# Associations of patterns of daily life, physical fitness and body composition of primary school age children

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## Attestation of Authorship

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

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Geoff Kira

# Glossary

| Term | Meaning |
|---|---|
| Resting metabolic rate | The amount of energy (kilocalories or kilojoules) expended to maintain bodily function at rest, usually over a period of 24 hours (for example 2304 $kcal.day^{-1}$) |
| Energy balance | Energy input (food) – Energy output (Energy expenditure) = positive/negative energy balance. A positive energy balance adds excess weight and negative energy balance removes weight. |
| Physical fitness | The ability to participate in physical activity. For the purposes of this thesis, physical fitness is both cardiorespiratory fitness and muscle strength fitness. |
| Bone integrity | See stiffness index |
| Stiffness index | Strength of bone as measured by Quantitative Ultrasound (QUS) |
| Body Mass | "Body mass" is the preferred term as weight may vary dependent on the environment (gravitational field) in which the "mass" resides. The standard is at sea level (one atmosphere) where body mass = body weight. Thus from this point on, body mass (BM) will be used to refer to body weight. |
| Weight | See body mass (BM) |
| Glucose homeostasis | Measures of glucose, insulin and any derived measures such as homeostatic model assessment (HOMA) or insulin resistance. |

# Abbreviations

| Abbreviation | Definition |
|---|---|
| ANCOVA | analysis of covariance |
| ATP | American treatment panel |
| BM | body mass |
| BMC | bone mineral content |
| BMD | bone mineral density |
| BMI | body mass index |
| BOC | British oxygen company Ltd |
| BOT | board of trustees |
| BP | blood pressure |
| BUA | broadband ultrasound attenuation |
| CD | compact disc |
| CDC | Centers for disease control and prevention |
| CI | confidence interval |
| Cm | centimetre(s) |
| CRF | cardiorespiratory fitness |
| CT | computerised tomography |
| CVD | cardiovascular disease |
| DBP | diastolic blood pressure |
| DEXA | dual energy x-ray absorptiometry |
| EE | energy expenditure |
| EOI | expression of interest |
| FFM | fat free mass |
| FGIR | fasting glucose/insulin ratio |
| FIRI | fasting insulin resistance index |
| FM | fat mass |
| FQ | food quotient |
| FSIVGTT | frequently sampled intravenous glucose tolerance test |

| | |
|---|---|
| GE | General electric ltd |
| HDL-C | high density lipoprotein cholesterol |
| HHQ | household questionnaire |
| HOMA | homeostatic model assessment |
| HOMA2 | homeostatic model assessment version 2 |
| HOMA2-B% | homeostatic model assessment version 2 beta cell function |
| HOMA2-S% | homeostatic model assessment version 2 insulin sensitivity |
| IANZ | International accreditation new zealand |
| IDF | International diabetes federation |
| IOTF | International obesity task force |
| IQR | interquartile range |
| IR | insulin resistance |
| IRS | insulin resistance syndrome |
| ISI | insulin sensitivity index |
| kcal | kilocalorie |
| kg | kilogram(s) |
| LDL-C | low density lipoprotein cholesterol |
| M | metre(s) |
| Min | minute(s) |
| MSF | muscular strength fitness |
| NEAT | Non exercise activity thermogenesis |
| OR | odds ratio |
| PA | physical activity |
| PAL | physical activity level |
| PBF | percentage body fat |
| PF | physical fitness |
| PP | pulse pressure |
| PTH | parathyroid hormone |
| % | percent |

| | |
|---|---|
| ± | plus or minus |
| QUICKI | quantitative insulin sensitivity check index |
| QUS | quantitative ultrasound |
| RDI | recommended dietary intake |
| RER | respiratory exchange ratio |
| RHR | resting heart rate |
| RIA | radio-immuno assay |
| RMR | resting metabolic rate |
| SBP | systolic blood pressure |
| SD | standard deviation |
| SDS | standard deviation score |
| SES | socioeconomic status |
| Si | insulin sensitivity |
| SOS | speed of sound |
| SPSS | statistical package for the social sciences now PASW (predictive analytics software) |
| SRT | shuttle run test |
| TEE | total energy expenditure |
| TEM | technical error of measurement |
| TG | triglyceride |
| TV | television |
| T2DM | type 2 diabetes mellitus |
| $VCO_2$ | carbon dioxide production rate ($mL.min^{-1}.kg^{-1}$) |
| $VO_2$ | oxygen uptake ($mL.min^{-1}.kg^{-1}$) |
| WHO | World health organisation |

# Co-authored works

Rush, E.C., Scragg, R., Schaaf, D., **Juranovich, G.**, and Plank, L.D. (2008). *Indices of fatness and relationships with age, ethnicity and lipids in New Zealand European, Māori and Pacific children*. European Journal of Clinical Nutrition, Feb 27. [Epub ahead of print].

Graham, D., **Kira, G.**, Conaglen, J., McLennan, S., & Rush, E. (2009). *Vitamin D status of Year 3 children and supplementation through schools with fortified milk.* Public Health Nutrition, 24, 1-6.

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# Ethics approvals

*Chapter 3:*

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*Chapter 4:*

Approved by the Northern Y Regional Ethics Committee (NTY/05/12/108) on the 27th of September 2006 and subsequently through the Auckland University of Technology Ethics Committee.

*Chapter 5:*

Ethical approval for Phase One (NTY/06/10/100) was provided by the Northern Region Y Ethics committee in July 2006 (2006). Phase Two ethics was approved by the Northern Region Y Ethics committee in November 2006 as an addendum to Phase One.

# Abstract

The daily patterns of life, for example, food, physical activity and inactivity and sleep as well as physical fitness are associated with the accumulation of excess body fat in children. A positive energy balance between food (energy intake) and metabolism, particularly physical activity (energy output) is the accepted explanation. The reality of daily life for children is excessive calorie consumption, imbalances in macronutrient intake and missed opportunities for activity are being driven by the physical and social environment.

Excess body fat tracks into adulthood and is associated with decreased insulin sensitivity, which may lead to increased risk of insulin resistance and chronic disease. Resting metabolic rate and substrate utilisation (measured by respiratory exchange ratio) are implicated in the prediction of weight gain in adults, but these relationships have been rarely explored in children. Both insulin and respiratory exchange provide insights into the pathways of accumulation of body fat.

The purpose of this body of work presented here was to explore and explain how lifestyle patterns, substrate metabolism, physical fitness attributes and insulin resistance are related to excess body fat accumulation in children.

“Project Energize” is a Waikato District Health Board-initiated through-school nutrition and physical activity intervention, operated in a growing number of primary schools throughout the Waikato. Data was collected from Project Energize control and programme schools between 2004 and 2006. The children that participated in this body of work were aged between 5 and 12 years of age (20% Māori). Fat mass (FM) change in Hamilton and Waikato primary school age children over a two year period is reported in study one (n=618). Study two (n=69) and three (n=169) are cross-sectional sub-studies of low decile schools (<3) that employ two methods of metabolic assessment; indirect calorimetry and glucose homeostasis; to investigate the relationships between food, activity, fitness with body composition and metabolic risk.

More than 70% of the increase in body mass index (BMI) and percentage body fat (PBF) could be explained by the same measures two years earlier and more than 10% of the reported food, activity and sleep behaviours were able to be predicted from the responses two years earlier. There were no clear associations found with resting respiratory exchange ratio (RER), but resting metabolic rate (RMR) was best explained (45%) by fat free mass with a

further 3% explained by cardiorespiratory fitness. Children with longer legs (as represented by height) travelled further, but FM attenuated final speed. Children with more fat had higher insulin resistance. Physical fitness was not associated with insulin resistance. Overall, a pattern of increased FM was linked to

1. FM two years previously
2. a lesser speed attained in the 20m Shuttle Run Test and
3. higher insulin resistance.

A focus on weight gain rather than change in FM and FFM, fitness and metabolic markers as the outcome of interventions is unlikely to show short or medium term changes. Therefore it is recommended that when instigating school-based nutrition and physical activity programmes, there must also be a focus on the daily patterns of life alongside community, family and culture-based partnerships to support sustainable behavioural change.

# 1. Introduction

Global concerns are being raised of the increasing number of children experiencing obesity and have high risk of cardiovascular disease (CVD) and type 2 diabetes mellitus (henceforth termed diabetes). Research has confirmed that it is likely that obese children will be obese adults (Freedman et al., 2005; Fuentes, Notkola, Shemeikka, Tuomilehto, & Nissinen, 2003; Rosenbloom, Joe, Young, & Winter, 1999) and there is a close relationship between body mass, cardiovascular disease and diabetes (Garrison, Higgins, & Kannel, 1996; Glowinska, Urban, & Koput, 2002; Hotu, Carter, Watson, Cutfield, & Cundy, 2004) (Figure 1.) Thus there is a body of research that has focused upon the accumulation of body mass in children (Darnton-Hill, Nishida, & James, 2004; McCarthy et al., 2007).

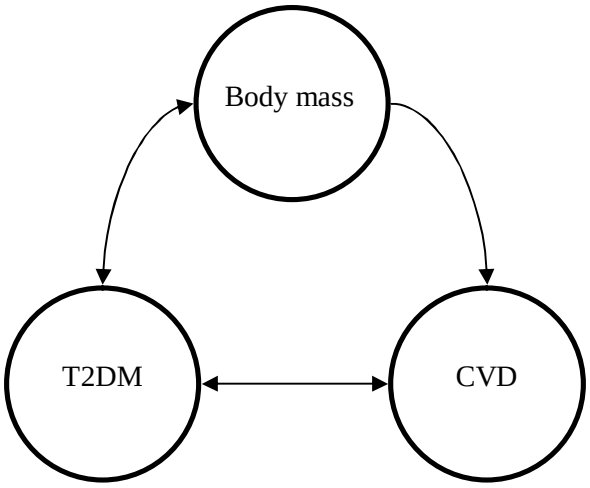


**Figure 1. The relationships of body mass, cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM).**

## 1.1 Body Mass Status

The body mass of growing children is a dynamic process and a measure of body mass status that is assessed relative to age and external standards are a more accurate reflection of changes in body mass. The most common measure of body mass is the body mass index or BMI. It is calculated as weight (kilograms) divided by height squared (metres). Many studies have now utilised an international standard of BMI from the International Obesity task Force (IOTF) (Cole, Bellizzi, & Flegal, 2000). It relates the use of standardized scores to classify body mass into thinness (Grade 1 and 2), normal, overweight and obese. This score is based on prediction of BMI at age 18 years. If the predicted BMI is between 25 and 30kg/$m^2$ then

the classification is overweight and if greater than or equal to 30 kg/m$^2$ the classification is obese. This is based on world health organization definitions of overweight and obesity in adults (World Health Organisation, 2000). A popular alternative has been the use of the United States Centers for Disease Control and Prevention (CDC) percentiles (85$^{th}$: at risk for overweight; 95$^{th}$: overweight; 99$^{th}$; obese). Janssen et al. (2005) found that both sets of reference norms were powerful predictors of obesity and coronary heart disease risk factors in young adulthood and difference between the two are negligible. In contrast, Clarke and Lauer (1993) and Voss (2006) highlight methodological and modeling issues and question the use of BMI in children. Research has revealed that there are many determinants of body mass (Budd & Hayman, 2006; Moreno et al., 2004; Rennie, Johnson, & Jebb, 2005), and nutrition and physical activity are the most common factors studied. This is because they represent the modifiable aspects part of the energy balance equation (Figure 2). Metabolic rate and diet-induced thermogenesis are very difficult to modify, hence there is less focus on those aspects in interventions. An additional confounding factor in the energy balance equation for children is the energy requirement for normal linear growth (Goran & Treuth, 2001).

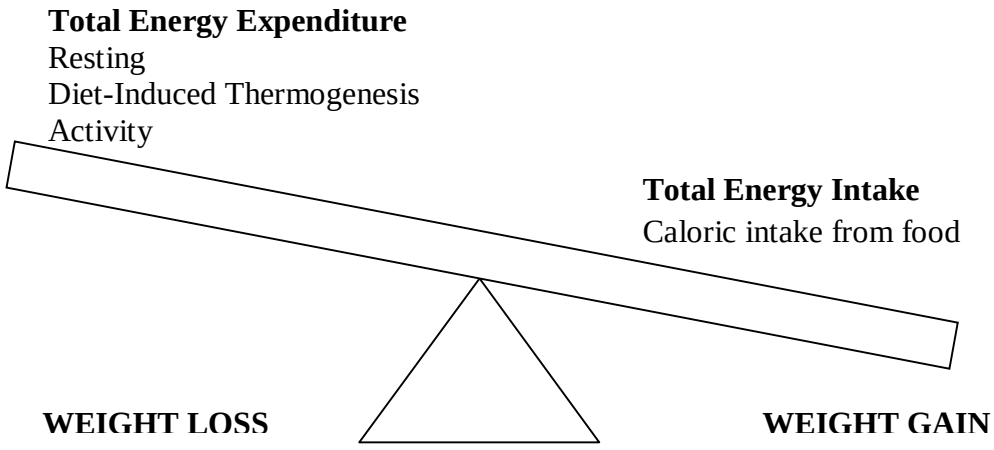


**Figure 2. Energy-based determinants of the energy balance equation**

In New Zealand, the national survey of New Zealand children (5-14 year olds) (Ministry of Health, 2003) found that 21.3% of all children were overweight and 9.8% were obese by IOTF BMI cut-off points for obesity. Ethnic disparity is evident in that 24.9% of Māori children were overweight and 16.2% obese. However, non-European children are likely to live in lower socioeconomic circumstances, than New Zealand European, where the

prevalence of overweight and obesity is higher. Thus there is an interaction between ethnicity and socioeconomic status.

### 1.2 Food and activity patterns

Causation models for obesity include low physical activity and excess calories but measurement is difficult. The model for physical activity has progressed further, than the excess calorie intake model, with the advent of objective measures such as pedometry and accelerometry. In contrast, it is much more difficult to develop causative models using food intake. In a naturalistic setting children's food intake must be measured subjectively using recall, but subjective recall is not precise (Livingstone & Black, 2003) and children, especially overweight and obese, tend to under-report (Dietz, 1991). The issues associated with the measurement of food patterns may be the reason behind the conflicting results from research.

In a 17-year long study of two to eighteen year olds (n=228), Alexy et al. (2004), found that dietary differences did not explain body mass status. Under-reporting, a small study sample and difficulty in identifying the small indulgences are probably responsible for the gradual increases in body mass status and are likely to be obstacles in the final analysis. Moreno et al. (2007) claims that carbohydrate intake is related to childhood obesity, whereas in Swiss children, protein consumption was found to be a positive correlate with weight gain (Aeberli, Kaspar, & Zimmermann, 2007). In contrast, fat intake also presented as a contributing factor to weight gain by Robertson et al. (1999) in American four to seven year olds. But Berkey et al. (2000) has demonstrated that the small and cumulative effects of excessive caloric intake in children (nine to fourteen years) do contribute to increases in BMI over time. Thus energy balance may be the ultimate indicator of weight gain.

### 1.3 Substrate utilisation/fuel partitioning

Interestingly, although a positive energy balance is commonly thought to be the sole factor contributing to weight gain, Westerterp (1993) suggests that there is likely to be an additional contribution to weight gain, namely substrate utilisation, that is accurately represented by twenty four hour respiratory exchange ratio (RER). This is a measure of the amount of carbon dioxide produced in relation to the oxygen utilized and is influenced by the oxidation of the macronutrients, carbohydrate, fat and protein. Ravussin and Bogardus (2000) relate that some of the variability in BMI can be explained by metabolic rate (12%) and fat oxidation (5%) showing some effect on energy balance (metabolic rate and another effect through an

alternative pathway - RER). Maffeis et al. (1995) found that obese prepubertal boys had a higher fat oxidation (18 g/day) than non-obese boys. Additionally, Tounian et al. (2003) also found that fat oxidation correlated with fat mass. This phenomenon was explained by diet; the relatively higher fat intake of obese children and therefore higher fat oxidation rate reflecting that total caloric intake exceeded expenditure (Maffeis, Pinelli, & Schutz, 1996).

### 1.4 Insulin Sensitivity

Insulin is an anabolic hormone with a major role in carbohydrate and fat metabolism. A high RER (elevated carbohydrate oxidation), a low metabolic rate, and reduced insulin sensitivity (Si) have all been identified as predictors in obesity (Bray, 1996). Measurement of insulin sensitivity is important as it enables the quantification of the point of insulin resistance; the point where cells are no longer able to respond to insulin and absorb glucose optimally. (See Figure 3). Insulin resistance (IR) is clinically defined as the "inability of a known quantity of exogenous or endogenous insulin to increase glucose uptake and utilization in an individual as much as it does in a normal population" (Lebovitz, 2001). Insulin resistance is implicated as a precursor to type 2 diabetes (Lillioja et al., 1993) and thus highlights the importance of measuring insulin sensitivity. In obese preadolescent children insulin resistance and hyperinsulinaemia have been found to coexist (Caprio, Bronson, Sherwin, Rife, & Tamborlane, 1996) and fat mass was shown to be the prime promoter of insulin dysfunction in children (Gallistl, Sudi, Mangge, Erwa, & Borkenstein, 2000). The liver and skeletal muscle are the major organs where insulin mediates glucose release, disposal and storage. These processes are dysfunctional in the insulin resistance state.

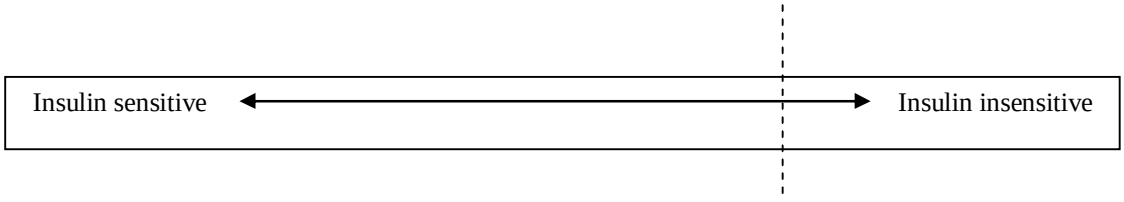


**Figure 3. Scale of insulin sensitivity with insulin resistance cutoff near low insulin sensitivity**

The gold standard measurement of insulin sensitivity, the euglycaemic-hyperinsulinaemic clamp method (DeFronzo, Tobin, & Andres, 1979), is an invasive, expensive, time consuming, and difficult assessment to perform (Conwell, Trost, Brown, & Batch, 2004). Many alternatives to the clamp method have been proposed, including fasting insulin ($I_0$)

(Cutfield & Hofman, 2005), Insulin-Glucose ratio (Legro, Finegood, & Dunaif, 1998), Frequently Sampled Intravenous Glucose Tolerance Test (FSIVGTT) (Bergman, Ider, Bowden, & Cobelli, 1979), Quantitative Insulin Sensitivity Check Index (QUICKI) (Katz et al., 2000), and the Homeostatic Model Assessment (HOMA) (Matthews et al., 1985a). HOMA, while still frequently used has been met with mixed reviews from researchers (Cutfield & Hofman, 2005; Inchiostro, 2005; Katsuki et al., 2002; Keskin, Kurtoglu, Kendirci, Atabek, & Yazici, 2005; Yokoyama et al., 2004). A modified version of HOMA, HOMA2 was developed to improve the predictive value of the original method (Wallace, Levy, & Matthews, 2004b). Fasting insulin and glucose samples may now be entered into a computerised DOS-based algorithm to produce insulin sensitivity (HOMA2-S%) and beta cell function (HOMA2-B%) values (Hines, Kennedy, & Holman, 2004). Normative results for adults are 100% for either variable (HOMA2-S% or HOMA2-B%), but no values have been charted for children using this method even though the new method has been utilised in paediatric studies (Benson, Torode, & Singh, 2006; Conwell et al., 2004).

Energy restricted high carbohydrate based diets have been found to be positively-related to insulin sensitivity in adults although equivalent investigations have not been conducted in children (McClenaghan, 2005). In contrast, dietary fat has been shown to reduce insulin sensitivity (Mayer-Davis et al., 1997) and increased consumption of wholegrain foods (Liese et al., 2003) and dietary fibre (Liese et al., 2005) have been shown to improve sensitivity. Additionally, physical activity has also shown to contribute positive effects to insulin sensitivity (Schmitz et al., 2002). In the developing child the close synergies between dietary quality and physical activity will mediate physical (and metabolic) fitness. There are now compelling reports that physical fitness is an important factor for insulin sensitivity in children (Ruiz et al., 2006) although fat mass may be a mediating component (Ball et al., 2004).

## 1.5 Physical Fitness

Physical fitness is generally defined as the “ability to perform physical activity”. The definition evolved to account for variants of physical fitness dependent on the perspective. A health-related physical fitness definition, such as that proposed by Bouchard and Shepard (1992), incorporates five components: morphological, muscular, motor, cardiorespiratory and metabolic. The metabolic (Si), cardiorespiratory fitness (CRF) and muscle strength fitness (MSF) are the components of health-related physical fitness explored in this thesis. Cardiorespiratory fitness is a measure of the “ability to perform large muscle, dynamic,

moderate-to-high exercise for a prolonged period" (American Council for Sports Medicine, 2005). Muscle strength is "maximal force that can be generated by a specific muscle or muscle group" (Vanhees et al., 2005) and relates both to muscle and body mass.

Physical fitness, specifically cardiorespiratory fitness (CRF - often known as aerobic fitness), has been a common focus of insulin sensitivity studies in children (Lee, Bacha, Gungor, & Arslanian, 2006). However very few studies include children younger than 10 years of age (Ball, et al., 2004; Carrel, Meinen, Garry, & Storandt, 2005; Eliakim, Scheett, Newcomb, Mohan, & Cooper, 2001) . While increased CRF does promote insulin sensitivity; increased fat mass has a negative effect. In addition to increased insulin sensitivity, cardiovascular risk factors (Froberg & Andersen, 2005) also seem to be reduced with increased cardiorespiratory fitness. Although few child studies have shown a positive association of muscular strength fitness (MSF) with insulin sensitivity (Benson, Torode, & Singh, 2006), it would be expected, similar to adults, that both increased CRF and strength would be associated with increased insulin sensitivity in children.

## 1.6 Demographics

Demographics play a vital role in aetiology of obesity apparently augmenting the impact of the environment (nutrition and physical activity) on phenotype (genetics). Many factors may be used to describe the population that is being studied, for example, sex, age, ethnicity, language, country of birth, early childhood care and education, shared parenting arrangements, primary caregiver's relationship to child, age, education, income support, employment or occupation status, and household characteristics (Ministry of Health, 2008b). However for the purpose of the thesis, age, sex, ethnicity and socioeconomic status (SES) were deemed the most important and accessible variables to control for in a study of children. Although age and sex are self-explanatory variables, the use and method of categorisation of ethnicity and SES requires elaboration.

### 1.6.1 Ethnicity

Ethnicity is defined, by Statistics New Zealand (Statistics New Zealand, 2005), as "the ethnic group that people identify with or feel they belong to" and as such "is a measure of cultural affiliation, as opposed to race, ancestry, nationality or citizenship". New Zealand has a unique multicultural environment, encompassing a large number of European-descent and smaller groups of Māori, Pacific, Asian and other peoples. Additionally, there are significant numbers of people who subscribe to multiple ethnicities. Māori and Pacific peoples have the

highest incidence and age-adjusted prevalence obesity and cardiovascular and type 2 diabetes events (Ministry of Health, 2002, 2004). Maori and Pacific children appear to be following the same trend of increased size and associated risk (Ministry of Health, 2008b). Further, research of Māori adults has found that the prevalence of insulin resistance is higher than in the European population (Tipene-Leach et al., 2004). A model of ethnic and biological interactions in the presence of a genetic inheritance of insulin resistance has had some support internationally. Studies conducted with Pima Indians of the American Southwest (Lillioja, et al., 1993) and first nation youth (Dean, Mundy, & Moffatt, 1992) show that native (American) Indians have a biological tendency to be more insulin resistant than their North American Caucasian counterparts. It is tempting to propose a hereditary ethnic relationship with insulin resistance, however the accumulation of central fat appears to be a better predictor (Haffner, 2006).

### 1.6.2 Socioeconomic status

In New Zealand, ethnicity and SES have a close relationship. Using 1996 census data which was categorized by New Zealand deprivation index (10 = most deprived), Riddell and North (Riddell & North, 2003) found that Māori were disproportionately represented (more than 50% of Māori) in deciles eight, nine and ten. Deprivation index has been through two revisions of definition since that time resulting in NZDep2006 (Salmond, Crampton, & Atkinson, 2007) and now accounts for the following factors: income, home ownership, support, employment, educational qualifications, living space, access to a phone and car. Similarly, the New Zealand Ministry of Education has instituted population-based funding of state schools with the use of deprivation index-style decile rating with 10 being the least deprived. The school decile represents the average SES of all the students that live in the surrounding area and uses census data of household incomes, occupation, household crowding, educational qualifications, and income support (Ministry of Education, 2008).

Globally SES has received a lot of attention in research related to obesity (Cecil et al., 2005; Dietz, 1991; Eckersley, 2001; Kumanyika & Grier, 2006; Patrick & Nicklas, 2005; Power, Manor, & Matthews, 2003; Sobal & Stunkard, 1989). Several studies have found a negative association between SES and obesity (Blomquist & Bergstrom, 2007; Danielzik, Czerwinski-Mast, Langnase, Dilba, & Muller, 2004; Kinra, Nelder, & Lewendon, 2000) through an indirect effect of cultural or social norms, nutrition in infancy and childhood, and psychological factors (Power & Parsons, 2000). Further, environmental effects of macro- and community level factors (economic, legal and government policy) have created a major

nutrition transition from high energy expenditure:low energy intake to low energy expenditure:high energy intake (Popkin, Duffey, & Gordon-Larsen, 2005).

## 1.7 Background

The research presented in this thesis was drawn from and aligned with a New Zealand regional initiative called Project Energize. The project was funded by the Waikato District Health Board to be delivered in local primary schools. This Waikato through-school health initiative was based on social marketing of nutrition and physical activity as well as improving the primary school health environment (Graham et al., 2008). National nutrition and physical activity messages have been combined to promote positive health to a specific target audience, Waikato primary school students. The delivery was through trained "Energizers" who were allocated up to eight of the initial 62 intervention schools to help facilitate change priorities identified by the school. The students in the evaluation arm of the project are predominantly of New Zealand European origin (59 %), and Maori, Pacific Island and Asian contribute 34%, 4%, and 3% (Graham, et al., 2008) of the Project Energize population, respectively. In 2001 the New Zealand population under the age of 15 was 75% European, 24% Maori, 11% Pacific and 7% Asian. These numbers do not add up to 100% as 21% of the children identified with more than one ethnicity.

## 1.8 Thesis rationale

Primary school age children of lower socioeconomic status have been targeted as the population of interest in this thesis. In New Zealand, Māori are over-represented in low socioeconomic regions. The primary school age group is increasingly the focus of school-based nutrition and activity interventions in New Zealand and worldwide but there are varying degrees of success. This is in part because knowledge about body fat accumulation in children is limited to the energy balance theory. In addition, few studies consider multiple aspects of energy balance simultaneously. Parental influence, the built environment, physical activity, food intake, physical inactivity, sleep, physical fitness, and socioeconomic status have all been touted as potential drivers of body mass. Alongside increase in body mass, the development of bone in childhood and its implications for bone health are unknown and may provide new understanding. . Thus a multi-faceted study is required to amass holistic knowledge of the accumulation of excess body fat and bone growth in children in a real-life setting.

To provide a knowledge bridge from drivers of body mass to changes in body mass, measures of the physiological pathways need to be undertaken. Energy homeostasis measures of resting metabolic rate and substrate utilisation (by resting respiratory exchange ratio) have provided some insights into adult weight gain and may elucidate the weight gain pathways in children. Further, insulin resistance is a major feature of increased body fat, so the inclusion of glucose homeostasis measures of fasting insulin and glucose offer an alternative perspective of the physiologic pathway of weight gain. Energy and glucose homeostais measures in New Zealand children in combination with patterns of daily life and physical fitness measures offer a unique, comprehensive and valuable set of information that not been attempted previously in this age group. Data of this magnitude adds significantly to the knowledgebase and understanding of children and their health.

## 1.9 Purpose of the research

The primary objective of this investigation was to explore and try to explain how demographic and lifestyle factor relationships affect body mass status of children. Two physiologic pathways, substrate utilization (measured by resting respiratory exchange) and indices of insulin action were the focus. According to Bray (Bray & Champagne, 2005), body mass status is explained by energy balance, however there are indications that respiratory exchange data may provide additional information (Ravussin & Bogardus, 2000). Further, studies (Marra, Scalfi, Contaldo, & Pasanisi, 2004; Seidell, Muller, Sorkin, & Andres, 1992; Zurlo et al., 1990) have shown that RER may be predictive of future weight gain. Respiratory exchange studies that collect both resting metabolic rate (RMR) and resting respiratory exchange ratio (RER) in children are not common. Research that has investigated measures of insulin and insulin sensitivity in children in association with body mass are more common and there is strong evidence that in children high insulin concentrations are associated with an elevated body mass status. The complexity of demographic and lifestyle determinants of body mass status, physical fitness and links with glucose homeostasis are required to obtain a better understanding of the areas where interventions may be most effective.

Secondary objectives of this body of work were to:

1. evaluate the differences and relationships between the two modes of measurement of body mass status, BMI SDS and PBF SDS.
2. investigate the effect of physical fitness (muscle strength (MSF) and cardiorespiratory fitness (CRF) on respiratory exchange and insulin data.
3. uncover the determinants of bone integrity

### 1.10 Thesis structure

The following chapter reviews previous literature in the context of how body mass status may be measured in children. Body mass status of children is then related to childhood obesity, metabolism, insulin sensitivity and bone integrity. An analysis of longitudinal, two year measurements of the children in Project Energize and aspects of their daily life in relation to their body mass status are reported in chapter three. To extend the work carried out in chapter three, physical fitness assessments were added to the aligned investigations reported in chapters four and five to provide another factor associated with the accrual of body mass. The crux of chapter four, was the utility of a non-invasive measurement of whole-body resting energy expenditure (RMR – resting metabolic rate by indirect calorimetry) and macronutrient oxidation (RER - respiratory exchange ratio) to deduce metabolic pathways to explain environmental influence (demographic and lifestyle) on children's body mass status. Therefore in chapter five, a different metabolic aspect to respiratory exchange, that is, insulin sensitivity was explored. To add a different dimension and to further the investigation into the body mass-insulin-fitness relationship, a measurement of the integrity of bone (calcaneal quantitative ultrasound) was a secondary outcome of the study. Finally, the last chapter provides a summation of the previous chapters and brings together the data to present the evidence as an argument to help explain the development of body mass in primary school age children.

# 2 Patterns of daily life: associations with body mass in primary school-age children – a review of literature

This review of the literature is a summary of evidence that food and activity patterns, are determinants of body mass in primary school age children. Activity patterns include those belonging to sport, exercise and play activities, inactivity and sleep. In addition, a review of physical fitness and bone integrity are also included. Methods of measurement have also been confined to those that are possible to perform in the naturalistic setting of a large through-school intervention programme. Where possible, to maintain a contemporary perspective, only those articles that have been published from 1990 onwards were included.

## 2.1 Body mass and its health consequences in children

The term "body mass" is synonymous with "body weight" however there is a difference between the two terms (page ix). As well as total BM measured in kilograms several "standardisations" of BM are used , to describe the human form, for example Fels index, Quetelet index or body mass index (BMI), fat free mass (FFM) and fat mass (FM). Most often BMI, which is a ratio of weight (in kilograms) divided by height (in metres) squared is used to describe mass to height with the assumption that BMI is not related to height.

Non-invasive but more technical equipment such as for bioimpedance analysis (BIA), allows BM to be further compartmentalised into water, fat free mass (FFM) and fat mass (FM). BIA is a portable noninvasive unit that uses the measure of impedance of the body to an alternating current to derive the proportion of fat free to fat mass from a measure of body water.

In children, the understanding of body composition change is complicated by their growth patterns. Children change body shape as they develop with age and there is sexual dimorphism. Use of reference populations provide a useful guide to estimating the body mass status of children but the identification of children who, because of body size for age, are at risk of developing risk for chronic disease, obesity must be defined. There are a variety of methods and definitions of overweight and obesity.

### 2.1.1 Defining Obesity

The World Health Organisation (WHO) defines obesity "as the disease in which excess body fat has accumulated to such an extent that health may be adversely affected" (World Health Organisation, 2000). Adult obesity reference ranges are published with objective and absolute

definitions of obesity using both body mass index and percent body fat. BMI has been the measure of choice by the WHO as it is convenient and has a positive relationship with risk of chronic diseases such as type 2 diabetes and cardiovascular disease.

The issue of defining obesity in children is an especially difficult task as linear and axial growth rates vary with age and between the sexes. In addition, some researchers claim that BMI may not provide information regarding risk of chronic disease for children. Various institutions use different reference populations and classification methods to calculate prevalence which adds to the difficulty of global comparison. The two international reference norms are provided by the International Obesity Task Force (IOTF) (Cole, et al., 2000) and the Centers for Disease Control and Prevention (CDC). Both employ BMI as their reference unit of measure.

The IOTF method uses a multiethnic population and standard deviation scores (SDS) to classify obesity (at +2 standard deviations) on curves extrapolated to predicted BMI at age 18 years. The CDC version is United States national data which classify overweight at the 95$^{th}$ percentile (+1.96 standard deviations), and at risk of overweight by 85$^{th}$ percentile. Thus both the IOTF and CDC methods are age and sex adjusted using SDS. Neither reference population included Australasian populations, for example, Australian aborigine, New Zealand Māori or Pacific people. An addition to these two reference norms is a percentage body fat (PBF) classification method (McCarthy, Cole, Fry, Jebb, & Prentice, 2006) drawn from a low socioeconomic status English population. This method also allows SDS to be determined.

Although these classification methods differ in origin of their reference population and their calculation method their use allows the increasing global prevalence of childhood obesity to be determined. The United States has the highest recorded proportion of IOTF overweight and obese children (>35%) (Lobstein & Jackson-Leach, 2007) and New Zealand in 2006/2007 followed with 29.1% (Ministry of Health, 2008b). The Ministry reported that the prevalence of overweight and obese children in New Zealand has appeared to have stabilised over the past 5 years because in the 2002 National Children's Nutrition Survey (Ministry of Health, 2003) 31.1% of children were recorded as overweight or obese children. However the absolute number of overweight and obese children continues to climb, thus the drivers of excess mass are maintaining their adverse effect and the consequences are an ever present problem.

Obesity, as excess body mass, is recognised as a primary risk factor for chronic disease such as cardiovascular disease (CVD), Type 2 diabetes (T2DM), osteoarthritis and some cancers (World Health Organisation, 2000). Although this tenet may be true in adults, the relationship of excess body mass with children's health is a more tenuous one as the relationships are not temporally or functionally close. As explained by Tobias, biologic causes are easier to measure due to their close proximity to the problem, whereas behavioural and environmental causes, are further removed and more difficult to measure (Nelson, 2005). The behavioural drivers that will be included in this review are the patterns of daily life - physical activity and inactivity, food and sleep. The demographic causes of socioeconomic status and ethnicity are also addressed.

### 2.1.2 Drivers of obesity

Upstream drivers of disease (including obesity) that require wholesale changes to the environmental and social culture have been cited in the literature (Agras & Mascola, 2005; Anderson & Butcher, 2006; Aranceta et al., 2007 ; Deckelbaum & Williams, 2001; Drewnowski & Darmon, 2005; Economos et al., 2007). A comprehensive schematic of the interactions between social and economic determinants of obesity from global to individual impact was presented by Ritenbaugh and colleagues (1999) and consequently published by Kumanyika et al. (2002).Socioeconomic status is a major driver of upstream causation.

*Socioeconomic status*

Socioeconomic status (SES) of children may be ranked into ordinal categories by various combinations of indicators. Shrewsbury and Wardle (Shrewsbury & Wardle, 2008) in their review of epidemiological studies from western developed countries found that parental education, parental occupation, family income, composite SES and neighbourhood SES were all used in different SES classification systems whereas in New Zealand the index of deprivation (NZDep06) (Salmond, et al., 2007) is used. The NZDep06 used measures of lack of income, employment, communication, transport, support, qualifications, owned home and living space to derive a score for a small, geographic area (meshblock). The least deprived areas are in quintile 1 and the most deprived in quintile 5. This geographical score is similar to the decile ranking system for New Zealand schools (Ministry of Education, 2008) where the most deprived decile is ranked at one. School decile is a national percentile ranking that is calculated from census data of local households within selected meshblocks that house children of school age. Although not a precise individual classification of SES both NZDep06

and school decile provide a general, non-invasive and current indication of children's socioeconomic environment.

Data published from the Children's Nutrition Survey (Utter, Scragg, Schaaf, Fitzgerald, & Wilson, 2007) found direct evidence that SES was linked to overweight and obesity (Ministry of Health, 2003) and that the highest number of obese children were in the lowest SES category (NZDep5: 17.7% vs NZDep1: 4.7%). Duncan et al. (2008) found that 54% of low SES (by decile) children aged 5-11 years, from 27 Auckland primary schools in New Zealand, were overfat compared to the high SES children (21%). Low SES children were two times more likely to be overfat when adjusted for all other risk factors in the study  This strong relationship between deprivation and health is recognised by the Ministry of Health (Tobias & Howden-Chapman, 1999) (New Zealand) and has relevance for some ethnic communities such as Māori and Pacific peoples who were at higher risk than others since they were over-represented in the lower socioeconomic areas (Gray, 2003).

Children overseas are also subject to the same SES pressures. In 2631 German 5-7 year olds Danielzik et al. (2004) found that 45% of obese children were of low SES compared to middle (26.6%) and high (28.2%) SES (Danielzik, et al., 2004). Similarly Kinra et al. (2000) demonstrated in 20,973 English girls and boys that obesity prevalence increased with age and children in more deprived areas were up to twice as likely to become obese. In addition to obesity, SES appeared to limit growth in height; in a study of 2454 boys and girls in Dundee, low SES children were 1.26 cm shorter than their higher SES counterparts (Cecil, et al., 2005).

A cofactor in the obesity-SES relationship may be the interactions of socio-cultural behaviours such as television viewing hours which is a factor that is associated with inactivity, low nutrient-high calorie eating habits and exposure to junk food advertising as demonstrated in 60 children, grouped into low and high TV consumption (Group 1: ≤1h/day; Group 2: >1h/day) (Grund, Krause, Siewers, Rieckert, & Muller, 2001). The children with parents of lower education spent longer periods watching television. Patrick and Nicklas (Patrick & Nicklas, 2005) added evidence that lower parental education and reduced time available for food preparation was associated with impaired ability to choose healthy foods which in turn was associated with increased fat intake and reduced household income. This pattern of association may explain the positive relationship between a child's BMI and their mothers BMI (explained variance: 4%) and household income (explained variance: (1.1%) found by Lin et al. (2004). Parents have no direct control over the interaction of SES and

intergenerational obesity which drive the development and growth of children and therefore the prevalence of obesity (Kumanyika & Grier, 2006).

In New Zealand, the close association of ethnicity with SES and geographical location means that the association between SES and obesity is confounded.

*Ethnicity*

Ethnicity, a sociocultural and self-identified construct, and SES are closely associated in New Zealand. Māori and Pacific are overly represented in the lower SES communities (Gray, 2003). Certainly both factors are associated with increased risk of obesity and illness (Duncan, Schofield, Duncan, Kolt, & Rush, 2004; McLaren, 2007). Māori and Pacific peoples have the highest age-adjusted rates of cardiovascular disease (Tobias, Sexton, Mann, & Sharpe, 2006), T2DM (Ministry of Health, 2002), and cancer (Robson, Purdie, & Cormack, 2006) in New Zealand. Also, more Māori and Pacific peoples are obese (Ministry of Health, 2008b) and have earlier morbidity and mortality (Bascand, 2008) than non-Māori.

The hypothesis that Māori and Pacific peoples are genetically 'thrifty' (Neel, 1962) has been used to explain the predisposition for the accumulation of excess mass in the contemporary obesogenic environment (Pearce, Foliaki, Sporle, & Cunningham, 2004). Certainly evidence has been provided that Māori may have higher levels of insulin resistance (Mann et al., 2006) than non-Maori, however it has been argued that level of adiposity and distribution of fat are the overriding factors (Simmons, Thompson, & Volklander, 2001). Maori and Pacific peoples were disproportionately represented in the low socioeconomic group and thus ethnic differences interact with SES in explaining the higher prevalence of obesity and overweight.

Cultural factors, that are ethnic and socioeconomic in nature, may directly influence the development of obesity at home. The preferential dietary behaviour of Māori and Pacific peoples to overfeed guests and families with low cost, high calorie food such as fatty meats, highly-processed grains and sugary soft drinks (Utter, Scragg, Schaaf, & Fitzgerald, 2006) may be interpreted as a sign of hospitality and parental care within the culture. A possible cultural preference for the larger sized person (Metcalf, Scragg, Willoughby, Finau, & Tipene-Leach, 2000) as a demonstration of 'natural' growth and the belief that overfeeding is an example of the 'care' by parents may also contribute to the accumulation of excess mass in Māori and Pacific peoples.

Furthermore, when addressing the issue of obesity in Māori and Pacific cultural needs must be considered. Tikanga (culture) and kawa (protocol) are being developed for promotion of

Māori health and fitness (Durie, 2007) by Māori for Māori, which is important to strengthen community action and may be an easier adjustment for the general Māori public to make when assimilating national messages for healthy dietary and activity practices into diverse cultures and communities.

### 2.1.3 Childhood to Adulthood

The evidence for tracking of obesity from childhood to adulthood is present but variable. It is thought that early environmental effects in-utero may permanently programme (epigenetic) adaptive responses such as obesity and possibly lead to the increased risk of chronic disease in adulthood (2007). Early research work indicated that when an infant exceeded the 75$^{th}$ weight percentile a significant increase (chi-square=17.2, $p$<0.001) in adult (20 to 30 years of age) obesity could be expected.(Charney, Goodman, McBride, Lyon, & Pratt, 1976). A systematic review (Baird et al., 2005) of 24 studies found that both "overweight as an infant and rapid growth during infancy are predictors of adult obesity". However Poskitt and Cole (Poskitt & Cole, 1977) report that few obese infants went on to be obese as five year olds. Similarly, Fuentes et al. (2003), also suggest that borth weight is not predictive of BMI during childhood. As a child ages, better predictions of adult obese state can be made. Serdula et al. (1993) found that at older ages, an obese child is at higher risk (42-63%) of continuing that obese state into adulthood..

Longitudinal studies (Casey, Dwyer, Coleman, & Valadian, 1992; Deshmukh-Taskar et al., 2006; Guo et al., 2000; Magarey, Daniels, Boulton, & Cockington, 2003; Trudeau, Shephard, Arsenault, & Laurencelle, 2001) including those from New Zealand (Williams, 2001), demonstrate that obesity tracks from childhood into adulthood but the strength of prediction varies greatly.

For example Williams (Williams, 2001) cohort of 558 participants, with a BMI over the 75$^{th}$ percentile at seven years of age, had 6.8 times the relative risk of being overweight at 21 years of age. Patterns of change in BMI, from 2 to 25 years of age, including minimum BMI at adiposity rebound phase and BMI at maximum velocity during pubescence predicted BMI at 35 to 45 years old and were stronger for females (odds ratio, 95% CI=2.07, 1.24-3.46; 3.64, 2.04-6.47 respectively) than males (odds ratio=1.07, CI not quoted; 2.03, 1.37-2.99) for 338 Fels study participants (Guo, et al., 2000).

Tracking of classification of body size by BMI is also supported by longitudinal studies. Tracking of normal weight to normal weight and overweight to overweight BMI from

childhood (9 to 11 years old) to adulthood (19 to 35 years old) was strong in 361 European American women (72.8%) similar between 94 African American men and 173 women (59.6% vs. 59.5% respectively) and lowest in 213 European American men (48.8%) (Deshmukh-Taskar, et al., 2006). Trudeau et al. (2001) also suggests that the BMI of females tracks better from childhood (10 to 12 years of age) to adulthood than males (r = 0.64-0.70, P < 0.001 vs. r = 0.43-0.49). However, Casey et al. (1992) found that female BMI at 50 years of age could not be predicted from childhood BMI ($R^2$=0.002, $p$>0.05) but male BMI could ($R^2$=0.17, $p$=0.008). When males and females (n=155) were tracked throughout childhood to 20 years of age and found that BMI at age 6 was a good correlate (males $r^2$=0.61, $p$<0.001; females $r^2$=0.71, $p$<0.001) with BMI at 20 years of age (Magarey, et al., 2003).

In summary, BMI does track from childhood to adulthood but the application of these studies to contemporary children is not valid as globally dietary and physical activity behaviours and background prevalence have changed (Guo, et al., 2000).

### 2.1.4 Consequences of obesity

Less is known about the health consequences of obesity in children than in adults. For the purposes of this thesis the scope of consequences will be limited to cardiovascular disease risk, insulin resistance syndrome (IRS) and bone health. Voss et al. (2006) suggest that the IOTF cutoffs are not suitable as markers for insulin resistance, thus a better understanding of insulin resistance in children is required.

*Cardiovascular disease and Insulin Resistance Syndrome*

Cardiovascular disease was the leading cause of death in New Zealand in 2000 (Hay, 2004) and although the prevalence of ischaemic heart disease has decreased in the past 40 years, there are warnings about a predicted increase in cardiovascular disease-related disability and death driven by the increase in obesity (Tobias, et al., 2006). Overweight children with metabolic complications of dyslipidaemia, hypertension and insulin resistance are at higher risk of adult health issues (Nathan & Moran, 2008). Self-reported cardiac events were tracked over 25 years by the Princeton follow-up study using the Lipid Research Clinics cohort from 1973-1976 (Morrison, Friedman, & Gray-McGuire, 2007). The 31 children who had IRS (defined by ATPIII criteria with BMI obesity as a surrogate for waist) at age 12.9 ±3.4 years , were 14.6 times more likely to incur cardiovascular disease (defined as myocardial infarction, coronary artery bypass graft, angioplasty, or stroke) 25 years later. Twenty percent from the paediatric IRS group (n=6) experienced cardiac events whereas only 2% (n=11) of the

paediatric non-IRS group (n=740) had cardiac events while prevalence of IRS syndrome had increased to 27.2% over 25 years.

Overweight in adolescence (13 to 18 years) was demonstrated to have an increased relative risk of all cause mortality amongst men (1.8) and from coronary heart disease (2.3) but not women (Must, Jacques, Dallal, Bajema, & Dietz, 1992). Similarly, cardiovascular risk factors found in 384 eight to 18 year olds (51% boys), including age and sex adjusted weight (odds ratio, 95% CI=3.0, 1.3-6.7) and measures of lipid biomarkers, led to a higher risk of developing coronary artery calcification between 29 and 37 years of age. Risk was higher in males with 31% of men and 10% of women showing calcification (Mahoney et al., 1996).

The role of increased total and central body fat as a phenotypic indicator of childhood cardiovascular risk factors (BMI, dyslipidaemia, and hypertension) to predict the development of vascular endothelial dysfunction in young people was further supported in a study of 93 autopsies (aged 2 to 39 years) (Berenson, Srinivasan, Bao, Newman, Tracy, & Wattigney, 1998). These cardiovascular risk factors were strongly associated with lesions in the aorta and coronary arteries ($r=0.70$, $p<0.001$). As the risk factors accumulated so did the extent of atherosclerosis. In contrast, Kirk et al. (2005), found that when they decreased the BMI of 394 obese (BMI >95$^{th}$ percentile) 5 to 19 year olds over 5.6 months, risk factors of cardiovascular disease were significantly ($p<0.001$) reduced (BMI SDS=-0.15 ±0.15; blood pressure – systolic=-3.9 ±9.6 mmHg, diastolic=-4.2 ±10.0 mmHg; triglycerides=-14.7 ±53.5 dl/mL; LDL cholesterol=-7.8 ±18.9 dl/mL; fasting insulin=-4.3 ±14.2 $\mu$U/mL). This study shows that inroads to improved cardiovascular health as adults can be made in childhood by the reduction of body mass index early through physical activity and dietary behavioural intervention.

*Glucose homeostasis*

Glucose homeostasis in this thesis is an umbrella term to include measures of glucose, insulin and derived measures such as homeostatic model assessment or insulin resistance. Insulin resistance is a recognised pathological state where insulin receptors in liver, muscle and fat tissue do not respond optimally to the stimulus of blood-borne insulin and consequently concentrations of glucose in the blood increase. An increase in blood glucose concentration stimulates the β cells of the pancreas to produce more insulin. Over a long period of time, insulin production falters to the point of not producing enough insulin to cope with the elevated glucose levels. A compensatory mechanism is the release of fatty acids into the

blood stream, hence raised serum lipid values in insulin resistance syndrome (Nathan & Moran, 2008).

Insulin resistance is described as a primary pre-cursor to the development of type 2 diabetes (Rappaport & Usher, 2006). In a laboratory setting, insulin resistance is measured by a 'gold standard' hyperinsulinaemic euglycaemic clamp, whereby insulin is intravenously infused at a known rate (DeFronzo, et al., 1979). Enough glucose is also infused to maintain blood glucose at a safe level to maintain euglycaemia and the rate of blood glucose infused over a 30-minute period determines the insulin sensitivity of the individual. Insulin resistance is regarded as insulin insensitivity, the reciprocal of the state of being insulin sensitive (Wallace, et al., 2004b). The hyperinsulinaemic euglycaemic clamp is costly and time consuming and less complex procedures are required in the clinical setting (Keskin, Kurtoglu, Kendirci, Atabek, & Yazici, 2005). These less complex procedures usually involve a single fasting blood sample to measure the relationship between insulin and glucose. Common examples of diagnostic indices are fasting glucose/insulin ratio (FGIR) (Legro, et al., 1998; Silfen et al., 2001), fasting insulin resistance index (FIRI) (Duncan, Singh, Wise, Carter, & Alaghband-Zadeh, 1995), quantitative insulin sensitivity check index (QUICKI) (Katz, et al., 2000), insulin sensitivity index (ISI) (Soonthornpun et al., 2003), and homeostatic model assessment (HOMA) (Matthews, et al., 1985a).

An addition to these alternative models of insulin sensitivity, resistance and beta cell function has been the evolution of HOMA-IR to HOMA2 (Wallace, et al., 2004b). Both models require fasting measures of glucose and insulin . Remodelling of the original HOMA method to suit modern assay methods of insulin has lead to the original method being superceded by the updated version (HOMA2) which has been validated against the standard euglycaemic clamp method (Matthews et al., 1985b; Stumvoll et al., 2000; Wallace, Levy, & Matthews, 2004a). HOMA2 consists of two results, insulin sensitivity (HOMA2-S%) and beta cell function (HOMA2-B%) that together best explain the condition of insulin resistance. Beta-cell function is the estimation of insulin secretion from the pancreatic islets of Langerhans. It is recommended by the authors that 100% in either variable is the normative value for adults and that the results must be reported in tandem as the different permutations of the results explain whole-body insulin dynamics. For example, although most people with normal glucose values may achieve the norm of 100% HOMA2-S% and 100% HOMA2-B%, an individual that was slim and fit may attain a highly insulin sensitive value of 200% HOMA2-S% and a corresponding lower beta-cell function (50% HOMA2-B%) as a result of higher

sensitivity. Reporting of the HOMA2-B% alone may erroneously portray failing beta-cell function when the beta cells have naturally reduced insulin production because of increased insulin sensitivity. HOMA2 has not been validated in children thus it is difficult to know whether the estimate of 100% is applicable for children, but it does provide a wider picture of glucose homeostasis of individuals when using convenient, less invasive measures. However many researchers still use the single 1985 HOMA index (HOMA-IR) (fasting insulin (μU/mL) x fasting glucose (mmol/L)] / 22.5) but this index does not reliably predict insulin sensitivity in those already hyperglycaemic (ref)

Using these convenient measures, prevalence of insulin resistance has been investigated in very few paediatric studies, the largest of which (Lee, Okumura, Davis, Herman, & Gurney, 2006) investigated 1802 adolescents (12-19 years of age). HOMA-IR was the outcome measure for this study and a 2 standard deviation cutoff point (HOMA-IR =4.39) of the normal weight group (n=1169; BMI <85$^{th}$ percentile) was applied to determine the prevalence of insulin resistance. Of the obese group (n=340; BMI ≥95$^{th}$ percentile) 52.1% were found to be insulin resistant after adjusting for age, sex, and race. Sung et al. (2003) found that the prevalence of insulin resistance amongst Hong Kong-based 9-12 year olds (n=271) was 77% in the obese group (n=129) using the HOMA method but did not report their cutoff marker. Prevalence of insulin resistance (HOMA) was much lower in obese Greek children (31.0%) (Manios et al., 2008). Manios et al. found that 9.2% of 522 ten to twelve year old Greek children were insulin resistant. They implemented a cutoff point based on the 97.5$^{th}$ percentile of normal weight children which was >2.15 (HOMA-IR).

The proportion of insulin resistant children increased with increasing body mass (BMI) in all three studies and more so for the older cohort of Lee et al. (2006). This may be because of pubertal influences on obesity and insulin resistance. Guzzaloni et al. (2002) found that progression through puberty demonstrated that children become more resistant to insulin and less insulin sensitive. Overwhelmingly, waist circumference and therefore visceral adiposity was shown to be a primary component in predicting insulin resistance. This was confirmed by Lee et al. (2006), where insulin resistance as measured by the euglycaemic clamp method showed for white subject's that insulin resistance was associated with vascular damage but was not able to be shown in black subjects. Possibly this was because there is less visceral adiposity and lower circulating triglyceride concentrations in black children. Aggoun (2007) contends that vascular damage cannot be explained by insulin-mediated mechanisms.

