2010

Dietary patterns and markers for the metabolic syndrome in Australian adolescents

Gina L. Ambrosini
Curtin University of Technology, Gina.Ambrosini@mrc-hnr.cam.ac.uk

Rae-Chi Huang
University of Western Australia, rhuang@meddent.uwa.edu.au

Trevor A. Mori
University of Western Australia, trevor.mori@uwa.edu.au

Beth P. Hands
University of Notre Dame Australia, bhands@nd.edu.au

Therese O'Sullivan
University of Western Australia, tosullivan@ichr.uwa.edu.au

See next page for additional authors

This article was originally published as:
Ambrosini, G. L., Huang, R., Mori, T. A., Hands, B. P., O'Sullivan, T., de Klerk, N. H., Beilin, L. J., & Oddy, W. H. (2010). Dietary patterns and markers for the metabolic syndrome in Australian adolescents. *Nutrition, Metabolism and Cardiovascular Diseases, 20* (4), 274-283.
http://doi.org/10.1016/j.numecd.2009.03.024

ARTICLE IN PRESS
+ MODEL
Nutrition, Metabolism & Cardiovascular Diseases (2009) xx, 1–10

available at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/nmcd

Nutrition, Metabolism & Cardiovascular Diseases

Dietary patterns and markers for the metabolic syndrome in Australian adolescents☆

G.L. Ambrosini [a,b], R-C. Huang [c], T.A. Mori [c], B.P. Hands [d], T.A. O'Sullivan [b], N.H. de Klerk [b], L.J. Beilin [c], W.H. Oddy [b,*]

[a] School of Public Health, Curtin University of Technology, Western Australia, Australia
[b] Telethon Institute for Child Health Research, Centre for Child Health Research, University of Western Australia, Australia
[c] School of Medicine and Pharmacology, Royal Perth Hospital Unit, University of Western Australia, Australia
[d] School of Health Sciences, University of Notre Dame, Fremantle, Western Australia, Australia

Received 5 February 2009; received in revised form 22 March 2009; accepted 27 March 2009

KEYWORDS
Adolescents;
Metabolic syndrome;
Dietary patterns;
Cardiovascular disease;
Biochemistry;
Cohort study;
Raine Study

Abstract *Background and aims:* Overweight and other risk factors for cardiovascular disease (CVD) as well as their clustering, are increasingly prevalent among adolescents. We examined dietary patterns, CVD risk factors, and the clustering of these risk factors in 1139 14-year-olds living in Western Australia.
Methods and results: Usual dietary intake was assessed using a food frequency questionnaire. Two dietary patterns, 'Western' and 'Healthy', were identified using factor analysis. Associations between these dietary patterns and BMI, waist circumference, systolic blood pressure, fasting levels of serum glucose, insulin, total cholesterol, HDL-C, LDL-C, triglycerides and insulin resistance were assessed using ANOVA. Cluster analysis identified a high risk group (the 'high risk metabolic cluster') with features akin to adult metabolic syndrome. Belonging to the 'high risk metabolic cluster' was examined in relation to dietary patterns using logistic regression, adjusting for aerobic fitness and socio-demographic factors. Higher 'Western' dietary pattern scores were associated with greater odds for the 'high risk metabolic cluster' (p for trend = 0.02) and greater mean values for total cholesterol (p for trend = 0.03), waist circumference (p for trend = 0.03) and BMI (p for trend = 0.02) in girls, but not boys. Scores for the 'Healthy' dietary pattern were not related to the 'high risk metabolic cluster' but were

☆ Sources of support: Telethon Institute for Child Health Research, Raine Medical Research Foundation, Telstra Research Foundation of Australia, Australian Rotary Health Research Fund, Western Australian Health Promotion Research Foundation (Healthway), and Australian National Health and Medical Research Council (Program Grant).
* Corresponding author. Telethon Institute for Child Health Research, P.O. Box 855, West Perth WA 6872, Australia. Tel.: +61 8 9489 7879; fax: +61 8 9489 7700.
E-mail address: wendyo@ichr.uwa.edu.au (W.H. Oddy).

0939-4753/$ - see front matter
doi:10.1016/j.numecd.2009.03.024

inversely associated with serum glucose in boys and girls (p for trend = 0.01 and 0.04, respectively) and were positively associated with HDL-C in boys (p for trend = 0.02).
Conclusions: Dietary patterns are associated with CVD risk factors and the clustering of these risk factors in adolescence.

Introduction

The pathological process of cardiovascular disease (CVD) begins during childhood [1] and the prevalence of CVD risk factors, including overweight, central adiposity, hyperlipidemia, hypertension and markers of impaired glucose metabolism, is increasing among children and adolescents [1,2]. The clustering of these risk factors (among others) is referred to as the metabolic syndrome, and increases the risk of CVD and type 2 diabetes mellitus beyond that of its individual components [3]. Although there is currently no universally accepted definition for the metabolic syndrome in childhood, there is evidence that the metabolic syndrome is increasing in prevalence among children and adolescents [1,2]. As these risk factors are known to track into adulthood [2], identifying ways of reducing their prevalence is of high public health importance.

Obesity is central to the metabolic syndrome and the increasing prevalence of the metabolic syndrome has mirrored that of the global overweight and obesity epidemics [2,3]. Obese and overweight children have the highest risk for the metabolic syndrome, and prevalence studies report that 9%–38% have some form of the condition [2]. Therefore, in addition to physical activity, dietary composition may be an important modifier of risk. The research on diet and the metabolic syndrome in children or adolescents is limited, although high intakes of fruits and vegetables have been linked with a lower risk for the metabolic syndrome [4–8].

Dietary patterns determined using factor analysis have been shown to be useful for assessing diet-disease relationships [9]. Analyses using dietary patterns consider the total diet and avoid attempts to separate effects from individual foods and nutrients, which are likely to be highly correlated or too small to detect [9]. The aim of this study was to investigate relationships between dietary patterns and markers for the metabolic syndrome and CVD risk, including body mass index (BMI), waist circumference (WC), systolic blood pressure (SBP), serum lipids, glucose, insulin, and insulin resistance in adolescents participating in the Western Australian Pregnancy Cohort (Raine) Study.

