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Adolescent Dietary Intakes Predict Cardiometabolic Risk Clustering

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Abstract

Purpose: To prospectively examine the relation between adolescent dietary intake and cardiometabolic risk (CMR) clustering at the end of adolescence.

Methods: Data from the NHLBI Growth and Health Study on 1369 girls enrolled at ages 9–10 in 1987–1988 and followed for 10 years were used to estimate the relative risk of having multiple (2 or 3) risk factors in late adolescence associated with usual food intake patterns from 9–17 years of age. Mean food intakes were derived from multiple 3-day diet records and CMR factors included larger waist circumference, insulin resistance, low high-density lipoprotein-cholesterol, high low-density lipoprotein-cholesterol, high triglycerides, and elevated systolic or diastolic blood pressures.

Results: Of 1,369 subjects, 18.4% girls had 3–6 prevalent risk factors by the end of adolescence and 35.0% had at least two. Higher intakes of fruit and non-starchy vegetables, dairy, and grains were independently associated with having fewer risk factors as were eating patterns characterized by higher combined intakes of these food groups. After adjusting for age, race, socio-economic status, height, physical activity, and television watching, girls with high intakes of dairy and fruits and non-starchy vegetables (vs. those with lower intakes of both) were nearly 50% less likely to have three or more CMR factors in late adolescence; girls with higher intakes of grains plus fruits and non-starchy vegetables were nearly 60% less likely.

Conclusion: These results suggest that healthy food consumption patterns during adolescence may prevent accumulation of cardiometabolic risk.

Keywords

Nutrition; central obesity; blood pressure; lipids; insulin resistance; youth

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Introduction

While the metabolic syndrome as a diagnostic construct has come under question, clustering of cardiovascular risk factors continues to serve as the basis for adult cardiovascular risk assessment. The roots of cardiovascular disease are established early and cardiovascular risk clustering persists from early childhood through adolescence and adulthood [1;2]. The presence of multiple cardiovascular risk factors in younger populations is associated with concurrent vascular dysfunction and therefore may serve as an important tool for identifying adolescents at higher cardiovascular risk [3].

The role of diet patterns in the development of cardiometabolic risk (CMR) in adults has been examined in a number of studies [4–6]. These studies have explored the impact of specific food groups as well as related patterns of intake of foods, macronutrients and other nutrients [7]. In contrast, the role of food-based consumption in the development of CMR clustering amongst children has been infrequently studied.[8] Previous analyses of data from the National Growth and Health Study (NGHS) have examined the effects of dietary patterns on separate adolescent health outcomes including weight, blood pressure, and lipid levels but the effect of dietary intakes on the clustering of CMR factors is unknown [9–11].

The goal of the current analysis was to examine the effects of usual food group intakes in earlier adolescence on the clustering of CMR factors in later adolescence.

Methods

The analyses were conducted using data from the National Heart, Lung and Blood Institute's Growth and Health Study (NGHS), a longitudinal study of the development of obesity and other cardiovascular-related outcomes in adolescent girls. Beginning in 1987–1988, the NGHS enrolled 2,379 subjects at 9–10 years of age from three representative urban and suburban clinical sites. Approximately equal numbers of blacks and whites were enrolled; subjects were followed annually until ages 19–20. Details of the study design have been previously published [12]. The current analyses were approved by the Institutional Review Board of Boston University and include 1,369 girls with complete data on diet and six CMR factors described below.

Dietary Intake

Diet was assessed using 3-day diet records during eight of the 10 study years. Data were entered into the Nutrition Data System (NDS) nutrient calculation program (of the Nutrition Coordinating Center of the University of Minnesota) [13] using version 19 food tables (the version most appropriate to the period of data collection). Each subject's food intake was calculated in Food Pyramid servings by the investigators at Boston University through linking NDS food codes with those from the USDA's "MyPyramid Equivalents Database, Version 2.0 for USDA Survey Food Codes"[14]. The final Food Pyramid data set contains each subject's intake in the five major food groups (i.e., fruit, vegetables, dairy, meat/other proteins, grains) and all subgroups.

