Korcarz, C. E., Hurst, R. T., Lonn, E., Kendall, C. B., Mohler, E. R., . . . Post, W. S. (2008). Use of carotid ultrasound to identify subclinical vascular disease and evaluate cardiovascular disease risk: A consensus statement from the American Society of Echocardiography Carotid Intima-Media Thickness Task Force endorsed by the Society for Vascular Medicine. *Journal of the American Society of Echocardiography*, *21*, 93-111. doi:10.1016/j.echo.2007.11.011

- Stevens, J., Cai, J., Evenson, K. R., & Thomas, R. (2002). Fitness and fatness as predictors of mortality from all causes and from cardiovascular disease in men and women in the Lipid Research Clinics Study. *American Journal of Epidemiology*, *156*, 832-841. doi:10.1093/aje/kwf114
- Stoner, L., Credeur, D., Dolbow, D. R., & Gater, D. R. (2015). Vascular health toolbox for spinal cord injury: Recommendations for clinical practice. *Atherosclerosis*, *243*, 373-382. doi:10.1016/j.atherosclerosis.2015.10.013
- Stoner, L., Gaffney, K., Wadsworth, D., & Page, R. (2014). Should obesity be considered a disease? *Perspectives in Public Health*, *134*, 314-315. doi:10.1177/1757913914551917
- Stoner, L., Lambrick, D. M., Faulkner, J., & Young, J. (2013). Guidelines for the use of pulse wave analysis in adults and children. *Journal of Atherosclerosis and Thrombosis*, *20*, 404-406. doi:10.5551/jat.16295
- Stoner, L., Lambrick, D. M., Westrupp, N., Young, J., & Faulkner, J. (2014). Validation of oscillometric pulse wave analysis measurements in children. *American Journal of Hypertension*, *27*, 865-872. doi:10.1093/ajh/hpt243
- Stoner, L., Matheson, A., Hamlin, M., & Skidmore, P. (2016). Environmental determinants of childhood obesity: A specific focus on Māori and Pasifika in New Zealand. *Perspectives in Public Health*, *136*, 18-20. doi:10.1177/1757913915616734
- Stoner, L., Rowlands, D., Morrison, A., Credeur, D., Hamlin, M., Gaffney, K., . . . Matheson, A. (2016). Efficacy of exercise intervention for weight loss in overweight and obese adolescents: Meta-analysis and implications. *Sports Medicine*, *46*, 1737-1751. doi:10.1007/s40279-016-0537-6

- Stoner, L., Stoner, K. R., Young, J. M., & Fryer, S. (2012). Preventing a cardiovascular disease epidemic among indigenous populations through lifestyle changes. *International Journal of Preventive Medicine*, 3, 230-240. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3354392/>
- Stoner, L., Weatherall, M., Skidmore, P., Castro, N., Lark, S., Faulkner, J., & Williams, M. A. (2017). Cardiometabolic risk variables in preadolescent children: A factor analysis. *Journal of the American Heart Association*, 6(e007071). doi:10.1161/JAHA.117.007071
- Stratton, G., Canoy, D., Boddy, L. M., Taylor, S. R., Hackett, A. F., & Buchan, I. E. (2007). Cardiorespiratory fitness and body mass index of 9-11-year-old English children: A serial cross-sectional study from 1998 to 2004. *International Journal of Obesity*, 31, 1172-1178. doi:10.1038/sj.ijo.0803562
- Streppel, M. T., Ocké, M. C., Boshuizen, H. C., Kok, F. J., & Kromhout, D. (2008). Dietary fiber intake in relation to coronary heart disease and all-cause mortality over 40 y: The Zutphen Study. *American Journal of Clinical Nutrition*, 88, 1119-1125. doi:10.1093/ajcn/88.4.1119
- Tang, W. W., Kitai, T., & Hazen, S. L. (2017). Gut microbiota in cardiovascular health and disease. *Circulation Research*, 120, 1183-1196. doi:10.1161/CIRCRESAHA.117.309715
- Tedgui, A., & Mallat, Z. (1999). Atherosclerotic plaque formation. *La Revue du Praticien*, 49, 2081-2086. Retrieved from www.larevuedupraticien.fr/

- Thijssen, D. H., Black, M. A., Pyke, K. E., Padilla, J., Atkinson, G., Harris, R. A., . . . Green, D. J. (2011). Assessment of flow-mediated dilation in humans: A methodological and physiological guideline. *American Journal of Physiology-Heart and Circulatory Physiology*, *300*, H2-H12. doi:10.1152/ajpheart.00471.2010
- Thosar, S. S., Johnson, B. D., Johnston, J. D., & Wallace, J. P. (2012). Sitting and endothelial dysfunction: The role of shear stress. *Medical Science Monitor*, *18*, RA173-RA180. doi:10.12659/MSM.883589
- Threapleton, D. E., Greenwood, D. C., Evans, C. E., Cleghorn, C. L., Nykjaer, C., Woodhead, C., . . . Burley, V. J. (2013). Dietary fibre intake and risk of cardiovascular disease: Systematic review and meta-analysis. *BMJ*, *347*(f6879), 1-12. doi:10.1136/bmj.f6879
- Thurn, D., Doyon, A., Sözeri, B., Bayazit, A. K., Canpolat, N., Duzova, A., . . . Melk, A. (2015). Aortic pulse wave velocity in healthy children and adolescents: Reference values for the vicorder device and modifying factors. *American Journal of Hypertension*, *28*, 1480-1488. doi:10.1093/ajh/hpv048
- Trandafir, L. M., Frasinariu, O. E., Corciovă, C., Boiculese, L. V., & Moscalu, M. (2017, June). *Prevention of cardiovascular risk factors in childhood obesity*. Paper presented at the E-Health and Bioengineering Conference (EHB), Sinaia, Romania. doi:10.1109/EHB.2017.7995524
- Trayhurn, P., & Wood, I. S. (2004). Adipokines: Inflammation and the pleiotropic role of white adipose tissue. *British Journal of Nutrition*, *92*, 347-355. doi:10.1079/BJN20041213

- Tremblay, M. S., Colley, R. C., Saunders, T. J., Healy, G. N., & Owen, N. (2010). Physiological and health implications of a sedentary lifestyle. *Applied Physiology, Nutrition, and Metabolism*, *35*, 725-740. doi:10.1139/H10-079
- Tremblay, M. S., LeBlanc, A. G., Janssen, I., Kho, M. E., Hicks, A., Murumets, K., . . . Duggan, M. (2011). Canadian sedentary behaviour guidelines for children and youth. *Applied Physiology, Nutrition, and Metabolism*, *36*, 59-64. doi:10.1139/H11-012
- Trumbo, P., Schlicker, S., Yates, A. A., & Poos, M. (2002). Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. *Journal of the American Dietetic Association*, *102*, 1621-1630. doi:10.1016/S0002-8223(02)90346-9
- U.S. Food and Drug Administration. (2018a). *Nutrition facts label programs and materials*. Retrieved from <https://www.fda.gov/Food/LabelingNutrition/ucm20026097.htm>
- U.S. Food and Drug Administration. (2018b). *Vitamins*. Retrieved from https://www.accessdata.fda.gov/scripts/InteractiveNutritionFactsLabel/factsheets/Vitamin_and_Mineral_Chart.pdf
- Urbina, E. M., Williams, R. V., Alpert, B. S., Collins, R. T., Daniels, S. R., Hayman, L., . . . McCrindle, B. (2009). Noninvasive assessment of subclinical atherosclerosis in children and adolescents. *Hypertension*, *54*, 919-950. doi:10.1161/hypertensionaha.109.192639
- Van de Water, A. T. M., Holmes, A., & Hurley, D. A. (2011). Objective measurements of sleep for non-laboratory settings as alternatives to polysomnography—A systematic review. *Journal of Sleep Research*, *20*, 183-200. doi:10.1111/j.1365-2869.2009.00814.x