Methods

Study population

Details of the Raine Study have been published previously [10]. In brief, 2900 women at 16–20 weeks gestation were recruited through the public antenatal clinic and local private clinics in Perth, Western Australia between 1989 and 1991. A total of 2804 women (97%) had 2868 live births, and these children have been followed up at birth and ages one, two, three, five, eight, 10 and 14 years. This paper uses data collected at the 14-year follow up.

All data collection for the Raine Study occurred in accordance with Australian National Health and Medical Research Council Guidelines for Ethical Conduct in Human Research and was approved by the ethics committees of King Edward Memorial Hospital for Women and Princess Margaret Hospital for Children, Perth, Western Australia. Informed consent was obtained from the adolescent and their primary caregiver.

Dietary patterns

We have previously reported how dietary patterns were derived in this cohort at age 14 years [11]. In brief, an evaluated (Ambrosini G.L. et al, *manuscript under review*) semi-quantitative food frequency questionnaire (FFQ) was mailed to the study adolescents' primary caregiver to complete with the adolescent. The FFQ collected information on intake over the previous year of 212 foods or dishes, which were later collapsed into 38 food groups defined *a priori* [11]. Using PROC FACTOR in SAS [12] we applied common factor analysis (maximum likelihood method) on all 38 food group intakes to identify major dietary patterns. The factor solution was limited to factors with an eigen value > 1. The scree plot assisted with determining the number of factors to retain. Varimax rotation was applied to the factor solution to improve interpretability and retain independent patterns. The two major dietary patterns; 'Western' and 'Healthy' explained 84% of the total variance in food group intakes. Foods with a factor loading greater than |0.30| were the main contributors to dietary pattern scores and thus, best characterise each pattern (Table 1). These dietary patterns were similar when analysed separately for boys and girls [11], therefore, dietary patterns based on boys and girls combined, are presented. Every subject received a score for each dietary pattern measured on the z-scale. These scores were categorised into quartiles based on the total sample distribution.

Biochemistry

Blood samples were collected from consenting study participants after fasting overnight. Samples were analysed in the PathWest Laboratories at Royal Perth Hospital for serum insulin, glucose, triglycerides, total cholesterol, HDL-cholesterol (HDL-C) and calculated LDL-cholesterol (LDL-C), using standardised methodologies. Homeostasis Model Assessment (HOMA) was calculated as a measure of insulin sensitivity using the formula: [fasting insulin (μU/ml) × fasting glucose (mM)]/22.5 [13]. Full details of serum assay methods have been published previously [14].

Please cite this article in press as: Ambrosini GL, et al., Dietary patterns and markers for the metabolic syndrome in Australian adolescents, Nutr Metab Cardiovasc Dis (2009), doi:10.1016/j.numecd.2009.03.024

Table 1 Dietary patterns and their factor loadings in the Raine Cohort at 14 years of age.

Food group	Factor loadings[a]	
	'Healthy' pattern	'Western' pattern
Whole grains	*0.39*	−0.12
Yellow or red vegetables	*0.56*	0.12
Leafy green vegetables	*0.49*	0.00
Tomato	*0.49*	0.00
Cruciferous vegetables	*0.48*	0.27
Other vegetables	*0.66*	0.22
Fresh fruit	*0.48*	−0.02
Legumes	*0.43*	0.19
Fish, steamed, grilled or tinned	*0.33*	0.05
Red meat	0.14	*0.46*
Processed meats	−0.02	*0.41*
Takeaway foods	−0.20	*0.53*
Refined grains	0.03	*0.42*
Potato, fried e.g. french fries	−0.25	*0.39*
Potato, not fried	0.21	*0.34*
Confectionery	−0.14	*0.46*
Crisps	−0.22	*0.39*
Soft drinks	−0.18	*0.37*
Cakes, biscuits	0.10	*0.34*
Sauces and dressings	0.13	*0.34*
Full fat dairy products	0.00	*0.30*
Low fat dairy products	0.22	−0.10
Milk dishes	0.13	0.20
Fish, fried or battered	0.02	0.23
Poultry	0.01	0.29
Meat dishes	0.26	0.15
Eggs	0.20	0.24
Soups	0.26	0.26
Canned fruit	0.26	0.11
Dried fruit	0.23	0.00
Mineral water	0.23	−0.05
Juices	0.19	−0.02
Added sugar	0.13	0.21
Nuts	0.17	−0.02
% Variance	50	34

Written permission to reproduce this table from [11] has been provided by Cambridge University Press.

[a] Those foods with a factor loading $>|0.30|$ are shown in italics.

Anthropometry and physical activity

All adolescents eligible for the 14-year follow up were requested to attend the Telethon Institute for Child Health Research in Perth, Western Australia, for physical examinations. These included calibrated measurements of height and weight using electronic chair scales and stadiometer. WC was measured by horizontally positioning a tape measure across the belly button and at the smallest girth at the back, with the average of two measurements being used. Blood pressure was measured after resting supine for 5 min. A Dinamap recorder automatically recorded blood pressure every 2 min, and the average of the second and subsequent SBP readings was used for this analysis.

Physical inactivity is an independent risk factor for the metabolic syndrome in adolescents [8]. We used an objective estimate of aerobic fitness which was measured on a bicycle ergometer using the Physical Working Capacity 170 (PWC 170) test, whereby the power output (watts) required at a heart rate of 170 beats per minute was estimated. This measure is highly correlated with self-reported physical activity level in this cohort [15].

Identifying adolescents at high risk for the metabolic syndrome

Controversy exists over defining the metabolic syndrome in adolescents and to date, there is no universally accepted criteria [1,2]. As an alternative to definitions for the metabolic syndrome, a two-step cluster analysis was previously conducted in this cohort using BMI, SBP, serum triglycerides and HOMA measured at 14 years of age [14]. This classified adolescents into one of two clusters (or groups) indicating risk for the metabolic syndrome: the 'high risk metabolic cluster' and 'low risk metabolic cluster' [14]. The differences between these groups are shown in Table 2.

Statistical methods

We hypothesised that the 'Western' dietary pattern would be positively associated with the 'high risk metabolic cluster' and with components of the metabolic syndrome including: higher BMI, WC, SBP, fasting insulin, glucose, triglycerides, total cholesterol, LDL-C and HOMA, and lower HDL-C. We also hypothesised that the opposite would be true for the 'Healthy' dietary pattern.

