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Short Communication

Breakfast and fast food consumption are associated with selected biomarkers in adolescents

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ABSTRACT

Objective: Skipping breakfast and consuming fast food are related to the risk of obesity and are common adolescent behaviors. The relationship between these behaviors and biomarkers related to diabetes and CVD is understudied in this population. *Methods*: Data are from a study of the etiologic factors related to obesity risk in adolescents. Breakfast and fast food consumption were assessed using a self-report survey. Anthropometrics, fasting lipids, glucose, insulin, and homeostatic model assessment for insulin resistance (HOMA-IR) were assessed. Multivariate analyses were used to examine the relationship between dietary behaviors and selected biomarkers, controlling for calories consumed, body mass index (BMI), and demographic covariates. *Results*: 367 adolescents (11 to 18-years; mean 14.7 ± 1.8 years) were assessed at the University of Minnesota-Twin Cities from 2006–2008. Breakfast consumption was significantly associated with lower BMI, body fat, insulin, HOMA-IR, and metabolic syndrome (MetS) cluster score, while fast food consumption was associated with higher BMI, body fat, low-density lipoprotein cholesterol, triglycerides, glucose, insulin, HOMA-IR, and MetS cluster score. Some gender differences were observed. *Conclusion*: Breakfast and fast food consumption appear to be related to important metabolic syndrome biomarkers for chronic disease in a sample of healthy adolescents. The importance of this finding needs to be validated by examining the stability of this pattern over time and to assess the pattern in other populations.

Introduction

The prevalence of overweight and obese youth has increased dramatically over the past three decades (Wang and Lobstein, 2006) and has grown to be one of the most important public health concerns of our time (Ogden et al., 2014). Indeed, youth obesity is associated with a myriad of health risks such as elevated blood pressure, dyslipidemia, hyperglycemia, and insulin resistance that contribute to increased metabolic risk (Ogden et al., 2014). Similar to adults, overweight and obese youth often display a "clustering" of individual risk factors that may act together towards metabolic dysfunction (Grundy, 2005). Since obese youth are more likely to become obese adults and will likely exhibit similarly increased risk for adverse cardiovascular outcomes as observed in adults (Freedman et al., 2001; Guo and Chumlea, 1999), a better understanding of the determinants of youth obesity is necessary to develop intervention approaches for obesity prevention and translation to adulthood.

Adolescence and young adulthood are also life periods that often coincide with unhealthy dietary patterns and increased physical inactivity (Nelson et al., 2008) that may contribute to increased cardiovascular and metabolic risk. Indeed, declines in breakfast consumption have been reported during adolescence (Siega-Riz et al., 1998) and the proportion of energy consumed at restaurants or fast food establishments increases significantly while at-home meals decrease during this crucial life stage (Bauer et al., 2009; Nielsen et al., 2002). Current research links fast food consumption with increased obesity risk (Ebbeling et al., 2004) and report improved adiposity indexes and selected metabolic profiles with increased breakfast consumption (Cho et al., 2003; Papoutsou et al., 2014; Szajewska and Ruszczynski, 2010). Additionally, adolescents are also undergoing physical and metabolic changes associated with puberty that may also contribute to overall cardiometabolic risk (Steinberger et al., 2001).

Abbreviations: BMI, Body mass index; CVD, Cardiovascular disease; DBP, Diastolic blood pressure; HDL, High-density lipoprotein cholesterol; HOMA-IR, Homeostasis model assessment for insulin resistance; LDL, Low-density lipoprotein cholesterol; MetS, Metabolic syndrome; PBF, Percent body fat; SBP, Systolic blood pressure; TG, Triglycerides.