Measurement of CMR Factors

The following factors related to CMR were considered: waist circumference, blood pressure, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides (TG), and insulin resistance (IR). Mean values for each risk factor at the final exam served as primary outcomes. Waist circumference was measured in duplicate at the narrowest part of the torso against the skin. Blood pressure was measured by standardized protocol with a mercury sphygmomanometer. Fasting lipids and glucose were analyzed at a Johns Hopkins laboratory (a participant in CDC/NHLBI Lipid Standardization Program). LDL cholesterol was calculated using a modified Friedewald equation: $[LDL] = [(TC)] - [HDL] - [TG]/6.5$ [15]. IR was estimated using the homeostasis model assessment (HOMA-IR) [16].

Potential Confounders

Potential confounders included age, race, socio-economic status (SES), height, physical activity, and television/video viewing time. Girls were self-described as “black” or “white”. Annual household income and education were used to classify SES as low, moderate or high. Physical activity was assessed during eight years using a validated Health Activity Questionnaire (HAQ) [17]. Usual time spent watching television/video was assessed annually by questionnaire. There was no confounding by energy, macronutrient intakes, or family medical history variables so these variables were dropped from the final models.

Statistical Analysis

Usual dietary intake was estimated as a mean of all-available diet records from ages 9 to 17 years. Up to eight sets of 3-day diet records were collected between 9 and 17 years of age. On average, the girls completed 18 days of diet records. The mean intake was used to derive a more stable estimate of usual intake patterns and to minimize the effects of measurement error, seasonal variability in intake, and changes in consumption linked with periods of growth. The clustering of six CMR factors in late adolescence (ages 18–20) was examined in relation to selected diet patterns based on usual intake over the course of the study. Each girl was classified as being at elevated risk (or not) for each of six factors as follows: waist circumference (≥ 88 cm), systolic and/or diastolic blood pressure (≥ 90 th percentile for age, sex and height), LDL (≥ 110 mg/dL), HDL (<50 mg/dL), serum TG (≥ 110 mg/dl), and HOMA-IR (≥ 4). Cutoff values were selected from several sources. “At risk” waist circumference and TG were derived from the National Cholesterol Education Program (NCEP) guidelines [18]. Low HDL followed the International Diabetes Federation guidelines for late adolescent girls [19]. “At risk” blood pressure was defined using the National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescence [20]. The elevated LDL criterion was based on the NCEP classification of “borderline high” levels for children and adolescents [21]. While there is no single accepted value for defining insulin resistance using HOMA-IR among adolescents, we chose a cutoff value of 4.0 based on previously-published studies as well as the data distribution in the current study [22]. Subjects received a score of one for each risk factor in which their value fell in the at-risk range or a zero otherwise. The sum of the six risk factors served as an overall risk clustering score.

Multiple logistic regression analysis was used to estimate the relative risk (RR) of having multiple (2 or 3) risk factors in later adolescence (18–20 years of age) associated with selected diet patterns including dairy, fruits and non-starchy vegetables (FnsV), and grains. Total dairy is presented in the results since no differences were observed in effects of low-fat vs. fullfat dairy. Patterns of intake were explored for two-way combinations of the above food groups, with intake of each food dichotomized (e.g., <2 vs. ≥2 cup-equivalents for dairy and <1.5 vs. ≥1.5 cup-equivalents per day for FnsV). In this way, four mutually-exclusive categories were derived (e.g., (a) low intakes of both dairy and FnsV, (b) low intake of dairy and high intake of FnsV, (c) high intake of dairy but low intake of FnsV, and (d) high intakes of both dairy and FnsV). Other diet patterns combined dairy and grain consumption as well as FnsV and grains. Since whole grain consumption was very low in this cohort, total grains were analyzed instead. Cutpoints were based on distribution of levels of intake to optimize analytic power.

Results

Table 1 provides descriptive data on subjects according to three food groups—dairy, FnsV, and grains. Girls reporting the highest intakes of dairy or FnsV were more likely to be white. They also had a lower mean BMI, were more active and watched less television than girls with lower intakes in either of these food groups. Somewhat similar, but weaker, patterns were found for higher intakes of grains. Dairy intakes were directly associated with energy-adjusted protein intakes and inversely associated with the percent of calories from fat.