To describe the dietary patterns, selected nutrient intakes, BMI and WC were compared across quartiles of the dietary pattern scores using ANOVA and adjusting for total energy intake. Logistic regression was used to estimate the odds of being in the 'high risk metabolic cluster' according to both dietary patterns using PROC LOGISTIC in SAS [12]. Analysis of variance was used to compare mean values for individual components of the metabolic syndrome according to quartiles of the dietary pattern scores using PROC GLM in SAS, with p-values adjusted for multiple comparisons using the Dunnett-Hsu method. Variables showing non-normal distributions, including serum insulin and triglycerides, HOMA, BMI and WC, were log-transformed before analyses and exponentiated for reporting.

We have previously reported that the 'Healthy' and 'Western' dietary patterns in this cohort are correlated with socio-demographic and lifestyle factors, including sex, maternal education, being in a two parent family and hours spent watching television per day [11]. Therefore, we considered these variables as potential confounders, along with aerobic fitness (watts) and total energy intake (MJ). Total energy intake was positively correlated with both dietary patterns [11] and may be an independent risk factor for the outcomes examined in this study.

BMI and WC were included in models where biochemical measurements or blood pressure was the outcome. Both BMI and WC may be important predictors of the metabolic syndrome [16] but the two are highly correlated in this cohort. Therefore, we classified individuals into one of four

Please cite this article in press as: Ambrosini GL, et al., Dietary patterns and markers for the metabolic syndrome in Australian adolescents, Nutr Metab Cardiovasc Dis (2009), doi:10.1016/j.numecd.2009.03.024

Table 2 Characteristics of subjects with FFQ and metabolic syndrome data in the Raine Cohort 14-year follow up.

	'High risk metabolic cluster' ($n = 318$)	'Low risk metabolic cluster' ($n = 821$)	
	n (%)[a]	n (%)[a]	p-Value[b]
Gender			
Female	173 (54.4)	373 (45.4)	0.007
Male	145 (45.6)	448 (54.6)	
Combined BMI and waist circumference			
Lean or normal weight, acceptable waist	78 (24.9)	664 (81.7)	<0.0001
Lean or normal weight, unacceptable waist	25 (8.0)	66 (8.1)	
Overweight or obese, acceptable waist	17 (5.4)	21 (2.6)	
Overweight or obese, unacceptable waist	193 (61.7)	62 (7.6)	
Maternal education			
10 years or less	173 (56.2)	541 (67.8)	<0.001
>10 years	135 (43.8)	257 (32.2)	
Single parent family			
No	221 (69.9)	662 (80.8)	<0.0001
Yes	95 (30.1)	157 (19.2)	
Hours of TV per day			
<1 or none	32 (10.1)	166 (20.3)	<0.001
1—2	109 (34.4)	265 (32.4)	
>2—3	124 (39.1)	286 (35.0)	
>3	52 (16.4)	101 (12.3)	
	mean (SD)	mean (SD)	p-value[c]
Weight (kg)	70.0 (15.3)	53.4 (8.5)	<0.0001
Height (m)	1.65 (0.07)	1.64 (0.08)	0.04
Body mass index	25.5 (4.9)	19.8 (2.3)	<0.0001
Waist circumference (cm)	86.2 (12.3)	71.7 (6.6)	<0.0001
Arm circumference (cm)	28.2 (4.3)	24.0 (2.8)	<0.0001
Systolic blood pressure	114 (11.0)	110 (9.5)	<0.0001
PWC 170 (watts)[d]	111.7 (26.7)	111.2 (30.1)	0.78
Total energy intake (KJ/day)	9426 (3002)	9722 (3037)	0.14
Fasting serum concentrations			
Glucose (mmolL)	4.9 (0.69)	4.7 (0.4)	<0.0001
Insulin (mU/L)	18.5 (10.9)	9.3 (3.9)	<0.0001
HOMA[e]	4.2 (3.0)	1.9 (0.9)	<0.0001
Total cholesterol (mmol/L)	4.4 (0.8)	4.1 (0.7)	<0.0001
HDL-cholesterol (mmol/L)	1.2 (0.2)	1.5 (0.3)	<0.0001
Calculated LDL-cholesterol (mmol/L)	2.5 (0.7)	2.3 (0.6)	<0.0001
Triglycerides (mmol/L)	1.4 (0.8)	0.8 (0.3)	<0.0001

[a] For some variables the number of subjects does not add up to total due to small numbers of subjects with missing data.
[b] p-Value for chi-squared test.
[c] p-Value for t-test.
[d] Physical Working Capacity 170 (PWC 170) test estimates physical work capacity at a heart rate of 170 beats per minute and is reported in power output (watts). The test is completed on a bicycle ergometer as a measure of aerobic fitness, and a higher value indicates greater aerobic fitness.
[e] Homeostasis Model Assessment.

groups: lean or normal weight with acceptable WC, lean or normal weight with unacceptable WC, overweight or obese with acceptable WC and overweight or obese with unacceptable WC [17]. Lean, normal, overweight and obese status was determined by applying age- and sex-specific cut offs for BMI developed by Cole et al. [18]. An unacceptable WC was defined as being greater than the 80th percentile for age and sex [19]. This has been shown to be a highly sensitive and specific cut-off for identifying high trunk fat mass in Australian children and adolescents when compared to dual-energy X-ray absorptiometry measurements [19].

ANOVA and logistic regression models began with the full model including all potential confounders. Using a manual, backward process, only those that explained a significant amount of variation ($p < 0.05$) were retained in the model, however, dietary pattern quartiles and total energy intake

Please cite this article in press as: Ambrosini GL, et al., Dietary patterns and markers for the metabolic syndrome in Australian adolescents, Nutr Metab Cardiovasc Dis (2009), doi:10.1016/j.numecd.2009.03.024

were retained in all models. Because of possible sex differences related to body composition and pubertal status in this age group, we conducted separate analyses for boys and girls.

