The Combating Obesity in Māori and Pasifika Adolescent School-children Study: COMPASS Methodology and Study Protocol

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ABSTRACT

Background: Lifestyle modifications including, physical activity can reduce obesity-related morbidity and subsequent cardiovascular disease in youth. This study will investigate the efficacy of a culturally-sensitive, non-contact, boxing-orientated training program on obesity and related cardio-metabolic conditions in Māori and Pasifika adolescents. Details of the methodological aspects of recruitment, inclusion criteria, randomization, cultural sensitivity, intervention program, assessments, process evaluation, and statistical analyses are described.

Methods: This study will be a community based, New Zealand, randomized control trial (RCT). Male and female obese (body mass index >95th percentile) Māori and Pasifika adolescents aged 14-16 years will be recruited and the sample size will be confirmed through a feasibility study. Combating Obesity in Māori and Pasifika Adolescent School-children Study (COMPASS) is a 6-month, theory-based program, conducted 3-times/week in a culturally appropriate setting. Each session includes 40 min boxing-orientated training and 30 min resistance training. Assessments will be made at baseline, 3-months, 6-months, 12-months, and 24-months. Main outcomes include abdominal obesity, endothelial function, and insulin resistance. Other outcomes include arterial stiffness, lipid profile, inflammatory biomarkers, well-being, and aerobic fitness. Control measures include physical activity, sleep behavior, and dietary intake.

Results: As a protocol paper there are no specific results to present, our purpose is to share our RCT design with the scientific community.

Conclusions: COMPASS will be used to provide direction for exercise prescription policy in at-risk Māori and Pasifika adolescents.

Keywords: Cardiovascular, exercise, indigenous, lifestyle, metabolic syndrome, pediatrics

INTRODUCTION

Within New Zealand, much higher rates of obesity have been reported for Pasifika (22.3%) and Māori (11.8%)
children (2-14 years) than their counterparts of European ancestry (5.5%).[1] The increased prevalence places these cohorts at greater risk for obesity-related cardio-metabolic conditions, including dyslipidemia, hypertension, and type 2 diabetes mellitus[2] [Figure 1].[3] These conditions independently and additively increase cardiovascular disease (CVD) risk,[3-7] even in children and adolescents.[5,8-17] Indeed, many of the pro-inflammatory and pro-atherogenic disorders associated with vascular disease in adults have also been demonstrated in obese children.[5,8-17] For example, overweight children were found to have elevated levels of interleukin-6 (IL-6) and tumor necrosis factors-alpha (TNF-α) receptors, and plasma levels of C-reactive protein (CRP) that are approximately 3 times greater than normal-weight children.[5]

Although genetic factors might influence the susceptibility of individuals to weight gain,[18] there is a consensus that changes in lifestyle activities have driven the current obesity epidemic.[19] Dietary modification has been shown to be relatively ineffective in the long-term treatment of obesity in adults,[20,21] and it has been suggested that obesity prevention in childhood and adolescence should focus on physical activity rather than diet because of fears relating to eating disorders.[22] Dieting can also lead to loss of fat-free mass, which is of particular concern when considering an increase in muscle strength (and by proxy, muscle mass) decreases insulin resistance in children aged 10-15 years.[23] Conversely, exercise maximizes the proportion of weight lost from fat mass and minimizes the loss of fat free mass.[22] The World Health Organization (WHO), in recognition of declining physical activity levels and the subsequent rise in cardio-metabolic conditions, including obesity, published the Global Recommendations on Physical Activity and Health.[24] The WHO recommends that children aged 6-17 years participate in at least 60 min of moderate-to-vigorous physical activity every day, and to perform vigorous exercise (high intensity), muscle-strengthening, and bone strengthening exercise, on at least 3 days/week.[24] However, obese children and adolescents are disadvantaged by physical and cardiovascular constraints, simply due to the excess body weight and the effort involved in moving a large mass, thus, limiting the capacity of these individuals to comply with WHO recommendations.

A meta-analysis of exercise treatment programs in obese children and adolescents has shown that the most effective exercise paradigm for this population incorporates high repetition resistance training combined with low-intensity, long-duration aerobic exercise.[25] Resistance training has been shown to be well-tolerated by this population and results in positive changes to body composition,[26-28] as well as an improvement in elements of metabolic syndrome (i.e., levels of glycated hemoglobin,[29] insulin resistance,[28,29] low-density lipoprotein (LDL) cholesterol,[30] triglycerides,[30] total cholesterol,[30] intrahepatic lipid,[28] both systolic and diastolic blood pressure[29-31]) in obese adults. Low-intensity, long-duration aerobic exercise has been shown to have positive results on body weight and composition in obese children,[22] but ultimately is limited in capacity.
when compared to high-intensity exercise for decreasing obesity, cardio-metabolic conditions, and the progression of CVD. There remains a pressing need to develop and assess vigorous exercise programs, which are safe, tolerable, enjoyable, and sustainable for at-risk obese children and adolescents. A mixed-modality program that incorporates both resistance and high-intensity aerobic training is optimal for producing the greatest impact to the health of obese adolescents.

Exercise programs designed for children and adolescents from indigenous backgrounds, including Māori and Pasifika, should not only be physiologically appropriate, but also culturally sensitive. An argument can be made that sport can be used as a vehicle to experience, discover, and reconnect to indigenous cultural heritage. For example, Māori are attracted to sport not only because of their love of whakataetae (competition) and achievement (whakatutukitanga), but also, because it provides a forum to experience feelings of whanau (extended family). In particular, many Māori show a decided preference for sports, which involve a team environment (whanaungatanga/kotahitanga) and that include bodily contact. Boxing training is characterized by high-intensity, intermittent activities, and, in previously untrained individuals, has been shown to result in favorable changes in gait and balance, cardiovascular fitness, and lipid profiles. Besides health/fitness (hauora/oranga), boxing training can also lead to increased self-confidence (mana), self-discipline, character development, and comradery. A non-contact, boxing-orientated program presents an accessible, relatively inexpensive, fun and engaging option for promoting healthy lifestyles that aligns with tikanga (Māori culture).

Research objectives
This study will investigate the efficacy of a culturally-sensitive, non-contact, boxing-orientated training program Combating Obesity in Māori and Pasifika Adolescent School-children Study (COMPASS) on obesity and related cardio-metabolic conditions in Māori and Pasifika adolescents aged 14-16 years. Three null hypotheses will be tested: H1: There will be no relationship between COMPASS participation and endothelial function (CVD risk), H2: There will be no relationship between COMPASS participation and abdominal adiposity; H3: There will be no relationship between COMPASS participation and insulin resistance. An important objective is to also determine the strength of association between endothelial function, abdominal adiposity, and insulin resistance associated with the COMPASS intervention. Findings from COMPASS will provide direction for future exercise prescription policy in this at-risk cohort.

