Available online at www.sciencedirect.com
ScienceDirect
Metabolism Clinical and Experimental 57 (2008) 511 – 516
Metabolism
Clinical and Experimental
www.elsevier.com/locate/metabol

Body mass index status is effective in identifying metabolic syndrome components and insulin resistance in Pacific Island teenagers living in New Zealand

Andrea M. Grant[a], Finau K. Taungapeau[b], Kirsten A. McAuley[c], Rachael W. Taylor[d], Sheila M. Williams[e], Margaret A. Waldron[d], Ashley W. Duncan[d], Michelle J. Harper[d], Ailsa Goulding[a,*]

[a]*Department of Medical and Surgical Sciences, University of Otago, PO Box 913, Dunedin 9054, New Zealand*
[b]*Otago Pacific Peoples' Health Trust, Dunedin 9054, New Zealand*
[c]*The Edgar National Centre for Diabetes Research at the University of Otago, PO Box 913, Dunedin 9054, New Zealand*
[d]*Department of Human Nutrition, University of Otago, PO Box 913, Dunedin 9054, New Zealand*
[e]*Department of Preventive and Social Medicine, University of Otago, PO Box 913, Dunedin 9054, New Zealand*

Received 17 May 2007; accepted 7 November 2007

Abstract

Although adults of Pacific ethnicity living in New Zealand have more than double the prevalence of diabetes and cardiovascular disease than the general population, little is known regarding the presence of risk factors for these disorders among young Pacific Islanders. The study aim was to examine relationships between body composition, glucose and lipid metabolism, and components of the metabolic syndrome (MS) in a community sample of Pacific Island (PI) teenagers living in Dunedin. Anthropometry, body composition (dual-energy x-ray absorptiometry), glucose and lipid metabolism, insulin resistance (homeostasis model assessment [HOMA2], McAuley index), and components of MS were assessed in 80 PI teenagers (aged 15-18 years). Results showed that 6 participants had full MS, 2 had high fasting blood glucose values (>7.0 mmol/L), 55 had high adiposity, and 21 had insulin resistance. Assessment of the components of MS by body mass index (BMI) status showed that obese participants (n = 29) had a high prevalence (86.2% had one or more component), whereas only 10.5% of those with healthy BMI status (n = 19) had any MS component. Elevated fat mass had substantial effects on fasting insulin values, HOMA2, and the McAuley index because in data adjusted for age, sex, and lean mass, a 10% greater fat mass was associated with a 4.7% increase in fasting insulin, a 5.3% rise in HOMA2, and a 2.3% decrease in the McAuley index. Our results suggest that the antecedents of cardiovascular disease and type 2 diabetes mellitus occur frequently in young Pacific Islanders having high adiposity. We conclude that community studies of PI adolescents should focus on assessing risk factors whenever BMI values are high.

1. Introduction

Although adults of Pacific ethnicity have more than double the prevalence of diabetes (10% vs 4% for the New Zealand [NZ] population) [1,2], cardiovascular disease (390 per 100 000 vs 176 per 100 000 for the NZ population) [2,3], and metabolic syndrome (MS) (odds ratio, 2.54; 95% confidence interval [CI], 1.93-3.35) [4] than adult NZ Europeans, little is known regarding the presence of risk factors for these disorders among young Pacific Islanders. This is a concern given the rising prevalence of type 2 diabetes mellitus in overweight adolescents in many populations [5]. Young Pacific Islanders in NZ have extremely high levels of obesity [6]. In a recent representative survey [7], more than 60% of 1429 youngsters aged 5 to 14 years were overweight (males, 33.9%; females, 32.9%) or obese (males, 26.1%; females, 31%) from body mass index (BMI) [8]. Moreover, the prevalence of type 2 diabetes mellitus in adolescents attending Auckland diabetes clinics rose from 1.8% in 1996 to 12.5% in 2002, with all new cases being of Pacific Island (PI) ethnicity [9].

Insulin resistance (IR) generally precedes the onset of diabetes, and a clustering of risk factors known as the *metabolic syndrome* is independently associated with both type

* Corresponding author. Tel.: +64 3 474 0999x8516; fax: +64 3 474 7641.
E-mail address: ailsa.goulding@stonebow.otago.ac.nz (A. Goulding).