However, there are no disagreements with the contention that obesity, insulin resistance, and cardiovascular disease are related and start early in life.

*Bone Health*

The health of bone is partly dependent on body mass status. Wearing et al (Wearing, Hennig, Byrne, Steele, & Hills, 2006) states that obesity is a risk factor in the onset and development of musculoskeletal disorders. They suggest that excess weight loading is responsible for musculoskeletal injuries. A combined nutrition and physical activity intervention as successful in increasing the bone strength (21.5 ±21.6 vs. - 87.0 ±37 m/s, $p = 0.023$) of obese children with a slowing in the rate of weight gain (0.01 ±0.7 vs. 2.3 ±0.6 kg, $p = 0.033$) (Nemet, Berger-Shemesh, Wolach, & Eliakim, 2006).

The relationship between body composition and disease risk in children is becoming relatively clear but the mechanism is more complex and lies in energy homeostasis and balanced nutrition. A method to explore energy homeostasis is to measure metabolic rate and the mix of macronutrients (substrates of fat, carbohydrate and protein) oxidised. The specific combination of energy expenditure (resting metabolic rate) and proportional use of fuel (substrate utilisation) provides a unique perspective into physiologic pathways of energy metabolism. For example with insulin resistance proportionally more fat than glucose is burnt and glucose takes longer to be cleared from the blood.

### 2.1.5 Resting metabolic rate and substrate utilisation in children

Resting metabolic rate and substrate utilisation, as represented by resting RER (also known as RQ or respiratory quotient), are useful tools in the investigation into body mass. RER is measured at the mouth whereas RQ is normally measured at a localized area such as the leg or arm. RER and RQ are both calculated by the carbon dioxide output divided by oxygen input and are equivalent in a steady state such as rest. RER and RQ begin to diverge when the body enters a dynamic state like the onset of exercise. Surprisingly little work has been performed in children under 12 years of age. Of the little research that has been conducted we know that obese children usually have a similar RMR (body mass adjusted) to non obese children (DeLany, Bray, Harsha, & Volaufova, 2002; Fontvieille & Ravussin, 1993; Stensel, Lin, & Nevill, 2001).

Kleges et al. (1993) reported that physical inactivity such as television viewing lowers RMR, but Dietz et al. (1994) contends that physical inactivity does not appear to affect RMR.

Constitutionally lean children have a low metabolic rate and RQ was no different between lean, normal weight and obese groups (Tounian, et al., 2003). This is probably due to fat-free mass being the main determinant of RMR. (Molnar & Schutz, 1997). Though a high fat oxidation (low) was found to be associated with higher levels of body fat (Maffeis, et al., 1996).

No RMR differences were observed between New Zealand ethnicities (Rush, Plank, Davies, Watson, & Wall, 2003) and between American Hispanic, African American and American European girls (Treuth, Butte, & Wong, 2000). However (Yanovski, Reynolds, Boyle, & Yanovski, 1997) did find that normal weight African American girls had a lower metabolic rate (-92 ±32 kcal.day) than European American girls. Patterns of daily life in New Zealand children

## 2.2 Patterns of daily life in New Zealand children

While obesity in children is driven by social, environmental, genetic, biological and behavioural aspects (World Health Organisation, 2000), the interaction and level of influence among these determinants on the energy balance equation, food intake and energy expenditure; have a profound and complex effect on body mass and composition (Budd & Hayman, 2006). In this section, the primary determinants that will be considered are food habits, physical activity and fitness (Ortega, Ruiz, Castillo, & Sjostrom, 2008), and socioeconomic status (Duncan, et al., 2008). Other acknowledged determinants not considered in this review are parental BMI, in-utero effects of maternal nutrition, and the built-environment; however these other determinants may also have indirect and direct relationships with SES (Budd & Hayman, 2006) adding to the complexity of predicting risk, targeting and designing interventions to reduce relative body size in childhood.

### 2.2.1 Food patterns

How much and what a child eats is one side of the energy balance equation – caloric input. Children's eating patterns can further be categorised into locations, frequency, quantity and type of food at home and away from home are primary considerations when assessing dietary patterns of children (Rosenkranz & Dzewaltowski, 2008). Data from the 2002 National Children's Nutrition Survey (Ministry of Health, 2003) found that the New Zealand children from a lower socioeconomic background were more likely to obtain their lunch at school and that their households were the least food 'secure', that is, they were less likely to be able to afford to eat properly. This was supported by children from high deprivation households

reporting consumption of more fat and low nutrient-value carbohydrate foods than children from those less deprived. Also children from high deprivation households appear to have an increased calorie intake, more energy dense foods and a potential reduction in the intake of micro-nutrients and antioxidants which are essential for optimal growth and development.

A recent review of international literature by Newby (2007) found very few studies have examined the effect of dietary patterns on the development of childhood obesity and evidence for food-based interventions was lacking. Low socio-economic American children consumed a diet high in sugar and fat and low in vegetables, fruits and lean meat; inconsistent with the American food based pyramid dietary recommendations (Knol, Haughton, & Fitzhugh, 2005). Whereas the dietary practices identified for similar low-SES immigrant population in Sweden were that consuming high level of sweet drinks and missing breakfast (Magnusson, Hulthen, & Kjellgren, 2005) were associated with increased body size. Janssen et al.'s (2005) review of studies of overweight and obese childrens from 34 countries showed no obvious differences in dietary patterns by body size. This lack of association of reported diet and body size was also supported by Kelley et al. (2004) in their analysis of 297 U.S. children and Alexy et al.'s (2004) study who indicated that possibly under-reporting and the dietary assessment tools used may be an issue in analysis. In spite of this worldwide lack of evidence to demonstrate caloric overconsumption is key to weight gain, Berkey et al. (2000) claims that excessive caloric intake is one of two determinants, the other being lack of physical activity, that produce overweight children. Further, Swinburn et al.'s (2006a) mathematical modeling of total energy expenditure and mean body mass in three studies of 963 youth (4 to 18 years) showed that there was a positive relationship with EnFlux (a representation of energy balance) with body mass. The positive relationship supported the hypothesis that total energy intake (TEI) was the principle factor for weight gain in youth.

Although caloric intake and nutrition are important factors in the aetiology of obesity and health of children, the ability to precisely record dietary intake remains a problem through under-reporting, selective reporting and inaccuracy of dietary records. Nutrient quality of foods is also very variable and the nutrient composition of many foods and recipes are either inconsistent or unknown. There is some indication that excessive calorie intake may be a factor however no single macronutrient can be highlighted as a culprit. It may be possible that food security, affected by socio-economic factors, in the family food environment will be underlying a dietary relationship with obesity (Patrick & Nicklas, 2005).

### 2.2.2 Physical activity

Physical activity is a multi-dimensional behaviour that involves any movement of the body through space and is most often seen in terms of energy expenditure (Malina & Katzmarzyk, 2006). Subsets of physical activity include the more organised domains of exercise, physical education and sport participation but there are many other physical activities including play and active transport that make up the physical activity component of total daily energy expenditure (TDEE). Non-exercise activity thermogenesis or NEAT is classified as all activity that is not purposeful exercise such as sports or prescribed exercise programmes (Levine, 2003). NEAT is perceived to be important for it's greater contribution (than formal exercise and sports) to total energy expenditure and perceived health benefits, such as lipoprotein regulation. However, physical activity (activity energy expenditure) is relatively small proportion of TDEE. Resting metabolic rate and the thermic effect of food may make up to 90% of TDEE in a sedentary child. In a very active child resting metabolic rate and the thermic effect of food make up 60% of total energy, leaving 40% as the maximum activity energy expenditure.

Many paediatric intervention studies designed to reduce weight gain employ physical activity as their primary focus (Boreham et al., 2004; Donnelly et al., 1996; Eliakim, Nemet, Balakirski, & Epstein, 2007; Florea, 2005; Gutin, Yin, Johnson, & Barbeau, 2008; Sallis et al., 1997; Verstraete, Cardon, De Clercq, & De Bourdeaudhuij, 2007; Zahner et al., 2006). On the output side of energy balance, physical activity is perceived to be the primary, modifiable factor contributing to energy output, thus increased physical activity is claimed to be an important factor in the prevention of weight gain (Flodmark, Marcus, & Britton, 2006) and for cardiovascular health (Eisenmann, 2004; Francis, 1996; Twisk, Kemper, & van Mechelen, 2002b). It may be important to attain high levels of physical activity in young children as physical activity levels generally decline in adolescence and prevent the onset of weight gain during that time (Amisola & Jacobson, 2003; Eisenmann, 2004).

*Consequences of reduced physical activity*

Despite the perceived beneficial effect of increased physical activity, Twisk et al. (2002a) calculated that physical activity in childhood does not predict cardiovascular health in adulthood. Leonard (2001) counters by stating that methodological limitations may develop an "under-estimation" of the efficacy of physical activity so standardisation of measurement of study outcomes may be required (Thomas, 2006). Similar findings on the weight change

outcomes of physical activity interventions were reviewed by Thomas (2006) and Doak et al. (2006) and the conclusion was that the "modest and mixed" benefits of physical activity may be due to methodological and analytical differences among studies.

In complementary physical activity investigations, Trost et al. (2001) and Grund et al. (2001) both raise the important issue of physical inactivity or sedentary behaviours such as TV viewing time, independent of weight, as risk factors for obesity and cardiovascular disease (Froberg & Andersen, 2005). Proctor and colleagues (2003) also observed a similar effect in 4 year old children. Confirming international research, Duncan et al. (2008) found that New Zealand children are twice as likely to be overfat when inactive than active.

### 2.2.3 Physical inactivity

Physical inactivity is distinct from physical activity as it is the absence of physical movement and participating in sedentary activities such as sitting or lying down. The most common pursuits that are inactive involve screen time such as television (TV) watching, computer use or game console (fixed or portable). Hamilton (2007) reports that adults are more susceptible to cardiovascular disease when they have a sedentary lifestyle and children may be the same. Additionally, Tremblay and Willms (Tremblay & Willms, 2003) find that there is a positive relationship between physical inactivity and body mass in Canadian children and to counter this relationship Koezuka et al. (2006) recommend reducing TV viewing time. Interestingly, Droomers et al. (1998) identify psychosocial (for example emotional social support and coping styles) and material factors (for example employment status and financial problems) related to physical inactivity in adults and this may be overcome by introducing interventions that account for the psychosocial and material factors in children.

### 2.2.4 Bone development and health in children

Diet and physical activity are both related to the development and function of the musculoskeletal system in children. Paediatric bone-health is an area that requires more attention as like obesity there is increased risk of acute (fractures) and chronic disease when bone health is not optimal (Zhu, Greenfield, Du, & Fraser, 2001). In bone-health research physical activity interventions have been found to be beneficial for those with a low BMI but not those with high BMI (Ondrak & Morgan, 2007). However, Nemet et al. (2006) were able to show that obese children had gains in bone strength (quantitative ultrasound speed of sound 21.5 ±21.6 vs -87.0 ±37.0 m/s, $p=0.023$) from physical activity even whilst losing weight, Additionally, an 8-month physical education class that included jumping, skipping,

and hopping as part of the curriculum demonstrated a 4.4% vs 3.2% increase in bone mineral density from baseline (McKay et al., 2000). Bone mass and structure are determined during growth and physical activity has an important role to optimise bone health and other body functions during childhood.

### 2.2.5 Sleep

The inverse relationship of habitual daily sleep and body mass and the influence of the activities of daily life are not well understood. Several authors (Chen, Beydoun, & Wang, 2008; Knutson & Van Cauter, 2008; Nixon et al., 2008) suggest a physiologic action mediated by reduced metabolic rate, increased insulin resistance, or poor appetite control. Whereas Horne (2008) disputes that there could be a physiologic difference and puts any significant result down to bad sleep habits affecting patterns of daily life - diet and activity.

### 2.2.6 Recording patterns of daily life

Tools developed to record patterns of daily life vary markedly in their appearance, delivery and reliability. A questionnaire that reflected the New Zealand culture, young children of the Waikato and it's great diversity was not currently available at the development of the Project Energize. Thus food and physical activity frequency questions were pooled from the respective experts in the area and published in the original baseline questionnaire.

## 2.3 Physical fitness

Closely aligned with physical activity and the patterns of daily life is physical fitness. Physical fitness is the capacity to perform physical activity and reduced physical fitness is associated with obesity and cardiovascular disease (Ortega, et al., 2008). Dawson et al. (2001) report that the physical fitness of New Zealand children is on a decline which is not different from international trends (Olds, Ridley, & Tomkinson, 2007). There are many aspects to physical fitness (Simons-Morton, Parcel, O'Hara, Blair, & Pate, 1988) but for the purposes of this review, only two aspects, cardiorespiratory fitness (CRF) and muscle strength (MSF), will be considered in the context of BMI and body composition.

### 2.3.1 Cardiorespiratory fitness

Measurement of cardiorespiratory fitness in children requires different methods for different features. A measure of oxygen uptake on maximal exertion requires ergometers (treadmill and cycle) and real-time respiratory gas and volume measurement. $VO_2$ max is the direct, gold standard, measure of cardiorespiratory fitness in children, adolescents and adults and is

expressed in milliliters of oxygen per kilogram of body weight per minute. Often less technically and resource dependent indirect methods are utilised. Examples include maximal tests like the 20m shuttle run test (20m SRT) (Léger, Mercier, Gadoury, & Lambert, 1988) and the one mile run. Other indirect methods used for children are submaximal such as the physical work capacity 170 test (PWC170) cycle ergometer (Rowland, Rambusch, Staab, Unnithan, & Siconolfi, 1993) and bench step (Jette, Campbell, Mongeon, & Routhier, 1976), and these validated methods can used to predict a fitness value from the heart rates attained. Issues of location, availability of equipment, time, cost, expertise and numbers of children to be tested may limit the applicability of the test method.

Prevention of illness and improvement of health rather than treatment for obesity is the focus of this review therefore only studies of children who are both normal weight and overweight have been included in this section. A prospective study of Canadian elementary school children (n= 135; 6 to 10 years) over a year found that although fitness levels, measured in a 20m SRT, did change throughout the year, children at risk of overweight (BMI >85$^{th}$ percentile) consistently scored as having lower fitness than the normal weight group (Baseline = 1.8 vs 2.7 stage.level, p=<0.01). Ball et al. (2005) conducted a 12-month long study considering fitness between normal weight (n=115) and overweight children (n=20). Their repeated measures study (0, 3, 6 and 12 months) of children aged six to ten years old from seven Canadian elementary schools showed that normal weight children were not only fitter but increased in fitness faster than overweight children (F=14.3, $p$<0.001). However, Carrel et al. (2007) warns that benefit from physical fitness may be lost during the long summer break. Similarly, a 3-year after-school physical activity intervention held in 18 schools found that although there were body fat, bone density and physical fitness benefits to physical activity these tended to be attenuated by the summer break (Gutin, et al., 2008). The Georgia Fitkid after-school programme obtained a significant group by time interaction between lower body fat levels (p<0.05) and higher aerobic fitness (p<0.01) and improved bone mineral density than controls after three years of intervention focused on increasing MVPA, but intervention children reverted to near-control levels in all aspects after summer vacation (Gutin, et al., 2008.). Treuth et al. (Treuth, Butte, Adolph, & Puyau, 2004) observed that girls at 8 years of age appeared to maintain fitness over 2 years, however if they had obese parents they tended to be less fit.

Fitness, like mass, tracks over time but the interaction with body size is less clear and is often inferred from cross-sectional analyses (Kathleen F. Janz, Dawson, & Mahoney, 2000; Jos W. R. Twisk, Kemper, & van Mechelen, 2000).

### 2.3.2 Association of cardiorespiratory fitness with obesity

The entire spectrum of opinion exists from "fatness mediates the effects of fitness" (S. Lee, et al., 2006; Rizzo, Ruiz, Hurtig-Wennlof, Ortega, & Sjostrom, 2007) to "fitness protects against the adverse effects of excess body fat" exist (Chen, Fox, Haase, & Wang, 2006; DuBose, Eisenmann, & Donnelly, 2007; Halle, Korsten-Reck, Wolfarth, & Berg, 2004; Isasi et al., 2003). Furthermore while others agree that a relationship exists they opine that there is little to suggest which of these two physiological mechanisms (fat mediation of fitness effects or fitness protection from excess fat) are responsible (Eisenmann, Welk, Ihmels, & Dollman, 2007), and most offer no explanation of process (Ara, Moreno, Leiva, Gutin, & Casajus, 2007; Brunet, Chaput, & Tremblay, 2007; Kim et al., 2005; Nassis, Psarra, & Sidossis, 2005). Tomakidis (2006) simply proffered the comment that both a reduction in BMI and an increase physical fitness would be beneficial to child health.

Investigations in young children of possible mediation by hormones of the relationship between cardiovascular fitness and obesity have found that fitness training was associated with changes in the growth hormone – insulin-like growth factor axis and that this may help explain the relationship of cardiorespiratory fitness with insulin sensitivity (Eliakim, et al., 2001; Moran et al., 2002). Young girls that were fit and trained were insulin responsive and had a higher growth hormone function than untrained girls who were observed "to pass through a neuroendocrine state" before attaining the trained state (Eliakim, et al., 2001). For children information about molecular mechanisms including glut 4 interactions is not available at present.

High fitness is linked with higher insulin sensitivity (Carrel et al., 2005) which, by proxy, may mean that increased fitness would be associated with a reduction in insulin resistance syndrome (Moran, et al., 2002; Ruiz et al., 2007) often known as metabolic syndrome (Ortega, et al., 2008). Froberg and Andersen (2005) confirm that fitness has a causal role in metabolic risk and Eisenmann (2007) believes that this may be due to genes, adipocytokines and mitochondrial function. This belief is partly supported by studies that show raised inflammation markers in the presence of obesity, and fitness has been found reduce inflammation in obese children (Halle et al., 2004; Ruiz, Ortega, Warnberg, & Sjostrom,

2007). Other studies have found that there is a close relationship between increased abdominal adiposity and risk of metabolic syndrome with a protective function of fitness (Benson et al., 2006; Nassis, Psarra et al., 2005). In obese and unfit children metabolic syndrome and classic cardiovascular risk factors such as low HDL cholesterol, high total cholesterol, increased blood pressure and C-reactive protein have been demonstrated (Chen, et al., 2006; Isasi, et al., 2003; Thomas, Cooper, Williams, Baker, & Davies, 2007). Little is known in children about the fat-fit phenotype (Eisenmann 2007) in relation to the oxidative capacity of muscle, whole body insulin sensitivity and its relevance to future health. This area needs to be explored further.

While a few overweight and obese children perform better than their non-overweight counterparts in cardiorespiratory fitness testing, this is a small (<5%) proportion. (Stratton et al., 2007). Most cardiorespiratory tests are weight-bearing and therefore the excess mass that did not contribute to the activity would require additional energy expenditure. In non-weight bearing testing (i.e. cycle ergometry), overweight and obese children continued to be outperformed by their normal weight peers (DuBose et al., 2007; K. F. Janz, Dawson, & Mahoney, 2002; Ruiz, Rizzo et al., 2006) , although Norman et al. (2005) found that the fitness results were similar between weight categories in their study. It has been suggested that cardiorespiratory fitness would assist in reducing the prevalence of obesity (Janz, Dawson, & Mahoney, 2002) however others state the mechanism might be the reverse, that a reduction in obesity would improve performances in fitness testing (Stratton, et al., 2007). Olds et al. (2007) believe that although excess body fat has a detrimental effect, there are other issues involved in fitness performance such as training effect of assessments and reduced physical activity that need to be considered (Norman, et al., 2005). However it is clear that children that are obese and unfit are likely to be at higher risk of developing cardiovascular risk factors and insulin insensitivity. The consensus is that within a population increased physical fitness is strongly associated with a reduction in risk factors for cardiovascular disease and insulin resistance whatever the body size independent of the type of cardiorespiratory fitness test or technique of body mass assessment employed by researchers.

### 2.3.3 Muscle strength

Compared with cardiorespiratory fitness, static, resistive muscle strength is a factor that has not been considered to a large degree in obesity prevention programme. Yet increased, and metabolically fit, muscle mass is associated with more efficient disposal of glucose in adults (Snowling & Hopkins, 2006). How muscular strength interacts with obesity and child health may be important in the development of effective interventions to prevent chronic disease. Prista et al. (2003) and Deforche et al. (2003) found that obese subjects had higher grip strength but less strength in weight bearing tasks (such as standing broad jump, bent-arm hang and endurance shuttle run) than non-obese subjects. Lesser strength of bent leg curl-ups in obese children was also observed in Taiwanese children by Chen et al. (2006). Whereas Grund et al. (2001) found no difference in leg strength in higher weight children. Inability to pass the upper body strength testing (Amateur Athletic Union and Fitnessgram) was associated with overweight in 5 to 14 year old children (Kim, et al., 2005). Wearing et al. (2006) state that these differences demonstrate the reduced strength performance of obese children, especially if strength values are adjusted by body weight.

The correlation of muscle strength and health outcomes with obesity in children is difficult to review because of the lack of any relevant and in-depth publications. The most relevant report from Benson and team (2006) claims that increases in strength improve insulin sensitivity and that resistance training may provide some helpful health benefits for children, however the research evidence is equivocal at this point in time. Absolute strength from daily activity is different from strength gains from resistance training programmes and evidence obtained from the application of both modes would be useful for developing and improving intervention programmes (Snowling & Hopkins, 2006).

## 2.4 Summary

Overweight and obesity in childhood is increasing worldwide although in New Zealand, it appears the proportion of overweight and obese children may have reached a plateau (Ministry of Health, 2008b), the number of Maori and Pacific and low SES children is increasing. Lifestyle patterns and body mass in childhood have a high likelihood of being carried into adulthood and this may place more adults at risk of developing insulin resistance, a precursor to T2DM and cardiovascular disease and cancer, which presages earlier mortality and reduced quality of life. More ill people will increase pressure on health care organisations to provide treatment. Although body composition changes, that are mediated by nutrition (caloric input) and physical activity (caloric output), may be explained by the direct relationship with energy balance, the functional relationship of physical fitness with body composition in young children is unclear. More research is needed to identify the metabolic processes and associations with the accumulation of excess fat, risk for cardiovascular disease, and insulin resistance. Protective factors need to be identified and incorporated into health promotion and intervention programmes. Studies that investigate the physiologic relationship of food patterns and physical fitness and activity with weight gain will help provide evidence to inform the design of more effective health interventions for children.

# 3 Patterns of daily life in primary age school children

This chapter reports contemporary food and physical activity and sleep patterns as reported in questionnaires by parents of children in the *Project Energize* evaluation. In 2004 schools were randomized to either control or to receive the Project Energize programme. Follow up measurements were made in 2006. This report concerns those children whose parents completed questionnaires at both time points and where the children did not change from a control to programme school or vice versa. Questions asked included demographics and food, physical activity, physical inactivity and sleep habits. Children had measures of age, height, weight and body fatness by bioimpedance at both points. Popular foods, physical activity choices and pattern are described. The primary outcome measure was a comparison between control and programme schools of two year change in standard deviation scores (SDS) for body mass index (BMI) and percentage body fat (PBF). These scores were derived from the Centers for Disease Control (CDC) growth charts (Kuczmarski et al., 2000) and English population norms respectively, which are adjusted for sex and precise chronological age at time of measurement.

## 3.1 Literature review and rationale

According to several researchers (Hill, 2006; Ravussin & Bogardus, 2000; Spiegelman & Flier, 2001; Uauy & Diaz, 2005) weight gain in adults and children can be explained by a "gap" in between energy in and energy out over a long period of time (Figure 2). Resting energy expenditure (REE) and diet-induced thermogenesis (DIT) components of total energy expenditure (TEE) are relatively fixed as they are determined by total body mass and familial inheritance (Goran, 1997). However in children there are energy demands both for growth and when mass is increased. Nonetheless the factors of food intake and physical activity are the primary means by which weight management is deemed possible. A disproportionate number of children's studies have focussed on a single factor, such as physical activity (Dencker et al., 2006; Jurg, Kremers, Candel, Van der Wal, & De Meij, 2006; Rennie, Wells, McCaffrey, & Livingstone, 2006; Ruiz, Rizzo et al., 2006; Yin, Hanes et al., 2005; Zahner et al., 2006; Ziviani, Macdonald, Ward, Jenkins, & Rodger, 2008), inactivity (Berkey, Rockett, Gillman, & Colditz, 2003; Gordon-Larsen, Adair, & Popkin, 2002; Hussey, Bell, Bennett, O'Dwyer, & Gormley, 2007; Koezuka, et al., 2006; Prentice & Jebb, 2006; Tremblay, Barnes, Copeland, & Esliger, 2005) and food (Casazza & Ciccazzo, 2006; J. A. Kim et al., 2007; Theodore et al., 2006; Van Horn, Obarzanek, Friedman, Gernhofer, & Barton, 2005;

Weigensberg et al., 2005) patterns, rather than explore the interrelationships of all three of the modifiable factors - food, physical activity and inactivity (Aeberli, et al., 2007; Berkey, et al., 2000; Gibson & Neate, 2007; Goldfield et al., 2006; Janssen, Katzmarzyk, Boyce, King, & Pickett, 2004; Utter, Scragg, Schaaf, et al., 2006) with body size. While interpretation and analysis of interactions among determinants of mass gain may be difficult, it is essential to inform a more complete picture of real-world obesity issues. Bar-or et al. (1998) suggest that behaviour modification techniques encompassing the lifestyle factors of food and activity patterns of children would be the most effective method of reducing childhood and future adulthood obesity and obesity-related issues. A call was also made by these authors to consider the upstream social pressures that promote inactivity and overconsumption of food. There are several researchers (Davison & Birch, 2002; Green et al., 2003; Rennie, Johnson, et al., 2005) who provide evidence that parents have the most influence to improve their child's health through food and activity both as role models and via the environment within which the child operates.

### 3.1.1 Interventions to change daily life patterns

A Canadian community study by Merchant et al. (2007) considered the environment of children in a comparative and naturalistic setting. Two schools were enrolled in this investigation; 'school A/low SES' (n=48) was considered a low socioeconomic and, 'school B/high SES' (n=112), high. There was no difference between BMI z-scores (BMI SDS) of children of the two schools but perceptions and reported behaviours were very different. In comparison to school B/high SES parents the parents of 'school A/low SES' children were less educated and had lower income, and perceived the built environment of the school A/low SES neighbourhood to be less "walkable". Furthermore "school A/low SES' children consumed more baked goods, chips, sodas, confectionary and spent more time in front of TV and the computers than their school B/high counterparts. The mean age of school A/low SES children was 11.2 years (SD ±2.0) and school B/high SES, 8.2 years (SD ±1.5) but apart from the use of BMI SDS to compare there was no apparent adjustment for the three year difference in age or any confounding of sex. While school A/low SES children reported "sitting" for longer than school B/high SES children (weekend days 61 ±31 mins/day vs 43 ±51 mins/day, $p<0.05$; weekdays 268 ±672 mins/day vs 134 ±144 mins/day, $p$=NS) age and sex were not considered as confounders. The statistical analysis that was applied in this study may not have been appropriate for a non-normally distributed data set however the identification of differences in patterns of inactivity and the impact of features and

perceptions of the built-environment were important to inform possible food supply and environmental interventions. This study emphasises the complexity of the obesity issue, highlights methodological issues and identifies potential modifiable actual and perceptual lifestyle and environmental factors that are very relevant to the New Zealand situation.

Six year tracking of 975 six to thirteen year old Chinese children (Wang, Ge, & Popkin, 2000) showed that those who remained in the upper quartile of BMI throughout were more likely to have at least one obese parent (OR 1.9 95% CI 1.1, 3.1) and dietary fat intake of no more than 10% of total energy intake also tracked (OR 2.2, 95% CI 1.2, 4.0). In comparison, increased consumption of protein, quantity of meat products in the diet, time spent viewing television and playing personal computer games were found to be associated with overweight in 142 Swiss children (Aeberli, et al., 2007). Twenty four hour dietary recall data in 1385 six to eleven year old Chinese children showed that not only are Chinese children less over weight than US children, but they are more active and are less likely to consume snacks (Waller, Du, & Popkin, 2003). Wrotniak et al. (2007) applied regression analysis to the relationships amongst physical activity, health and food patterns between children and also their parents. No food or physical activity relationship could be found with the percentage overweight outcome variable in children, however parents physical activity (in MET's) explained 46.2% of the variance of children's physical activity. Perhaps the lack of food effect could be explained by Janssen's 2005 review (Janssen, Katzmarzyk, Boyce, et al., 2005) of cross-sectional studies involving 137,593 children aged 10 to 16 years. In this appraisal, physical activity and television viewing, not food patterns, was found to be related to overweight. Limitations recognised by the authors include that food frequency recalls may be subject to under-reporting bias by overweight children compounded by no measures of portion sizes consumed. The observation by Berkey et al.'s (2000) in 9 to 14 year old American girls (n=6,149) and boys (n=4620) that time spent watching TV and videos, and playing electronic games contributed to an increased BMI is in agreement with Janssen et al. (2005) and Aeberli et al. (2007). Berkey and colleagues (ibid) also found that, over a year, an increase in calories consumed and inactivity time was associated with an increase in BMI. Although the magnitude of the effect size was small ($<0.04 kg/m^2$), the authors proposed that these small effects were cumulative and over many years could potentially develop into significant weight gain. These studies demonstrate that although it may be logical reasoning to assume food and activity patterns contribute to fat accumulation and an excessive increase in body size, to conclusively determine that outcome in children over a relatively short time is

a difficult task. The various determinants of body size may have such a complex interaction that current methodologies and statistics may not be appropriate to demonstrate the relationships of these lifestyle behaviours (Newby, 2007). Investigation and intervention in other lifestyle behaviours including sleep patterns also have inherent difficulties. Throughout the life time, but particularly with children, more than one third of total time spent is spent sleeping.

### 3.1.2 Sleep

Habitually short sleep duration is a relatively novel concept as a lifestyle behaviour risk factor for obesity. The basic construct is that reduced night–time sleep promotes obesity based on two physiological processes that have been confirmed by experimental studies in adults, 1) promotion of the wake-promoting and appetite-stimulating effects of the orexin system (Sakurai, 2005) and 2) upregulation of the sympathetic nervous system (Rechtschaffen & Bergmann, 1995) . All data collected of sleep duration and analysis of the association with obesity in children are from epidemiological studies. The oldest study (Locard et al., 1992) involved 1031 five year old French children and relative risk factors were drawn from interview recorded constitutional factors (parental and birth overweight) and environmental factors (family structure, socio-economic and daily lifestyle). Parental overweight and short sleep duration elicited estimated relative risk factors of 3.1 and 4.9 respectively. Also, five to six year old German children (von Kries, Toschke, Wurmser, Sauerwald, & Koletzko, 2002) that slept 10.5-11.0 hours (OR 0.77, 0.59-0.99; 0.53, 0.35-0.80 respectively) or more than 11.0 hours (OR 0.54, 0.40-0.73; 0.45, 0.28-0.75 respectively) were found to be less likely to be overweight and obese. Similarly, Duncan et al. (2008) observed that less than ten hours sleep during weekdays was associated with overfatness (equal to or more than percentage body fat 25% in boys and 30% in girls) in New Zealand children (n=1229) aged five to eleven years (Adjusted OR 7.03, 1.63-30.4). Similarly Sekine et al. (2002) found, in 8,274 Japanese six to seven year olds that sleep duration of nine to ten hours, eight to nine hours and less than eight hours all increased the risk of developing obesity (1.49, 95% CI 1.08, 2.14; 1.89, 95% CI 1.34, 2.73; 2.87, 95% CI 1.61, 5.05 respectively; $p<0.001$). Comparable odds ratios were calculated by Eisenmann et al. (2006) who investigated sleep duration and overweight data from 6,324 Australian children (aged 7.5 to 16.5 years). A significant sleep duration and overweight odds ratio was uncovered for males (adjusted OR 3.06, 95% CI 2.11, 4.46 respectively), but not for females (adjusted OR 1.09, 95% CI 0.68, 1.20). Canadian girls aged five to ten years that slept between eight and ten hours per day had

lower odds to be overweight or obese (OR 3.15, 95% CI 2.06,4.43) than boys of the same age (OR 5.65, 95% CI 4.23, 6.75) in Chaput et al.'s (2006) study. Nixon et al. (2008) are the only authors to employ objective measures of sleep parameters using an Actigraph in 519 seven year old New Zealand children. Actigraph measures of night-time sleep and daytime activity estimated an overweight/obese odds ratio of 3.92 (95% CI 1.91, 8.06) for less than 9 hours sleep. One of two longitudinal studies that employed sleep as a covariate for obesity, the AVON longitudinal study (Reilly, Armstrong, et al., 2005), was able to identify that less than 10.5 hours sleep per day at three years of age (n=909), led to an increased risk of obesity at age seven (OR 1.45, 95% CI 1.10, 1.89). Agras et al. (2004) also found that reduced sleep duration as an infant appeared to contribute to obesity outcomes in 150 children tracked from birth to 9.5 years of age. The habitual sleep duration of three to twelve year old American children (n=2281) (Snell, Adam, & Duncan, 2007) decreased (weekday: 2.5 hours per night; weekend: 40 mins per night) over five years and early sleep habits (baseline bedtime R=0.116, $p<0.01$; baseline waketime R=-0.120, $p<0.05$) appeared to contribute to subsequent increases in BMI. In contrast, Horne (Horne, 2008) provides a critical review of the sleep evidence stating that some of the studies (Agras, et al., 2004; Locard, et al., 1992; Reilly, Armstrong, et al., 2005; von Kries, et al., 2002) presented above focus on statistical significance whereas the results may not be clinically significant and therefore have no practical bearing on obesity treatment. Issues he raises are: 1) extrapolation of the physiological evidence from the short-term experimental studies to the results found in longitudinal studies is erroneous because the experimental studies consisted of drastically reduced sleep periods (for example <4hrs) and did not continue for more than one week and children's sleep duration in the longitudinal research did not reduce to the extent found in the experimental studies 2) Children's sleep duration was self-reported in the longitudinal studies and therefore prone to error 3) the mean difference in habitual sleep duration between non-obese and obese children, although statistically significant, is negligible. For example, Lumeng et al. (2007) uncovered a significant difference of 14.4 minutes per day in sleep duration between overweight and normal weight American children at 12 years of age. Thus sleep is a factor that does need to be considered for any population study into paediatric obesity; however the causal pathways, and the amount of sleep reduction required, are unknown. Additionally, most of the studies of sleep have been conducted with children of European origin, thus ethnic differences are an unknown aspect.

### 3.1.3 Ethnicity

The 2008 New Zealand Health Survey (Ministry of Health, 2008b) presented results that showed the prevalence of obesity to be 23.3% in Pacific children (aged 2 to 14 years) and 11.8% in Maori whereas European and others the prevalence was less than 6.0%. Issues have been raised as to whether the IOTF BMI cutoffs are suitable for ethnicities such as Pacific and Māori. Rush et al. (2003) has shown that for the same BMI Maori and Pacific girls have a higher fat free mass than European girls. It is also known the Māori and Pacific girls have their first menses at a much earlier age than European (Ministry of Health, 2003). Globally, minority ethnicities are most at risk for developing obesity and the situation for children is no different. It is also widely recognised that minority populations are often the poorest (Kumanyika & Grier, 2006), have restricted access to medical care, limited food security (Bronte-Tinkew, Zaslow, Cappsa, & Horowitz, 2007), reduced opportunities for physical activity (Kumanyika & Grier, 2006) and high rates of television viewing. Similarly in New Zealand, Pacific and Maori children together make up the majority of the low socioeconomic youth population (Utter, Scragg, et al., 2007).

### 3.1.4 Socioeconomic status

Socioeconomic status (SES) has long been associated with obesity however studies still struggle to determine the mechanism of the relationship (Shrewsbury & Wardle, 2008). The developmental level of the country may determine which socioeconomic factors contribute most to obesity (McLaren, 2007). There appears to be a number of contributing factors in low socioeconomic dwellings that promote obesity such as parental behavior and education, closer proximity of fast food outlets and less healthful food vendors in the neighbourhood, unsafe streets, lack of facilities that promote activity and higher levels of television viewing (Kumanyika & Grier, 2006). Magnsson et al. (2005) suggest that lack of awareness about healthy lifestyle amongst low socioeconomic groups may be a reason for high obesity prevalence and Drewnowski (2005) adds that the low cost of high energy density-low nutrient value foods remains a tempting target for those with less disposable income.

### 3.1.5 Consequences of obesity

The association of adult obesity with the development of disease is well known and it is also known that most children that are obese will continue as obese adults (Magarey, et al., 2003). Freedman found similar rates obesity tracking into adulthood in their cohort that were followed over 17 years as well as identifying Caucasian versus African American differences.

This places fatter children at high risk for developing cardiovascular disease, osteoarthritis and metabolic disease such as type 2 diabetes. The association of excess fat in childhood (both BMI and body fat methods) with future health issues cannot be ignored. It is important to know the context of increased childhood fatness so that research can inform both treatment and prevention.

## 3.2 Introduction

This chapter addresses the association of demographics and lifestyle patterns on fat accumulation and body size of primary school age children participating in a nutrition and physical activity intervention. The intervention, *Project Energize* (Graham, et al., 2008), set out to improve the health of primary school children by working with schools to provide more opportunities for improved food choices and increased physical activity opportunities effected by an "Energizer" working within the school. The focus of this chapter is not the effect of the intervention (which will be reported elsewhere), but the factors that are related to body mass both cross-sectionally and over time. Food intake and physical activity patterns represent opposing arms of the energy balance equation and are important factors in fat accumulation. Other factors that will be considered are habitual sleep patterns, ethnicity, socioeconomic status represented by school decile, and inactivity represented by 'screen time'. This study utilises the data collected at baseline and followup two years later of two groups, five and ten year old children, from the New Zealand District Health Board region of Waikato. Further, boys and girls are considered separately, for the most part, considering their biological and behavioural differences.

New Zealand children are amongst the most overweight and obese in the world (Wang & Lobstein, 2006). Although the prevalence of overweight and obese (aged 5 to 14 years) in New Zealand has appeared to plateau from 2002 (boys: 20.0% and 9.0%; girls: 22.8% and 10.7% respectively) (Ministry of Health, 2003) to 2007 (boys: 20.5% and 8.0%; girls: 21.3% and 8.7% respectively) (Ministry of Health, 2008b), the consequences of an 'excess fat' state in children is likely to lead to dire cardiovascular and metabolic circumstances in adulthood. Adult rate of obesity in New Zealand is one in five (Ministry of Health, 2008b). There are remarkably few published New Zealand dietary (Boulton & Magarey, 1995; Grant, Ferguson, Toafa, Henry, & Guthrie, 2004; Theodore et al., 2006) or physical activity studies. An early food pattern study (Boulton & Magarey, 1995) of New Zealand European children had found that dietary fat intake did not detrimentally affect growth including weight. In a longitudinal study of preschool Pacific children (Grant, et al., 2004) dietary factors were not found to be

associated with increased body mass, although the statistical analysis was not clear how this conclusion was made. The contribution of dietary sugar to total energy intake was found to be very high (2-3 year olds: 30.3%; 4-5 year olds: 25.9%) in this pre-school study population. Theodore et al. (2006) revealed that the foods consumed by 549 pre-school New Zealand European children were remarkably similar to those reported by school-aged children in the National Children's Nutrition Survey. The similarity may indicate that food patterns are started early, presumably because of the influence of the caregiver and that continuation of those patterns is merely habituation rather than choice. Analysing data from the National Children's Nutrition Survey, Utter et al. (2007) show that purchasing school food from shops or school canteen predicted a 0.53 increase in BMI ($p$=0.044), and lack of breakfast at home was associated with increased BMI by 1.14 kg.m$^2$ ($p$=0.007). However, watching TV more than 2 hours per day (Utter, Scragg, & Schaaf, 2006) was associated with an increased risk of children, aged 5 to 10 years, consuming soft drinks more than five times per week (OR 2.2, 1.2 to 4.0, $p$=0.029), sweets and snacks and selected fast foods. Additionally, in a separate study (Utter, Schaaf, et al., 2007), those children who purchased their food from the school canteen were much more likely to purchase high fat (pies etc ≥3 times per week: OR 6.0, 2.7 to 13.1) and/or high sugar foods (soft drinks ≥5 times per week: OR 4.0, 2.0 to 8.0; confectionary ≥four times per week: OR 3.9, 1.5 to 10.3) than choose products that promoted fruit or vegetable intake.

On the other side of the energy balance scale, physical activity has had much more research conducted in New Zealand. Relationships between physical activity level (PAL, as measured by doubly labeled water and calorimetry, PAL= TEE:RMR) and body composition (by isotope dilution) were analysed and the authors (Rush, Plank, et al., 2003) found that Māori and Pacific children had a higher BMI than, but similar body fat percentage to, European children. Māori also had a higher PAL than either European or Pacific children. In addition, low levels of physical activity were inversely related to body fat in boys, but not girls. A longitudinal study assessing New Zealand European children at birth, 1.0, 3.5 and 7.0 years of age (baseline n=871) has been assessed by Blair et al. (2007). This particular study did not include food pattern data, but showed that a high weight for gestational age (>4000g; 8.8% body fat, 4.8 to 12.3) and more than 3 hours of TV watching per day (5.2% body fat, 1.2 to 9.1) were independently associated with high percent body fat at 7 years of age. Maternal factors of high maternal BMI (>30 kg.m2; 4.0% body fat, 0.4 to 7.7), maternal age (30-34 years; 2.5% body fat, -0.2 to 5.3) and being a female child (2.4% body fat, 0.7 to 4.0) also

increased risk of accumulating body fat. Utter et al. (2006) shows that TV use ($p$=0.07) or less physically active behaviours ($p$=0.22) were not significantly associated with BMI in multivariate analysis, when adjusted for demographic variables.

Notwithstanding the work presented in the literature review and introduction above, there is a serious lack of knowledge regarding lifestyle habits (food and physical activity) of young children. This is especially so in New Zealand where the ethnic minorities (Māori and Pacific) are over-represented in obesity and illness statistics. Consistent relationships amongst food intake, physical activity and body composition are yet to be confirmed by research. Also longitudinal studies of children are conspicuous by their rarity. It is vital that New Zealand-specific research be conducted that accounts for the wide variance and changes in food, activity, inactivity and sleep behaviours in children so that we may determine the factors that increase the risk for excess fat accumulation.

### 3.3 Aim

The principal aim of this study was to determine interrelationships of demographics (that is, ethnicity and socioeconomic status) and lifestyle behaviours (that is, food, physical activity and physical inactivity and sleep patterns) with the accumulation of body mass as represented by BMI and PBF SDS in two age groups, five and ten year olds, over the period of two years.

### 3.4 Hypotheses

1. Baseline BMI SDS and PBF SDS will predict BMI SDS and PBF SDS two years later for all children
2. Ethnicity of Māori and low decile children will predict notably higher BMI SDS and PBF SDS levels
3. Consumption of high energy (fat and sugar) foods will be higher, and low energy foods (fruit and vegetables) will be lower, in obese (IOTF criteria) children than not obese children
4. Participation in physical activity will be lower and physical inactivity higher in obese (IOTF criteria) children than not obese children
5. Obese children will sleep less than not obese children

## 3.5 Method

The Waikato Ethics Committee approved this randomized control longitudinal study of the effectiveness of Project Energize programme to be conducted across primary schools in the District Health Board region of Waikato. The programme was through-school although only children aged five and ten were invited through the schools to participate in the research. Both parents and children provided informed voluntary consent to participate. Measures of anthropometry, blood pressure and heart rate (cardiovascular profiling) were collected at baseline and at 24 months in concert with the distribution and collection of questionnaires.

Design, programme randomization and evaluation methodology have been reported previously (Graham, et al., 2008). Method relevant to this analysis is detailed below. The researcher (GK) was introduced to Project Energize in 2005, and involved with measurements of anthropometry, and cardiovascular profiling in 2006. GK had no input into the design and method of the main study and thus the intervention is to be reported elsewhere and is not part of this body of work.

### 3.5.1 Population

Cluster (by geographical area) randomisation of schools that accepted the invitation to participate was stratified by decile, location, ethnicity and size. The Energize programme was delivered to 62 schools and 62 schools were enrolled as control: there were approximately 11,000 children in each arm of the study. The evaluation cohort was restricted to only five and ten year old children, a total of 6456, of whom 3034 (47%) children gave assent with parental consent to have measures of body size made at school. These age groups were selected to represent the two extremes of (full) primary school age from 5 to 12 years of age over the two year study period.

Ethnicity was defined as the singular choice of self-selected ethnicity by the caregiver/parent. If more than one ethnicity was chosen, those that included Māori were defined as Māori. Those not selecting European or Māori as one of the ethnicities (10.8%, n=327/3,034) were excluded from this analysis.

Children who did not change from a programme to control school, for whom ethnicity was recorded as Māori or European and measurements (BMI and PBF) were made at both points totaled 1315, and of these 384 (29.2%) were Māori

Household questionnaires which included questions related to food and activity, were sent to all parents who provided consent and were returned by 618 (117, 18.9% Māori) parents

meeting the above criteria at both baseline and two years later. This represents 20.4% of those children originally providing assent. It is this, the "complete data", that is included in the analysis presented in this chapter.

Table 1 displays the proportion of children in the control and programme groups, by age group and ethnicity. Māori five year old children represent 18.0% of all five year olds included in this study and Māori ten year olds represent 21.3% of all ten year olds.

**Table 1. Children by age group and ethnicity in intervention group (N=618).**

| Age group | Control (number boys) | Programme (number boys) |
|---|---|---|
| Child5 | | |
| European | 173 (86) | 188 (92) |
| Maori | 37 (16) | 42 (25) |
| Child10 | | |
| European | 88 (46) | 52 (24) |
| Maori | 20 (9) | 18 (10) |

Data will be discussed using the following combinations of coding: children (all children regardless of sex), boys (male only), girls (female only), 5 (baseline five year olds), 7 (five year olds assessed two years later), 10 (baseline ten year old) and 12 (ten year olds assessed two years later). For example, when discussing the five year old boys data at final assessment, the coding - boys7, will be applied. Ten year old children at baseline, regardless of sex, will be coded - child10.

### 3.5.2 Socioeconomic status

A surrogate measure of socioeconomic status is school decile (Ministry of Education, 2008). Decile is determined from the deprivation rating of census- selected meshblocks near their respective school. Decile one is described as the most deprived and decile 10 the least deprived. Although not a precise individual classification of SES, school decile provides a general and current indication of a child's socioeconomic level and the area that they reside in. The decile used in this analysis is that one allocated to each school by the Ministry of Education in 2003. Decile was further categorized into low (1 to 3), medium (5 to 7) and high (8 to 10) for purpose of these analyses.

### 3.5.3 Anthropometry

All children removed excess clothing and shoes prior to the anthropometric measures. Height (cm) was assessed by stadiometer that was regularly checked for accuracy and precision (±0.1cm). Body mass (kg) was measured using personal weight scales (±0.1kg; TIHD316 Personal scales, Wedderburn NZ). All measurements were recorded in duplicate and repeated if not within the defined tolerance. Height and body mass values were used to calculate body mass index (BMI $kg.m^{-2}$). BMI values were converted to age- and sex-adjusted BMI SDS utilizing the Center for Disease Control 2000 (Kuczmarski, et al., 2000) centiles.

Classification of obesity, overweight or normal body mass index was by application of the international obesity task force (IOTF) criteria of Cole et al. (2000). Body fat (%) was derived from single frequency (50kHz) measurements of hand to foot resistance (Imp5, Impedimed, Queensland) using an equation previously validated in a similar population (Rush, Puniani, Valencia, Davies, & Plank, 2003). Briefly, electrodes were placed on the hand and foot, at the carpal/tarsal crease and at the distal third meta-carpal/tarsal. Resistance, reactance, phase angle and impedance values were recorded as raw data. Because these values are mathematically related dimensions of the same impedance vector, data integrity was able to be verified to correct recording of the resistance measurement validated. Percentage body fat (PBF) values were converted to age- and sex-adjusted PBF SDS applying the McCarthy et al. (2006) body fat reference curves. The advantage of using standardized scores is that age and sex variables are accounted for in individual and group values. Both BMI SDS and PBF SDS were used as outcome variables for the statistical analysis.

### 3.5.4 Household Questionnaires

Responses to demographic, food and physical activity and physical inactivity questions to investigate patterns of behaviour were provided by the household questionnaires. The baseline questionnaire is represented as HHQ1 (See Appendix B) and the follow-up questionnaire two years later is represented as HHQ2 (See Appendix C). To investigate associations with body composition change selected household questions were grouped (See Appendix A, Table 33 and below for detail) to reflect eleven categories of lifestyle behaviours. Six categories of food pattern were determined from the frequency of consumption of foods categorised as fruit, vegetables, healthy, takeaway (fast food), high energy carbohydrate and high energy fat. Each category was independent from all others, for example high energy fat foods do not include any question item that could potentially belong

in high energy carbohydrate foods. For instance, biscuits are potentially high in both sugar and fat however they were arbitrarily allocated to the carbohydrate category only. Five categories were related to physical activity and inactivity behaviours, sleep and resources. Two of those five were determined from the frequency of participation in occasions categorized as physically active (sport and play) and time spent physically inactive (screen time). Habitual sleep duration was calculated from time to bed and time of waking averaged for week and weekend days. The presence of resources that promote physical activity (for example, play equipment) and inactivity (for example, multimedia devices in the bedroom) formed the two final categories, activity and inactivity promoters. Detail of the coding and categorization process is provided in the following sections.

### 3.5.5 Food Patterns

As responses to frequency questions (HHQ1 sect 4, 24 to 31 which were exactly the same as HHQ2 63 to 71) had possible responses that ranged from never and less than once a month, to twice or more times a day, responses were weighted to fractions of portions in a day. For example, if a child consumed a food product once per month, the weighting applied is $1/30^{th}$ of a day. Some food frequency questions have been ordered into general cumulative categories (See Table 33) of fruit, vegetables, health promoting, takeaway, milk, carbohydrate, and fat foods. The daily frequency of foods within each food category was summed to give frequency/day - scaled variables. These were used as independent variables to explain the dependent variable - body mass (BMI SDS and PBF SDS).

*Fruit and vegetables*

Questionnaire HHQ2 was amended regarding vegetable and fruit consumption, where HHQ1 contained one question each regarding the consumption of fruit or vegetables, HHQ2 expanded that question to enquire of the intake of specific fruit and vegetables. Thus HHQ2 fruit and vegetables questions were summed to present a single total which is much higher than the single question amount from HHQ1. For this reason HHQ2 total was not able to be compared to HHQ1 fruit and vegetable intake frequency because of validity issues surrounding the survey question amendment. Consumption of these foods is weighted by intake frequency per day.

*Health promoting foods*

Health promoting foods is a broad category including foods that contain nutrients that are thought to promote general good health including water, nuts, seeds, and vegemite. Consumption of these foods is weighted by intake frequency per day.

*Carbohydrate food frequency*

The carbohydrate foods are products that are energy dense foods that contain a higher proportion of carbohydrate (e.g. sugary foods and cereals) than other macronutrients of fat and protein. Consumption of carbohydrate foods have been linked to less incidence of overweight. Each weighted carbohydrate food frequency was summed to indicate relative consumption per day.Flavoured sweetened milk and sugary soft drinks were included in this category.

*Fat food frequency*

The fat foods were defined as foods where the most energy was derived from fat compared with carbohydrate and protein, e.g. fried foods, crisps. Overconsumption of fatty foods may predispose an individual to weight gain by contributing to a positive energy balance. Each weighted fat food frequency was summed to indicate relative consumption per day.

*Milk food frequency*

All milk-only products except for flavoured sweetened milk were included in this variable. Other dairy products such as yoghurt or cream were also excluded (See ). Milk and water are promoted as a healthy alternative to other fluids. Each weighted milk food result was summed to indicate consumption per day.

*Takeaway food frequency*

Takeaway food frequency included ready-to-eat meals purchased at restaurants and fast food convenience restaurant brands such as McDonalds, Kentucky Fried Chicken, Pizza hut, and ethnic-based food outlets. High frequency of purchase would be considered to promote fat accumulation and be detrimental to health. Each weighted takeaway food result was summed to indicate relative consumption per day.

### 3.5.6 Physical Activity Patterns

*Physical Activity*

Physical activity was assessed, in this study, in a similar way to food frequency. Activity questions were weighted by their frequency per day and summed for each pattern. In addition, promoters of physical activity, i.e. the presence of equipment or facilities that a child may freely access in order to be more active were summed for total number of physical activity promoters present in or near their home.

*Physical Inactivity*

Although all physical inactivity cannot be accurately recorded by recall, certain elements may be able to be categorised as sedentary such as watching television, playing electronic games, or using a computer. These questions in the household questionnaire asked for an estimated hourly use of the abovementioned items in questions separated into weekdays and weekends. The hours were collated into a total estimated physically inactive hours per week by the following equation:

Physical inactivity (hours per week) = (weekday hours * 5 [days per week]) + (weekend hours * 2 [days per week]) (1)

Further, total number of physical inactivity promoters (for example, television or phone in the bedroom) present in or near their home was also recorded.