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Given obese youth are more likely to become obese adults and have increased risk for adverse cardiovascular disease outcomes, it is important to understand adolescent lifestyle behaviors such as breakfast and fast food consumption, and their relationship to cardiovascular and metabolic risk. While past studies have examined the relationship between breakfast and fast food consumption separately, to our knowledge no studies to date have examined the associations of both breakfast and fast food consumption on cardiovascular and metabolic biomarkers in the same adolescent population as they relate to the metabolic syndrome. Therefore, the primary objective of this study is to evaluate the relationship between both breakfast and fast food consumption on selected biomarkers and important cardiovascular and metabolic risk factors among healthy adolescents, and further examine the relationship between these dietary behaviors and the known risk factor clustering that occurs with the metabolic syndrome. We hypothesize that increased breakfast and decreased fast food consumption would be significantly associated with lower cardiovascular risk and a healthier metabolic profile as assessed through a fasting blood draw, thereby allowing examining of the metabolic syndrome in youth.

Methods

Sample and measures

Subjects were from two large etiologic studies of childhood obesity investigating individual, social, and environmental influences on adolescent obesity risk (Lytle, 2009). Data for this research includes 367 youth between ages 11 and 18-years (mean 14.7 ± 1.8 years) for which fasting blood draws were available. Recruitment was conducted within a 7-county metropolitan area from Minneapolis/St. Paul, Minnesota from 2006 to 2008. Youth were invited to participate regardless of weight status but were required to participate with one adult with whom they spent a significant amount of time (e.g., a parent/guardian, or other relative or adult that cares for them). Adult/youth pairs were excluded from participating if they planned to move from the area in the next 3 years, had a medical condition that affected their growth, were non-English speaking, and/ or had any other physical or emotional condition that would affect their diet/activity levels, or make it difficult to complete measurements (Lytle, 2009; Dengel et al., 2010). Height, weight, body composition, and seated blood pressure were measured (Lytle, 2009; Dengel et al., 2010) and body mass index (BMI) was calculated as weight in kilograms (kg) divided by height in meters-squared (m^2). BMI-percentiles were derived (CDC Growth Charts, 2000). Pubertal status was assessed using validated self-report measures (Lytle, 2009).

Breakfast and fast food consumption were assessed using a self-report survey using validated questions (Nelson and Lytle, 2009). Breakfast consumption was expressed as average number of days/week that breakfast was consumed. Fast food consumption was reported as the number of times that fast food (over the counter or drive-thru restaurants) was eaten over the last month (never/rarely, 1×/month, 2–3×/month, 1–2×/week, 3–4×/week, 5–6×/week, 1×/day, 2×/day, 3×/day or more). Average caloric consumption was calculated from averaging three 24-h recalls (2 weekdays, 1 weekend day) as previously described (Lytle, 2009).

A 12-h fasting blood draw was administered. Plasma samples were measured for glucose and insulin, as well as fasting lipids (triglycerides (TG), low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL)). Homeostasis model assessment for insulin resistance (HOMA-IR) was calculated as previously reported (Matthews et al., 1985). A metabolic syndrome (MetS) cluster score was generated via the summation of sample-specific z-scores including percent body fat (PBF), insulin, HDL (negative), TG, and systolic blood pressure (SBP) in an effort to evaluate risk factor clustering. Previous studies have incorporated a MetS cluster score using modified ATP III risk factors (Kelly et al., 2011). A higher individual MetS cluster score indicates risk factor clustering in higher sections of the sample distributions, representing heightened metabolic risk. The study was approved by the University of Minnesota Institutional Review Board (IRB) and adhered to HIPAA guidelines.

Statistical analysis

SAS Software Package (Version 9.4, SAS Inc., Cary, NC, USA) was used for statistical analysis. Demographic characteristics are expressed as mean ± SD. A generalized linear model was used to assess the relationship between breakfast and fast food consumption, and selected biomarkers, adjusting for study cohort, age, sex, pubertal status, parent/guardian level of education, and average caloric consumption. An additional adjustment for BMI was used for glucose, insulin, HOMA-IR, and lipids. BMI, PBF, lipids (TG, LDL, and HDL), SBP, diastolic blood pressure (DBP), glucose and insulin, HOMA-IR, and MetS cluster score were the designated outcome biomarkers of interest. Cross-sectional data from one time point were used in the analysis. An alpha value of 0.05 was used to signify statistical significance.