0026-0495/$ – see front matter © 2008 Elsevier Inc. All rights reserved.
doi:10.1016/j.metabol.2007.11.013

2 diabetes mellitus and cardiovascular disease [10]. Our study examines the relationships between body composition estimated by dual-energy x-ray absorptiometry, measures of glucose and lipid metabolism, and components of MS in a community sample of PI teenagers living in Dunedin, NZ.

2. Materials and methods

The University of Otago Ethics Committee approved the study protocol. Pacific Island volunteers (40 females, 40 males) aged 15 to 18 years were recruited by PI community workers who invited individuals of appropriate age and ethnicity to take part. This sample represents approximately one third of PI teenagers of this age living in Dunedin and was considered representative of the community. After a 10-hour overnight fast, each participant was weighed (electronic scale, Seca Corp, Ontario, CA) and measured (Harpenden stadiometer, Holtain Ltd, Crymych, UK) without shoes in light clothing. Body mass index (in kilograms per square meter) was calculated and graded as normal, overweight, or obese according to international cut points for age and sex [8]. Waist and hip girths were measured as described previously [11], and blood pressure (BP) was determined in a sitting position after a 10-minute rest (random zero sphygmomanometer). Fasting venous blood was taken for estimations of glucose, insulin, lipids, and C-reactive protein as described previously [12]. Adiponectin was measured by radioimmunoassay (Linco Diagnostics, St Charles, MO; intraassay coefficient of variation, 9.9%). Plasma samples were stored at −70°C before analysis.

Participants had an oral glucose tolerance test: after ingesting 75 g of dextrose, glucose and insulin were measured in venous blood at 120 minutes. Insulin resistance was estimated by the homeostatic model assessment (HOMA2) [13] using the HOMA2 calculator (using fasting glucose and fasting insulin, http://www.dtu.ox.ac.uk/homa) and by the McAuley index using fasting insulin and triglycerides, where predicted insulin sensitivity is expressed as exponent (2.63 − 0.28 ln[fasting insulin] − 0.31 ln[fasting triglycerides]) [12]. Homeostatic model assessment values >2 or McAuley index ≤6.3 indicated IR.

A total body scan was taken by dual-energy x-ray absorptiometry (DPX-L scanner, Lunar Corp, Cincinnati, OH) to measure total fat mass (in kilograms), lean mass (in kilograms), and fat percentage [11]. Scanning was performed by a single experienced operator and analyzed using the software package 1.35 (Lunar, Cincinnati, OH). Our in vivo coefficients of variation obtained from 10 repeated measurements of the total body taken on different days from a group of young adults are 2.6% for fat mass and 0.88% for lean mass.

Participants completed an interviewer-administered questionnaire concerning ethnicity, Tanner stage of pubertal development [13], general health, smoking (at least one cigarette daily), alcohol consumption (>3 drinks per week), family history of diabetes, habitual physical activity, time spent in vigorous activity, and time spent watching TV or playing computer/video games. Participants ranked their usual physical activity relative to youths of similar age and sex on a 5-point Likert scale (1 = least active, 5 = most active) [14].

Definitions for MS described by Cruz and associates [15] were used. These were based on the Adult Treatment Panel III recommendations [10] applying age- and sex-specific modifications derived from appropriate percentile cut points in National Health and Nutrition Examination Survey (NHANES) III adolescents. A diagnosis of MS required 3 or more of the following: abdominal obesity (waist circumference ≥90th percentile [16] or ≥88 cm for females and ≥102 cm for males), hypertriglyceridemia (triglycerides ≥90th percentile for age and sex [17]), low high-density lipoprotein (HDL) cholesterol (≤10th percentile [17]), hypertension (systolic or diastolic BP ≥90th percentile for height, age, and sex [18] or BP ≥130 systolic or ≥85 diastolic), or impaired glucose tolerance (IGT) (2-hour glucose ≥7.8 mmol/L) [10].

Statistical analysis was performed using the STATA statistical software package 7.0 (Stata, College Station, TX). Raw data are shown as mean ± standard deviation. Two-tailed ($P = .05$) tests of significance were used. Sensitivity, specificity, and positive predictive values were calculated by receiver operating characteristic analysis. Data were log-transformed before analysis. As both dependent and independent variables were log-transformed, the regression analysis can be interpreted in terms of percentage change in the dependent variable associated with a 10% elevation in body fat.

