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Regular Consumption from Fast Food Establishments Relative to Other Restaurants Is Differentially Associated with Metabolic Outcomes in Young Adults[1,2]

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Abstract

Although away-from-home eating is adversely associated with weight, other comorbidities have not been examined; therefore, we sought to determine the associations of fast food (e.g. Wendy's, McDonalds) and restaurant (sit-down style) consumption (times per week) with weight and multiple metabolic outcomes, including homeostatic model assessment insulin resistance (HOMA-IR), waist circumference, and plasma triglycerides (TG), LDL cholesterol, and HDL cholesterol (HDL-C). We used 3 waves of data (exam y 7, 10, and 20) from the Coronary Artery Risk Development in Young Adults Study, a prospective cohort study of black and white young adults [aged 25–42 y in 1992–93, n = 3643 (men, 1659; women, 1984)]. Individuals in the highest (vs. lowest) quartile of baseline (defined as the mean of y 7 and 10) fast food consumption had higher y 20 weight [adjusted mean (95% CI): 5.6 kg (CI, 2.1, 9.2); P = 0.002], HOMA-IR [0.9 (CI, 0.4, 1.3); $P < 0.001$], waist circumference [5.3 cm (CI, 2.8, 7.9); $P < 0.000$], TG concentrations [0.25 mmol/L (CI, 0.10, 0.40), 22.7 mg/dL (CI, 9.1, 36.3); P = 0.001], and lower HDL-C concentrations [−0.014 mmol/L (CI, −0.215, −0.067), 5.4 mg/dL (CI, −8.3, −2.6); $P < 0.000$]. Baseline restaurant consumption was unrelated to y 20 outcomes. Adjusted change in weekly restaurant ($P < 0.05$) and fast food intake ($P < 0.001$) was associated with 13-y changes in body weight [0.09 kg (CI, 0.02, 0.17) and 0.15 kg (CI, 0.06, 0.24), respectively] and waist circumference [0.08 cm (CI, 0.02, 0.14) and 0.12 cm (CI, 0.04, 0.20), respectively]. Fast food consumption may be an important target for the prevention of adverse metabolic health outcomes. J. Nutr. doi: 10.3945/jn.109.109520.

Introduction

Away-from-home food (available in fast food places and restaurants) contributes considerably to daily energy intake (1) and accounts for roughly one-third of energy intake among certain subpopulations, particularly young adult males (2,3). Fast food consumption has been associated with adverse health outcomes including increased risk of excess weight, body fatness, poor dietary quality, and insulin resistance/diabetes (1,2,4–8). Mechanisms for the direct contribution of fast food intake to the development of diabetes and other obesity-related comorbidities have included low unsaturated:saturated fat ratio, greater portion sizes (9), and lower fiber content of fast food (10).

Cross-sectional (11,12) studies have demonstrated an association between away-from-home food consumption with weight and glucose outcomes, but these studies have limited ability to address causality due to concurrent assessment of exposure and outcome. Prospective observational studies (4,13) have also demonstrated an association between away-from-home food consumption with weight and glucose, but only 1 study differentiated between restaurant and fast food intake (13) and it was limited by a short time duration. To our knowledge, not a single prospective or longitudinal study has collected detailed food intake data by location and food source so that the impact of the type of foods eaten at restaurants or fast food outlets could be studied, representing a major gap in the literature. Although we cannot address this limitation, this study fills another important gap, in that, to our knowledge, that no study exists on the relationship between away-from-home food consumption and a broad set of metabolic outcomes.

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The purpose of this study was to examine the association between baseline (defined as mean of exam y 7 and 10) away-from-home food (restaurant and fast food) consumption on 13-y health outcomes and the change in away-from-home food consumption with 13-y changes in health outcomes. Based on previous research in this population (4,13), we hypothesized that fast food and restaurant consumption would be differentially associated with weight, homeostatic model assessment insulin resistance (HOMA-IR)[7] score, and plasma triglycerides (TG), LDL cholesterol (LDL-C), and HDL cholesterol (HDL-C) concentrations.