METHODS

Study design
The COMPASS intervention will be evaluated using a randomized control trial (RCT) study design [Figure 2]. A randomization envelope will be prepared by a member of the research team and an independent third party will allocate participants to the treatment or control (wait list) groups. Full assessments [Table 1] will be made on both groups at baseline, and at 3-months and 6-months. Follow-up assessments will be conducted on the intervention group at 12-months and 24-months. Analysis will be by intention to treat. Ethical approval will be obtained from the regional Health and Disabilities Ethics Committee.

![Figure 2: Design of the combating obesity in Māori and Pasifika Adolescent school-children study](image-url)
Study participants and recruitment

Male and female adolescents aged 14-16 years will be recruited. Participants will be categorized as obese (Body mass index [BMI] >95th percentile), based on the age- and gender-specific recommendations of the International Obesity Task Force. A self-reported Tanner scale will be used to determine pubertal status. Participants who have not reached Tanner IV staging will not be eligible. Participants receiving insulin or other pharmacological treatment for diabetes will be excluded, but will be eligible if diabetes is diet-controlled. Participants will also be excluded from the study, if they have had an orthopaedic injury or surgery that has prohibited full function within 4 weeks of commencing the intervention, or have been diagnosed with a neuromuscular disease or cardiovascular conditions that would exclude them from participating in high-intensity exercise. All female participants will be asked to submit a urine sample for a basic pregnancy test as pregnant participants are not eligible for participation.

Participants will be recruited from the greater Wellington area through press releases on local radio and in newspapers, by physician referral, publicly posted advertisements, and via direct contact with the Māori and Pasifika boxing community. Recruitment will continue over a period of several months, with rolling study commencement occurring at 1-month intervals.

Cultural sensitivity

A number of steps will be followed to ensure cultural sensitivity and long-term viability of COMPASS:

1. Māori academic consultants. To advise on cultural issues pertaining to the program and translate common boxing terminology to Te Reo (Māori) and Pasifika.

2. A steering committee. Comprised of Māori and Pasifika adolescents and parents who are currently involved with the boxing club or from the greater community.

3. Adherence to kawa and tikanga. Kawa are the protocols that apply in different situations. For boxing training (fight training) there are certain rules and conditions that Māori apply regarding expectations of behavior, outcomes, and standards, similar to martial arts training. Tikanga or customs apply in the treatment of individuals and groups and this would include awhi (inclusion), whakawhanaungatanga (relationship building), mana (self-esteem and empowerment), and noa (states of wellbeing). The merging of Māori culture and physical exercise serves to validate the interface between mātauranga Māori and science.

Adherence

To ensure adherence we will: (1) Have the participants sign an accountability contract, (2) ensure the program is fun and culturally sensitive, and (3) utilize social media group to aid social facilitation. The accountability contract will be independent of the ethics consent form. By signing this contract between the participant and program coordinator, the participant is agreeing to attend scheduled training sessions on time, or contact the program coordinator, if they will be late or absent. The participant also agrees to attend sessions in appropriate dress (i.e., gym attire) and maintain a positive attitude throughout the training sessions. The social media will be participant-led,
with minimal input from the research team except to vet the suitability of entries. Participants will be provided with pocket cameras and encouraged to share their personal journeys.

**Pilot studies**

Two pilot studies will be conducted prior to the primary intervention trial.

**Feasibility study**

A 3-month trial of the COMPASS intervention will be recruit 14 male/female adolescents who meet the study criteria outlined above. Assessments will be made at baseline, 1-month and 3-months. The outcomes will be: Abdominal obesity (ultrasound, central arterial stiffness (pulse wave analysis [PWA]), central blood pressures (PWA), carotid arterial stiffness (ultrasound), and insulin resistance (oral glucose tolerance test [OGTT]). Daily physical activity (accelerometry), nutrition (food frequency questionnaire) and sleep behavior and quality (accelerometry) will also be collected to ensure the COMPASS program does not interact with important lifestyle factors. A focus group will be held at the conclusion of the study, utilizing standardized semi-structured questions. This study will provide the data needed to adequately power the primary intervention trial, and to address unforeseen cultural issues.

**Brunel mood scale validation**

Mood will be assessed using the Brunel Mood Scale, which is a 24-item scale that assesses anger, confusion, depression, fatigue, tension, and vigor. The BRUMS was designed with the intention of producing a scale that was easy to complete. Studies support the validity of use with children in different languages including, Italian, Hungarian, Malaysian, and Australian. Consistent with the notion that the validation is an on-going process and that researchers should not assume that validity transfers from culture to another, we will investigate the factorial validity of BRUMS for use in New Zealand with the Māori and Pasifika population. Data will be gathered from a sample of 200 Pākehā (non-indigenous) and 30 Māori/Pasifika. BRUMS will be translated to Te Reo (Māori). A member of the research team is a Chartered Psychologist with the British Psychological Society.

**Treatment group**

COMPASS is a 6-month mixed-modality exercise training program for Māori and Pasifika adolescents. The participants will attend three 70 min training sessions per week, on non-consecutive days. Each session will consist of 40 min standard boxing training, consisting of skipping (3 min × 2 min), shadow boxing (3 min × 2 min), bag work (3 min × 2 min), and one-on-one focus-pad work (3 min × 2 min). The boxing training will be standardized in terms of content, and intensity will be gradually increased each month. Training intensity will be monitored using ratings of perceived exertion (RPE) and heart rate monitors. Session-RPE has been validated for use in range of intermittent sports, including combat sports. Then the participants will complete a 30 min progressive, closed kinetic, resistance training routine, consisting of 5 lower extremity, 5 upper extremity, and 3 abdominal exercises. Intensity will be increased non-linearly. One of the research team is a certified Athletic Trainer, and two members are accredited exercise physiologists with the British Association of Sport and Exercise Sciences. The training sessions will be held at Petone Sports and Boxing Club (Lower Hutt, Wellington, New Zealand). Compared to New Zealand as a whole, Lower Hutt has a higher proportion of Māori (14.7% vs. 17.1%, respectively) and Pasifika (6.9% vs. 10.5%, respectively). Training sessions will be coordinated by the head coach of Petone Sports and Boxing Club, one of the co-investigators (a Level 1 boxing coach), and a trained research assistant.

**Control (wait list) group**

The control group will continue at their typical levels of physical activity. The control group will have an opportunity to participate in COMPASS after the initial 6-month period and post-testing has been completed.