3. Results

Eighty-three percent of the adolescents invited to take part were enrolled. All participants were of PI ethnicity (Tongan, Samoan, Niuean, Cook Islanders, Tokelauan, Kiribati, or Fijian). Body mass index criteria indicated that 40% were overweight and a further 36% were obese. Eighteen participants weighed >100 kg, and 13 had BMI values >35 kg/m^2. Participants with higher BMI values had greater adiposity, higher waist and hip girths, higher fasting insulin and a greater degree of IR, lower HDL cholesterol, and greater triglyceride concentrations (Table 1).

Participants were at late stages of puberty (Table 2). About one fifth of the sample reported smoking, low physical activity, and regular alcohol consumption. Many (68%) had first- or second-degree relatives with type 2 diabetes mellitus.

High waist circumference was the most common component of MS, followed by low HDL, elevated systolic BP, and elevated triglycerides (Table 2). Although 2 females had elevated fasting glucose values (>7.0 mmol/L), only one had an abnormal oral glucose tolerance test result. Eighteen individuals had one MS component (22.5%) and 15 (18.8%) had 2 components, whereas 6 (7.5%) fully met the criteria for

Table 1
Characteristics of the 80 study participants grouped according to BMI status

BMI status [8]	Healthy (n = 19)	Overweight (n = 32)	Obese (n = 29)	*P* ANOVA*
Male, females (n)	11, 8	16, 16	13, 16	.68
Age (y)	16.7 (1.3)	16.3 (1.1)	16.4 (1.1)	.4
Height (cm)	173.0 (7.24)	169.9 (6.67)	172.3 (7.85)	.3
Weight (kg)	67.3 (6.82)	76.1 (8.06)	103.9 (15.9)	**.001**
BMI (kg/m^2)	22.4 (1.5)	26.3 (1.8)	35.0 (5.0)	
BMI *z* score	0.69 (0.55)	1.77 (0.34)	3.04 (0.50)	
Waist girth (cm)	76.0 (5.3)	83.7 (4.6)	101.9 (9.4)	**.001**
Hip girth (cm)	98.4 (4.7)	104.0 (4.7)	118.9 (9.1)	**.001**
Waist to -hip ratio	0.77 (0.05)	0.81 (0.04)	0.86 (0.06)	**.001**
Systolic BP (mm Hg)	109.6 (13.2)	112.5 (11.4)	116.4 (12.0)	.15
Diastolic BP (mm Hg)	56.5 (9.8)	59.9 (9.6)	65.6 (9.1)	**.005**
Fat mass (kg)	15.04 (6.11)	22.00 (7.92)	41.35 (11.98)	**.001**
Fat percentage	23.0 (9.7)	29.2 (9.9)	40.2 (9.1)	**.001**
Lean mass (kg)	48.41 (9.52)	50.17 (9.07)	57.80 (11.74)	**.003**
Fasting glucose (mmol/L)	4.44 (0.27)	4.61 (0.71)	4.72 (0.73)	.34
Fasting insulin[a] (μIU/mL)	8.0 (0.5)	11.3 (0.8)	17.0 (0.7)	**.002**
2-h glucose (mmol/L)	4.37 (0.91)	4.21 (0.96)	4.77 (1.36)	.15
2-h insulin (μIU/mL)	49.8 (19.7)	44.1 (32.5)	75.4 (63.0)	**.02**
McAuley index[b]	8.99 (1.95)	8.02 (2.07)	6.48 (1.72)	**.001**
HOMA2[b]	1.13 (0.78)	1.56 (1.13)	2.23 (1.32)	**.005**
HOMA (%S)[a]	102.9 (0.52)	79.2 (0.65)	52.9 (0.59)	**.001**
HOMA (%B)[a]	121.9 (0.28)	141.8 (0.45)	178.8 (0.40)	**.005**
Total cholesterol (mmol/L)	3.85 (0.69)	4.08 (0.56)	4.10 (0.64)	.33
LDL cholesterol (mmol/L)	2.25 (0.72)	2.62 (0.50)	2.53 (0.45)	.06
HDL cholesterol (mmol/L)	1.27 (0.25)	1.08 (0.26)	0.98 (0.20)	**.001**
Triglycerides (mmol/L)	0.72 (0.28)	0.85 (0.38)	1.27 (0.72)	**.001**
CRP (mg/L)	0.38 (0.28)	0.72 (0.92)	1.25 (2.16)	.11
Adiponectin (μg/mL)	6.00 (3.33)	6.37 (3.19)	6.00 (3.77)	.89
Minutes TV/d	124 (91)	185 (107)	170 (123)	.15
Minutes computer time/d	54 (71)	92 (120)	51 (71)	.18
Total inactivity (computer plus TV) min/d	178 (150)	278 (171)	221 (144)	.08