Participants and Methods

Study population. Data were taken from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a prospective study of the determinants and evolution of cardiovascular risk. Recruitment procedures were designed to create balanced representation of age, gender, ethnicity, and education groups within each of the 4 study sites. A total of 5115 young adults aged 18–30 y completed the first survey (exam y 0, 1985–1986). Follow-up examinations were conducted at 2, 5, 7, 10, 15, and 20 y later. Data from exam y 7, 10, and 20 (retention rates 81, 74, and 72%, respectively) were used for this study. Detailed descriptions of the sampling plan and cohort characteristics are described elsewhere (14,15). Sample sizes differed for each modeling framework and outcome variable (described below). All analyses were in accordance with the ethical standards of the University of North Carolina at Chapel Hill; the study was approved by the Institutional Review Board at the University of North Carolina at Chapel Hill.

Away-from-home eating. Our main exposure, frequency of restaurant and fast food consumption, was ascertained at exam y 7, 10, and 20 using an interviewer-administered questionnaire. Participants were asked "How many times in a week or month do you eat breakfast, lunch or dinner in a place such as McDonald's, Burger King, Wendy's, Arby's, Pizza Hut, or Kentucky Fried Chicken?" and subsequently "How many times in a week or month do you eat breakfast, lunch, or dinner at a restaurant or cafeteria?" These questions were open-ended, but answers were calculated to reflect a "per-week" frequency.

Anthropometrics, insulin resistance, and blood lipids. Measured height (nearest 0.5 cm) and weight (nearest 0.1 kg) were collected by trained technicians. Waist circumference (measured in cm) was measured midway between the iliac crest and the lowest lateral portion of the rib cage using the average of 2 measurements.

Venous blood was drawn from fasting participants for measurement of plasma insulin, glucose, and blood lipids. Glucose was measured using hexokinase coupled to glucose-6-phosphate dehydrogenase. Insulin measurements were performed by using an RIA with an overnight, equilibrium-incubation format. The HOMA-IR was calculated as [glucose (mmol/L) $\times$ insulin (μU/L)]/22.5 (16).

Plasma total cholesterol and TG were measured using enzymatic assays (17) at each follow-up visit. HDL-C was assayed after dextran sulfate-magnesium precipitation (18) and LDL-C was estimated from the Friedewald equation (19). LDL-C concentrations were not calculated for individuals who had plasma TG concentrations >400 mg/dL (n = 68).[8]

Covariates. Using standardized questionnaires, self-reported information on sociodemographic characteristics and selected health behaviors were collected at exam y 7, 10, and 20, including age, education (highest level achieved), smoking status (current, former, never), family structure (married, single, married with children, single with children), and sedentary behavior (hours of TV watched per day). Race (black vs. white) and gender were verified at each follow-up exam. Total energy intake was derived from the CARDIA FFQ, an interviewer administered questionnaire that assesses usual intake over the past month. The CARDIA FFQ was administered at exam y 7 and 20, with values for y 10 carried forward from y 7 for modeling purposes. Physical activity (PA), presented as exercise units (EU) per week, was assessed using the CARDIA PA questionnaire (20). For reference, 300 EU roughly approximates the American College of Sports Medicine recommendations for the amount of exercise needed to support weight loss [5 sessions of 300 kcal (1260 kJ) of weekly energy expenditure] (21). Due to nonlinearity with the outcome, PA was dichotomized as high ($\geq$474 EU/wk) vs. low ($<$474 EU/wk).