Results

Youth were predominantly white (88%); mid- or advanced-pubertal developmental stages (84%); and had at least one parent/guardian having obtained a college education (91%). Clinical and dietary characteristics are further presented in Table 1. About one-quarter of the sample was overweight or obese. Average breakfast consumption was about 3 times per week while the sample reported consuming fast food was about 3 times each month. All clinical measures were within normal reference ranges for the study sample.

Model summary statistics are displayed in Table 2. More frequent breakfast consumption was significantly associated with lower BMI, PBF, insulin, HOMA-IR, and MetS cluster score. And more frequent fast food consumption was significantly associated with higher BMI, PBF, LDL, TG, glucose, insulin, HOMA-IR, and MetS cluster score. Blood

Table 1
Clinical and dietary characteristics.

n (sample size)	367
Male (n, %)	187 (51%)
BMI (kg/m^2)	21.8 ± 4.5
BMI-percentile (n, %)	
Underweight (<5th)	8 (2.2%)
Normal weight (5 to 85th)	271 (73.8%)
Overweight (85 to 95th)	47 (12.8%)
Obese (>95th)	41 (11.2%)
PBF (%)	20.9 ± 10.2
Systolic BP (mmHg)	114.8 ± 9.6
Diastolic BP (mmHg)	54.5 ± 7.5
LDL (mg/dL)	85.4 ± 24.9
HDL (mg/dL)	49.7 ± 11.3
TG (mg/dL)	80.3 ± 43.1
Fasting glucose (mg/dL)	80.0 ± 7.4
Fasting insulin (mU/L)	8.6 ± 5.8
HOMA-IR	1.7 ± 1.2
MetS cluster score	−0.42 ± 2.52
Dietary consumption	
Average caloric intake (kcal)[Ω]	1955.4 ± 600.5 [1893.8, 2017.1]
Breakfast (days/week)[§]	3.3 ± 1.0 [3.1, 3.4]
Fast Food (times/month)[†]	2.9 ± 1.0 [2.8, 3.0]

Measures listed as Mean ± SD, unless otherwise noted. Dietary consumption variables include 95% confidence intervals. BMI, body mass index; PBF, percent body fat; BP, blood pressure; LDL, low-density lipoprotein cholesterol; HDL, high-density lipoprotein cholesterol; TG, triglycerides; HOMA-IR, homeostatic model assessment-insulin resistance; MetS, metabolic syndrome. [Ω]Average energy intake (kcal) calculated from averaging 3 34-h recalls. [§]Days/week (including weekends) breakfast was typically eaten. [†]Categorical number of times buying fast food over past month (1 = never/rarely, 2 = once/month, 3 = 2–3×/month, 4 = 1–2×/week, 5 = 3–4×/week, 6 = 5–6×/week, 7 = 1×/day, 8 = 2×/day, 9 = 3×/day or more).

Table 2
Clinical and dietary characteristics (n = 367).

Clinical outcome variables	Dietary variables	
	Breakfast	Fast food
BMI (kg/m^2)	−0.479*	0.599*
PBF (%)	−0.972*	1.051*
Systolic BP (mmHg)	−0.896	−0.045
Diastolic BP (mmHg)	−0.048	−0.187
LDL (mg/dL)†	−0.956	3.198*
HDL (mg/dL)†	−0.231	−0.550
TG (mg/dL)†	−2.266	3.006*
Glucose (mg/dL)†	0.138	0.942*
Insulin (mU/L)†	−0.609*	0.374*
HOMA-IR†	−0.129*	0.100*
MetS cluster score	−0.258*	0.353*

Generalized linear model β coefficients adjusted for covariates including age, gender, pubertal status, BMI (for glucose, insulin, HOMA-IR, and lipids only†), highest parent level of education, average caloric intake, and cohort.
* Signifies statistical significance ($P < 0.05$).

pressure and HDL were not associated with either fast food or breakfast consumption.