Results

There were 2337 adolescents eligible (alive and not withdrawn from the study) for the 14-year follow up. The FFQ was completed by 1631 adolescents. Serum assays were completed for 1377 adolescents (their distributions have been reported previously [14]). Seventy nine (79) subjects who had not fasted were removed from this analysis. In total, 1139 adolescents (546 girls and 593 boys) provided biochemical and dietary pattern data. Of these, 318 (173 girls and 145 boys) fell into the 'high risk metabolic cluster' and 821 fell into the 'low risk metabolic cluster'; their characteristics are shown in Table 2. Adolescents in this analysis did not differ according to sex, BMI, WC, family income or single parent family status, compared with other participants who responded to the 14-year follow up (data not shown).

Nutrient profiles for 'Healthy' and 'Western' dietary patterns are shown in Appendix 1. The main differences between the two patterns pertained to fat, sugar, folate, fibre and sodium intakes. With increasing score for the 'Healthy' pattern, the mean percentage of total energy intake from total fat, saturated fat, monounsaturated fat and refined sugars decreased ($p < 0.001$), while percentage of total energy from protein, total carbohydrate, natural sugars, folate and fibre increased ($p < 0.01$). With increasing 'Western' pattern score, the mean percentage of total energy intake from total fat, saturated fat, monounsaturated fat, refined sugars and sodium increased ($p < 0.0001$) and percentage of energy from total carbohydrate, natural sugars, folate and fibre decreased ($p < 0.0001$). Mean WC and BMI did not vary significantly according to quartiles of either dietary pattern.

Associations between dietary patterns and odds of being in the 'high risk metabolic cluster' are shown in Table 3. For girls, the odds were approximately 2.5 times higher ($p < 0.05$) in the highest quartile of the 'Western' pattern compared with the lowest. An increasing trend in odds was observed across quartiles of the 'Western' pattern both before ($p = 0.01$) and after ($p = 0.02$) adjustment for multiple confounders. No relationship was seen with the 'Western' pattern in boys. Neither boys nor girls showed statistically significant relationships between the 'Healthy' pattern and being in the 'high risk metabolic cluster'.

Girls showed increases in mean WC (p for trend $= 0.03$) and BMI (p for trend $= 0.02$) with increasing quartile for the 'Western' dietary pattern (Table 4). There were no relationships for boys between mean WC, BMI and SBP and either dietary pattern (Table 4).

Mean serum concentrations of lipids, glucose, insulin and HOMA according to dietary pattern quartiles are presented in Table 5. The 'Western' pattern appeared more important for girls, whereas the 'Healthy' pattern appeared more important for boys. A consistent finding was that with increasing 'Healthy' dietary pattern score, mean serum glucose decreased for boys (p for trend $= 0.01$) and girls (p for trend $= 0.04$). Among girls, higher 'Western' pattern scores were associated with an increasing trend in total cholesterol (p for trend $= 0.03$) and a statistically non-significant increasing trend in insulin concentration (p for trend $= 0.06$). Among boys, higher 'Healthy' pattern scores were associated with greater mean HDL-C concentrations (p for trend $= 0.02$) and were inversely associated with HOMA, however, this was not statistically significant (p for trend $= 0.08$). For both boys and girls, the variable combining different levels of BMI and waist circumference was a statistically significant predictor ($p < 0.05$) for all serum concentrations, except total cholesterol. Subjects in the 'overweight or obese with unacceptable WC' category had significantly higher levels of glucose, insulin, HOMA, LDL-C, TG and SBP, and significantly lower levels of HDL-C.

Table 3 Dietary pattern score and odds of 'high risk metabolic cluster' vs. 'low risk metabolic cluster'.

Dietary pattern		Model 1[a]	Model 2[b]
		OR (95% CI)	OR (95% CI)
Girls			
'Healthy'	*n* (*N*)[c]	1.20 (0.95–1.51)[d]	1.27 (0.98–1.65)[d]
Q1	41 (78)	1.00	1.00
Q2	45 (106)	0.86 (0.51–1.45)	0.91 (0.51–1.65)
Q3	36 (102)	0.75 (0.43–1.30)	0.78 (0.42–1.45)
Q4	51 (87)	1.38 (0.80–2.38)	1.51 (0.82–2.78)
p for trend[e]		0.35	0.24
'Western'	*n* (*N*)[c]	1.82 (1.28–2.60)[d]	1.72 (1.17–2.54)[d]
Q1	53 (141)	1.00	1.00
Q2	46 (89)	1.56 (0.94–2.60)	1.84 (1.04–3.26)
Q3	37 (76)	1.64 (0.89–3.04)	2.11 (1.08–4.18)
Q4	37 (67)	2.67 (1.22–5.89)	2.50 (1.05–5.98)
p for trend[e]		0.01	0.02
Boys			
'Healthy'	*n* (*N*)[c]	0.95 (0.76–1.19)[d]	1.04 (0.83–1.31)[d]
Q1	43 (126)	1.00	1.00
Q2	38 (97)	1.16 (0.69–1.94)	1.31 (0.75–2.28)
Q3	34 (108)	0.94 (0.56–1.59)	1.15 (0.66–2.01)
Q4	30 (117)	0.77 (0.44–1.35)	0.98 (0.54–1.79)
p for trend[e]		0.32	0.92
'Western'	*n* (*N*)[c]	1.20 (0.88–1.64)[d]	1.07 (0.77–1.49)[d]
Q1	25 (72)	1.00	1.00
Q2	42 (110)	1.05 (0.57–1.93)	0.94 (0.50–1.78)
Q3	32 (120)	0.73 (0.37–1.45)	0.55 (0.27–1.13)
Q4	46 (146)	1.01 (0.49–2.20)	0.66 (0.30–1.49)
p for trend[e]		0.81	0.17

[a] Logistic regression, adjusting for total energy intake.
[b] Logistic regression, adjusting for total energy intake (MJ), television viewing time (hrs/day), aerobic fitness (estimated power output (watts) at HR = 170 bpm), single parent status and maternal education (>10 or ≤10 years).
[c] *n* = number of subjects in 'high risk metabolic cluster', *N* = number in 'low risk metabolic cluster'.
[d] Dietary pattern score analysed as a continuous variable.
[e] Test for trend where dietary pattern quartile was analysed as a continuous variable.