The first row in Table 2 shows that of the 1,369 girls at the end of follow up, 30.1% had no CMR factors while 34.9% had only one, 16.6% had two and 18.4% had three or more. While the proportion of girls with clustering of risk factors was very similar in whites and blacks, the types of risk factors observed were different (data not shown). Increased waist size, elevated BP and insulin resistance were twice as common in blacks as in whites. In contrast, abnormal lipid levels were more common in whites than blacks; in fact, 27.5% of white girls had at least two lipid related abnormalities compared with 18.2% of black girls. In particular, white girls were more than twice as likely to have elevated triglyceride levels. Table 2 also shows the degree of CMR clustering according to dietary intakes in three food groups. Unfortunately, the distribution of FnsV intake among these girls did not allow us to evaluate particularly high intakes of these foods. Girls consuming 1.5 cups of FnsV per day had fewer risk factors than girls consuming less. In contrast, 23.3% of girls consuming less than one cup per day had 3 or more prevalent risk factors by late adolescence. There were similar but weaker trends for intakes of dairy and grains.

Table 3 shows CMR clustering associated with three empirically-defined dietary patterns consisting of the following: (a) combined dairy and FnsV intakes, (b) combined dairy and grain intakes, and (c) combined FnsV and grain intakes. Higher combined intakes in each of these three healthy diet patterns were associated with less clustering of CMR. For example, only 9.3% girls with high FnsV and high grain intakes had three or more CMR factors compared with 21.1% girls with low intakes of both in early adolescence. Similarly, only 10.2% girls with higher intakes of both dairy and FnsV had three or more risk factors compared with 19.5% of those with low intakes. Diet patterns characterized by high intakes

of FnsV alone or high dairy alone were somewhat less beneficial than the combined diet patterns.

Table 4 shows the effect of diet patterns on the risk of CMR clustering after adjusting for age, race, SES, height, physical activity, and hours spent watching television/video. Girls with any of the three healthy eating patterns shown here were about 40% less likely to have two or more CMR factors than girls with lower intakes in these food groups. They were even less likely to have three or more clustered risk factors. For example, girls with high intakes of dairy and FnsV (vs. those with lower intakes) were nearly 48% less likely (RR=0.52; 95% CI: 0.30, 0.89) to have three or more CMR factors.

Discussion

These results suggest that earlier adolescent diet patterns are important predictors of subsequent clustering of cardiometabolic risk. Girls with higher usual intakes of total dairy, fruit and nonstarchy vegetables and grains had less accumulated risk by the time of late adolescence. Specifically, higher combined intakes of dairy and FnsV as well as higher intakes of FnsV and grains were associated with a 40–60% lower risk of CMR clustering.

Food-based dietary determinants of cardiometabolic risk clustering in younger populations have not been widely studied. Published data from the IDEFICS study of over 16,000 children ages 2–9 from eight European countries examined the association between individual food components and the clustering of cardiovascular risk.[8] Higher intakes of breakfast cereals as well as chocolate, nut-based spreads, and honey were positively associated with lower cardiovascular risk clustering, particularly among older subjects. Recently published cross-sectional data from another large European study of adolescents suggested that higher intakes of dairy were linked with less clustering of cardiovascular risk, particularly among female subjects.[23]

Few studies have used longitudinal data to examine the association between food intake patterns during the adolescent years and the clustering of CMR factors in late adolescence. Several studies have compared the association between dietary patterns derived from factor analyses, typically a “healthy” diet vs. a “less healthy” diet, and selected cardiovascular risk factors. Cross-sectional data from one Australian adolescent cohort found that a “Western” diet pattern was associated with a greater likelihood of metabolic risk clustering [24]. Since that study did not provide data on individual food groups, it is not possible to compare results directly. Analysis of 1999–2002 NHANES cross-sectional data suggests a lower prevalence of metabolic syndrome in adolescents with higher diet quality as measured by the Healthy Eating Index [25].

Several additional studies have examined the effects of selected food groups on risk of metabolic syndrome in adolescents and young adults. Results from the cross-sectional CASPIAN Study of school-aged children showed that increased consumption of dairy as well as fruits and vegetables was associated with a decreased risk of metabolic syndrome [26]. Another cross-sectional study found that markers of inflammation and oxidative stress were also inversely associated with fruit and vegetable intake among adolescents [27]. In the

Bogalusa Heart Study, higher fruit and vegetable consumption was also linked with a lower prevalence of metabolic syndrome in adolescents and young adults (ages 19–38 years) [28]. In the CARDIA study of young adults, diet patterns that included higher dairy intakes led to a lower risk of insulin resistance syndrome [29].