- Van der Ploeg, H. P., Chey, T., Korda, R. J., Banks, E., & Bauman, A. (2012). Sitting time and all-cause mortality risk in 222 497 Australian adults. *Archives of Internal Medicine*, *172*, 494-500. doi:10.1001/archinternmed.2011.2174
- Van der Ploeg, H. P., & Hillsdon, M. (2017). Is sedentary behaviour just physical inactivity by another name? *International Journal of Behavioral Nutrition and Physical Activity*, *14*(142), 1-8. doi:10.1186/s12966-017-0601-0
- Vanhecke, T. E., Franklin, B. A., Zalesin, K. C., Sangal, R. B., DeJong, A. T., Agrawal, V., & McCullough, P. A. (2008). Cardiorespiratory fitness and obstructive sleep apnea syndrome in morbidly obese patients. *Chest*, *134*, 539-545. doi:10.1378/chest.08-0567
- Van Herpen, N. A., & Schrauwen-Hinderling, V. B. (2008). Lipid accumulation in non-adipose tissue and lipotoxicity. *Physiology & Behavior*, *94*, 231-241. doi:10.1016/j.physbeh.2007.11.049
- Van Hoye, A., Nicaise, V., & Sarrazin, P. (2014). Self-reported and objective physical activity measurement by active youth. *Science & Sports*, *29*, 78-87. doi:10.1016/j.scispo.2013.01.010
- Vanhoutte, P. M., Shimokawa, H., Tang, E. H. C., & Feletou, M. (2009). Endothelial dysfunction and vascular disease. *Acta Physiological*, *196*, 193-222. doi:10.1111/j.1748-1716.2009.01964.x
- Vaulont, S., Vasseur-Cognet, M., & Kahn, A. (2000). Glucose regulation of gene transcription. *Journal of Biological Chemistry*, *275*, 31555-31558. doi:10.1074/jbc.R000016200.

- Verloigne, M., Van Lippevelde, W., Maes, L., Yildirim, M., Chinapaw, M., Manios, Y., . . .
- De Bourdeaudhuij, I. (2012). Levels of physical activity and sedentary time among 10- to 12-year-old boys and girls across 5 European countries using accelerometers: An observational study within the ENERGY-project. *International Journal of Behavioral Nutrition and Physical Activity*, 9(34), 1-8. doi:10.1186/1479-5868-9-34
- Viitasalo, A., Lakka, T. A., Laaksonen, D. E., Savonen, K., Lakka, H.-M., Hassinen, M., . . .
- . Laukkanen, J. A. (2014). Validation of metabolic syndrome score by confirmatory factor analysis in children and adults and prediction of cardiometabolic outcomes in adults. *Diabetologia*, 57, 940-949. doi:10.1007/s00125-014-3172-5
- Vlachopoulos, C., Aznaouridis, K., O'Rourke, M. F., Safar, M. E., Baou, K., & Stefanadis, C. (2010). Prediction of cardiovascular events and all-cause mortality with central haemodynamics: A systematic review and meta-analysis. *European Heart Journal*, 31, 1865-1871. doi:10.1093/eurheartj/ehq024
- Von Elm, E., Altman, D. G., Egger, M., Pocock, S. J., Gøtzsche, P. C., & Vandenbroucke, J. P. (2007). The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: Guidelines for reporting observational studies. *The Lancet*, 370(9596), 1453-1457. doi:10.1016/S0140-6736(07)61602-X
- Von Elm, E., Altman, D. G., Egger, M., Pocock, S. J., Gøtzsche, P. C., Vandenbroucke, J. P., & STROBE Initiative. (2014). The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: Guidelines for reporting observational studies. *International Journal of Surgery*, 12, 1495-1499. doi:10.1016/j.ijsu.2014.07.013

- Von Haehling, S., Doehner, W., & Anker, S. D. (2007). Nutrition, metabolism, and the complex pathophysiology of cachexia in chronic heart failure. *Cardiovascular Research*, *73*, 298-309. doi:10.1016/j.cardiores.2006.08.018
- Wang, Z., & Nakayama, T. (2010). Inflammation, a link between obesity and cardiovascular disease. *Mediators of Inflammation*, *2010*(535918), 1-17. doi:10.1155/2010/535918
- Warren, T. Y., Barry, V., Hooker, S. P., Sui, X., Church, T. S., & Blair, S. N. (2010). Sedentary behaviors increase risk of cardiovascular disease mortality in men. *Medicine and Science in Sports and Exercise*, *42*, 879-885. doi:10.1249/MSS.0b013e3181c3aa7e
- Waters, E., De Silva-Sanigorski, A., Burford, B. J., Brown, T., Campbell, K. J., Gao, Y., . . . Summerbell, C. D. (2011). Interventions for preventing obesity in children. *Cochrane Database of Systematic Reviews*, *12*, 1-226. doi:10.1002/14651858.CD001871.pub3
- Wearing, S. C., Hennig, E. M., Byrne, N. M., Steele, J. R., & Hills, A. P. (2006). Musculoskeletal disorders associated with obesity: A biomechanical perspective. *Obesity Reviews*, *7*, 239-250. doi:10.1111/j.1467-789X.2006.00251.x
- Weber, D. R., Leonard, M. B., & Zemel, B. S. (2012). Body composition analysis in the pediatric population. *Pediatric Endocrinology Reviews*, *10*, 130-139. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4154503/>

- Williams, A. J., Henley, W. E., Williams, C. A., Hurst, A. J., Logan, S., & Wyatt, K. M. (2013). Systematic review and meta-analysis of the association between childhood overweight and obesity and primary school diet and physical activity policies. *International Journal of Behavioral Nutrition and Physical Activity*, *10*, 1-22. doi:10.1186/1479-5868-10-101
- Wilson, A. C., Urbina, E., Witt, S. A., Glascock, B. J., Kimball, T. R., & Mitsnefes, M. (2008). Flow-mediated vasodilatation of the brachial artery in children with chronic kidney disease. *Pediatric Nephrology*, *23*, 1297-1302. doi:10.1007/s00467-008-0815-0
- Wilson, R. P. (2003). Amino acids and proteins. In J. E. Halver & R. W. Hardy (Eds.), *Fish Nutrition* (3rd ed., pp. 143-179). San Diego, CA: Academic Press.
- Wittmann, M., Dinich, J., Merrow, M., & Roenneberg, T. (2006). Social jetlag: Misalignment of biological and social time. *Chronobiology International*, *23*, 497-509. doi:10.1080/07420520500545979
- Wolfson, A. R., Carskadon, M. A., Acebo, C., Seifer, R., Fallone, G., Labyak, S. E., & Martin, J. L. (2003). Evidence for the validity of a sleep habits survey for adolescents. *Sleep*, *26*, 213-216. doi:10.1093/sleep/26.2.213
- Wong, J. E., Parnell, W., Black, K. E., & Skidmore, P. M. (2012). Reliability and relative validity of a food frequency questionnaire to assess food group intakes in New Zealand adolescents. *Nutrition Journal*, *11*, 1-9. doi:10.1186/1475-2891-11-65
- Wong, J. E., Parnell, W. R., Howe, A. S., Black, K. E., & Skidmore, P. M. (2013). Development and validation of a food-based diet quality index for New Zealand adolescents. *BMC Public Health*, *13*, 1-10. doi:10.1186/1471-2458-13-562

- World Cancer Research Fund & American Institute for Cancer Research. (2007). *Food, nutrition, physical activity, and the prevention of cancer: A global perspective*. Washington, DC: American Institute for Cancer Research. Retrieved from <http://discovery.ucl.ac.uk/4841/1/4841.pdf>
- World Health Organization. (2011). *Waist circumference and waist-hip ratio: Report of a WHO expert consultation*. Retrieved from http://apps.who.int/iris/bitstream/10665/44583/1/9789241501491_eng.pdf?ua=1
- World Health Organization. (2015). *Healthy diet*. Retrieved from <http://www.who.int/mediacentre/factsheets/fs394/en/>
- World Health Organization. (2017a). *Cardiovascular diseases (CVD)*. Retrieved from <http://www.who.int/mediacentre/factsheets/fs317/en/>
- World Health Organization. (2017b). *Noncommunicable diseases*. Retrieved from <http://www.who.int/mediacentre/factsheets/fs355/en/>
- World Health Organization. (2017c). *Obesity and overweight*. Retrieved from <http://www.who.int/mediacentre/factsheets/fs311/en/>
- World Health Organization. (2017d). *Physical activity*. Retrieved from <http://www.who.int/mediacentre/factsheets/fs385/en/>
- World Health Organization. (2018a). *BMI-for-age (5-19 years)*. Retrieved from http://www.who.int/growthref/who2007_bmi_for_age/en/
- World Health Organization. (2018b). *Physical activity*. Retrieved from http://www.who.int/topics/physical_activity/en/
- World Health Organization. (2018c). *Promoting fruit and vegetable consumption around the world*. Retrieved from <http://www.who.int/dietphysicalactivity/fruit/en/index2.html>