Please cite this article in press as: Ambrosini GL, et al., Dietary patterns and markers for the metabolic syndrome in Australian adolescents, Nutr Metab Cardiovasc Dis (2009), doi:10.1016/j.numecd.2009.03.024

Table 4 Waist circumference, BMI and SBP by quartile of dietary pattern score[a]

Dietary pattern	WC (cm)	BMI (kg/m^2)	SBP (mmHg)
Girls			
'Healthy'			
Q1	75.9 (73.9, 77.8)	21.8 (21.0, 22.5)	111 (109, 114)
Q2	74.3 (72.6, 76.0)	21.0 (20.3, 21.6)	112 (110, 114)
Q3	74.0 (72.3, 75.7)	21.3 (20.7, 22.0)	112 (110, 114)
Q4	77.0 (75.2, 78.8)	22.5 (21.8, 23.2)	113 (111, 115)
p for trend[b]	0.38	0.09	0.33
'Western'			
Q1	73.5 (71.5, 75.4)	20.9 (20.2, 21.7)	112 (109, 114)
Q2	75.2 (73.4, 77.2)	21.4 (20.7, 22.1)	113 (111, 115)
Q3	74.7 (72.8, 76.7)	21.6 (20.9, 22.4)	112 (110, 114)
Q4	77.7 (75.3, 80.2)	22.6 (21.6, 23.6)	112 (110, 115)
p for trend[b]	0.03	0.02	0.89
Boys			
'Healthy'			
Q1	76.8 (75.1, 78.6)	21.4 (20.7, 22.0)	117 (115, 119)
Q2	77.2 (75.3, 79.1)	21.8 (21.1, 22.5)	117 (115, 119)
Q3	77.2 (75.2, 79.2)	21.5 (20.8, 22.2)	117 (115, 119)
Q4	76.2 (74.2, 78.2)	21.1 (20.4, 21.8)	117 (115, 119)
p for trend[b]	0.68	0.58	0.80
'Western'			
Q1	75.5 (73.1, 77.9)	21.2 (20.3, 22.1)	118 (116, 121)
Q2	78.0 (76.1, 80.0)	21.9 (21.2, 22.6)	117 (115, 119)
Q3	76.1 (74.2, 78.1)	21.2 (20.6, 22.0)	115 (113, 117)
Q4	77.7 (75.6, 79.9)	21.4 (20.6, 22.2)	118 (115, 120)
p for trend[b]	0.36	0.93	0.58

[a] Mean values (95% CI) estimated using ANOVA adjusting for: total energy intake (MJ), aerobic fitness (estimated power output (watts) at HR = 170 bpm), single parent status, maternal education (>10 or ≤10 years), body mass index and waist circumference combined (SBP only).
[b] Obtained by analysing the dietary pattern quartile as a continuous variable.

Discussion

This study indicated that a 'Western' dietary pattern was associated with a greater risk for metabolic syndrome and some of its components, including serum total cholesterol, higher BMI and higher WC, among female adolescents. A 'Healthy' dietary pattern was associated with lower levels of serum glucose in both male and female adolescents.

The inverse association between a 'Healthy' dietary pattern and fasting serum glucose concentration is a new and important finding in this age group. Long-term hyperglycemia, even in individuals who do not develop diabetes mellitus, is directly related to the risk of developing microvascular and macrovascular complications such as CVD [20]. Vegetables, fruits, legumes and whole grains were dominant in the 'Healthy' dietary pattern. These foods are important sources of nutrients that may affect glucose metabolism, e.g., soluble fibre [21] and potentially influence development of the metabolic syndrome and diabetes mellitus. A dietary pattern similar to our 'Healthy' pattern has been associated with decreased risks for type 2 diabetes mellitus in adults [22,23].

An explanation of why these two independent dietary patterns show different relationships in boys and girls is beyond the scope of this paper. However, possibilities include hormonal effects on the metabolic syndrome related to the fact that most of the girls in this study were post-pubertal [24], differences between boys and girls in lipid accumulation [25], or unrecognised lifestyle confounders.

To date, three other studies have examined dietary patterns using factor analysis and CVD risk factors or the metabolic syndrome in adolescents. An Australian study reported three major dietary patterns using national nutrition data from 764 adolescents: (1) fruit, fish, cereal and salad, (2) high fat and sugar, and (3) vegetables [5]. Adolescents in the upper tertile for the first pattern had significantly lower diastolic blood pressure than those in the lowest tertile, after adjustment for age, sex and physical activity. BMI, WC and SBP were not related to these dietary patterns [5]. In a study of 4811 Iranian adolescents, hypertriglyceridemic waist (HW) phenotype, i.e., abnormal triglyceride concentration coupled with high WC (>75th percentile), was used to define the presence of metabolic syndrome. Two dietary patterns were identified: one high in salty/fatty snacks, sweets, fast foods and animal protein, the other high in dairy, vegetables and fruit. The 'unhealthy' dietary pattern was positively associated with the HW phenotype, while there was a weak inverse association between HW phenotype and the dairy, vegetable and fruit pattern [6]. These findings were generally repeated in subsequent analyses of food group intakes and the metabolic syndrome defined using a modified Adult Treatment Panel III in the same study population [7]. However, BMI and physical activity were not considered in either analysis. The Bogalusa Heart Study (US) included 1275 adolescents and young adults aged 12–24 years [4]. Twelve dietary patterns were observed in food intakes measured between 1981 and 1983. A 'fruit and vegetable' pattern was associated with higher levels of HDL-cholesterol and lower levels of triglycerides and LDL-cholesterol. 'Sugary foods' and 'fats and pasta' patterns were positively associated with higher LDL-cholesterol levels [4].

These studies generally support our findings and those from adult populations [26–28]. However, comparisons between studies on adolescents are limited due to the use of different definitions for the metabolic syndrome and lack of control for confounding factors. Furthermore, none of the reviewed studies reported separate results for boys and girls whereas, our results suggest sex differences in the relationships between dietary patterns and the metabolic syndrome in adolescents.