Discussion

The finding suggests that fast food and breakfast consumption are associated with some metabolically important chronic disease risk factors in healthy adolescents. To our knowledge, this is the first study to examine the association of both breakfast and fast food consumption on cardiovascular and metabolic biomarkers as they relate to metabolic syndrome clustering. The findings of the present study are consistent with some studies in youth of similar ages (Berkey et al., 2003; Laska et al., 2012; Papoutsou et al., 2014; Siega-Riz et al., 1998; Smith et al., 2010), but not all (Resnicow, 1991), of the previously conducted cross-sectional studies reporting greater obesity risk in adolescents who skip breakfast than those who consumed breakfast. Resnicow (1991) reported higher plasma cholesterol concentrations among children who skipped breakfast but we observed no association between breakfast consumption and fasting lipids within the present study. Unique to past research studies, we found that higher levels of breakfast consumption were significantly associated with lower HOMA-IR and metabolic syndrome cluster score, while fast food consumption was significantly associated with higher HOMA-IR and metabolic syndrome cluster score.

Previous studies in youth have reported associations between skipping breakfast and increased BMI, adiposity, and the metabolic syndrome (Alexander et al., 2009; Boutelle et al., 2002; Fiore et al., 2006; Shafiee et al., 2013; Summerbell et al., 1996; Vanelli et al., 2005), yet not all studies are in agreement (Albertson et al., 2007; Berkey et al., 2003). Specifically, Albertson et al. (2007) report girls who ate breakfast more often displayed lower BMI at the end of the study, whereas Berkey et al. (2003) found that overweight children who never ate breakfast reported decreased BMI over the study yet normal weight children who never ate breakfast actually gained weight relative to those who ate breakfast. Possible mechanisms for increased weight gain over time may be linked to the potential up-regulation of appetite as a result of skipping breakfast (Timlin and Pereira, 2007). Nonetheless, a lack of a universal definition of breakfast consumption, as well as different methods for measuring breakfast consumption and meal composition, has led to differing results in many studies examining the link between breakfast consumption and composition on health outcomes (Timlin and Pereira, 2007), making interpretation of results difficult across studies. Additionally, observed differences between studies may also be partially due to varying study designs (longitudinal vs. cross-sectional) (Must et al., 2009).

Previously conducted studies also support our findings surrounding fast food consumption, specifically higher consumption and the association with higher body fatness (Pereira et al., 2005). Because fast food and meals eaten away from home are usually higher in energy density, cholesterol, saturated fat, and sodium (Hearst et al., 2013), it is reasonable to postulate that frequent and habitual consumption of fast food would lead to increased weight gain and a poorer metabolic profile. Indeed, studies have reported strong positive associations between fast food consumption and insulin resistance, as well as weight gain (Pereira et al., 2005). Nonetheless, fast food consumption within our adolescent population is likely low compared to previous reports; specifically, French et al. (2001) reported that 75% of a sample of 5000 youth, 11 to 18 years of age, self-reported eating fast food during the previous week. Additionally, Paeratakul et al. (2003) reported that youth 11 to 18 years of age eat at fast food outlets an average of twice per week.

The present study is indeed the first study to examine the association of both breakfast and fast food consumption on cardiovascular and metabolic biomarkers as they relate to the potential metabolic syndrome in youth, and poses the potential use of a metabolic syndrome cluster score to assess these often clustering biomarkers. Future studies would need to evaluate the usage of such a metabolic cluster score and confirm the potential relevance of this cluster score in youth outcomes, as well as to possible longitudinal outcomes in adulthood.

Study limitations include the relatively homogenous sample population and potential underreporting with dietary recall. The frequency with which our sample reported consuming fast food is lower than other population estimates and may reflect a sample bias; we are one of the few studies of adolescents that examine parent and adolescent dyads. We also did not control for level of physical activity within our model. Our findings support our hypothesis that breakfast and fast food consumption are associated with the metabolic profile in expected directions. It is important to see that these metabolic risk factors are negatively impacted by infrequent breakfast consumption and more frequent fast food consumption even in healthy adolescents. Future research should examine how these relationships track over time, possibly as they relate to the metabolic syndrome in youth, and their long-term impact on chronic disease risk.

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Conflict of interest statement

The authors declare that there are no conflicts of interest.

Transparency Document

The Transparency document associated with this article can be found, in the online version.

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