A number of potential mechanisms could explain these results. First, fruits and vegetables are key sources of nutrients, fiber, and other phytochemicals and thus have the potential to lower oxidative stress, decrease platelet aggregation, and reduce cholesterol via lowered hepatic cholesterol synthesis and/or increased excretion of fecal fatty acids. Magnesium in particular, which is present in many fruits and vegetables as well as dairy, is thought to act as a natural calcium channel blocking agent and to improve vascular resistance [30], thereby benefiting blood pressure. Fruits and vegetables are also nutrient-rich, low energy-dense foods that may lead to reductions in overall energy intake, providing benefits to the regulation of body weight [31]. Fruits and vegetables are also a key source of fiber which is believed to benefit lipid profiles, particularly in terms of lowering LDL [32].

While dairy has been linked with lower levels of body fat in some studies of children and adolescents [33], others have found no effect. Two recent reviews found that while there is insufficient evidence to conclude that dairy has a beneficial effect on body weight/fat, there is substantial evidence that it has no adverse effect [34;35]. The protein content of dairy is one of several mechanisms by which it may convey benefits to body fat or composition [36]. In addition to its magnesium content, dairy is also an important source of both calcium and potassium, minerals that have been associated with lower blood pressures through the promotion of vasodilation and reduction of vascular resistance. Since a low potassium-to-sodium ratio decreases synthesis of nitric oxide and increases renal sodium retention, it may be responsible for elevations in blood pressure. Consequently, higher potassium intake may reduce salt sensitivity and lower blood pressure by reducing peripheral vascular resistance. Dairy-derived tripeptides are another potential pathway to improvements in blood pressure. A meta-analysis of randomized controlled trials demonstrated important hypotensive effects of such tripeptides [37], which lead to inhibition of the renin-angiotensin system, thereby lowering blood pressure.

Numerous studies address the metabolic health benefits of whole grains in adults. One such study amongst adolescents found beneficial effects of whole grain consumption on both body mass and insulin sensitivity, particularly among the heaviest subjects [38]. Few studies have examined the effects of total grains on cardiometabolic risk in children or adolescents. Data from the third National Health and Nutrition Examination Survey showed that total grain consumption was inversely associated with anthropometric measures of central adiposity [39]. While several studies of children and adolescents have found beneficial effects of overall grain consumption, most studies have attributed those benefits to whole grains [40]. A recently published review concluded that the vast majority of prospective studies of refined grain consumption (largely amongst adults) found either no effect or a protective effect of higher levels of intake on CVD risk [41]. Potential beneficial effects of refined grains may stem from its nutrient composition (e.g., B-vitamins) or from an association with other dietary habits (e.g., lower fat intakes).

The importance of cardiovascular risk clustering is well established and risk scores have been used to predict the development of coronary heart disease amongst adults for a number of years. Longitudinal data from the Bogalusa Heart Study further indicates an association between the number of cardiovascular risk factors in childhood and the likelihood of atherosclerotic lesions in early adulthood [42]. Data also suggests that a cluster score derived from the components of the metabolic syndrome in late childhood (waist circumference, triglycerides, HDL, fasting glucose, and blood pressure) was a better predictor of early adult cardiovascular risk than the simple identification of “metabolic syndrome” or any single component of the cluster [43]. A study examined cross-sectional data among 474 adolescents and found that those subjects who had ≥ 2 cardiovascular risk factors (vs. less) had greater vascular stiffness and higher wall thickness compared with those having few risk factors [3].

This study has several important strengths including its prospective design with ten years of follow up throughout adolescence, the large number of 3-day diet records which enhance the precision of estimated dietary intakes, and the replicate measures of cardiovascular risk factors and potential confounders. Calculation of food group intakes by linking NDS data from study subjects with USDA Food Pyramid that underlies the analysis is also an important strength. This study is, however, subject to several limitations that are common to epidemiologic studies of dietary exposures. Dietary data are of necessity obtained by self-report and are thus subject to potential misreporting such as underestimation of energy intake. A further specific limitation of the study is the low levels of intake in certain food sub-groups (e.g., whole grains) which limited the power of certain sub-analyses related to these exposures.