- Wu, H. C., Chen, H. M., & Shiau, C. Y. (2003). Free amino acids and peptides as related to antioxidant properties in protein hydrolysates of mackerel (*Scomber austriasicus*). *Food Research International*, *36*, 949-957. doi:10.1016/S0963-9969(03)00104-2
- Yates, T., Khunti, K., Wilmot, E. G., Brady, E., Webb, D., Srinivasan, B., . . . Davies, M. J. (2012). Self-reported sitting time and markers of inflammation, insulin resistance, and adiposity. *American Journal of Preventive Medicine*, *42*, 1-7. doi:10.1016/j.amepre.2011.09.022
- Zhu, W., Plowman, S. A., & Park, Y. (2010). A primer-test centered equating method for setting cut-off scores. *Research Quarterly for Exercise and Sport*, *81*, 400-409. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/21268463>

Appendix A
PACMAC Questionnaire



This questionnaire asks about the things that you eat and drink, how well you sleep, and how much and what kinds of physical activity you do.

This questionnaire must be filled out by the participant (child) and a parent/guardian - together as a team unless noted.

- Please answer the questions honestly and as accurately as you can.
- This is not a test - there are no right or wrong answers to the questions.
- We will not tell anyone your answers.
- The questions about your parent/guardian are for whom you live with or who takes care of you.

Demographic:

- **PARENTS** please answer all the questions as honestly and accurately as you can.
- Please do not leave any lines blank, so if you are a single parent put N/A in parent 2 questions.

1. Participant's (child) name: _____
2. Date of birth of child (dd/mm/yy): _____/_____/_____
3. Child sex:
 - Male
 - Female
4. Child's ethnicity:
 - New Zealand European
 - Māori
 - Samoan
 - Cook Island Māori
 - Tongan
 - Chinese
 - Indian
 - Other _____
5. Parent/Guardian marital status:
 - Single, not living with a partner
 - Single, living with a partner
 - Married
 - Divorced
 - Never Married
6. Parent/Guardian (parent 1), occupation/job: _____
7. Parent/ Guardian (parent 1), please tick all school/academic qualifications:
 - None
 - NCEA Level 1 /5th Form
 - NCEA Level 2 /6th Form
 - NCEA Level 3 /7th Form
 - Degree
 - Honours Degree
 - Post-Graduate Diploma
 - Master's Degree
 - Doctorate

8. Parent/Guardian (parent 2), occupation/job: _____

9. Parent/ Guardian (parent 2), please tick all academic qualifications:

- None
- NCEA Level 1 /5th Form
- NCEA Level 2 /6th Form
- NCEA Level 3 /7th Form
- Degree
- Honours Degree
- Post-Graduate Diploma
- Master's Degree
- Doctorate

10. Household income:

- \$0
- \$1 - \$5,000
- \$5,001 - \$10,000
- \$10,001 - \$15,000
- \$15,001 - \$20,000
- \$20,001 - \$25,000
- \$25,001 - \$30,000
- \$30,001 - \$35,000
- \$35,001 - \$40,000
- \$40,001 - \$50,000
- \$50,001 - \$60,000
- \$60,001 - \$70,000
- \$70,001 - \$100,000
- \$100,001 - \$150,000
- \$150,001 - Above

11. Child home street address: _____

12. Child home flat/house number: _____

13. Child home postal code: _____

14. School name: _____

Physical Activity - Part 1

- The following questions are about the activities the participant (child) usually does.
- Parent AND Participant please fill this section out together.
- Please answer all questions as honestly and accurately as you can.
- Please tick a box on every line in the questionnaire.

AVAILABILITY

1. Do you consider your school walking distance from your home?

Yes?

No?

2. Are there playgrounds or parks within walking distance from your home where you can play?

Yes?

No?

How many times did you do visit playgrounds or parks close to your home in the PAST 7 DAYS?

	Each day that you did this, how long did you normally do it for?	How many days did you do this activity?			
		Never	Once	2 to 3 times	4 or more times
<input type="checkbox"/> How often do you visit playgrounds or parks close to your home	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

How many times did you do the following PHYSICAL activities
in the PAST 7 DAYS?

3. ACTIVITIES AT SCHOOL IN THE PAST 7 DAYS

☐	Each day that you did this, how long did you normally do it for?	How many days did you do this activity?			
		Never	Once	2 to 3 times	4 or more times
PE class	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Walk to and from school	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Cycle to and from school	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Travel to and from school by car / bus	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Travel to and from school by skateboard/scooter/ bike		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

4. ACTIVITIES OUTSIDE SCHOOL IN THE PAST 7 DAYS

☐	Each day that you did this, how long did you normally do it for?	How many days did you do this activity?			
		Never	Once	2 to 3 times	4 or more times
<i>SPORTS ACTIVITIES (NOT AT SCHOOL)</i>					
Aerobics	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Softball / Tee ball	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Basketball / Volleyball	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Cricket	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Dancing	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Football	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Gymnastics	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Hockey (field or ice)	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Martial arts (e.g. karate or judo)	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Netball	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

☐	Each day that you did this, how long did you normally do it for?		How many days did you do this activity?			
			Never	Once	2 to 3 times	4 or more times
Rugby/League	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Running or jogging	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Swimming lessons	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Swimming for fun	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Tennis/badminton/squash/ other racquet sport	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
LEISURE TIME ACTIVITIES						
Bike riding (not to or from school)	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Trampolining	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Bowling	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Household or farm chores	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Playing on playground equipment	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Playing with pets or horse riding	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Rollerblading / roller-skating	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Playing on scooter	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Skateboarding (not to or from school)	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Skiing, snowboarding, or sledging	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Skipping	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Walking the dog	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Walking for exercise	hrs	mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

☐	Each day that you did this, how long did you normally do it for?	How many days did you do this activity?			
		Never	Once	2 to 3 times	4 or more times
Art & craft (e.g. pottery, sewing, drawing, painting)	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Doing homework	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Listening to music	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Playing indoors with toys	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Playing board games / cards	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Playing musical instrument	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Reading	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Sitting talking	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Talking on the phone	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Any other activities you do (Please write it in here)	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

5. ACTIVITIES OUTSIDE SCHOOL ON A SCHOOL DAY IN THE PAST 7 DAYS

☐	On the SCHOOL DAYS that you did this, how long did you normally do it for?	On how many SCHOOL DAYS did you do this activity?			
		Never	One day	2 to 3 days	4 or more times
Playing computer games (like Xbox, Play station / game boy/ DS/ PSP)	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Using computer / internet	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Watching TV / DVDs	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

6. ACTIVITIES OUTSIDE SCHOOL ON A WEEKEND DAY IN THE PAST 7 DAYS

☐	On the WEEKEND DAYS that you did this, how long did you normally do it for?	On how many WEEKEND DAYS did you do this activity?		
		Never	One day	Two days
Playing computer games (like Xbox, play station / game boy/ DS/ PSP)	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Using computer / internet	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Watching TV / DVDs	hrs mins	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Physical Activity - Part 2

- The following questions are about the activities the participant (child) usually does.
- Parent AND Participant please fill this section out together.
- Put a check by the number that best describes your response to the question.
- Answers are on a point system from 1-4.
- 1 being the lowest given score.
- 4 being the highest given score.

E.g. I like to eat ice cream more than anything else.