Results are mean (SD) for each group. ANOVA indicates analysis of variance; %S, insulin sensitivity; %B, β-cell function; CRP, C-reactive protein.
* Significant associations shown in bold.
[a] Geometric means.
[b] Excluded the 2 participants with IFG (glucose >6.1 mmol/L).

MS. Forty-one teenagers (51.3%) had no MS component. The MS components were predominantly observed in individuals with high adiposity (Fig. 1). In females, components of MS were only detected in overweight or obese subjects, whereas in males, 2 subjects with healthy BMI values had MS components (one high BP and one low HDL). Indeed, the sensitivity, specificity, and positive predictive value of an obese BMI predicting one or more MS component were 62.5% (95% CI, 45.8%-77.3%), 90% (95% CI, 76.3%-97.2%), and 86.2% (95% CI, 68.3%-96.1%).

Insulin resistance affected a quarter of the participants, whereas 35 (44% of the sample) had elevated fasting insulin (>12 μIU/mL). Moreover, 29 of the 55 participants with high percentage of body fat had high fasting insulin values. All participants with MS had both hyperinsulinemia and a high body fat percentage. Few participants with hyperinsulinemia had a healthy body fat percentage (n = 6). Only 19 of the 80 participants had neither elevated adiposity or high fasting insulin values. The sensitivity, specificity, and positive predictive value of an obese BMI predicting high fasting insulin were 54.3% (95% CI, 36.6%-71.2%), 77.8% (95% CI, 62.9%-88.8%), and 65.5% (95% CI, 45.7%-82.1%).

Fasting plasma insulin values were positively correlated with percentage of body fat (Table 3), waist girth, triglycerides, diastolic BP, and fasting glucose. Tanner stage was not associated with fasting plasma insulin values. High-density lipoprotein values were negatively correlated with fasting plasma insulin levels. Adiponectin showed a negative association with 2-hour glucose.

Elevated fat mass had substantial effects on fasting insulin values, HOMA2, and the McAuley index. In data adjusted for age, sex, and lean mass, a 10% greater fat mass was associated with a 4.7% increase in fasting insulin, a 5.3% rise in HOMA2, and a 2.3% decrease in the McAuley index. The effect of variations in lean mass on these measures was tested, but no significant effects were found.

4. Discussion

In this community sample of PI adolescents, abnormalities of MS and hyperinsulinemia were rare in those

Table 2
Weight status, pubertal status, and prevalence of risk factors in the sample (n = 80)

	Females (n = 40)	Males (n = 40)	Both (n = 80)
Healthy BMI, overweight, obese (n)	8, 16, 16	11, 16, 13	19, 32, 29
Tanner stage 3, 4, 5 (n)	3, 25, 12	8, 23, 9	11, 48, 21
Current or previous smoker	11	4	15
Current alcohol	3	11	14
Low self-rated physical activity (score <3)	9	8	17
1st degree, 2nd degree, or no family history of type 2 diabetes mellitus (n)	6, 24, 10	6, 19, 15	12, 43, 25
Components of MS (n [%])			
High waist girth [a]	18 (45.0)	9 (22.5)	27 (33.8)
High triglycerides [b]	3 (7.5)	4 (10.0)	7 (8.8)
Low HDL [c]	15 (37.5)	6 (15.0)	21 (26.3)
Raised systolic BP [d]	3 (7.5)	8 (20.0)	11 (13.8)
Raised diastolic BP [d]	2 (5.0)	1 (2.5)	3 (3.8)
IGT [e]	1 (2.5)	0	1 (1.3)
Any component of MS	21 (52.5)	18 (45.0)	39 (48.8)
Full MS [f]	4 (10.0)	2 (5.0)	6 (7.5)
IR (n [%])			
IR from McAuley index [g]	13 (34.2)	8 (20.0)	21 (26.9)
IR from HOMA2 [g]	14 (36.8)	7 (17.5)	21 (26.9)