The results of this study support the hypothesis that food intake patterns in early to midadolescence may have long-term effects on the clustering of cardiometabolic risk in older African-American and Caucasian adolescent girls. These findings may have important implications for the prevention of cardiovascular disease and diabetes in these populations.

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Table 1.

Subject characteristics according to usual mean daily dietary intakes at ages 9–17 years

	Dairy Servings ^a			Fruit & Non-Starchy Vegetable Servings ^a			Grains Servings ^a		
	<1.0 (n=223)	1.0–<2.5 (n=959)	>2.5 (n=187)	<1.0 (n=392)	1.0–<1.5 (n=449)	>1.5 (n=528)	<5.0 (n=299)	5.0–<7.0 (n=721)	>7.0 (n=349)
N (column percent)									
Race									
White	45 (20.2%)	440 (45.9%)	154 (82.4%)	166 (42.3%)	186 (41.4%)	287 (54.4%)	115 (38.5%)	357 (49.5%)	167 (47.9%)
Black	178 (79.8%)	519 (54.1%)	33 (17.6%)	226 (57.7%)	263 (58.6%)	241 (45.6%)	184 (61.5%)	364 (50.5%)	182 (52.1%)
SES									
Low	44 (19.7%)	221 (23.1%)	28 (15.0%)	113 (28.8%)	115 (25.6%)	65 (12.3%)	68 (22.7%)	141 (19.6%)	84 (24.1%)
Moderate	109 (48.9%)	403 (42.0%)	63 (33.7%)	182 (46.4%)	197 (43.9%)	196 (37.1%)	137 (45.8%)	306 (42.4%)	132 (37.8%)
High	70 (31.4%)	335 (34.9%)	96 (51.3%)	97 (24.8%)	137 (30.5%)	267 (50.6%)	94 (31.5%)	274 (38.0%)	133 (38.1%)
Mean (s.d.) ^b									
BMI (kg/m ²)	23.1 (5.4)	22.0 (4.8)	20.8 (4.2)	22.5 (5.1)	22.0 (4.6)	21.7 (4.8)	23.0 (5.1)	21.9 (4.9)	21.3 (4.3)
Activity (METS)	17.4 (9.2)	19.6 (9.8)	22.6 (11.0)	17.4 (9.1)	18.9 (9.5)	22.0 (10.5)	18.7 (8.9)	19.9 (10.0)	20.1 (10.8)
Television (hrs/day)	5.3 (1.9)	4.7 (2.1)	3.4 (2.1)	5.0 (2.0)	4.9 (2.0)	4.1 (2.2)	4.9 (2.0)	4.5 (2.0)	4.6 (2.3)
Total energy (kcal/day)	1745 (331)	1889 (361)	2099 (374)	1751 (320)	1897 (364)	1998 (376)	1565 (255)	1876 (289)	2214 (338)
% kcal-protein	13.7 (2.0)	14.0 (1.8)	14.9 (1.9)	14.1 (2.05)	14.0 (1.9)	14.2 (1.1)	14.6 (2.2)	14.0 (1.8)	13.8 (1.7)
% kcal-carbohydrates	51.2 (5.1)	51.8 (4.9)	52.1 (4.9)	50.0 (4.9)	51.2 (4.3)	53.4 (5.0)	50.8 (5.2)	51.8 (4.7)	52.3 (5.1)
% kcal-fat	36.0 (4.0)	35.2 (3.9)	34.2 (4.2)	36.7 (3.7)	35.7 (3.5)	33.7 (4.1)	35.6 (4.1)	35.1 (3.8)	35.0 (4.4)
Fruit	0.83 (0.6)	0.89 (0.6)	1.1 (0.7)	0.37 (0.2)	0.72 (0.2)	1.5 (0.6)	0.80 (0.6)	0.89 (0.6)	1.0 (0.7)
Vegetables	1.1 (0.5)	1.0 (0.4)	1.0 (0.4)	0.84 (0.3)	1.1 (0.4)	1.2 (0.5)	0.94 (0.4)	1.0 (0.4)	1.1 (0.4)
Grains	5.4 (1.2)	6.2 (1.4)	6.8 (1.6)	5.7 (1.2)	6.1 (1.4)	6.5 (1.5)	4.3 (0.5)	6.0 (0.6)	8.0 (1.0)
Dairy products	0.78 (0.2)	1.6 (0.4)	3.0 (0.5)	1.6 (0.7)	1.6 (0.7)	1.8 (0.8)	1.4 (0.6)	1.7 (0.7)	1.9 (0.7)
Meat products	5.0 (1.7)	4.6 (1.6)	4.3 (1.6)	4.4 (1.4)	4.7 (1.6)	4.8 (1.7)	4.3 (1.4)	4.5 (1.5)	5.1 (1.8)

