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).

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 (m²). 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/m²)	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. ΩAverage 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).