*Sleep*

Habitual daily sleep duration was calculated from estimated time to bed and time of waking for both weekdays and weekend days as recorded by the parent. Weekday and weekend day values are utilised as explanatory variables in statistical analysis. Total estimated weekly sleep is calculated using the following calculation:

Total Estimated Weekly Sleep = (Weekday sleep [hours] *5 [days per week]) + (Weekend day sleep [hours] * 2 [days per week]) (2)

### 3.5.7 Statistical Analysis

Data were analysed using SPSS software, gradpack version 15.0 (SPSS, USA). Results with P values <0.01 were considered significant to build-in a tolerance for the high number of statistical analyses performed. Comparison of frequency among groups was examined using the chi squared test. The distribution of the continuous variables for example, anthropometry was examined within group for example, programme/control school, age group, sex, and ethnicity for normality, outliers and homogeneity of variance. A normal distribution was found in 128 (73%) of a possible 176 categories of anthropometric variables [(control and programme) 2 x (male and female) 2 x (Māori and Non-māori) 2 x Age group (5 yo and 10 yo) 2 x (Age, Ht, Ht SDS, Wt, Wt SDS, BMI, BMI SDS, PBF, PBF SDS, Arm, Waist) 11]. Descriptive statistics are presented as mean values and standard deviations and where data within groups was not normally distributed either the data was transformed before analysis or non parametric tests used. Where variables were normally distributed, comparisons were made using the general linear model including analysis of covariance (ANCOVA = multivariate linear regression) to explore associations and differences, between groups defined by treatment, age group and sex. Changes over time were analysed using the baseline value of the variable of interest as a covariate. This technique adjusts for any differences between groups in the baseline data. Associations between normally distributed variables were examined using Pearsons r and non-normal using Spearmans's rho to examine patterns of association. For non parametric data frequency, median, interquartile range are reported and comparison by group using the Mann Whitney U-test for example, for programme, sex and ethnicity as the grouping variables. Ranking of dietary patterns (food frequency) and physical activity recall data were tabulated in a descriptive fashion and no statistical analysis was performed on the ranking data between control and programme groups and between not obese and obese. Kruskal-Wallis analysis was undertaken to calculate significant differences in dietary patterns and physical activity between decile group.

## 3.6 Results

Cross-sectional analysis of baseline and final results of both five and ten year old children assessed in this study appear in section 3.6.1. Within each age-group and at each measurement point, descriptive data precede bivariate analysis for differences among groups followed by longitudinal multivariate comparisons.

### 3.6.1 Descriptive data

Questionnaires from both time points were returned by 618 parents with children attending low (24.1%), mid (32.8%) and high (43.0%) decile schools. More European than Māori returned questionnaires (Table 2). Because of this "biased response" subgroup analysis by the fixed effects of ethnicity and/or decile and the interaction of ethnicity and decile analysis within age group was only examined for body size outcomes. To be considered for analysis each sub-group total had to be more than 40.

**Table 2. Ethnic summary by age group of returned household questionnaires**

| | child5 | child10 |
|---|---|---|
| European | 361 | 140 |
| Maori | 79 | 38 |

Descriptive data for child5 and child10 children by programme and control treatments are shown in Table 3. At both measurement points average BMI SDS and PBF SDS were greater than zero which indicates in general a higher BMI and PBF in these study children adjusted by age and sex than the two reference populations. Distribution of children by decile grouping was not different by sex but compared with Māori there were more European in the high decile group (decile eight to ten - 93% European, 7% Māori) and less in the low decile group; (deciles one to three) 57% European compared with 43% Māori.

Using the Cole criteria the classification of children as normal, obese or overweight over two years did not change within groups defined by treatment, age group and sex, Table 3.

**Table 3. Anthropometric characteristics of five year olds at baseline and final measurement (N=440)**

| | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | Baseline | | Final | | Baseline | | Final | |
| | Control (n=102) | Programme (n=117) | Control | Programme | Control (n=108) | Programme (n=113) | Control | Programme |
| Age (months) | 67.6 (±3.8) | 68.3 (±3.7) | 92.0 (±3.8) | 92.6 (±3.5) | 67.8 (±3.7) | 68.1 (±3.5) | 92.1 (±3.7) | 92.1 (±4.0) |
| Height (cm) | 114.5 (±5.4) | 115.4 (±4.8) | 126.9 (±6.0) | 128.1 (±5.4) | 114.1 (±5.1) | 115.0 (±4.5) | 126.2 (±5.8) | 127.6 (±5.4) |
| Height (SDS) | 0.31 (±1.01) | 0.41 (±0.98) | 0.17 (±0.99) | 0.32 (±0.97) | 0.35 (±0.93) | 0.49 (±0.79) | 0.07 (±0.97) | 0.30 (±0.84) |
| Weight (kg) | 21.5 (±3.0) | 21.9 (±2.6) | 27.4 (±5.0) | 28.0 (±4.1) | 21.2 (±2.9) | 22.0 (±3.5) | 27.2 (±5.0) | 28.0 (±5.5) |
| Weight (SDS) | 0.47 (±0.88) | 0.56 (±0.83) | 0.43 (±0.94) | 0.59 (±0.83) | 0.47 (±0.80) | 0.62 (±0.83) | 0.38 (±0.87) | 0.53 (±0.87) |
| BMI ($kg.m^2$) | 16.4 (±1.3) | 16.4 (±1.4) | 16.9 (±2.1) | 17.1 (±1.9) | 16.3 (±1.4) | 16.6 (±1.9) | 17.0 (±2.2) | 17.1 (±2.6) |
| BMI (SDS) | 0.58 (±0.77) | 0.61 (±0.84) | 0.47 (±0.85) | 0.56 (±0.79) | 0.55 (±0.79) | 0.64 (±0.83) | 0.45 (±0.79) | 0.47 (±0.86) |
| PBF (%) | 18.3 (±4.87) | 18.0 (±4.7) | 21.0 (±5.0) | 20.1 (±5.3) | 20.7 (±4.0) | 20.4 (±5.6) | 23.6 (±5.5) | 23.1 (±6.1) |
| PBF (SDS) | 0.50 (±2.26) | 0.49 (±1.93) | 0.89 (±1.04) | 0.62 (±1.26) | 0.77 (±1.20) | 0.44 (±1.74) | 0.60 (±1.19) | 0.40 (±1.53) |
| FFM (kg) | 17.6 (±2.5) | 17.9 (±2.1) | 21.6 (±3.3) | 22.2 (±2.5) | 16.8 (±2.1) | 17.4 (±2.1) | 20.6 (±2.9) | 21.3 (±2.9) |
| FM (kg) | 3.9 (±1.3) | 4.0 (±1.2) | 5.9 (±2.3) | 5.7 (±2.2) | 4.4 (±1.2) | 4.69 (±2.0) | 6.6 (±2.6) | 6.7 (±3.1) |
| Arm (cm) | 18.7 (±1.6) | 19.0 (±1.6) | 20.1 (±2.3) | 20.6 (±2.1) | 19.1 (±1.7) | 19.5 (±2.1) | 20.5 (±2.2) | 21.1 (±2.5) |
| Waist (cm) | 54.7 (±3.5) | 55.4 (±3.5) | 58.6 (±5.5) | 59.4 (±5) | 55.2 (±4) | 56.5 (±5.2) | 59.1 (±5.9) | 59.9 (±6.7) |
| Normal/OW/ Obese (%)† | 70.5/24.5/5.0 | 67.5/28.2/4.3 | 65.7/25.6/7.8 | 65.0/25.6/9.4 | 70.4/25.0/4.6 | 68.1/21.2/7.7 | 67.6/23.1/9.3 | 68.1/19.5/12.4 |

† IOTF criteria (Cole, et al., 2000) OW = overweight
*Control significantly different from programme ($p<0.01$)

**Table 4. Anthropometric characteristics of ten year olds at baseline and final measurement (N=178).**

| | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | Baseline | | Final | | Baseline | | Final | |
| | Control (n=55) | Programme (n=34) | Control | Programme | Control (n=53) | Programme (n=36) | Control | Programme |
| Age (months) | 127.9 (±3.9) | 128.4 (±3.9) | 152.3 (±4.2) | 152.8 (±3.9) | 126.9 (±4.2) | 127.5 (±3.7) | 151.2 (±4.3) | 151.9 (±3.8) |
| Height (cm) | 142.8 (±6.5) | 145.5 (±6.8) | 154.5 (±7.8) | 157.3 (±8.0) | 142.1 (±6.4) | 145.3 (±7.0) | 154.7 (±7.3) | 158.2 (±6.7) |
| Height (SDS) | 0.14 (±0.91) | 0.49 (±0.95) | 0.07 (±0.97) | 0.41 (±1.02) | 0.09 (±0.84) | 0.50 (±0.94) | -0.02 (±0.97) | 0.41 (±0.94) |
| Weight (kg) | 37.8 (±8.4) | 40.5 (±9.4) | 47.8 (±10.9) | 50.5 (±12.6) | 36.8 (±8.5) | 40.9 (±10.1) | 47.5 (±11.4) | 53.1 (±11) |
| Weight (SDS) | 0.25 (±0.98) | 0.54 (±1.02) | 0.21 (±1.04) | 0.43 (±1.11) | 0.03 (±0.91) | 0.47 (±0.89) | 0.15 (±0.97) | 0.67 (±0.88) |
| BMI ($kg.m^2$) | 18.4 (±2.9) | 18.9 (±3.2) | 19.8 (±3.3) | 20.2 (±3.7) | 18.1 (±2.9) | 19.2 (±2.9) | 19.7 (±3.4) | 21.1 (±3.4) |
| BMI (SDS) | 0.30 (±0.97) | 0.47 (±1.00) | 0.32 (±0.99) | 0.38 (±1.03) | 0.15 (±0.85) | 0.51 (±0.74) | 0.19 (±0.87) | 0.59 (±0.83) |
| PBF (%) | 21.0 (±7.1) | 22.6 (±6.7) | 24.5 (±7.0) | 25.3 (±6.4) | 22.8 (±5.6) | 25.9 (±5.3) | 26.2 (±4.8) | *29.5 (±5.9) |
| PBF (SDS) | 0.13 (±1.93) | 0.57 (±1.48) | 1.01 (±1.22) | 1.19 (±0.80) | -0.24 (±1.34) | *0.49 (±0.93) | 0.40 (±0.91) | 0.94(±0.97) |
| FFM (kg) | 29.5 (±5) | 31 (±5.9) | 35.8 (±7.7) | 37.6 (±7.5) | 28.2 (±5.2) | 29.9 (±5.3) | 34.7 (±6.7) | 37 (±5.7) |
| FM (kg) | 8.3 (±4.5) | 9.5 (±4.4) | 12 (±5.5) | 13.2 (±6.3) | 8.7 (±4) | 11 (±5.1) | 12.8 (±5.4) | 16.1 (±6.1) |
| Arm (cm) | 22.5 (±3.1) | 23.3 (±3.3) | 24.7 (±3.2) | 25.1 (±3.6) | 23.0 (±2.9) | 24.2 (±3.4) | 24.6 (±3.2) | 26.3 (±3.5) |
| Waist (cm) | 66.0 (±7.8) | 68.3 (±9.5) | 71.5 (±8.7) | 73.6 (±11.0) | 65.0 (±7.6) | 68.3 (±9.2) | 70.3 (±9.4) | 75.5 (±9.9) |
| Normal/OW/Obese (%)† | 69/22/9 | 53/29/18 | 67/20/13 | 53/32/15 | 72/19/9 | 56/28/17 | 68/23/9 | 53/25/22 |

† IOTF criteria(Cole, et al., 2000) OW = overweight
*Control significantly different from programme ($p<0.01$)

### 3.6.2 Change in BMI SDS over two years

When adjusted for intervention group (programme or control), decile (low, mid, or high), and ethnicity (European or Māori), the intervention did not demonstrate any significant effect on final BMI SDS in both the five year old age group ($p$=0.218) and the ten year old age group ($p$=0.503). Baseline BMI SDS was found to be the most influential predictor ($p$<0.0001) determining BMI SDS two years later in all children. The ethnicity of Maori was a significant determinant in the model ($p$=0.013) with, on average, Māori children increasing 0.148 BMI SDS units more than European over these two years. The same effect of ethnicity could not be found in the ten year old age group ($p$=0.177). Regression analysis confirmed the tracking effect of baseline BMI SDS to final BMI SDS in both age groups explaining 76.9% and 86.7% percent of the variance respectively.

**Table 5. Coefficient output of multiple regression analysis of dependent variable - final BMI SDS for child5 participants**

| | ***b*** | ***SE b*** | ***Standardised β*** |
|---|---|---|---|
| Constant | 0.002 | 0.068 | |
| Baseline BMI SDS | 0.879 | 0.023 | 0.866** |
| Ethnicity (Māori) | 0.138 | 0.052 | 0.064* |
| Sex | -0.066 | 0.038 | -0.04 |
| Decile | -0.014 | 0.025 | -0.013 |
| Intervention group | 0.004 | 0.039 | 0.003 |

Note. $R^2$= .772. * $p < 0.01$, ** $p < 0.01$

### 3.6.3 Change in PBF SDS over two years

Similar ANCOVA adjustments and results that appeared for BMI SDS were produced using final PBF SDS as the outcome variable and baseline PBF SDS as an explanatory variable. Baseline PBF SDS was a significant factor that influenced final PBF SDS in child5 ($p$<0.0001) and child10 ($p$<0.0001). Identification as Māori in child5 was associated with a higher increase (0.378 of PBF SDS units) than European ($p$=0.015). In regression analysis, baseline PBF SDS explained a smaller variance in final PBF SDS than BMI (3.6.2 above) for child5 (30.3%) and child10 (36.2%). Māori ethnicity appeared to contribute higher than European (p=0.015) in child5 but not child10.

**Table 6. Coefficient output of multiple regression analysis of dependent variable - final BMI SDS for child10 participants**

| | *b* | *SE b* | *Standardised β* |
|---|---|---|---|
| Constant | 0.081 | .090 | |
| Baseline BMI SDS | .960 | .030 | .925** |
| Sex | .081 | .052 | .043 |
| Ethnicity (Māori) | .046 | .072 | .020 |
| Intervention group | -.016 | .053 | -.009 |
| Decile | -.046 | .037 | -.039 |

Note. $R^2$= .868. * $p < 0.01$, ** $p < 0.01$

### 3.6.4 Ranking of Food and Physical Activity patterns

Food and activity patterns are descriptively explored in relation to child age group, intervention, sex and not obese or obese classification by IOTF criteria. For the following analyses no differences between Māori and European were observed.

The top ten foods that were reported as consumed by each age group were very similar and did not change markedly over two years or by intervention, Table and Table 7. Consistently the top five foods most frequently consumed by all groups were fruit, vegetables, water, white bread and butter. The only exception was the child10 programme group, where the fifth ranked food item was standard milk, however the sixth ranked item was butter. White bread was consumed more frequently than wholemeal/grain at baseline. In 2006, the bread most frequently consumed was white, but wholemeal or wholegrain breads were also reported as frequently consumed. Biscuits were high on the list of frequently consumed foods (ranking 6$^{th}$ to 8$^{th}$). Jam appeared in child5 and child7 lists (See Table) but not in child10 or child12 lists (See Table 7). Conversely, crisps were apparent in child10 and child12 lists but not in child5 or child7 lists. For the majority of children in 2004 standard fat milk was reported as a staple food and the median daily consumption of standard milk had reduced by one third in the child5 cohort from 0.9 serves/day to 0.6 serve a day two years later. In the older children, between 10 and 12 years of age, total milk intake decreased by one fifth. In both control and programme schools and reduced fat milk consumption increased

over two years by about 0.2 times per day in the child5 cohort and 0.1 times per day in the child10 cohort.

The obese children's parents often reported a lower mean daily intake frequency of foods, Table 8 and Table 9 than the not obese children. Differences in food consumption appeared, not in the first five ranked food items, but in the items ranked six to ten. Powdered drink made an appearance in child5 obese as did meat and tomato sauce for child7 obese (See Table 8). Child5 and child7 maintained very similar food items over two years, with the only difference in the tenth ranked item (child5: peanut butter; child7: weetbix).

When analysed by ethnicity (New Zealand European and Māori) the data showed that Milk, fruit and vegetable intake were similar between groups. Māori consumed carbohydrate-based foods more frequently than NZ European children especially in the older years [child10 Māori median 5.1 (IQR 3.9, 7.0) per day vs. NZ Euro 3.7 (3.2, 5.4), $p$=0.002; 4.0 (2.5, 5.3) vs. 2.9 (2.2, 4.1), $p$=0.016]. Although young Māori children consumed significantly more fast food than New Zealand European but fatty and fast foods were similar daily frequencies (between 4.3 and 4.8 or 0.1 and 0.2 respectively). In the early years (child5 and child7) Māori children were more sedentary (as represented by screen time) [child5 Māori: 17.5 (11.8, 25.0) vs. NZ Euro 14.0 (11, 20), $p$<0.001; child10 Māori: 19.5 (13.4, 28.1) vs. NZ Euro 16.0 (11.5, 21.0), $p$<], however this was less noticeable in child10 and child12 ($p$>0.01). Māori were more physically active than New Zealand European children in child10 (2.1 (1.3, 3.3) vs. 1.3 (0.8, 2.0), $p$=0.001) and child12 (1.6 (0.8, 3.4) vs. 1.1 (0.7, 1.9)) but very similar at child5 and child7.

The same data was also analysed by decile group (low, mid and high). High decile children were significantly less sedentary than mid and low decile children [child 5: 13.5 (10.5, 18.5) vs. 17.0 (11.5, 23.1) vs. 16.5 (11.5, 24.0), $p$=0.003; child7: 15.0 (11.1, 19.5) vs. 18.5 (12.5, 23.0) vs. 18.5 (12.5, 26.0), $p$=0.001]. Although fruit and vegetable frequency was statistically significantly different across decile, the practical significance was low (median 2.0 to 3.0 times per day, $p$<0.01).

**Table. Daily intake of top ten individual foods for five year olds at baseline (child5) and final (child7) summed and ranked by intervention group.**

| | Child5 | | | | Child7 | | | |
|---|---|---|---|---|---|---|---|---|
| | Control (n=102) | | Programme (n=227) | | Control (n=202) | | Programme (n=222) | |
| Sum Rank | Food | Mean daily consumption | Food | Mean daily consumption | Food | Mean daily consumption | Food | Mean daily consumption |
| 1 | Water | 1.75 | Water | 1.75 | Fruit | 5.10 | Vegetable | 5.24 |
| 2 | Fruit | 1.47 | Fruit | 1.51 | Vegetable | 4.96 | Fruit | 5.06 |
| 3 | Vegetable | 1.30 | Vegetable | 1.32 | Water | 1.80 | Water | 1.82 |
| 4 | Butter | 1.12 | Butter | 1.05 | Butter | 0.91 | Butter | 0.88 |
| 5 | Bread, White | 0.93 | Bread, White | 0.98 | Bread, White | 0.69 | Bread, White | 0.73 |
| 6 | Milk, Standard | 0.89 | Milk, Standard | 0.96 | Biscuits | 0.63 | Bread, WG | 0.62 |
| 7 | Biscuits | 0.66 | Biscuits | 0.70 | Milk, Standard | 0.65 | Biscuits | 0.61 |
| 8 | Bread, WG | 0.58 | Bread, WG | 0.54 | Bread, WG | 0.61 | Milk, Standard | 0.54 |
| 9 | Jam | 0.52 | Jam | 0.51 | Weetbix | 0.49 | Milk, Trim | 0.52 |
| 10 | Peanut Butter | 0.49 | Vegemite | 0.51 | Jam | 0.44 | Jam | 0.49 |

Bread, WG = Bread, Wholemeal/grain

**Table 7. Daily intake of top ten individual foods for ten year olds at baseline (child10) and final (child12) summed and ranked by intervention group.**

| | Child10 | | | | Child12 | | | |
|---|---|---|---|---|---|---|---|---|
| | Control (n=104) | | Programme (n=68) | | Control (n=105) | | Programme (n=69) | |
| Sum Rank | Food | Mean daily consumption | Food | Mean daily consumption | Food | Mean daily consumption | Food | Mean daily consumption |
| 1 | Water | 1.67 | Water | 1.63 | Vegetable | 5.07 | Vegetable | 5.81 |
| 2 | Vegetable | 1.26 | Fruit | 1.28 | Fruit | 4.20 | Fruit | 4.54 |
| 3 | Fruit | 1.24 | Vegetable | 1.20 | Water | 1.65 | Water | 1.61 |
| 4 | Butter | 0.97 | Bread, White | 0.95 | Butter | 0.89 | Butter | 0.83 |
| 5 | Bread, White | 0.87 | Milk, Standard | 0.88 | Bread, White | 0.77 | Bread, White | 0.77 |
| 6 | Milk, Standard | 0.67 | Butter | 0.87 | Milk, Standard | 0.62 | Bread, WG | 0.69 |
| 7 | Biscuits | 0.64 | Biscuits | 0.70 | Bread, WG | 0.61 | Biscuits | 0.62 |
| 8 | Crisps | 0.50 | Bread, WG | 0.53 | Biscuits | 0.54 | Milk, Standard | 0.52 |
| 9 | Drink, Powdered | 0.49 | Drink, Powdered | 0.51 | Milk, Trim | 0.46 | Sauce, Tomato | 0.49 |
| 10 | Vegemite | 0.45 | Crisps | 0.46 | Crisps | 0.44 | Peanut Butter | 0.45 |

Bread, WG = Bread, Wholemeal/grain

**Table 8. Daily intake of top ten individual foods for five year olds at baseline (child5) and final (child7) summed and ranked by IOTF obesity classification. (N=440)**

| | Child5 | | | | Child7 | | | |
|---|---|---|---|---|---|---|---|---|
| | Not obese (n=413) | | Obese (n=27) | | Not obese (n=397) | | Obese (n=43) | |
| Sum Rank | Food | Mean daily consumption | Food | Mean daily consumption | Food | Mean daily consumption | Food | Mean daily consumption |
| 1 | Water | 1.75 | Water | 1.75 | Vegetables | 5.14 | Vegetables | 4.83 |
| 2 | Fruit | 1.49 | Fruit | 1.51 | Fruit | 5.12 | Fruit | 4.70 |
| 3 | Vegetables | 1.32 | Vegetables | 1.21 | Water | 1.80 | Water | 1.89 |
| 4 | Butter | 1.09 | Butter | 1.10 | Butter | 0.91 | Butter | 0.75 |
| 5 | Bread, White | 0.95 | Bread, White | 0.98 | Bread, White | 0.72 | Bread, White | 0.63 |
| 6 | Milk, Standard | 0.94 | Milk, Standard | 0.77 | Bread, WG | 0.62 | Biscuits | 0.63 |
| 7 | Biscuits | 0.70 | Drink, Powdered | 0.68 | Biscuits | 0.62 | Bread, WG | 0.58 |
| 8 | Bread, WG | 0.56 | Bread, WG | 0.56 | Milk, Standard | 0.61 | Sauce, Tomato | 0.53 |
| 9 | Jam | 0.53 | Biscuits | 0.50 | Jam | 0.47 | Meat, CNO | 0.44 |
| 10 | Peanut Butter | 0.50 | Vegemite | 0.48 | Weetbix | 0.46 | Milk, Standard | 0.42 |

Bread, WG = Bread, Wholemeal/grain; Meat, CNO = Meat, cooked, no oil

**Table 9. Daily intake of top ten individual foods for ten year olds at baseline (child10) and final (child12) summed and ranked by IOTF obesity classification. (N=178)**

| | Child10 | | | | Child12 | | | |
|---|---|---|---|---|---|---|---|---|
| | Not obese (n=156) | | Obese (n=22) | | Not obese (n=153) | | Obese (n=25) | |
| Sum Rank | Food | Mean daily consumption | Food | Mean daily consumption | Food | Mean daily consumption | Food | Mean daily consumption |
| 1 | Water | 1.70 | Water | 1.34 | Vegetables | 5.40 | Vegetables | 5.10 |
| 2 | Fruit | 1.29 | Butter | 1.07 | Fruit | 4.35 | Fruit | 4.24 |
| 3 | Vegetables | 1.28 | Fruit | 0.99 | Water | 1.64 | Water | 1.59 |
| 4 | Bread, White | 0.91 | Vegetables | 0.88 | Butter | 0.87 | Butter | 0.83 |
| 5 | Butter | 0.91 | Bread, White | 0.81 | Bread, White | 0.80 | Bread, WG | 0.68 |
| 6 | Milk, Standard | 0.76 | Milk, Standard | 0.66 | Milk, Standard | 0.65 | Bread, White | 0.60 |
| 7 | Biscuits | 0.70 | Crisps | 0.55 | Bread, WG | 0.63 | Sauce, Tomato | 0.49 |
| 8 | Drink, Powdered | 0.50 | Vegemite | 0.53 | Biscuits | 0.60 | Crisps | 0.46 |
| 9 | Bread, WG | 0.48 | Bread, WG | 0.50 | Sauce, Tomato | 0.44 | Meat, CNO | 0.45 |
| 10 | Crisps | 0.48 | Peanut Butter | 0.50 | Peanut Butter | 0.43 | Milk, Trim | 0.45 |

Bread, WG = Bread, Wholemeal/grain; Meat CNO = Meat, cooked, no oil

Active games, outdoor play and biking were undertaken on average once every two or three days by child5 and child7 and once to twice a week by child10 and child 12. Swimming was popular in the child10 group. Whilst ball games, skateboarding and dance remained popular with child5 to child7, gymnastics and swimming appeared to wane in popularity over these two years. Organised sports such as soccer and rugby become more commonplace amongst child7 top ten activity lists but do not appear in child5 lists.

Outdoor chores, gymnastics and swimming are ranked lower in final assessment for child12 than child10, whereas ball games and indoor chores become the more common activities over time. As with child7, child12, but not child10, children appear to be involved in more organized sport as evidenced by the appearance of rugby and basketball in the child12 top ten activity list.

The lists in Table 10 and Table 11 show that not obese children tended to be more active than obese in all age groups. There were few differences in choices of activities between child5 obese and not obese at baseline and final assessment. Data from Table 11 suggest that child10 and chld12 obese children spend more time indoors (for example, longer duration indoors chores, and shorter duration outdoors chores and outdoors play).

Girls tended to be less active than boys across age and time-points (child5: 1.94 vs 2.47 times per day; child7: 1.53 vs 1.92 times per day; child10: 1.71 vs 1.95 times per day; child12: 1.34 vs 1.76 times per day, respectively). Total physical activity frequency reduced overall from child5 and child10 to child7 and child12 respectively. Active games, ball games, biking, outdoor play and swimming were consistently included in the top ten activities for both sexes and at all time points. Gymnastics was a top ten activity for both sexes until the child12 time-point. Similarly outdoor chores were frequently participated in for all but child7. Skateboarding was a popular top ten activity for both sexes except boy10 and girl12. Girls were slightly more consistent, than boys, in their top ten activities participating in five common activities (boys = four activities) across all four groups (two age groups x two time-points = four groups) and four activities in three out of the four groups (boys = three activities).

Dance was almost an exclusive top ten activity for all girls with the exception of boy5's. From the first age group (child5), boys engaged in organised sports more than 0.05 times per day (more than once a month), whereas girls only attained this frequency of participation, in organised sports, at the child12 time-point. The organised sports for

young boys were soccer and rugby, and although rugby continued to be a popular sport with the older boys, it was basketball, not soccer that was ranked in the top ten activities for child10 and child12.

The mean total reported weekly sleep duration was much less in older children than the younger children in both age groups (child5 = 79.00 hours vs child10 = 73.75 hours; child7 = 77.50 hours vs. child12 = 72.25 hours). Younger children tended to sleep longer during weekends (child5 = 11.50 hours; child7 = 11.25) than weekdays (child5 = 11.00 hours; child7 = 10.75 hours) but older children had similar mean weekday and weekend hours (child10 = 10.50 vs.10.50 hours; child12 = 10.50 vs. 10.25 hours). Younger girls reported longer mean total weekly sleeping duration than younger boys (Girl5 = 79.50 hours vs. boy5 = 78.75 hours; girl7 = 78.25 vs.boy7 = 77.00), but this phenomenon was reversed in the older age group (Girl10 = 71.75 hours vs. boy10 = 73.00; girl12 = 72.50 vs. boy12 = 74.50).

Programme children reported slightly less mean total weekly sleep than control children at every instance (Table 14) and mean daily weekday and weekend hours were no different than 15 minutes (0.25 hours). Similarly, marginally less mean total weekly sleep hours were reposted by obese children as compared with not obese children (Table 14). Typically, a difference of no more than 15 minutes separated the daily (weekday and weekend day) sleeping hours of not obese and obese children.

Figure 4 displays the differences in time to bed during week days and weekend days between age groups. Obese children report slightly later time to bed to not obese children. All children reported an earlier time to bed during weekend days than week days.

**Table 10. Summed total and ranked top ten daily physical activities for child5 and child7 by intervention group. Mean average of each activity provided. (N=440)**

| | Child5 | | | | Child7 | | | |
|---|---|---|---|---|---|---|---|---|
| | Control (n=199[a]) | | Programme (n=220[a]) | | Control (n=170[a]) | | Programme (n=180[a]) | |
| Sum Rank | Activity | Mean daily frequency | Activity | Mean daily frequency | Activity | Mean daily frequency | Activity | Mean daily frequency |
| 1 | Active games | 0.44 | Active games | 0.44 | Active games | 0.37 | Active games | 0.37 |
| 2 | Outdoors play | 0.38 | Outdoors play | 0.39 | Outdoors play | 0.30 | Outdoors play | 0.32 |
| 3 | Biking | 0.35 | Biking | 0.31 | Biking | 0.24 | Biking | 0.24 |
| 4 | Gymnastics | 0.29 | Gymnastics | 0.25 | Ball games | 0.14 | Skateboarding | 0.14 |
| 5 | Skateboarding | 0.14 | Swimming | 0.18 | Skateboarding | 0.11 | Soccer | 0.14 |
| 6 | Swimming | 0.13 | Skateboarding | 0.14 | Dance | 0.11 | Ball games | 0.13 |
| 7 | Dance | 0.10 | Ball games | 0.10 | Soccer | 0.10 | Rugby | 0.11 |
| 8 | Ball games | 0.11 | Dance | 0.09 | Swimming | 0.09 | Dance | 0.09 |
| 9 | Outdoors chores | 0.09 | Outdoors chores | 0.06 | Gymnastics | 0.07 | Swimming | 0.07 |
| 10 | Organised exercise | 0.07 | Organised exercise | 0.05 | Rugby | 0.06 | Gymnastics | 0.08 |

[a]number of participants that completed activity questions – some missing data

**Table 11. Summed total and ranked top ten daily physical activities for child10 and child12 by intervention group. Mean average of each activity provided. (N=178)**

| | Child10 | | | | Child12 | | | |
|---|---|---|---|---|---|---|---|---|
| | Control (n=105[a]) | | Programme (n=67[a]) | | Control (n=97[a]) | | Programme (n=50[a]) | |
| Sum Rank | Activity | Mean daily frequency | Activity | Mean daily frequency | Activity | Mean daily frequency | Activity | Mean daily frequency |
| 1 | Active games | 0.35 | Active games | 0.32 | Active games | 0.25 | Biking | 0.26 |
| 2 | Outdoors play | 0.23 | Swimming | 0.23 | Biking | 0.21 | Active games | 0.18 |
| 3 | Biking | 0.22 | Biking | 0.22 | Outdoors play | 0.18 | Outdoors play | 0.16 |
| 4 | Swimming | 0.20 | Outdoors play | 0.18 | Ball games | 0.15 | Indoors chores | 0.13 |
| 5 | Gymnastics | 0.19 | Gymnastics | 0.17 | Swimming | 0.11 | Ball games | 0.14 |
| 6 | Ball games | 0.13 | Ball games | 0.12 | Indoors chores | 0.10 | Rugby | 0.11 |
| 7 | Outdoors chores | 0.08 | Organised exercise | 0.10 | Rugby | 0.10 | Outdoors chores | 0.09 |
| 8 | Dance | 0.07 | Indoors chores | 0.09 | Dance | 0.09 | Swimming | 0.08 |
| 9 | Rugby | 0.07 | Outdoors chores | 0.08 | Basketball | 0.08 | Tennis | 0.08 |
| 10 | Organised exercise | 0.06 | Skateboarding | 0.08 | Tennis | 0.07 | Skateboarding | 0.08 |

[a]number of participants that completed activity questions – some missing data

**Table 12. Top ten daily physical activities for child5 and child7 by summed total and ranked. Mean average of each activity provided. N=440**

| | Child5 | | | | Child7 | | | |
|---|---|---|---|---|---|---|---|---|
| | Not obese (n=393[a]) | | Obese (n=26[a]) | | Not obese (n=313[a]) | | Obese (n=33[a]) | |
| Sum Rank | Activity | Mean daily frequency | Activity | Mean daily frequency | Activity | Mean daily frequency | Activity | Mean daily frequency |
| 1 | Active games | 0.45 | Active games | 0.35 | Active games | 0.37 | Active games | 0.32 |
| 2 | Outdoors play | 0.39 | Biking | 0.34 | Outdoors play | 0.32 | Biking | 0.23 |
| 3 | Biking | 0.33 | Outdoors play | 0.30 | Biking | 0.24 | Outdoors play | 0.21 |
| 4 | Gymnastics | 0.27 | Gymnastics | 0.26 | Soccer | 0.13 | Gymnastics | 0.12 |
| 5 | Swimming | 0.15 | Swimming | 0.19 | Skateboarding | 0.12 | Skateboarding | 0.12 |
| 6 | Skateboarding | 0.14 | Outdoors chores | 0.10 | Ball games | 0.13 | Ball games | 0.13 |
| 7 | Dance | 0.10 | Skateboarding | 0.09 | Dance | 0.10 | Swimming | 0.09 |
| 8 | Ball games | 0.10 | Ball games | 0.08 | Rugby | 0.09 | Dance | 0.07 |
| 9 | Outdoors chores | 0.07 | Dance | 0.04 | Swimming | 0.08 | Tennis | 0.08 |
| 10 | Organised exercise | 0.06 | Rugby | 0.02 | Gymnastics | 0.07 | Soccer | 0.06 |

[a]number of participants that completed activity questions – some missing data

**Table 13. Top ten daily physical activities for child10 and child12 by summed total and ranked. Mean average of each activity provided. N=178**

| | Child10 | | | | Child12 | | | |
|---|---|---|---|---|---|---|---|---|
| | Not obese (n=152[a]) | | Obese (n=21[a]) | | Not obese (n=126[a]) | | Obese (n=21[a]) | |
| Sum Rank | Activity | Mean daily frequency | Activity | Mean daily frequency | Activity | Mean daily frequency | Activity | Mean daily frequency |
| 1 | Active games | 0.35 | Swimming | 0.24 | Biking | 0.24 | Indoor chores | 0.21 |
| 2 | Biking | 0.22 | Active games | 0.23 | Active games | 0.23 | Active games | 0.19 |
| 3 | Outdoors play | 0.21 | Biking | 0.21 | Outdoors play | 0.18 | Ball games | 0.18 |
| 4 | Swimming | 0.21 | Outdoors play | 0.20 | Ball games | 0.14 | Biking | 0.14 |
| 5 | Gymnastics | 0.19 | Ball games | 0.13 | Rugby | 0.11 | Swimming | 0.12 |
| 6 | Ball games | 0.12 | Gymnastics | 0.13 | Indoor chores | 0.10 | Skateboarding | 0.13 |
| 7 | Outdoors chores | 0.08 | Dance | 0.11 | Swimming | 0.09 | Dance | 0.12 |
| 8 | Organised exercise | 0.07 | Indoor chores | 0.10 | Dance | 0.08 | Rugby | 0.10 |
| 9 | Skateboarding | 0.07 | Outdoors chores | 0.10 | Basketball | 0.07 | Outdoors play | 0.10 |
| 10 | Rugby | 0.06 | Organised exercise | 0.09 | Outdoors chores | 0.07 | Outdoors chores | 0.10 |

[a]number of participants that completed activity questions – some missing data

**Table 14. Mean average sleep duration for five and ten year olds at baseline and final assessment by intervention and by IOTF criteria obesity classification (N=618).**

| | Child5 | | Child7 | | Child10 | | Child12 | |
|---|---|---|---|---|---|---|---|---|
| | Control (n=210) | Programme (n=230) | Control | Programme | Control (n=108) | Programme (n=70) | Control | Programme |
| Total Weekly Sleep (hours) | 78.50 | 77.25 | 79.50 | 77.75 | 74.00 | 72.25 | 73.50 | 72.00 |
| Weekday Sleep (hours) | 11.00 | 10.75 | 11.25 | 11.00 | 10.50 | 10.50 | 10.50 | 10.50 |
| Weekend Sleep (hours) | 11.25 | 11.25 | 11.5 | 11.25 | 10.50 | 10.25 | 10.50 | 10.25 |
| | Not obese (n=413) | Obese (n=27) | Not obese | Obese | Not obese (n=156) | Obese (n=22) | Not obese | Obese |
| Total Weekly Sleep (hours) | 79.25 | 77.75 | 73.75 | 73.50 | 77.75 | 76.75 | 72.25 | 71.50 |
| Weekday Sleep (hours) | 11.25 | 11.00 | 10.50 | 10.75 | 10.75 | 10.75 | 10.50 | 10.50 |
| Weekend Sleep (hours) | 11.50 | 11.25 | 10.50 | 10.50 | 11.25 | 11.00 | 10.25 | 10.00 |

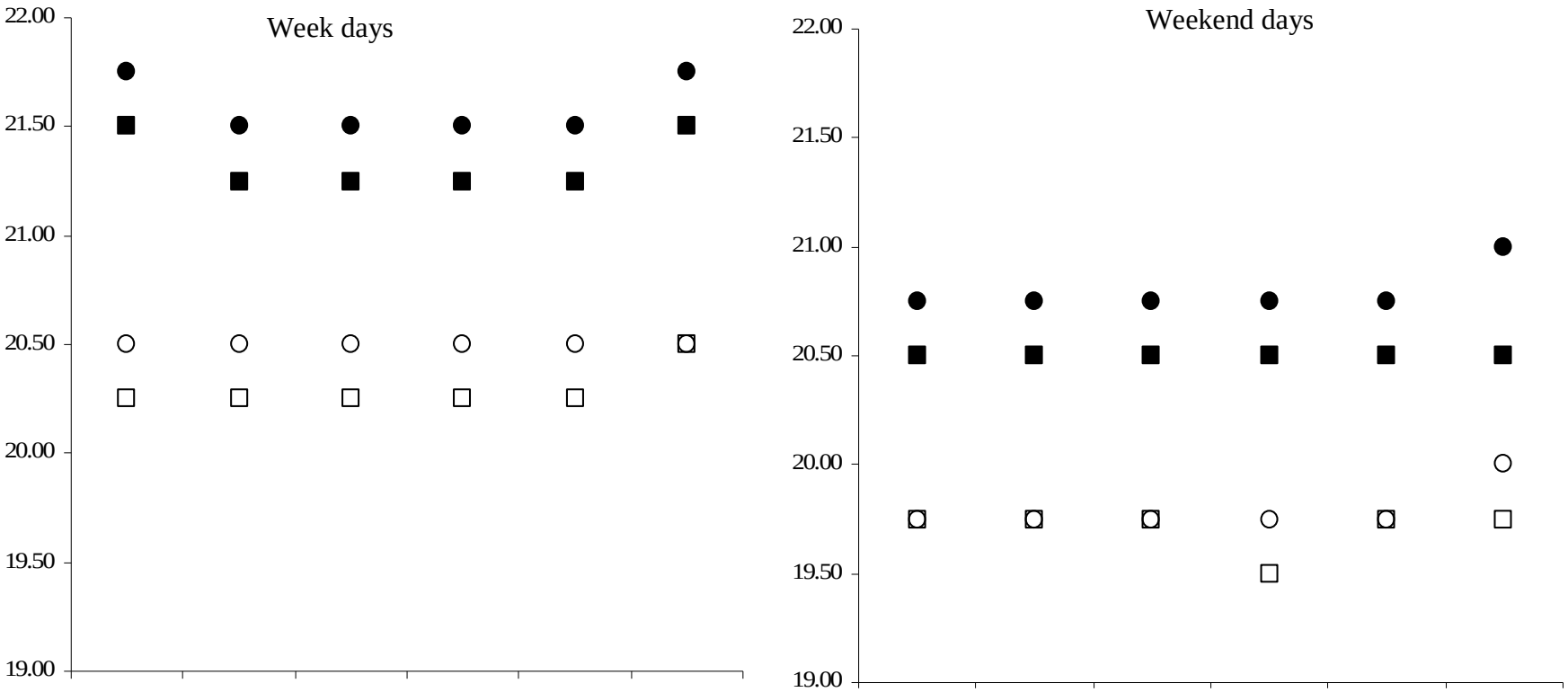


**Figure 4. Time to bed during week days and weekend days of boys and girls, control and programme, and not obese and obese by baseline and final assessment of five year olds (child5 and child7) and ten year olds (child10 and child12).**

### 3.6.5 Tracking of Food and Physical Activity patterns

All baseline food pattern score variables were significantly correlated ($p$<0.001) with their corresponding final result for both age groups which demonstrates good internal reliability of the questionnaire and tracking of dietary information (See Table 15). Similarly, all baseline physical activity pattern variables were significantly correlated ($p$<0.001) with their corresponding final result for both age groups which demonstrates good internal reliability of the questionnaire and tracking of physical activity information (See Table 16).

There were no Spearman's rho correlations of any food pattern scores with BMI SDS for child5 or child7; however final PBF SDS was significantly correlated with baseline and final healthy food (rho=-0.139, $p$=0.004; rho=-0.14, $p$=0.003) and vegetable intake (rho=-0.145, $p$=0.003; rho=-0.132, $p$=0.005). Child5 PBF SDS was inversely correlated with baseline healthy food (rho=-0.12) and vegetable intake (rho=-0.12) however the relationships were not significant (p=0.017 and p=0.010 respectively). However, Child10 and child12 BMI SDS was significantly negatively related to baseline vegetable intake (rho=-0.20, $p$=0.007; rho=-0.25, $p$=0.001).

**Table 15. Correlation coefficient of baseline food frequency variables with their respective final frequency variables for child5 (n=440) and child10 (n=178)**

| Baseline variables | Final variables 5 year olds | Final variables 10 year olds |
|---|---|---|
| Carbohydrate foods | 0.575* | 0.556* |
| Fast foods | 0.597* | 0.568* |
| Fat foods | 0.563* | 0.431* |
| Healthy foods | 0.461* | 0.497* |
| Milk | 0.422* | 0.477* |
| Fruit | 0.415* | 0.460* |
| Vegetables | 0.343* | 0.338* |

*All correlates (Spearman's rho) significant p<0.01

Physical activity variables were not correlated with any measure of BMI and PBF SDS for child10 or child12. However, child7 BMI and PBF SDS were negatively correlated with total weekly sleep (rho=-0.14, *p*=0.002; rho=-14, *p*=0.002) and weekday sleep hours (rho=-0.15, *p*=0.001; rho=-0.15, *p*=0.002). Baseline physical activity was negatively correlated with child5 and child7 PBF SDS only. Additionally, child7 PBF SDS was positively related to baseline physical inactivity promoters (rho=0.12, *p*=0.00998) and final physical inactivity (rho=0.15, *p*=0.001).

**Table 16. Correlation coefficient of baseline activity scores with their respective final variables for child5 and child10 (n=618)**

| Baseline variables | Final variables 5 year olds | Final variables 10 year olds |
|---|---|---|
| Physical activity promoters | 0.61* | 0.70* |
| Physical activity | 0.44* | 0.45* |
| Physical inactivity promoters | 0.47* | 0.64* |
| Physical inactivity | 0.72* | 0.75* |
| Total weekly sleep | 0.66* | 0.63* |
| Weekday sleep | 0.58* | 0.56* |
| Weekend sleep | 0.61* | 0.55* |

*All correlates (Spearman's rho) significant p<0.01

### 3.7 Discussion

This study shows that for the 618 children whose parents completed questionnaires and had anthropometric measurements made in 2004 and 2006 in Project Energize:

1. prior status of body mass, BMI SDS or PBF SDS, tracked over two years and was the major determinant of change of body mass status of these children.
2. child5 Māori increased in BMI SDS and PBF SDS at a greater rate than their European peers.
3. obese children appeared to consume similar foods to not obese children and, in general, displayed a tendency to consume fruit and vegetables less often and high fat/sugar more often. This was especially evident in older children.
4. obese children reported less physical activity participation than the not obese children. Additionally, the study data suggested the obese children spent more time indoors than outdoors.
5. obese children slept less than their not obese peers. Sleep was related to BMI SDS and PBF SDS in younger children but not older children.

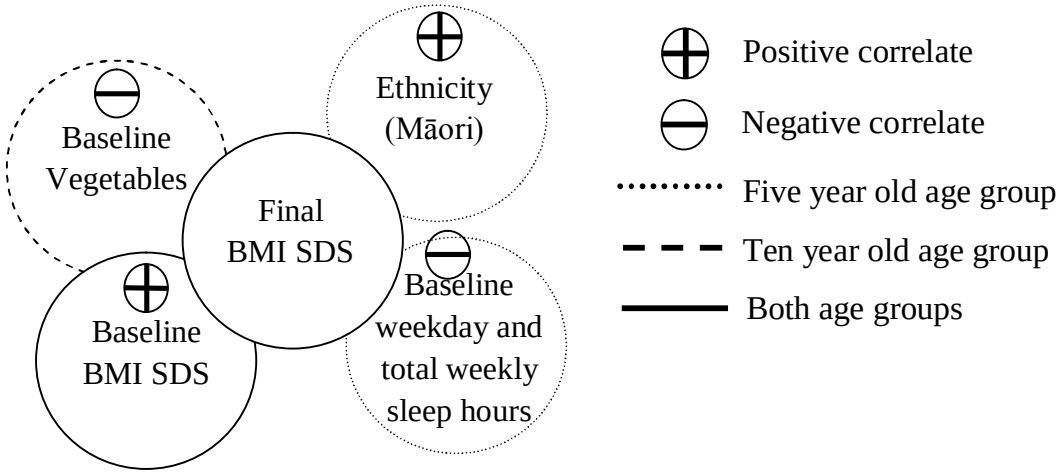


**Figure 5. Demographic, food and physical activity factors that may contribute to final BMI SDS. Where circles touch or arrows are used indicates relationships.**

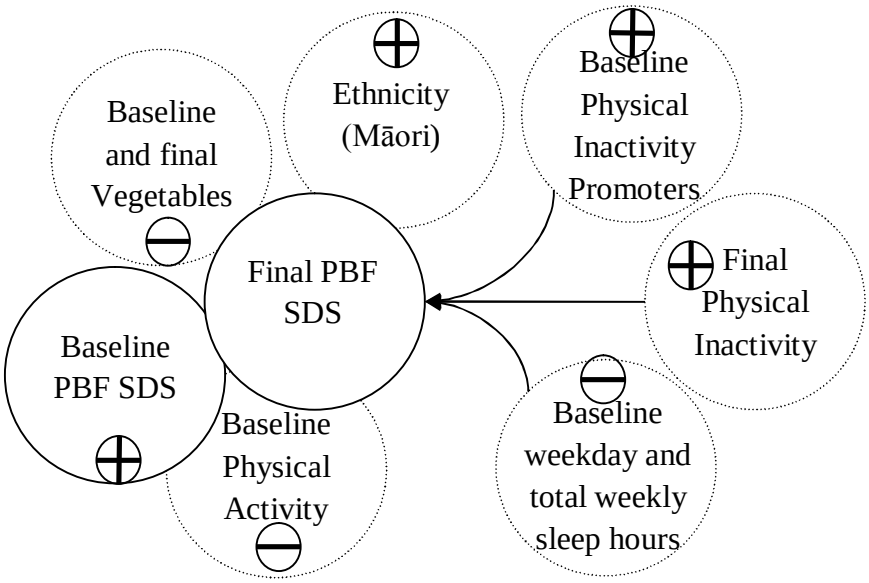


**Figure 6. Demographic, Food and Physical Activity factors are related to final PBF SDS. Where circles touch or arrows are used indicates relationships.**

Further observations were:

1. Child5 and child7 BMI SDS was associated with physical activity variables and PBF SDS was related to food pattern variables. Child10 and child12 BMI SDS was associated with food patterns but not to physical activity patterns. PBF SDS was not associated with any food or physical activity variable.
2. The pattern of food consumption and selection of physical activities tracked over the two years.
3. Older children selected team sports more often than younger children. Girls participated less in team sports than boys at an earlier age.

The pattern of all associations with BMI SDS and PBF SDS have been presented schematically in Figure 5 and Figure 6 respectively. The associations with PBF SDS appear to be more complex/comprehensive indicating changes in PBF SDS may be a more sensitive measure of influences of food, physical activity and sleep.

**Sleep negative and BMI**

### 3.7.1 Tracking of BMI and PBF SDS

As evidenced in this study, body mass status (BMI and PBF SDS) at a younger age tracks and is the best predictor of body mass status of the child two years later. This tracking has been shown in other children. The initial BMI of Chinese children (6 to 13 years old) was also a significant predictor of their BMI 6 years later (Wang, et al., 2000). Similarly Blair et al. (2007) in the Auckland Birth Cohort found that PBF at 3.5

years of age was significantly related to PBF at 7 years of age (r=0.67). These authors also showed that birth weight was positively associated with BMI and also PBF at 9 months, 3.5y and 7 y. The Blair investigation was confined to European children. The data presented here from Project Energize adds to this knowledge that between ages 5 and 7 (rho = 0.68) and 10 and 12 (rho = 0.62) PBF SDS scores also track strongly for both Māori and European children. These findings lend support to the proposition that interventions should be present during the entire lifespan (Blair, et al., 2007), improving the adult BMI and PBF outcome for very young children.

Māori consistently record a higher BMI for age than European children in New Zealand (Ministry of Health, 2008b). This study has shown that the contribution of ethnicity to BMI SDS and PBF SDS wanes as children become older. This may indicate that the difference between Māori and European is primarily due to early environmental factors (for example, food and physical activity) rather than hereditary (for example, genetic) factors.

### 3.7.2 Tracking of food and physical activity behaviour patterns

Patterns (frequency) of food and physical activity were recorded from parental recall in a questionnaire at baseline and final stages. Food choices were similar between age groups and over time, which may indicate the influence of parental control of children's access to food. The stability of food choice also suggests target foods of interest for those who wish to influence food intake of children. Baseline vegetable intake was found to be inversely related to child7 PBF SDS and child10 and child12 BMI SDS. Final vegetable intake was also inversely related to child7 PBF SDS.

Mean daily consumption of foods for child5 did not appear to differ markedly between programme and control group. Mean fruit and vegetable frequency did not exceed 2.83 times per day, well below the dietary guidelines of 3 servings of vegetables and 2 servings of fruit per day. The HHQ2 gathered much more detail regarding fruit and vegetable frequency and exceeded 10 times per day. Similarly child10 participants mean vegetable and fruit intake did not exceed 2.99 times per day, however their Mean daily consumption exceeded 9 times per day two years later. These differences may be an artifact of a more detailed section about fruit and vegetables intake in the followup questionnaire, HHQ2.

Within the child10 and child12 groups, physical activity variables did not register any relationships with BMI or PBF SDS. This may be an effect of the recall tool used to record physical activity. Sleep was significantly related to child7 BMI and PBF SDS.

Additionally, baseline physical activity promoters and final physical inactivity were also related to child7 PBF SDS. A far reaching relationship was found in baseline physical activity which was inversely related to both child5 and child7 PBF SDS.

Total activity at baseline did not exceed a daily mean of 2.5 times per day betweeb control and programme groups of child5 and at child7 mean daily activity did not exceed 1.8 times per day. For the older children mean daily activity was worse, 1.9 times per day at baseline dropping off to 1.5 times per day at final assessment. One drawback of this activity frequency measure is the inability to estimate duration of activity. Thus no attempt to align these study results with current phsyical activity recommendations has been made.

There was little difference in dietary patterns between Māori and NZ European children in this study. Māori consumd more carbohydrate foods, and slightly more fast foods, but similar frequencies of fatty foods than New Zealand European.Young low decile children (child 5) consumed less fruit and vegetables (one unit less per day) but there were no other dietary patterns differences found. It does not appear that low decile and Māori children have a much worse nutritional intake than New Zealand European as described in the New Zealand Health Survey (Ministry of Health, 2008b)

The relationships found in the data of this study add to the complexity of the problem of weight gain, however valuable insights were obtained. Tracking of body mass status was confirmed with strong results and ethnicity appeared to be only related to BMI SDS in the younger age group. The tools to record food and physical activity patterns may not have been robust, but patterns emerged to show that early food and physical activity are related to body mass status. Both physical activity and inactivity are related to PBF SDS and sleep is related to growth (BMI and PBF SDS) in young children but not in older children. These insights contribute to the growing evidence that weight gain in children should be treated holistically and that no one factor may be responsible.

Food patterns of food frequency and activity patterns of physical activity frequency and inactivity duration were related to the younger age group body mass SDS moreso than the older children. Previous studies have shown ambiguous results for the influence of food patterns in obesity. In this study, the significant relationships between food and obesity were weak and varied dependent on age group, sex, and obesity classification (BMI and PBF SDS), however they were present and should not be discounted in future research. What are lacking are appropriate and precise tools to assess food intake. The use of objective measures of physical activity may produce stronger results as shown in

other studies (Christodoulos, Flouris, & Tokmakidis, 2006; Reilly, Kelly, et al., 2005; Rennie et al., 2005). This global and indirect relationship is representative of the complex interaction that is obesity in children. Additionally, the environmental aspect, such as access to activity promoters and removal of physical inactivity promoters may significantly reduce obesity prevalence (Trost, et al., 2001).

Food choices were similar over time and between age groups suggesting target foods for social marketing to parents or behavioural interventions for choices made by children. The similarity over time confirmed Theodore et al.'s (2006) finding that foods consumed pre-school New Zealand children did not differ to school-aged children and that food patterns are started early.