SES = socioeconomic status; BMI = body mass index; METs = metabolic equivalents derived from physical activity scores

^aDairy and fruit & non-starchy vegetables expressed as cup equivalents (i.e., for fruit & non-starchy vegetables, 1 cup raw or cooked non-starchy vegetable or fruit, ½ cup dried vegetable or fruit, or 1 cup (8 fl. oz.) 100% vegetable or fruit juice, or 2 cups leafy salad greens); grain intakes expressed as ounce equivalents (i.e. amount equivalent to 1 bread slice, 1 cup cereal, or 1/2 cup cooked pasta/rice)^bMean values from ages 9–17 years

Table 2.

Clustering of cardiometabolic risk factors at 18–20 years of age overall and by categories of dietary intakes

	N	Number of Risk Factors ^a				4-6
		0	1	2	3	
N (row percent)						
Overall	1369	412 (30.1%)	478 (34.9%)	227 (16.6%)	152 (11.1%)	100 (7.3%)
Whites	639	187 (29.3%)	242 (37.9%)	103 (16.1%)	76 (11.9%)	31 (4.9%)
Blacks	730	225 (30.8%)	236 (32.3%)	124 (17.0%)	76 (10.4%)	69 (9.5%)
<u>Dairy (cup-eq/day)^b</u>						
<1.0	223	67 (30.0%)	75 (33.6%)	38 (17.0%)	28 (12.6%)	15 (6.7%)
1.0-<2.5	959	283 (29.5%)	333 (34.7%)	165 (17.2%)	98 (10.2%)	80 (8.3%)
>2.5	187	62 (33.2%)	70 (37.4%)	24 (12.8%)	26 (13.9%)	5 (2.7%)
p-value for χ^2		0.1200				
<u>FnsV (cup-eq/day)^b</u>						
<1	392	104 (26.5%)	127 (32.4%)	70 (17.9%)	52 (13.3%)	39 (10.0%)
1-<1.5	449	133 (29.6%)	161 (35.9%)	74 (16.5%)	51 (11.4%)	30 (6.7%)
>1.5	528	175 (33.1%)	190 (36.0%)	83 (15.7%)	49 (9.3%)	31 (5.9%)
p-value for χ^2		<0.001				
<u>Grains (oz-eq/day)^b</u>						
<5	299	90 (30.1%)	97 (32.4%)	61 (20.4%)	27 (9.0%)	24 (8.0%)
5-<7	721	211 (29.3%)	248 (34.4%)	109 (15.1%)	90 (12.5%)	63 (8.7%)
>7	349	111 (31.8%)	133 (38.1%)	57 (16.3%)	35 (10.0%)	13 (3.7%)
p-value for χ^2		<0.0960				

FnsV = fruit and non-starchy vegetables; BP = blood pressure; HDL = high-density lipoprotein; LDL = low-density lipoprotein; TG = triglycerides; HOMA-IR = homeostasis model assessment-insulin resistance

^a Risk factors include the following: waist 88cm, high BP 90th percentile for age, sex and height, HDL <50 mg/dL, LDL-C 110mg/dL, TG 110mg/dL, HOMA-IR 4.

^b Mean intakes ages 9–17

Table 3.

Clustering of cardiometabolic risk factors at 18–20 years associated with dietary patterns