Statistical analysis. All statistical analyses were conducted in Stata (version 10.0). Differences in sociodemographic characteristics between exam y 7, 10, and 20 were determined using Student's t test (continuous) and chi-squared tests (categorical) where appropriate, with significance set at $P < 0.05$. Using ordinary least-squares regression models, we first examined the association between fast food and restaurant consumption [mean of y 7 and 10 times per week, divided into quartiles: lowest quartile (referent)] with y 20 outcomes (weight, HOMA-IR, waist circumference, TG, LDL-C, and HDL-C) adjusting for y 10 sociodemographic (age, race, education, family structure, and CARDIA exam center) and behavioral [smoking status, PA, hours of television viewing, total energy, y 10 value (continuous) of the outcome variable, and change in fast food and restaurant consumption between y 10 and 20] covariates. Estimates were similar in sign and significance if y 7 and 10 frequencies were independently modeled. The interaction between exposure and gender was not significant, so combined estimates are presented here. Adjustment for macronutrients (total and saturated fat) or selected lipid-containing food groups (dairy, meat) did not change our estimates and are excluded for parsimony. Year 20 outcome values were obtained from each estimated β coefficient using the ADJUST command in Stata.

For each outcome, we used participants with complete data. Of those with complete exposure and outcome data [n = 2439 (weight), n = 2238 (HOMA), n = 2437 (waist circumference), n = 2193 (TG and HDL-C), n = 2170 (LDL-C)], we excluded those missing covariate data [n = 1341 (weight), n = 122 (HOMA), n = 133 (waist circumference), n = 117 (TG and HDL-C), n = 125 (LDL-C)] or information on change in restaurant and/or change in fast food exposure between exam y 10 and 20 [n = 672 (weight), n = 606 (HOMA), n = 667 (waist circumference), n = 603 (TG and HDL-C), n = 596 (LDL-C)], resulting in final sample sizes of n = 1633 (weight), n = 1510 (HOMA), n = 1637 (waist circumference), n = 1473 (TG and HDL-C), and n = 1458 (LDL-C).

[7] Abbreviations used: CARDIA, Coronary Artery Risk Development in Young Adults; EU, exercise unit; HOMA-IR, homeostatic model assessment of insulin resistance; PA, physical activity; TG, triglyceride.

[8] To convert LDL-C and HDL-C to mmol/L, multiply by 0.0259. To convert TG to mmol/L, multiply by 0.0113.

Respondents who were included and those who were not did not differ.

Estimates obtained from standard regression models (e.g. those described above) are useful for providing an estimate of the average effect of away-from-home food consumption on subsequent health outcomes, but these estimates can be influenced by unobserved (e.g. an individual's motivation to be healthy) or unmeasured (e.g. knowledge of the risk of being overweight) variables, particularly those that change over time. Longitudinal, repeated-measures conditional regression models (also known as fixed-effect longitudinal models) account for these unmeasured variables. Conditioned on the participant, these models estimate parameters for characteristics that are time-variant within the individual (e.g. weekly fast food consumption), whereas time-invariant parameters (e.g. gender) are not estimated.

For each model, time-varying fast food and restaurant consumption (continuous) were regressed on time-varying outcome variables (continuous), adjusting for time-varying demographic (including age, education, and family structure) and lifestyle factors (including PA, sedentary behavior, total energy, and smoking status) collected at each exam year. Using all available data across 13 y and 3 exam periods provided the following sample sizes [observations (subjects)]: weight: 8489 (3987); HOMA-IR: 7921 (3873); waist: 8472 (3982); TG and HDL-C: 8152 (3926); and LDL-C: 7466 (3450).

Results

As the population aged, there were expected increases in level of education, BMI, and the proportion that were overweight and obese (**Table 1**). The difference in weekly consumption between the lowest and highest quartile of fast food and restaurant consumption decreased over time, from 4.3 times/wk at exam y 7 to just 2 times/wk at exam y 20. Decreases were similar for restaurant consumption (Table 1).

Compared with the lowest quartile of fast food consumption, participants in the higher quartiles has significantly higher exam y 20 weight and waist circumferences, HOMA-IR scores, and TG concentrations and significantly lower plasma HDL-C concentrations (**Table 2**). For example, compared with the lowest quartile of fast food intake, persons in the highest quartile were 5.7 kg (95% CI: 2.1, 9.2; $P = 0.002$) heavier, had 5.3 cm (95% CI: 2.8, 7.9; $P < 0.001$) larger waist circumference, 22.7 mg/dL (95% CI: 9.1, 36.3; $P = 0.001$) higher plasma TG concentrations, and 5.5 mg/dL (95% CI: −8.3, −2.6; $P < 0.001$) lower plasma HDL-C concentrations. Conversely, the associations with greater weekly restaurant food consumption tended to be in the opposite direction as those with increasing fast food intake.