This study benefits from being a population-based study in which a range of socio-demographic information has been collected, thus allowing a thorough investigation of possible confounders. Other studies show that dietary

Please cite this article in press as: Ambrosini GL, et al., Dietary patterns and markers for the metabolic syndrome in Australian adolescents, Nutr Metab Cardiovasc Dis (2009), doi:10.1016/j.numecd.2009.03.024

Table 5 Serum concentrations by quartile of dietary pattern score[a]

Dietary pattern	Glucose (mmol/L)	Insulin (mU/L)	HOMA	Cholesterol (mmol/L)	HDL-cholesterol (mmol/L)	LDL-cholesterol (mmol/L)	Triglycerides (mmol/L)
Girls							
'Healthy'							
Q1	4.86	12.3	2.6	4.35	1.35	2.53	1.07
	(4.75, 4.97)	(11.1, 13.6)	(2.4, 2.9)	(4.21, 4.50)	(1.28, 1.42)	(2.38, 2.67)	(0.99, 1.16)
Q2	4.86	11.8	2.5	4.28	1.44	2.42	1.00
	(4.76, 4.96)	(10.7, 13.0)	(2.3, 2.8)	(4.15, 4.41)	(1.37, 1.50)	(2.28, 2.55)	(0.93, 1.08)
Q3	4.81	11.5	2.5	4.30	1.43	2.46	0.96
	(4.71, 4.91)	(10.4, 12.7)	(2.2, 2.7)	(4.18, 4.43)	(1.37, 1.50)	(2.32, 2.60)	(0.88, 1.03)
Q4	4.75	12.6	2.7	4.36	1.36	2.53	1.02
	(4.64, 4.85)	(11.4, 14.0)	(2.4, 3.0)	(4.23, 4.48)	(1.29, 1.42)	(2.39, 2.67)	(0.94, 1.11)
p for trend[b]	0.04	0.67	0.97	0.90	0.97	0.92	0.27
'Western'							
Q1	4.87	11.3	2.4	4.21	1.38	2.43	0.98
	(4.76, 4.99)	(10.2, 12.6)	(2.2, 2.7)	(4.07, 4.36)	(1.31, 1.45)	(2.28, 2.58)	(0.90, 1.06)
Q2	4.82	11.6	2.5	4.20	1.35	2.39	1.02
	(4.71, 4.93)	(10.5, 12.8)	(2.2, 2.8)	(4.06, 4.33)	(1.28, 1.42)	(2.25, 2.54)	(0.95, 1.11)
Q3	4.80	12.0	2.6	4.49	1.45	2.60	0.99
	(4.69, 4.92)	(10.8, 13.4)	(2.3, 2.9)	(4.35, 4.63)	(1.38, 1.52)	(2.45, 2.74)	(0.91, 1.07)
Q4	4.77	13.3	2.8	4.39	1.39	2.51	1.06
	(4.64, 4.91)	(11.8, 15.1)	(2.5, 3.2)	(4.21, 4.57)	(1.30, 1.47)	(2.33, 2.69)	(0.95, 1.17)
p for trend[b]	0.17	0.06	0.12	0.03	0.48	0.19	0.36
Boys							
'Healthy'							
Q1	4.94	11.0	2.5	4.09	1.25	2.39	0.98
	(4.84, 5.03)	(10.0, 12.2)	(2.2, 2.7)	(3.98, 4.20)	(1.19, 1.31)	(2.27, 2.52)	(0.91, 1.06)
Q2	4.97	11.2	2.4	4.02	1.30	2.32	0.91
	(4.85, 5.08)	(10.0, 12.6)	(2.1, 2.7)	(3.90, 4.14)	(1.23, 1.36)	(2.17, 2.46)	(0.83, 1.00)
Q3	4.92	10.9	2.3	3.98	1.30	2.26	0.95
	(4.82, 5.02)	(9.8, 12.1)	(2.1, 2.6)	(3.86, 4.11)	(1.24, 1.37)	(2.12, 2.39)	(0.87, 1.03)
Q4	4.77	10.5	2.2	4.19	1.35	2.43	0.92
	(4.66, 4.88)	(9.4, 11.8)	(1.9, 2.4)	(4.07, 4.32)	(1.28, 1.41)	(2.29, 2.57)	(0.85, 1.01)
p for trend[b]	0.01	0.43	0.08	0.45	0.02	0.99	0.38
'Western'							
Q1	4.97	11.5	2.4	3.99	1.22	2.34	0.98
	(4.84, 5.10)	(10.0, 13.2)	(2.1, 2.8)	(3.83, 4.15)	(1.14, 1.29)	(2.17, 2.51)	(0.89, 1.09)
Q2	4.87	11.0	2.3	4.02	1.33	2.28	0.92
	(4.77, 4.98)	(9.8, 12.2)	(2.1, 2.6)	(3.90, 4.13)	(1.27, 1.39)	(2.15, 2.41)	(0.85, 1.00)
Q3	4.87	10.4	2.2	4.18	1.36	2.41	0.93
	(4.76, 4.97)	(9.3, 11.5)	(2.0, 2.5)	(4.05, 4.30)	(1.29, 1.42)	(2.27, 2.54)	(0.85, 1.01)
Q4	4.89	10.9	2.4	4.10	1.30	2.36	0.93
	(4.77, 5.01)	(9.7, 12.3)	(2.1, 2.7)	(3.96, 4.24)	(1.23, 1.37)	(2.21, 2.51)	(0.85, 1.03)
p for trend[b]	0.46	0.47	0.79	0.31	0.22	0.67	0.70

[a] Mean values (95% CI) estimated using ANOVA adjusting for: total energy intake (MJ), aerobic fitness (estimated power output (watts) at HR = 170 bpm), single parent status, maternal education (>10 or ≤10 years), television viewing time (hours/day), body mass index and waist circumference (combined).

[b] Obtained by analysing dietary pattern quartile as a continuous variable.

patterns based on factor analysis may be repeatable and valid [29,30]. We have avoided the use of arbitrary definitions for the metabolic syndrome, which allowed a larger number of at-risk children to be identified, and we have examined individual components of the metabolic syndrome. As this study is cross-sectional in design, causal associations cannot be definitely established. However, we will examine longitudinal relationships using follow up data currently being collected from 17-year-old adolescents participating in the Raine Study.

In this study of Australian adolescents, dietary patterns were associated with individual risk factor components of the metabolic syndrome and their clustering. Longitudinal studies are required to confirm the role of dietary patterns

Please cite this article in press as: Ambrosini GL, et al., Dietary patterns and markers for the metabolic syndrome in Australian adolescents, Nutr Metab Cardiovasc Dis (2009), doi:10.1016/j.numecd.2009.03.024

in the development of these risk factors. This information will be invaluable for the development of strategies to reduce risk factor prevalence among adolescents and decrease lifelong risk of chronic disease.