**Main outcome measures**

**Endothelial function**

The flow-mediated dilation (FMD) test is the standard tool used to assess endothelial function. Reduced FMD is an early marker of atherosclerosis and has been noted for its capacity to predict future CVD events. Furthermore, an impaired vascular response has been demonstrated in children as young as 7 years old with familial hypercholesterolemia. Physical activity has been shown to improve FMD in obese children with exercise-induced
improvement in FMD reversing after only 6 weeks of inactivity.\cite{64,65} FMD will be conducted using high-resolution B-mode ultrasound (t3200; Terason, Burlington, Massachusetts) equipped with a 15-4 MHz linear array transducer (15L4), by a highly trained technician who has developed FMD guidelines.\cite{67} In past research FMD has been expressed as percentile, and this is the basis of the sample size calculation. A change of 1% is a clinically relevant difference\cite{61} and past research has found a wide range for the standard deviation (SD) for FMD in similar populations, of between 0.8% and 2.5%.\cite{63,65} We have used the largest SD in the sample size calculation for the feasibility study. We will also express FMD as the absolute change in diameter and in analysis use the baseline diameter as a covariate.\cite{68}

**Abdominal obesity**

The classification of an individual as obese is usually determined by BMI, not by a direct measure of adiposity. Whilst BMI is a surrogate of total body fatness it does not account for the differential health risks of excess fat within the abdominal and gluteal regions and is less predictive of health risk than measures of abdominal fat.\cite{69} Visceral adiposity decreases the sensitivity of target tissues to insulin while subcutaneous abdominal fat has a more direct influence on insulin sensitivity.\cite{69} While total body fat is important, studies have shown that visceral adiposity poses a higher risk for developing obesity related disorders than overall adiposity,\cite{70-72} including in adolescents and children.\cite{73-76} Therefore, intra-abdominal and subcutaneous abdominal adipose tissue will be measured by a highly trained sonographist using a portable, high-resolution, B-mode ultrasound device (t3200; Terason, Burlington, MA) equipped with a 6-1 MHz curved array transducer (6C1). Intra-abdominal thickness will be defined as the distance between the anterior wall of the aorta and the posterior surface of the rectus abdominis muscle, measured 1-5 cm above the umbilicus at the xiphio-umbilical line. These measurements strongly agree with computed tomography (CT),\cite{77-79} and magnetic resonance imaging (MRI),\cite{80,81} based estimations and are reliable,\cite{79,82,83} with high interobserver (P = 0.97, 95% CI: 0.90-0.99) and intraobserver (Intraclass Correlation Coefficient: 0.97, 95% CI: 0.88-0.99) reliabilities.\cite{83} CT and MRI are reference methods, both MRI and CT are high-cost technologies, and CT requires radiation exposure. The clinically relevant difference in adiposity measured by ultrasound is unknown. The association between adiposity and FMD will be used to estimate the likely size of the clinically important difference for this variable.

**Insulin resistance**

There are many methods for estimating insulin sensitivity with reference methods including the hyperinsulinaemic euglycemic clamp\cite{85} or intravenous glucose tolerance test with minimal modelling.\cite{86} Both of these methods are invasive, time consuming and expensive. A variety of surrogate methods are based on either fasting state insulin and glucose concentrations, or insulin and glucose concentrations during an OGTT.\cite{87,89} The homeostasis model assessment (HOMA) is perhaps the most commonly used simple method, and correlates well with the euglycemic clamp in those without diabetes.\cite{80} The Matsuda index provides a more integrated measure combing both fasting and dynamic changes in glucose and insulin.\cite{87} We will use both of these measures in the pilot study. This will enable accurate sample size calculations for the main study, and also an assessment of tolerability and acceptability of the 5 point OGTT in this adolescent population. To enable 5 blood samples to be collected at 30 min intervals an indwelling intravenous cannula will be inserted. Fasting plasma glucose and insulin will be analysed using standard commercial assays, and HOMA will use a computer generated program to calculate insulin resistance based on insulin and glucose values. Insulin resistance has been related to FMD in children,\cite{66,90,91} adolescents\cite{90} and adults. For example, a strong, negative relationship between HOMA and FMD has been reported in adults with chronic kidney disease ($R^2 = 0.91$, $\beta = -0.24$, $P = 0.008$).\cite{92} The association between HOMA, Matsuda index and FMD will be used to estimate the likely size of the clinically important difference for this variable in obese adolescents.

**Other outcome measures**

**Well-being**

Available evidence indicate that pleasant emotions are associated with good physical and psychological health.\cite{96} The present study will use a measure of Total Mood Disturbance by subtracting the sum of unpleasant states (anger,
confusion, depression, fatigue, and tension) from vigor scores as assessed by the BRUMS.

Aerobic fitness
Cardiovascular health risk has been associated with low levels of cardiorespiratory fitness in youth. Therefore, participants will complete 2 phases of a maximal discontinuous cycle protocol. During the first phase, the initial workload will be set at 50 Watts and each stage will include an increased workload of 25 Watts. Stages will consist of 3.5 min of continuous cycling followed by 1.5 min of rest. During the cycling component, the participant will maintain a cadence of at least 60 rpm. When the cadence minimum is not maintained, the stage is considered incomplete and phase one is terminated. The participant will be given several minutes rest before a second attempt at the previous workload (Phase 2). If a participant completes this stage, then the workload is increased appropriately and protocol continued as described above. When the participant is unable to complete a stage (i.e., maintain a cadence of 60 rpm for 3.5 min), the testing session will be finished. A valid maximal test will have been achieved if at least 3 of the following criteria were met during the final 30 s of the last completed stage: Respiratory equivalent ratio less than 1.15, RPE less than 18, heart rate within 11 bpm of the age-predicted maximum (208 – [0.7 × age]), an increase in VO$_2$ that is less than 50% of what would be expected for the change in mechanical work.\[97\]

Central blood pressure and arterial stiffness
The FMD test can be used to evaluate the functional health of the vascular system, whereas indicators of arterial stiffness and central blood pressures are used to assess structural characteristics.\[98\] PWA is a simple,\[98\] non-invasive, valid,\[99-101\] and reliable\[102-104\] technique that has been widely used in epidemiological\[105\] and interventional studies\[106\] to investigate central blood pressures and arterial stiffness. The PulseCor R7 CardioScope (PulseCor, Auckland, New Zealand) measures brachial artery pressure waves using the oscillometric method from an upper arm cuff and incorporates a POEM2 module (Welch Allyn, Skaneateles Falls, New York). Central blood pressures, derived from a generalized transfer function, have been validated against invasive catheter measurements and exceed the requirements of the Association for the Advancement of Medical Instrumentation (sphygmomanometer committee 0) for measurement accuracy.\[107,108\] Using this device, Lydakis et al.\[109\] reported that obesity and adherence to the Mediterranean diet independently predicts arterial stiffness in 12-year-old children.