Physical activity in both age groups displayed a trend to team sports even though the top five activities were the same. This trend may be phenomenon of general through-school physical education policy moving toward organised sports as a physical activity alternative than demonstrating individual choice. There were several instances of decile and ethnic differences: Māori were more significantly more sedentary (more screentime) but participated in slightly more physical activity than New Zealand European children which has been highlighted in recent national survey data (Ministry of Health, 2008b). A higher proportion of New Zealand European children are in high decile schools so it followed that children in high decile schools were also less sedentary than mid or low decile children.

Decile and ethnicity show strong links with BMI SDS and PBF SDS and these links are borne out in other published research (Cecil et al., 2005; Danielzik et al., 2004; E. Duncan et al., 2004; Kumanyika, 2008; Lamerz et al., 2005; S. J. Taylor et al., 2005). However regression analysis failed to identify decile as a predictor of either BMI or PBF. Ethnicity was the only demographic variable that was found to explain a proportion of child7 BMI SDS. With the admixture of ethnicities in New Zealand it may be faulty logic to classify these children into European or Māori as they potentially could have mixed biological lineage. Add to this, that there is an ethnic cultural aspect that may be modified by family culture. These factors of admixture and family culture may render the use of ethnicity in this type of research ineffective. It might be more prudent to delve more deeply into the family structure and culture to ascertain their attitudes and behaviour around food and activity.

Habitual sleep duration links with obesity has been a topic that has drawn much attention (Horne, 2008; Knutson & Van Cauter, 2008). Complex interactions may also

be at work regarding habitual sleep duration and propensity towards obesity. This study observed the significant regression relationship of sleep with BMI in baseline five year olds however this relationship is no longer present at final assessment (child7) or in ten year olds (child10 and child12). The results show that there is never more than 30 minutes difference in daily sleep quotas between obese and non-obese children, no matter which age group or assessment period (baseline or final), and that this difference is not always significant. This small difference is unlikely to be the basis for a physiological pathway that produces increased adiposity or insulin resistance as daily differences in experimental studies are much larger (Horne, 2008). The alternative underlying funcational pathway that is suggested in this data is that obese children may be either staying up longer or sleeping less or both and this leads to less energy to participate in physical activities. In contrast, children that are more active may sleep better and longer (Gupta, Mueller, Chan, & Meininger, 2002). Without strong evidence of this pathway, the concept is supposition only, however based on the current evidence it is likely that this is the norm in most cases. Although Spearman's correlations in this study revealed that sleep was linked to physical inactivity and BMI and PBF SDS, the relationships were fleeting and variable. This data contradicts current published work (Chaput, et al., 2006; Duncan, et al., 2008; Nixon, et al., 2008; Reilly, Armstrong, et al., 2005). However, so little research has been conducted in this area of sleep and obesity that all data will be helpful to draw a comprehensive picture of the issue.

### 3.7.3 Strengths and limitations

Strengths of this study are the large number and variety of variables, quantity of children and two age groups followed over two years which provide a wider and more comprehensive picture of the data. The concurrent limitation is that a large number of statistical tests have been applied but by applying a 0.01 level of significance this limitation should have been reduced (Krebs et al., 2007). The rate of growth of children is not linear and differs by age and sex. This difficulty has been overcome by comparing these children to reference growth curves and BMI SDS and PBF SDS were derived. This also allows comparison with other populations. Particularly the use of the PBF SDS is a significant step to demonstrating the differences in growth between age groups and ethnicities. Weaknesses include missing data and methodological limitations of food and activity questions. Precision of activity data collection could have been overcome with objective measures of physical activity and inactivity, for example accelerometers. Food observations would have been difficult with this number of

participants and their spread throughout the region. The lifestyle behaviours investigated by the household questionnaire were reliant on parents having good knowledge of their child's daily habits, but their recall may not be reliable or accurate.

### 3.7.4 Summary

In summary, there is strong evidence that body mass status tracks from a very young age. Thus interventions should start with early age groups and continue into adolescence to have full impact. This may require programmes which follow a pre-natal to young adulthood life course. Further, despite the indications that food and activity patterns, representing the energy balance equation, in this study are partly responsible for the development of obesity, many environmental, cultural and hereditary factors act upon these patterns creating a complex picture. The importance of the environment especially access to physical activity and nutritious food and the removal of those factors that promote sedentary lifestyles is emphasised. The ambiguous food, activity and habitual sleep duration results in this study may be due to the inaccuracy that is derived from self-reported recall lifestyle questionnaires which highlights the need for objective, and therefore more precise, tools to conduct this type of research.

Thus to prevent obesity, the most appropriate intervention setting may need to be family-based, at home (Sothern, 2004), and as early in life as possible. School-based programmes may support the family-based intervention by providing an environment that also promotes healthful eating and activity in association with their peers. Healthful changes in food patterns of children is a recognised issue for the health and body size of children (Lioret, Touvier, Lafay, Volatier, & Maire, 2008) and Patrick and Nicklas (2005) suggest that the parent plays a vital role in making these healthful changes. Although an authoritative stance regarding food choices of children is not recommended for parents, providing a healthful environment where suitable choices may be accessible, and chosen, by the child could be an effective preventive measure for obesity.

# 4 Resting substrate utilisation and metabolic rate in young children: relationships with body composition and food and physical activity patterns

## 4.1 Background

This chapter describes the investigation of a possible relationship between resting metabolic rate and substrate oxidation, physical fitness and anthropometry, specifically body mass index (BMI) and percentage body fat (PBF).

The understanding of these relationships is important because children with higher fitness levels also show improved cardiovascular risk outcomes (lipid profile) and lower adiposity than their less fit peers (Andersen et al., 2008). Furthermore there is a lack of information about the context of the relationship of substrate oxidation lifestyle (for example, food, physical activity and inactivity and sleep patterns) and relative body size, that is, BMI and PBF z-scores (SDS).

In children, because of age-related changes in body proportions and composition, standardised BMI SDS are frequently utilised to allow comparisons adjusted for age and sex within the study group and with an external standard. However, PBF which is a measure of fat and lean mass rather than mass relative to height may also be expressed as SDS.

Based on the schematic proposed by Sun et al. (2004) (Figure 7) it is hypothesised that an unhealthy food pattern (high fat or carbohydrate) will be associated with a high BMI SDS (Bowman, Gortmaker, Ebbeling, Pereira, & Ludwig, 2004). In other words, It is Sun et al's hypothesis that weight gain from an unhealthy diet may be explained through elevated substrate utilisation which is represented by resting RER. Addtionally, physical fitness may have a modulating effect on resting RER and by extension, weight.

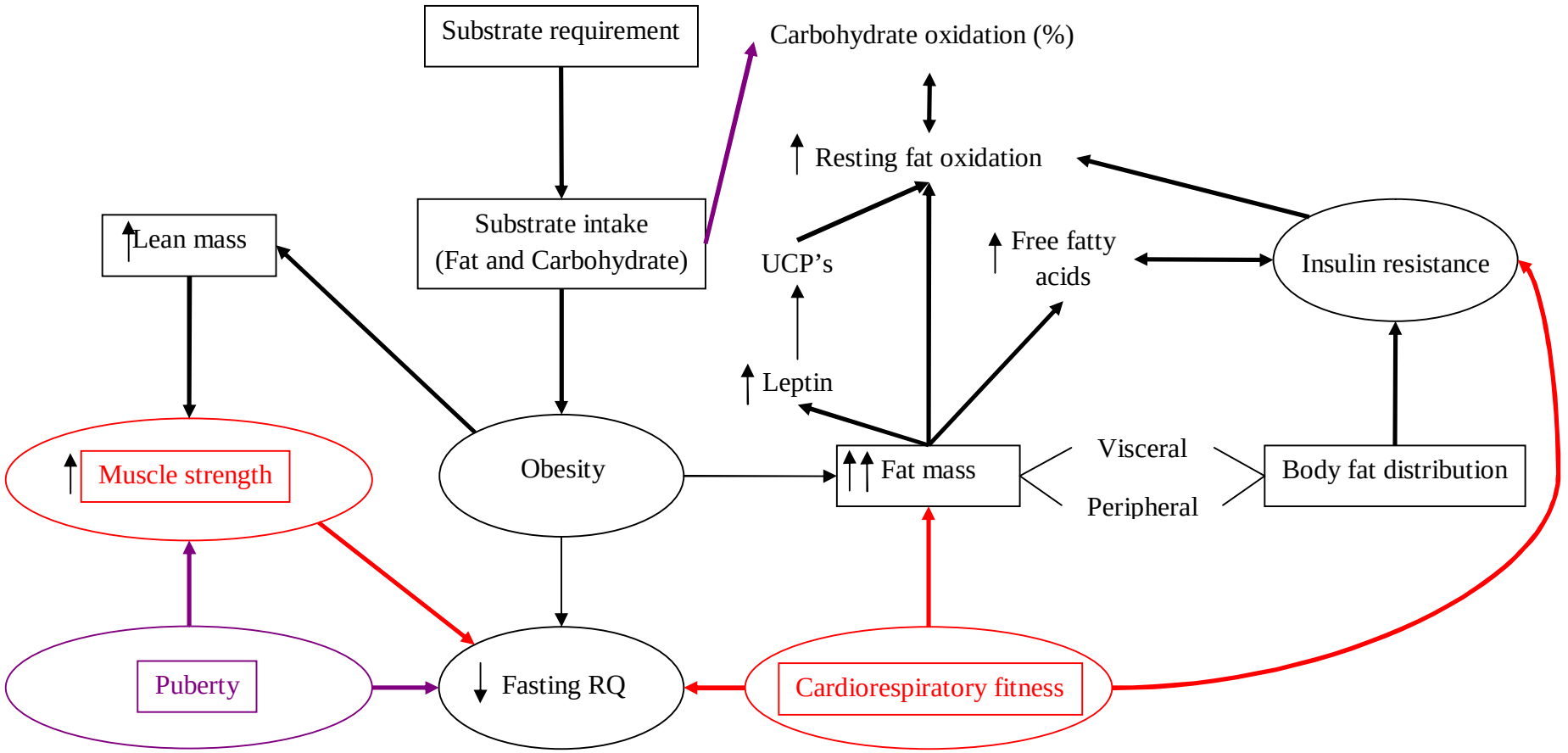


**Figure 7. Body composition, metabolic and hormonal responses regulating substrate balance.**
UCP, uncoupling protein; RQ, respiratory quotient. Adapted from Sun et al. (2004). Those items in black denote the original schematic and all other colours are added physiological pathways. Red = hypothesised, purple =confirmed.

Weight gain and obesity are a much more complex issue than the simple arithmetic of a positive energy balance when energy intake is greater than energy expenditure. Especially in children, who are growing and developing, there are many other changing physiological and environmental factors that confound the relationship between energy in and energy out (World Health Organisation, 2000). Energy in food is stored in the covalent bonds of the macronutrients; carbohydrate, fat and protein. This energy is released in a controlled way in the presence of oxygen by the cascade of enzyme controlled catabolic reactions that is intermediate metabolism. The end products of this oxidative process are carbon dioxide and water. In the resting and fasting state both the rate and type of substrates metabolized may be evaluated by indirect calorimetry. Indirect calorimetry is the measurement over time of the volume of expired air and the expired concentrations of carbon dioxide and oxygen. From this information both metabolic rate in terms of energy expended over time can be determined and the respiratory exchange ratio or RER, the ratio of carbon dioxide produced to oxygen utilized is derived. Over a long period of time RER indicates the proportion of glucose, protein or lipids that are metabolised. Fat has an RER of 0.7 and carbohydrate is 1.0. Over a short period, when the subject is fasted, oxidation of protein may be ignored (Frayn, 1983). Resting RER is measured after at least four hours of fasting and not exercising and has a physiologic range of 0.71 to 1.0 (Matarese, 1997), with a modal value of 0.85 or a 50:50 mix of carbohydrate and lipid utilisation (Flatt, 1995).

## 4.2 Literature review

Physiological moderators of weight gain are dependent on the interrelationships of body composition, substrates metabolized, macronutrient composition of foods consumed and metabolic pathways related to physical fitness and growth. A schematic showing the complexity of the interrelationships of these factors is provided in Figure 7. The following paragraphs will explore the literature that supports this model focusing on what is known about dietary fat, carbohydrate, weight gain as a measure of energy balance and possible pathways. The simplistic proposition is that a high RER infers carbohydrate burning and weight gain and a low RER is associated with fat burning and weight loss.

Several adult studies have identified a high RER as a potential predictor of weight gain in adults (Hainer et al., 2000; Marra, Scalfi, Contaldo, & Pasanisi, 2004; Seidell, Muller, Sorkin, & Andres, 1992; Valtuena, Salas-Salvado, & Lorda, 1997; Zurlo et al., 1990), but in children, few studies have been reported. In 72 prepubertal, eight to eleven

year old children, the obese (n=35) had a higher postabsorptive fat oxidation rate than the nonobese (31.4 +/- 9.7 mg/min vs 21.9 +/- 10.2 mg/min; $p < 0.001$) (Maffeis, et al., 1995). That study was followed by another Maffeis et al. article (1996) that found increased fat oxidation in eight to ten year olds children (N=82; obese=36.6%) was linked to a higher fat intake (as represented by percentage of energy intake). In another study, 48 pre-pubertal boys and girls (aged 4.8-11.4 years) categorised by BMI into overweight (>90th centile; bioimpedance body fat = 32.4 ±4.2%) and normal weight (bioimpedance body fat = 18.8 ±4.6%) no difference was found (0.88 ±0.04 vs 0.90 ±0.04) (Muller et al., 2002). It did not appear that RER had been included in further analysis for correlation or linear regression analysis of body size

In contrast, in 20 malnourished Kenyan babies and infants (5 to 79 months) fed a food that was 70% carbohydrate, resting RER was raised compared with post prandial RER (mean ±SD, 0.98 ±0.026 cf 0.94±0.022) (Duggan & Milner, 1986). This study has been included because it was the only study that clearly shows in the resting state that de novo lipogenesis can occur. Obesity may be considered a form of malnourishment by reasoning of lack of nutrients for adequate health (Eckhardt, 2006; Sherry et al., 2007).

The macronutrient mix of the food may be described by the food quotient (FQ) (Treuth et al., 2003; Westerterp, 1993) which over a long period of time should equal RER that is, for a stable weight all macronutrients consumed are oxidised if body composition does not change. However, Goris and Westerterp (Goris & Westerterp, 2000) were able to show that energy balance measured as change in body mass was positively related to RER, but not FQ. Although dietary carbohydrate determines short term carbohydrate oxidation (Westerterp, 1993) no such mechanism is in place for dietary fat (Prentice, 1998). Fat oxidation is less dependent on intake than carbohydrate oxidation because there is limited opportunity for carbohydrate storage as glycogen but relatively unlimited potential for fat to be stored. This has been proposed to explain the association between increased fat oxidation with higher fat mass (Westerterp, 1993).

Sun et al. (2004) have opined that it is the quality (glycemic index) and quantity of dietary carbohydrate that influences RER and, in turn, substrate utilisation. They suggest that a food high in simple carbohydrate would stimulate a high glucose utilisation and that it appears that dietary fat has no such short term effect on substrate utilisation. Ingestion of a food high in fat may result in increased fat oxidation, storage of fat which will increase fat mass and, in the long term, indirectly reduce carbohydrate utilisation by favouring insulin resistance (Figure 7). Few authors discuss the

phenomenon that high intake of carbohydrate may result in de novo lipogenesis and a resting RER over 1.0.

The relationship of RER and body mass in children has been seldom considered. Grund et al (Grund et al., 2000) compared the resting RER of overweight (OW) and obese (OB) children ($90^{th}$-$97^{th}$ and >$97^{th}$ body mass index [BMI] centiles, respectively) with non-overweight children and found no difference (OW 0.86 ±0.03, OB 0.86 ±0.1 and NO 0.89 ±0.04, $p$>0.05). This is in contrast to RER in "constitutionally" lean children as compared to constitutionally obese children (0.88 ±0.04 vs. 0.83 ±0.05, $p$=0.02 respectively) (Tounian, et al., 2003); supporting the observation in adults that there may be a familial influence on RER (Toubro, Sorensen, Hindsberger, Christensen, & Astrup, 1998). Both studies provide an insight into the resting RER:obesity relationship, however the use of different overweight and obesity classifications prevent a direct comparison. It is interesting to note that the OW and OB children in the Grund et al study have a slightly higher (non-significant) resting RER than the NO children whereas Tounian et al. found a significantly lower resting RER in their obese children (compared with lean children). Ultimately only Tounian et al. had the aim of investigating resting energy metabolism whereas the use of RER in the study for Grund et al appears to be for descriptive purposes.

Within obese children, RER can differ significantly (Perseghin et al., 2006). Resting RER was assessed in 54 obese children and those children with non-alcoholic fatty liver disease (NAFLD) were significantly higher than obese children with normal intra-hepatic fat (IHF) levels (0.83±0.08 vs. 0.77 ±0.05 $p$<0.01). But in non-obese children (6 to 9 years and 13 to 16 years) have no relationship of substrate utilisation to fat mass, puberty and fitness no mater the macronutrient composition of their diet (2003). Again, a different references for obesity classification were used for these studies to the ones highlighted in the previous paragraph. Despite this methodological difference, intra-hepatic fat accumulation may be a point of difference that affects RER. Fat mass (particularly hepatic) may need a tipping point to affect RER whereas in normal weight children their metabolic flexibility (of substrate utilisation) may prevent weight gain via de novo lipogenesis. Weight gain in metabolically flexible children may occur through a positive energy balance, whereas some obese children may no longer be metabolically flexible. Their weight loss could be hampered by liver fat and identified by RER. Comparison of these studies is extremely difficult, for example, all four studies utilise a different classification of overweight and obesity. Three use cross-sectional designs

which can only infer associations and to draw conclusions about pathways to weight gain via RER would be erroneous.

In 88 children, divided into tertiles of cardiorespiratory fitness determined by the ratio of oxygen uptake to pulse rate, substrate oxidation (RER) was not different (0.88 ±0.05 vs 0.86 (±0.05) vs. 0.87 (±0.05) (Grund et al., 2000). Treuth et al. (1998) also observed no difference in RER or substrate utilisation after five months strength training of 10 pre-pubertal girls (7 to 10 years). No other studies regarding fitness and resting RER in children could be found and this represents a gap in knowledge.

Substrate oxidation may be further modified by many factors including hereditary factors, possibly through human growth hormone mechanisms (Christiansen et al., 2002), sex (Treuth et al., 2003), resting serum free fatty acids and plasma lactate, fibre type and muscle (Goedecke et al., 2000) and liver glycogen concentrations (Stubbs, Murgatroyd, Goldberg, & Prentice, 1993). Resting RER of 24 children (13 to 16 years) was reduced after a high fat:low carbohydrate diet but increased after a high carbohydrate:low fat diet. A mechanistic explanation for the relationship between fat intake, fat oxidation and fat mass was proposed by Nistche et al. (2007). These authors were able to demonstrate with short term weight loss on a hypocaloric diet, both the appetite hormone ghrelin and resting RER increased and the concomitant rate of increase was correlated ($p$=0.029). This association may mean that low fat oxidation, and therefore low RER, in obese individuals may also signify a stimulated appetite (low ghrelin concentrations), explaining the tendency to consume a higher fat (high calorie) diet.

Curtailed sleep duration has also been touted as a mechanism that could affect appetite regulation via the orexin system as well as the Ghrelin and Leptin ratio (Taheri, Lin, Austin, Young, & Mignot, 2004). Additionally, sympathetic nervous system and cortisol and growth hormone secretion can also be activated (Spiegel et al., 2000; Spiegel, Leproult, & Van Cauter, 1999). Resting energy expenditure is hypothesised to be lower after an intensive period of sleep deprivation (Knutson & Van Cauter, 2008) however no studies have considered RMR or resting RER (or substrate utilisation) as a possible pathway for excess weight gain from chronically impaired sleep duration.

Very little is known about the relationships of substrate utilisation with body mass and composition, food patterns (carbohydrate and fat foods) and physical or metabolic fitness for children. Cross sectional data, which is often not adjusted for possible confounders and may utilise different approaches with the variables of interest, are the

only evidence to date. No longitudinal studies have looked at this question. The associations described in the review (above) make these relationships worth exploring further to provide insights into possible pathways of energy imbalance and inappropriate weight gain.

### 4.3 Aim

The aim of this study was to determine the interrelationships of body mass and percentage body fat with substrate oxidation as measured by resting respiratory exchange ratio (RER), resting metabolic rate (RMR), physical fitness (cardiorespiratory and strength) and self-reported activity and food patterns.

### 4.4 Hypotheses

1. Measured RMR values will be predicted by body mass (fat free mass and fat mass); furthermore RMR will be higher in those with greater physical fitness
2. Lower cardiorespiratory fitness adjusted for age and height (stride length) will be negatively associated with body mass (BM, FFM, FM) and an elevated RER at rest
3. Increased sleep, physical activity and fitness, and high fat foods including fast foods will be inversely associated with resting RER
4. Physical inactivity and carbohydrate food intake e.g. sugary drinks, will be positively associated with resting RER

Hypotheses two through four convey that a higher RER at rest, which represents higher carbohydrate oxidation, will be associated with increased mass for age. Further, a higher RER at rest will be explained by time spent in physical inactivity, carbohydrate food intake and decreased intake of fat foods, low physical activity/fitness and reduced sleep duration.

### 4.5 Method and process

Ethics approval was obtained through the Northern Region Y Ethics Committee (NTY/05/12/108) on the 27$^{th}$ of September 2006 and subsequently through the Auckland University of Technology Ethics Committee. Locality assessments and agreements were required from each participating primary school as part of the ethics approval process to demonstrate consultation and due diligence with the schools and their students. The principal and the board of trustees (BOT) of each school were petitioned for permission to conduct the study on school grounds. Once each locality

assessment was signed, it was then passed onto the Northern Y ethics committees' administrator for "running approval" to conduct the study. The data collection began on Friday the 31st of October 2006 and finished on the 14th of March 2007, which included a summer break of seven weeks.

### 4.5.1 The reality of conducting research in schools

The length of time to process the locality assessments from contacting the principal and meeting with them, allowing time for the study information to be presented to the school BOT's meeting and then for the locality assessment to be signed and returned to this investigator (GK) took three months. This included time delays because documents were lost by the schools in the petitioning process or there were further questions from the BOT. The most effective approach was found to be formal, repectful, friendly, prepared and face-to-face meetings. Correspondence by email or letter was invariably left aside and sometimes forgotten.

Key elements for a proposal to schools to be acceptable needed to put the priorities of the school, students and the parents first by showing respect at all times and avoiding any expense or time-wasting activities. When the research proposal was presented in an attractive manner to potential participants, their parents and schools, they were more likely to be recruited. Successful strategies included, the important work on the ground such as taking time to speak with school staff (reception, caretakers, and teachers), management (principals, deputies and senior teachers) and parents and their children. This investigator was provided with the excellent facilities and accommodating staff for every aspect of the research work that was undertaken the participating schools.

The initial proposal to the principals and BOT's included consent for blood sampling. The BOT's expressed a reservation that most parents would not be prepared to have blood samples to be taken from their child. Thus the study was modified to accommodate this reservation of schools and participants.

Of the six schools that initially agreed to participate, one school withdrew citing a busy schedule. The five primary schools in this study were in the Hamilton city metropolitan area and at the time of study, rated decile three or below (Table 16.) and were participating in a control or programme group in the Project Energize Programme (Graham, et al., 2008).

One of the original proposals for this study was to measure metabolic differences between control and intervention children at two time points in alignment with the

Project Energize intervention. The time delays, relatively small numbers recruited, inability to sample blood and preliminary findings in the main study (Chapter 3, p.50) that a trivial effect of the intervention on BMI SDS required a renegotiation of the aims of the research with participants, their caregivers, school staff, Project Energize staff, and the relevant ethics committees. Updated and frequent contact using the preferred communication mode of the recipient played a major role in allowing continued free-flowing access into the schools.

**Table 17. Demographic details of the five primary schools involved.**

| School | Control/ Intervention | Decile | Roll | NZ European | Maori | Pacific | Asian | Other |
|---|---|---|---|---|---|---|---|---|
| 1 | C | 1 | 328 | 16% | 71% | 4% | 5% | 2% |
| 2 | C | 3 | 594 | 56% | 35% | 4% | 2% | 0% |
| 3 | C | 2 | 285 | 34% | 48% | 2% | 14% | 1% |
| 4 | I | 2 | 547 | 38% | 48% | 5% | 7% | 2% |
| 5 | I | 2 | 150 | 37% | 51% | 5% | 5% | 2% |

### 4.5.2 Recruitment

Prior to obtaining informed consent, contact with parents was made by letter from the school principal who invited expressions of interest (EOI) from parents and included the study information sheet and consent form (Appendix E). Simultaneously, a notice informing that a research study was about to be held at the school and request for volunteers were also included in the school newsletter. Where possible, this newsletter notice was repeated multiple times to stimulate interest. Potential participants were canvassed, when permitted, by entering school classrooms and presenting the study to children.

The principal investigator observed the school staff strongly encouraging the children to have their parents read the take-home forms that were distributed. These were also the same forms that were mailed out with the letter from the principal. This strategy was undertaken following advice from participating schools that the children's caregivers were notoriously lax in the return of documents. Even if the intention by the caregiver was to return the documents, strong encouragement and constant reminders were needed. As a result one hundred and seventy two expressions of interest (EOI) were returned to school offices over a period of three months and a registry list of names was constructed with individual identification numbers allocated to each potential

participant. Each caregiver who returned an EOI was contacted by the principal investigator (GK) and invited to a group meeting at their respective school to discuss the study. The schools supported this action by providing a room to meet parents to discuss the research work.

It was apparent at the group meetings that the majority of caregivers wanted to participate however they neither had the time nor the inclination to read through a two page information sheet. Also they wanted to meet the people who would be interacting with their child. These issues enforced the importance of interpersonal relationships and trust-building in research work as a whole, not only in paediatric studies. The group meeting was also a third opportunity to distribute information sheets and consent forms. In addition, Household questionnaires (Appendix B and described in Table 33) were also distributed. At every stage of the study questions and discussions were encouraged, research equipment was displayed and its operation described. Caregiver contact and demographic details were confirmed at this time and, in almost every case signed consent forms were handed over at these meetings. Some caregivers could not attend the meetings and in these cases either a personal visit or explanation via the telephone was sufficient to encourage a signed consent form to be handed into the school office at the caregiver's convenience.

Once consent was received an appointment was made, usually no more than one week in advance. A follow-up phone call the evening before was made as a reminder for the appointment, the requirement for fasting and that the child could be later home the next day. Since there were a number of assessments required for each child and usually only one assessor, no more than 5 children per day could be studied. The participants were informed that they would be required for one day only, in the morning prior to school starting (usually 8.30 a.m.) and in the afternoon of the same day immediately after school finishing (usually 3 p.m.) for the fitness testing.

The convenience sampling resulted in more overweight male and obese children, but less overweight female children participating in this study. Therefore comparison with a recent national nutrition survey is not valid (Ministry of Health, 2003). This bias may be due to three factors 1) more athletic (fit) females were apt to volunteer, 2) a school effect – less overweight and obese females in some schools. Thirdly, a likely explanation for a lower number of overweight girls is the lack of intrinsic motivation to participate in a physical fitness study, because of social stigma of being overweight

compounded by a common social expectation that it is not feminine for girls to participate in physical tasks.

### 4.5.3 Fasting

Each child was requested to fast from their dinner meal in the evening (commonly between 5 p.m. and 7 p.m.) avoiding all food and drink except water until the breakfast provided post-assessment. Some children (n=8) had not fasted (when asked when and what they had last ate and drank) and in that case they were assessed anyway but also requested to make another appointment in a fasted state. The non-fasting measurements were not included in analyses.

### 4.5.4 Questionnaires

Household questionnaires were distributed to parents at the parent meetings, at the assessment appointments and through the school administration offices. The survey utilised was the “About your child” section, the same used in the Project Energize research study and contained questions of health, food and physical activity patterns and the time of the child going to bed and rising on weekdays and weekend days (Appendix C). By the end of the testing period, 49 (71%) of 69 had returned questionnaires with useable information. Not all these questionnaires were returned complete and this led to some missing data in the final analysis of section B. Thirty-two (46%) respondents completed all items in the questionnaire.

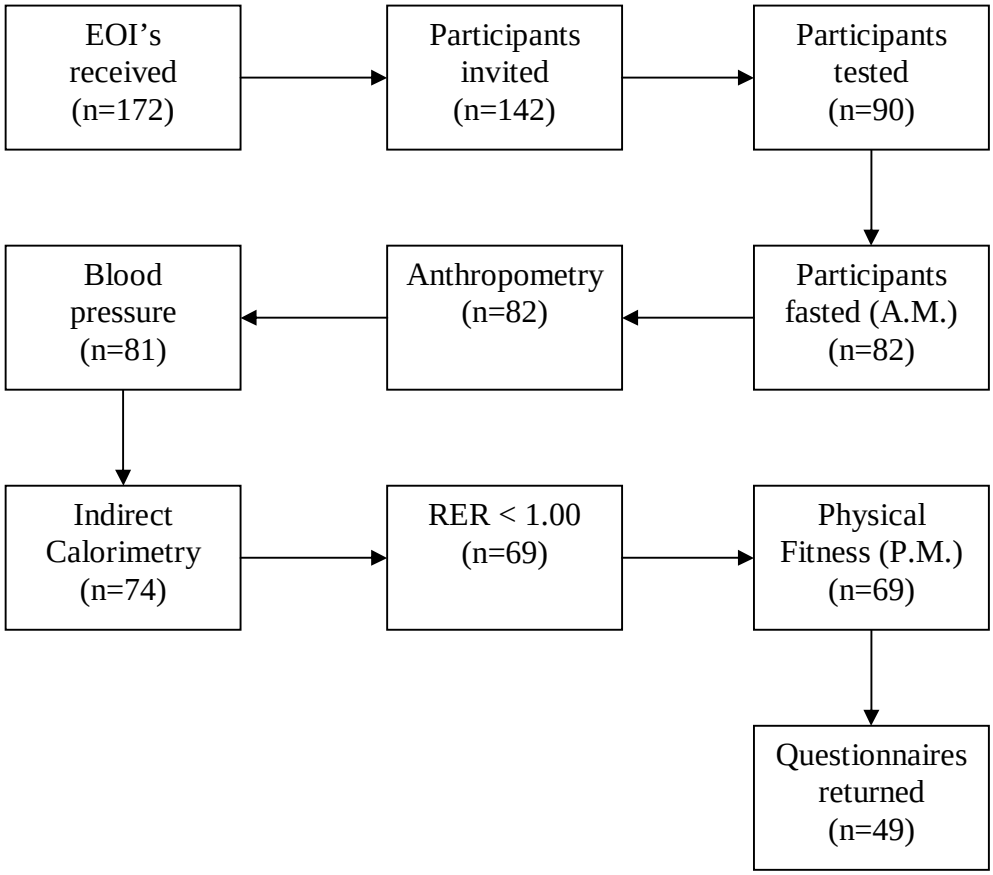


**Figure 8. Consort flowchart of measurement process. Anthropometry, BP and calorimetry assessment was carried out in the morning (A.M.) after an overnight fast and before school. Physical fitness assessment occurred directly after school in the afternoon (P.M.)**

### 4.5.5 Protocol of measurements

Each school made a room available for testing that was quiet and private. This was organised in the 'conduct of research' brief discussed with all participating schools before appointments with candidates were booked. Upon arrival in the morning each child was asked whether they had fasted since dinner the evening before and was requested to attend the rest rooms to void any urine or faeces. When returning to the assessment room, measurement of anthropometry, blood pressure and heart rate (cardiovascular), and indirect calorimetry (metabolic) aspects began. Once all morning tests were complete, breakfast was provided to the children (cereal, yoghurt and fruit). They were also reminded of the physical fitness testing in which they were to participate after school in the afternoon that same day. The principal investigator returned in the afternoon to collect the children for outdoor physical fitness testing. Since muscle strength assessment has a localised effect it was selected as the first of the physical tests.

### 4.5.6 Anthropometry

Height was assessed three times (TEM = 0.31 cm) by free-standing stadiometer (Laboratory-produced stadiometer, AUT University, Auckland – SECA 2m wall-mounted measuring tape) with the tape brought to the crown of the head, whilst the child inhaled and stood with the zygomatic process parallel with the lobe of the ear. Measurements were repeated to ±0.5cm. Weight was assessed three times (±0.1kg, TEM = 0.32 kg) with a SECA personal weight scales (150kg max. ±100gm). Bioimpedance analysis (BIA) was performed three times using an (Impedimed DF50, Impedimed, Queensland, Australia) 50Hz single phase hand to foot method to obtain body fat percentage and to determine fat free mass (FFM) and fat mass (FM) in kilograms. Resistance (TEM = 0.73 ohms), height and weight were used to predict percent body fat (PBF), fat mass (FM) and fat free mass (FFM) in kilograms. (Rush, Puniani, et al., 2003). One subject's data was removed from analyses because of a discrepancy between the written record of resistance and phase angle values.

BMI SDS and PBF SDS was calculated from algorithms provided by Microsoft Excel LMSgrowth progam version 2.6 developed by Huiqi Pan and Tim Cole. BMI SDS was derived from Centers for Disease Control and Prevention in multiple national surveys of American children (Kuczmarski, et al., 2000) and PBF SDS were derived from English school children data (McCarthy, et al., 2006)

### 4.5.7 Blood pressure measurement

The child rested for a minimum of five minutes in a seated position after participating in the anthropometry. Resting heart rate (RHR) and blood pressure (BP) were assessed three times using an electronic sphygmomanometer (SEM-1, Omron, Kyoto, Japan) complete with arm cuff. All measures were taken in a seated position on the left arm. Where there was difficulty in obtaining a measure or large disagreements in values were present, retesting was performed on the opposite arm. Systolic (SBP) and diastolic blood pressures (DBP) were used to calculate pulse pressure (PP) as a variable that predicts risk of future cardiovascular disease (Zakopoulos et al., 2001).

### 4.5.8 Measurement of metabolic rate and substrate utilisation at rest

Respiratory exchange assessment was performed using a Vacu-Med Mini-CPX calorimeter (Vacu-Med Ltd, Ca). The turbine pneumotach was calibrated at the AUT University laboratory prior to assessments and at regular intervals using a one litre syringe. Beta gas was obtained from BOC Ltd (British Oxygen Company Ltd, Otahuhu,

Auckland) at a 15.0% oxygen, 5.0% carbon dioxide and nitrogen balance mix. The calorimeter was 2-point gas calibrated (beta gas and fresh air) every morning approximately 45 minutes prior to assessments. Ambient barometric pressure, temperature and humidity were measured and entered into the Vacumed software programme. For the respiratory exchange measured the child lay supine on a thin soft mattress on the floor. A blanket was made available if they felt cold and a cushion to ensure a reclining posture for a rested, but not sleeping, attitude. RHR to ensure a stable resting state during testing was recorded from an $O_2$ pulse oximeter (Nonin 9500, Nonin Medical, Mn) clipped to the forefinger. RHR was used as an indicator of resting state. During testing the child was constantly monitored to ensure they did not sleep but were comfortable. The child was permitted to read during this time. A respiratory mask was held firmly on the face by way of elastic bands attached to the mask and around the head, holding the mask firmly to the face. Once the child had spent ten minutes or more in this position the pneumotach was attached to the mask and the gas measurement begun. All tests were for no less than 20 minutes and a minimum of five minutes of stable resting data were selected from each test.

Resting metabolic rate (RMR) was calculated as absolute kilocalories per day and resting RER derived from the $VCO_2/VO_2$ from the breath by breath measurements recorded using the Vacumed software. The equations of Frayn (1983) were used to calculated carbohydrate oxidation (1) and fat oxidation (2) from the non-protein RER, $VCO_2$and $VO_2$ measurements (expressed in milliliters per minute) assume that oxidation of protein was negligible.

Fat oxidation mg.min-1 = $1.6946*VO_2 - 1.7012*VCO_2$ (1)

Carbohydrate oxidation mg.min-1 = $4.585*VCO_2 - 3.2255*VO_2$ (2)

Eight children (of 90) were not able to take part in metabolic testing due to forgetfulness, time clashes or equipment malfunction. Their anthropometry and fitness

results have not been included in the analyses. Five sets of indirect calorimetry data were not included in analyses because they were higher than 1.00 (Figure 9). Although a result of >1.00 may be possible, it is more likely indicative of hyperventilation (which is unlikely to be prolonged) or de novo lipogenesis due to recent consumption of carbohydrate. While those with an RER of more than 1.0 reported that they were fasted at the time of assessment. RHR was elevated suggesting that the children may have consumed food shortly before assessment. Some children openly admitted consuming food and their results have been withdrawn from analysis (4.5.3).

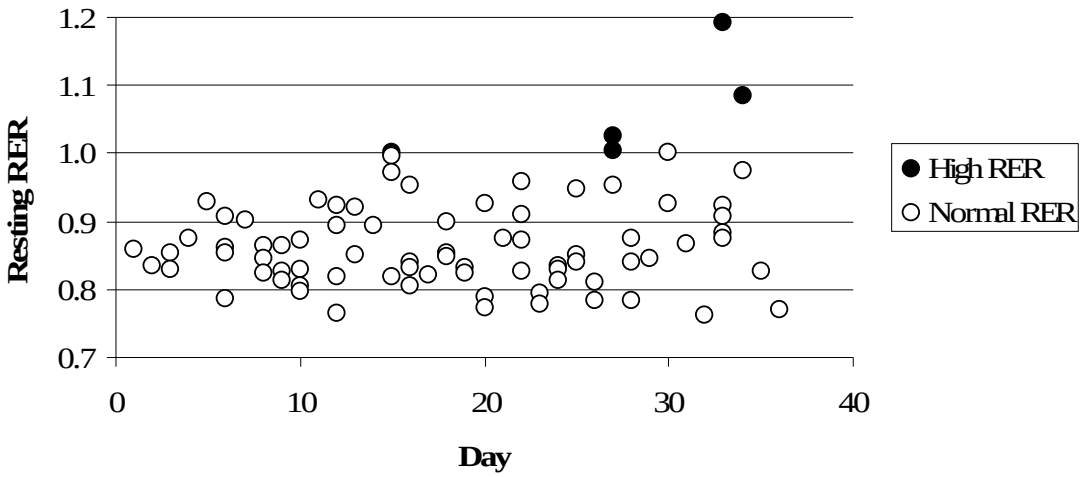


**Figure 9. Resting RER data from all fasting participants (n=74)**
High recorded RER (>1.00) participants (n=5) are identified as filled circles and were removed from analysis, participants with resting RER between 0.75 and 1.00 are identified as open circles.

### 4.5.9 Strength

Muscle strength fitness (MSF) was assessed by portable handgrip dynamometer (Mentone, Melbourne) for grip strength. Standard handgrip dynamometer protocol was applied viz. the dynamometer was grasped in the hand and held directly above the head with a fully extended elbow. The subjects back and legs are resting against a flat wall at all times during the assessment. The opposing arm was relaxed by the side and was not allowed to move at any stage during assessment. Upon a verbal cue "3…2…1…GO!" the child squeezed the handgrip trigger and simultaneously brought the dynamometer forward and down to their side, maintaining full extension at the elbow. On the verbal cue "STOP", the child released the trigger and the dynamometer was reset for the next measure. Each measure was performed three times and on both arms (left TEM = 1.55 kg; right TEM = 2.12 kg). The maximum result obtained for either arm was considered grip maximum for use in data analysis.

### 4.5.10 Cardiorespiratory

The 20m shuttle run test (20m SRT) was selected as the preferred assessment of cardiorespiratory fitness. Preference was given to the 20m SRT for it's:

1. Portability – The need for 4 markers, 20m tape measure and a CD player are the maximum equipment required for the 20m SRT making it a very portable and inexpensive fitness assessment.
2. Specificity - children's play is very rarely linear and thus running in a straight line or around a track change may be more a measure of speed rather than fitness. Employing the 20m SRT is an attempt to replicate the change of direction that children often perform in daily play.
3. Weight bearing – The majority of movement by children is on their feet (weight-bearing) as shown by common activities published by the Children's Nutrition Survey (Ministry of Health, 2003). The usefulness of using an assessment that applies similar physical pressures to daily movement is apparent with the utility of the 20m SRT.
4. Validity and Reliability – The multi-stage fitness test has been validated as a reliable assessment of cardiorespiratory fitness in children (Boreham, Paliczka, & Nichols, 1990; van Mechelen, Hlobil, & Kemper, 1986)
5. Convenience – The children can be tested as a group rather than as individuals. This reduces the time spent conducting tests for the investigator. The added benefit of this convenience is that each child that participates acts as a motivator for the other children to do their best.

A 20m SRT courtyard that was part of a hard asphalt or concrete court was measured and marked with cones as 20m in length. Width was dependent on the number of children, but in this study the width did not exceed 10m in any 20m SRT. The children were assessed for injuries and proper footwear. A briefing was performed prior to beginning the test which included:

1. showing the boundaries of the 20m court,
2. informing the students that a "pacer" will be present during the test to assist them and,
3. running the children through the assessment whilst playing a sample of the CD (this constituted a warmup for the children).

The “pacer” (GK) was introduced to provide the children with information about the test as they run and also as motivation to encourage the children to do their utmost to work to their potential. Most of the children performed to the best of their ability, accentuated by collapsing on nearby grass verges upon completion. For some children it was very obvious that they had not performed maximally, demonstrated by returning to the 20m SRT to join their peers, soon after they had dropped out. The number of laps completed (completion of one 20m length from cone to cone is counted as a one lap) determined the individual laps. Total laps were included in the calculation of the final speed of the participant using the following equation (Stone, 2007):

$$Vmax\ (km.h^{-1}) = V + Vstep \times (Rcom - Rtotal/2)/Rtotal \quad (3)$$

The final speed value value (Vmax) was used in the analyses. V is the velocity of the final stage in $km.h^{-1}$. Vstep is the $km.h^{-1}$ increase in speed between every stage. Rcom represents the number of laps completed within the final stage. Finally, Rtotal is the total number of laps within the final stage.

One child was not able to participate in the 20m SRT, however they were also a non-fasting particiant and their data was not included in the analyses.

### 4.5.11 Statistical Analysis

All statistical analyses were performed using SPSS version 15 graduate pack (SPSS Inc., Chicago IL.). Continuous variables were explored for skewness and kurtosis and Kolmogorov-Smirnov tests of normality were applied to the analysed variables. It was expected that some subject characteristics would not be normally distributed as there were low numbers of participants, males are different to females and the sample may have homogeneity as selection of participants was not by randomisation. Approximately half the variables (age, height, PBF and CRF) were normally distributed however weight, BMI, MSF, and resting RER were not normally distributed. When subgroups are compared e.g. male female, obese non obese, nonparametric statistics – mean, interquartile range, U-test statistic are used. Spearman’s rho determined bivariate correlation. For investigation of more complex relationships such as adjustment for age, sex, fat mass, CRF, MSF, RER and RMR, the general linear model ANCOVA was

used and for prediction, stepwise multiple regression. Following the conventions of Cohen (Cohen, 1988) and Hopkins (the following descriptors of the correlation coefficient of the relationships are used: <0.09 = trivial, 0.10-0.29 = small, 0.30-0.49 = moderate, 0.50-0.69 = large, 0.70-0.89 = very large, and 0.90 to 1.0 = nearly perfect. A P value of 0.05 was used to determine statistical significance.

## 4.6 Results

To maximise the information obtained from this study the findings are presented in two sections; Section A is for those subjects (n=69) who completed all assessments (4.6.1) and Section B those (n=49) that completed all assessments and returned questionnaires (4.6.2). The subjects in the latter group (Section B) are a subsample of the first group (Section A).

### 4.6.1 Section A Results – Physical fitness: association with RER and body composition

As described in the above paragraph the data in this section relates to the 69 children (43.5%) who completed all physical assessments, including anthropometry, the fitness testing and indirect calorimetry measurements. The order of presentation is demographics of the study population, body composition, RMR and RER, physical fitness and contribution of physical fitness and RER to body composition.

*Study population*

Applying the IOTF obesity classification, children in this study were classified as obese (n=10) or not obese (n=59). Two NZ European (3%) and 8 Māori children (12%) were included in the obese group. Absolute Ht was not significantly different ($p$=0.091) between IOTF groups but all other body size measures were significantly higher for the obese group including Ht SDS (As shown by both the BMI SDS and PBF SDS both the not obese and obese children on average were taller, heavier and fatter than the reference children (Table 17). The relationship of BMI SDS with PBF SDS shown as a scatterplot (Figure 10) demonstrates a large and positive association (spearman's rho = 0.77, $p$<0.001) between these two measures. By inspection of the graph it is seen that variation in the relationship for children of lower relative body fatness and BMI (<1.00 SDS) was higher than for the children of higher body size suggesting a more reliable prediction of fatness by BMI SDS for the larger children.

Table 18). Obese females were significantly taller than their NO counterparts [135.6 cm (129.7, 139.3) vs. 126.0 cm (118.5, 135.6), $p$=0.04, respectively] but height was nonsignificant in boys [OB: 139.8 cm (120.2, 152.3) vs. NO: 134.6 (124.9, 138.8), $p$=0.60].

Absolute RMR was higher in the obese children (mean difference=225 kcal.day, $p$=0.045) but when adjusted for FFM it was found to be lower in the obese children (mean difference=-11.4 kcal.day$^{-1}$, $p$=0.001). No significant difference in substrate utilisation and blood pressure was apparent between groups. However CRF was higher in the not obese children (CRF difference: 0.26 km.h$^{-1}$, $p$=0.023). NO boys were much faster than their obese peers (CRF difference: 0.68 km.h$^{-1}$) but the difference was less so for girls (CRF difference: 0.22 km.h$^{-1}$). Strength was higher in the obese children irrespective of sex (MSF: 4.0 kg, $p$=0.021).

As shown by both the BMI SDS and PBF SDS both the not obese and obese children on average were taller, heavier and fatter than the reference children (Table 17). The relationship of BMI SDS with PBF SDS shown as a scatterplot (Figure 10) demonstrates a large and positive association (spearman's rho = 0.77, $p$<0.001) between these two measures. By inspection of the graph it is seen that variation in the relationship for children of lower relative body fatness and BMI (<1.00 SDS) was higher than for the children of higher body size suggesting a more reliable prediction of fatness by BMI SDS for the larger children.

**Table 18. Descriptive characteristics of children by IOTF BMI obesity classification (Cole, et al., 2000) (N=69)**

| | Not Obese (n=59) | Obese (n=10) | U test | Sig. |
|---|---|---|---|---|
| Age (years) | 8.3 (6.9, 9.5) | 8.34 (7, 10.27) | 268.5 | 0.651 |
| Ht (cm) | 129.3 (120.7, 137.1) | 136.5 (128.5, 141.9) | 196.0 | 0.091 |
| Ht SDS | 0.13 (-0.35, 0.99) | 1.46 (0.22, 1.62) | 151.0 | 0.014* |
| Wt (kg) | 28.0 (23.2, 34.5) | 45.4 (38.4, 53.2) | 43.0 | 0.001** |
| Wt SDS | 0.53 (-0.10, 1.04) | 2.54 (2.25, 2.86) | 9.0 | <0.001** |
| BMI (kg.m2) | 17.1 (15.8, 18.4) | 25.0 (22.9, 25.7) | 2.0 | <0.001** |
| BMI SDS | 0.67 (-0.14, 1.04) | 2.76 (2.52, 3.19) | 0.0 | <0.001** |
| PBF (%) | 21.3 (17.6, 25.1) | 33.9 (30.2, 36.4) | 14.0 | <0.001** |
| PBF SDS | 0.72 (-0.33, 1.33) | 2.36 (2.01, 2.55) | 24.0 | <0.001** |
| FFM (kg) | 21.9 (19, 25.5) | 30.3 (26.5, 33.5) | 77.0 | <0.001** |
| FM (kg) | 6.1 (4.5, 8.4) | 15.7 (10.5, 19.2) | 19.0 | <0.001** |
| RMR ($kcal.day^{-1}$) | 1462 (1272, 1639) | 1687 (1490, 1834) | 177.5 | 0.045* |
| RMR.FFM ($kcal.kgFFM.day^{-1}$) | 66.4 (60, 73.9) | 55.0 (51.4, 59) | 92.0 | <0.001** |
| Resting RER | 0.845 (0.819, 0.898) | 0.873 (0.81, 0.923) | 262.0 | 0.574 |
| CHO Ox ($mg.min^{-1}$) | 148 (126, 183) | 206 (141, 220) | 217.0 | 0.184 |
| Fat Ox ($mg.min^{-1}$) | 45 (26, 62) | 45 (27, 79) | 284.0 | 0.851 |
| SBP (mmHg) | 107 (100, 113) | 110 (104, 126) | 202.0 | 0.113 |
| DBP (mmHg) | 64 (60, 72) | 70 (65, 77) | 215.5 | 0.175 |
| PP | 41.0 (37.0, 46.0) | 44.8 (38.5, 50.3) | 232.0 | 0.283 |
| RHR (bpm) | 79 (74, 86) | 77 (74, 87) | 292.5 | 0.966 |
| CRF (km.h-1) | 9.56 (9.13, 10.25) | 9.30 (8.80, 9.50) | 161.5 | 0.023* |
| MSF (kg) | 15.5 (12.5, 19.0) | 19.5 (15.4, 22) | 159.5 | 0.021* |

Mean (Interquartile range) displayed
*Obese significantly different from Not Obese ($p<0.05$)
**Obese significantly different from Not obese ($p<0.001$)
Ht: Height; BM: Body Mass; BMI: Body mass index; FFM: Fat free mass; FM: Fat Mass; RMR: Resting metabolic rate; RMR.FFM: Relative resting metabolic rate per kg fat free mass; RER: Respiratory exchange ratio; CHO ox: Carbohydrate Oxidation; Fat Ox: Fatty acid oxidation; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; PP: Pulse pressure; RHR: Resting heart rate; CRF: Cardiorespiratory fitness; MSF: Muscle strength fitness; SGR: Shoulder girdle retraction; SGP: Shoulder girdle protraction.

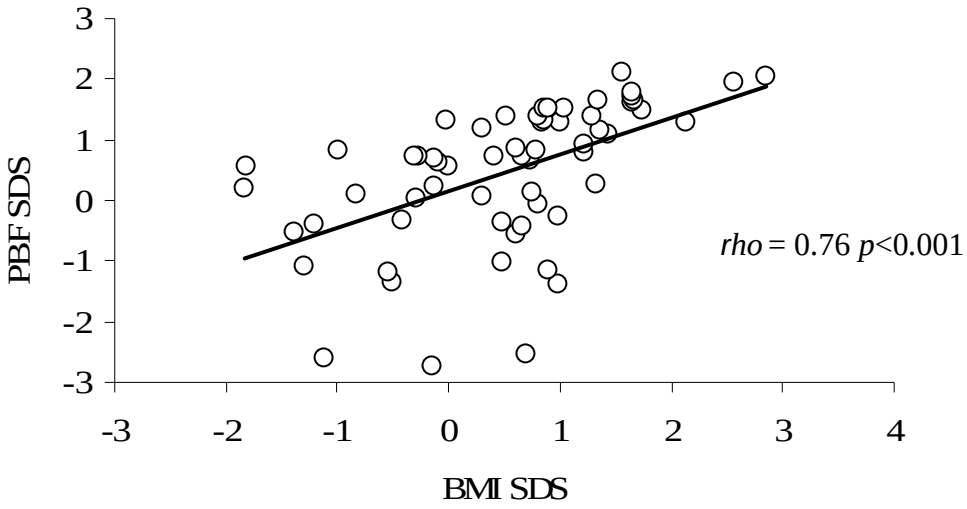


**Figure 10. Paired BMI SDS and PBF SDS of all participants (N=69)**

*Associations among physical measurements*

The pattern and direction of relationships between physical measurements was examined by bivariate correlations, Spearmans rho (Table 19). Resting RMR was moderately to strongly associated with all physical measures. Conversely, there were no significant relationships observed between resting RER and any of the physical measurements including BMI, PBF, FFM or FM. However, both oxidation variables (fat and carbohydrate) were moderately associated with Ht and FFM. Similarly CRF was significantly related with two variables only, age and Ht but there were no significant associations with any body mass variable including BMI, PBF, FFM or FM. However a different picture arose in MSF; all physical measures were strongly associated with MSF. The same broad-ranging associations were observbed in SBP but few for DBP. RHR was inversely associated with age only.