1	2	3	✓ 4
Always NO	Sometimes NO	Sometimes YES	Always YES

1. I like playing outdoor games and sports.

1	2	3	4
----------	----------	----------	----------

2. I like getting sweaty when I exercise or play hard.

1	2	3	4
----------	----------	----------	----------

3. I have more fun playing games and sports than anything else.

1	2	3	4
----------	----------	----------	----------

4. I like to exercise lots.

1	2	3	4
----------	----------	----------	----------

5. I am told that I am good at games and sports.

1	2	3	4
----------	----------	----------	----------

6. I feel really tired after I play games and sports.

1	2	3	4
----------	----------	----------	----------

7. I get nervous and worried about playing games and sports.

1	2	3	4
----------	----------	----------	----------

8. I get teased by other kids when I play games and sports.

1	2	3	4
----------	----------	----------	----------

9. I think that the more exercise you get the better.

1	2	3	4
----------	----------	----------	----------

10. I make a lot of friends when I play games and sports.

1	2	3	4
----------	----------	----------	----------

11. I enjoy exercise a lot.

1	2	3	4
---	---	---	---

12. I try to stay in good **shape** (explained as having a good looking body).

1	2	3	4
---	---	---	---

13. I wish I could play more games and sports.

1	2	3	4
---	---	---	---

14. I think that I will feel really good after I play hard.

1	2	3	4
---	---	---	---

15. I do not mind getting out of breath after I play hard.

1	2	3	4
---	---	---	---

16. I think it is very important to always be in good shape.

1	2	3	4
---	---	---	---

17. Playing games and sports is my favourite thing.

1	2	3	4
---	---	---	---

18. I really like to run a lot.

1	2	3	4
---	---	---	---

19. I think exercise is very important for my **health** (Explain as well being & strong).

1	2	3	4
---	---	---	---

20. I look forward to playing sports and games.

1	2	3	4
---	---	---	---

21. I like to burn lots of energy by playing hard.

1	2	3	4
---	---	---	---

22. I think that exercise is the most important thing for good health.

1	2	3	4
---	---	---	---

23. I really like to exercise.

1	2	3	4
---	---	---	---

24. I feel good when I run hard.

1	2	3	4
---	---	---	---

25. I am popular when I play games and sports.

1	2	3	4
---	---	---	---

Nutrition:

- We would like to know about your general eating habits.
- Please answer all questions as honestly and accurately as you can.
- Please tick a box for every question on the questionnaire.
- Questions 1-11 are for the CHILDREN to answer with parent's assistance if needed.
- Questions 12-37 are for the PARENT/GUARDIAN to answer.

1. Do you like the taste of onions?

Yes?

No?

2. Do you like the taste of broccoli?

Yes

No

3. How often do you usually have these meals (more than a glass of milk or fruit juice) during the WEEK?
Please choose only one of the following per meal:

	Breakfast	Lunch	Dinner
Never	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
One day	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Two days	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Three days	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Four days	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Five days	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

4. How often do you usually have these meals (more than a glass of milk or fruit juice) during the WEEKEND?
Please choose only one of the following per meal:

	Breakfast	Lunch	Dinner
Never	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
One day	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Both days	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

5. On school days during lunch break do you generally...

Please tick one from the following:

Eat food bought at school or ordered through the school
(E.g. Subway, Pita Pit, Sushi etc.)

Eat a packed lunch brought from home

Go home for lunch

Eat food bought on the way to school

Don't eat lunch

6. For each of these questions, please tick which answer you think is right.

	None	Hardly any	Some	Quite a lot	Lots
Do you think that you eat a lot of fruit?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Do you think that you eat a lot of vegetables?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

7. For each of these questions, please tick which answer you think is right

	Never	Not often	Sometimes	Quite often	Always
If you ask for fruit that you like will your parents buy it for you?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
If you ask for vegetables that you like will your parents buy it for you?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Are there usually different kinds of fruit available at home?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Are there usually different kinds of vegetables available at home?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Is there usually fruit at home that you like?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Are there usually vegetables at home that you like?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

8. In the past week, at home.....Tick one box per line

	Yes	No
a. were there fruit or vegetables on the kitchen counter or somewhere in the open	<input type="checkbox"/>	<input type="checkbox"/>
b. was there fruit juice, fruit or cut up vegetables in the fridge as a snack?	<input type="checkbox"/>	<input type="checkbox"/>

9. How much do you like each of these foods? Tick one box per question.

	Like	They're ok	Dislike		Like	They're ok	Dislike
a. Apples	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	m. Butter	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
b. Oranges	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	n. Pasta	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
c. Bananas	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	o. Rice	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
d. Strawberries	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	p. Cake	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
e. Grapes	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	q. Pizza	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
f. Pears	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	r. Sausages	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
g. Peas	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	s. Chicken	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
h. Carrots	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	t. Potatoes*	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
i. Broccoli	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	u. Fish	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
j. Salad	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	v. Milk	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
k. Tomatoes	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	w. Ice Cream	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
l. Sweetcorn	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	x. Chocolate	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

* Not hot chips/potato chips

10. How healthy do you think each of these foods are? Tick one box per question

	Healthy	They're ok	Not healthy		Healthy	They're ok	Not Healthy
a. Apples	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	m. Butter	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
b. Oranges	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	n. Pasta	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
c. Bananas	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	o. Rice	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
d. Strawberries	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	p. Cake	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
e. Grapes	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	q. Pizza	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
f. Pears	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	r. Sausages	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
g. Peas	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	s. Chicken	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
h. Carrots	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	t. Potatoes*	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
i. Broccoli	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	u. Fish	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
j. Salad	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	v. Milk	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
k. Tomatoes	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	w. Ice Cream	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
l. Sweetcorn	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	x. Chocolate	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

* Not hot chips/potato chips

11. How many times a week do you usually eat or drink...? (Please tick ONE box for each item)

		Never	Less than once a week	Once a week	2 - 4 days a week	5 - 6 days a week	Every day, once a day	Every day, more than once
1	Fruits							
2	Vegetables (excluding potato)							
3	Trim milk (green and yellow cap) [including on cereals, milo, hot chocolate]							
4	Milk (blue) [including on cereals, milo, hot chocolate]							
5	Cheese							
6	Yoghurt							
7	Ice-cream							
8	Processed meat (such as meat pies, sausage, sausage roll, salami, luncheon, bacon, ham)							
9	Other meats (such as mince, beef, chicken)							
10	Fish (including canned tuna or salmon, fish cakes, fish fingers, fish pie, battered fish)							
11	Fruit juice (such as Orange juice, Apple juice, Raro, Refresh, Keri, Twist, Ribena)							
12	Diet fizzy drinks (such as Diet Coke, Pepsi Max, Sprite Zero and any other "light" or "sugar free" varieties.							
13	Fizzy drinks (such as Coke, Pepsi, Sprite, L&P, Fanta, Ginger Beer)							
14	Breakfast cereals							
15	White bread							
16	Brown /Wholemeal bread							
17	Rice, rice based dishes							
18	Pasta (such as spaghetti, macaroni), noodles							
19	Potato (such as mashed, boiled)							
20	Potato chips, potato snacks, corn chips							
21	Hot chips, wedges, French fries							
22	Biscuits, cakes, muffins, doughnuts, fruit pies							
23	Snack bars (such as muesli bar, fruit bar, rice bubble bar)							
24	Lollies							
25	Chocolate, Chocolate bars							
26	Tomato sauce, Ketchup							
27	Peanut butter, Nutella							
28	Jam, Honey							

Questions 12-37 are for the PARENT/GUARDIAN to answer.