[a] Waist girth ≥90th percentile for age and sex or >88 cm for females and >102 cm for males.
[b] Triglycerides ≥90th percentile for age and sex.
[c] High-density lipoprotein cholesterol ≤10th percentile for age and sex.
[d] Systolic or diastolic BP ≥90th percentile for height, age, and sex, or BP >130 systolic or >85 diastolic.
[e] Two-hour glucose >7.8 mmol/L.
[f] Three or more components.
[g] Excludes the 2 participants with IFG (fasting glucose >6.1 mmol/L).

having healthy BMI, but common in overweight or obese participants, in agreement with other work [19]. These results suggest that the increased risks of cardiovascular disease and type 2 diabetes mellitus present in adult Pacific Islanders are related to high adiposity, particularly central adiposity, starting in adolescence [20,21]. All participants with full MS had both high percentage of fat and hyperinsulinemia.

Others have reported a higher prevalence of MS and hyperinsulinemia with increased severity of adolescent obesity of different ethnicity [22-26]. Furthermore, the severity of IR appears to affect risks for type 2 diabetes mellitus and cardiovascular disease. Bacha and associates [27] found that youths with severe IR displayed greater visceral adiposity, poorer glucose disposal, a more abnormal lipid profile, and lower adiponectin values than those with greater insulin sensitivity but similar BMI values. In our study, one or more elements of MS were observed in a significantly higher proportion of youths with IR than in those with greater insulin sensitivity and healthy HOMA2 or McAuley index values. Adiponectin values did not differ in groups of different BMI status, but were generally low as seen in high-risk populations [28].

Although a study limitation is that our sample was not randomly selected, we do not consider the sample to be selected from a particular weight range; and indeed, the prevalences of overweight and obesity we observed were consistent with findings in a recent representative nationwide survey [7], suggesting that our findings have wide application to the public health of contemporary PI adolescents living in NZ. Strengths of our study are that comprehensive measures of body composition, IR, and glucose and lipid metabolism were obtained. In addition, our sample was drawn from the community, not from adolescents attending obesity clinics. Evidence that obese BMI status was associated in PI adolescents with higher health risks than healthy BMI status should facilitate wider identification of individuals with risks for cardiovascular disease and type 2 diabetes mellitus because age- and sex-specific BMI values can be categorized readily in large samples. In addition, BMI is less intrusive and simpler than undertaking a fasting blood sample; and in our sample, a nonobese BMI had a high specificity to exclude adolescents without any component of the MS. Although the merits of defining MS as a clinical entity may be in dispute [29], the importance and value of identifying and treating the components are beyond question.

Although Pacific Islanders often have greater lean mass for a given BMI [30] or height [31] than whites, dual-energy x-ray absorptiometry measurements established that participants with higher BMI values and MS components had elevated body fat percentages, showing that these youngsters had high adiposity and not merely a high lean mass [32]. We did not detect any significant decrease in plasma insulin with increasing lean mass, although high lean mass may enable better glucose disposal than ethnic groups with lower lean mass.

Genetic and lifestyle factors each contribute to known ethnic differences in the prevalences of diabetes and cardiovascular disease. Some ethnic groups, such as Pima Indians [33] and Hispanics [15], have been reported to show

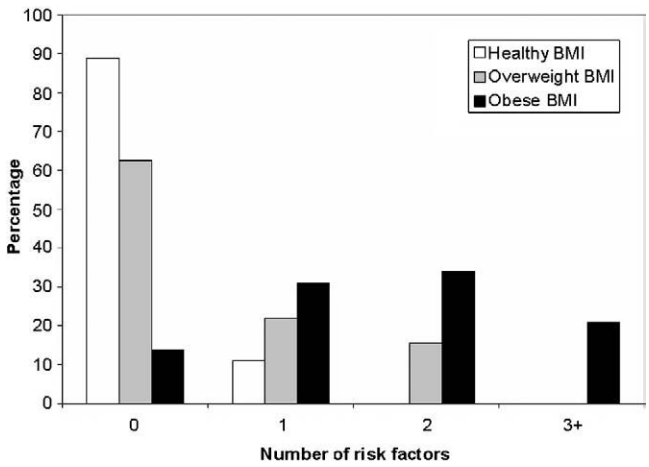

Fig. 1. Number of abnormalities for the MS present in groups of different BMI status.