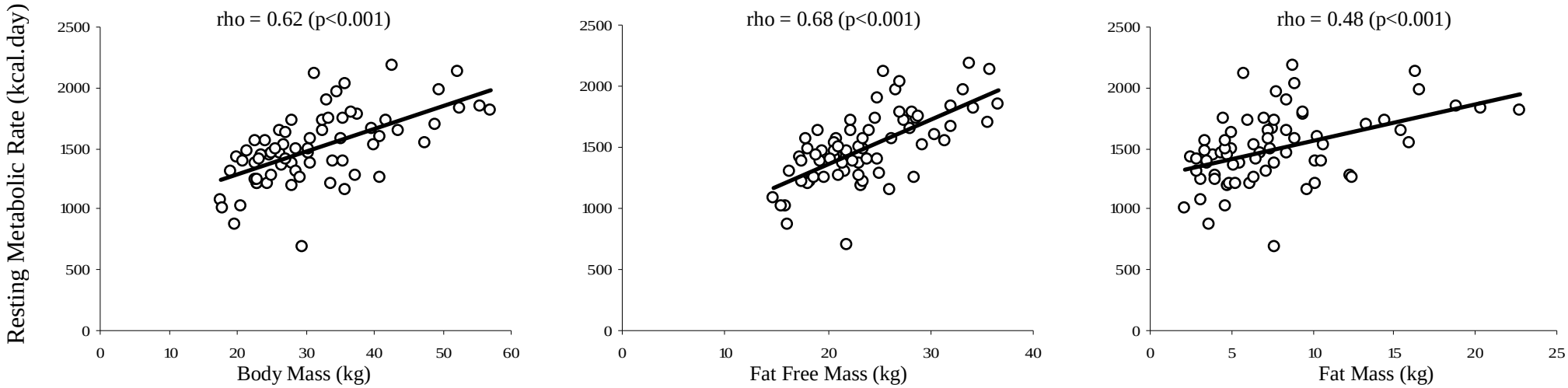


**Figure 11. Spearman's rho bivariate correlates of RMR with BM, FFM and FM.**

**Table 19. Spearmans rho (p value) correlates of anthropometry with physical fitness, blood pressure and metabolic variables (N=69).**

| | Age (years) | Ht (cm) | BM (kg) | BMI (kg.m2) | PBF (%) | FFM (kg) | FM (kg) |
|---|---|---|---|---|---|---|---|
| Age (years) | - | 0.77 (<0.001)** | 0.64 (<0.001)** | 0.33 (0.005)* | 0.43 (<0.001)** | 0.65 (<0.001)** | 0.57 (<0.001)** |
| CRF ($km.h^{-1}$) | 0.39 (<0.001)** | 0.34 (0.005)* | 0.13 (0.281) | -0.09 (0.476) | -0.17 (0.173) | 0.21 (0.084) | -0.004 (0.971) |
| MSF (kg) | 0.68 (<0.001)** | 0.79 (<0.001)** | 0.78 (<0.001)** | 0.56 (<0.001)** | 0.49 (<0.001)** | 0.80 (<0.001)** | 0.69 (<0.001)** |
| RHR (bpm) | -0.29 (0.014)* | -0.19 (0.123) | -0.15 (0.210) | -0.05 (0.690) | -0.02 (0.893) | -0.18 (0.130) | 0.10 (0.407) |
| SBP (mmHg) | 0.42 (<0.001)** | 0.48 (<0.001)** | 0.48 (<0.001)** | 0.36 (0.002)* | 0.30 (0.012)* | 0.49 (<0.001)** | 0.42 (<0.001)** |
| DBP (mmHg) | 0.17 (0.170) | 0.15 (0.210) | 0.25 (0.067) | 0.27 (0.023)* | 0.15 (0.234) | 0.26 (0.033)* | 0.20 (0.108) |
| PP | 0.35 (0.003)* | 0.43 (<0.001)** | 0.33 (0.006)* | 0.16 (0.187) | 0.22 (0.071) | 0.34 (0.004)* | 0.32 (0.008)* |
| Resting RER ($VCO_2/VO_2$) | 0.13 (0.283) | -0.02 (0.841) | 0.06 (0.643) | 0.05 (0.711) | 0.12 (0.324) | 0.03 (0.828) | 0.09 (0.446) |
| CHO Ox ($gm.min^{-1}$) | 0.28 (0.019)* | 0.24 (0.045)* | 0.29 (0.015)* | 0.19 (0.125) | 0.19 (0.122) | 0.30 (0.013)* | 0.24 (0.049)* |
| FAT Ox ($gm.min^{-1}$) | 0.04 (0.721) | 0.30 (0.012)* | 0.22 (0.067) | 0.13 (0.283) | 0.06 (0.641) | 0.25 (0.033)* | 0.16 (0.194) |
| RMR ($kcal.day^{-1}$) | 0.44 (<0.001)** | 0.65 (<0.001)** | 0.62 (<0.001)** | 0.44 (<0.001)** | 0.29 (0.014)* | 0.68 (<0.001)** | 0.48 (<0.001)** |

Ht: Height; BM: Body mass; BMI: Body mass index; PBF: Percentage body fat; FFM: Fat free mass; FM: Fat mass; CRF: Cardiorespiratory fitness; MSF: Muscle strength fitness; RHR: resting heart rate; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; PP: Pulse pressure; CHO Ox: Carbohydrate oxidation; FAT Ox: Lipid oxidation; RER: resting respiratory exchange ratio; RMR: Resting metabolic rate.
*significant $p<0.05$
** significant $p<0.001$

*Relationships with measures of physical fitness*

Examination of the patterns of the relationships of physical fitness with blood pressure and respiratory exchange show that as fitness increases, resting metabolic rate increased but resting heart rate decreased (Table 20). Muscle strength showed stronger associations with diastolic and pulse pressures than cardiorespiratory fitness – which is explained by the high correlation of MSF with body mass previously shown (Table 19). The collinear relationship of blood pressure and muscle strength with body mass infers that they should be regarded as alternative outcomes of growth and not independent explanatory variables.

**Table 20. Spearmans rho correlates of fitness parameters with cardiovascular and metabolic variables.**

| | CRF | MSF |
|---|---|---|
| RHR (bpm) | -0.26 (0.036)* | -0.24 (0.063) |
| SBP (mmHg) | 0.10 (0.438) | 0.11 (0.397) |
| DBP (mmHg) | 0.25 (0.046)* | 0.49 (<0.001)** |
| PP | 0.16 (0.202) | 0.45 (<0.001)** |
| CHO Ox ($gm.min^{-1}$) | 0.08 (0.543) | 0.30 (0.015) |
| FAT Ox ($gm.min^{-1}$) | 0.21 (0.096) | 0.23 (0.070) |
| Resting RER ($VCO_2/VO_2$) | -0.08 (0.534) | 0.08 (0.518) |
| RMR (kcal.day) | 0.34 (0.006)* | 0.65 (<0.001)** |

CRF: Cardiorespiratory fitness; MSF: Muscle strength fitness, RHR: resting heart rate; DBP: Diastolic blood pressure; SBP: Systolic blood pressure; PP: Pulse pressure; CHO Ox: Carbohydrate oxidation; FAT Ox: Lipid oxidation; RER: resting respiratory exchange ratio; RMR: Resting metabolic rate.
*significant $p<0.05$
**significant $p<0.001$

*Explanation of physical fitness by outcome variables- multiple regression*

Physical fitness (CRF and MSF) were investigated for relationships with anthropometry RMR and RER. Univariate analysis of the dependent variable CRF adjusted by age and sex (which were not significant), and added Ht and BM to the model. Ht (cm) had a significant positive relationship with CRF ($\beta=0.043$, $p=0.003$) and BM (kg) was equally but inversely related to CRF ($\beta=-0.045$, $p=0.002$). Stepwise linear regression with BM

separated into two FFM and FM showed that the speed attained was best predicted by FM (10%) and Ht which explained a further 17% of the variance.

The prediction equation for final speed in beep test ($km.hr^{-1}$) was:

4.75 +0.042 * height(cm) -0.084 * fat mass (kg) $R^2$=0.27 SEE = 0.634 $km.hr^{-1}$ (4)

FFM did not add any further explanation to this model. A similar process of exploration of explanatory variables for MSF gave rise to a different model, compared with CRF, where FFM explained 59% and age, a further 5% of MSF. The prediction equation for muscular strength fitness was:

-0.415 + 0.428 *fat free mass (kg) +0.765 age (years) $R^2$=0.66 SEE = 2.434 kg (5)

Neither CRF or MSF were explained by sex.

*Modelling RMR and resting RER*

The first model examined was the effect of body mass, FFM and FM on resting metabolic rate and then age, sex and CRF were explored. The equations derived were:

RMR $kcal.day^{-1}$.= 903 + 18.9 * body mass (kg) $R^2$=0.36 SEE = 234 $kcal.day^{-1}$ (6)

RMR $kcal.day^{-1}$.= 627 + 36.7 * FFM $R^2$=0.45 SEE = 217 $kcal.day^{-1}$ (7)

Fat mass was not a significant predictor when added to the above model and neither were age or sex. Finally the variable of CRF was added to the model as it is known that

fit people have a higher resting metabolic rate (De Lorenzo et al., 1999). Age, height and sex were not significant predictors and were not included in the model. CRF added a further 3% of explanation to this mode.

RMR kcal.day$^{-1}$.= -133 + 34.5 * FFM (kg) + 84.6 * final speed(km.hr$^{-1}$); $R^2$=0.48 SEE = 217 kcal.day$^{-1}$;standardised $\beta$ = 0.638 (FFM) and 0.193 (CRF). (8)

### 4.6.2 Section B Results – Food and activity pattern association with resting RER and body composition

*Study population*

This section explores food and activity (including physical inactivity and sleep) patterns reported by the parents of the children, however the data is limited to the number of questionnaires (n=49). Table 20 shows that parents of Not Obese children reported that their children consumed fat more frequently than Obese children (mean difference = 1.27 times per day, $p$=0.008). Further, non-significant trends were observed in reported time spent in physical inactivity and frequency of physical activities. Obese children tended to spend more time in inactivity and were less frequently reported participation in activities than Not Obese children.

**Table 21. Food and activity differences between IOTF criteria BMI not obese and BMI obese 5 to 12 year olds (N=49)**

| | Not Obese (n=45) | Obese (n=4) | U test | Sig. |
|---|---|---|---|---|
| Male/female (%) | 48.9/51.1 | 75.0/25.0 | | |
| Age (years) | 8.4 (6.8, 9.6) | 8.5 (6.4, 10.5) | 79.0 | 0.712 |
| BMI SDS | 0.61 (-0.20, 1.25) | 2.78 (2.45, 3.22) | 0.0 | <0.001** |
| Foods | | | | |
| Carbohydrate (times per day) | 4.33 (3.36, 6.21) | 5.16 (4.17, 6.21) | 69.0 | 0.469 |
| Fat (times per day) | 3.80 (2.95, 4.68) | 2.55 (2.10, 2.93) | 29.0 | 0.023* |
| Healthy food (times per day) | 5.07 (3.68, 6.14) | 5.01 (2.59, 5.86) | 79.0 | 0.712 |
| Milk times per day) | 1.00 (0.79, 1.79) | 0.93 (0.32, 1.00) | 65.5 | 0.385 |
| Non-diet Soft Drink (times per day) | 0.07 (0.01, 0.14) | 0.04 (0, 0.07) | 68.0 | 0.447 |
| Diet Soft Drink (times per day) | 0.01 (0, 0.07) | 0.07 (0.01, 0.43) | 30.5 | 0.128 |
| Takeaway Food (times per day) | 0.09 (0.03, 0.16) | 0.02 (0.02, 0.03) | 40.0 | 0.076 |
| Activities | | | | |
| Physical Activity (times per day) | 4.72 (2.38, 6.79) | 3.78 (1.96, 8.58) | 28.0 | 0.676 |
| Physical Inactivity (hours per week) | 14.0 (6.25, 22) | 18.5 (14.5, 19.9) | 66.0 | 0.437 |
| Sleep (hours per week) | 76.5 (73.5, 79.3) | 74.5 (71.6, 77.4) | 69.0 | 0.469 |

*significant $p<0.05$
**significant $p<0.001$

Few food and activity correlates showed any association with anthropometry and RMR and resting RER data (Appendix F). PBF was moderately positively related with healthy food frequency (rho=0.32, $p$=0.034). Total weekly sleep hours had a small to moderate inverse relationship with all anthropometry, with the exception of BMI ($p$=0.198). Additionally, this sleep variable was also significantly inversely correlated with carbohydrate oxidation (rho=-0.36, $p$=0.017).

## 4.7 Discussion

In a sample of young children of a wide range of body size and physical fitness, this study explored patterns of association of body size, physical fitness and food and activity patterns with the biomarkers of measured resting metabolic rate and resting substrate oxidation. The main findings were that, independent of age and sex, cardiorespiratory fitness, but not muscle strength fitness, is associated with an increased RMR. Additionally, RMR and fitness were confirmed as the difference between the obese and not obese groups. Specifically with reference to tests of the four hypotheses the following observations were made in this study:

1. RMR values were predicted by fat free mass ($R^2$=0.45) but not fat mass. FFM and CRF together were the best predictors of RMR ($R^2$=0.48)
2. No negative association was recorded between CRF and body size (BM, FFM, FM) or RER. Bigger children had stronger muscles (MSF).
3. RER was not associated with fat intake, physical activity and fitness and sleep
4. Physical inactivity and carbohydrate and fast food intake were not positively associated with RER

Thus for the three hypotheses concerning substrate oxidation the null hypothesis was supported. The neglible effect size observed add to the body of knowledge that informs measurements of outcomes of healthy eating and physical fitness programmes in children. Resting RER is not a measure that is associated with body mass, but elevation of RMR through increased cardiorespiratory fitness may be able to help close the energy gap thought to be responsible for weight gain (Swinburn et al., 2006b) More research is required in this area and the directions for those studies have been included in the following text.

### 4.7.1 RMR and resting RER

In growing children there is complexity in the interpretion of measurements in the context of age and development stage. Measurement of physical fitness is a particular challenge. Firstly, there are no published reports of a “beep test” in children as young as five and secondly adjustment for body size (particularly stride length/height) was an important moderator. The close relationship of resting metabolic rate with fat free mass

and cardiorespiratory fitness exemplifies the physiologic pathway by which fitness and therefore improved muscle function may influence health. Molnar and Schutz (Molnar & Schutz, 1997) have confirmed in children that fat free mass is a major contributor to resting metabolic rate and that there is also an age effect. Aerobic (CRF) fitness training appears to drive RMR in a positive fashion which in turn may contribute to weight management (Speakman & Selman, 2003). An increase in RMR from CRF exercise seems to be generated from voluntary participation in activity, rather than an enforced, more demanding regimen.

A common research perspective has been to consider how exercise (aerobic or resistance training) could affect RMR (Speakman & Selman, 2003). This study utilised current level of fitness (CRF or MSF) as the explanatory variable. One published study has performed a similar analysis to this one although they reported daily energy expenditure (DEE) instead of RMR (Sharp, Reed, Sun, Abumrad, & Hill, 1992). Sharp et al. (1992) found that while both FFM and $VO_2$max (representing CRF) were significantly correlated with DEE, multiple regression analysis of DEE, adjusted by FFM, could not find an independent effect of $VO_2$max. No studies involving children, fitness level and RMR could be uncovered that would elicit useful information.

In contrast, muscle strength was unable draw any such relationship with RMR in this study and this was despite strong bivariate correlates between MSF and RMR. The general linear model found that after adjustment for FFM, MSF was no longer significant. The conclusion must be drawn that MSF in children does not contribute to RMR, but CRF may.

Resting RER was unrelated to body mass. None of the environmental factors drawn from the questionnaire, appeared to be related to resting RER. This is consistent with the findings of three published studies of PF, body composition and resting RER in children (Grund et al., 2000; Salbe et al., 2002; Treuth et al., 2003). The resting RER in five to ten old overweight (0.86 ±0.03; n=12) and obese children (0.86 ±0.01; n=54) when compared to similar age, normal weight children (n=22; 0.89 ±0.04) was not significantly different (Grund, et al., 2000).

Neither fitness, body fat nor puberty were related to resting RER in 12 six to nine year olds and 12 thirteen to sixteen year olds, but resting RER was linked to sex and macronutrient composition (Treuth et al., 2003). Furthermore, Salbe et al. (2002) found no association with resting RER and adiposity or body mass in a 5-year followup of five year olds ($R^2$=0.545, $p$=0.12). The results of this study show that resting RER is not

representative of the cumulative effect of the environmental factors of food and activity patterns and fitness on body size and that indications are that it may not be utilised as a predictor of body fat accumulation as found in adults (Marra et al., 2004; Seidell et al., 1992; Valtuena et al., 1997).

### 4.7.2 Physical fitness

In this study, physical fitness is related to body size and its significance may be due to the objective measures that were utilised to assess physical fitness. Physical fitness is represented by two elements in this study, muscle strength fitness (MSF) and cardiorespiratory fitness (CRF). How physical fitness is related to body size is one of the differentiating features of this study.

Adjustment for age and sex showed that an inverse correlation between CRF and fat mass is clear. CRF was related to reduced body fat and BMI in 6 to 13 year olds (Nassis, Psarra, et al., 2005) and Ara et al. (2004) found that fitness ($VO_2$max) in 114 pre-pubescent boys was predictive of percent body fat. The difference between study assessment methods and data analysis does not assist comparison of results however these studies and the current study suggest that CRF does influence body size in terms of FM, but the pathway are unclear. What is known is that FM appears to impede a true reading of fitness in children.

Height also demonstrated a strong relationship with CRF. This may be due to taller children having a longer leg length (and therefore stride length) and contributes positively to performance in the 20m SRT (Grieve & Gear, 1966). The significantly higher height of the obese children, particularly obese females (n=7), can positively contributed to the relatively small CRF difference compared to non-obese children. Early pubertal growth driven by excess fat accumulation may have contributed to this height difference (Dunger, Lynn Ahmed, & Ong, 2005).

The absence of strong relationships between physical fitness and the questionnaire variables suggests that any preventive approach to weight management would need to consider multiple aspects, such as food, physical activity, fitness and sleep, in conjunction with each other.

The other aspect of physical fitness, muscle strength, showed a parallel relationship with all anthropometry, including BM, FFM and FM. As indicated above MSF did not to contribute to RMR and it's bivariate correlations were most probably due to the close relationships of strength to FFM and FFM to RMR. Twelve obese, pre-pubertal girls (7

to 10 years) undergoing a strength programme for 5 months not only increased in strength (Bench press: 19.6%; Leg press: 20.0%; Isometric strength: 35.2%) but also increased in percentage body fat (baseline: 38.9% ±6.6; final: 39.2% ±5.9) and overall weight (baseline: 46.6 kg ±9.4; final: 50.6 kg ±10.3). Thus an increase in body size generally begets an increase in muscle mass and more muscle can apply more force.

### 4.7.3 Food and activity patterns

A paucity of relationships was observed between the food and activity data with objective measures of body size and RMR and resting RER. Their lack of association with these outcomes may be explained by statistics or methods. From a statistical perspective, 1) there may not be any true or significant relationship or 2) that food and activity variables could interact in such a way to be confounded by one another. Methods wise, the data could be affected by external factors such as biased reporting, a common problem with parental- and self-reported data.

The effect of reported food data with objective outcomes such as body size or energy expenditure (for example RMR), has been ambiguous in research, whereas activity research outcomes are less vague. This may be because of the introduction of more objective measures and standardised protocols. Activity can be well measured by doubly labelled water or accelerometry however food has no such objective measure. Reported food intake is the only source of energy intake data in a naturalistic environment. Inaccuracy of dietary recall may be for two reasons 1) lack of parental knowledge about their child's patterns or 2) biased responses based on what a researcher may want to see. The latter may explain the reported physical activity, fat-based food intake and healthy food intake of overfat children that was superior to their non-overfat peers.

Further, the grouping of foods into fat-based, carbohydrate-based and healthy foods could potentially confound the outcome of the results as it could be argued that some fat-based foods could, for example, be categorised as carbohydrate or some other category. The use of food frequency may have also predisposed this study to the questionnaire variables having weaker relationships with other variables. Food frequency does not account for portion size or volume of food; however it has been known to provide an indicator of food habits. Perhaps the best alternative is to research valid, and where possible objective, tools for use in a free-living context to inform food research.

### 4.7.4 Limitations and strengths

Limitations of this study were that the study population were volunteers from low decile schools, thus there may be bias in recruitment. All children who assented, met the inclusion criteria and had parental consent were included in the study. Recruitment was stopped when there was a pragmatic need for an end date for the study. The questionnaires asked for parental recall of their children's food and activity patterns. Although parental report is more valid than self-report in pre-pubertal children (Lafay et al., 1997; Livingstone, Robson, & Wallace, 2004), recall of any lifestyle factor is fraught with error and may not be reliable.

Strengths of this study were that the schools and caregivers were very supportive of the principal investigator, possibly because of the empathetic and non-invasive approach and also that they appeared to be truly interested and concerned about the health of their children. This study's use of examination of patterns by correlation followed by continuous data analysis (regression and ANCOVA) with careful selection of explanatory variables that are not collinear has enabled a more powerful analysis than categorical or odds ratio analysis. The objective anthropometric and physiologic measures paired with survey results was inclusive of exercise science, nutrition and paediatric health and exemplified the multi-disciplinary perspective of this study. The originality of investigating RER as a predictor for obesity and body fat accumulation in children is also a strength.

### 4.7.5 Summary

In this study, food and activity were not seen as influential factors for RMR or resting RER. These lifestyle variables had very little in common with any outcome, including body mass and fitness. This lack of commonality may be due to inaccuracies and biases that are inherent in reported data. It is much more desirable to implement objective measures to investigate the relationship of food and physical activity with outcome variables such as energy expenditure or body mass.

Cardiorespiratory fitness in children has a relationship, mediated by age, with body mass that is probably directed via the physiologic pathway of resting metabolic rate. This finding supports current evidence that there is a relationship between fitness and body mass in young children (Hussey, Bell, Bennett, O'Dwyer, & Gormley, 2007; J. Ruiz et al., 2006), but until now the pathway through which this phenomenon may exert itself was an educated guess. In addition, muscle strength had no relationship with RMR

or resting RER and the strong correlation observed with body mass may be considered as an indirect association.

# 5 Body mass is associated with insulin, fitness and bone integrity in young children

## 5.1 Background

In 2004, the Waikato District Health Board launched a region-wide through-school programme of nutrition and physical activity education and participation. This programme was known as Project Energize, and was initially implemented in 124 primary schools throughout the Waikato (Graham, et al., 2008). In late 2005 a 'Milk in Schools' programme was established within eleven low decile (a measure of lower socioeconomic status) Project Energize schools. Children were given 300 mL of 'Mega-Milk', which was provided by Fonterra™, each school day over a period of two years. Each 300 mL tetrapak of Mega-milk was enriched in calcium (60% adult RDI) and vitamin D (15% RDI) and low in total fat (6 g) and saturated fat (4.2 g). An additional eleven control schools, who did not receive milk, were matched for decile and included in the Project Energize programme from the beginning of 2007.

## 5.2 Literature review

The consumption of dairy product, particularly milk, for health benefits has had equivocal support (Huang & McCrory, 2005). The relationship between milk consumption and overweight and obesity has been investigated by several researchers (Barba et al., 2005; Cook, Altman, Jacoby, & Holland, 1975; Lanou, Berkow, & Barnard, 2005; Rush, Paterson, & Obolonkin, 2008). Barba et al. (2005) observed a significant inverse relationship (t=-2.964, $p$=0.003) between frequency of milk intake and BMI standard deviation score (SDS) in young children (N=884) aged three to eleven years. Conversely, Rush et al. (2008) identified that dairy consumption was positively related to body fat and BMI. A survey of 396 English primary school children (8 to 11 years) found no relationship of school milk consumption with obesity, skinfolds, height, weight and arm girth (Cook, et al., 1975).

The mechanisms by which calcium may exert a weight control influence continue to elude researchers; however a few potential pathways have been suggested from animal and in vitro models. Dietary calcium affects weightloss via physical and biological approaches. Calcium ions bind to fatty acids in the gut thereby preventing fat uptake (Denke, Fox, & Schulte, 1993; Jacobsen, Lorenzen, Toubro, Krog-Mikkelsen, & Astrup, 2005; Papakonstantinou, Flatt, Huth, & Harris, 2003; Welberg et al., 1994), but since the effect of this action is small it does not fully explain weight loss found in

population studies (Zemel, 2005). An explanation may be found post-absorption where serum calcium attenuates calcitrol (1,25-$(OH)_2$-D) concentrations (and possibly parathyroid hormone) which in turn regulates a series of adipocyte cellular processes. Firstly, MARRS (Membrane associated rapid response to steroid) protein activity is suppressed preventing entry of calcium to drive fatty acid synthase expression. By preventing fatty acid synthase function dietary calium promotes lipolysis and avoids de novo lipogenesis (Nemere et al., 2004; Nemere, Safford, Rohe, DeSouza, & Farach-Carson, 2004; Shi, Norman, Okamura, Sen, & Zemel, 2001). Low concentrations of calcitrol also allows uncoupling protein 2 (UCP2) (Shi, Norman, Okamura, Sen, & Zemel, 2002) to function and maintains mitochondrial thermogenesis rates (even in low-calorie environments) by increases fat cell apoptosis rates via UCP2 (Sun & Zemel, 2004) and mitochondrial potential (Shi, DiRienzo, & Zemel, 2000). Calcium supplements can bring about the same effect of weightloss however a greater effect has been found with dietary calcium sources such as dairy foods and this greater effect has been attributed to the bioactive ingredients found in dairy products.

Bioactive ingredients of dietary calcium sources (specifically dairy products), such as ACE (angiotensin converting enzyme inhibitory peptides) and branched chain amino acids (BCAA's), have been found to work well in conjunction with calcium to promote weightloss. Whey contains ACE inhibitors that inhibit adipocyte fatty acid synthase expression (Causey & Zemel, 2003) and BCAA's may encourage protein synthesis and provide energy substrate (Anthony, Anthony, Kimball, & Jefferson, 2001). Animal models demonstrate faster weightloss in calorie-restricted diets with the use of whole dairy products (than without dairy), but also achieve weightloss in energy-balanced (eucaloric) diets (Zemel, 2005).

In addition to overweight and obesity, insulin resistance has also been linked with milk consumption. Pereira et al. (2002) showed that increased dairy consumption in (N=3157) overweight young adults (18 to 30 years) over a ten year period has an inverse association (OR 0.79, 95% CI 0.70-0.88) with insulin resistance syndrome and may provide a protective health benefit. Suggested mechanisms to explain the effect found in their study included weight regulation of dairy products (as specified above), as yet unrecognized boactive substances in dairy, or dairy intake associated dietary patterns such as low glycaemic index. However no mechanism has been identified linking dairy or milk consumption with insulin resistance in children.

Although milk has not been shown to improve insulin resistance in children, fitness has. An eight-week trial with (N=14) obese children showed a significant improvement in insulin sensitivity (8.20 ±3.44 to 10.03 ±4.33 mg.kg.min$^{-1}$) without a change in fat mass (Bell et al., 2007). It is also associated with a reduction in the risk of early mortality in patients with existing disease (Lavie & Milani, 2005). In children the association of physical fitness with future risk is not as clear as the disease outcomes are not usually manifested at a young age. More convincing evidence from longitudinal studies in children have shown that an increase in muscle strength fitness (MSF) or CRF is associated with improved insulin sensitivity and can lead to improved glycaemic control and cardiovascular disease biomarkers (Allen et al., 2007 ; Bell, et al., 2007; Benson, et al., 2006; Carrel, Clark, et al., 2005; S. Lee, et al., 2006; Nassis et al., 2005 ). CRF was a significant predictor for fasting insulin ($p$<0.001) in 106 middle school (Age ±SD = 12.8 ±1.4 years) children (Allen et al., 2007).

Combining nutrition and exercise is also effective, for example, 50 obese six to eighteen year olds participated in an intervention that involved nutrition education focusing on the food guide pyramid and physical education classes based on lifestyle activities, and playing down competitive games, five times a fortnight for 45 minutes duration. This study demonstrated that fasting plasma insulin concentrations, from baseline to post-intervention, decreased in the programme group but increased in control (-5.1 ±5.2 vs. 3.0 ±14.3 $\mu$IU/mL), however the wide age range and limited number of participants make it difficult to ascertain the true benefit for prepubescent children (Carrel, Meinen, et al., 2005).

Likewise, a cross-sectional study conducted with older New Zealand children and adolescents (10 to 15 years) showed that high levels of physical fitness were associated with enhanced insulin sensitivity – HOMA2-S% (Benson, et al., 2006). Also, Lee et al. (2006) in North American children aged 8 to 17 years of age (n=122) demonstrated positive associations of fitness with insulin sensitivity but when multiple regression analysis included fat mass, measured by DEXA and CT, the association with CRF no longer held as an important mediator. Finally, Nassis et al. (2005) was able to demonstrate that 12 weeks of aerobic training in 19 overweight and obese girls (aged between nine and fifteen years of age) CRF (PWC170) was increased by 18.8% (mean PWC ±SD = 1.30 ±0.42 vs. 1.30 ±047). For these girls, insulin (area under the curve - AUC) dropped by 23.3% (AUC ±SD = 9799.0±4918.6 vs. 12781.7 ±7454.2 µU.min.mL$^{-1}$).

Additionally, Vitamin D status is known to be positively associated with insulin sensitivity, and this relationship appears to be fat mass-mediated in children aged 6.0 to 17.9 years (Alemzadeh, Kichler, Babar, & Calhoun, 2008). These insulin studies have almost exclusively reported findings from middle school or older children, however most large-scale interventions now regularly include children as young as 5 years of age (Graham, et al., 2008; Sharma, 2006).

Another biomarker of health and fitness status is adequate bone mineralization which is associated with adequate nutrition including improved CRF (Yin et al., 2005) and increased MSF, vitamin D and calcium (Cadogan, Eastell, Jones, & Barker, 1997). Indeed, bone mineral density at a young age tracks to better bone health and less osteopenic disease as adults (Ondrak & Morgan, 2007). Increases in fitness, measured by heart rate change in a bench-step test, were concomitant with increases in bone mineral density (BMD: 0.008, 95% CI 0.001, 0.005) in high attendance participants (N=447) of the Georgia Fitkid Project (Yin, et al., 2005).

Serum vitamin D and calcium concentrations are known to affect bone remodeling in adults but vitamin D and calcium interactions are not well understood in children as longitudinal studies are lacking (Holick, 2004 ; Weaver, 2007). Most studies in children are based on adolescent girls, particularly because it is known that peak bone mass in this age and sex is important for adult life. Cadogan et al. (1997) conducted an open randomised controlled trial of 82 females from four high schools. Programme girls were asked to consume as much of a daily home-delivered pint (586 mL) of milk as possible for 18 months. Dietary intake was assessed by seven-day weighed intake and physical activity was recorded by seven-day recall. Intervention children had greater increases than control in bone mineral density (BMD) (9.6% vs 8.5%, $p$=0.017) and bone mineral content (BMC) (27.0% vs. 24.1%, $p$=0.009).

Bone remineralisation (programme: 22.8% ±6.9% vs. control: 12.9% ±8.3%) was found to take place in 12 month randomized controlled trial (RCT) of 48 girls (Chan, Hoffman, & McMurry, 1995). Similarly, a two year RCT of 91 adolescent girls (15 to 18 years) was able to bring about a higher bone mineral density (trochanter: 4.6% and lumbar spine: 1.5%) in the programme group with dairy supplementation (mean 1160 mg of calcium/day) (Merrilees et al., 2000). Sandler et al. (1985) concurs with these findings and suggests that peri- or postmenopausal bone mineral density may be attributable to childhood or adolescent milk consumption.

However a review of milk and dairy consumption by Lanou et al. (2005) failed to find any evidence that increased consumption of milk improved bone mineralisation outcomes in children or adolescents despite government policy that promoted otherwise. Further, use of dairy products as an intervention may need to consider the lactose intolerance aspect of some ethnicities (Brand & Darnton-Hill, 1986).

Calcaneal quantitative ultrasound (QUS) has been employed as a tool to non-invasively observe changes in bone structure in a few paediatric studies (Lehtonen-Veromaa, Mottonen, Kautiainen, Heinonen, & Viikari, 2001; Magkos, Manios, Babaroutsi, & Sidossis, 2005; Rautava et al., 2006), however none have studied a children's population of less than 10 years of age. It should be acknowledged that the QUS outcome is not a measure of bone mineral density (Masud & Francis, 2000) and therefore cannot diagnose osteopenic disease. However, it is able to differentiate those most at risk of developing osteopenia (Lopez-Rodriguez, Mezquita-Raya, de Dios Luna, Escobar-Jimenez, & Munoz-Torres, 2003). To date, Magkos et al. (2005) have published normative QUS data from 1205 Greek children and adults but no data exists for New Zealand children.

To date there is an absence of research to show whether supplemented calcium and vitamin D status and inherent fitness is associated with fat mass, insulin sensitivity, insulin resistance and bone density in young pre-pubescent children.

## 5.3 Introduction

In 2007, ten of the intervention and eight of the control schools agreed to participate in this cross-sectional study. Although some control schools had been introduced to the intervention during 2007, none of the control schools involved in this study had been enrolled into the intervention arm of Project Energize. To assist in determining glucose homeostasis (Insulin sensitivity [HOMA2-S%] and beta cell function [HOMA2-B%]), lipid profile, and bone related biochemistry (parathyroid hormone [PTH], Calcium and vitamin D [25-hydroxy Vit D]), fasting blood was sampled, bone density assessed and body composition and physical fitness measurements made. Subsequently, the changes in fasting blood vitamin D and lipid concentration as a result of receiving the milk was reported (Graham, Kira, Conaglen, McLennan, & Rush, 2009). To extend this work, this chapter primarily explores the associations of body mass with insulin homeostasis, fitness and bone integrity in children as a result of receiving the milk.

## 5.4 Aims

The aims of this study were to investigate in young children whether:

1. a two year fortified milk supplementation programme influences lipids, body composition, glucose homeostasis and bone integrity
2. the fitness parameters of CRF and MST are associated with lipid profile, body composition, glucose homeostasis and bone integrity.

## 5.5 Hypotheses

Frequent and regular consumption of a fortified milk product will result in:

1. an improved lipid profile (that is, lower triglycerides and LDL-C and higher HDL-C).
2. an improved stiffness index (as a measure of bone strength) as measured by QUS
3. a lower BMI and PBF SDS, and
4. a higher fitness (CRF and MST) level than non-consumers

Children classified as obese will:

1. have adverse HOMA and lipid profiles, and
2. perform worse than children classified as not obese in fitness tests

Fitness parameters are:

1. inversely associated with BMI and PBF SDS
2. inversely associated with beta cell function and positively associated with insulin sensitivity, and
3. positively associated with stiffness index.

## 5.6 Method

### 5.6.1 Design

A case-control comparison of children in low decile Project Energize programme schools who received milk for up to two years with children in low-decile Project Energize control schools who did not. Measurements were made on the same children at two time points – Phase One and Phase Two. Phase One measurements (5.6.5) were conducted approximately two years after the introduction of the milk supplement. An administrative delay in the ethics approval for the fitness measures meant that Phase Two measurements were conducted, on average, fifteen weeks after Phase One for each individual. Between phases there were two consecutive weeks of holidays and thirteen

weeks of school. All assessments were conducted at the school the child attended for ease of transport; curriculum continuation and child/parent familiarity. School halls were the usual assessment location as they were spacious enough to hold large numbers of children; assessment stations could be located throughout the hall with plenty of space and relative privacy and contained chairs for seating, facilities for washing hands and toileting, and kitchen facilities to provide breakfast.

### 5.6.2 Recruitment

Letters and consent forms were handed to all potential participants (n=1040) and when completed were returned to the school administration (Appendix D). Two control schools were not allocated for testing as few or no consent forms were received. Twenty seven percent (n=282) of distributed consent forms were returned complete. Subsequently, seventy four percent (n=169) of consenting participants presented in a fasted state at Phase One of the assessment which included anthropometry, QUS, and blood sampling. Phase Two was held approximately 15 weeks after Phase One and consisted of anthropometry (repeated from Phase One) and fitness assessments.

### 5.6.3 Study population

Ethical approval for Phase One (NTY/06/10/100) was provided by the Northern Region Y Ethics committee in July 2007 (Northern Y Ethics Committee, 2007). Phase Two ethics was approved by the Northern Region Y Ethics committee in November 2007 (Northern Y Ethics Committee, 2007) as an addendum to Phase One. Table 22 shows that there was a 39.1% dropout of participants between Phase One and Two, however the proportion of ethnicities was similar between both phases. Reasons for the lesser number of participants in Phase Two are: No consent for Phase Two, students unavailable (moved school, did not attend school on testing date) and participants could have bene unaware that Phase Two was a continuance of Phase One.

**Table 22. Subjects by phase, intervention, sex and ethnicity**

| Ethnicity | Phase One (n=169) | | Phase Two (n=103)* | |
|---|---|---|---|---|
| | Control (number of boys) | Programme (number of boys) | Control (number of boys) | Programme (number of boys) |
| European | 17 (8) | 17 (7) | 10 (4) | 15 (6) |
| Maori | 61 (36) | 58 (30) | 38 (22) | 28 (13) |
| Pacific | 3 (1) | 9 (5) | 2 (1) | 7 (4) |
| Other | 1 (1) | 3 (2) | 1 (1) | 2 (1) |

*Participants in Phase Two also participated in Phase One

### 5.6.4 Questionnaire

A questionnaire to determine demographic data, for example the date of birth, sex and ethnicity of the child, and the lifestyle of the child, such as their frequency of physical activity and intake of different foods, especially those known to contain vitamin D and calcium, was completed by the parent or caregiver. The questionnaire can be found in Appendix C.

### 5.6.5 Phase One procedure

All children were requested to fast overnight (~10 hrs) prior to Phase One. Upon arrival at school, on the morning of the assessment, participants were registered. For those providing blood samples, a topical anaesthetic cream (Amethocaine 4% w/w, Smith and Nephew, New Zealand) was applied to the antecubital area. Anthropometry, stiffness index and blood sampling in turn were subsequently assessed.

*Anthropometry*

As per chapter 3.5.3.

*Stiffness Index*

Ultrasound screening of the right os calcis (heel) was carried out using a portable quantitative ultrasound device (QUS, GE Achilles Insight, Lunar Corporation, Madison, WI, USA) and measurements made as described by Baroncelli (Baroncelli, 2008). The equipment and training in its use was provided by Fonterra. This measurement derives a stiffness index (StIn) from broadband ultrasound attenuation (BUA) expressed in dB/MHz and the speed of sound (SOS) expressed in m/s. StIn is a percentage of the mean value in a young adult, and is calculated from BUA and SOS in the following equation:

$$\text{Stiffness index (StIn)} = 0.67 \times (\text{BUA}) + 0.28 \times (\text{SOS}) - 420 \quad (9)$$

Although StIn may not predict bone mineral density, it shows good precision and can discriminate between "normal" and osteopenic children aged 6 to 13 years (Jaworski, Lebiedowski, Lorenc, & Trempe, 1995). Each child was visually pre-screened for abrasions or lesions on the right heel and where there were open sores or cuts, the left leg was used. Prior to QUS, the childs heel and ankle was sprayed liberally with a 70% alcohol solution and then immediately positioned into the plastic well of the ultrasound device. At the ankle, two "balloons" either side of the foot filled with warm water and enveloped the foot so that skin of the balloon and the foot formed a bond via the alcohol solution. Once the balloon-filling procedure was complete, the child was advised to remain motionless whilst the scan proceeded. The duration of each scan varied between 10 to 30 seconds dependent on the seal contact or the ability of the participant to remain still. After the scan, the child's foot was wiped clean with tissue and they continued on to the next assessment.

*Blood Biochemistry*

A mobile clinic was used to provide a private and comfortable room to obtain blood samples from the participants. An experienced and qualified paediatric phlebotomist from the Waikato District Health Board provided the service. A number of biochemical measures were derived from these samples. Serum 25-hydroxyvitamin D (vitamin $D_3$) was determined using an RIA kit (DiaSorin, Immunodiagnostics, PO Box 101 St Peters, NSW 2044 Australia), referenced to standards and averaged duplicates reported. Total cholesterol, HDL-C and triglyceride were determined using a homogeneous enzymatic colorimetric system (Roche modular P800, PO Box 62-089, Mt Wellington, Auckland). Low density lipoprotein cholesterol (LDL-C) was then derived using the formula: LDL-C= [total cholesterol - (triglyceride/2.2) – HDL-C]. Serum insulin levels were verified using a solid phase, 2 site chemiluminescent immunometric assay (Siemens, PO Box 14-046 Panmure 1741, Auckland). Following standard storage procedures samples were transported to the IANZ-accredited Waikato District Health Board laboratory for analysis. The HOMA (Homeostatic Model Assessment) is a mathematical model of beta-cell function and insulin sensitivity interactions that utilises fasting glucose and insulin values (Matthews, et al., 1985a). An updated model (HOMA2) and its

corresponding analysis of beta-cell function (HOMA2-B%) and insulin sensitivity (HOMA2-S%) was calculated using a dos-based application (Wallace, et al., 2004b).

### 5.6.6 Phase Two procedure

No fasting was required for this phase. This procedure involved the same measures of anthropometry (as described above) plus an additional assessment of physical fitness. By repeating the anthropometric measures from Phase One in Phase Two, within-subject differences between the two phases is reduced using change in anthropometry as an adjusting factor. Anthropometry was measured first, followed by the physical fitness assessment. All measurements were repeated twice except when the variance was too large (Ulijaszek & Lourie, 1994) and then they repeated a third time. Our technical error of measurement for Phase Two was height = 0.0075, BM =0.0016 and hand grip = 0.027.

*Physical Fitness*

Physical fitness, in this study, consisted of determining cardiorespiratory fitness and muscle strength fitness. Muscle strength was assessed by maximal forearm grip strength using a portable dynamometer (TTM, original dynamometer, Tokyo) held with a straight arm above the head whilst the participant stood against a wall. On the command "go" the participant squeezed the dynamometer trigger and brought the dynamometer down to their side in an 180° arc in the frontal plane maintaining a straight arm. This exercise was repeated on the opposite arm with two additional repetitions per side. Positive verbal encouragement was provided throughout by the tester. Following this test, CRF was determined using a 20m shuttle run test (20-m SRT) published by the Australian Coaching Council and validated by Anderson et al. (1992). The primary outcome of this test is to perform as many 20 m shuttles as possible in time with the audio signal which is played by a compact disc player to control the running speed between two points that are 20m apart. Early drop-out was avoided by holding this test as a group activity (other children encouraging their peers) and with an adult "pacer". The "pacer" remained in the test until the last child could not continue and positively encouraged all children to stay in the assessment for as long as they could. The test was stopped when the participant either voluntarily discontinued the test or they could not complete 2 consecutive laps within the time allowed. In both cases the previous recorded lap was used as their final lap. The first stage is started at a speed of 8.0 $km.h^{-1}$, the second stage is performed at a speed of 9.0 $km.h^{-1}$ and then increases by 0.5 $km.h^{-1}$

[1] every stage thereafter. Lap speed is the recommended and validated result utilised for analysis and is calculated using the following formula (Stone, 2007):

$$Vmax\ (km.h^{-1}) = V + Vstep \times (Rcom - Rtotal / 2) / Rtotal \quad (10)$$

Where V represents the velocity of the stage in $km.h^{-1}$; Vstep represents the increase in velocity between each stage in $km.h^{-1}$; Rcom represents the number of shuttle runs the participant completed during the stage; and Rtotal is the number of shuttle runs corresponding to the stage. Lap speed was calculated so that data from this study could be compared to international norms (Olds, Tomkinson, Léger, & Cazorla, 2006).

### 5.6.7 Statistical Analysis

All statistical analyses were performed using SPSS version 15 (SPSS Inc., Chicago IL.). The data file was split between male and female subjects and an exploration of normal distribution of the data was made using Kolmogorov-Smirnov distribution statistic. Age, height, calcium, LDL cholesterol, and HOMA2-S% were all normally distributed. Body mass, BMI, FM, Alkaline Phosphatase, HOMA2-B%, glucose, folate, insulin, quantitative ultrasound, and triglycerides were not normally distributed and were analysed accordingly. Male PBF, FFM, and Vit D and female FM, Total cholesterol and HDL cholesterol were identified as sex-specific variables which were not normally distributed. Data was not analysed for differences between ethnicities because the numbers in each ethnicity were very low. Table 22 shows that there was a 39.1% dropout of participants between Phase One and Phase Two, however the proportion of ethnicities was similar between both phases. A large proportion of the data contained outliers and were not normally distributed, thus the non-parametric Mann-Whitney U test was utilised to analyse differences in all descriptive and test data and presented as medians and interquartile ranges (IQR). Spearman's rho correlation of anthropometry, fitness parameters and biochemical markers of bone, cardiovascular and metabolic health was determined. Logistic regression was employed to estimate odds risk ratio for dependent variables HOMA2-B%, HOMA2-S% and stiffness index. Continuous outcome variables were converted into dichotomous categories for logistic regression analysis.

## 5.7 Results – Phase One for 169 children

Children who received milk were slightly, but significantly (P=0.006, Table 23) older than their control group peers. In addition, BMI SDS was significantly higher in milk children. No between-group differences in anthropometry were observed when analysed by intervention. Similar proportions of normal weight and overweight were found in both groups.

**Table 23. Age and anthropometry of Phase One subjects by intervention (N=169)**

| | Control (n=82) | Milk (n=87) | U Test | Sig. |
|---|---|---|---|---|
| Age (years) | 7.2 (6.6, 7.8) | 7.5 (7.1, 8.1) | 2693.0 | 0.006* |
| Ht (cm) | 125.4 (120.3, 129.1) | 127.0 (121.8, 130.9) | 3136.0 | 0.175 |
| Ht SDS | 0.44 (-0.37, 0.84) | 0.42 (-0.29, 0.90) | 3436.5 | 0.681 |
| BM (kg) | 26.6 (23.7, 31.2) | 28.2 (24.2, 32.2) | 3129.0 | 0.168 |
| BM SDS | 0.61 (0.01, 1.19) | 0.80 (0.29, 1.42) | 3038.0 | 0.096 |
| BMI ($kg.m^{-2}$) | 17.1 (16.0, 18.6) | 17.3 (16.1, 19.6) | 3244.0 | 0.310 |
| BMI SDS | 0.72 (0.08, 1.21) | 0.90 (0.38, 1.57) | 2933.0 | 0.046* |
| PBF (%) | 21.6 (19.2, 26.4) | 22.3 (19.1, 27.3) | 3291.0 | 0.385 |
| PBF SDS | 1.25 (0.65, 1.70) | 1.17 (0.54, 1.72) | 3397.0 | 0.593 |
| FFM (kg) | 21.2 (18.7, 23.4) | 21.8 (19.0, 24.5) | 3144.0 | 0.183 |
| FM (kg) | 5.7 (4.6, 7.7) | 6.4 (4.8, 8.3) | 3147.0 | 0.186 |
| Normal/OW/ Obese (%)† | 65.8/23.2/11.0 | 63.3/22.9/13.8 | | |

NB: Mann Whitney U Test - Mean (IQR) values displayed
†IOTF criteria OW = overweight
*significant $p<0.05$
**significant $p<0.001$

In milk children, HDL-C and HDL/LDL ratio was significantly higher than control children (Table 24). Vitamin D was also higher in milk children (P=0.01). No significant differences were observed between milk and control groups regarding variables related to glucose homeostasis.

**Table 24. Comparison of Phase One lipids, glucose homeostasis and bone measures by intervention**

| | Control (n=82) | Programme (n=87) | U Test | Sig. |
|---|---|---|---|---|
| Lipids | | | | |
| HDL-C (mmol/L) | 1.31 (1.14, 1.52) | 1.47 (1.20, 1.68) | 278.9 | 0.014* |
| LDL-C (mmol/L) | 2.10 (1.80, 2.60) | 2.10 (1.70, 2.50) | 3333.5 | 0.462 |
| HDL/LDL ratio | 0.62 (0.48, 0.80) | 0.68 (0.54, 0.94) | 2900.0 | 0.036* |
| TG (mmol/L) | 0.60 (0.50, 0.90) | 0.70 (0.50, 0.90) | 3192.5 | 0.236 |
| T-C (mmol/L) | 3.80 (3.38, 4.25) | 4.00 (3.50, 4.30) | 3250.0 | 0.318 |
| Glucose Homeostasis | | | | |
| Glucose (mmol/L) | 4.60 (4.30, 4.80) | 4.60 (4.30, 4.70) | 3541.0 | 0.934 |
| Insulin (IU/L) | 22.00 (14.00, 38.25) | 23.00 (14.00, 40.00) | 3194.5 | 0.232 |
| HOMA2-S (%) | 241.65 (136.53, 374.55) | 226.30 (131.50, 373.40) | 3180.5 | 0.224 |
| HOMA2-B (%) | 63.10 (52.30, 85.13) | 67.20 (53.20, 92.90) | 3249.5 | 0.318 |
| HOMA-IR | 49.01 (31.27, 85.11) | 51.27 (31.28, 88.98) | 3180.0 | 0.223 |
| Bone | | | | |
| QUS | 78.50 (73.00, 87.00) | 82.00 (77.00, 87.00) | 3062.0 | 0.112 |
| Vit D (nmol/L) | 42.00 (34.00, 54.00) | 48.00 (38.00, 59.00) | 2746.0 | 0.010* |
| Ca (mmol/L) | 2.43 (2.38, 2.48) | 2.43 (2.40, 2.48) | 3256.0 | 0.327 |
| PTH (pmol/L) | 3.40 (2.40, 4.20) | 3.10 (2.30, 4.10) | 2137.5 | 0.371 |

NB: Mann Whitney U Test applied; Median (IQR) values displayed
HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol; TG: triglycerides; T-C: Total Cholesterol; HOMA2-S: homeostatic model assessment - insulin sensitivity; HOMA2-B: homeostatic model assessment – beta cell function; HOMA-IR: homeostatic model assessment - insulin resistance; QUS: Quantitative Ultrasound; VIt D: 25-hydroxy Vitamin D; Ca: Calcium; PTH: parathyroid hormone.
*significant $p<0.05$
**significant $p<0.001$

The absolute PBF (%) and FM (kg) was significantly lower in boys than girls (Table 25), however no significant difference was found in PBF SDS, which is age and sex adjusted.

**Table 25. Age and anthropometry of Phase One subjects by sex**

| | Boys (n=90) | Girls (n=79) | U Test | Sig. |
|---|---|---|---|---|
| Age (years) | 7.4 (6.9, 8.0) | 7.4 (6.9, 7.9) | 3440.5 | 0.718 |
| Ht (cm) | 125.8 (121.5, 129.6) | 126.9 (121.3, 131.4) | 3359.5 | 0.538 |
| Ht SDS | 0.52 (-0.45, 0.93) | 0.26 (-0.27, 0.76) | 3296.0 | 0.414 |
| BM (kg) | 26.3 (23.8, 30.9) | 29.0 (24.3, 32.6) | 2956.0 | 0.059 |
| BM SDS | 0.68 (0.25, 1.30) | 0.78 (0.04, 1.36) | 3528.5 | 0.934 |
| BMI ($kg.m^{-2}$) | 17.0 (15.8, 18.3) | 17.6 (16.1, 20.2) | 3008.0 | 0.085 |
| BMI SDS | 0.90 (0.32, 1.31) | 0.77 (0.27, 1.35) | 3440.0 | 0.717 |
| PBF (%) | 20.7 (18.5, 22.6) | 25.4 (21.5, 29.0) | 1927.0 | <0.001** |
| PBF SDS | 1.16 (0.59, 1.58) | 1.25 (0.49, 1.77) | 3297.5 | 0.417 |
| FFM (kg) | 21.3 (18.8, 24.4) | 21.5 (18.9, 23.8) | 3541.0 | 0.965 |
| FM (kg) | 5.4 (4.5, 6.5) | 6.9 (5.6, 9.7) | 2184.0 | <0.001** |

NB: Mann Whitney U Test - Median (IQR) values displayed
*significant $p<0.05$
**significant $p<0.001$

Girls had significantly higher LDL-C and total cholesterol than boys (Table 25). Significant differences in glucose homeostasis were also observed with lower glucose concentrations and higher beta-cell function in girls. Concentrations of calcium were also higher in girls. However, after ANCOVA adjustment for intervention and age the differences did not exist, except for glucose, between boys and girls.

**Table 26. Comparison of lipids, glucose homeostasis and bone measures by sex**

| | Boys (n=90) | Girls (n=79) | U Test | Sig |
|---|---|---|---|---|
| Lipids | | | | |
| HDL-C (mmol/L) | 1.42 (1.21, 1.58) | 1.34 (1.13, 1.59) | 3295.5 | 0.413 |
| LDL-C (mmol/L) | 2.0 (1.6, 2.43) | 2.3 (1.9, 2.6) | 2852.0 | 0.027* |
| HDL/LDL ratio | 0.68 (0.55, 0.93) | 0.64 (0.48, 0.82) | 2968.5 | 0.065 |
| TG (mmol/L) | 0.6 (0.4, 0.9) | 0.7 (0.5, 0.9) | 2945.0 | 0.530 |
| T-C (mmol/L) | 3.8 (3.3, 4.2) | 4.0 (3.6, 4.4) | 2834.0 | 0.023* |
| Glucose Homeostasis | | | | |
| Glucose (mmol/L) | 4.6 (4.5, 4.8) | 4.5 (4.2, 4.7) | 2808.5 | 0.018* |
| Insulin (IU/L) | 20 (14, 34) | 25 (14, 43) | 2998.5 | 0.074 |
| HOMA2-S (%) | 256.9 (155.0, 374.3) | 209.6 (121.8, 374.3) | 3171.0 | 0.226 |
| HOMA2-B (%) | 61.7 (48.3, 83.75) | 74.8 (55.9, 99.6) | 2627.5 | 0.003* |
| HOMA-IR | 45.7 (31.3, 75.7) | 55.7 (31.3, 95.7) | 3172.0 | 0.227 |
| Bone | | | | |
| QUS | 80 (75, 86) | 82 (76, 88) | 3147.0 | 0.198 |
| Vit D (nmol/L) | 45.5 (37.0, 55.3) | 45.0 (35.0, 59.0) | 3539.5 | 0.961 |
| Ca (mmol/L) | 2.4 (2.4, 2.5) | 2.4 (2.4, 2.5) | 2882.5 | 0.034* |
| PTH (pmol/L) | 3.2 (2.33, 4.18) | 3.4 (2.35, 4.5) | 2222.0 | 0.677 |

NB: Mann Whitney U Test applied; Median (IQR) values displayed
HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol; TG: triglycerides; T-C: Total Cholesterol; HOMA2-S: homeostatic model assessment - insulin sensitivity; HOMA2-B: homeostatic model assessment – beta cell function; HOMA-IR: homeostatic model assessment - insulin resistance; QUS: Quantitative Ultrasound; VIt D: 25-hydroxy Vitamin D; Ca: Calcium; PTH: parathyroid hormone.
*significant $p<0.05$
**significant $p<0.001$

The TG concentration was significantly lower in non-obese children, but no other lipid variable differed between obese and non-obese groups (See Table 27). Comparison of bone measurements revealed no significant differences. In contrast, apart from glucose concentration, glucose homeostasis variables displayed many significant differences between the obese and non-obese children. Specifically, obese children had higher insulin, HOMA2-B% and HOMA-IR than non-obese children. Conversely, HOMA2-

S% was significantly lower in obese children. Adjustment for intervention, age and sex could not explain differences in TG, insulin and HOMA2-S%.