Lipid profile
A fasting blood sample will be collected at the time the cannula is inserted for analysis of lipid profile. Total cholesterol (TC), high-density lipoprotein (HDL) cholesterol, and triglyceride (TG) concentrations will be determined using commercial assays. Low-density lipoprotein (LDL) cholesterol will be calculated combing values from the above measurements of TC, HDL cholesterol, and TG, using Friedwald's formula.\[110\]

Inflammation
Pro-inflammatory cytokines (i.e., IL-1β, IL-6 and TNF-α) are secreted from adipose tissue macrophages and impair insulin signaling as well promote endothelial dysfunction. CRP and other markers of oxidative stress have been associated with increases in adiposity and atherosclerosis progression.\[111\] Contracting skeletal muscle releases myokines that lower blood pressure, enhance insulin sensitivity and lipid oxidation and protect against cardiovascular morbidity. These markers have been previously linked to published exercise interventions in obesity management.\[112-116\] Therefore, a clinical phlebotomist with experience in children and adolescents will collect serum from the arms of participants. Serum samples will be analysed, in duplicate, for TNF-α, IL-6, CRP, using high sensitivity enzyme linked immunosorbent assays (ELISA). Fasting plasma insulin and IL-1 will be analysed using standard ELISA kits. The adipokines (adiponectin, leptin, resistin, plasminogen activator-1) will be analysed using a human adipocyte multiplex kit.

Genomics
Genetic studies have revealed links between genetics and response to physical training\[117\] and obesity.\[118\] The COMPASS study provides an opportunity to add to this data in a Maori/Pasifika population. A whole blood sample will be collected by a clinical phlebotomist and genomic DNA isolated and stored as previously described\[119\] for future analysis with single nucleotide polymorphism/copy number variant (SNP/CNV)
chip and/or next generation sequencing technology. The study will focus on genetic analysis of genes that have been shown to be associated with response to physical training, including Alpha-actinin-3, IL-6, insulin-like growth factor-2, vitamin D receptor, peroxisome proliferator-activated receptor alpha, and angiotensin-converting enzyme. Genes implicated in associations with obesity will also be analyzed, including fat mass and obesity-associated protein, uncoupling protein 2 and 3, transmembrane protein 18, pro-opiomelanocortin, and melanocortin receptor 4 genes.

**Control measures**

Other important lifestyle factors, including physical activity, nutrition, and sleep behavior, may be strongly associated with our main outcome variables. For example, a recent systematic review and meta-analysis reported that physical activity interventions have had only a small effect on children's overall activity levels. Statistical adjustment for these factors will be necessary to account for possible uneven distribution between the randomized groups and to increase the precision of the estimates of the effect of the randomized treatment. As these factors will only be measured as part of the study design it will not be possible to stratify randomization by these factors.

**Physical activity**

Accelerometry, in combination with a physical activity questionnaire will be used to interpret changes in physical activity. For both measurements, data collection will take place over 7-days at baseline, then over 7-days at the end of each stage. The participants will be instructed to wear a 3-axis accelerometer (wActiSleep +; ActiGraph LLC, Fort Walton Beach, FL) at the right hip during their waking hours, but to remove when bathing, showering, or participating in water sports. A 60 s sampling period will be used throughout, and the count data expressed in counts per minute. This is a validated objective measure of physical activity for use with young people.

Physical activity type and context (e.g., modality) will be determined using the International Physical Activity Questionnaire (IPAQ) Long Form (http://www.ipaq.ki.se/ipaq.htm), a cross-national monitoring tool, which has been validated for use in children and adolescents. The IPAQ will be applied during an interview because there is evidence that interview measures have stronger characteristics than self-administered measures.

**Nutritional intake**

Direct observation is considered the “gold standard” for monitoring dietary intake. However, this approach can be time consuming and impractical for use in large-scale studies. Alternatively, food frequency questionnaires are considerably less burdensome in both time and cost in than other measurement tools. Therefore, food choice will be assessed using 12 questions from the Health Behavior in School Children (HBSC) questionnaire that has been validated for use in New Zealand adolescents aged 14-18 years. The frequency of consumption of food items will be recorded by asking the respondent how many times weekly each item is ate or drank. The food frequency questionnaire will be applied during an interview, along with the IPAQ.

**Sleep behaviour**

Increasing evidence has indicated that short sleep duration may be related to childhood obesity, high blood pressure, and decreased insulin sensitivity among children and adolescents. Sleep behavior will be assessed using the ActiSleep monitor described above. When worn during sleep episodes, the ActiSleep will monitor sleep onset, sleep latency, total sleep time, number and duration of awakenings, and sleep efficiency. An integrated ambient light sensor provides information on subject environment. Study participants will wear the ActiSleep monitor on their non-dominant wrists for intervals of seven consecutive days to provide objective estimates of sleep. Participants will be instructed to keep a record of logging time in bed and time out of bed for each measured sleep episode and to return a completed sleep log with the ActiSleep monitor to the research staff.

**Process evaluation**

The feasibility of COMPASS will be examined using measures of recruitment, retention, adherence, and satisfaction. Evaluation questionnaires will also be administered to determine perceptions of the programme. A 6-point Likert scale format will be used with responses ranging from “Strongly Disagree” through to “Strongly Agree.” Focus group interviews involving 5-6 students and lasting...
5-10 min will also be conducted by trained research assistants. The groups will be based on friendship groups (both single-sex and mixed-sex groups) and will utilize standardized semi-structured questions. The anonymous verbal responses will be recorded by the research assistant. At the end of the session the participants will also be asked if they have anything else to add or would like to discuss anything further.

**Sample size**

The sample size for the feasibility study is based on FMD. Based on the research from a similar population,[63,65] for 90% power, alpha 5% a total of 14 participants, seven in each group are needed to detect a difference of 5% with an SD of 2.5%. The sample size calculation for the main study will be based on FMD and we plan to confirm the SD for this variable in the feasibility study as the range of SD in past research was so wide. If possible we also plan to estimate the SD for abdominal obesity and insulin resistance (OGTT). A sample size of 14 has reasonable precision to estimate variance. The variances and appropriate 95% confidence intervals will be estimated by Chi-square statistics.