Table 3
Correlations and partial correlations (n = 80)

	Fat %	Waist girth	LnHDL	LnTag	Syst BP	Diast BP	LnFasting glucose	Ln2-hr glucose
Ln insulin	**0.423**	**0.470**	**−0.324**	**0.451**	0.099	**0.408**	**0.556**	**0.383**
Adiponectin	0.01	−0.094	0.137	0.009	0.072	−0.163	−0.167	**−0.237**
Ln insulin [a]	**0.353**	**0.490**	**−0.309**	**0.445**	0.211	**0.410**	**0.586**	0.210
Adiponectin [a]	0.026	−0.121	0.176	0.016	0.128	−0.126	−0.135	**−0.242**

Significant associations shown in bold.
[a] Adjusted for age and sex.

more severe disorders of lipid and glucose metabolism in adolescents than we observed. This may relate to the greater severity of obesity in samples studied. Alternatively, we speculate that Pacific Islanders may retain better insulin secretory reserves at this age.

In 2430 US adolescent participants of NHANES III, Cook and associates [34] found that 4.2% had MS and 28.7% of those with BMI values >95th percentile for age had this diagnosis. A subsequent survey of a large sample of US adolescents from NHANES 1999-2000 reported MS in 6.4%, with 32.1% of obese participants having this condition [35]. A recent survey in Iran found 10.1% of 3036 adolescents had full MS [36] with more than 60% of obese individuals having MS. Invitti and associates [37] reported that 23% of 588 obese Italian children had MS. In our study, 6 of 80 teenagers had MS (7.5%); and the prevalence was 20.7% among those with obesity.

Duncan and associates [35] found that 1.1% of their sample from NHANES 1999-2000 displayed elevated fasting plasma glucose. In our study, which included many severely obese adolescents, only 2 individuals had high fasting glucose values, with only one of the affected female being obese. This is a smaller proportion than was reported in the United States where 21% of a group of 112 markedly obese adolescents attending obesity clinics had this condition [22]. Similarly, 10.3% of 126 obese British children had IGT and elevated fasting glucose [38]. Some have reported a higher prevalence of IGT among obese children with a parental history of type 2 diabetes mellitus [36,38], although others, as in the present study, found no association between family history of diabetes with hyperinsulinemia or abnormalities of MS [39].

The observation that only a small proportion of our PI teenagers had aberrant glucose metabolism despite the high prevalence of obesity suggests that interventions in adolescence have the potential to prevent progression to type 2 diabetes mellitus. This is especially important because youths with type 2 diabetes mellitus have more health complications than those with type 1 diabetes mellitus [40], despite a shorter duration of diabetes. Moreover, adolescents who are already obese have the potential to improve their risk profiles because others have shown that lifestyle interventions can improve risk factors for MS and IR in adolescents [41], as can a combination of lifestyle change and metformin [42].

To conclude, this research establishes that metabolic abnormalities are frequently present in PI teenagers, particularly those with high adiposity. Risk factors for diabetes and cardiovascular disease should therefore be assessed early in all individuals with high BMI values.

Acknowledgment

The study was supported by an Otago Research Grant and a Distinguished Researcher Award from the University of Otago.