**Table 27. Phase One biochemistry [median (IQR)] of children by IOTF obesity classification**

| | Not Obese (n=136) | Obese (n=32) | U Test | Sig. |
|---|---|---|---|---|
| Lipids | | | | |
| HDL-C (mmol/L) | 1.40 (1.21, 1.60) | 1.24 (1.06, 1.53) | 1720.5 | 0.058 |
| LDL-C (mmol/L) | 2.1 (1.7, 2.5) | 2.3 (1.9, 2.6) | 1830.5 | 0.146 |
| HDL/LDL ratio | 0.67 (0.52, 0.88) | 0.63 (0.43, 0.75) | 1754.0 | 0.079 |
| TG (mmol/L) | 0.6 (0.5, 0.8) | 0.9 (0.6, 1.2) | 1329.0 | <0.001** |
| T-C (mmol/L) | 0. 7 (0.5, 0.9) | 0.6 (0.4, 0.8) | 1754.0 | 0.079 |
| Glucose Homeostasis | | | | |
| Glucose (mmol/L) | 4.60 (4.30, 4.80) | 4.65 (4.33, 4.80) | 2053.0 | 0.570 |
| Insulin (IU/L) | 19 (14, 33) | 39 (27, 61) | 1003.5 | <0.001** |
| HOMA2-S (%) | 274.8 (156.6, 375.3) | 135.0 (76.6, 190.2) | 998.5 | <0.001** |
| HOMA2-B (%) | 62.0 (52.1, 80.8) | 97.8 (74.8, 120.6) | 960.0 | <0.001** |
| HOMA-IR | 42.4 (31.3, 74.6) | 87.9 (61.3, 135.6) | 999.0 | <0.001** |
| Bone | | | | |
| QUS | 80 (76, 87) | 83 (74, 95) | 1951.0 | 0.333 |
| Vit D (nmol/L) | 46.0 (36.0, 58.5) | 43.5 (38.0, 46.7) | 1823.5 | 0.139 |
| Ca (mmol/L) | 3.4 (2.5, 4.5) | 2.8 (2.1, 3.7) | 1326.0 | 0.072 |
| PTH (pmol/L) | 2.4 (2.4, 2.5) | 2.5 (2.4, 2.5) | 1758.0 | 0.081 |

NB: Mann Whitney U Test applied; Median (IQR) values displayed
HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol; TG: triglycerides; T-C: Total Cholesterol; HOMA2-S: homeostatic model assessment - insulin sensitivity; HOMA2-B: homeostatic model assessment – beta cell function; HOMA-IR: homeostatic model assessment - insulin resistance; QUS: Quantitative Ultrasound; VIt D: 25-hydroxy Vitamin D; Ca: Calcium; PTH: parathyroid hormone.
*significant $p<0.05$
**significant $p<0.001$

A low response (98 of 168) to food and physical activity items in the questionnaire prevented any formal statistical analysis. A description of the results is provided (Table 28) Comparison of food and activity patterns by intervention group showed that the

milk group reported on average consuming more milk and fizzy drink than the control group; however they consumed less cheese, ice cream, water and yoghurt. Schools reported high compliance however no record was kept for individual children. Control children appeared to be more time in inactivity and less time active than milk children.

**Table 28. Descriptive patterns of food, activity and inactivity by intervention group[a].**

| | Control (n=42) | Programme (n=56) |
|---|---|---|
| Cheese (times/day) | 0.340 (0.005, 2.00) | 0.250 (0.005, 2.000) |
| Fizzy drink (times/day) | 0.132 (0.005, 0.786) | 0.163 (0.005, 2.000) |
| Food fizzy drink (times/day) | 0.095 (0.005, 0.429) | 0.068 (0.005, 0.429) |
| Ice cream (times/day) | 0.188 (0.005, 0.786) | 0.125 (0.005, 1.000) |
| Total milk (times/day) | 3.622 (0.348, 6.148) | 4.558 (1.072, 8.000) |
| Water (times/day) | 0.214 (0.005, 2.000) | 0.077 (0.005, 0.786) |
| Yoghurt (times/day) | 0.503 (0.005, 1.000) | 0.384 (0.005, 2.000) |
| Total weekly physical inactivity (hours per week) | 22.9 (5.5, 51.0) | 22.5 (4.5, 58.5) |
| Total physical activity (times/day) | 2.277 (0.225, 4.515) | 2.671 (0.638, 4.867) |

[a]Means (minimum, maximum) displayed

When split by obesity status, obese children reported a higher mean frequency of cheese consumption, but no other food, than the not obese group. Physical inactivity was also higher in obese children and the frequency of physical activity slightly less than not obese children.

**Table 29. Descriptive patterns of food, activity and inactivity by IOTF obesity classification[a].**

| | Not Obese (n=80) | Obese (n=18) |
|---|---|---|
| Cheese (times/day) | 0.273 (0.005, 2.00) | 0.36 (0.005, 2.000) |
| Fizzy drink (times/day) | 0.153 (0.005, 2.000) | 0.134 (0.005, 0.429) |
| Diet fizzy drink (times/day) | 0.082 (0.005, 0.429) | 0.069 (0.005, 0.492) |
| Ice cream (times/day) | 0.154 (0.005, 1.000) | 0.141 (0.005, 0.786) |
| Total milk (times/day) | 4.200 (0.348, 7.429) | 4.055 (1.072, 8.000) |
| Water (times/day) | 0.142 (0.005, 2.000) | 0.104 (0.005, 0.786) |
| Yoghurt (times/day) | 0.451 (0.005, 2.000) | 0.365 (0.005, 1.000) |
| Total weekly physical inactivity (hours per week) | 22.3 (4.5, 51.0) | 24.4 (4.5, 58.5) |
| Total physical activity (times/day) | 2.506 (0.291, 4.500) | 2.457 (0.225, 4.867) |

[a]Means (minimum, maximum) displayed

### 5.7.1 Relationships between key measures

Spearman's rho correlations were used to explore paired relationships of the significantly different variables related in Table 23 to Table 26. Milk consumption frequency was positively related to HDL-C (rho=0.30, p=0.0045) and physical activity (rho=0.29, p=0.03) but negatively related to fizzy drink consumption (rho=-0.28, p=0.01). In contrast, no variable of body mass, glucose homeostasis, or bone integrity was associated with milk frequency.

The stiffness index was not associated with any food or activity variable, including total milk consumption frequency ($p$=0.657). The BMI SDS (rho=0.21, $p$=0.005) appeared to be a significant positive correlate with bone integrity, however PBF SDS was nonsignificant ($p$=0.051). Absolute insulin (rho=0.16, p=0.043) and HOMA2-B% (rho=0.17, $p$=0.026) were positive correlates with StIn but the relationship with HOMA2-S% was not significant (p=0.12).

The glucose homeostasis variables of glucose, insulin, HOMA2-B% and HOMA2-S% had significant relationships with SDS of height, weight, BMI, and PBF, as well as absolute fat free and fat mass (Table 30). Insulin and HOMA2-S% (a calculated derivative of insulin) were strongly related, positively and negatively respectively, with all measures of mass. Absolute insulin and HOMA2-S% had similar strengths of

relationships with measures of mass, although insulin was slightly stronger against FFM and FM. Because of this widespread strength of significance of association and the absence of the need for calculations to obtain an insulin result (compared with HOMA2) absolute insulin was selected as the best measure that represented the relationship between glucose homeostasis and mass.

**Table 30. Spearman's rho correlates (sig.) between glucose homeostasis variables (glucose, insulin, HOMA2-S% and HOMA2-B%) and measures of mass.**

| | Glucose (mmol/L) | Insulin (IU/L) | HOMA2-S (%) | HOMA2-B (%) |
|---|---|---|---|---|
| Ht SDS | 0.22 (0.005)* | 0.17 (0.029)* | -0.17 (0.023)* | 0.10 (0.194) |
| BM SDS | 0.18 (0.017)* | 0.20 (0.009)* | -0.21 (0.008)* | 0.13 (0.083) |
| BMI SDS | 0.13 (0.09) | 0.17 (0.032)* | -0.17 (0.027)* | 0.11 (0.139) |
| PBF SDS | 0.09 (0.262) | 0.24 (0.002)* | -0.24 (<0.001)** | 0.21 (0.005)* |
| FFM (kg) | 0.20 (0.01)* | 0.45 (<0.001)** | -0.44 (<0.001)** | 0.39 (<0.001)** |
| FM (kg) | 0.14 (0.07) | 0.45 (<0.001)** | -0.43 (<0.001)** | 0.41 (<0.001)** |

Ht: Height; BM: Body Mass; BMI: Body Mass Index; PBF: Percentage body fat; FFM: Fat free mass; FM: fat mass; HOMA2-S: Homeostatic model assessment – insulin sensitivity; HOMA2-B: Homeostatic model assessment – beta cell function;
*significantly different ($p<0.05$)
**significantly different ($p<0.001$)

### 5.7.2 Logistic regression of Insulin

Obese children tended to have lower insulin sensitivity and beta cell function than non obese (See Figure 13A). This was also borne out by PBF SDS. However children with high BMI SDS (BMI SDS of 2 or more) were more evenly spread throughout the group (See Figure 13C). This even spread for BMI SDS and aggregation for PBF SDS indicated a PBF influence. To confirm this, logistic regression was applied to insulin.

Insulin appeared to be the most significant biochemical variable associated with BMI SDS and PBF SDS. In addition, PBF SDS was the only Phase One variable to be significantly ($p$=0.002) related to insulin (Figure 12). Further, PBF SDS was the best logistic regression variable to predict stiffness index (OR=1.79, 95% CI 1.23, 2.59).

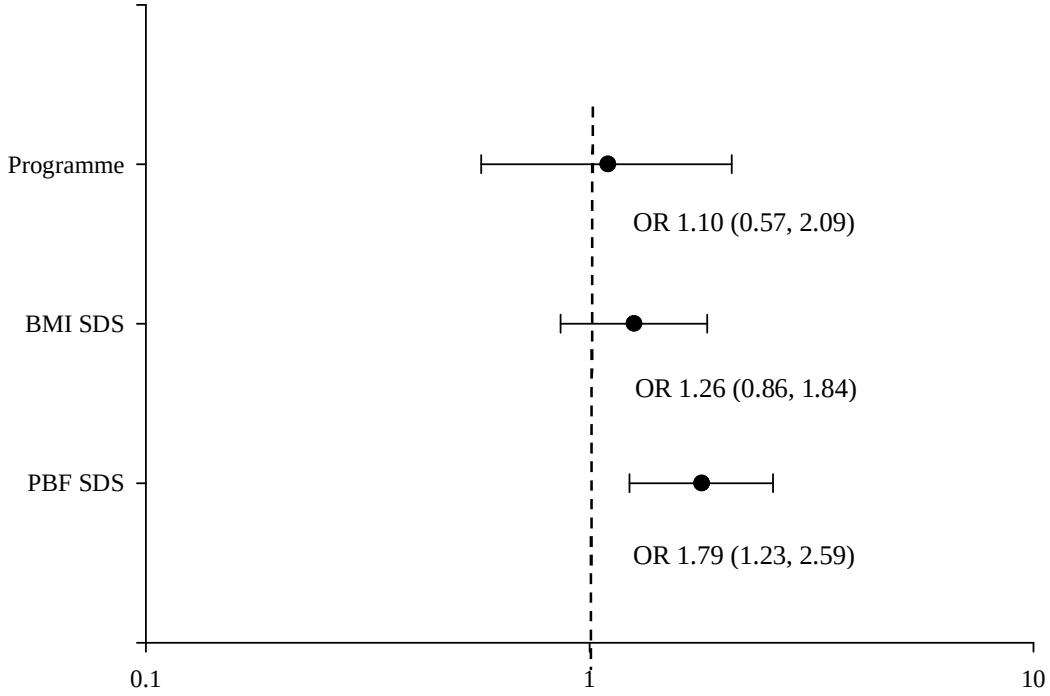


**Figure 12. Odds risk (95% CI) of variables to predict insulin ≥23 IU/L**

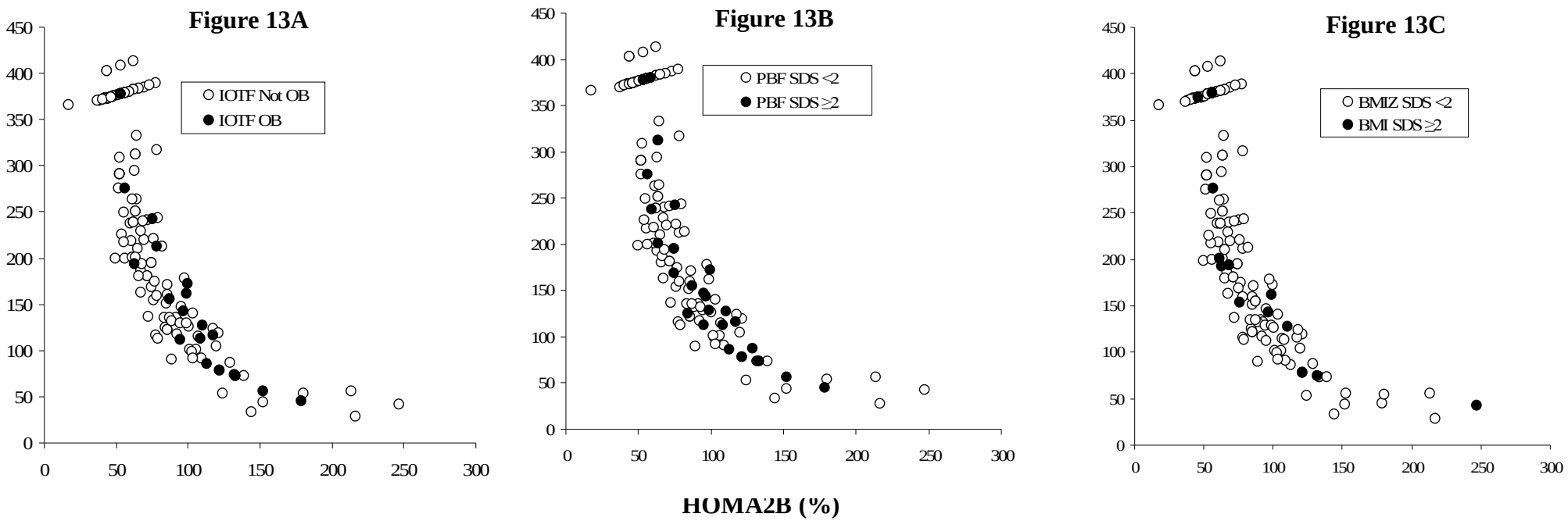


**Figure 13. HOMA2S and HOMA2B represented in three figures defined by IOTF (A) , PBF SDS (B), and BMI SDS (C)**
Black dots represent obese (n=32) in figure 9A, participants with PBF SDS 2 or more (n=27) in figure 9B, participants with BMI SDS 2 or more (n=13) in figure 9C and clear dots represent all others.

## 5.8 Results – Phase Two for 98 children

A total of 98 children (female = 49.0%) participated in all aspects of this study (Phase One and Phase Two). As explained in 5.6.1, this phase was defined by the introduction of fitness parameter measures, the 20m SRT as a measure of CRF and handgrip dynamometry as a measure for MSF. The age and anthropometry of the Phase Two participants were not significantly different from the Phase One children that did not participate in Phase Two (Table 31). The programme group was significantly older than controls, which is consistent with Phase One. There were no other demographic or anthropometric differences between groups.

Phase Two programme children had a significantly higher vitamin D concentration than Phase Two control children. TG was the only lipid variable to be significantly higher in obese children and this did not change after adjustment for sex and intervention group. Glucose homeostasis results for obese children were all significantly less functional than those for not obese children except for glucose itself. Bone data were not significantly different between IOTF obesity classifications. Of the fitness parameters only the muscle strength fitness was significantly higher in obese children. No significant difference in CRF was observed by IOTF obesity classification.

Figure 14 relates the comparison of Phase Two 20m SRT results with international means. The international results do not include 5 years old children in their data, however the Phase Two 5 year old data is displayed to demonstrate differences in CRF between age and sex. Phase Two CRF in boys steadily increased from 5 years of age with a small decline at 8 years of age. The trend to higher CRF continues at 9 years of age. In Phase Two girls, from a high 9.38 $km.h^{-1}$ at 5 years of age, there is a drop in final lap running speed at 6 years of age (9.25 $km.h^{-1}$). From 6 years of age, however, the mean final lap running speed steadily improves to 8 years of age (10.06 $km.h^{-1}$). Up until 8 years of age, both Phase Two boys and girls record faster mean final lap running speeds than their international peers. At 9 years of age, the final mean running speeds of both Phase Two and international boys are very similar.

**Table 31. Descriptive characteristics of participants by phase and by intervention.**

| | Phase | | | | Intervention | | | |
|---|---|---|---|---|---|---|---|---|
| | Phase One (n=70) | Phase Two (n=98) | U Test | Sig. | Control (n=49) | Programme (n=49) | U Test | Sig. |
| Age (years) | 7.5 (7.1, 8) | 7.3 (6.8, 7.8) | 2921.5 | 0.102 | 7.0 (6.5, 7.8) | 7.5 (7.0, 8.1) | 844.5 | 0.011* |
| Height (cm) | 126.0 (121.7, 129.9) | 126.3 (121.3, 130.8) | 3423.0 | 0.982 | 124.9 (120.5, 128.8) | 127.9 (121.7, 131.8) | 1010.5 | 0.177 |
| Height SDS | 0.25 (-0.50, 0.84) | 0.50 (-0.18, 0.96) | 2831.5 | 0.054 | 0.50 (-0.10, 0.91) | 0.47 (-0.25, 1.12) | 1184.5 | 0.909 |
| Weight (kg) | 26.8 (23.5, 31.2) | 28.3 (24.1, 32.4) | 3126.5 | 0.329 | 26.8 (24.0, 33.2) | 29.5 (24.2, 31.5) | 1125.5 | 0.594 |
| Weight SDS | 0.55 (0.01, 1.15) | 0.80 (0.23, 1.43) | 2861.0 | 0.067 | 0.67 (0.21, 1.27) | 0.94 (0.36, 1.46) | 1044.0 | 0.266 |
| BMI (kg.m2) | 17.0 (15.7, 18.8) | 17.4 (16.2, 19.2) | 2970.0 | 0.139 | 17.4 (16.1, 20.2) | 17.3 (16.4, 18.9) | 1195.0 | 0.969 |
| BMI SDS | 0.74 (0.08, 1.29) | 0.86 (0.37, 1.37) | 3024.0 | 0.191 | 0.75 (0.23, 1.27) | 0.92 (0.49, 1.56) | 1011.5 | 0.179 |
| PBF (%) | 22.3 (19.1, 26.7) | 22.1 (19.5, 26.2) | 3356.0 | 0.812 | 22.2 (19.5, 27.1) | 22.0 (18.8, 25.6) | 1186.0 | 0.918 |
| PBF SDS | 1.23 (0.66, 1.62) | 1.22 (0.47, 1.72) | 3392.0 | 0.903 | 1.27 (0.74, 1.82) | 1.17 (0.26, 1.71) | 1055.0 | 0.301 |
| Fat Free Mass (kg) | 20.9 (18.7, 23.4) | 21.8 (18.9, 24.3) | 3059.0 | 0.233 | | | | |
| Fat Mass (kg) | 5.9 (4.7, 7.6) | 5.9 (4.8, 8.8) | 3342.0 | 0.777 | | | | |

NB: Mann Whitney U Test applied; Median (IQR) values displayed
*significant $p<0.05$
**significant $p<0.001$

**Table 32. Metabolic, lipid, bone and fitness results for Phase Two children by intervention group and IOTF cutoff**

| | Inetervention | | | | IOTF cut-off | | | |
|---|---|---|---|---|---|---|---|---|
| | Control (n=49) | Programme (n=49) | U-Test | Sig. | Not Obese (n=76) | Obese (n=22) | U-Test | Sig. |
| HDL (mmol/L) | 1.36 (1.22, 1.55) | 1.47 (1.2, 1.7) | 1039.5 | 0.253 | 1.46 (1.22, 1.68) | 1.28 (1.13, 1.52) | 606.0 | 0.050 |
| LDL (mmol/L) | 2.1 (1.8, 2.7) | 2.1 (1.75, 2.45) | 1121.5 | 0.574 | 2.1 (1.8, 2.5) | 2.3 (2.0, 2.6) | 711.5 | 0.288 |
| TG (mmol/L) | 0.6 (0.5, 0.9) | 0.6 (0.5, 0.8) | 1171.5 | 0.835 | 0.6 (0.5, 0.8) | 0.85 (0.6, 1.1) | 506.0 | 0.005* |
| T-C (mmol/L) | 3.9 (3.45, 4.3) | 4 (3.5, 4.3) | 1142.0 | 0.677 | 3.9 (3.5, 4.3) | 4.1 (3.6, 4.3) | 756.5 | 0.498 |
| HDL/LDL ratio | 0.63 (0.48, 0.75) | 0.67 (0.52, 0.89) | 996.5 | 0.147 | 0.68 (0.50, 0.86) | 0.60 (0.46, 0.75) | 657.0 | 0.127 |
| Glucose (mmol/L) | 4.5 (4.25, 4.8) | 4.6 (4.3, 4.75) | 1094.0 | 0.447 | 4.5 (4.2, 4.7) | 4.7 (4.4, 4.8) | 636.0 | 0.087 |
| Insulin (IU/L) | 21 (14, 41.5) | 25 (14, 44) | 1077.0 | 0.369 | 17 (14, 37) | 46 (30, 72) | 343.0 | <0.001** |
| HOMA2-S (%) | 250.6 (127.25, 377.6) | 209.6 (119.25, 374.3) | 1067.0 | 0.343 | 299.6 (141.7, 377.6) | 114.3 (73.4, 174.5) | 332.0 | <0.001** |
| HOMA2-B (%) | 63.7 (53.2, 98.6) | 69.1 (53.2, 99.2) | 1130.5 | 0.619 | 61.7 (52.2, 82.6) | 108.8 (81.1, 132.2) | 340.5 | <0.001** |
| HOMA IR | 46.81 (31.26, 92.3) | 55.69 (31.27, 97.87) | 1066.5 | 0.341 | 39.0 (32.3, 83.4) | 103.4 (67.9, 160.6) | 332.5 | <0.001** |
| QUS | 81 (73, 93) | 83 (76, 88) | 1153.0 | 0.736 | 82 (75, 88) | 83 (74, 97) | 751.0 | 0.469 |
| Ca (mmol/L) | 2.44 (2.38, 2.49) | 2.43 (2.39, 2.49) | 1172.0 | 0.839 | 2.43 (2.38, 2.48) | 2.44 (2.40, 2.51) | 717.5 | 0.312 |
| Vitamin D (nmol/L) | 42 (34, 51.5) | 50 (38.5, 62) | 849.0 | 0.012* | 47.5 (37, 58) | 43.5 (38, 46.25) | 676.5 | 0.174 |
| PTH (pmol/L) | 3.5 (2.4, 5.15) | 3.2 (2.3, 3.8) | 674.0 | 0.211 | 3.4 (2.4, 4.6) | 3.3 (2.0, 3.8) | 541.5 | 0.253 |
| MSF (kg) | 14.5 (12.0, 16.0) | 15.0 (13.0, 17.0) | 1018.5 | 0.195 | 14.5 (12.0, 16.0) | 15.5 (14.0, 17.0) | 620.5 | 0.066 |
| CRF ($km.h^{-1}$) | 9.38 (9.28, 9.92) | 9.69 (9.25, 10.31) | 1000.0 | 0.154 | 9.50 (9.31, 10.08) | 9.38 (9.00, 9.81) | 652.5 | 0.118 |
| $VO_2max$ ($ml.kg^{-1}.min^{-1}$) | 49.2 (47.6, 51.0) | 49.1 (46.6, 52.0) | 1150.0 | 0.851 | 49.7 (47.6, 51.6) | 47.9 (47.2, 50.6) | 641.0 | 0.113 |

NB: Mann Whitney U Test applied; Median (IQR) values displayed

HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol; TG: triglycerides; HOMA-IR: homeostatic model assessment - insulin resistance; Ca: Calcium; PTH: parathyroid hormone; MSF: muscle strength fitness; CRF: cardiorespiratory fitness; $VO_2max$: Maximal oxygen uptake *significant $p<0.05$; **significant $p<0.001$

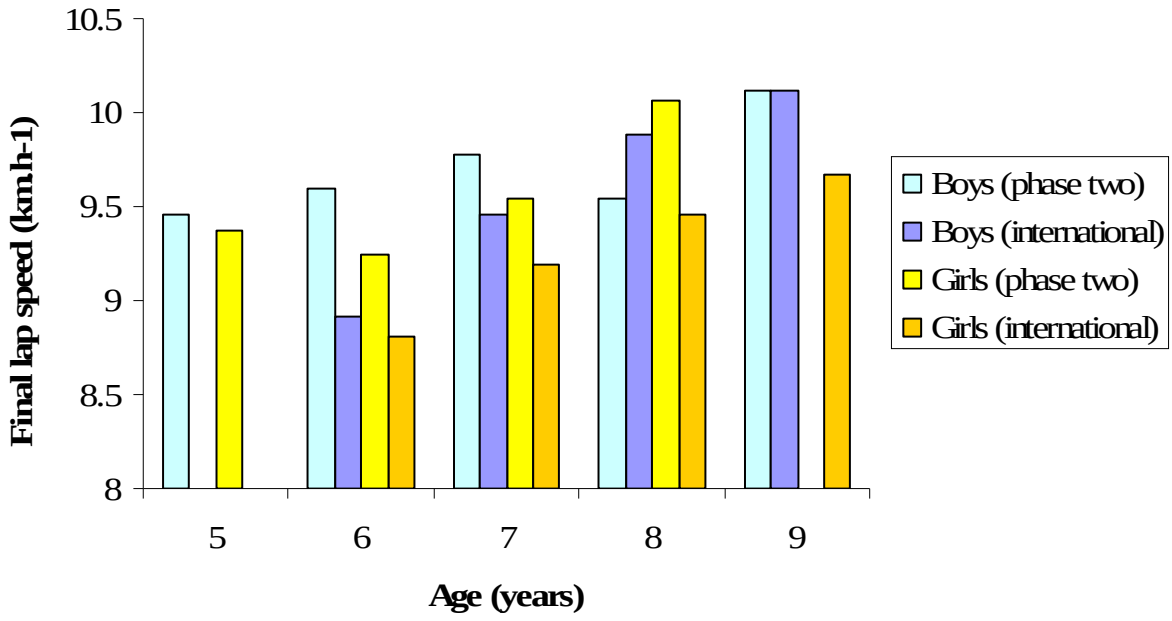


**Figure 14. Phase Two 20m SRT average means compared to international means by age and sex**

### 5.8.1 Relationships between key measures

Spearman's rho correlations revealed a low to moderate negative relationship between Phase Two BMI SDS and CRF (rho=-0.23, $p$=0.022) and a positive relationship with MSF (rho=0.28, $p$=0.005). Additionally, MSF was positively correlated with weight SDS (rho=0.41, $p$<0.001), but not CRF score. Phase Two PBF SDS was not related to either CRF or MSF.

The BM SDS, BMI SDS and PBF SDS data for both Phase One and Phase Two were positively correlated (rho=0.52, $p$<0.001; rho=0.50, $p$ <0.001; rho=0.88, $p$ <0.001 respectively) after 15 weeks. Phase One biochemical data was compared by correlation with Phase Two anthropometry and fitness parameter data and the following correlations were found. MSF was inversely related to insulin sensitivity (rho=-0.28, $p$=0.005). Positive relationships were found between MSF and BMI SDS (rho=0.25, $p$=0.013), insulin (rho=0.27, $p$=0.007), and QUS (rho=0.28, $p$=0.005).

### 5.8.2 Logistic regression of Insulin

Phase Two BMI SDS was the only variable to provide a significant odds ratio (OR=2.11, CI 95% 1.21, 3.68) for insulin (cutoff at the 50$^{th}$ percentile for this group ≤23 IU/L). Other nonsignificant covariates included in the analysis were age, sex, and intervention group. Fitness parameters were also among the nonsignificant variables.

MSF outweighed all other Phase Two variables (BMI SDS, PBD SDS, and CRF) in logistic regression analysis (OR=1.21, CI 95% 1.05, 1.38) to be the sole factor to explain QUS (cutoff at $50^{th}$ percentile of stiffness index ≤81).

### 5.9 Discussion

This two-part study (Phase One n=169, Phase Two n=98), of pre-pubertal boys and girls shows that:

1. Frequent and regular consumption of a fortified milk product:
    a. will result in higher vitamin D and HDL-C, but not lower triglycerides and LDL-C
    b. does not result in an enhanced stiffness index as measured by QUS
    c. does not result in lower BMI and PBF SDS
    d. is not associated with fitness (CRF or MST)
2. Children classified as obese:
    a. have adverse glucose homeostasis profiles
    b. have poorer fitness test performance than children classified as not obese
3. Fitness parameters:
    a. were not associated with BMI and PBF SDS
    b. of MST, but not CRF, were positively associated beta-cell function and inversely associated with insulin sensitivity.
    c. of MST, but not CRF, were positively associated with StIn.

The results from this study suggest that the children that consumed the fortified milk product had better vitamin D and lipid profiles than their control counterparts. This significant difference is reported in a published article (Graham, et al., 2009) and will not be discussed further in this thesis. Frequent and regular consumption of the fortified milk product did not result in the expected elevated stiffness index values, however Phase One PBF SDS appeared to predict stiffness index. In Phase Two, fitness parameters were introduced as explanatory variables and by including covariates of adjustments for change in height and weight between Phase One and two, MSF was calculated to be the sole predictor for stiffness index.

### 5.9.1 Intervention

The consumption of milk may be perceived to be a healthful habit; certainly milk intake (and also water) is currently promoted by the Ministry of Health as a healthy alternative to other fluids (Ministry of Health, 2008a). As none of the food or physical activity patterns reported were related to stiffness index, BMI SDS or PBF SDS, or to fitness parameters, it is conceivable that milk may not be as powerful a food as first thought.

The lack of efficacy of the milk intervention could be due to an association of milk consumption with biological changes may have a "tipping point" such as a daily or weekly target where change may be observed. This point may not have been attained by the milk children. Alternatively, a combination of physical activity and milk consumption may also be needed to make full use of the food. Compliance for milk consumption was not recorded, and although there are anecdotal reports from schools that the milk was widely accepted and consumed, there is no physical evidence of the amount, frequency or proportion of programme children that drank the intervention milk. Possible intervention design issues are discussed in Limitations and strengths (5.9.6).

### 5.9.2 Glucose homeostasis

Obese children recorded lesser Insulin and HOMA2 measures compared to not obese children, with the exception of glucose itself (no difference). This phenomenon, and Phase One insulin predictor – PBF SDS, demonstrate that an elevated insulin concentration was closely associated with the fat content of children. An explanation of why PBF SDS was the defining variable for insulin in Phase One, yet with the addition of the fitness parameters in Phase Two BMI SDS was identified as the sole predictor for insulin, is difficult to elucidate. Body fat, represented as PBF SDS, is known to have a physiological influence on insulin dynamics (Wagenknecht et al., 2003).

BMI SDS however is a different representation of body mass to body fat as it includes both fat and lean body tissue. Perhaps when there is a large body mass, there are less insulin receptors and a consummate increase in insulin production is required to compensate. This may result in resistance to the action of insulin. No variables other than BMI SDS and PBF SDS had the strength to bear influence over glucose homeostasis.

BMI SDS and PBF SDS strength of influence over insulin reveals an important issue – the action of insulin on lean tissue in young children may not behave in the same way as

in adolescents and adults. Myoblast cultures from pre-pubescent children show very different insulin-related characteristics than those cells from adults (Grohmann et al., 2005). Furthermore, Dunger et al. (2006) point out that puberty attenuates insulin action on muscle and that this is a normal part of growth and development. Thus the effect of fitness may be more pronounced on the peri-pubertal insulin resistance muscle in year 1 and year 5 children (Kriemler et al., 2008).

The relationship between fitness and glucose homeostasis variables, particularly insulin, was also enlightening. Insulin was positively related to MSF but had no relationship with CRF. Thus CRF and MSF may confer no glucose homeostasis benefit to this group of young children. Interestingly the obese children in this study had a similar CRF to the not obese children and tended to be stronger than their peers. In most studies of children and CRF, obese children are considered to have lower levels of fitness (Andersen et al., 2008; Ortega, et al., 2008). However few of these studies include children aged less than 9 years.

Where it has been demonstrated that the glucose homeostasis of adolescents and adults reacts positively to the effect of activity, fitness and food interventions (Ruiz, et al., 2006), this phenomenon cannot be demonstrated in young children from the data in this study. Excess mass (fat and lean) in young children appears to regulate glucose homeostasis with much more effect than functional and environmental factors such as physical activity, fitness and food patterns. There may be two reasons for this:

1. the lean active tissue in young children is not sufficiently developed (Grohmann, et al., 2005) to receive the metabolic benefit of increased physical activity, fitness and healthy food or;
2. the effect of mass is so very strong that it effectively cancels out the benefit of physical activity, fitness and healthy food when mass passes a certain tipping point (Blair, et al., 2007).

Upon the emergence into puberty, a morphological change occurs in lean active tissue and the benefits of fitness, physical activity and healthy food may be amplified to a level that can challenge the influence of mass. For children who are obese long-term, the benefits may be difficult to find in adolescence as excess mass may attenuate the effectiveness of fitness, physical activity and healthy food (Huus, Ludvigsson, Enskär, & Ludvigsson, 2007). Thus it is imperative that interventions begin at an early age to ensure maximal effect. The list of possible confounders and influences could literally be endless. Confounders are a typical characteristic of a naturalistic study. Tighter control

of the distribution and observation of the consumption of the food product may possibly have resulted in a more favourable outcome for the hypotheses presented in this study.

### 5.9.3 Obese and Non-Obese children

The International Obesity Taskforce obesity classification was implemented to classify the children from the study into obese and not obese groups (Cole, et al., 2000). While few differences were observed by intervention and sex, the obesity group classification revealed some other important findings.

*Lipid profile*

Specifically, obese children had higher triglyceride concentrations than not obese children. Elevated triglycerides are an important cardiovascular risk factor (Berenson, Srinivasan, Bao, Newman, Tracy, & Wattigney, 1998), however no other lipid variable differed between obese and non-obese groups (Table 27). Although elevated serum triglycerides may indicate the extent of atherosclerosis in young adults (Berenson et al., 1998), it is much more difficult to demonstrate this finding in young children (less than 10 years of age). Both Freedman (1999) and Botton (Botton et al., 2007) found higher levels of triglycerides in overweight and obese children than normal weight children. High triglycerides may also indicate a diet high in carbohydrate (Casazza, Dulin-Keita, Gower, & Fernandez, 2009).

*Glucose homeostasis*

Glucose homeostasis variables, with the exception of glucose itself, displayed many significant differences between the obese and non-obese children. Specifically, obese children had higher insulin, HOMA2-B% and HOMA-IR than non-obese children. Conversely, HOMA2-S% was significantly lower in obese children. HOMA2 (-S% and –B%) and HOMA-IR were correlated to the very same variables as insulin, but the relationships were less strong. This may be because insulin is a primary constituent of the algorithms that formulate HOMA-IR and HOMA2. PBF SDS was the singularly most influential factor (by logistic regression) related to insulin (See Table 9). However this changed in Phase Two where BMI SDS was the most influential factor on insulin. The most obvious answer for this change in predictor for insulin is a change in body mass from Phase One and Phase Two. Table 31 shows that there is no difference between the two groups, but perhaps the magnitude of change did not have to be statistically significant. Phase Two logistic regression was performed with addition of

height and weight change scores and fitness parameters and this may have made a difference to the outcome.

Adjustment for intervention, age, and sex could not explain the differences in TG, insulin and HOMA2-S%. Both triglycerides and insulin were highly correlated with BMI and body fat in the work of Botton et al. (2007). Botton et al. suggested that subcutaneous body fat may be a modifying factor in insulin resistance. Triglycerides and absolute insulin were correlated with BMI and PBF SDS in this study but PBF SDS was computed to be the most powerful predictor (alongside with age) of insulin levels in Phase One and Phase Two. Thus a relatively (PBF SDS) higher accumulated body fat level appears to attenuate insulin sensitivity and in turn elevate insulin.

*Insulin and body mass*

The children in this study were more insulin sensitive (HOMA2-S%) when their body weight was lower. Furthermore, strength was related to elevated insulin levels and lower insulin sensitivity, but CRF was associated (inversely) with BMI SDS only. BMI SDS was a weak correlate with insulin. These factors (strength and BMI SDS) were directly correlated with stiffness index in this study. Evidence from Thraikill et al. (2005) suggests that insulin may act as an anabolic catalyst to bone modeling. Both Leger et al. (2006) and Saha et al. (2008) found that type 1 diabetic children, children that lack insulin production, were at risk of lower BMC than non-diabetics. The mechanism of action appears to be at the receptor level (insulin receptor 1 to 4) of osteoblasts, which are highly insulin receptive (Thomas, Hards, Rogers, Ng, & Best, 1996). Additionally there is a significant positive correlate between strength and insulin. This relationship is most probably the product of the anabolic effect of the hormone, insulin, on muscle hypertrophy and therefore improving muscle strength. With this evidence, it is no small stretch of the mind to visualise that the insulin–BMI SDS link also goes further to affect stiffness index and strength. Ultimately, odds ratio of BMI SDS was the only significant variable to explain insulin and the insulin-derived metabolic measures of the homeostatic models assessment (HOMA-IR, insulin sensitivity and beta cell function).

*Physical fitness*

This phenomenon of an underwhelming relationship of fitness with metabolic factors provides a different perspective to that impugned by Benson et al. (2006), where both muscle strength and CRF were found to be significantly related to insulin sensitivity in New Zealand children. However, the children were older (between 10 and 15 years)

than the ones in this study and it is this factor that may explain the difference amongst young pre-pubertal and peri-pubertal children and older post-pubertal adolescents' metabolic response to fitness. Younger children (9 to 10 years of age, n = 873) were investigated by Ruiz et al. (2006) and high cardiorespiratory fitness quartiles was found to be significantly and negatively associated with quartiles of insulin resistance (HOMA-IR). Regression analysis had not been carried out in this instance and thus the strength of the relationship is not known. However given that the CRF data had been divided into quartiles and significance could only be found in the lowest CRF quartile (and the second lowest quartile for girls) there may be other factors, such as body mass, that override this significance as has been shown in this study.

*Bone*

While comparisons of bone health data revealed no significant differences, stiffness index was seen to have low to moderate correlation with BMI SDS and muscle strength. It is well-documented that muscle tension stimulates bone remodeling (Schoenau & Frost, 2002) and this may very well be the case observed in this study as demonstrated by logistic regression analysis. Research, published by Fuchs et al. (2001), found that a jumping programme for 5 to 9 year olds, elicited better bone mineral content (BMC) than controls (Femoral neck:4.5%; Lumbar spine: 3.1%). Furthermore, female gymnasts 8 to 17 years of age had significantly higher BMD (13-28%) and BMC (24-51%) than active controls (Nurmi-Lawton et al., 2004). It is unknown whether the relationship of strength to stiffness index is purely a matter of size. A large person is more likely to be stronger than a smaller one. Perhaps an indication of whether strength is a valuable contributor to stiffness index may be observed in the logistic regression results. BMI SDS was the only variable to predict stiffness index. Thus the great value of impact stress from a larger mass is perhaps significant for bone integrity in young children.

### 5.9.4 The difference between young children and adolescents

Perhaps the time between Phase One and two dampened the effect of both fitness parameters, but there was a clear difference, in relationship to BMI SDS, between CRF and muscle strength. This difference implies a factor of muscle morphologic maturity, where, in young pre-pubertal children, their muscles may not respond in the same physiologic pattern as peri-pubertal or post-pubertal adolescents. Certainly Stephens et al. (2006) found significant metabolic differences (carbohydrate and lipid oxidation rates) in children of differing maturity that underwent fitness testing. Thus the metabolic

response of muscle in pre-pubertal children to fitness should be seriously investigated in order to develop efficacious preventive interventions.

In this study, the sole and significant factor affecting metabolic health in young children is body mass. Smaller children were more insulin sensitive and had low beta cell function compared to their larger peers regardless of sex, age and body composition. Puberty reduces insulin sensitivity and it may be possible that a number of the obese children in this study experience early onset of puberty. However, the oldest child was 9.4 years and it is extremely unlikely that the rate of maturation would have affected this young population. Low beta cell function and high insulin sensitivity at a young age may prevent development of cardiovascular disease and type 2 diabetes in adulthood (Deshmukh-Taskar, et al., 2006). In contrast, stiffness index in the same children increases with every kilogram of body mass, which has potential to delay the development of osteopenia later in life. This positive and negative factoring of body mass may, on the surface, present a "catch-22" scenario for paediatric health workers, however there are mitigating circumstances that can serve weight loss and bone development (Nemet, et al., 2006) and these can be overcome by implementing BMI-SDS specific guidelines.

### 5.9.5 Further research

Paediatric studies of metabolic factors (insulin resistance – HOMA-IR, insulin sensitivity – HOMA2-S% and beta cell function - HOMA2-B%) and fitness suggest that fitness improves (Allen et al., 2007 ; Bell et al., 2007; Benson et al., 2006; AL Carrel et al., 2005; Lee et al., 2006; Nassis et al., 2005 ), but fat mass degrades, metabolic health. However the methodological utilisation of HOMA may be questioned (Wallace, et al., 2004b) and its application in this study, of pre-pubertal children, shows that HOMA is a lesser cousin to the measure of absolute insulin. Additionally the bulk of paediatric studies are conducted with older children who may be affected by the hormonal influence of peri- or post-puberty. Thus future research should be focused upon:

1. The division of maturity in children especially in metabolic studies
2. Expanding fitness studies to investigate muscle morphology and physiologic muscular response to activity in prepubertal children
3. Developing appropriate measures of metabolic variables (for example HOMA) in children

It is incumbent upon health researchers to encourage studies to investigate this issue further as young children are currently being exposed to health interventions that

purport to prevent issues such as insulin resistance and by proxy, type 2 diabetes, without relevant supporting evidence or information.

### 5.9.6 Limitations and strengths

Limitations of this study were the extended period of time between blood sampling and fitness testing (approx 15 weeks). This was unavoidable as the ethics approval was required for the additional fitness testing. As unavoidable as this period was, it may have had a deterimental effect on the relationship between glucose homeostasis measures and fitness. However the children had little time out (two weeks) for school holidays during the 15-week delay, thus the children were exposed to the physical activity intervention for approximately 13 weeks  A 9-month fitness intervention increased mean average $VO_2$max in 17 American middle school children (age ±SD=12.0, 0.5 years) by approximately 1.0 mL.kg$^{-1}$.min$^{-1}$ (*p*=N.S.) from baseline (31.2 ±5.2 mL.kg$^{-1}$.min$^{-1}$) (Carrel, et al., 2007). On return from a three-month (summer break) period, they recorded a mean average decrease in $VO_2$max of 9.5% (-3.2 mL.kg$^{-1}$.min$^{-1}$, *p*=0.007). In contrast, fitness (predicted $VO_2$max from 20m SRT) in 178 Greek children (%) age ±SD 8.0 ±1.4 years did not change significantly over the holidy period (May to September = four months). High physical activity children increased (nonsignificant) $VO_2$max by 0.7 mL.kg$^{-1}$.min and low physical activity children increased (nonsignificant) $VO_2$max by 0.3 mL.kg$^{-1}$.min$^{1}$ and 0.2 mL.kg$^{-1}$.min$^{1}$ for Males and females respectively. Further, test-retest reliability co-efficients are 0.89 for children (n=139 male and female) aged six to sixteen (Léger, et al., 1988). Thus noise of the reliability co-efficient may conceal any true change change in fitness.  Based on the information available, it does not seem likely that there would be a large change in fitness levels over a period of 15 weeks (approximately 3.5 months). Programme children were encouraged into physical activity but these physical activity events were not directed to fitness.

By including in the analysis the change in anthropometric factors of height and weight in the analysis, statistical adjustments for time were made. The 20m SRT was limited as a maximal test as it cannot be stated with absolute confidence that all children exerted maximal effort. However, maximal performance is extremely difficult to achieve in young children (Armstrong, 1994). To combat this issue, the researcher (GK) developed a “pacer” protocol to motivate the young participants to maximal volition. This innovative and unique procedure involved one of the researchers taking part in the 20m SRT with the children. The strategy allowed for control over the children’s behaviour

(keeping “pace” with the audio CD) and motivating them to perform to their maximal ability. The 20m SRT results demonstrated that the children in this study were mostly fitter than those overseas, thus the difference could be due to two reasons: 1) the “pacer” protocol may have been successful or 2) the children in this study were fitter. The cross-sectional approach taken in this study eliminates the ability to infer causality; however relationships may be adequately defined.

A potential limitation of this study was the frequency of milk consumption. The milk was placed in front of the children in the programme group to actively encourage them to drink. However, children could refuse to consume the school milk at any time. No record of school milk consumption was taken, thus it is not known how often this scenario took place.

This study is the first study in New Zealand to investigate relationships between CRF and glucose homeostasis in primary school-age children. This is an extremely important detail as there are very few studies that have included children of this age group. Because of the scarcity of data in young children, adolescent studies appear to be “representing” children. Another first for New Zealand is the use of the non-invasive, quantitative ultrasound in a pediatric health study. This new innovation and will help contribute to new knowledge of childrens’ bone strength and dynamics as they grow, much about which very little is known.

The design strengths of this study include the employment of an internationally recognised and validated cardiorespiratory fitness assessment (the 20m SRT) that was weight-bearing (Anderson, 1992). Utilising a weight-bearing assessment is important to elicit the effect of fitness on bone strength because it includes the gravitational effect on body mass. Also, weight-bearing activity is a common activity for children (moreso than cycling) and thus the 20m SRT is more “specific” as an assessment. The sex hormone influence of puberty on growth, biochemical concentrations and fitness were avoided by recruiting a young participant group (Ministry of Health, 2003). Glucose homeostasis measures in New Zealand children are rare and the glucose and insulin data provided by this study allows a unique perspective into the metabolic health of young New Zealand children. The blood sampling and analyses were carried out by an accredited laboratory and trained staff so the quality control was excellent.

### 5.9.7 Summary

In summary, contrary to adolescent and adult studies, CRF in young children does not appear to be related to insulin, insulin sensitivity or insulin resistance. However MST

was positively related to insulin and inversely related to insulin sensitivity. This may be as a result of the large body mass in bigger children promoting higher production of insulin which encourages the development of more muscle, and in turn, creates a stronger child. Thus the insulin-grip strength relationship is probably one of indirect association. BMI SDS was related to many factors, especially those of cardiovascular risk, glucose homeostasis dysfunction and bone integrity. Fitness parameters did not appear to modify the metabolic profile in young children. Thus the continued use of BMI SDS is important to ascertain proportion of risk and aiding the prescription of modified behaviours to prevent future illness. Research in this area of metabolic health is limited and given the far-reaching implications of growth and development in youth on their health status at adulthood, it is imperative to widen the scope of determinants included in analysis and to confirm the results presented in this chapter.

# 6 Discussion and Conclusion

The purpose of this thesis was to investigate the drivers of body mass and fat mass in primary school children. Determinants included lifestyle factors in the form of responses to diet and physical activity questionnaires, and measures of physical function in the form of fitness tests, body composition, resting metabolic rate, substrate utilisation and glucose homeostasis. Specifically, the intent was to explore explanations for accumulation of body mass. The most accepted dogma to explain change in mass is the energy balance equation. A positive energy balance imposes an increase in mass and a negative energy balance creates a decrease in mass. Whilst the energy balance theory gains approval via its logic and simplicity (Bellisle, McDevitt, & Prentice, 1997; Hill, 2006; Spiegelman & Flier, 2001), a few prospective studies (Molnar, 1992; Sun, Ukkola, Rankinen, Joanisse, & Bouchard, 2002; Toubro, et al., 1998) show that change in body mass is more complex than the energy balance equation.

This thesis sought to explore in children possible physiological causes for relationships between increase in body mass and risk for future chronic disease. Some new information has been provided, by this body of work, for the following questions.

1. What are the determinants of BMI or PBF SDS of primary school age children in a naturalistic setting? (chapter 3)
2. Does RMR and resting RER represent a possible physiologic pathway linking lifestyle and demographic determinants to body mass and fat mass? (Chapter 4)
3. Is body mass or fat mass influenced by physical fitness and insulin sensitivity?
4. Can physical fitness affect RMR and resting RER and insulin sensitivity?
5. What determinant(s) affect bone integrity in a naturalistic setting?

## 6.1 Summary of the studies

To answer the questions posed above, the three studies reported were related by the dependent variables BMI SDS and PBF SDS and by being included in the intervention, "*Project Energize*". To enable a well-rounded investigation of the determinants and physiological pathways of body and fat mass, a multiple-perspective approach was employed to explain relationships between selected determinants and BMI and PBF

SDS (Figure 15). Both BMI SDS and PBF SDS were a common point of reference throughout this thesis. The SDS unit from the CDC database (BMI) and (PBF) was utilised to adjust for age and sex differences in mass and to provide a common reference base for other studies. The four perspectives, all of which related to body mass and fat mass are presented in Figure 15.

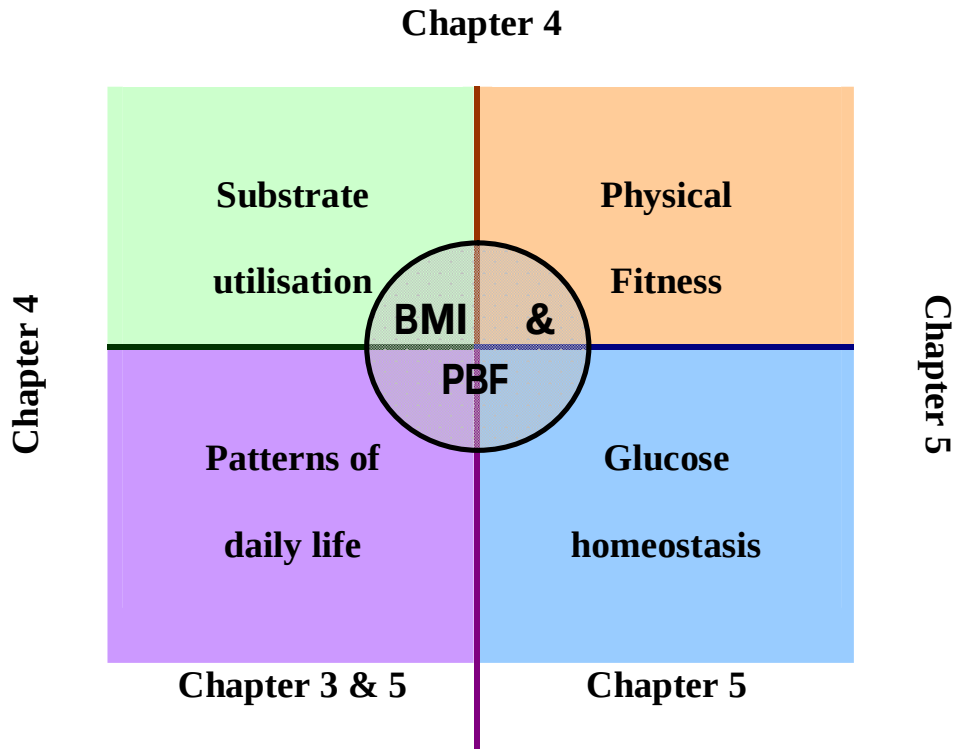


**Figure 15. Use of a multiple perspective approach to inform determinants relationship with dependent variables, BMI and PBF.**

All participants attended programme or control primary schools of the *"Project Energize*". To set the scene, the 618 five and ten year olds, whose parents returned questionnaires in 2004 and 2006, as part of the large-scale longitudinal evaluation of the nutrition and physical intervention Project Energize were examined (Chapter 3). This chapter reports the relationships that parent-reported patterns of daily life, i.e. food and activity had with the dependent variables, BMI SDS and PBF SDS. The next chapter (Chapter 4) considered resting metabolic rate and substrate utilisation as an explanatory pathway for relationships between patterns of daily life and BMI and PBF. It was in this chapter that physical fitness (CRF and MSF) was introduced as a possible determinant of mass. Finally, chapter 5 reports an investigation of glucose homeostasis as an alternate explanatory pathway for relationships between patterns of daily life and BMI SDS and PBF SDS. Physical fitness measures were also included in this study.

Three studies, each exploring associations from different viewpoints, were commissioned to answer several existing questions (see previous section) regarding the determinants of mass in young children and their physiologic pathways. BMI and PBF are common themes that link the studies and substrate utilisation and glucose homeostasis represent links between determinants and mass.