12. How often do you usually have these meals (more than a drink) DURING THE WEEK?

Please choose only one of the following:

	Breakfast	Lunch	Dinner
Never	<input type="text"/>	<input type="text"/>	<input type="text"/>
One day	<input type="text"/>	<input type="text"/>	<input type="text"/>
Two days	<input type="text"/>	<input type="text"/>	<input type="text"/>
Three days	<input type="text"/>	<input type="text"/>	<input type="text"/>
Four days	<input type="text"/>	<input type="text"/>	<input type="text"/>
Five days	<input type="text"/>	<input type="text"/>	<input type="text"/>

13. How often do you usually have these meals (more than a drink) DURING THE WEEKEND?

Please choose only one of the following:

	Breakfast	Lunch	Dinner
Never	<input type="text"/>	<input type="text"/>	<input type="text"/>
One day	<input type="text"/>	<input type="text"/>	<input type="text"/>
Both days	<input type="text"/>	<input type="text"/>	<input type="text"/>

14. On work days during lunch break do you generally:

Please choose only one of the following:

- Eat food bought at work
- Eat a lunch brought from home
- Go home for lunch
- Eat food bought on the way to work
- Don't eat lunch

15. When you eat takeaways, do you usually pick healthier options if these are available?

Please choose only one of the following:

- Yes
- No
- I don't eat takeaways

16. How often do you eat takeaways (such as McDonalds, KFC, Fish 'n' chips, Domino's Pizza, Hell Pizza, Pizza Hut, Country Fried Chicken, and Asian Takeaways)? Please choose only one of the following:

	Alone or with friends Please choose one	With family Please choose one
Never	<input type="checkbox"/>	<input type="checkbox"/>
Less than once a week	<input type="checkbox"/>	<input type="checkbox"/>
Once a week	<input type="checkbox"/>	<input type="checkbox"/>
2-4 days a week	<input type="checkbox"/>	<input type="checkbox"/>
5-6 days a week	<input type="checkbox"/>	<input type="checkbox"/>
Once a day	<input type="checkbox"/>	<input type="checkbox"/>
More than once a day	<input type="checkbox"/>	<input type="checkbox"/>

Section 9: Dietary Habits

This section is about your usual eating patterns. When answering these questions please think back over the past 4 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.

17. On average, how many slices of bread/toast OR bread rolls do you eat per day?

- None, I don't eat bread or toast
- Less than one per day
- 1-2 per day
- 3-4 per day
- 5-6 per day
- 7 or more per day
- Don't know

18. What type of bread, rolls or toast do you eat most of?

- White
- High fibre white
- Light grain bread (e.g. Molenburg, Freya's, Ploughmans, And MacKenzie High Country)
- Heavy grain bread (e.g. Vogels and Burgen)
- Other
- Don't know

19. In the past four weeks, which of the following have you eaten at all? (choose at that apply)

- Red meat- such as beef, pork, mutton, lamb and goat
- Chicken- such as chicken breast, drumsticks, or whole chickens, but not chicken nuggets or chicken roll
- Processed meats- such as ham, bacon, sausages, chicken roll, luncheon, canned corned beef, pastrami, and salami
- Seafood- such as fish or shellfish
- None
- Don't know

20. How often do you EAT:

	Red meat	Chicken
Never		
Less than once per week		
1-2 times per week		
3-4 times per week		
5-6 times per week		
7 or more times per week		
Don't know		

21. How often do you REMOVE:

	Excess fat from meat	Skin from chicken
Never		
Rarely		
Sometimes		
Regularly		
Always		
Don't know		

22. How often do you eat processed meat products? Processed meat includes ham, bacon, sausages, luncheon, canned corned beef, pastrami, and salami.

- Never
- Less than once per week
- 1-2 times per week
- 3-4 times per week
- 5-6 times per week
- 7 or more times per week
- Don't know

23. How often do you eat fresh or frozen fish or shellfish? Do not include battered/fried or canned fish or shellfish.

- Never
- Less than once per week
- 1-2 times per week
- 3-4 times per week
- 5-6 times per week
- 7 or more times per week
- Don't know

24. How often do you eat battered or fried fish or shellfish? This may include battered or deep fried fish bought from the 'Fish and Chip' shop.

- Never
- Less than once per week
- 1-2 times per week
- 3-4 times per week
- 5-6 times per week
- 7 or more times per week
- Don't know

25. How often do you eat canned fish or shellfish? Canned fish includes products such as tuna, salmon, and sardines.

- Never
- Less than once per week
- 1-2 times per week
- 3-4 times per week
- 5-6 times per week
- 7 or more times per week
- Don't know

26. On average, how many servings of fruit- fresh, frozen, canned or stewed- do you eat per day?
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. Do not include fruit juice or dried fruit.

- Never, I don't eat fruit
- Less than one serving per day
- 1 serving

-
- 2 servings
 - 3 servings
 - 4 or more servings
 - Don't know

27. On average, how many servings of vegetables- fresh, frozen or canned- do you eat per day?

A serving is the same as one potato/kumara, half a cup of peas, or a cup of salad. For example, 2 potatoes + $\frac{1}{2}$ cup of peas = 3 servings. Do not include vegetable juices.

- Never, I don't eat vegetables
- Less than one serving per day
- 1 serving
- 2 servings
- 3 servings
- 4 or more servings
- Don't know

28. What type of milk do you use the most of?

- None, I don't use milk
- Whole or standard milk (dark blue or silver)
- Reduced fat (light blue)
- Skim or trim (green or yellow)
- Soy milk
- Other (such as rice, goats milk)
- Don't know

29. What type of butter or margarine spread do you use the most of?

- Never, I don't use butter or margarine as spread
- Butter (including semi-soft)
- Butter and margarine blend
- Margarine- Full fat (e.g. Canola, Sunflower, and Olive oil based)
- Light or reduced fat margarine (e.g. Canola, Sunflower, and Olive oil based)
- Plant sterol margarine- full and low fat varieties (e.g. Proactive or Logicol)
- Don't know

30. What type of fat or oil do you use most often when cooking?

- None, I don't use fat or oil
- Butter
- Margarine
- Butter blend
- Oil
- Dripping or Lard
- Other
- Don't know

31. How often do you add salt to your food after it has been cooked or prepared?

- Never
- Rarely
- Sometimes
- Regularly
- Always
- Don't know

32. How often do you choose low or reduced fat varieties of foods instead of the standard variety?

- Never
- Rarely
- Sometimes
- Regularly
- Always
- Don't know

33. How often do you choose low or reduced salt varieties of foods instead of the standard variety?

- Never
- Rarely
- Sometimes
- Regularly
- Always
- Don't know

34. How often do you eat hot chips, French fries, wedges, or kumara chips? Think about lunch, dinner, and snacks.

- Never
- Less than once per week
- 1-2 times per week
- 3-4 times per week
- 5-6 times per week
- 7 or more times per week
- Don't know

35. How often do you drink fruit juices and drinks? *Do not include diet or diabetic varieties.* 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. **Excludes-** 'diet varieties', soft drinks and energy drinks, flavoured waters (e.g. H2Go), and sports waters (e.g. Charlies Sports Water, Mizone, and Aqua-shot).

- Never
- Less than once per week
- 1-2 times per week
- 3-4 times per week
- 5-6 times per week
- 7 or more times per week
- Don't know

36. How often do you drink soft drinks or energy drinks? *Do not include diet varieties.* Soft drinks are often carbonated or 'fizzy' and include Coca-Cola, Pepsi, Lemonade, Ginger Beer, Energy drinks (e.g. 'V', Red Bull, and Lift Plus), PowerAde, E2, and G-force.

- Never
- Less than once per week
- 1-2 times per week
- 3-4 times per week
- 5-6 times per week
- 7 or more times per week
- Don't know

37. How often do you eat lollies, sweets, chocolate, and confectionary?

- Never
- Less than once per week
- 1-2 times per week
- 3-4 times per week
- 5-6 times per week
- 7 or more times per week
- Don't know

Sleep:

- The following questions are to be answered by the PARENT about their child's sleep habits in the past week. If last week was unusual for a specific reason, choose the most recent typical week.
- Please **tick a box for every question** on the questionnaire.
- Always if something occurs every night.
- Usually if it occurs 5 to 6 times a week.
- Sometimes if it occurs 2 to 4 times a week.
- Rarely if it occurs once a week.
- Never if it occurs less than once a week.