References

[1] Simmons D, Thompson CF, Volklander D. Polynesians: prone to obesity and type 2 diabetes mellitus but not hyperinsulinaemia. Diabet Med 2001;18:193-8.
[2] Ministry of Health and Ministry of Pacific Island Affairs. Tupu Ola Moui: Pacific health chart book 2004. Wellington: Ministry of Health; 2004.
[3] Bell AC, Swinburn BA, Simmons D, Wang W, Amosa H, Gatland B. Heart disease and diabetes risk factors in Pacific Island communities and associations with measures of body fat. N Z Med J 2001;114: 208-13.
[4] Gentles DGR, Metcalf PA, Dyall L, Sundborn G, Schaaf D, Black PN, et al. Metabolic syndrome prevalence in a multicultural population in Auckland, New Zealand. N Z Med J 2007;120:8. http://www.nzma.org.nz/journal/120-1248/2387/.
[5] Cruz ML, Shaibi GQ, Weigensberg MJ, Spruijt-Metz D, Ball GDC, Goran MI. Pediatric obesity and insulin resistance: chronic disease risk and implications for treatment and prevention beyond body weight modification. Ann Rev Nutr 2005;25:435-68.
[6] Gordon FK, Ferguson EL, Toafa V, Henry TE, Goulding A, Grant AM, et al. High levels of childhood obesity observed among 3- to 7-year-old New Zealand Pacific children is a public health concern. J Nutr 2003; 133:3456-60.
[7] Ministry of Health. NZ food NZ children: key results of the 2002 National Children's Nutrition Survey. Wellington: Ministry of Health; 2003.
[8] Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ 2000;320:1240-3.
[9] Hotu S, Carter B, Watson PD, Cutfield WS, Cundy T. Increasing prevalence of type 2 diabetes in adolescents. J Paediatr Child Health 2004;40:201-4.
[10] Reaven GM. The metabolic syndrome: is this diagnosis necessary? Am J Clin Nutr 2006;83:1237-47.
[11] Taylor RW, Jones IE, Williams SM, Goulding A. An evaluation of waist circumference, waist-to-hip ratio and the conicity index as screening tools for high trunk fat, as measured by dual-energy x-ray absorptiometry, in children aged 3-19 yr. Am J Clin Nutr 2000;72: 490-5.