Schematics to visually describe the relationships between determinants, explanatory pathways and mass, BMI (Figure 16) and PBF (Figure 17) are presented below to assist in the explanation of interrelationships observed in all three studies reported in this thesis.

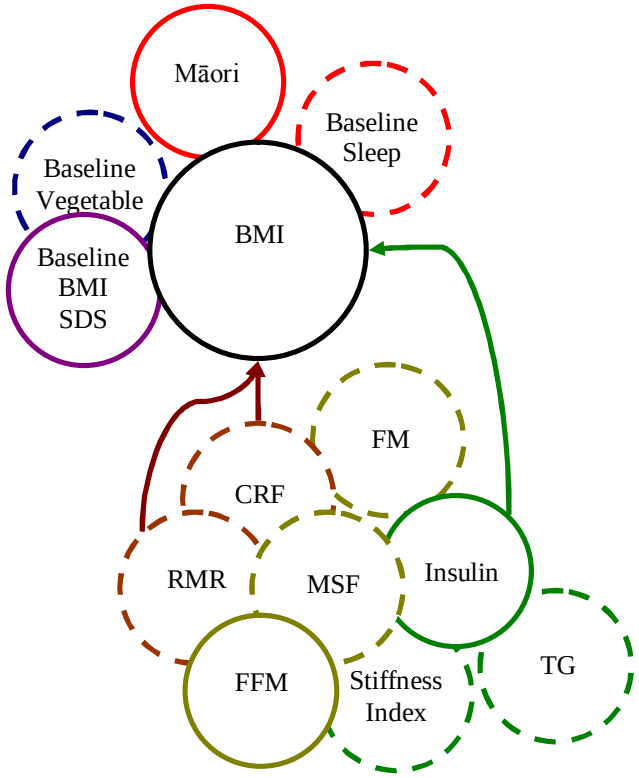


**Figure 16. Balloon schematic of determinants and physiologic interactions of fat mass as represented by BMI SDS.**
Relationships are represented by overlapping circles, dashed lines are bivariate correlations and solid lines are regression relationships. Black = BMI; Red = chapter three child5; Blue: chapter three child10; Purple = chapter three children; Brown = chapter four; Olive = chapter four and five; Green = chapter five.

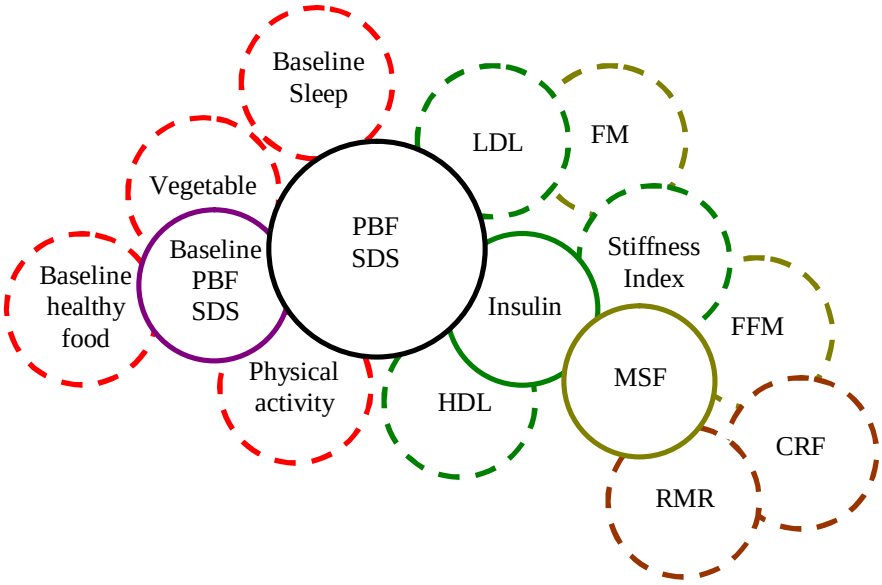


**Figure 17. Balloon schematic of determinants and physiologic interactions of fat mass as represented by PBF SDS.**
Relationships are represented by overlapping circles, dashed lines are bivariate correlations and solid lines are regression relationships. Black = PBF SDS; Red = chapter three determinants 5 year olds; Purple = chapter three determinants 5 and 10 year olds; Brown = chapter four metabolic variables; Olive = chapter four and five; Green = chapter five insulin resistance variables.

## 6.2 Significance of the findings

The primary finding of this thesis is that the most important determinant of BMI SDS and PBF SDS of young pre-adolescent children was their BMI SDS and PBF SDS two years previously. That is, once a child is overweight or obese, there are strong odds that they will remain on this growth trajectory unless there are purposeful, dramatic, and consistent changes to their environment. In addition, ethnicity was an independent predictor of change over a two year period in BMI SDS and PBF SDS of children aged five. Māori children increased in mass faster than European children. But the same ethnic difference was not seen in the ten year old cohort. There was a confounding relationship between ethnicity and decile and this was because of an over-representation of Māori in low decile communities. This notion is further explored in 6.4. Some modifiable factors were identified in the studies that could be the focus and target of future interventions.

Very few relationships of daily lifestyle patterns with BMI were seen. Small inverse relationships were found between final BMI SDS and Child5 total weekly sleep and also, child10 vegetable frequency. Interestingly, no activity or any other frequency of consumption of food groups were related to BMI SDS. The low number of correlates suggests that it is too simplistic to state, for instance, high body mass for age, sex and

height is due to the frequent consumption of high fat foods; the energy balance theory is not a satisfactory explanation for mass accumulation in isolation and a genetic predisposition may have an influence. In contrast, there were four times as many child5 correlates with PBF SDS, but none in child10. Again, small inverse relationships were seen between child5 PBF SDS with the explanatory variables, physical activity, sleep and vegetable frequency. Physical inactivity was positively aligned with PBF SDS in a weak correlation. It seems these factors affect FM more readily than BM.

FM was a significant negative predictor of 20m SRT performance (CRF), but taller children (for their age and sex) performed better. This finding supports the conclusion of Olds et al. (2007) who partially attribute fatness to the decline in fitness of children (aged 10 to15). Unsurprisingly, FFM and to a lesser extent body mass (BM) contributed significantly to the MSF result. The positive relationship between muscle size and MSF is well-known. The obese children had more FFM and therefore they were much stronger, in absolute terms, than the not obese children. Age was also a significant predictor of MSF however because mass also increases with age, collinearity may be the primary driver of this particular relationship.

Both MSF and PBF SDS were significantly related to stiffness index (StIn). Evidence that muscle strength also encourages bone remodeling is supported in chapter 5. However the link between StIn and PBF SDS is an unknown. No physiological or physical factor can be produced to state that fat status encourages bone remodeling. Research demonstrates that bone mineral content is independently predicted by both FM and FFM; however FFM has the stronger relationship (Ackerman et al., 2006).

The investigation of substrate utilisation (chapter 5) could not confirm that resting RER was an explanatory pathway for the accumulation of body fat by macronutrient intake, physical activity and inactivity, sleep or physical fitness. This may be because the explanation of the large variation in resting RER (in this study 0.76 to 1.00) could be due to other factors including genetic (Jacobson et al., 2006), CRF, food intake, muscle glycogen store, and proportion of type 1 muscle fibres (Goedecke, et al., 2000).

CRF and FFM were both found to be significant determinants of RMR. It was not surprising that FFM predicted RMR as the literature has already shown that relationship in adults. However, the finding that CRF predicted RMR is new in studies of children. Although CRF did not explain much of the variance, the importance of CRF is that it is a modifiable variable. MSF was highly correlated with RMR but failed to demonstrate a

relationship in regression analysis. This is most likely due to the collinearity of the relationships that MSF has with FFM, BP and Ht.

MSF, BMI, and insulin, from the glucose homeostasis study (chapter 5), are intimately implicated in what appears to be a positive feedback loop (Figure 18). Insulin sensitivity in young children was very high in comparison to standardized adult norms for HOMA2-S%. Children are able to adapt to changes in macronutrient composition of food better than adults (Treuth, et al., 2003) – high insulin sensitivity may provide a protective mechanism against periods of high carbohydrate intake or physical inactivity by "building-in" a large safety zone of insulin sensitivity. However, after being subjected to frequent (and long) periods of high carbohydrate intake or physical inactivity, the body may become more insulin resistant at a much younger age. This scenario places the individual at risk for developing T2DM. Insulin is a known anabolic hormone and as such stimulates tissue growth. This tissue growth implies strength enhancement. However there are indications from this study that a large mass also encourages higher insulin secretion and thus positively feeding back stimuli. That is, leading to pressure the pancreas to produce higher and higher levels of insulin which stimulates mass, which produces more insulin.

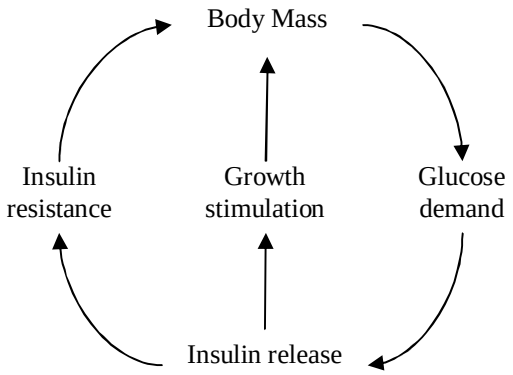


**Figure 18. A hypothesised relationship of body mass and insulin.**

In addition, BMI SDS and insulin were positively related to triglycerides, but PBF SDS was positively linked to LDL cholesterol and inversely with HDL cholesterol. Again, this intimate liaison between biomarkers and body mass status reveals the complexity of the physiologic pathways that are associated with obesity. It appears fat mass is an indicator of lipid profile health in children. CRF was not associated with insulin or any HOMA variant, however MSF was inversely associated with HOMA2-S% but positively with insulin and the other HOMA variants, in contrast to previous research. Based on the observations made in this study, physical fitness had little effect on glucose homeostasis of the young children reported here. A simplistic answer is that the

skeletal muscle mass of the children was not sufficiently mature to take on the benefits of fitness to improve glucose homeostasis. Alternatively, the hormonal conditions were not present to be able to have measurable physical fitness benefits as there has been for adults and adolescents.

### 6.3 New knowledge and translation into public health action

This body of work confirms evidence from the existing, international literature and contributes to understanding the challenges of improving child health and function in the New Zealand context – in particular, for children attending primary schools in the Waikato district which has a high Māori population. The strong tracking of body size and physical activity and food choice behaviours with and without intervention, the positive contribution of cardiorespiratory fitness measured by a 20m shuttle run test with resting energy expenditure and the association of increased plasma insulin with increased body fat underscore the importance of early intervention.

More children with increased fatness were in the lower decile schools, which also had a higher proportion of Māori. This stratification of risk justifies targeting within Project Energize, schools of lower decile and having Māori community participation in the design and delivery of interventions.

Training can improve cardiorespiratory fitness and one of the keys to cardiorespiratory fitness is proficiency in fundamental movement skills such as running (Okely et al., 2004).

Other important associations with BMI and PBF included inactivity, vegetable consumption and sleep.

The strong tracking of BMI, PBF and food and physical activity patterns identifies target areas for future planning of paediatric public health interventions. Healthy body mass and optimal physiologic function (Fowden, Giussani, & Forhead, 2006; Taylor & Poston, 2007) is primed by adequate maternal nutrition and physical activity as mandated by the World Health Organisation lifecourse approach to health (Darnton-Hill, Nishida, James, & James, 2004). Additionally, static food patterns over time like those found in the chapter three study cohort indicate that government policy could be directed to improving the quality of, and access to, the food supply e.g. nutritional fortification, cheaper variety of whole foods. Physical activity of all age groups was also static and this indicates that sports play a minor role in physical activity. Perhaps sports

participation needs to be promoted or spontaneous physical activity needs to be encouraged more often.

Based on this developmental premise, options for preventive programmes could entertain a number of different methods:

1. transform the physical environment – to promote healthful food and physical activity.
2. establish interventions that are focused on women of child-bearing age
3. provide health promotion and social marketing campaigns targeted at young families

Ethnicity classification may be an impediment to interpretation of results especially concerning Māori and Pacific peoples. Methodological problems regarding selection and interpretation of ethnicity may confound interpretation of outcomes (Section 6.4). It is necessary for explicit guidelines for the use of ethnicity in research before conclusions can be drawn from results such as those observed in chapter 3. Similarly, self-reported food intake needs to reflect an individual's food more accurately. Accuracy is doubly difficult because parents usually report their perception of food choices of their children, and biased 3$^{rd}$ person reporting may occur whether purposeful (i.e. saying what the researchers want to hear) or accidental (i.e. not knowing what foods their child has consumed).

CRF can be used to improve RMR and which may, by proxy, manage BM in young children. Resting ER did not show any indication that it was a useful tool to predict weight gain. However, insulin sensitivity was not moved by either CRF or MSF, which may indicate that primary school children could have physically immature muscle to benefit from such treatment. Adjustments for height should be made when making comparisons between children. did not contribute tcannot be Given that fat mass impairs the slows a child down

## 6.4 Methodological limitations and strengths

The findings and their implications outlined above add valuable knowledge to the current database of information about children and effects of their environment on their body size. However, recognising the methodological issues confronted by this body of work will help to further understand the context and perspectives in which the information was collected.

All studies were in a naturalistic setting which provides external validity to the outcomes and relationships. Experimental laboratory studies e.g. when food and activity are controlled provide have some advantages because control of confounders in naturalistic settings is extremely difficult, thus there will issues of precision in measurement in some cases. Study one was longitudinal while studies two and three were cross sectional which limits the conclusions for these studies to associations. The instrument used for demographic, food, activity, and sleep were parental-recall questionnaires which while having the advantage of being relatively cheap and easy to administer to a number of parents have the limitation of respondents being the more health conscious, better educated parents who may not recall, know or report information about their child accurately. The anthropometric measures have better validity and quality control as do the measures of fitness, biochemistry, substrate utilization and energy expenditure. That children reported fasting to these last measures was a strength of the conduct of the study.

Conducting respiratory exchange in a non-clinical environment posed a challenge but a challenge with unexpected benefits. Initially the use of a room at the school was a matter of convenience, that is, the child was going to be there later that morning and all children with an appointment could report to a single location that was known to them. Being at the school provided a sense of safety and comfort for the children, much like a second home and this aided their relaxation, even though they had a respiratory mask over their face and a pulse oximeter fitted to their hand. Additionally, if a child forgot their appointment, they could be accessed in their classroom and reminded. A drawback of using the schools were that the rooms were not always well-ventilated and temperature controlled, thus the constant monitoring of climate variables and numerous calibrations were required.

Sampling fasting blood for measures of glucose and lipid homeostasis in very young children was a challenge, which included cultural and empathic concerns for safe handling of the blood. This information is therefore a “taonga”, a treasure of knowledge with added value. Ethnicity and cultural considerations are essential in research in naturalistic settings but also pose problems. As an example, sufficient time needs to be set aside to allow the consultation process with minority ethnicities to provide enough information for all parties (participants, their community leaders and the researchers) to be satisfied that their needs are going to be met.

There were differences observed between Māori and European children, however these differences were made non-significant by associations of BMI SDS with socioeconomic status. This information casts doubt over the use of ethnicity in New Zealand health research, especially of New Zealand European and Māori children who are attending the same schools. The range of genetic variation within the named ethnicities, coupled with the escalating admixture of ethnicities in New Zealand, informs that ethnicity-related biological traits of body mass are unlikely. Ethnic cultural behaviour that might encourage an increasing body mass status may also be subject to other influences such as whanau (extended family) culture or socioeconomic (financial or education status) culture, thus the pursuit of ethnic comparisons in body mass status is convoluted and possibly erroneous. In New Zealand, Maori and Pacific peoples are disproportionately represented in the low socioeconomic status group. In five year olds, Maori children were significantly increased the risk of obesity, but this was not seen in the analysis of ten year olds. There is a risk, in these circumstances, of concluding that there is a genetic basis for the ethnic difference. The selection of ethnicity is a complicated issue in itself. Restriction to one ethnic group convolutes the results when it is obvious that admixture is apparent, especially in a country such as New Zealand, where everyone originates from immigrants. Choice of ethnicity, as a determinant of obesity and health risk may be overridden by measures of social and family cultures and behaviours (Deckelbaum & Williams, 2001).

## 6.5 Future Research Directions

There are a number of future research opportunities that make themselves apparent in this thesis. The high prediction value of historical body mass status (BMI or PBF SDS) suggests that contemporary body mass status may be determined much earlier than childhood and adolescence. Thus longitudinal studies from preconception to young adulthood would be the most valid research method to elicit the primary determinants of body mass status. In addition to variables of demographics and lifestyle, that is ethnicity, decile, food and activity patterns, there are genetic and epigenetic factors that may well represent much of the historical body mass and provide a more complete picture of the development of body mass.

Secondly physical fitness has shown that it has a place in the development of body mass. Strength did not appear to be a protective feature against body mass status nor cardiovascular risk factors as suggested in previous research. Strength was positively related to both BMI and PBF SDS by weak coefficients. In contrast, CRF was inversely

related to BMI and PBF SDS and insulin, which belies a possible protective feature in the development of body mass. Thus future research should investigate CRF's physiologic relationship with anthropometry and employ insulin as a point of focus. Many more studies of insulin, insulin sensitivity and beta cell function need to be conducted to provide a complete picture of this dynamic in young children. HOMA2-S% (insulin sensitivity) was much higher in children that in adults, however absolute insulin concentrations were more highly associated with body mass and strength than HOMA scores. It was interesting that insulin's relationship with lipids was different dependent on whether total or fat mass was considered. Insulin drives growth, body mass increases and therefore a higher production of insulin by beta cells of the pancreas is required. Overstimulation of this positive feedback pathway in childhood may have a cumulative effect on cardiovascular risk factors such as lipid levels. While no association of fasting insulin with cardiorespiratory fitness was seen future work is needed to define the dynamics of these relationships.

Although there was little evidence in this thesis to confirm respiratory exchange as an indicator of a primary physiologic pathway to body mass change, the observation that resting metabolic rate might be attenuated in overweight and obese children. For child weight management programmes improving fundamental movement skills and fitness, increases in resting metabolic rate and cardiorespiratory fitness may be useful outcome measures.

A multitude of physiologic mechanisms have been ascribed to explain why reduced habitual sleep duration is associated with weight gain. However, the small difference in sleep duration has not been studied in a laboratory situation nor has a causal pathway between sleep duration and body mass status been demonstrated. Future work should follow up on the hypothesised pathways of how sleep contributes to body mass development.

Objective measures of physical activity should be pre-requisite in studies of children. To be reliant on memory recall of a child or their parent only works to the detriment of the study as the information is subjective and does not support rational scientific method. Any one of several methods such as observation, pedometers, global positioning systems (GPS), laser positioning and accelerometers can provide more precise estimates of physical activity patterns than recall.

Similarly, food patterns drawn from participant recall suffer from the very same issues as physical activity data drawn from participant recall (see paragraph above). However

objective measures of food patterns are unable to be performed in a naturalistic setting. Additionally, measurement of food quotient and energy content is just as difficult. To develop and validate a system that could precisely measure the energy content and FQ of a food in a naturalistic setting would be extremely helpful in settling issues of lifestyle patterns and its relationship with body mass.

Time spent in physical inactivity demonstrated inverse associations with BMI and PBF SDS. Although physical inactivity in this study was drawn from recall data, the result may indicate interplay amongst sedentary behaviour, inadequate duration and poor quality of sleep and convenience food intake that combine to affect body mass status. This issue must be investigated further as it has consequences for the instilling of unhealthy behaviours, subsequent weight gain and development of cardiovascular disease.

There are a number of suggested research areas that have been identified and detailed in the previous paragraphs. Some, like the development of a food pattern system that is precise, does not rely on participant recall and can be performed in a naturalistic environment, may be an unreasonable request. However, the knowledge to be attained by investigating relationships among physical inactivity, insulin, sleep and respiratory exchange in children is necessary to develop recommendations that encourage healthy body mass status and prevent obesity related disorders in adulthood.

Future work to better understand ethnic, socioeconomic, family and cultural influences of lifestyle patterns in relation to weight gain needs to occur. Mixed method and complementary investigations are required to identify barriers and motivators for family involvement to support school-based interventions.

## 6.6 Conclusion

Body mass status, physical inactivity, sleep and food patterns in children aged 5 and 10 years tracked over two years. There were patterns of association of body mass status with resting metabolic rate, fitness and glucose homeostasis but not resting substrate utilisation.

This work is the first in New Zealand to utilise the internationally validated 20m SRT and measures of insulin to investigate associations with body mass and food and activity patterns in primary school age children. Six major findings with implications for nutrition and physical activity in schools were provided.

The first major insight arising from this thesis was that at least seventy percent of body mass status was explained by body mass status two years earlier. This predetermination was much higher than expected and underpins the recommendation of this thesis that prevention programmes need to start with preconception education and intervention. Secondly, physical inactivity (50%), sleep (36%) and food patterns (>10%) tracked over two years and there were small interrelationships among these behaviours. This reflects the importance of connecting environment, aetiology and physiologic effect. Thirdly, more data has been collected on insulin and insulin sensitivity in young children where very little has been reported in the literature. Fourthly, strength was strongly associated with insulin and body mass status but cardiorespiratory fitness was less as fat mass (but not fat free mass) increased. Fifthly, the innovative use of resting respiratory exchange data in children to identify predictors of body mass status suggested that resting metabolic rate (energy balance), but not substrate utilisation, was a likely mechanism for weight management in young children. Lastly, the innovative use of portable, non-invasive assessment of bone integrity, calcaneal quantitative ultrasound, in children was a first in New Zealand. The lack of precision in the measurement meant that any effect of milk supplementation on healthy bone formation in children was not able to be detected but associations of bone strength with body mass were evident. Two schematic diagrams (see Figure 16 & Figure 17) were developed based on the results of the studies and are focused on those explanatory variables that are related BMI SDS and PBF SDS.

Overall, this thesis has added new knowledge concerning the progression of body mass status, demographic, lifestyle, fitness and metabolic aspects in young New Zealand children. The perspectives of the evidence presented built on previous literature and

have been synthesized into a paradigm which may further explain interrelationships of determinants of the evolution of body mass.

A focus on weight gain as the outcome of interventions is unlikely to show short or medium term changes. Interventions that target up-stream determinants such as fat and sugar and whole foods in the food supply and more immediate metabolic and functional outcomes such as glucose homeostasis and cardiorespiratory fitness may reduce the future burden of disease and improve quality of life for children. Therefore it is recommended that when instigating school-based nutrition and physical activity programmes, there must also be a food and activity environment focus alongside community, family and culture-based partnerships to support sustainable behavioural change. The holistic and integrated approach of this thesis could help inform both design and delivery of public health programmes to improve health and function across the lifecourse for future generations of New Zealand children.

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## Appendix A. Questionnaire categories

**Table 33. Definition of questionnaire categories**

| Category | Related Questions | Description |
|---|---|---|
| Physical Activity frequency | HHQ1Q45-46<br>HHQ2Q83,85 | Frequency of involvement physical activities such as play i.e. biking, swimming, gymnastics, exercising, basketball, softball/baseball, netball, rugby, volleyball, football/soccer, hockey, racquet sports, ball games, active play, play outdoors, martial arts, dance, chores outdoors/indoors, skating/skateboarding, cricket and other. These were summed and a total derived for estimated frequency of current activity per day. |
| Physical Activity promoters | HHQ1Q38<br>HHQ2Q76 | Environment or items that may promote physical activity e.g. play equipment. These were summed to derive a total number of items in the immediate environment that may encourage physical activity. |
| Sedentary duration | HHQ1Q40-41<br>HHQ2Q78-79 | Frequency watching TV including DVD's; playing electronic games; using a computer. This estimate amounted to average time spent during weekdays and weekends participating in these sedentary activities. Both weekday and weekend times were added for a hours per week unit. |
| Sedentary promoters | HHQ1Q37<br>HHQ2Q75 | Environment or items that may promote sedentarianism e.g. multi-media devices in the bedroom |
| Health food consumption | HHQ1Q37<br>HHQ2Q75 | Frequency of consumption of foods considered to confer a health benefit i.e. Vegemite, nuts and water. The frequencies of consumption of these foods were totalled and represented as a total frequency per day |
| Fruit | HHQ1Q37<br>HHQ2Q75 | HHQ1 was a single question including all fruits. HHQ2 was a selection of a variety of fruits, therefore the results were different. |
| Vegetables | HHQ1Q37<br>HHQ2Q75 | HHQ1 was a single question including all vegetables. HHQ2 was a selection of a variety of vegetables, therefore the results were different. |

| | | |
|---|---|---|
| Takeaway or fast food | HHQ1Q31<br>HHQ2Q71 | Consumption frequency of food not prepared at home such as fish and chips, KFC, McDonalds, Indian, Asian and other restaurants. |
| Carbohydrate consumption | HHQ1Q24a-24f,24h-24i,24r,27a-27n,30e-30g,30i,30m,31a-31f<br>HHQ2Q63a-63f,63h-63i,64e,67a-67n,70e-70g,70i,70m.71a-71f | Frequency of consumption of high energy foods with a high proportion of carbohydrate i.e. Fruit juice, fruit drink, powdered drink, cordial, soft drinks, sports drink, flavoured milk, muffin, bread white/wholemeal, pasta, weetbix, cornflakes, un/toasted muesli, special K, bran, just right, nutrigrain, puffed wheat, porridge, muesli bar, chocolate, confectionary, and jam. |
| Fat consumption | HHQ1Q28-30f, 30h, 30j, 30l-m<br>HHQ2Q68-70f, 70h, 70j, 70l-m | Frequency of consumption of high energy foods with a high proportion of fat i.e. hamburger, meat pie, meat - fried with/without oil/boiled/stir fried, Mayonnaise, Salad dressing, meat gravy, white/cheese sauce, coconut milk, biscuits, cakes, hot chips, pizza, butter, peanut butter, and crisps. |
| Milk consumption | HHQ1Q24n-w<br>HHQ2Q64 | Frequency of Milk consumption – all types, with the exception of flavoured milk (see carbohydrate consumption) |
| Sleep | HHQ1Q47-50<br>HHQ2Q86-89 | Estimated average hourly duration of sleep during weekdays derived from bedtime and waking time during weekdays and weekends. Weekday estimate is multiplied by 5 (weekdays) and weekend estimate is multiplied by 2 (weekend days) and then added to provide a weekly estimate of sleeping hours. |

HHQ1 = HouseHold Questionnaire at baseline
HHQ2 = HouseHold Questionnaire at 2 years post-baseline
NB Equivalent questions in the same questionnaires did not have the same question numbers

## Appendix B. Project Energize Household Questionnaire 2004

# Project Energize Household Questionnaire

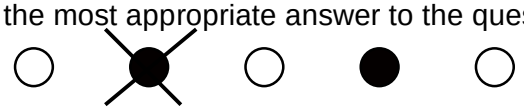
Dear Parent/caregiver

This is the household questionnaire that is part of Project Energize (Project Energize is the new name for Activ8 - we had to change it). This questionnaire should be completed by the child's usual caregiver (most often the mother). The questionnaire will take about 30 minutes to complete. You might like to involve your child when you fill it in. If you have difficulty with any part of the questionnaire, or have any questions, freephone 0800 228 488 for assistance.

The questionnaire responses are either in the form of circles to fill in, or squares to enter letters or numbers.

To answer a question where there are a number of circles to choose from, fill in completely the circle which is the most appropriate answer to the question.

If you need to chance your answer, put a cross through the first answer, and fill in the circle which is the most appropriate answer to the question.



To answer a question where there are boxes to write in, put one letter or number in each box. It's best to write in capital letters.

| 2 | 3 | A | | A | C | A | C | I | A | | S | T | R | E | E | T | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|

If you need to change your answer, put a line through the first answer, and write your corrections above or below the altered boxes.

1 9 D BOWEN AVENUE

| ~~2~~ | ~~3~~ | ~~A~~ | | ~~A~~ | ~~C~~ | ~~A~~ | ~~C~~ | ~~I~~ | ~~A~~ | | S | T | R | E | E | T | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|

If you have more than one child involved in the study, you only need to complete

Section 1: About you
Section 2: About your home
Section 3: About your family

once only.

We do need you to complete Section 4: About your child for <u>each child</u> who is in the study.

When you have completed the questionnaire, place it in the attached stamped addressed envelope, and mail it.

Thanks for your assistance

Dr Dave Graham
Paediatrician
For Project Energize

# Project Energize

## Section 1: About you

**PLEASE ANSWER ALL THE FOLLOWING QUESTIONS**

**Please complete in BLOCK CAPITALS using a BLACK Pen shading circles like this ●**

### Primary Care Giver Details

**1. What is your name? First Name**

**Last Name**

**2. What is your gender?** ○ **Male** ○ **Female**

**3. What is your date of birth?** ☐☐ / ☐☐ / ☐☐☐☐ **Date of birth**

**e.g.** 13 / 07 / 1976 **Date of birth**

**4. With which ethnic group(s)do you BEST identify with?**

New Zealand European, Pakeha ○

New Zealand Maori ○

Pacific ○ please specify _____

Other European ○

Asian ○ please specify _____

Other ○ please specify _____

**5. Were you born in New Zealand?**

Yes ○ No ○

**6. How many years have you lived in New Zealand?** ☐☐

**7. What is your highest level of academic qualification?**

None ○

NZ School Certificate in one or more subjects ○

NZ Sixth Form Certificate in more or more subjects ○

NZ University entrance or equivilent ○

NZ A or B Bursary scholarship ○

NZQA or trade certificate or diploma ○

Bachelors degree ○

Postgraduate degree, diploma, certificare ○

Other - ○ please specify _____

**8. Which of the following categories BEST describes your CURRENT situation?**

- A worker for pay ◯
- A student, on no benefits ◯
- A student receiving a living costs Student Loan ◯
- A student receiving a Student Allowance/Community Wage Benefit ◯
- A student NOT receiving a Student Allowance/ Community Wage Benefit ◯
- Unemployed receiving a Work and Income NZ Benefit ◯
- Unemployed NOT receiving a Work and Income NZ Benefit ◯
- A home-maker receiving a Work and Income NZ Benefit ◯
- A home-maker NOT receing a Work and Income NZ Benefit ◯

**9. How many hours per week do you usually work for pay?**

- I don't work for pay ◯
- Less than 8 hours ◯
- 8 to 15 hours ◯
- 16 to 24 hours ◯
- 25 to 32 hours ◯
- 33 to 40 hours ◯
- 41 to 48 hours ◯
- 49 to 56 hours ◯
- More than 56 hours ◯

**10. And, how many hours is that usually each day?**

- I don't work for pay ◯
- Less than 3 hours ◯
- 3 to 5 hours ◯
- 6 to 8 hours ◯
- 9 to 10 hours ◯
- More than 10 hours ◯

**11. What is your usual work pattern for paid work?**

- I don't work for pay ◯
- Daytime - no shifts ◯
- Rotating shifts without nights ◯
- Rotating shifts with nights ◯
- Permanent nights ◯
- Irregular or variable ◯

**12. How do you usually travel to each of the following?**

| | Walking | Bicycle | Car | Bus |
|---|---|---|---|---|
| Nearest Diary | ○ | ○ | ○ | ○ |
| Nearest Take away outlet | ○ | ○ | ○ | ○ |
| Nearest Grocery store | ○ | ○ | ○ | ○ |

**13. And for each of these, by the way you usually travel how long would the trip take going one way?**

| | 1-5 Minutes | 6 - 10 Minutes | 11-15 Minutes | 16 - 20 Minutes | 21 -30 Minutes | More than 30 Minutes |
|---|---|---|---|---|---|---|
| Nearest dairy | ○ | ○ | ○ | ○ | ○ | ○ |
| Nearest Take away outlet | ○ | ○ | ○ | ○ | ○ | ○ |
| Nearest Grocery store | ○ | ○ | ○ | ○ | ○ | ○ |

**14. We want to know what you think about various sorts of food and drink**

| | Can have as much as possible | Can have frequently | Can eat regularly | OK to have occasionally | Can have very little of this |
|---|---|---|---|---|---|
| Milk | ○ | ○ | ○ | ○ | ○ |
| Fruit Juice | ○ | ○ | ○ | ○ | ○ |
| Fruit | ○ | ○ | ○ | ○ | ○ |
| Bread | ○ | ○ | ○ | ○ | ○ |
| Vegetables | ○ | ○ | ○ | ○ | ○ |
| Eggs | ○ | ○ | ○ | ○ | ○ |
| Lollies | ○ | ○ | ○ | ○ | ○ |
| Meat | ○ | ○ | ○ | ○ | ○ |
| Fish | ○ | ○ | ○ | ○ | ○ |
| Potato/Rice/Kumara/Taro/ Pasta | ○ | ○ | ○ | ○ | ○ |
| Muesli Bars | ○ | ○ | ○ | ○ | ○ |
| Soft drinks (fizzy) and Cordial | ○ | ○ | ○ | ○ | ○ |
| Chicken | ○ | ○ | ○ | ○ | ○ |
| Roll-ups and Dried Fruit | ○ | ○ | ○ | ○ | ○ |
| Chocolate | ○ | ○ | ○ | ○ | ○ |

## Section 2: About your Home

**1. Which BEST describes your family's current home?**

Stand alone home ◯ Block of flats ◯

**2. Which BEST describes your family's home location**

We live in the country/ rural district ◯

We live in a town ◯

We live in a city i.e. Hamilton ◯

**3. Does your home have an outdoor play area, and is it fenced or enclosed?**

We have an outdoor play area, and it is fenced or enclosed ◯

We have an outdoor play area, but it is not fenced or enclosed ◯

We do not have an outdoor play area ◯

**4. Do you have a vegetable garden?**

Yes ◯ No ◯

**5. For food storage and preparation, does your household have (in working order) a:**

| | Yes | No |
|---|---|---|
| Fridge | ◯ | ◯ |
| Stove (cooking rings) | ◯ | ◯ |
| Microwave | ◯ | ◯ |
| Oven | ◯ | ◯ |
| Freezer | ◯ | ◯ |

**6. How many of the following are in your home?**

| | None | 1 | 2 | More than 2 |
|---|---|---|---|---|
| Television | ◯ | ◯ | ◯ | ◯ |
| Video/DVD player | ◯ | ◯ | ◯ | ◯ |
| Computer | ◯ | ◯ | ◯ | ◯ |
| Internet access | ◯ | ◯ | ◯ | ◯ |
| Telephone (land line) | ◯ | ◯ | ◯ | ◯ |
| Cellphone | ◯ | ◯ | ◯ | ◯ |

**7. Is the water in your home fluoridated?**

Yes ◯ No ◯ Don't know ◯

**8. How many times has your family shifted in the last 5 years?**

0 ◯ 1 ◯ 2 ◯ 3 ◯ 4 ◯ 5 ◯ More than 5 ◯

**9. Does your neighbourhood have public recreation facilities (such as public swimming pools, parks, walking trials, bike paths?)**

| | Yes | No |
|---|---|---|
| Public parks or gardens | ◯ | ◯ |
| Public School Playground | ◯ | ◯ |
| Public/community Swimming pool | ◯ | ◯ |
| Bike Paths | ◯ | ◯ |
| Walking trails | ◯ | ◯ |
| Recreation Centre or Community Hall | ◯ | ◯ |

## Section 3: About your Family

*For the purpose of this questionnaire family refers to the people living in your household*

**1. How many people of what age are currently living in your household?** *Include yourself, and the child in ths study* - Write the number that applies , for each age group.

| | | | |
|---|---|---|---|
| Under 1 year | ☐☐ | 15 to 19 years | ☐☐ |
| 1 to 4 years | ☐☐ | 19 to 24 years | ☐☐ |
| 5 to 9 years | ☐☐ | 25 to 39 years | ☐☐ |
| 10 to 14 years | ☐☐ | 40 to 59 years | ☐☐ |
| | | 60 years and older | ☐☐ |

---

**2. What is your taxable household income (2003-2004)?**

*While this information is really useful to us, and will be stored completely securely, we understand it is sensitive information, so this is an optional question.*

| | | | |
|---|---|---|---|
| Less than $5,000 | ○ | $50,000 - $59,999 | ○ |
| $5,000 - $9,999 | ○ | $60,000 - $69,999 | ○ |
| $10,000 - $19,999 | ○ | $70,000 - $79,999 | ○ |
| $20,000 - $29,999 | ○ | $80,000 - $89,999 | ○ |
| $30,000 - $39,999 | ○ | $90,000 + | ○ |
| $40,000 - $49,999 | ○ | | |

---

**3. *Is your family as a whole* trying at present to make any changes to their choice of ther following foods?**

| | Trying to increase the amount eaten | Not trying to change | Trying to decrease the amount eaten |
|---|---|---|---|
| Fruit | ○ | ○ | ○ |
| Vegetables | ○ | ○ | ○ |
| Bread -white bread | ○ | ○ | ○ |
| Bread -whole grain | ○ | ○ | ○ |
| Cereals - plain (e.g. weetbix, porridge) | ○ | ○ | ○ |
| Cereals processed - (e.g. honeypuffs, cocopops) | ○ | ○ | ○ |
| Butter, Margarine, Oils | ○ | ○ | ○ |
| High fat foods | ○ | ○ | ○ |
| Hamburgers, pizza, fried chicken | ○ | ○ | ○ |
| High sugar foods | ○ | ○ | ○ |

**The following questions relate to the buying of food and drink and whether your family has sufficient resources to obtain the food required. the questions relate to the family over the past year**

**4. The household can afford to eat healthily**

Always ◯ Often ◯ Sometimes ◯ Never ◯

**5. Food runs out in the household due to lack of money (this refers to basic food items such as bread and potatoes)**

Always ◯ Often ◯ Sometimes ◯ Never ◯

**6. We eat less because of a lack of money**

Always ◯ Often ◯ Sometimes ◯ Never ◯

**7. The variety of food the household is able to eat is limited by a lack of money**

Always ◯ Often ◯ Sometimes ◯ Never ◯

**8. Social occasions for the household revolve around visiting takeaway establishments (e.g. Pizza Hut, KFC)**

Always ◯ Often ◯ Sometimes ◯ Never ◯

**9. The household cannot provide the food required for social occasions**

Always ◯ Often ◯ Sometimes ◯ Never ◯

**10. Family members eat watching TV**

Always ◯ Often ◯ Sometimes ◯ Never ◯

**11. The household is too busy to provide meals you want to provide**

Always ◯ Often ◯ Sometimes ◯ Never ◯

**12. The family eats together**

Always ◯ Often ◯ Sometimes ◯ Never ◯

## Section 4: About your Child

**This section refers to your child who is in the study. Remember, if more than one of your children is in the study, we need you to complete this section for *each* child**

**1. What is your child's name?** **First Name**

**Last Name**

**2. What is your child's gender?** **Male** ○ **Female** ○

**3. What is your child's date of birth?** ☐☐ / ☐☐ / ☐☐☐☐ **Date of birth**

**e.g.** 13 / 07 / 1976 **Date of birth**

**4. With which ethnic group(s)does your child BEST identify with?**

New Zealand European, Pakeha ○

New Zealand Maori ○

Pacific ○ please specify _____

Other European ○

Asian ○ please specify _____

Other ○ please specify _____

**5. Was your child born in New Zealand?**

Yes ○ No ○

**6. How many years has your child lived in New Zealand?**

| | | |
|---|---|---|
| <1 year ○ | 4 years ○ | 8 years ○ |
| 1 Year ○ | 5 years ○ | 9 years ○ |
| 2 Years ○ | 6 years ○ | 10 years ○ |
| 3 years ○ | 7 years ○ | |

**7. How would you rate your childs health and fitness?**

| | Very Poor | Poor | Average | Good | Very Good |
|---|---|---|---|---|---|
| Health | ○ | ○ | ○ | ○ | ○ |
| Fitness | ○ | ○ | ○ | ○ | ○ |
| Eating Habits | ○ | ○ | ○ | ○ | ○ |
| Body Shape | ○ | ○ | ○ | ○ | ○ |

**8. Does your child have a health problem or condition lasting six months or more, that causes difficulty for your child doing everyday activities that children his/her age USUALLY do?**

| | No health problem of this type | Has problem but doesn't limit activity | Mild Limitation Sometimes can't join in usual activities | Moderate limitation often can't join in usual activites | Severe limitation can't participate in most things |
|---|---|---|---|---|---|
| Lung or breathing | ○ | ○ | ○ | ○ | ○ |
| Heart | ○ | ○ | ○ | ○ | ○ |
| Bone or Joint | ○ | ○ | ○ | ○ | ○ |
| Brain, spine or nerve | ○ | ○ | ○ | ○ | ○ |
| Muscle | ○ | ○ | ○ | ○ | ○ |
| Overweight | ○ | ○ | ○ | ○ | ○ |
| Diabetes | ○ | ○ | ○ | ○ | ○ |
| Hyperactivity | ○ | ○ | ○ | ○ | ○ |
| Bowel ("poos") | ○ | ○ | ○ | ○ | ○ |
| Night-time bladder (wetting) | ○ | ○ | ○ | ○ | ○ |
| Day-time bladder (wetting) | ○ | ○ | ○ | ○ | ○ |

Other - please specify _____

**9. Has your child ever had a broken bone or fracture?**

No ○ Once ○ Twice ○ More than twice ○

**10. How many attacks of wheezing has your child had in the *past 12 months?***

None ○ 1 to 3 ○ 4 to 12 ○ More than 12 ○

**11. IN THE PAST 12 MONTHS, how often, on average has your child's sleep been disturbed due to wheezing?**

Never ○ Less than 1 night/week ○ One or more nights/week ○

**12. IN THE PAST 12 MONTHS, has wheezing ever been severe enough to limit your child's speech to only one or two words at a time between breaths?**

Yes ○ No ○

**13.** **IN THE PAST 12 MONTHS, has your child's chest sounded wheezy during or after exercise?**

Yes ◯ No ◯

**14.** **IN THE PAST 12 MONTHS, has your child had a dry cough at night, apart from a cough associated with a cold or a chest infection?**

Yes ◯ No ◯

**15.** **Has your child EVER had a wheezing or whistling in the chest at any time in the past?**

Yes ◯ No ◯

**16.** **Has your child ever had asthma?**

Yes ◯ No ◯

## Your child's general eating habits

**17.** **How would you describe your child's eating schedule?**

3 meals and snacks daily ◯

3 meals daily ◯

2 meals daily ◯

Less than 2 meals per day ◯

**18.** **How would you describe your child's eating pattern?**

Eats a variety of foods at each meal ◯

Eats a variety of foods, but can be fussy at some meals ◯

Is fussy about food at most meals ◯

Is fussy at every meal ◯

**19.** **Does your child eat breakfast?**

Every day ◯

5-6 days a week ◯

3-4 days a week ◯

1 - 2 days a week ◯

Never ◯

**20. Where does your child eat breakfast most often?**

- My child doesn't usually eat breakfast ○
- Provided at home ○
- Provided at school ○
- Purchased from a shop on the way to school ○

**21. At School, how does your child usually have lunch?**

| | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | Every school day |
|---|---|---|---|---|---|
| My child doesn't eat lunch | ○ | ○ | ○ | ○ | ○ |
| My child eats lunch at home | ○ | ○ | ○ | ○ | ○ |
| Packed lunch from home | ○ | ○ | ○ | ○ | ○ |
| Purchsed from a school lunch programme | ○ | ○ | ○ | ○ | ○ |
| Purchased from a shop on the way to school | ○ | ○ | ○ | ○ | ○ |
| Purchased from a school canteen | ○ | ○ | ○ | ○ | ○ |
| Lunch is provided by school | ○ | ○ | ○ | ○ | ○ |

***We're interested to know how much your child eats that hasn't had to be bought -which might be from a vegetable garden, family chickens, caught or hunted, or traded at the markets***

**22. On average how often does your child eat foods such as fish, meat, fruit and/or vegetables that were harvested, traded, caught or hunted?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Meat - chicken/beef/lamb etc | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Fish and shellfish | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Fruit | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Vegetables | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Eggs | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Milk | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

**23. To what age was your child breastfed?**
Fill in the circle that BEST applies

- 0-1 month ○
- 1+ -3 months ○
- 3+ - 6 months ○
- 6+ months ○
- N/A (the child was not breast fed) ○

**24. How many times would your child usually eat these foods?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Fruit Juice e.g. Just Juice, Fresh-up, Robinson's or Rio Gold | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Vegetable juice e.g. Tomato Juice | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Fruit Drink e.g. Golden Circle, Choice etc | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Powdered drinks e.g. Raro, Vita-Fresh | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Low-calorie cordial | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Cordial | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Diet carbonated drink e.g. Diet Sprite, Diet Coke | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Non-diet carbonated drink e.g. Sprite, Coke | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Gatorade, Powerade | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Water, including unflavoured mineral water, soda water, tap water | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Coffee | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Coffee- decaffeinated | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Tea | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Milk, standard, homogenized milk (Blue top) | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Trim Milk (light blue top) | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Super Trim or skim milk (light green top) | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Calci trim (yellow top) | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Flavoured milk (eg Primo) | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Powdered low fat milk | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Powdered whole milk | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Whole milk (silver top) | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| 'Slim and Fit' | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Soy milk | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| **25.** **Fruit** (fresh, frozen,canned, stewed) Serving =1 medium piece, 2 small pieces, 1/2 cup | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| **26.** **Vegetables** (fresh, frozen, canned) Serving =1 medium potato/kumara, 1/2 cup cooked, 1 cup fresh | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

| **27. Breads and Cereals** | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Muffins or scones | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| White bread/buns/pita bread | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Wholemeal/wholegrain/buns/pita bread | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Pasta, rice, noodle, couscous | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Weetbix | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Cornflakes/Ricebubbles | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Toasted muesli | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Untoasted muesli | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Special K | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| All Bran, San-Bran, Bran Flakes, Weetbix, Hi Bran | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Just right, Light n Tasty or Good Morning | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Nutrigrain, Cocoa pops or Honey Puffs | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Puffed Wheat, Mini-Wheats | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Porridge | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

## 28. Meat, Fish, Poultry and Eggs

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Hamburger | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Meat pie, sausage roll, or other savoury pastries | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Meat, fish or poultry, fried or roasted in added fat or oil | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Meat, fish or poultry, fried or roasted without added fat or oil | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Meat, fish or poultry stir-fried | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

## 29. Dressings and Sauces

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Mayonnaise | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Salad dressing | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Gravy | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| White sauce/cheese sauce | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Coconut cream | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

## 30. Miscellaneous

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Sweet biscuits | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Cakes or slices | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Hot chips | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Pizza | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

## 30. Miscellaneous - continued

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Muesli bar e.g. Fruit splits, roll-ups, Uncle Tobys | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Chocolate (including chocolate bars e.g. Moro bars) | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Other confectionery | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Butter or margarine | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Jam, honey, marmalade | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Peanut butter, other nut spreads | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Vegemite or marmite | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Nuts | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Potato chips, corn chips, Twisties etc | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

## 31. Takeaway Foods

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| KFC, Southern Fried Chicken or similar | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| McDonalds, Burger King or similar | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Fish n Chips | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Valentines or other 'All you can eat' establishments | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Takeaway curry | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Takeaway chinese food | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Restaurant | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

## About what your child does

**32. How often does your child eat with the television or video on?**

Always ◯ Often ◯ Sometimes ◯ Never ◯ Don't know ◯

**33. Does your child have their own toothbrush?**

Yes ◯ No ◯

**34. How often does your child USUALLY brush their teeth?**

Less than once a day ◯

Once a day ◯

Twice a day ◯

More than twice a day ◯

**35. How does your child USUALLY travel to each of the following?**

| | Walking | Bicycle | Car | Bus |
|---|---|---|---|---|
| **Childs school** | ◯ | ◯ | ◯ | ◯ |
| **Nearest Play Area** | ◯ | ◯ | ◯ | ◯ |
| **Nearest Friend** | ◯ | ◯ | ◯ | ◯ |
| **Nearest Relative** | ◯ | ◯ | ◯ | ◯ |
| **Nearest sports field or club** | ◯ | ◯ | ◯ | ◯ |

**36. And for each of these, by the way your child usually travels, how long would the trip take going one way?**

| | 1-5 Minutes | 6 - 10 Minutes | 11-15 Minutes | 16 - 20 Minutes | 21 -30 Minutes | More than 30 Minutes |
|---|---|---|---|---|---|---|
| **Childs school** | ◯ | ◯ | ◯ | ◯ | ◯ | ◯ |
| **Nearest Play Area** | ◯ | ◯ | ◯ | ◯ | ◯ | ◯ |
| **Nearest Friend** | ◯ | ◯ | ◯ | ◯ | ◯ | ◯ |
| **Nearest Relative** | ◯ | ◯ | ◯ | ◯ | ◯ | ◯ |
| **Nearest sports field or club** | ◯ | ◯ | ◯ | ◯ | ◯ | ◯ |

**37. Does your child have the following *in their bedroom?***

| | Yes | No |
|---|---|---|
| Television | ○ | ○ |
| Video/DVD player | ○ | ○ |
| Computer | ○ | ○ |
| Internet access | ○ | ○ |
| Telephone (land line) | ○ | ○ |
| Portable hand held electronic games (e.g. Nintendo) | ○ | ○ |

**38. Does your child have any of the following to play with at home?**

| | Yes | No |
|---|---|---|
| Bicycle or scooter | ○ | ○ |
| Skateboard, rollerblades | ○ | ○ |
| Winter sport equipment e.g. rugby, netball, scoccer etc | ○ | ○ |
| Summer sport equipment e.g. cricket, tennis, softball etc | ○ | ○ |
| Swing or slide | ○ | ○ |
| Trampoline | ○ | ○ |
| Pool or paddling pool | ○ | ○ |
| Tree hut | ○ | ○ |
| Tent | ○ | ○ |

**39. Does your child have their own cellphone?**

Yes ○ No ○

**40. During a normal day, how long does your child watch television, videos and/or DVD's?**

| | 0 -30 minutes | 30-60 minutes | 1 - 1.5 hours | 1.5 - 2 hours | 2 - 2.5 hours | 2 .5- 3 hours | 3 - 3.5 hours | 3.5- 4 hours | 4 - 4.5 hours | 4.5 -5 hours | over 5 hours |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Monday-Friday | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Saturday -Sunday | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

**41. During a normal day, how long does your child spend on computer, playstation, X-box, hand held games etc?**

| | 0 -30 minutes | 30-60 minutes | 1 - 1.5 hours | 1.5 - 2 hours | 2 - 2.5 hours | 2 .5- 3 hours | 3 - 3.5 hours | 3.5- 4 hours | 4 - 4.5 hours | 4.5 -5 hours | over 5 hours |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Monday-Friday | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Saturday -Sunday | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

**42. On average, how long per day would your child spend with an adult, with adult and child just playing together?**
Fill in the circle that best applies

- Less than 5 minutes ◯
- 5 to 10 minutes ◯
- 11 to 15 minutes ◯
- 16 to 20 minutes ◯
- 21 to 25 minutes ◯
- 26 to 30 minutes ◯
- More than 30 minutes ◯

**43. Regarding my child's level of physical activity:**

- During leisure time , my child is more active than other children of their age ◯
- During leisure time, my child is just as active as other children of their age ◯
- During leisure time, my child is less active than other children of their age ◯
- During leisure time, my child is much less active then other children of their age ◯
- Don't know ◯

**44. During leisure time does your child exercise to breathlessness?**

- Very often ◯
- Often ◯
- Sometimes ◯
- Seldom ◯
- Never ◯

**45. During leisure time does your child play organised or team sports?**

- less than once per month ◯
- 1-3 times per month ◯
- Once per week ◯
- 2-4 times per week ◯
- 5-6 times per week ◯
- Once per day ◯
- 2 or more times per day ◯

**46. What activities does your child do?**

*Please tick all the relevant activities the child has participated in for at least 20 minutes at a time, in the past month*

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Bicycling | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Swimming | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Gymnastics: bars, beam, tumbling, trampoline | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Exercise: push-ups, sit-ups, jumping rope | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Basketball | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Baseball/softball | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Netball | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Rugby | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Vollyball | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Soccer | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Hockey | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Racket Sport: badminton, tennis | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Ball playing: four square, dodge, ball, kickball | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Active games: chase, running, tag, hopscotch | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Outdoor play: climbing trees, hide and seek | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Judo, karate, boxing etc | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Dance | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Outdoor chores: mowing, raking, gardening, farm work | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Indoor work: vaccuming, sweeping | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Skateboaring, scootering | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |

47. **On the night before a school day, when does your child go to bed?**

☐☐ : ☐☐ **(pm)**

48. **On a school day, when does your child wake up?**

☐☐ : ☐☐ **(am)**

49. **On the night before a week-end day or holiday when does your child go to bed?**

☐☐ : ☐☐ **(pm)**

50. **On a week-end day or holiday when does your child wake up?**

☐☐ : ☐☐ **(am)**

**Thank you for taking the time to complete this questionnaire. Could you just take a moment to check all sections are complete, then place it in the stamped addressed envelope and mail it.**

## Appendix C. Project Energize Household Questionnaire 2006

Child ID [ ][ ][1][2]

## Section 1A: Food and Drink

This section asks you about different types of food and drink, and whether you and your family are trying to make any changes to your diet.