Unlike the previous results (Table 2), 1 additional weekly fast food or restaurant consumption eating occasion was positively associated with 13-y changes in weight and waist circumference (**Table 3**). In the fully adjusted model (model 2), an increase of 3 times/wk consuming fast food (restaurant) was associated with a 0.45 kg ± 0.15 kg weight gain. Changes in waist circumference were small but roughly equivalent between restaurant and fast food. Changes in fast food and restaurant consumption were unrelated to changes in HOMA-IR and cholesterol concentrations.

Discussion

Using prospective data spanning 13 y, we found that higher baseline (mean of y 7 and 10) fast food, but not baseline restaurant, consumption was positively associated with y 20 health outcomes, including weight and waist circumference, HOMA-IR score, and TG concentrations and negatively associated with y 20 HDL-C concentrations. These associations are clinically relevant. For example, someone with a height of 1.87 m weighing 84.4 kg at exam y 20 would go from a y 20 BMI of 24.2 {20 y BMI = [84.4/(1.87 m^2)] = 24.1} to a predicted y 20 BMI of 25.8 {predicted 20 y BMI = [(84.4+5.7 kg)/(1.87 $m)^2$] = 25.8} if they were in the highest compared with the lowest quartile of fast food intake.

Similarly, the mean increase in TG of 22.7 mg/dL in the highest (4th) compared with 9.4 mg/dL for persons in the second-lowest (2nd) quartiles, respectively, resulted in a 4% increase in the proportion of the sample classified as having high y 20 TG concentrations (≥150 mg/dL) according to the National Cholesterol Education Program ATP III Guidelines (22) [predicted TG ≥150 mg/dL: quartile 4, 175 of 981 persons (17.8%); quartile 2, 111 of 829 persons (13.4%)]. For individuals with additional coronary heart disease risk factors, this could be the difference between needing and not needing medication.

One additional weekly away-from-home eating event was also associated with greater 13-y anthropometric changes. In longitudinal repeated-measures conditional regression models, restaurant and fast food consumption were unrelated to changes in HOMA insulin score and cholesterol concentrations over the 13-y period. As a side note, fast food consumption was also associated with incident metabolic syndrome. Compared with the lowest quartile of intake, persons in the 3rd and 4th quartiles of weekly fast food intake had greater odds of incident metabolic syndrome between exam y 10 and 20 [3rd quartile: odds ratio, 1.90 (95% CI: 1.11, 3.26; $P = 0.019$); 4th quartile: odds ratio, 2.14 (95% CI: 1.24, 3.70; $P = 0.006$)]. Further examination of additional incident health measures may prove valuable in understanding the full extent to which fast food and restaurant consumption affect health outcomes.

For weight and insulin resistance outcomes, previous studies in this cohort report comparable findings. Pereira et al. (4) found frequent consumers of fast food at baseline and during 15 y of follow-up had greater weight gain than did infrequent consumers, but they did not consider restaurant food consumption. The current finding that greater restaurant consumption is positively associated with 13-y changes in weight and waist circumference likely results from the use of more complex modeling strategies in which we modeled change in weight, not BMI, over a longer time span than previous research with BMI only (13).

Despite adjusting for many individual-level factors, significant relationships of weight, waist circumference, TG, HDL-C, and HOMA-IR with fast food consumption were observed. These findings may result from unmeasured area-level factors (e.g. neighborhood socioeconomic status, price of fast food) that could influence whether restaurants compared with fast food outlets were commonly patronized (23–25) and we cannot discount the fact that such factors may be partially responsible for the differences in the associations between consumption frequency and change in weight (26,27). Individuals might also eat differently when visiting a fast food compared with a sit-down style restaurant. Unfortunately, we are not able to examine the specific foods consumed from these 2 away-from-home sources (i.e. to determine whether the reported hamburger was purchased from Burger King or made at home on the grill) and therefore cannot accurately describe the dietary intakes of fast food compared with restaurant consumers. In fact, to our