Dietary Intakes ^b (ages 9–17 yrs)	N	Number of Risk Factors ^a				
		0	1	2	3	4–6
<i>N (row percent)</i>						
Dairy & FnsV						
Low Da / Low FnsV	635	177 (27.9%)	216 (34.0%)	118 (18.6%)	70 (11.0%)	54 (8.5%)
Low Da / High FnsV	341	107 (31.4%)	120 (35.2%)	53 (15.5%)	36 (10.6%)	25 (7.3%)
High Da / Low FnsV	206	60 (29.1%)	72 (35.0%)	26 (12.6%)	33 (16.0%)	15 (7.3%)
High Da / High FnsV	187	68 (36.4%)	70 (37.4%)	30 (16.0%)	13 (7.0%)	6 (3.2%)
<i>p-value for χ^2</i>						
Dairy & Grains				0.0046		
Low Da / Low Gr	775	224 (28.9%)	263 (33.9%)	133 (17.2%)	85 (11.0%)	70 (9.0%)
Low Da / High Gr	201	60 (29.9%)	73 (36.3%)	38 (18.9%)	21 (10.5%)	9 (4.5%)
High Da / Low Gr	245	77 (31.4%)	82 (33.5%)	37 (15.1%)	32 (13.1%)	17 (6.9%)
High Da / High Gr	148	51 (34.5%)	60 (40.5%)	19 (12.8%)	14 (9.5%)	4 (2.7%)
<i>p-value for χ^2</i>						
FnsV & Grains				0.0087		
Low FnsV / Low Gr	664	186 (28.0%)	223 (33.6%)	115 (17.3%)	80 (12.1%)	60 (9.0%)
Low FnsV / High Gr	177	51 (28.8%)	65 (36.7%)	29 (16.4%)	23 (13.0%)	9 (5.1%)
High FnsV / Low Gr	356	115 (32.3%)	122 (34.3%)	55 (15.5%)	37 (10.4%)	27 (7.6%)
High FnsV / High Gr	172	60 (34.9%)	68 (39.5%)	28 (16.3%)	12 (7.0%)	4 (2.3%)
<i>p-value for χ^2</i>						
				<0.001		

FnsV = fruit and non-starchy vegetables; BP = blood pressure; HDL = high-density lipoprotein; LDL = low-density lipoprotein; TG = triglycerides; HOMA-IR = homeostasis model assessment -insulin resistance

^aRisk factors include the following: waist 88cm, BP 90th percentile for age, sex and height, HDL <50 mg/dL, LDL-C 110mg/dL, TG 110mg/dL, HOMA-IR 4.

^bLow vs. high dairy=<2 vs. 2 cup-eq; low vs. high FnsV=<1.5 vs. 1.5 cup-eq; low vs. high grain=<7 vs. 7 oz-eq (based on mean dietary intakes ages 9–17).

Table 4.

Relative risk of two or more cardiometabolic risk factors at 18–20 years associated with dietary patterns

Dietary Intakes ^a (ages 9–17 yrs)	# with >2 risk factors ^b		# with >3 risk factors ^b	
	RR ^c	95% CI	RR ^c	95% CI
Dairy & FnsV				
Low Da / Low FnsV	1.00	-	1.00	-
Low Da / High FnsV	0.87	0.65–1.16	0.98	0.69–1.39
High Da / Low FnsV	0.91	0.64–1.28	1.28	0.85–1.91
High Da / High FnsV	0.62	0.42–0.91	0.52	0.30–0.89
Dairy & Grains				
Low Da / Low Gr	1.00	-	1.00	-
Low Da / High Gr	0.84	0.60–1.17	0.67	0.43–1.03
High Da / Low Gr	0.94	0.68–1.30	1.06	0.71–1.56
High Da / High Gr	0.57	0.37–0.86	0.56	0.33–0.97
FnsV & Grains				
Low FnsV / Low Gr	1.00	-	1.00	-
Low FnsV / High Gr	0.84	0.57–1.14	0.78	0.50–1.19
High FnsV / Low Gr	0.86	0.65–1.15	0.89	0.63–1.26
High FnsV / High Gr	0.58	0.40–0.86	0.41	0.23–0.71

FnsV = fruit and non-starchy vegetables; Gr = grains; BP = blood pressure; HDL = high-density lipoprotein; LDL = low-density lipoprotein; TG = triglycerides; HOMA-IR = homeostasis model assessment-insulin resistance

^aLow vs. high dairy=<2 vs. 2 cup-eq; low vs. high FnsV=<1.5 vs. 1.5 cup-eq; low vs. high grain=<7 vs. 7 oz-eq (based on mean dietary intakes ages 9–17).

^bRisk factors include the following: waist 88cm, BP 90th percentile for age, sex and height, HDL <50 mg/dL, LDL-C 110mg/dL, TG 110mg/dL, HOMA-IR 4.

^cAdjusted for age (at time of diet assessment), race, SES, mean height, physical activity, and television/video watching (hours/day).