**8. How often do you think the following foods SHOULD be eaten?**

| | As much as possible | Frequently | Regularly | Occasionally | Very little |
|---|---|---|---|---|---|
| Milk | ☐ | ☐ | ☑ | ☐ | ☐ |
| Fruit Juice | ☐ | ☐ | ☐ | ☑ | ☐ |
| Fruit | ☐ | ☑ | ☐ | ☐ | ☐ |
| Bread | ☐ | ☑ | ☐ | ☐ | ☐ |
| Vegetables | ☑ | ☐ | ☑ | ☐ | ☐ |
| Eggs | ☐ | ☐ | ☐ | ☐ | ☐ |
| Lollies | ☐ | ☐ | ☐ | ☐ | ☑ |
| Meat | ☐ | ☐ | ☑ | ☐ | ☐ |
| Fish | ☐ | ☐ | ☑ | ☐ | ☐ |
| Potato/Rice/Kumara/Taro/ Pasta | ☐ | ☑ | ☐ | ☐ | ☐ |
| Muesli Bars | ☐ | ☐ | ☐ | ☐ | ☑ |
| Soft drinks (fizzy) and Cordial | ☐ | ☐ | ☐ | ☐ | ☑ |
| Chicken | ☐ | ☐ | ☑ | ☐ | ☐ |
| Roll-ups and Dried Fruit | ☐ | ☐ | ☐ | ☐ | ☑ |
| Chocolate | ☐ | ☐ | ☐ | ☐ | ☑ |

**9. Are you and your family trying to make any changes to your choice of the following foods?**

| | Increasing amount eaten | Not changing | Decreasing amount eaten |
|---|---|---|---|
| Fruit | ☑ | ☐ | ☐ |
| Vegetables | ☑ | ☐ | ☐ |
| Bread - white | ☐ | ☑ | ☐ |
| Bread - wholegrain | ☐ | ☑ | ☐ |
| Cereals - plain (eg weetbix, porridge) | ☐ | ☑ | ☐ |
| Cereals - processed (eg honey puffs, cocopops) | ☐ | ☐ | ☑ |
| Butter, margarine, oils | ☐ | ☑ | ☐ |
| High fat foods | ☐ | ☐ | ☑ |
| Hamburgers, pizza, fried chicken | ☐ | ☐ | ☑ |
| High sugar foods | ☐ | ☐ | ☑ |

Child ID | | | 1 | 2 |

## Section 1B: Your Home

This section asks you about your household and home environment.

**10. Which BEST describes your family's current home?**

☑ Stand alone house ☐ Block of flats

**11. Which BEST describes your family's home location?**

☑ We live in the country/rural district

☐ We live in a town (e.g. Te Awamutu)

☐ We live in a city (e.g. Hamilton)

**12. How many times has your family moved house in the last 5 years?**

☑ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ More than 5

**13. Does your home have an outdoor play area, and is it fenced or enclosed?**

☑ We have an outdoor play area, and it is fenced or enclosed

☐ We have an outdoor play area, but it is not fenced or enclosed

☐ We do not have an outdoor play area

**14. How do you usually travel to each of the following?**

| | Walk | Cycle | Car | Bus |
|---|---|---|---|---|
| Nearest dairy | ☐ | ☐ | ☑ | ☐ |
| Nearest take away food outlet | ☐ | ☐ | ☑ | ☐ |
| Nearest grocery store | ☐ | ☐ | ☑ | ☐ |

**15. Using your usual method of travel, how long would the trip take going one way?**

| | 1-5 mins | 6-10 mins | 11-15 mins | 16-20 mins | 21-30 mins | 30+ mins |
|---|---|---|---|---|---|---|
| Nearest dairy | ☑ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Nearest take away outlet | ☑ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Nearest grocery store | ☐ | ☑ | ☐ | ☐ | ☐ | ☐ |

**16. Does your neighbourhood have public recreation facilities, such as swimming pools, parks, walking tracks, bike paths?**

| | Yes | No |
|---|---|---|
| Public parks or gardens | ☐ | ☑ |
| Public playground | ☐ | ☑ |
| Public swimming pool | ☐ | ☑ |
| Bike paths | ☐ | ☑ |
| Walking trails | ☐ | ☑ |
| Recreation centre or community hall | ☐ | ☑ |

**17. How many of the following are in your home?**

| | None | 1 | 2 | More than 2 |
|---|---|---|---|---|
| Television | ☐ | ☐ | ☑ | ☐ |
| Video/DVD player | ☐ | ☑ | ☐ | ☐ |
| Computer | ☐ | ☑ | ☐ | ☐ |
| Games console (e.g. Playstation, Xbox) | ☑ | ☐ | ☐ | ☐ |
| Internet access | ☐ | ☑ | ☐ | ☐ |
| Telephone (land line) | ☐ | ☐ | ☐ | ☑ |
| Cellphone | ☐ | ☐ | ☐ | ☑ |

**18. Do you have a vegetable garden?**

☑ Yes ☐ No

**19. Do you have the following household appliances for food storage and preparation?**

| | Yes | No |
|---|---|---|
| Fridge | ☑ | ☐ |
| Stove - cooking rings | ☑ | ☐ |
| Microwave | ☑ | ☐ |
| Oven | ☑ | ☐ |
| Freezer | ☑ | ☐ |

Child ID [ ][ ][1][2]

## Section 1C: Employment and Household Income

**This section asks you about your employment, the number of hours you regularly work and a general idea of your household income.**

**20. Which of the following catagories BEST describes your CURRENT situation? You may choose more than one.**

- ☐ I work fulltime or part-time for wages
- ☐ I am a student, on no benefits
- ☐ I am a student receiving a benefit (e.g. student allowance, living costs loan)
- ☐ I am unemployed and receiving a Work and Income NZ benefit
- ☐ I am unemployed and do NOT receive a Work and Income NZ benefit
- ☐ I am a homemaker receiving a Work and Income NZ benefit
- ☑ I am a homemaker NOT receiving a Work and Income NZ benefit

**21. How many hours per week do you usually work for pay?**

- ☑ I don't work for pay - please go to question 24
- ☐ Less than 8 hours
- ☐ 8 to 15 hours
- ☐ 16 to 24 hours
- ☐ 25 to 32 hours
- ☐ 33 to 40 hours
- ☐ 41 to 48 hours
- ☐ 49 to 56 hours
- ☐ More than 56 hours

**22. And, how many hours is that usually each day?**

- ☐ Less than 3 hours
- ☐ 3 to 5 hours
- ☐ 6 to 8 hours
- ☐ 9 to 10 hours
- ☐ More than 10 hours

**23. What is your usual work pattern for paid work?**

- ☐ Daytime - no shifts
- ☐ Rotating shifts without nights
- ☐ Rotating shifts including nights
- ☐ Permanent night shift
- ☐ Irregular or variable

Child ID [ ][ ][1][2]

**24. What was your household income before tax for the last 12 months?**

- ☐ Less than $5,000
- ☐ $5,000 - $9,999
- ☐ $10,000 - $19,999
- ☐ $20,000 - $29,999
- ☐ $30,000 - $39,999
- ☐ $40,000 - $49,999
- ☐ $50,000 - $59,999
- ☐ $60,000 - $69,999
- ☐ $70,000 - $79,999
- ☐ $80,000 - $89,999
- ☑ $90,000 +

**25. Using the following age groups, identify how many people live in your household, including yourself and the child in this project.**

| | | | |
|---|---|---|---|
| Under 1 year | [ ][ ] | 19 to 24 years | [ ][ ] |
| 1 to 4 years | [ ][ ] | 25 to 39 years | [ ][ ] |
| 5 to 9 years | [ ][1] | 40 to 59 years | [ ][2] |
| 10 to 14 years | [ ][1] | 60 years and older | [ ][ ] |
| 15 to 19 years | [ ][1] | | |

## Section 2: Food Resources and Availability

This section asks about how the resources available to you and your family may affect your choices about food. The questions relate to your experiences over the last 12 months. Please place a cross in the box that best describes your situation.

**26. The household can afford to eat healthily.**

☒ Always ☐ Often ☐ Sometimes ☐ Never

**27. The household runs out of basic food items, such as potatoes or bread, due to a lack of money.**

☐ Always ☐ Often ☐ Sometimes ☒ Never

**28. We eat less because of a lack of money.**

☐ Always ☐ Often ☐ Sometimes ☒ Never

**29. The variety of food available to the household is limited by a lack of money.**

☐ Always ☐ Often ☐ Sometimes ☒ Never

**30. The variety of food eaten in our household is limited by our lack of cooking skills.**

☐ Always ☐ Often ☐ Sometimes ☒ Never

Child ID | | | 1 | 2 |

**31. We use food labels to help us decide what to buy.**

☐ Always ☐ Often ☒ Sometimes ☐ Never

**32. The household is too busy to provide the meals you would like to provide.**

☐ Always ☐ Often ☒ Sometimes ☐ Never

**33. The household cannot contribute to the food required for social occasions. This may be because of a lack of money, time or cooking ability.**

☐ Always ☐ Often ☐ Sometimes ☒ Never

**34. Social occasions for the household revolve around visiting takeaway venues (e.g. Pizza Hut, KFC, McDonalds)**

☐ Always ☐ Often ☒ Sometimes ☐ Never

**35. The members of your family eat meals together.**

☐ Always ☒ Often ☐ Sometimes ☐ Never

**36. Family members eat while watching television.**

☐ Always ☐ Often ☒ Sometimes ☐ Never

## Section 3: About your child

This section is all about your child who is involved with Project Energize. If more than one of your children is taking part in Project Energize, please fill in this section for *each* child.

Child ID | | | 1 | 2 |

**40. Which ethnic group(s) does your child BEST identify with?**
*You may tick more than one.*

- [x] New Zealand European, Pakeha
- [ ] New Zealand Maori
- [ ] Pacific Islander
- [ ] Other European
- [ ] Asian, please specify _____
- [ ] Other, please specify _____

**41. Was your child born in New Zealand?** [x] Yes [ ] No

**42. How many years has your child lived in New Zealand?**

| | | |
|---|---|---|
| [ ] less than 1 year | [ ] 4 years | [ ] 8 years |
| [ ] 1 year | [ ] 5 years | [ ] 9 years |
| [ ] 2 years | [ ] 6 years | [ ] more than 10 years |
| [ ] 3 years | [x] 7 years | |

## Section 3A: Your child's health.

This section asks about your child's health and whether or not this affects their physical activity.

**43. How would you rate your child's health and fitness?**

| | Very Poor | Poor | Average | Good | Very Good |
|---|---|---|---|---|---|
| Health | [ ] | [ ] | [ ] | [ ] | [x] |
| Fitness | [ ] | [ ] | [ ] | [ ] | [x] |
| Eating habits | [ ] | [ ] | [ ] | [ ] | [x] |
| Body shape | [ ] | [ ] | [ ] | [ ] | [x] |
| Weight | [ ] | [ ] | [ ] | [ ] | [x] |

**44. Has your child ever had a broken bone or fracture?**

☒ No ☐ Once ☐ Twice ☐ More than twice

**45. IN THE PAST 12 MONTHS, Has your child had any attacks of wheezing?**

☒ None - Go to question 48

☐ 1 to 3

☐ 4 to 12

☐ More than 12

**46. IN THE PAST 12 MONTHS, how often, on average has your child's sleep been disturbed due to wheezing?**

☐ Never ☐ Less than 1 night per week ☐ 1 or more nights per week

**47. IN THE PAST 12 MONTHS, has wheezing ever been severe enough to limit your child's speech to only one or two words at a time between breaths?**

☐ Yes ☐ No

**48. IN THE PAST 12 MONTHS, has your child's chest sounded wheezy during or after exercise?**

☐ Yes ☒ No

**49. IN THE PAST 12 MONTHS, has your child had a dry cough at night, apart from a cough associated with a cold or chest infection?**

☐ Yes ☒ No

**50. Has your child EVER had a wheezing or whistling in the chest at any time in the past?**

☒ Yes ☐ No

**51. Has your child ever had asthma?**

☐ Yes ☒ No

**52. Does your child have their own toothbrush?**

☒ Yes ☐ No

**53. How often does your child USUALLY brush their teeth?**

- ☐ Less than once a day
- ☐ Once a day
- ☒ Twice a day
- ☐ More than twice a day

**54. Does your area have a flouridated water supply?**

☐ Yes ☒ No ☐ Don't know

**55. In the past six months or more, have any of the following health problems made it difficult for your child to do everyday activities that children their age usually do?**

| | No health problem of this type | Has problem but doesn't limit activity | Mild limitation (sometimes can't join in) | Moderate limitation (often can't join in) | Severe limitation (can't participate in most things) |
|---|---|---|---|---|---|
| Lung or breathing | ☒ | ☐ | ☐ | ☐ | ☐ |
| Heart | ☒ | ☐ | ☐ | ☐ | ☐ |
| Bone or joint | ☒ | ☐ | ☐ | ☐ | ☐ |
| Brain, spine or nerve | ☒ | ☐ | ☐ | ☐ | ☐ |
| Muscle | ☒ | ☐ | ☐ | ☐ | ☐ |
| Overweight | ☒ | ☐ | ☐ | ☐ | ☐ |
| Diabetes | ☒ | ☐ | ☐ | ☐ | ☐ |
| Hyperactivity | ☒ | ☐ | ☐ | ☐ | ☐ |
| Bowel ("poos") | ☒ | ☐ | ☐ | ☐ | ☐ |
| Night-time bladder (wetting) | ☒ | ☐ | ☐ | ☐ | ☐ |
| Day-time bladder (wetting) | ☒ | ☐ | ☐ | ☐ | ☐ |
| Other | ☐ | ☐ | ☐ | ☐ | ☐ |

If other - please specify _____

**56. What was your child's birth-weight?** Please indicate whether the measurement is in kilograms or pounds by circling one or the other.

4150 ~~kgs~~ / ~~lbs~~

9lb 2½ oz

**57. To what age was your child breastfed?**

- ☐ My child was not breastfed
- ☐ Birth to 1 month
- ☐ More than 1 month but less than 3 months
- ☐ More than 3 months but less than 6 months
- ☒ More than 6 months

## Section 3B: Your child's general eating habits

This section asks about the type of food your child eats and how often they eat such food.

**58. How would you describe your child's usual eating schedule?**

- ☒ 3 meals and snacks daily
- ☐ 2 meals daily
- ☐ 3 meals daily
- ☐ Less than 2 meals per day

**59. How would you describe your child's usual eating pattern?**

- ☐ Eats a variety of foods at each meal
- ☒ Eats a variety of foods, but can be fussy at some meals
- ☐ Is fussy about food at most meals
- ☐ Is fussy at every meal

**60. Does your child eat breakfast?**

- ☒ Every day
- ☐ 1-2 days a week
- ☐ 5-6 days a week
- ☐ Never
- ☐ 3-4 days a week

**61. Where does your child usually eat breakfast?**

- ☐ My child doesn't usually eat breakfast
- ☒ At home
- ☐ At school
- ☐ Purchased from a shop on the way to school

**62. During the school week, how does your child usually have lunch?**

- ☐ My child doesn't eat lunch
- ☐ My child comes home for lunch
- ☒ Packed lunch from home
- ☐ Purchased from a school lunch programme
- ☐ Purchased from a shop on the way to school
- ☐ Purchased from a school canteen or tuck shop
- ☐ Lunch is provided free of charge by the school

**63. How many times does your child usually drink the following?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Fruit juice e.g. Just Juice, Fresh-Up | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Vegetable juice e.g. tomato juice | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Fruit drink e.g. Golden Circle, Choice | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Powdered drinks e.g. Raro, Vita-Fresh | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Low-calorie cordial | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Cordial | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Diet fizzy drink e.g. Diet Coke, Diet Sprite | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Non-diet fizzy drink e.g. Coke, Sprite | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Energy drinks e.g. V, Red Bull | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Sports drinks e.g. Gatorade, Powerade | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Water, including unflavoured mineral water, carbonated water, tap water | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ |
| Coffee | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Decaffeinated coffee | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Tea - ordinary | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Tea - herbal | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

Child ID [ ][ ][1][2]

**64. How often does your child drink the following types of milk?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Standard milk (blue top) | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Trim milk (light blue top) | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ |
| Super trim or skim milk (light green top) | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Calci Trim milk (yellow top) | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Flavoured milk e.g. Primo | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| 'Slim and Fit' | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Powdered low fat milk | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Powdered whole milk | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Soy milk | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

**65. How often does your child eat at least one serving of the following fruit when it is in season?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Apples or pears | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ |
| Banana | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ |
| Orange or mandarin | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Kiwifruit | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Nectarine, peach, plum or apricot | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Strawberries or other berries | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Canned or cooked fruit e.g. canned peaches | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Dried fruit e.g. raisins | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Other fruit | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

If other, please specify _____

Child ID | | | 1 | 2 |

**66. How often does your child eat at least one serving of the following vegetables when they are in season?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Potatoes | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Kumara | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Taro | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Carrots | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Pumpkin | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Sweet corn | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Peas | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ |
| Silverbeet or spinach | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Puha or watercress | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Green beans | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Broccoli | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Cauliflower | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Cabbage | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Mixed vegetables | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Capsicum (green, yellow or red) | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Lettuce or green salad | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Tomatoes | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Avocado | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Other | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

If other - please specify _____

Child ID | | | 1 | 2 |

**67. How often does your child eat at least one serving of the following breads and cereals?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| White bread, buns, pita bread | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Wholemeal or wholegrain bread, buns, pita bread | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ |
| Muffins or scones | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Pasta, rice, noodles, couscous | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Untoasted muesli | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Toasted muesli | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Cornflakes, ricebubbles | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Special K | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Weetbix, All Bran, San-Bran, Bran Flakes, Hi Bran | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Just Right, Light n Tasty, Good Morning | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Nutrigrain, Cocoa Pops, Honey Puffs | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Puffed Wheat, Mini-wheats | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Porridge | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

**68. How often does your child eat at least one serving of meat, fish, poultry and eggs?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Hamburger | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Meat pie, sausage roll, or other savoury pastries | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Meat, fish or poultry, fried or roasted in added fat or oil | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Meat, fish or poultry, fried or roasted without added fat or oil | ☐ | ☐ | ☐ | ■ | ☒ | ☐ | ☐ | ☐ |
| Boiled meat, fish or poultry | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Stir-fried meat, fish or poultry | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

Child ID | | | 1 | 2 |

**69. How often does your child have the following dressings and sauces on their food?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Mayonnaise | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Tomato sauce | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Salad dressing | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Gravy | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| White sauce or cheese sauce | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Coconut cream | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

**70. How often does your child eat the following types of snacks and spreads?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| Sweet biscuits | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Cakes or slices | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Hot chips | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Pizza | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Muesli bar e.g. Uncle Tobys, Fruit splits, Roll-ups | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Chocolate, including bars e.g. Moro | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Other confectionary | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Butter or margarine | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ |
| Jam, honey, marmalade | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Peanut butter, other nut spreads | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ |
| Vegemite | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Nuts | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Potato chips, corn chips, Twisties etc | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |

**71. How often does your child eat the following purchased meals?**

| | Never | less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|
| KFC, Southern Fried Chicken or similar | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| McDonalds, Burger King or similar | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Fish and chips | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Indian food | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Chinese or Asian food | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Valentines or other 'all you can eat' establishments | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Restaurant and cafes | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

## Section 4: Your child's physical activity.

This section asks questions about the activities your child does in during a normal day.

**72. How does your child USUALLY travel to each of the following?**

| | Walking | Bicycle | Car | Bus |
|---|---|---|---|---|
| Child's school | ☐ | ☐ | ☒ | ☐ |
| Nearest playground | ☐ | ☐ | ☒ | ☐ |
| Nearest friend | ☐ | ☐ | ☒ | ☐ |
| Nearest relative | ☐ | ☐ | ☒ | ☐ |
| Nearest sports field or club | ☐ | ☐ | ☒ | ☐ |

**73. When your child goes by their USUAL method of transport, how long does the trip take going one way?**

| | 1 - 5 minutes | 6 - 10 minutes | 11 - 15 minutes | 16 - 20 minutes | 21 - 30 minutes | More than 30 minutes |
|---|---|---|---|---|---|---|
| Child's school | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Nearest playground | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Nearest friend | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Nearest relative | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ |
| Nearest sports field or club | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |

**74. How often does your child eat a meal while watching television or a video/DVD?**

☐ Always ☐ Often ☒ Sometimes ☐ Never

**75. Does your child have the following <u>in their bedroom</u>?**

| | Yes | No |
|---|---|---|
| Television | ☐ | ☒ |
| Video/DVD player | ☐ | ☒ |
| Computer | ☐ | ☒ |
| Internet access | ☐ | ☒ |
| Telephone (land line) | ☐ | ☒ |
| Portable hand held electronic game e.g. PSP, Game Boy | ☐ | ☒ |
| Games console e.g. Playstation, Xbox | ☐ | ☒ |

**76. Does your child have any of the following to play with at home?**

| | Yes | No |
|---|---|---|
| Bicycle or scooter | ☒ | ☐ |
| Skateboard, rollerblades | ☐ | ☒ |
| Winter sport equipment e.g. rugby, netball, soccer etc | ☒ | ☐ |
| Summer sport equipment e.g. cricket, tennis, softball etc | ☒ | ☐ |
| Swing or slide | ☒ | ☐ |
| Trampoline | ☒ | ☐ |
| Pool or paddling pool | ☒ | ☐ |
| Tree hut, play house | ☒ | ☐ |

**77. Does your child have their own cellphone?**

☐ Yes ☒ No

Child ID | | | 1 | 2 |

**78. During a normal day, how long does your child watch television, videos and/or DVDs?**

| | 0-30 mins | 30-60 mins | 1-1.5 hours | 1.5-2 hours | 2-2.5 hours | 2.5-3 hours | 3-3.5 hours | 3.5-4 hours | 4-4.5 hours | 4.5-5 hours | over 5 hours |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Monday - Friday | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Saturday - Sunday | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

**79. During a normal day, how long does your child spend on a computer, Playstation, Xbox or hand-held games?**

| | 0-30 mins | 30-60 mins | 1-1.5 hours | 1.5-2 hours | 2-2.5 hours | 2.5-3 hours | 3-3.5 hours | 3.5-4 hours | 4-4.5 hours | 4.5-5 hours | over 5 hours |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Monday - Friday | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Saturday - Sunday | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

**80. On average, how long per day would your child spend playing with an adult?**

- ☐ Less than 5 minutes
- ☐ 5 to 10 minutes
- ☐ 11 to 15 minutes
- ☒ 16 to 20 minutes
- ☐ 21 to 25 minutes
- ☐ 26 to 30 minutes
- ☐ More than 30 minutes

**81. On average, how long per day would your child spend playing with other children, OUT OF SCHOOL HOURS?**

- ☐ Less than 5 minutes
- ☐ 5 to 10 minutes
- ☐ 11 to 15 minutes
- ☐ 16 to 20 minutes
- ☐ 21 to 25 minutes
- ☐ 26 to 30 minutues
- ☒ More than 30 minutes

**82. Which of the following BEST describes your child's level of physical activity?**

- ☐ During leisure time, my child is MORE active than other children of their age
- ☐ During leisure time, my child is JUST as active as other children of their age
- ☐ During leisure time, my child is LESS active than other children of their age
- ☐ During leisure time, my child is MUCH LESS active than other children of their age
- ☒ Don't know

Child ID | | | 1 | 2 |

**83. What activities does your child do?**
Please place a cross against all the activities your child has taken part in for at least 20 minutes at a time, in the past month.

| | Don't know | Never | Less than once per month | 1 - 3 times per month | Once per week | 2 - 4 times per week | 5 - 6 times per week | Once per day | 2 or more times per day |
|---|---|---|---|---|---|---|---|---|---|
| Cycling | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Swimming | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Gymnastics: bars, beam, tumbling, etc | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Exercise: push-ups, sit-ups etc | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Basketball | ☒ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Baseball/softball | ☒ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Netball | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Rugby | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Volleyball | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Soccer | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Hockey | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Cricket | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Racket sport e.g. tennis, badminton | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ |
| Ball games e.g. four square, dodgeball | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Active games e.g chase, tag, hopscotch | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ |
| Outdoor play e.g. hide & seek, | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ |
| Judo, karate, boxing, etc. | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Dance | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Skateboarding, scootering | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Outdoor chores e.g. mowing, raking | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Indoor chores e.g. vaccuming, sweeping | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Other | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☒ | ☐ |

If other - please specify TRAMPOLINE

Child ID | | | 1 | 2

**84. Does your child exercise to breathlessness?**

☐ Very often ☐ Often ☐ Sometimes ☒ Seldom ☐ Never

**85. Does your child play organised or team sports?**

☐ Never
☐ Less than once per month
☐ 1 to 3 times per month
☒ Once per week
☐ 2 to 4 times per week
☐ 5 to 6 times per week
☐ Once per day
☐ 2 or more times per day

**86. On the night before a school day, what time does your child usually go to bed?**

8:00 **pm/~~am~~**

**87. On a school day, what time does your child usually wake up?**

8:00 **am/~~pm~~**

**88. On the night before a weekend day or holiday, what time does your child usually go to bed?**

8:30 **pm/~~am~~**

**89. On a weekend day or holiday, what time does your child usually wake up?**

8:30 **am/~~pm~~**

**90. We would welcome any comments you would like to make about either Project Energize or this questionnaire.**

**Thank you for taking the time to fill in this questionnaire. Your answers will tell us about the health and fitness of children in the Waikato. This will help us to improve Project Energize and other programs, to help your children grow up to be healthy and active adults.**

**Please place your completed questionnaire into the prepaid addressed envelope provided and drop it into any postbox.**

## Appendix D. Chapter Four Recruitment and communication process

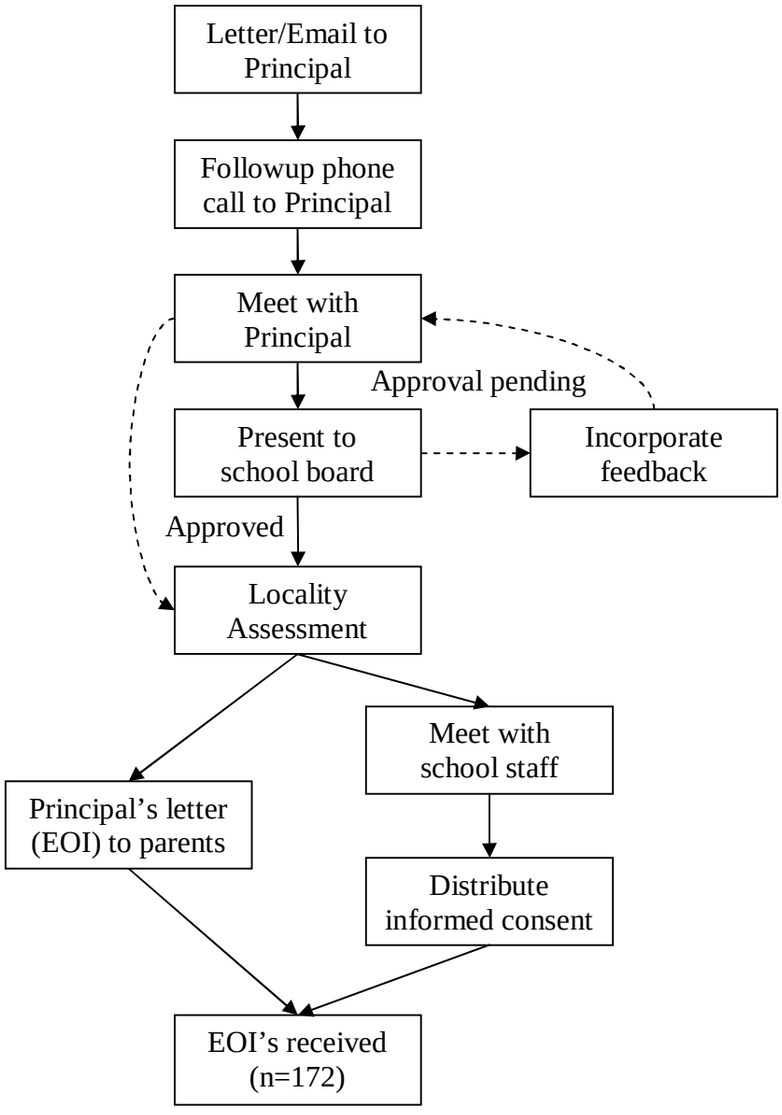



**Figure 19. Flowchart of communication and recruitment procedure with primary schools**

## Appendix E. Chapter Four Information sheet

**We will not be telling the children the measurements but we will contact you if there are any abnormal results that we think you should know about – just in case you would like us to refer your child to a doctor/clinic.**

You will also be asked to complete a questionnaire that will take about 30 minutes to fill in. The questionnaire is designed to find out what your child's usual eating and activity patterns are and a range of things that may affect these eating patterns.

**Will I get to find out the results of the study?**
Yes, if you would like, we will send you the results of the study once it is finished. Please indicate this on your consent form.

**Will the information collected be confidential?**
Yes. The information collected about you will be kept safe similar to your medical notes. We hope that we will be able to publish the results of the study in a scientific journal. If this happens the results published will be completely anonymous and participants' details will not be made public at all. No material which could personally identify you will be used in any reports on this study.

**Who should I contact if I need more information or help?**
You may call Mr Geoff Juranovich at the Project Energize helpline Phone: 0800 228 488

If you have any queries or concerns about your rights as a participant you may wish to contact a Health and Disability Services Consumer Advocate

| | |
|---|---|
| Northland to Franklin | 0800 555 050 |
| Mid to Lower North Island | 0800 423 638 |
| South Island | 0800 377 766 |

**Who has reviewed the study?**
This study has received ethical approval from the Northern Y Ethics Committee.

**Who are the researchers?**
The principal investigator is Geoff Juranovich. He is a post-graduate student from the Auckland University of Technology. He is conducting this research as part of his doctorate programme study.

His supervisor is Professor Elaine Rush also from the Auckland University of Technology. If you have any concerns about the principal investigators conduct or wish to know more about the doctorate programme at AUT you may contact Professor Rush by writing to her using the address on the front cover of this information pamphlet or phoning (09) 921 9999 x 8091.



# INFORMATION SHEET FOR PARENT/GUARDIAN

## Health Benefits from Project Energize

**Principal investigator:** Geoff Juranovich

**Address:** New Zealand Institute of Sport and Recreation Research
Auckland University of Technology
Private Bag 92006
Auckland
**Phone:** (07) 823 2510 or 0800 228 488

E mihi ana ki koutou, ngā mātua, i ngā ahuatanga o te wā, e pa ana kit e Atua Kaha Rawa; kit e Arikinui, a, Te Atairangikahu me te Kahui Ariki nui tonu.. Ka tukuna tonu ngā poroporoaki ki ngā mate – takoto mai koutou, moe mai, haere, haere, haere!
Rātou ki a rātou,; tātou ki a tātou!
Ka tahuri, kē, kit e kaupapa o tēnei tono ki a koutou, e pā ana kit e manaaki pai ā tātou tamariki/mokopuna.
No reira, āta panuihia, koa, ngā ahuatanga ka whakamāramahia i raro nei.
Ma te whaiwhakatutuki i aua ahuatanga, ka tae ate kii –
"Mauriora ki ā tātou tamariki/mokopuna!"

**Invitation**
Your child is invited to take part in a doctorate research study co-ordinated by Mr Geoff Juranovich. This study is a requirement for his postgraduate degree from the Auckland University of Technology. Before you decide whether you would like to take part it is important for you to understand why the research is being done, and what it will involve.

Please take time to read the following information carefully and discuss it with your child, mokopuna, family, whānau and friends if you wish. Please consider that there is no cost on your part for your child to be involved.

**What is Project Energize?**
Project Energize is a programme in some Waikato schools that promotes daily activity and encourages children to consume healthy food and drinks at school, and may also have an influence at home and the school's surrounding community.

This new study involves 5 additional assessments to the standard Project Energize programme and will provide information that tells us how changes in the body take place.

**Why are we doing this study?**
We would like to see firstly if the health of Waikato children/tamariki differs from each other and secondly how health-related changes take place over time in children attending a Project Energize school. We can only measure the difference Project Energize makes by comparing children who are in a Project Energize school with those who are NOT in a Project Energize school.

Your school is enrolled either as a Project Energize school, or as a control school. Older children in your school may already be enrolled in a Project Energize study, in this study we are looking for some different information to the study in which they are enrolled.

This study will help us understand not only how childrens bodies change, when they eat and drink well and are active, but also how their bodies make those changes.

**Who can take part in the study?**
Any child who is in the Project Energize programme. More than 3000 children are participating in the main Project Energize study already and we hope that up to 240 children will choose to participate in this special study.

We have asked your school that the researcher be allowed to introduce this study to the children. The students were asked to pass this information sheet to you to help you think about your child's participation in this study.

**How often will your child be measured?**
Now and in 52 weeks. It is possible that we may remain in contact, after the study has finished, to assess the future impact of Project Energize.

**What are the possible benefits of taking part?**
Your child will receive a personal profile report of all the measurements listed. In addition, the school will receive a summary of the results that does not identify the children. If Project Energize is successful, it may be funded to continue for longer in the schools where it already exists and be introduced into all the other Waikato schools.

**What are your child's rights as a study volunteer?**
Taking part in the study is purely voluntary. Your child does not have to take part. If they do agree to participate, with your consent, they will be free to withdraw from the study at any time, without having to give a reason and this will in no way affect you or your child's future health care.

**What is involved?**
The Project Energize programme will still be offered to all children in a Project Energize school, regardless of whether your child takes part in this study to find out how the programme changes your child's health.

We are asking your permission to allow us to measure your childs':

1. body weight using electronic scales (with your child wearing light clothing) and height using a stadiometer (no shoes).
2. body shape of the waist, hip and arm (using a tape measure on bare skin).
3. body size with skinfold callipers placed for 2 seconds at sites on the rear of the upper arm and below the shoulder blade.
4. blood pressure and heart rate (using an automated blood pressure machine).
5. capacity to walk or run in a standard fitness test.
6. build by using a machine called a bioelectrical impedance analysis (BIA). We would ask your child to lie down and then put a sticky pad on a hand, wrist, ankle and foot and turn the machine on for a few seconds. This quick, easy and painless test has been widely used to assess body composition in children and adults of all ages.
7. energy expenditure – i.e. how many calories your child burns by using a machine called a calorimeter. We would ask your child to lie down and then place a breathing mask over their mouth and nose. They will be able to comfortably breathe room air at all times, but will need to (fast) avoid food, fluid and exercise for 5 hours before taking part.
8. upper body strength by body weight resistance and dynanometers that measure how much they can push and pull.
9. growth using your child's Plunkett growth records

# HEALTH BENEFITS FROM PROJECT ENERGIZE
# PARENT/GUARDIAN CONSENT FORM

## REQUEST FOR INTERPRETER

| | | | |
|---|---|---|---|
| English | I wish to have an interpreter | Yes | No |
| Maori | E hiahia ana ahau ki tetahi kaiwhakamaori/kaiwhaka pakeha korero | Ae | Kao |
| Cook Island | Ka inangaro au i tetai tangata uri reo | Ae | Kare |
| Fijian | Au gadreva me dua e vakadewa vosa vei au | Io | Sega |
| Niuean | Fia manako au ke fakaaoga e taha tagata fakahokohoko kupu | E | Nakai |
| Samoan | Ou te mana'o ia i ai se fa'amatala upu | Ioe | Leai |
| Tokelaun | Ko au e fofou ki he tino ke fakaliliu te gagana Peletania ki na gagana o na motu o te Pahefika | Ioe | Leai |
| Tongan | Oku ou fiema'u ha fakatonulea | Io | Ikai |

1. I have read and I understand the information sheet dated _____ for children taking part in the study designed to investigate the changes in participants of the Project Energize programme.
2. I have had the opportunity to discuss this study with my child.
3. I am satisfied with the answers I have been given.
4. I have had the opportunity to use whanau support or a friend to help me ask questions and understand the study.
5. I understand that taking part in this study is voluntary (my choice) and that I may withdraw my child from the study at any time and this will in no way affect my future health care**.**
6. I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study.
7. I understand that the treatment, or investigation, will be stopped if it should appear harmful to my child.
8. I understand the compensation provisions for this study. (Insert for Form A and B trials)
9. I have had time to consider whether my child will take part. YES / NO
10. I wish to receive a copy of the results YES / NO

*Please be advised that a significant delay may occur between data collection and publication of the results.*

I agree to my GP or other current provider being informed of my childs participation in this study YES / NO

I _____ (full name) hereby consent to my child taking part in this study.
**PARENT/GUARDIAN**

Signature _____ Date _____
**PARENT/GUARDIAN**

Full Names of Researchers: **Mr. Geoffrey Juranovich**

Contact Phone Number for Researchers: **0800 228 488**

Project explained by: _____

Project role: _____

Signature _____ Date _____

***Please sign 2 copies –<u>Return ONE copy of this form and keep the other copy for your own record</u>***

# Appendix F. Chapter Four Section B correlates

**Table 34. Spearmans rho bivariate correlations (sig.) between food and activity data and anthropometry and RMR and resting RER**

| | Wt (kg) | BMI ($kg.m^2$) | PBF (%) | FFM (kg) | FM (kg) | RER ($VCO_2/VO_2$) | CHO Ox ($gm.min^{-1}$) | FAT Ox ($gm.min^{-1}$) | RMR (kcal.day) |
|---|---|---|---|---|---|---|---|---|---|
| Carbohydrate (times per day) | -0.03 (0.863) | -0.02 (0.918) | 0.16 (0.291) | -0.1 (0.525) | 0.08 (0.606) | -0.14 (0.363) | -0.14 (0.35) | 0.05 (0.72) | -0.12 (0.44) |
| Fat (times per day) | -0.26 (0.089) | -0.13 (0.392) | -0.01 (0.937) | -0.29 (0.05) | -0.15 (0.329) | -0.21 (0.161) | -0.29 (0.056) | 0.12 (0.432) | -0.11 (0.489) |
| Healthy food (times per day) | 0.16 (0.305) | 0.27 (0.075) | 0.32 (0.034)* | 0.09 (0.536) | 0.26 (0.082) | 0.22 (0.152) | 0.12 (0.446) | -0.05 (0.741) | 0.09 (0.546) |
| Milk (times per day) | -0.16 (0.297) | 0.1 (0.529) | 0.16 (0.307) | -0.23 (0.137) | 0.02 (0.894) | 0.09 (0.574) | -0.18 (0.224) | -0.06 (0.689) | -0.17 (0.266) |
| Takeaway Food (times per day) | 0.14 (0.353) | 0.12 (0.448) | 0.13 (0.409) | 0.15 (0.32) | 0.16 (0.305) | -0.01 (0.95) | 0.01 (0.932) | 0.10 (0.516) | 0.19 (0.221) |
| Physical Inactivity (hours per week) | 0.18 (0.255) | 0.06 (0.696) | 0.11 (0.485) | 0.20 (0.196) | 0.13 (0.416) | -0.02 (0.882) | 0.15 (0.331) | -0.10 (0.512) | 0.03 (0.867) |
| Physical Activity (times per day) | -0.10 (0.941) | 0.05 (0.785) | 0.03 (0.883) | 0.01 (0.975) | 0.02 (0.896) | -0.12 (0.485) | -0.11 (0.531) | 0.16 (0.36) | 0.01 (0.935) |
| Sleep (hours per week) | -0.36 (0.015)* | -0.20 (0.198) | -0.30 (0.048)* | -0.33 (0.028)* | -0.34 (0.021)* | -0.27 (0.077) | -0.36 (0.017)* | 0.12 (0.451) | -0.11 (0.49) |

*significant $p<0.05$
**significant $p<0.001$

## Appendix G. Chapter Five Information sheet and consent form

**Does fitness improve bone and blood health in children?**

Dear Parent or Carer,

Your child's school has been supporting Project Energize, a school physical activity and nutrition programme for the last two years. Some schools have been taking part in the Project Energize programme and others have been helping us to find out if the programme is working by being a control school. Younger children at programme schools have also been provided with milk and fruit daily.

We would like to invite your child to take part in a research study to find out if fitness can improve bone and blood health in children. This study is part of the bone health study that has been completed recently.

**Who do we want to take part?**

We would like to invite the same children that participated in the fruit and milk in schools bone health study to take part, so that we can find out more about the impact of fitness on child health in the Waikato. We really need your support with this study, with the aim of improving our children's health.

**If I take part what does my child have to do?**

Your child will take part in some physical measurements that they have already performed, that is:

1. Height and weight – using a standing height pole, tape measure slide and standard weight scales
2. Body composition by bioimpedance analysis – 4 gel stickers (electrodes) are placed on the skin (2 on the hand and 2 on the foot). A signal is passed through the electrodes to measure body fat levels. No sensation, discomfort or pain is felt during the test and all results are treated confidentially.
3. Bone scan (if equipment is available) by heel ultrasound. Much like an X-ray of the foot except that high frequency sound is used to create the image and there is no exposure to radiation.

plus an additional two fitness measurements:

1. An aerobic fitness test (the "beep test" ) – where they would be running laps of a 20m course for as long as they can. Duration: approx. 7 minutes
2. A hand grip strength test – where they would squeeze the handle of a device that measures how much pressure they can apply. Duration: approx. 5 minutes

All of these measurements will be taken as a group at your child's school by a trained and qualified professional and we would really like you to come along as well.

On the day of the measurements your child will need to come to school as usual. They will not need to fast this time as no blood is being taken. Your school may have a morning or an afternoon session and this time is noted below.

School_____ Date/Time:_____

*Version number 1, 4 September 2007*

**What will the information be used for?**

Your child is having their height, weight, body composition and bone scan (if equipment is available) re-measured to take into account any changes since the first phase of the bone health study.

The fitness measurements will be compared to the blood samples (from the first phase) and bone scan measures. We would like to know whether the fitness and strength of a child affects their heart health (e.g. cholesterol and triglycerides) and whether the fitness and strength of a child affects their bone health (bone mineral density).

All the measurement and test results will be kept by the research team, and the information will be completely confidential. Your child's results will be coded, which means that the results will not have their name with them and so they cannot be traced back to your child. When we have analysed the results, we will be reporting the study results back to the involved school communities.

Any information that has been recorded in the project will be kept for 7 years in a locked storage cabinet at AUT University as required by ethics procedures. No genetic tests will be taken.

**Do I have to take part?**

It is up to you and your child if they take part or not. If you decide to consent to your child taking part, you can withdraw your consent or your child can refuse to participate at any time during the project.

Please note that if you decide that your child should not take part, your decision will not have any adverse effect on the your access to or the provision of education or health services to you or your child. It might be useful to talk about this with your child and we would also encourage you to ring us to talk about the project in more detail.

## How can I find out more?

If you would like to know more about the project or if there is anything that is not clear, you can contact **Geoff Juranovich** on **021 403 889** during office hours, or leave a message and Geoff will call you back. You can also contact Geoff via email on **gjuranov@aut.ac.nz** or by writing to the following address:

**Geoff Juranovich**
**C/- Sport Waikato**
**PO Box 46**
**Hamilton**

If you have any concerns you'd like addressed independently, you can do this through the Health and Disability Advocacy Service, Hamilton (07) 834-3960

**NOTE: PLEASE RETURN SIGNED CONSENT FORMS TO THE SCHOOL OFFICE BY FRIDAY 12th OCTOBER IF YOU HAVE AGREED TO PARTICIPATE IN THIS STUDY. THANK YOU.**

## Appendix H. Chapter Five Questionnaire Sample

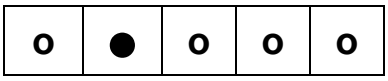
# Project Energize Bone Health Questionnaire

Dear Parent/caregiver

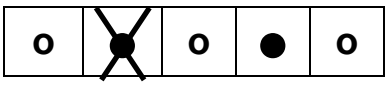
This study is looking at the bone health of children. We would like to know how food and activity affect bone development in children. It is important that you complete this form as openly as possible. This questionnaire should be completed by the child's usual caregiver (most often the mother). The questionnaire will take about 30 minutes to complete. You might like to involve your child when you fill it in. If you have difficulty with any part of the questionnaire, or have any questions, freephone 0800 228 488 for assistance.

The questionnaire responses are either in the form of circles to fill in, or squares to enter letters or numbers.

To answer a question where there are a number of circles to choose from, fill in completely the circle which is the most appropriate answer to the question.




If you need to change your answer, put a cross through the first answer, and fill in the circle which is the most appropriate answer to the question.




To answer a question where there are boxes to write in, put one letter or number in each box. It's best to write in capital letters.

| 2 | 3 | A | | A | C | A | C | I | A | | S | T | R | E | E | T | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|

If you need to change your answer, put a line through the first answer, and write your corrections above or below the altered boxes.

1 9 D B O W E N A V E N U E

| ~~2~~ | ~~3~~ | ~~A~~ | | ~~A~~ | ~~C~~ | ~~A~~ | ~~C~~ | ~~I~~ | ~~A~~ | | ~~S~~ | ~~T~~ | ~~R~~ | ~~E~~ | ~~E~~ | ~~T~~ | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|

If you have more than one child involved in the study, you only need to complete about your child for each child who is in the study.

When you have completed the questionnaire, please give it to your child or bring it into the school on the morning selected for your child's testing.

Thanks for your assistance


Dr Dave Graham

Paediatrician

For Project Energize

Geoff Juranovich

Post graduate student

For AUT University

**This questionnaire refers to your child who is in the study. Remember, if more than one of your children in the study, we need you to complete a separate questionnaire for *each* child**

## PLEASE ANSWER ALL THE FOLLOWING QUESTIONS

**Please complete in BLOCK CAPITALS using a Pen to shade circles like this ●**

### 1. What is your child's name?

**First Name**

**Last Name**

### 2. What is your child's gender?

**o Male** **o Female**

### 3. What is your child's date of birth?

| | | / | | | / | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **d** | **d** | | **m** | **m** | | **y** | **y** | **y** | **y** | **Date of birth** |

**e.g.**

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **1** | **3** | **/** | **0** | **7** | **/** | **1** | **9** | **7** | **6** | **Date of birth** |

### 4a. Which ethnic group does your child identify with?

*Mark the circle or circles which apply to your child.*

| | |
|---|---|
| New Zealand European | **o** |
| Mäori | **o** |
| Samoan | **o** |
| Cook Island Maori | **o** |
| Tongan | **o** |
| Niuean | **o** |
| Chinese | **o** |
| Indian | **o** |
| other such as *DUTCH, JAPANESE, TOKELAUAN*. Please state: | **o** _____ |

## 4b. Please select the ancestry of your <u>childs' grandparents</u> using the ethnicity descriptions provided above:

**If you are unable answer this question, please select this circle, and go to question 6.** **o**

**If you do not wish to answer this question, please select this circle, and go to question 6.** **o**

| **Mothers side** | | **Fathers side** | |
|---|---|---|---|
| **Grandfather** | **Grandmother** | **Grandfather** | **Grandmother** |
| | | | |

## 5. Was your child born in New Zealand?

**o Yes** **o No**

## 6. How many years has your child lived in New Zealand?

| | | | | | |
|---|---|---|---|---|---|
| <1 year | **o** | 3 years | **o** | 6 years | **o** |
| 1 Year | **o** | 4 years | **o** | 7 years | **o** |
| 2 Years | **o** | 5 years | **o** | 8 years | **o** |

## 7. Has your child ever had a broken bone or fracture (in the past 2 1/2 years)?

**o** No **o** Once

**o** Twice **o** More than twice

**This section asks about your child's eating patterns. It may be helpful to ask the person who does the cooking and shopping in your household to help you fill in the questions.**

## PLEASE DO NOT SKIP ANY FOODS

**Different foods may affect people's health. To help us understand these eating patterns, we would like you to think back over the *past 4 weeks* and answer the following questions about the foods your child eats.**

**Mark the circle which best tells HOW OFTEN your child usually eats the foods.**

**If they never or rarely eat a food, fill in the circle 'never or less than once a month', and go to the next question.**

**99. Milk (Not flavoured)**

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |



**Example only**

**If your child drinks milk 3 or 4 days each week, fill in the circle '3-4 times a week'.**

## 8a. Milk (Not flavoured)

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

## 8b. What **kind of milk** does your child **usually** drink?

- o Standard (dark blue top)
- o Trim (green)
- o Soy milk
- o Low fat (light blue)
- o Extra calcium
- o Other milk (please name) _____

## 9. Milk shake or Flavoured milk e.g. primo

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

## 10. How often do they drink water?

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

## 11a. Food drink e.g. Milo, Nesquik

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

**11b. With this drink do they use?**

- o All milk
- o ½ milk
- o ¼ or less milk

Is **sugar** added?

- o Yes
- o No

**12. How often do they drink *food* carbonated (fizzy) drinks?** Food Sprite, Food Coke

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

**13. How often do they drink *non-food* carbonated (fizzy) drinks? E.g.** Sprite, Coke

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

**14a. Bread (including toast and bread rolls)**

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

**14b.** What **type of bread** does your child **usually** eat? (fill one circle)

- o White
- o Wholemeal, wheatmeal or mixed grain
- o None

## 15a. Breakfast cereal

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

## 15b. What **kind of milk** was usually added to their cereal? (fill one circle)

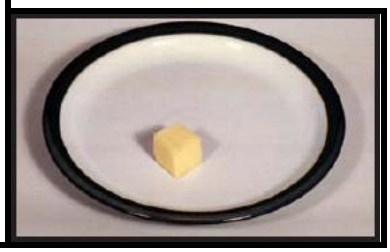
- o None
- o Low fat (light blue)
- o Extra calcium
- o Standard (dark blue top)
- o Trim (green)
- o Soy milk
- o Other milk (please name) _____

## 16. Ice cream

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

## 17. **Cheese,** e.g. Cheddar, Colby etc.

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |



## 18. **Yoghurt or Dairy food** (all types)

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

**We are now going to ask you some questions about your child's food habits.**

**19.** How **often** do they drink **milk provided** by their **school?**

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

**20a. Does your child take dietary supplements such as vitamins and minerals?**

o Yes o No

**If yes, please specify what kind**

**20b. How often would they take dietary supplements?**

| Never or less than once a month | 1-3 times a month | 1-2 times a week | 3-4 times a week | 5-6 times a week | Once a day | 2 or more times a day |
|---|---|---|---|---|---|---|
| o | o | o | o | o | o | o |

**21a. Are there any sorts of food your child does not eat?**

o Yes o No o Don't know

**21b. If yes, Which of the following foods do they <u>NOT</u> eat?**

o All meats

o Red meat

o Chicken

o Fish

o Dairy products

o Eggs

o Other

_____

**Now we will ask you questions about how often your child watches TV or videos or play on a computer when they are not at school.**

**22. During a normal day, how long does your child watch television, videos and/or DVD's?**

| | 0 -30 minutes | 30-60 minutes | 1 - 1.5 hours | 1.5 - 2 hours | 2 - 2.5 hours | 2 .5- 3 hours | 3 - 3.5 hours | 3.5- 4 hours | 4 - 4.5 hours | 4.5 -5 hours | over 5 hours |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Monday-Friday | o | o | o | o | o | o | o | o | o | o | o |
| Saturday-Sunday | o | o | o | o | o | o | o | o | o | o | o |

**23. During a normal day, how long does your child spend on computer, playstation, X-box, hand held games etc?**

| | 0 -30 minutes | 30-60 minutes | 1 - 1.5 hours | 1.5 - 2 hours | 2 - 2.5 hours | 2 .5- 3 hours | 3 - 3.5 hours | 3.5- 4 hours | 4 - 4.5 hours | 4.5 -5 hours | over 5 hours |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Monday-Friday | o | o | o | o | o | o | o | o | o | o | o |
| Saturday-Sunday | o | o | o | o | o | o | o | o | o | o | o |

**We are now going to ask you questions about the physical activities your child does in their spare time. Physical activities are 'sports, games, gym, dance or other activities that make them breathe harder, make their legs feel tired or make them sweat'.**

**We are going to ask you how many times in the last month your child has done each of the activities.**

**24.** Consider the **past month**, how **many hours** do they spend **outdoors** when **at home?**

(Monday-Friday = before and after school)

| | 0 -30 minutes | 30-60 minutes | 1 - 1.5 hours | 1.5 - 2 hours | 2 - 2.5 hours | 2 .5- 3 hours | 3 - 3.5 hours | 3.5- 4 hours | 4 - 4.5 hours | 4.5 -5 hours | over 5 hours |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **Monday-Friday** | o | o | o | o | o | o | o | o | o | o | o |
| **Saturday-Sunday** | o | o | o | o | o | o | o | o | o | o | o |

**25a. During leisure time, how often does your child play active games** e.g. chase, running, tag, hopscotch**?**

| | Never | Less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week |
|---|---|---|---|---|---|---|
| **Active games** | o | o | o | o | o | o |

**25b.** How **much time** do they spend **each time** they play **active games?**

| Less than one hour | 1–2 hours | 2–4 hours | 4-5 hours | 6+ hours | Don't know |
|---|---|---|---|---|---|
| o | o | o | o | o | o |

## 26. What activities does your child do?

*Please tick all the relevant activities the child has participated in for at least 20 minutes at a time, in the seven days*

| | Never | Less than once per month | 1-3 times per month | Once per week | 2-4 times per week | 5-6 times per week |
|---|---|---|---|---|---|---|
| **Team Sports e.g. soccer, rugby, basketball** | O | O | O | O | O | O |
| **Individual Sports e.g. Tennis** | O | O | O | O | O | O |
| **Gymnastics e.g. bars, beam, tumbling, trampoline** | O | O | O | O | O | O |
| **Outdoor chores e.g. mowing, gardening, farmwork** | O | O | O | O | O | O |
| **Indoor chores e.g. vacuuming** | O | O | O | O | O | O |
| **Skateboarding** | O | O | O | O | O | O |
| **Cycling** | O | O | O | O | O | O |
| **Walking** | O | O | O | O | O | O |
| **Ball-playing e.g. four square, dodgeball** | O | O | O | O | O | O |

**Please take a moment to check that you have answered all questions.**

# Thank you and your family taking part.

# Your participation is very much appreciated.

**Please do not forget to hand in this questionnaire to your school on the day that your child is to be assessed. If you cannot be there, please ensure your child has it with them when they come to the assessment day.**