TABLE 1 Sociodemographic and behavioral characteristics 3643 black and white young adults[1]

	All years	y 7 (1992–1993)	y 10 (1995–1996)	y 20 (2005–2006)
Black, *% (SE)*	51.6 (0.4)			
Female, *% (SE)*	54.5 (0.4)			
Away-from-home eating,[2] *n/wk*				
Fast food		1.9 ± 2.5[a]	1.8 ± 2.0[b]	1.7 ± 2.4[b]
Fast food, by quartile				
Quartile 1		0.2 ± 0.01	0.2 ± 0.01	0.6 ± 0.1
Quartile 2		0.8 ± 0.02	0.9 ± 0.02	1.4 ± 0.1
Quartile 3		1.8 ± 0.04	1.8 ± 0.04	1.8 ± 0.1
Quartile 4		4.5 ± 0.1	3.9 ± 0.1	2.6 ± 0.1
Change from previous exam		–[4]	−0.16 ± 2.4[a]	−0.13 ± 2.7
Restaurant		2.3 ± 3.2[a]	2.1 ± 2.3[b]	2.3 ± 2.5[a]
Restaurant, by quartile				
Quartile 1		0.3 ± 0.3	0.4 ± 0.01	1.4 ± 0.1
Quartile 2		1.1 ± 1.0	1.0 ± 0.03	2.0 ± 0.1
Quartile 3		2.0 ± 1.9	2.2 ± 0.1	2.2 ± 0.1
Quartile 4		5.7 ± 5.3	4.3 ± 0.1	3.4 ± 0.1
Change from previous exam		–[4]	−0.30 ± 3.4[a]	0.12 ± 2.8[b]
Total energy intake, *kJ/d*		2557 ± 939[a]	–[5]	2218 ± 862[b]
Demographics				
Age, *y*		32.0 ± 3.6	35.0 ± 3.7	45.2 ± 3.6
Education,[2] *% (SE)*				
Less than high school		5.8 ± 0.4[a]	6.3 ± 0.4[a]	4.3 ± 0.3[b]
Completed high school		23.4 ± 0.7[a]	23.2 ± 0.7[a]	19.9 ± 0.7[b]
More than high school		70.8 ± 0.7[b]	70.4 ± 0.7[b]	75.8 ± 0.7[a]
Smoking status,[2] *% (SE)*				
Current smoker		27.1 ± 0.7[a]	25.7 ± 0.7[a]	19.4 ± 0.7[b]
Former smoker		15.7 ± 0.6[b]	16.4 ± 0.6[b]	19.4 ± 0.7[b]
Never smoker		57.2 ± 0.8[b]	57.9 ± 0.8[b]	61.1 ± 0.8[b]
Family status,[2] *% (SE)*				
Married, no children		20.0 (0.6)[a]	17.3 (0.6)[b]	18.8 (0.7)[a]
Single, no children		31.3 (0.7)[a]	28.0 (0.7)[b]	23.8 (0.7)[b]
Married, with children		37.0 (0.8)[b]	42.7 (0.8)[a]	43.7 (0.8)[a]
Single, with children		11.7 (0.5)[b]	12.0 (0.5)[b]	13.7 (0.6)[a]
Anthropometrics				
BMI,[2] *% (SE)*		26.8 (6.1)[c]	27.5 (6.5)[b]	29.5 (7.2)[a]
BMI <25.0 kg/m^2		44.6 (0.8)[a]	39.6 (0.8)[b]	27.8 (0.8)[c]
BMI 25.0–29.9 kg/m^2		30.3 (0.7)[b]	31.9 (0.7)[b]	33.2 (0.8)[a]
BMI ≥30 kg/m^2		23.2 (0.7)[c]	26.5 (0.7)[b]	38.1 (0.8)[a]
Waist circumference, c*m*		84.0 ± 14.1[c]	85.9 ± 14.6[b]	91.9 ±15.6[a]
Plasma biomarkers				
Glucose,[3] *mg/dL*		90.1 ± 19.4[b]	86.0 ± 8.6[c]	93.2 ± 10.0[a]
HOMA-IR		3.5 ± 5.6[a]	3.1 ± 2.3[b]	3.4 ± 2.4[a,b]
TG, *mg/dL*		86.4 ± 75.7[c]	91.5 ± 73.5[b]	107.3 ± 78.2[a]
LDL-C, *mg/dL*		107.6 ± 31.6[c]	109.0 ± 31.8[b]	110.9 ± 32.0[a]
HDL-C, *mg/dL*		52.1 ± 14.2[b]	50.2 ± 14.0[c]	54.8 ± 16.8[a]
PA and sedentary behavior[2]				
Activity, *EU/wk*		338 ± 274	331 ± 275	336 ± 27
Television viewing, *h/d*		2.6 ± 1.8	2.5 ± 2.0	2.6 ± 2.3