**Statistical analysis**

The analysis will by intention to treat, namely that participants will be analyzed as by their randomized intervention allocation. A secondary analysis will be a per-protocol analysis, namely participants will be analyzed as by the intervention they actually completed. For missing data, we will take a multiple imputation approach based on the missing at random assumption for the main outcome variables at the main measurement time. The main outcome variables are: Abdominal obesity, endothelial function and insulin resistance. Other outcomes will include arterial stiffness, lipid profile, inflammatory biomarkers, well-being, and aerobic fitness. General linear models will examine the effect of randomized treatment on the outcome variables at the final measurement time. A sensitivity analysis will be by mixed linear models using all measurement time points as well as possible confounding variables: Daily physical activity, nutritional intake, and sleep behavior. Mixed linear models explicitly model the correlation of repeated measurements on the same individuals to examine rates of change with time and whether these differ by randomized treatment. Associations between adiposity, insulin resistance and endothelial function will be examined by general linear models and logistic regression. These associations will be used to predict the expected proportional reduction in obesity or insulin resistance, for example, attributable to the COMPASS intervention – assuming all else remains constant in the study population.

**DISCUSSION**

Multiple pediatric studies have demonstrated a clustering of cardio-metabolic complications with obesity.[2,141] The metabolic syndrome, manifested by the coexistence of central obesity, dyslipidemia, hypertension, and pre-diabetes, may affect as many as 30% of obese adolescents.[142,143] The symptoms of these conditions do not differ between adolescents and adults, however, the burden of disease is maintained for a longer period of time with less pharmaceutical options available for the younger population.[2] This increasing burden of obesity has created an urgent need to develop strategic prevention and management approaches for New Zealand youth of various ethnic backgrounds. Although lifestyle modification is considered the cornerstone of the management of obesity, there are significant gaps in our understanding of the optimal modalities of exercise to use with an adolescent population, particularly with at-risk, obese adolescents. COMPASS will be one of the first RCTs to address obesity and related cardio-metabolic and CVD concerns in Māori and Pasifika adolescents. The combined study objectives will provide a unique opportunity to gain robust evidence for developing safe and effective exercise options in this cohort.

**IMPLICATIONS**

This research study will provide much needed objective data in an area of research and population that to date has been largely ignored. Given the practical implications of the present study, we will examine the clinical significance for changes in each variable.[144]