Conflict of interest

The authors have no conflict of interests.

Acknowledgements

We are extremely grateful to all families who took part in this study and the entire Raine Study team, including data collectors, cohort managers, data managers, clerical staff, research scientists and volunteers. GLA conducted the statistical analysis, interpreted the data and prepared the manuscript for publication. RCH was responsible for the cluster analysis of metabolic syndrome and contributed to manuscript preparation. TAM contributed to data collection and analysis, interpretation of results and manuscript preparation. NdK, BH and TO'S contributed to the interpretation of the results and manuscript preparation. LJB contributed to study design, data collection and analysis, interpretation of results and manuscript preparation. WHO coordinated and was responsible for the collection of nutrition data and contributed to manuscript preparation.

Appendix 1

Mean BMI, WC and nutrient intakes according to quartile of dietary pattern score.[a]

Quartile of 'Healthy' pattern score					
	1	2	3	4	*p*-Value[b]
BMI	21.0 ± 1.0	20.8 ± 1.0	20.9 ± 1.0	21.3 ± 1.0	0.38
WC (cm)	75.3 ± 1.0	74.7 ± 1.0	74.7 ± 1.0	75.5 ± 1.0	0.81
Total energy intake (MJ)	9.1 ± 0.2	8.8 ± 0.2	9.7 ± 0.2	11.0 ± 0.2	<0.0001
% Energy from protein	15.9 ± 0.1	16.7 ± 0.1	17.6 ± 0.1	18.2 ± 0.1	<0.0001
% Energy from total fat	36.9 ± 0.3	35.0 ± 0.3	34.5 ± 0.3	32.6 ± 0.3	<0.0001
% Energy from saturated fat	16.8 ± 0.2	15.4 ± 0.2	15.1 ± 0.2	13.4 ± 0.2	<0.0001
% Energy from polyunsaturated fat	5.0 ± 0.1	5.3 ± 0.1	5.3 ± 0.1	5.7 ± 0.1	<0.001
% Energy from monounsaturated fat	12.9 ± 0.1	12.2 ± 0.1	11.9 ± 0.1	11.1 ± 0.1	<0.0001
% Energy from total carbohydrate	45.0 ± 0.3	46.0 ± 0.3	45.6 ± 0.3	46.9 ± 0.3	<0.001
% Energy from starch	20.3 ± 0.2	19.7 ± 0.2	19.2 ± 0.2	18.9 ± 0.2	<0.0001
% Energy from refined sugars	14.3 ± 0.3	12.9 ± 0.3	11.2 ± 0.3	9.8 ± 0.3	<0.0001
% Energy from natural sugars	10.1 ± 0.3	13.0 ± 0.3	14.8 ± 0.3	17.8 ± 0.3	<0.0001
Fibre (g)	17.8 ± 0.3	22.2 ± 0.3	25.1 ± 0.3	31.0 ± 0.3	<0.0001
Total folate (ug)	179 ± 3	228 ± 3	261 ± 3	325 ± 3	<0.0001
Sodium (mg)	3328 ± 34	3290 ± 34	3357 ± 34	3316 ± 35	0.87
Quartile of 'Western' pattern score					
	1	2	3	4	*p*-Value[b]
BMI	20.6 ± 1.0	21.0 ± 1.0	21.0 ± 1.0	21.4 ± 1.0	0.09
WC (cm)	73.7 ± 1.0	75.5 ± 1.0	74.8 ± 1.0	76.2 ± 1.0	0.07
Total energy intake (MJ)	7.1 ± 0.1	8.7 ± 0.1	10.0 ± 0.1	12.8 ± 0.1	<0.0001
% Energy from protein	17.2 ± 0.2	17.2 ± 0.2	17.1 ± 0.2	16.9 ± 0.2	0.29
% Energy from total fat	32.2 ± 0.3	34.2 ± 0.3	35.4 ± 0.3	37.1 ± 0.4	<0.0001

(*continued on next page*)

(*continued*)

Quartile of 'Western' pattern score					
	1	2	3	4	*p*-Value[b]
% Energy from saturated fat	14.0 ± 0.2	14.8 ± 0.2	15.5 ± 0.2	16.3 ± 0.2	<0.0001
% Energy from polyunsaturated fat	5.4 ± 0.1	5.5 ± 0.1	5.3 ± 0.1	5.1 ± 0.1	0.11
% Energy from monounsaturated fat	10.7 ± 0.1	11.8 ± 0.1	12.4 ± 0.1	13.2 ± 0.1	<0.0001
% Energy from total carbohydrate	48.2 ± 0.3	46.3 ± 0.3	45.2 ± 0.3	43.9 ± 0.4	<0.0001
% Energy from starch	19.1 ± 0.2	19.3 ± 0.2	19.6 ± 0.2	20.1 ± 0.2	0.01
% Energy from refined sugars	9.6 ± 0.3	11.5 ± 0.3	12.5 ± 0.3	14.7 ± 0.3	<0.0001
% Energy from natural sugars	18.9 ± 0.3	15.1 ± 0.3	12.8 ± 0.3	8.9 ± 0.3	<0.0001
Fibre (g)	27.6 ± 0.4	25.2 ± 0.4	23.0 ± 0.4	20.2 ± 0.5	<0.0001
Total folate (ug)	292 ± 5	265 ± 4	240 ± 4	196 ± 5	<0.0001
Sodium (mg)	3074 ± 37	3200 ± 33	3348 ± 34	3665 ± 40	<0.0001

[a] Mean ± SE adjusted for total energy intake (ANOVA).
[b] *p*-Value for trend (ANOVA F statistic).