[1] Values are means ± SD or % (SE), n = 3643. Values in a row with superscripts without a common letter differ, $P < 0.05$.
[2] Data are self-reported and may reflect differences in reporting as well as consumption away-from-home eating) or participation (PA and sedentary behavior).
[3] To convert mg/dL to mmol/L, multiply by 0.055, 0.0113, and 0.0259 for glucose, TG, and cholesterol, respectively.
[4] Cells are missing values because Exam Year 7 is the year used to calculate change at Exam Year 10. There is no change at Exam Year 7.
[5] Total energy intake was not measured at Exam Year 10.

knowledge, data with this level of detail does not exist in any longitudinal dataset (information on the location and type of food consumed), although they would prove useful in considering potential public health measures to address the health effects of frequent away-from-home consumption.

Further limitations of this study include self-reported away-from-home eating and other lifestyle factors, which may bias our results toward (28) or away from the null (29), potential residual confounding by unobserved, time-variant factors external to the individual, and an inability to differentiate between persons who

TABLE 2 Comparison of estimated associations of baseline quartile of fast food and restaurant food consumption with y 20 outcomes (dependent variable) in black and white young adults[1]

y 20 Outcomes	*n*	Quartile fast food consumption				Quartile restaurant food consumption			
		1	2	3	4	1	2	3	4
		Frequency, n/wk							
		0 to <0.5	0.5 to 1.2	1.2 to <2.5	≥2.5	0 to <0.7	0.7 to <1.5	1.5 to <3	≥ 3
Weight, *kg*	1633	83.1 ± 1.4	86.3 ± 1.0	86.7 ± 1.0*	88.9 ± 0.9*	87.0 ± 1.1	85.7 ± 1.1	87.7 ± 1.0	86.7 ± 1.0
HOMA-IR score	1510	3.0 ± 0.18	3.3 ± 0.12	3.6 ± 0.13*	3.9 ± 0.14*	3.7 ± 0.14	3.3 ± 0.14	3.5 ± 0.12	3.5 ± 0.13
Waist circumference, *cm*	1637	89.0 ± 1.0	91.8 ± 0.7*	93.2 ± 0.7*	94.4 ± 0.7*	93.5 ± 0.8	91.6 ± 0.8	92.8 ± 0.7	92.5 ± 0.7
TrG,[2] *mg/dL*	1473	94.8 ± 5.4	104.2 ± 3.9	113.5 ± 3.9*	117.5 ± 3.6*	111.8 ± 4.3	108.1 ± 4.2	107.3 ± 3.7	111.7 ± 4.0
LDL-C, *mg/dL*	1458	111.4 ± 2.3	111.8 ± 1.7	111.3 ± 1.7	113.4 ± 1.6	111.9 ± 1.8	111.3 ± 1.8	111.5 ± 1.6	113.8 ± 1.7
HDL-C, *mg/dL*	1473	57.5 ± 1.1	52.6 ± 0.8*	53.3 ± 0.8*	52.0 ± 0.7*	52.8 ± 0.9	53.7 ± 0.9	53.4 ± 0.8	53.2 ± 0.8