**REFERENCES**

1. Ministry of Health (2008). A portrait of health: Key results of the 2006/07 New Zealand Health Survey.
2. Dietz WH. Overweight in childhood and adolescence. N Engl J Med 2004;350:855-7.
3. Franco OH, Peeters A, Bonneux L, de Laet C. Blood pressure in adulthood and life expectancy with cardiovascular disease in men and women: Life course analysis. Hypertension 2005;46:280-6.
4. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. Hypertension 2003;42:1206-52.
5. McFarlin BK, Johnston CA, Tyler C, Hutchison AT, Kueht ML, Reeves R, et al. Inflammatory markers are elevated in overweight Mexican-American children. Int J Pediatr Obes 2007;2:235-41.
6. Nakagami T, Qiao Q, Tuomilehto J, Balkau B, Tajima N, Hu G, et al. Screen-detected diabetes, hypertension and hypercholesterolemia as predictors of cardiovascular mortality in five populations of Asian origin: The DECODA study. Eur J Cardiovasc Prev Rehabil 2006;13:555-61.
7. Teramoto T, Nakaya N, Yokoyama S, Ohashi Y, Mizuno K, Nakamura H, et al. Association between lowering low-density lipoprotein cholesterol with pravastatin and primary prevention of cardiovascular disease in mild to moderate hypercholesterolemic Japanese. J Atheroscler Thromb 2010;17:879-87.
8. Peña AS, Wiltshire E, MacKenzie K, Gent R, Piotto L, Hirte C, et al. Vascular endothelial and smooth muscle function relates to body mass index and glucose in obese and nonobese children. J Clin Endocrinol Metab 2006;91:4467-71.
9. Balagopal P, George D, Patton N, Yarandi H, Roberts WL, Bayne E, et al. Lifestyle-only intervention attenuates the inflammatory state associated with obesity: A randomized controlled study in adolescents. J Pediatr 2005;146:342-8.
10. Meyer AA, Kundt G, Steiner M, Schuff-Werner P, Kienast W. Impaired flow-mediated vasodilation, carotid artery intima-media thickening, and elevated endothelial plasma markers in obese children: The impact of cardiovascular risk factors. Pediatrics 2006;117:1560-7.
11. Peña AS, Belobradjic DP, Wiltshire E, Gent R, Hirte C, Couper J. Adiponectin relates to smooth muscle function and folate in obese children. Int J Pediatr Obes 2010;5:185-91.
12. Balagopal P, George D, Sweeten S, Mann KJ, Yarandi H, Mauras N, et al. Response of fractional synthesis rate (FSR) of fibrinogen, concentration of D-dimer and fibrinolytic balance to physical activity-based intervention in obese children. J Thromb Haemost 2008;6:1296-303.
13. Reinehr T, Kiess W, de Sousa G, Stoffel-Wagner B, Wunsch R. Intima media thickness in childhood obesity: Relations to inflammatory marker, glucose metabolism, and blood pressure. Metabolism 2006;55:113-8.
14. Mange H, Almer G, Haj-Yahya S, Pilz S, Gasser R, Möller R, et al. Preatherosclerosis and adiponectin subfractions in obese adolescents. Obesity (Silver Spring) 2008;16:2578-84.
15. Winer JC, Zern TL, Taksali SE, Dziera J, Cali AM, Wollschlager M, et al. Adiponectin in childhood and adolescent obesity and its association with inflammatory markers and components of the metabolic syndrome. J Clin Endocrinol Metab 2006;91:4415-23.
16. Oliver SR, Rosa JS, Milne GL, Pontello AM, Borntreger HL, Heydari S, et al. Increased oxidative stress and altered substrate metabolism in obese children. Int J Pediatr Obes 2010;5:436-44.
17. Giordano P, Del Vecchio GC, Cucinelli V, Delvecchio M, Altomare M, De Palma F, et al. Metabolic, inflammatory, endothelial and haemostatic markers in a group of Italian obese children and adolescents. Eur J Pediatr 2011;170:845-50.
18. Kumanyika S, Jeffery RW, Morabia A, Ritenbaugh C, Antipatis VJ, Public Health Approaches to the Prevention of Obesity (PHIPO) Working Group of the International Obesity Task Force (IOTF). Obesity prevention: The case for action. Int J Obes Relat Metab Disord 2002;26:425-36.
19. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment: Where do we go from here? Science 2003;299:853-5.
20. Schoenfeld-Warden N, Warden CH. Pediatric obesity. An overview of etiology and treatment. Pediatr Clin North Am 1997;44:339-61.
21. Rosenbaum M, Leibel RL, Hirsch J. Obesity. N Engl J Med 1997;337:396-407.
22. Watts K, Jones TW, Davis EA, Green D. Exercise training in obese children and adolescents: Current concepts. Sports Med 2005;35:375-92.
23. Benson AC, Torode ME, Singh MA. Muscular strength and cardiorespiratory fitness is associated with higher insulin sensitivity in children and adolescents. Int J Pediatr Obes 2006;1:222-31.
24. World Health Organisation. Global Recommendations on Physical Activity and Health. Geneva: World Health Organisation; 2010.
25. LeMura LM, Maziekas MT. Factors that alter body fat, body mass, and fat-free mass in pediatric obesity. Med Sci Sports Exerc 2002;34:487-96.
26. McGuigan MR, Tatasciore M, Newton RU, Pettigrew S. Eight weeks of resistance training can significantly alter body composition in children who are overweight or obese. J Strength Cond Res 2009;23:80-5.
27. Benson AC, Torode ME, Fiatarone Singh MA. The effect of high-intensity progressive resistance training on adiposity in children: A randomized controlled trial. Int J Obes (Lond) 2008;32:1016-27.
28. Lee S, Bacha F, Hannon T, Kuk JL, Boesch C, Arslanian S. Effects of aerobic versus resistance exercise without caloric restriction on abdominal fat, intrahepatic lipid, and insulin sensitivity in obese adolescent boys: A randomized, controlled trial. Diabetes 2012;61:2787-95.
29. Cauza E, Hanusch-Enserer U, Strasser B, Ludvik B, Metz-Schimmerl S, Pacini G, et al. The relative benefits of endurance and strength training on the metabolic factors and muscle function of people with type 2 diabetes mellitus. Arch Phys Med Rehabil 2005;86:1527-33.
30. Honkola A, Forsén T, Eriksson J. Resistance training improves the metabolic profile in individuals with type 2 diabetes. Acta Diabetol 1997;34:245-8.
31. Stewart KJ, Bacher AC, Turner K, Lim JG, Hees PS, Shapiro EP, et al. Exercise and risk factors associated with metabolic syndrome in older adults. Am J Prev Med 2005;28:9-18.
32. Murphy EC, Carson L, Neal W, Baylis C, Donley D, Yeater M. Effects of an exercise intervention using Dance Dance Revolution on endothelial function and other risk factors in overweight children. Int J Pediatr Obes 2009;4:205-14.
33. Tjønna AE, Stølen TO, Bye A, Volden M, Slørdahl SA, Odegård R, et al. The role of dance in the prevention and management of obesity and overweight in schoolchildren. Int J Obes (Lond) 2008;32:1016-27.
34. Montero D, Walther G, Perez-Martin A, Roche E, Vinet A. Endothelial dysfunction, inflammation, and oxidative stress in obese children and adolescents: Markers and effect of lifestyle intervention. Obes Rev 2012;13:441-55.
35. Bergin P. Maori sport and cultural identity in Australia. Aust J Anthropol 2002;13:257.
36. Te Rito P. Leadership in Māori, European cultures and the world of sport. MAI Review 2006;1:1-19.
37. Bellinger B, St Clair Gibson A, Oelofse A, Oelofse R, Lambert M. Energy expenditure of a noncontact boxing training session compared with submaximal treadmill running. Med Sci Sports Exerc 1997;29:1653-6.
38. Arseneau E, Mekary S, Léger LA. VO2 requirements of boxing exercises. J Strength Cond Res 2011;25:348-59.
39. Combs SA, Diehl MD, Staples WH, Conn L, Davis K, Lewis N, et al. Boxing training for patients with Parkinson disease: A case series. Phys Ther 2011;91:132-42.
40. Kravitz L, Greene L, Burkett Z, Wongsathikun J. Cardiovascular response to punching tempo. J Strength Cond Res 2003;17:104-8.
41. Chatterjee P, Banerjee AK, Majumdar P, Chatterjee P. Study of plasma lipid and lipoprotein profile in elite women boxers during a six weeks’ training programme. JNMA J Nepal Med Assoc 2007;46:25-30.
42. Sokol DK. The not-so-sweet science: The role of the medical profession in boxing. J Med Ethics 2004;30:513-4.
43. Hollis S, Campbell F. What is meant by intention to treat analysis? Survey of published randomised controlled trials. BMJ 1999;319:670-4.
44. Moreno LA, Blay MG, Rodríguez G, Blay VA, Mesana MI, Olivares JL, et al. Screening performances of the International Obesity Task Force body mass index cut-off values in adolescents. J Am Coll Nutr 2006;25:403-8.
45. Durie M. Exploring the interface between science and indigenous knowledge. Journal 2004;21.
46. University of Southern Queensland. User guide to Brunel Mood Scale. : University of Southern Queensland; 2010.
47. Terry PC, Lane AM, Fogarty GJ. Construct validity of the profile of mood States – Adolescents for use with adults. Psychol Sport Exerc 2003;4:125-39.
48. Lane AM, Soos I, Leibinger E, Karsai I, Hamar P. Validity of the Brunel Mood Scale for use with UK, Italian and Hungarian Athletes. In: Lane AM, editor. Mood and Human Performance: Conceptual, Measurement and Applied Issues. New York: Nova Science; 2007.
49. Hashim HA, Zulkifli EZ, Yusof Hanafi HA. Factorial validation of malaysian adapted brunel mood scale in an adolescent sample. Asian J Sports Med 2010;1:185-94.
50. Impellizzeri FM, Rampinini E, Coutts AJ, Sassi A, Marcora SM. Use of RPE-based training load in soccer. Med Sci Sports Exerc 2004;36:1042-7.
51. Scott TJ, Black CR, Quinn J, Coutts AJ. Validity and reliability of the session-RPE method for quantifying training in Australian football: A comparison of the CR10 and CR100 scales. J Strength Cond Res 2013;27:270-6.
52. Wallace LK, Slattery KM, Coutts AJ. The ecological validity and application of the session-RPE method for quantifying training loads in swimming. J Strength Cond Res 2009;23:33-8.
53. Perandinia LA. Use of session RPE to training load quantification and training intensity distribution in taekwondo athletes. Sci Sports 2012;27:e25-30.
54. Milanez VF, Pedro RE, Moreira A, Boullosa DA, Salle-Neto F, Nakamura FY. The role of aerobic fitness on session rating of perceived exertion in futsal players. Int J Sports Physiol Perform 2011;6:358-66.
55. Statistics New Zealand. QuickStats About Lower Hutt City. Available from: http://www.stats.govt.nz/Census/2006CensusHomePage/QuickStats/AboutAPlace/SnapShot.aspx?type=ta and ParentID=1000009 and tab=Education and id=2000046. [Last accessed on 2012 Jun 12].
67. Stoner L, Sabatier MJ. Use of ultrasound for non-invasive assessment of flow-mediated dilation. J Atheroscler Thromb 2012;19:407-21.