BEDTIME:

Write in your child's usual bedtime:

School night _____ - _____ pm

*School night = has school the next day

*Non-school night = has no school the next day

Non-school night _____ - _____ pm

	7 Always	5-6 Usually	2-4 Sometimes	1 Rarely	0 Never
1. Child goes to bed at the same time at night	()	()	()	()	()
2. Child falls asleep within 20 minutes after going to bed	()	()	()	()	()
3. Child falls asleep alone in own bed	()	()	()	()	()
4. Child falls asleep in parent's or sibling's bed	()	()	()	()	()
5. Child needs parent in the room to fall asleep	()	()	()	()	()
6. Child struggles at bedtime (cries, refuses to stay in bed, etc.)	()	()	()	()	()
7. Child is afraid of sleeping in the dark	()	()	()	()	()
8. Child is afraid to sleep alone	()	()	()	()	()

SLEEP BEHAVIOR:

Write in your child's usual amount of sleep each day. (Combining night time sleep and naps)

*School day = has school the next day _____ hours and _____ minutes

*Non-school day = has no school the next day _____ hours and _____ minutes

	7 Always	5-6 Usually	2-4 Sometimes	1 Rarely	0 Never
9. Child sleeps too little	()	()	()	()	()
10. Child sleeps the right amount	()	()	()	()	()
11. Child sleeps about the same amount each day	()	()	()	()	()
12. Child wets the bed at night	()	()	()	()	()
13. Child talks during sleep	()	()	()	()	()
14. Child is restless and moves a lot during sleep	()	()	()	()	()
15. Child sleepwalks during the night	()	()	()	()	()
16. Child moves to someone else's bed during the night (parent, brother, sister, etc.)	()	()	()	()	()
17. Child grinds teeth during sleep (your dentist may have told you this)	()	()	()	()	()
18. Child snores loudly	()	()	()	()	()
19. Child seems to stop breathing during sleep	()	()	()	()	()
20. Child snorts and/or gasps during sleep	()	()	()	()	()
21. Child has trouble sleeping away from home (visiting relatives, and holidays)	()	()	()	()	()
22. Child awakens during the night screaming, sweating, and inconsolable	()	()	()	()	()
23. Child awakens alarmed by a frightening dream	()	()	()	()	()

WAKING DURING THE NIGHT:

	7 Always	5-6 Usually	2-4 Sometimes	1 Rarely	0 Never
24. Child awakes once during the night	()	()	()	()	()
25. Child awakes more than once during the night	()	()	()	()	()

If you child wakes at night write the number of minutes a night waking usually lasts:

_____ hours and _____ minutes

MORNING WAKE UP

Write in the time your child usually wakes up in the morning:

*School day = has school that day

School day _____ - _____ am

*Non-school day = has no school that day

Non-school day _____ - _____ am

	7 Always	5-6 Usually	2-4 Sometimes	1 Rarely	0 Never
26. Child wakes up by him/herself	()	()	()	()	()
27. Child wakes up in a negative mood	()	()	()	()	()
28. Adults or siblings wake up child	()	()	()	()	()
29. Child has difficulty getting out of bed in the morning	()	()	()	()	()
30. Child takes a long time to become alert in the morning	()	()	()	()	()

Daytime Sleepiness:

	7 Always	5-6 Usually	2-4 Sometimes	1 Rarely	0 Never
31. Child seems tired	()	()	()	()	()

**During the past week, has your child has appeared very sleepy or fallen asleep during the following?
(Tick all that apply):**

	1 Not Sleepy	2 Very Sleepy	3 Falls Asleep
32. Watching TV	()	()	()
33. Riding in car	()	()	()

Birth History (Parents/Guardians)

- PARENTS, please answer these questions as best as you can.
- The following questions are in regards to the birth of your child.

1. Was your son/daughter a twin?

Yes?

No?

2. Do you know the exact weight of your son/ daughter at birth?

Yes?

No?

3. If yes to Q2, please state your child's weight: _____Kg

4. If no to Q2 was your child:

Less than 2.5 kg

More than 4 kg

5. Do you know the exact gestational week (e.g., 37 weeks is normal) that you delivered your son/ daughter?

Yes?

No?

6. If yes to Q5 please state the gestational age: _____weeks

7. If no to Q5, did you deliver you son/ daughter:

In less than 37 weeks

More than 37 weeks

Appendix B

Standard Operating Protocols

STANDARD OPERATING PROTOCOL: CARDIOMETABOLIC

Introduction

- When the heart contracts it generates a pulse wave that travels through circulation. The speed the pulse wave travels (pulse wave velocity, or PWV) is related to the stiffness of the arteries. It is easily measured in a noninvasive manner and is highly reproducible and reliable.
- Pulse Wave Analysis will be done using a PulseCor machine and will estimate central systolic blood pressure (cSBP) and central arterial wave reflection (augmentation index, AIx%).
- Biochemical markers give a good indication of overall cardio-metabolic health and risk.
- The markers measured:
 - Lipoproteins – measure good cholesterol (HDL) and bad cholesterol (LDL).
 - Triglycerides – fat that is used to store excess energy from diet. High levels of triglycerides in the blood are associated with atherosclerosis.
 - Serum glucose – measurement of glucose sugar. Elevated glucose levels can indicate diabetes or insulin inhibition.
 - HbA1c/glycated haemoglobin – measure blood glucose control over 6 weeks.
- In this study, biochemical markers will be collected using a well-tolerated finger pricks procedure and analysed with portable lipid/glucose (CardioChek, SensoCard) and HbA1c (Afinion HbA1c) analysers.

Responsibility

- It is the responsibility of the researchers to follow the standard operating procedures as outlined in this document. It is also expected that they review the document regularly to ensure they are up to date with current practices.

Pulse Wave Analysis

Equipment: PulseCor machine, power cord, child-sized cuff, and massage table.

Procedure:

1. Explain the procedure to the child and ask them to lie on the massage table for 20 minutes.
2. Ask the child to remove their jacket or jumper if it's covering the upper arm.
3. Wrap the cuff firmly around the upper arm at heart level.

4. Run the machine. Estimate central systolic blood pressure (cSBP) and central arterial wave reflection (augmentation index AIx %).
5. Each variable will be standardised as follows: standardised value = (value – mean)/SD. The z scores of the individual risk factors will be summed to create a cardio-metabolic risk factor score.
6. Record the results on the participant data sheet.

Biochemical Blood Markers

Equipment: Gloves, Swabs, alcohol wipes, sterol spray, rubbish bag, plasters, cholesterol and glucose readers, and cholesterol and glucose strips.

Procedure:

1. Wash hands thoroughly.
2. Explain the procedure to the child and ask them to choose a finger to be pricked. Get them to warm the finger by gently pulsing the end of it.
3. Insert a test strip into glucose metre.
4. Swab the child's finger with an alcohol wipe. Wait a few seconds for it to dry and then prick the child's finger using the smallest setting (twist to change) on the lancet.
5. Wipe the first drop of blood in case there's residual alcohol left.
6. Gently squeeze the finger until a drop of blood forms. Hold the edge of the test strip against the drop of blood and completely over the end. The machine will make a beeping noise when the test is done.
7. Fill the second strip (for cholesterol) with another drop of blood and insert this into the cholesterol tester. Wait for the machine to analyse.
8. Wipe the child's finger with a tissue and put a plaster on the prick site.

STANDARD OPERATING PROTOCOL: BODY FATNESS

Introduction

- An individual's body composition refers to their fat, water, bone and muscle proportions, influencing health and disease risk. There are multiple methods available, but the one chosen depends on resources available (e.g. cost, equipment, labour) and context (e.g. patient consult vs research study).
- This study is using the following procedures for measuring fatness: waist-to-hip ratio, waist-to-height ratio, body mass index (BMI) and bioelectrical impedance analysis (BIA).
- BMI, waist-to-hip ratios and waist-to-height ratios are indirect measures of body composition with minimal researcher and participant burden. These can be used to estimate fat and fat-free mass by studying body fat patterning, considering fat distribution (as central adiposity is associated with a greater risk of chronic disease).
- BIA is an effective and scientifically validated method for noninvasive measures of fat mass and fat-free mass, using the resistance of electrical flow through the body to estimate body fat. The BIA machine used in this study is BodyStat 1500MDD; Dual Frequency Body Composition and Wellness Monitoring Unit.

Responsibility

- It is the responsibility of the researchers to follow the standard operating procedures as outlined in this document. It is also expected that they review the document regularly to ensure they are up to date with current practices.

Waist-to-Hip Ratio

Equipment: Tape measure, pen, paper, and calculator.