[1] Values are predicted mean y 20 outcomes ± (SE) obtained using β coefficients from ordinary least squares regression models of y 20 outcome (continuous) comparing quartile of weekly fast food and restaurant food consumption [mean of exam y 7 and 10, quartile 1 (referent)], adjusting for age [32–34, 35–37, ≥38 y vs. <32 y (referent)], race [black vs. white (referent)], gender, education [less than high school, more than high school vs. high school/GED (referent)], family structure [married, married with children, single vs. single with children (referent)], CARDIA study center [Birmingham, Chicago, and Minneapolis vs. Oakland (referent)], PA [≥474 EU/wk vs. <474 EU/wk (referent)], television viewing (h/d, continuous), y 7 total energy (continuous), smoking status [current, former vs. never (referent)], and change in fast food and in restaurant food consumption (exam y 20 minus exam y 10). All models also include the exam y 7 measure of the outcome variable. Baseline intake is defined as the mean of exam y 7 and 10. *Different from referent group (quartile 1), $P < 0.05$.

[2] To convert mg/dL to mmol/L, multiply by 0.0113 for TG and by 0.0259 for cholesterol.

consumed healthier compared with less healthy meals at various fast food locations or persons who regularly consumed from fast food restaurants where healthier food options were available from those places where such offerings are not present.

The strengths associated with longitudinal analyses and our ability to rule out structural confounding as an explanation of our results. Note the considerable overlap in persons who are consumers of both fast food and restaurant food at each exam year [7 (82%), 10 (74%), and 20 (74%)] are advantages of this study.

To our knowledge, this is the first study to show that away-from-home food consumption is adversely associated with metabolic health outcomes, namely TG and HDL-C concentrations, and to report important differences between restaurant and fast food intake. Future research should examine differences in the types of individual foods that are available and consumed at restaurants compared with fast food places and consider programs and policies to improve healthfulness of fast food outlets.

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TABLE 3 Estimated longitudinal associations of weekly fast food and restaurant consumption with 13-y change in outcomes in black and white young adults[1]

Outcome changed over 13 y			Fast food		Restaurant food	
			Model 1[3]	Model 2[4]	Model 1[3]	Model 2[4]
	n	Obs[2]	$\beta \pm SE$			
Weight	3987	8489	0.15 ± 0.05*	0.15 ± 0.05*	0.09 ± 0.04	0.09 ± 0.04
HOMA-IR score	3873	7921	0.02 ± 0.01	0.02 ± 0.01	0.007 ± 0.01	0.01 ± 0.01
Waist circumference	3982	8472	0.13 ± 0.04*	0.12 ± 0.04	0.07 ± 0.03	0.08 ± 0.03
TG	3926	8152	0.24 ± 0.40	0.21 ± 0.40	0.18 ± 0.31	0.23 ± 0.31
LDL-C	3450	7466	0.16 ± 0.14	0.16 ± 0.14	−0.01 ± 0.11	0.004 ± 0.11
HDL-C	3926	8152	0.08 ± 0.06	0.09 ± 0.06	0.07 ± 0.05	0.06 ± 0.05

[1] Values are β coefficients ± SE, representing the change in outcome associated with the change in the frequency of consuming restaurant or fast food per week. Symbols indicate that coefficients differ from zero, *$P < 0.001$; †$P < 0.05$.

[2] Derived from number of observations (Obs) across 3 time periods (exam y 7, 10, and 20) per person (*n*). Values range from 1 to 3, with a mean of 2.1 observations/person.

[3] Model 1 is a repeated-measures, conditional longitudinal model of outcome (continuous) on weekly fast food and restaurant food consumption (continuous), adjusting for the time-variant factors age (continuous), education [less than high school, more than high school vs. high school/GED (referent)], and family structure [married, married with children, single vs. single with children (referent)].

[4] Model 2 is model 1 plus the time-variant lifestyle factors PA (EU/wk, continuous), television viewing (h/d, continuous), total energy (continuous), and smoking status [current, former vs. never (referent)].

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