References

[1] Daniels SR, Greer FR, the American Academy of Pediatrics Committee on Nutrition. Lipid screening and cardiovascular health in childhood. Pediatrics 2008;122:198–208.
[2] De Ferranti SD, Osganian SK. Epidemiology of paediatric metabolic syndrome and type 2 diabetes mellitus. Diab Vasc Dis Res 2007;4:283–4.
[3] Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet 2005;365:1415–28.
[4] Nicklas TA, Webber LS, Thompson B, Berenson GS. A multivariate model for assessing eating patterns and their relationship to cardiovascular risk factors: the Bogalusa Heart Study. Am J Clin Nutr 1989;49:1320–7.
[5] McNaughton SA, Ball K, Mishra GD, Crawford DA. Dietary patterns of adolescents and risk of obesity and hypertension. J Nutr 2008;138:364–70.
[6] Alavian S-M, Motlagh ME, Ardalan G, Motaghian M, Davarpanah AH, Kelishadi R. Hypertriglyceridemic waist phenotype and associated lifestyle factors in a national population of youths: CASPIAN Study. J Trop Pediatr 2008;54:169–77.
[7] Kelishadi R, Gouya MM, Adeli K, Ardalan G, Gheiratmand R, Majdzadeh R, et al. Factors associated with the metabolic syndrome in a national sample of youths: CASPIAN Study. Nutr Metab Cardiovasc Dis 2008;18:461–70. doi:10.1016/j.numecd.2007.02.014.
[8] Pan Y, Pratt CA. Metabolic syndrome and its association with diet and physical activity in US adolescents. J Am Diet Assoc 2008;108:276–86.
[9] Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. Curr Opin Lipidol 2002;13:3–9.
[10] Newnham JP, Evans SF, Michael CA, Stanley JF, Landau LI. Effects of frequent ultrasound during pregnancy – a randomised controlled trial. Lancet 1993;342:887–91.
[11] Ambrosini GL, Oddy WH, Robinson M, O'Sullivan TA, Hands BP, de Klerk NH, et al. Adolescent dietary patterns are associated with lifestyle and family psycho-social factors. Public Health Nutr January 23, 2009;. doi:10.1017/S1368980008004618 (published online ahead of print).
[12] SAS Institute Incorporated. SAS for Windows. v9.1.3. Cary, NC, USA: SAS Institute Incorporated; 2002–2003.
[13] Atabek ME, Pirgon O. Assessment of insulin sensitivity from measurements in fasting state and during an oral glucose tolerance test in obese children. J Pediatr Endocrinol Metab 2007;20:187–95.
[14] Huang RC, Mori T, Burke V, Newnham J, Stanley F, Landau L, et al. Synergy between adiposity, insulin resistance, metabolic risk factors and inflammation in adolescents. Diabetes Care 2009;32:695–701.
[15] Hands B, Larkin D, Parker H, Straker L, Perry M. The relationship among physical activity, motor competence and health-related fitness in 14-year-old adolescents. Scand J Med Sci Sports August 5, 2008;. doi:10.1111/j.1600-0838.2008.00847.x (published online ahead of print).
[16] Janssen I, Katzmarzyk PT, Srinivasan SR, Chen W, Malina RM, Bouchard C, et al. Combined influence of body mass index and waist circumference on coronary artery disease risk factors among children and adolescents. Pediatrics 2005;115:1623–30.
[17] Garnett SP, Baur LA, Srinivasan S, Lee JW, Cowell CT. Body mass index and waist circumference in midchildhood and adverse cardiovascular disease risk clustering in adolescence. Am J Clin Nutr 2007;86:549–55.
[18] 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.
[19] Taylor RW, Jones IE, Williams SM, Goulding A. Evaluation of waist circumference, waist-to-hip ratio, and the conicity index as screening tools for high trunk fat mass, as measured by dual-energy X-ray absorptiometry, in children aged 3–19 years. Am J Clin Nutr 2000;72:490–5.
[20] Porte D. Clinical importance of insulin secretion and its interaction with insulin resistance in the treatment of type 2 diabetes mellitus and its complications. Diabetes Metab Res Rev 2001;17:181–8.

Please cite this article in press as: Ambrosini GL, et al., Dietary patterns and markers for the metabolic syndrome in Australian adolescents, Nutr Metab Cardiovasc Dis (2009), doi:10.1016/j.numecd.2009.03.024

[21] Bjorck I, Granfeldt Y, Liljeberg H, Tovar J, Asp N. Food properties affecting the digestion and absorption of carbohydrates. Am J Clin Nutr 1994;59:S699—705.
[22] Nettleton JA, Steffen LM, Ni H, Liu K, Jacobs Jr DR. Dietary patterns and risk of incident type 2 diabetes in the Multi-Ethnic Study of Atherosclerosis (MESA). Diabetes Care 2008;31:1777—82.
[23] Brunner EJ, Mosdol A, Witte DR, Martikainen P, Stafford M, Shipley MJ, et al. Dietary patterns and 15-y risks of major coronary events, diabetes, and mortality. Am J Clin Nutr 2008;87:1414—21.
[24] Tfayli H, Arslanian S. Menstrual health and the metabolic syndrome in adolescents. Ann N Y Acad Sci 2008;1135:85—94.
[25] Regitz-Zagrosek V, Lehmkuhl E, Weickert MO. Gender differences in the metabolic syndrome and their role for cardiovascular disease. Clin Res Cardiol 2006;95:136—47.
[26] Panagiotakos DB, Pitsavos C, Skoumas Y, Stefanadis C. The association between food patterns and the metabolic syndrome using principal components analysis: the ATTICA Study. J Am Diet Assoc 2007;107:979—87.
[27] Esmaillzadeh A, Kimiagar M, Mehrabi Y, Azadbakht L, Hu FB, Willett WC. Dietary patterns, insulin resistance, and prevalence of the metabolic syndrome in women. Am J Clin Nutr 2007;85:910—8.
[28] Fung T, Rimm E, Spiegelman D, Rifai N, Tofler G, Willett W, et al. Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk. Am J Clin Nutr 2001;73:61—7.
[29] Hu FB, Rimm E, Smith-Warner SA, Feskanich D, Stampfer MJ, Ascherio A, et al. Reproducibility and validity of dietary patterns assessed with a food-frequency questionnaire. Am J Clin Nutr 1999;69:243—9.
[30] Khani BR, Ye W, Terry P, Wolk A. Reproducibility and validity of major dietary patterns among Swedish women assessed with a food-frequency questionnaire. J Nutr 2004;134:1541—5.

Please cite this article in press as: Ambrosini GL, et al., Dietary patterns and markers for the metabolic syndrome in Australian adolescents, Nutr Metab Cardiovasc Dis (2009), doi:10.1016/j.numecd.2009.03.024