68. Stoner L, Tarrant MA, Fryer S, Faulkner J. How should flow-mediated dilation be normalized to its stimulus? Clin Physiol Funct Imaging 2013;33:75-8.

69. Frayn KN. Visceral fat and insulin resistance: Causative or correlative? Br J Nutr 2000;83:S71-7.

70. Fox CS, Massaro JM, Hoffmann U, Pou KM, Maurovich-Horvat P, Liu CY, et al. Abdominal visceral and subcutaneous adipose tissue compartments: Association with metabolic risk factors in the Framingham Heart Study. Circulation 2007;116:39-48.

71. Fujimoto WY, Bergstrom RW, Boyko EJ, Chen KW, Leonetti DL, Newell-Morris L, et al. Visceral adiposity and incident coronary heart disease in Japanese-American men. The 10-year follow-up results of the Seattle Japanese-American Community Diabetes Study. Diabetes Care 1999;22:1808-12.

72. Kannel WB. Lipids, diabetes, and coronary heart disease: Insights from the Framingham Study. Am Heart J 1985;110:1100-7.

73. Goran MI. Visceral fat in prepubertal children: Influence of obesity, anthropometry, ethnicity, gender, diet, and growth. Am J Hum Biol 1999;11:201-7.

74. Gower BA, Nagy TR, Goran MI. Visceral fat, insulin sensitivity, and lipids in prepubertal children. Diabetes 1999;48:1515-21.

75. Goran MI, Gower BA. Relation between visceral fat and disease risk in children and adolescents. Am J Clin Nutr 1999;70:149S-56.

76. Owens S, Gutfin B, Ferguson M, Allison J, Karp W, Le NA. Visceral adipose tissue and cardiovascular risk factors in obese children. J Pediatr 1998;133:41-5.

77. Pontiroli AE, Pizzocri P, Giacomelli M, Marchi M, Vedani P, Cucchi E, et al. Ultrasound measurement of visceral and subcutaneous fat in morbidly obese patients before and after laparoscopic adjustable gastric banding: Comparison with computerized tomography and with anthropometric measurements. Obes Surg 2002;12:648-51.

78. Ribeiro-Filho FF, Faria AN, Azjen S, Zanella MT, Ferreira SR. Methods of estimation of visceral fat: Advantages of ultrasonography. Obes Res 2003;11:1488-94.

79. Leite CC, Wajchenberg BL, Radominski R, Matsuda D, Cerri GG, Halpern A. Intra-abdominal thickness by ultrasonography to predict risk factors for cardiovascular disease and its correlation with anthropometric measurements. Metabolism 2002;51:1034-40.

80. Stolk RP, Wink O, Zelissen PM, Meijer R, van Gils AP, Grobbee DE. Validity and reproducibility of ultrasonography for the measurement of intra-abdominal adipose tissue. Int J Obes Relat Metab Disord 2001;25:1346-51.
81. De Lucia Rolfe E, Sleigh A, Finucane FM, Brage S, Stolk RP, Cooper C, et al. Ultrasound measurements of visceral and subcutaneous abdominal thickness to predict abdominal adiposity among older men and women. Obesity (Silver Spring) 2010;18:625-31.
82. Tornaghi G, Raiteri R, Pozzato C, Rispoli A, Bramani M, Cipolat M, et al. Anthropometric or ultrasonic measurements in assessment of visceral fat? A comparative study. Int J Obes Relat Metab Disord 1994;18:771-5.
83. Bazzocchi A, Filonzi G, Ponti F, Sassi C, Salizzoni E, Battista G, et al. Accuracy, reproducibility and repeatability of ultrasonography in the assessment of abdominal adiposity. Acad Radiol 2011;18:1133-43.
84. Iacobellis G. Imaging of visceral adipose tissue: An emerging diagnostic tool and therapeutic target. Curr Drug Targets Cardiovasc Haematol Disord 2005;5:345-53.
85. DeFronzo RA, Tobin JD, Andres R. Glucose clamp technique: A method for quantifying insulin secretion and resistance. Am J Physiol 1979;237:E214-23.
86. Bergman RN, Prager R, Volund A, Olefsky JM. Equivalence of the insulin sensitivity index in man derived by the minimal model method and the euglycemic glucose clamp. J Clin Invest 1987;79:790-800.
87. Matsuda M, DeFronzo RA. Insulin sensitivity indices obtained from oral glucose tolerance testing: Comparison with the euglycemic insulin clamp. Diabetes Care 1999;22:1462-70.
88. McAuley KA, Williams SM, Mann JJ, Walker RJ, Lewis-Barned NJ, Temple LA, et al. Diagnosing insulin resistance in the general population. Diabetes Care 2001;24:460-4.
89. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: Insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985;28:412-9.
90. Akinci G, Coskun S, Akinci B, Hekimsoy Z, Bayindir P, Onur E, et al. Atherosclerosis risk factors in children of parents with the metabolic syndrome. Atherosclerosis 2007;194:e165-71.
91. Pacifico L, Anania C, Martino F, Cantisani V, Pascone R, Marcantonio A, et al. Functional and morphological vascular changes in pediatric nonalcoholic fatty liver disease. Hepatology 2010;52:1643-51.
92. Dogra G, Irish A, Chan D, Watts G. Insulin resistance, inflammation, and blood pressure determine vascular dysfunction in CKD. Am J Kidney Dis 2006;48:926-34.
93. Mattsson N, Rönnemaa T, Juonala M, Viikari JS, Jokinen E, Hutri-Kähönen N, et al. Arterial structure and function in young adults with the metabolic syndrome: The cardiovascular risk in young finns study. Eur Heart J 2008;29:784-91.
94. Balletshofer BM, Rittig K, Stock J, Lehn-Stefan A, Overkamp D, Dietz K, et al. Insulin resistant young subjects at risk of accelerated atherosclerosis exhibit a marked reduction in peripheral endothelial function early in life but not differences in intima-media thickness. Atherosclerosis 2003;171:303-9.
95. Vázquez LA, Pazos F, Berrazaeta JR, Fernández-Escalante C, García-Unzueta MT, Freijanes J, et al. Effects of changes in body weight and insulin resistance on inflammation and endothelial function in morbid obesity after bariatric surgery. J Clin Endocrinol Metab 2005;90:316-22.
96. Consedine NS, Moskowitz JT. The role of discrete emotions in health outcomes: A critical review. Appl Prev Psychol 2007;12:59-75.
haemodynamics: A systematic review and meta-analysis. Eur Heart J 2010;31:1865-71.