Procedure:

1. Explain the procedure to the child before taking measurements.
2. Ask the child to remove any extra layers (i.e., jumpers or jackets). Tell them to stand straight with their shoulders and stomachs relaxed.
3. The researcher should stand on the right of the child. The measurement should be taken on bare skin (ask the child to hold their shirt up) and at the end of normal exhalation. The measuring tape should be parallel to the floor, not twisted, and pulled to lay flat on the skin without compressing it (not too tight). The child should stand with their feet together and look straight ahead.
4. Measure **waist circumference** (to the nearest 0.1 cm) at the midpoint between the lowest rib and iliac crest (top of hip bone).
5. The mean of two measurements taken at this location should be used. If the measurements differ by more than 0.5(1/2) cm a third should be taken.
6. Measure **hip circumference** (to the nearest 0.1 cm) at the widest part of the buttocks using the same standardised measures as for waist.

7. The mean of two measurements taken at this location should be used. If the measurements differ by more than 0.5cm a third should be taken.
8. Divide waist/hip circumference to give the waist-to-hip ratio.

Body Mass Index (BMI)

Equipment: Stadiometer, pen, paper, calculator, and electric scales.

Procedure:

1. Explain the procedure to the child before taking measurements.
2. Take the height measurement follow steps 3-6 as for waist-to-height ratio.
3. To take weight, ask the child to remove excess clothing, shoes and empty all pockets. Zero the scales then ask the child to step on. Record the measurement to the nearest 0.1kg.
4. The mean of two measurements taken should be used. If the measurements differ by more than 0.1kg a third should be taken.
5. Convert the height measure to m (divide by 100) and square (m x m). Calculate BMI by dividing weight (kg) by height (m²).

Bioelectrical Impedance Analysis (BIA)

Equipment: BodyStat package (BIA dual frequency measuring unit, 2x red and black cable leads, disposable electrodes, BodyStat calibrator), pen, paper, and massage table.

Procedure:

1. Ensure this procedure is done after the other measurements have been taken.
2. Explain the procedure to the child (as a pulse wave that will flow through them and take a measurement, but it won't hurt) and prepare them for testing. The child should have no shoes and socks on, no jewellery on wrists, ankles or neck, and be well hydrated.
3. Set up the machine and turn it on. Record the test number on the data sheet.
4. Add information into the machine that was taken prior to the test (using the arrow buttons to select choices, then the enter key once option is selected): sex, age, height, weight, waist and hip circumferences, and physical activity level. Use the separate chart and ask the child to rate their physical activity on the given scale.
5. First procedure is to have the participant take a big drink of water before lying down (which usually allows 7-10 minutes time from water consumption to running the analyses).
6. Ask the child to lie on the table, stomach facing upwards. Ask them to position themselves so their thighs aren't touching, and their arms aren't touching their sides.
7. Get the cords and plug them into the device (cords are interchangeable).
8. Place 2 electrodes on the **right** foot, and 2 on the **right** hand. The first electrode should be placed on the distal end of the foot, under the 4th and 5th toes under the knucklebones, with the flat side of the electrode facing inwards. Place the

second electrode on the proximal end of the foot, facing in line with the first electrode, and in line with the medial and lateral malleoli (large protruding ankle bones).

9. Repeat with the electrodes on the hand, placing the first electrode under the 4th and 5th fingers under the knucklebones, and the second on the wrist just below the head of the ulna bone, with the flat side of the electrode facing inwards.
10. Attach the leads to the electrodes. Place the red crocodile clip on the distal electrode and the black one on the proximal end (for both hand and foot).
11. Before you run the test ensure the participant is positioned correctly and in a relaxed position. Let 5 minutes pass before pressing the enter button to allow fluid levels in the body to stabilise.
12. Press enter, and the analysis will run. The machine will display the result, with the optimal range underneath. Can scroll through the results and download to computer via Bluetooth.
13. Throw out the electrodes once the analysis is done.

STANDARD OPERATING PROTOCOL: PHYSICAL FITNESS

Responsibility

- It is the responsibility of all staff to follow the standard operating procedures as outlined in this document.
- It is expected that all staff review the document at regular intervals to ensure they are up to date with current practices.

20 Meter Shuttle Run Test

Introduction

- The beep test is a multistage fitness test used to measure cardiovascular fitness and maximum oxygen uptake ($\dot{V}O_{2\max}$ max).
- The Standard test has 21 levels, and each level consists of a different number of shuttles.
- The test is performed by running between two markers placed 20 meters (65.6 feet) apart, at an increasing pace as indicated by the beeps.
- The test ends when you can no longer keep pace, or level 21 is completed.
- 1 BEEP IS THE SOUND OF A NEW SHUTTLE, 3 BEEPS IS THE SOUND BEGINNING A NEW HIGHER LEVEL

Equipment: 4 marking cones, measuring tape, beep test audio, speaker, and clipboard, and recording sheets.

Procedure:

1. This test involves continuous running between two lines 20 meters apart timed to recorded beeps.
2. The subjects stand behind one of the lines facing the second line and begin running when instructed by the recording. The speed at the start is quite slow.
3. The subject continues running between the two lines, turning when signalled by the recorded beeps.
4. After about one minute, a sound indicates an increase in speed, and the beeps will be closer together. This continues each minute (level).
5. If the line is reached before the beep sounds, the subject must wait until the beep sounds before continuing.
6. If the line is not reached before the beep sounds, the subject is given a warning and must continue to run and try to catch up with the pace.
7. The participant is done when they fail to reach the line (within 2 meters) for two consecutive beeps.
8. The participant's final score is the last level and shuttle they completed before missing a beep.

Tips

1. Find area where there is plenty of width, so participants can turn or pivot without running into each other.
2. Measure course accurately as possible because 1 meter off can make a 10-20% difference in scoring accuracy.
3. Create lanes with numbers if possible.
4. Write down names and lanes of each participant so scoring can be accurate and less confusing.
5. Partner up students to assist with keeping track of which level and shuttle their partner completes.

Scoring

1. The athlete's score is the level and number of shuttles reached (e.g., Level 1 shuttle 5) before they were unable to keep up with the recording.
2. Record the last level completed (not necessarily the level stopped at).

Handgrip Strength

Introduction

- Handgrip strength measures the pull force able to be produced using the forearm and hand muscles and is associated with height, weight, and sex in childhood. Both the dominant and nondominant hands may be tested to produce an average result.

Equipment

- Hand dynamometer, recording sheet, chair, pen, and clipboard.

PARTICIPANT DATA SHEET

Participant Name _____

DOB: _____ Age: _____ Sex: M/F

ANTHRO:

Weight (kg) 1: _____ Weight 2: _____

Height (cm) 1: _____ Height 2: _____

Hip (cm) 1: _____ Hip 2: _____ Hip 3: _____

Hip Mean: _____

Waist (cm) 1: _____ Waist 2: _____ Waist 3: _____

Waist Mean: _____

BodyStat# _____ Body Fat: _____ %

PULSE WAVE ANALYSIS:

Resting	Total time rested (e.g., 10 min.)	Fasted (Y/N)	Water (Y/N)	Exercise (Y/N)
Start time (e.g., 10 a.m.)				
DBP (mmHg)				
SBP				
CBP (mmHg)				
Aix (%)				
Pulse Rate (BPM)				

- If AIX differs by more than 4% take a 3rd recording
- If BP differs by more than 5 mmHg take 3rd recording

BLOOD MARKERS:

Glucose	HbA1c	CHOL	HDL	Triglycerides	LDL	TC/HDL

PHYSICAL FITNESS:

Shuttle Run: Level: _____ Shuttle # _____

Handgrip strength test: Dominant Hand: Right: _____ Left: _____

Right 1: _____ Right 2: _____ Right 3: _____

Left 1: _____ Left 2: _____ Left 3: _____

Appendix C

Study Invitation Letter

Massey University
Wellington, NZ
College of Health
School of Sport and Exercise

August 2, 2015

Dear Principal,

My name is Nick Castro, and I am a PhD student at Massey University. My study consists of looking at the relationships between lifestyle behaviours (physical activity, physical fitness, nutrition, sleep), and cardiovascular health in children aged 8 to 10 years. This is a week-long study that puts an emphasis on the overall health of children by implementing a health and wellness week.

This health and wellness week will promote ways for children to live a long and healthy life by encouraging and educating children on the importance of eating healthy, exercising daily, staying fit, and getting enough sleep.