[12] McAuley KA, Williams SM, Mann JI, Walker RJ, Lewis-Barned NJ, Temple LA, et al. Diagnosing insulin resistance in the general population. Diabetes Care 2001;24:460-4.
[13] Levy JC, Matthews DR, Hermans MP. Correct homeostasis model assessment (HOMA) evaluation uses the computer program. Diabetes Care 1998;21:2191-2.
[14] Goulding A, Jones IE, Taylor RW, Williams SM, Manning PJ. Bone mineral density and body composition in boys with distal forearm fractures: a dual-energy x-ray absorptiometry study. J Pediatr 2001; 139:509-15.
[15] Cruz ML, Weigensberg MJ, Huang TTK, Ball G, Shaibi GQ, Goran MI. The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. J Clin Endocrinol Metab 2004;89:108-13.
[16] Fernandez JR, Redden DT, Pietrobelli A, Allison DB. Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. J Pediatr 2004;145:439-44.
[17] Hickman TB, Briefel RR, Carroll MG, Rifkind BM, Cleeman JI, Maurer KR, et al. Distributions and trends of serum lipid levels among United States children and adolescents ages 4-19 years: data from the Third National Health and Nutrition Examination Survey. Prev Med 1998;27:879-90.
[18] National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics 2004;114:555-76.
[19] Gutin B, Yin ZN, Humphries MC, Hoffman WH, Gower B, Barbeau P. Relations of fatness and fitness to fasting insulin in black and white adolescents. J Pediatr 2004;145:737-43.
[20] Cook S. The metabolic syndrome: antecedent of adult cardiovascular disease in pediatrics. J Pediatr 2004;145:427-30.
[21] Morrison JA, Friedman LA, Harlan WR, Harlan LC, Barton BA, Schreiber GB, et al. Development of the metabolic syndrome in black and white adolescent girls: a longitudinal assessment. Pediatrics 2005; 116:1178-82.
[22] Sinha R, Fisch G, Teague B, Tamborlane WV, Banyas B, Allen K, et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. N Engl J Med 2002;346:802-10.
[23] Goran MI, Ball GDC, Cruz ML. Cardiovascular endocrinology 2—obesity and risk of type 2 diabetes and cardiovascular disease in children and adolescents. J Clin Endocrinol Metab 2003;88:1417-27.
[24] Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, et al. Obesity and the metabolic syndrome in children and adolescents. N Engl J Med 2004;350:2362-74.
[25] Viner RM, Segal TY, Lichtarowicz-Krynska E, Hindmarsh P. Prevalence of the insulin resistance syndrome in obesity. Arch Dis Child 2005;90:10-4.
[26] Weiss R, Caprio S. The metabolic consequences of childhood obesity. Best Prac Res Clin Endocrinol Metab 2005;19:405-19.
[27] Bacha F, Saad R, Gungor N, Arslanian SA. Are obesity-related metabolic risk factors modulated by the degree of insulin resistance in adolescents? Diabetes Care 2006;29:1599-604.
[28] Lee S, Bacha F, Gungor N, Arslanian SA. Racial differences in adiponectin in youth. Relationship to visceral fat and insulin sensitivity. Diabetes Care 2006;29:51-6.
[29] Jones KL. The dilemma of the metabolic syndrome in children and adolescents: disease or distraction? Pediatr Diabetes 2006;7:311-21.
[30] Rush E, Plank LD, Chandu V, Laulu MS, Simmons D, Swinburn BA, et al. Body size, body composition, and fat distribution: a comparison of young New Zealand men of European, Pacific Island, and Asian Indian ethnicities. N Z Med J 2004;117:9. http://www.nzma.org.nz/journal/117-1207/1203/.
[31] Grant AM, Gordon FK, Ferguson EL, Williams SM, Henry TE, Toafa VM, et al. Do young New Zealand Pacific and European children differ in bone size or bone mineral? Calcif Tissue Int 2005;76:397-403.
[32] Freedman DS, Wang J, Maynard LM, Thornton JC, Mei Z, Pierson RN, et al. Relation of BMI to fat and fat-free mass among children and adolescents. Int J Obes 2005;29:1-8.
[33] Fagot-Campagna A, Burrows NR, Williamson DF. The public health epidemiology of type 2 diabetes in children and adolescents: a case study of American Indian adolescents in the Southwestern United States. Clin Chim Acta 1999;286:81-95.
[34] Cook S, Weitzman M, Auinger P, Nguyen M, Dietz WH. Prevalence of a metabolic syndrome phenotype in adolescents—findings from the Third National Health and Nutrition Examination Survey, 1988-1994. Arch Pediatr Adolesc Med 2003;157:821-7.
[35] Duncan GE, Li SM, Zhou XH. Prevalence and trends of a metabolic syndrome phenotype among U.S. adolescents, 1999-2000. Diabetes Care 2004;27:2438-43.
[36] Esmaillzadeh A, Mirmiran P, Azadbakht L, Etemadi A, Azizi F. High prevalence of the metabolic syndrome in Iranian adolescents. Obesity 2006;14:377-82.
[37] Invitti C, Maffeis C, Gilardini L, Pontiggia B, Mazzilli G, Girola A, et al. Metabolic syndrome in obese Caucasian children: prevalence using WHO-derived criteria and association with nontraditional cardiovascular risk factors. Int J Obes 2006;30:627-33.
[38] Sabin MA, Ford AL, Holly JMP, Hunt LP, Crowne EC, Shield JPH. Characterisation of morbidity in a UK, hospital based, obesity clinic. Arch Dis Child 2006;91:126-30.
[39] Goran MI, Coronges K, Bergman RN, Cruz ML, Gower BA. Influence of family history of type 2 diabetes on insulin sensitivity in prepubertal children. J Clin Endocrinol Metab 2003;88:192-5.
[40] Eppens MC, Craig ME, Cusumano J, Hing S, Chan AKF, Howard NJ, et al. Prevalence of diabetes complications in adolescents with type 2 compared with type 1 diabetes. Diabetes Care 2006;29: 1300-6.
[41] Monzavi R, Dreimane D, Geffner ME, Braun S, Conrad B, Klier M, et al. Improvement in risk factors for metabolic syndrome and insulin resistance in overweight youth who are treated with lifestyle intervention. Pediatrics 2006;117:E1111-8.
[42] Fu JF, Liang L, Zou CC, Hong F, Wang CL, Wang XM, et al. Prevalence of the metabolic syndrome in Zhejiang Chinese obese children and adolescents and the effect of metformin combined with lifestyle intervention. Int J Obes 2007;31:15-22.