106. Williams B, Lacy PS, Thom SM, Cruickshank K, Stanton A, Collier D, et al. Differential impact of blood pressure-lowering drugs on central aortic pressure and clinical outcomes: Principal results of the conduit artery function evaluation (CAFE) study. Circulation 2006;113:1213-25.

107. Lowe A, Harrison W, El-Aklouk E, Ruygrok P, Al-Jumailly AM. Non-invasive model-based estimation of aortic pulse pressure using suprasystolic brachial pressure waveforms. J Biomech 2009;42:2111-5.

108. Lin AC, Lowe A, Sidhu K, Harrison W, Ruygrok P, Stewart R. Evaluation of a novel sphygmomanometer, which estimates central aortic blood pressure from analysis of brachial artery suprasystolic pressure waves. J Hypertens 2012;30:1743-50.

109. Lydakis C, Stefanaki E, Stefanaki S, Thalassinos E, Kavousanaki M, Lydaki D. Correlation of blood pressure, obesity, and adherence to the Mediterranean diet with indices of arterial stiffness in children. Eur J Pediatr 2012;171:1373-82.

110. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972;18:499-502.

111. Kelishadi R, Hashemi M, Mohammadifard N, Asgary S, Rangul V, Holmen TL, Kurtze N, Cuypers K, Midthjell K. A genome-wide association meta-analysis identifies new childhood obesity loci. Nat Genet 2012;44:526-31.

112. Metcalf B, Henley W, Wilkin T. Effectiveness of intervention on physical activity of children: Systematic review and meta-analysis of controlled trials with objectively measured outcomes (EarlyBird 54). BMJ 2012;345:e5888.

113. Pate RR, O’Neill JR, Mitchell J. Measurement of physical activity in preschool children. Med Sci Sports Exerc 2010;42:508-12.

114. Dollman J, Okely AD, Hardy L, Timperio A, Salmon J, Hills AP. A hitchhiker’s guide to assessing young people’s physical activity: Deciding what method to use. J Sci Med Sport 2009;12:518-25.

115. Ottevaere C, Huybrechts I, De Bourdeaudhuij I, Sjöström M, Ruiz JR, Ortega FB, et al. Comparison of the IPAQ-A and actigraph in relation to VO2 max among European adolescents: The HELENA study. J Sci Med Sport 2011;14:317-24.

116. Rangel V, Holmen TL, Kurtze N, Cuypers K, Midtjell K. Reliability and validity of two frequently used self-administered physical activity questionnaires in adolescents. BMC Med Res Methodol 2008;8:47.

117. Hagstromer M, Bergman P, De Bourdeaudhuij I, Ruiz JR, Ortega FB, et al. Concurrent validity of a modified version of the international physical activity questionnaire (IPAQ-A) in European adolescents: The HELENA Study. Int J Obes (Lond) 2008;32:S42-8.

118. Ottevaere C, Huybrechts I, De Meester F, De Bourdeaudhuij I, Cuenca-Garcia M, De Henauw S. The use of accelerometry in adolescents and its implementation with non-wear time activity diaries in free-living conditions. J Sports Sci 2011;29:103-13.

119. Sallis JF, Saelens BE. Assessment of physical activity by self-report: Status, limitations, and future directions. Res Q Exerc Sport 2000;71:S1-14.

120. Gersovitz M, Madden JP, Smiciklas-Wright H. Validity of the 24-hr. dietary recall and seven-day record for group comparisons. J Am Diet Assoc 1978;73:48-55.
130. Frank GC. Taking a bite out of eating behavior: Food records and food recalls of children. J Sch Health 1991;61:198-200.
131. Mertz W. Food intake measurements: Is there a “gold standard”? J Am Diet Assoc 1992;92:1463-5.
132. Block G. A review of validations of dietary assessment methods. Am J Epidemiol 1982;115:492-505.
133. Kushi LH. Gaps in epidemiologic research methods: Design considerations for studies that use food-frequency questionnaires. Am J Clin Nutr 1994;59:180S-4.
134. Willett W. Nutritional Epidemiology. New York: Oxford University Press; 1998.
135. Howe AS, Mandic S, Parnell WR, Skidmore PM. Attitudes to food differ between adolescent dieters and non-dieters from Otago, New Zealand, but overall food intake does not. Public Health Nutr 2012;16:36-45.
136. Wong JE, Parnell WR, Black KE, Skidmore PM. Reliability and relative validity of a food frequency questionnaire to assess food group intakes in New Zealand adolescents. Nutr J 2012;11:65.
137. Chen X, Beydoun MA, Wang Y. Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. Obesity (Silver Spring) 2008;16:265-74.
138. Hitze B, Bosy-Westphal A, Bielfeldt F, Settler U, Plachta-Danielzik S, Pfeuffer M, et al. Determinants and impact of sleep duration in children and adolescents: Data of the Kiel Obesity Prevention Study. Eur J Clin Nutr 2009;63:739-46.
139. Javaheri S, Storfer-Isser A, Rosen CL, Redline S. Sleep quality and elevated blood pressure in adolescents. Circulation 2008;118:1034-40.
140. Javaheri S, Storfer-Isser A, Rosen CL, Redline S. Association of short and long sleep durations with insulin sensitivity in adolescents. J Pediatr 2011;158:617-23.
141. Wattigney WA, Webber LS, Srinivasan SR, Berenson GS. The emergence of clinically abnormal levels of cardiovascular disease risk factor variables among young adults: The Bogalusa Heart Study. Prev Med 1995;24:617-26.
142. Duncan GE, Li SM, Zhou XH. Prevalence and trends of a metabolic syndrome phenotype among U.S. Adolescents, 1999-2000. Diabetes Care 2004;27:2438-43.
143. Cook S, Weitzman M, Auinger P, Nguyen M, Dietz WH. Prevalence of a metabolic syndrome phenotype in adolescents: Findings from the third National Health and Nutrition Examination Survey, 1988-1994. Arch Pediatr Adolesc Med 2003;157:821-7.
144. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. Med Sci Sports Exerc 2009;41:3-13.
145. Stoner L, Stoner KR, Young JM, Fryer S. Preventing a Cardiovascular Disease Epidemic among Indigenous Populations through Lifestyle Changes. Int J Prev Med 2012;3:230-40.

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