Can we set up a time, so I can present my study and answer any questions you may have?

Thank you for your time and have a great day!

Best Regards,



Nick Castro

Nick Castro, M.A.Ed. | PhD Student
Massey University | College of Health
Private Bag 756 | Wellington 6140 | New Zealand
T: 04.801.5799 ext. 63905 | **E:** N.Castro@massey.ac.nz



Appendix D

Participant Paperwork

What is the relationship between excess bodyweight, and health in children aged 8 to 10 years?

We are inviting you and your child to take part in a study concentrating on the overall health of children. The study will promote ways for children to live a long and healthy life by being physically active, eating healthy, and getting the right amount of quality sleep.

In New Zealand, the number of overweight children is increasing. Research shows that lifestyle behaviours affect the overall health of children.

This study will look at the relationships between weight, lifestyle behaviours, and cardiovascular health in 400 children aged 8 to 10 years, from Wellington, Christchurch, and Dunedin.

If you wish for your child to take part in this study week, please carefully read through the enclosed forms. All the information you and your child provide is confidential.

Information Sheet – Parent/Guardian

What will I need to do if my child takes part?

You will be asked to fill out a consent form, a standardised health and physical activity readiness document for your child and a questionnaire at home with your child.

What will my child need to do if s/he takes part?

The study will take place at your child's school. All children will take part in two testing sessions. On one day during the week (45 mins) your child will take part in the following measurements:

- A finger prick to look at the levels of fats, and sugar in the blood
- Blood pressure measurement.
- Height, weight, hip, and waist circumference measurements
- Nutritional taste test
- A test that looks at how much fat and muscle in their body
- On the last day, participants will participate in a Health and Wellness workshop, fitness activity level measurement, and handgrip strength test
- Finally, at home you and your child will be asked to complete a questionnaire as a team. The questionnaire includes demographic, physical activity, nutritional, and sleeps behaviour questions.

CONSENT FORM – (PARENT / GUARDIAN)

I have read the Information Sheet concerning this Health and Wellness week and I understand what it is about. I have read the Information Sheet with or to my child..... (Insert Child's Name), and I am satisfied that they understand what they are required to do. I understand that I am free to request further information at any stage. I understand that it is my family's choice to participate, and my child or I can withdraw from participation at any time without giving any reason.

I have read the Information Sheet concerning this project and I understand what is required of me as a parent/guardian. I (Insert Your Name) am satisfied with what I am required to do. I understand that I am free to request further information at any stage. I understand that it is my choice for me and my child to participate in this study; I can withdraw from participation at any time without giving any reason.

If you have read and understood everything that we will be asked of you and your child, and you would like to take part in the study, please return the following:

- A signed Parent Consent Form
- An email address and or contact number
- The Health and Physical Activity Readiness document
- When completed a questionnaire

As a parent/ guardian I agree to participate in this study under the conditions set out in the Parent Information Sheet.

Please tick the box below if you agree to participate in this study under the conditions set out in the Parent Information Sheet:

I **AGREE** to my child participating in this study under the conditions set out.

I **DO NOT** agree to my child participating in this study under the conditions set out.

Parent/Guardian Full Name: _____

Parent/Guardian Signature: _____

Parent/Guardian Phone Number: _____

Parent/Guardian Email Address: _____

Participant's Rights

Your child is under no obligation to accept this invitation. If you decide to allow your child to participate, you and your child have the right to:

- Decline to answer any question
- Withdraw from the study at any time
- Ask any questions about the study at any time during participation
- Be given access to a summary of the project findings when it is concluded
- Request a summary of individual results

What will happen to this information?

All the information collected will be kept on a password locked computer. Your child's personal details and results will remain confidential and their names will not be disclosed on any documentation relating to this study. We may use the data that we collect in publications or during presentations, but no one will be able to tell which data are your child's.

**If the study identifies any abnormalities in a child the researchers will contact and consult with you to see whether it is appropriate to contact your general practitioner.

Please contact us at the School of Sport and Exercise if you have any questions about this study. Thank you for your time.

Best Regards,

Nick Castro
PhD Student
Massey University
School of Sport and Exercise
04.801.5799 Ext 63905
N.Castro@massey.ac.nz

**Health and Physical Activity Readiness – Questionnaire
Completed by a Parent/Guardian of Child**

NAME OF CHILD
CHILD DATE OF BIRTHCHILD’S AGE:

As your child is to be a participant in this study, would you please complete the following health & physical activity readiness questionnaire for your child?

Please Tick the Appropriate Box

Any information contained herein will be treated as confidential.

	YES	NO
Has the test procedure(s) that your child will participate in been fully explained to you?	<input type="checkbox"/>	<input type="checkbox"/>
1. Has your doctor ever said that your child has a heart condition and that your child should only do physical activity recommended by a doctor?	<input type="checkbox"/>	<input type="checkbox"/>
2. Does your child ever experience chest pain during physical activity?	<input type="checkbox"/>	<input type="checkbox"/>
3. Does your child ever lose balance because of dizziness or do they ever lose consciousness?	<input type="checkbox"/>	<input type="checkbox"/>
4. Does your child have a bone or joint problem that could be made worse by a change in their physical activity participation?	<input type="checkbox"/>	<input type="checkbox"/>
5. Does your child have uncontrolled asthma (i.e. asthma that is not easily controlled by an inhaler)?	<input type="checkbox"/>	<input type="checkbox"/>
6. Is your doctor currently prescribing any medication for your child’s blood pressure or a heart condition?	<input type="checkbox"/>	<input type="checkbox"/>
7. Do you know of any other reasons why your child should not undergo physical activity? This might include diabetes, a recent injury, or serious illness.	<input type="checkbox"/>	<input type="checkbox"/>

If you have answered **NO** to all questions, then you can be reasonably sure that your child can take part in the physical activity requirement of this project.

I..... declare that the above information is correct at the time of completing this questionnaire on date/...../.....
Please note: If your child's health changes so that you can answer YES to any of the above questions, notify the investigators and consult with your doctor regarding the level of physical activity that your child can participate in.

Signature of Parent/Guardian.....Date

The section below must be filled out ONLY if you answered YES to one or more questions on the front side:

Talk to your doctor in person discussing the questions you answered yes to regarding your child.

Ask your doctor if your child can participate in the physical activity requirements of the project.

Doctor's Name.....Date

Doctor's Signature

.....

Appendix E

Factor Loadings of Food Items Grouped into Three Identified Dietary Patterns in New Zealand Children

Food items/group	Processed Food	Fruit and Vegetables	Breakfast Food
Fruits	-0.02	0.41	-0.02
Vegetables (excluding potatoes)	-0.01	0.45	-0.02
Trim milk (green cap) [including on cereals, milo, hot chocolate]	0.01	0.22	-0.45
Milk (blue cap) [including on cereals, milo, hot chocolate]	0.11	0.07	0.54
Cheese	0.04	0.27	0.32
Yoghurt	0.19	0.27	-0.02
Ice cream	0.24	-0.07	-0.06
Processed meat (meat pies, sausage, sausage roll, salami, luncheon, bacon, ham)	0.29	0.13	-0.06
Other meats (mince, beef, chicken)	0.25	0.21	-0.06
Fish (including canned tuna or salmon, fish cakes, fish fingers, fish pie, battered fish)	0.27	0.04	-0.16
Nondairy drinks	0.31	-0.18	-0.05
Breakfast cereals	0.17	0.18	0.25
White bread	0.3	-0.19	0.03
Brown /Wholemeal bread	-0.12	0.37	0.09
Rice, rice-based dishes	0.25	0.02	-0.29
Pasta (such as spaghetti, macaroni), noodles	0.24	0.16	-0.31
Salty snacks	0.25	-0.13	0.03
Biscuits, cakes, muffins, doughnuts, fruit pies	0.12	0.15	0.25
Lollies	0.24	-0.19	0.16
Sweet snacks	0.33	-0.08	0.13
Spreads	0.28	0.11	0.08
Eigenvalue	3.5	2.4	1.49
Variance explained (%)	16.7	11.4	7.11

Note. Factors ≥ 0.20 are in boldface