# Research Brief

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## Fast Food Intake, Weight Gain, and Insulin Resistance in Young Adult African Americans and Caucasians

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Because of its rapid development among genetically stable populations, the obesity epidemic can be attributed to environmental factors affecting diet or physical activity level. One potentially important dietary factor may be consumption of "fast food," which can be defined as "convenience food purchased in self-service or carry-out eating places without wait service." From its origins in the 1950s, fast food has grown into a dominant dietary pattern, with a quarter million fast food restaurants in the United States. Consumption of fast food by children has increased from 2 percent of total energy in the late 1970s to 10 percent of energy in the 1990s.

A number of factors inherent to fast food may promote a positive energy balance and thereby increase risk for obesity and diabetes, including: excessive portion size, with single large meals often approaching or exceeding individual daily energy requirement; palatability, emphasizing primordial taste preferences for sugar, salt, and fat; high energy density; and high glycemic load. Dietary factors such as trans-fatty acids and high glycemic load may also increase risk for diabetes through energy-independent mechanisms.

However, surprisingly few studies have examined the effects of fast food consumption on energy balance or body weight, and most of these studies are of cross-sectional design. To our knowledge, there are no data regarding fast food consumption and diabetes-related endpoints. For these reasons, we examined the association between reported fast food habits and changes in body weight and insulin resistance over a 15-year period among young black and white adults in the United States.

Subjects for this study included 3031 young (ages 18 to 30 years in 1985/6) black and white adult participants in the Coronary Artery Risk Development in Young Adults (CARDIA) study. Participants were followed for 15 years with repeated dietary assessment. Fast food habits were assessed by the question: "How often do you eat breakfast, lunch, or dinner at places such as McDonald's, Burger King, Wendy's, Arby's, Pizza Hut, or Kentucky Fried Chicken?" Responses were recorded to the nearest frequency per week on a semi-continuous scale. We used multiple linear regression models to examine the associations of frequency of fast food restaurant visits at baseline and follow-up with 15-year changes in body weight and the homeostasis model for insulin resistance.

Fast food frequency was lowest for white women (mean ~ 1·3 times / week) compared to the other race-sex groups (mean ~ 2 times / week). After adjustment for many other lifestyle factors, baseline fast food frequency was directly associated with changes in body weight in both blacks ($p<.01$) and whites ($p<.01$). Change in fast food frequency over 15 years was directly associated with changes in body weight in whites ($p<.0001$), with a weaker association observed in blacks ($p=.10$). Changes in fast food were also directly associated with changes in insulin resistance in both blacks and whites ($p<.01$). In comparison to the average 15-yr weight gain among participants with infrequent (< 1 time per week) fast food restaurant use at baseline and follow-up (n=203), those with frequent (>2 times per week) fast food restaurant use at both baseline and follow-up (n=87) gained an extra 10 pounds of body weight ($p=.01$) and had a 104 perecnt greater increase in insulin resistance ($p<.01$) (See Figures 1 and 2). The associations appeared to be largely independent of other potentially confounding lifestyle factors, such as physical activity and television viewing.

The associations between fast food and insulin resistance were not fully explained by adjustment for a large number of other lifestyle factors, energy intake, nutrients, and food groups, raising the possibility that some unmeasured factor inherent to fast food may be mediating this effect. The single most obvious aspect of fast food that might lead to weight gain is the large

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portion size, with certain single-meal calorie levels approaching total daily energy intake requirements. Independent of energy intake, many other aspects of fast food, including direct effects of energy density on passive overconsumption, may also predispose to insulin resistance. Certain fast foods contain large amounts of partially hydrogenated oils, and this class of fatty acids may cause insulin resistance and increase risk of type 2 diabetes. Fast food also contains large amounts of highly refined starchy food and added sugar, carbohydrates that have been characterized as high in glycemic index. Consumption of a high glycemic index or high glycemic load diet has been linked to risk for diabetes, independent of body weight changes, in mechanistic studies, short term human trials, and observational studies, though this point remains controversial.

The strengths of our study include: long-term, prospective study design, with high rates of follow-up over 15 years; standardized, repeated, and detailed measurements of dietary practices; direct assessment of specific fast food habits, including changes in fast food; extensive data on covariates with which to explore confounders and mediators of the associations under investigation; and the demographics of the cohort – young adult black and white men and women from four U.S. metropolitan areas who have been examined during a period of life when substantial weight gain occurs and chronic diseases evolve.

Limitations of this study relate primarily to its observational nature, with the possibility of residual confounding precluding definitive conclusions about causality, and to reliance on self-reported diet and other lifestyle factors. Our conclusions are limited to frequency of fast food restaurant use because we were unable to analyze sufficiently the considerable range of available fast food items and their portion sizes. Nonetheless, it is quite possible that our results underestimate the true magnitude of the association due to measurement error and because of thorough adjustment for many covariates that may be on the causal pathway (e.g., food groups and nutrients). Measurement error, if non-differential, would tend to result in attenuated estimates of the strength of association between fast food intake and the several outcome variables. Similarly, analyses of changes are limited by floor and ceiling effects in that some people never eat fast food (so cannot decrease) or eat most meals at fast food restaurants (so cannot increase). These problems would also tend to attenuate observed associations compared to the true relation (such as would be observed in a clinical trial of investigator-controlled increase or decrease in fast food intake).

In conclusion, fast food habits have strong, positive and independent associations with weight gain and insulin resistance among young black and white adults. Fast food consumption can be linked to adverse health outcomes through plausible mechanisms, and results from other studies provide support for our findings. In view of the high and increasing rates of fast food consumption, further research into the effects of this dietary pattern on public health should be given priority.

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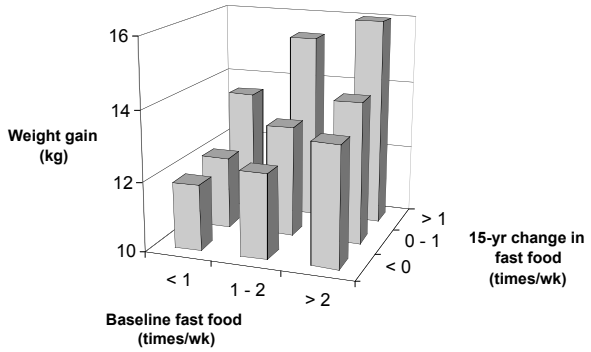


Figure 1. Joint association of year 0 fast food frequency and 15-year changes in fast food frequency with 15-year changes in body weight in the CARDIA Study, 1985-2000. The model is adjusted for the same covariates as in Model 2 of Table 3, with race as an additional covariate. Cell-specific sample sizes range from 87 (> 2 times per week at baseline and > 1 times / week change) to 730 (> 2 times per week at baseline and < 0 times / week change).

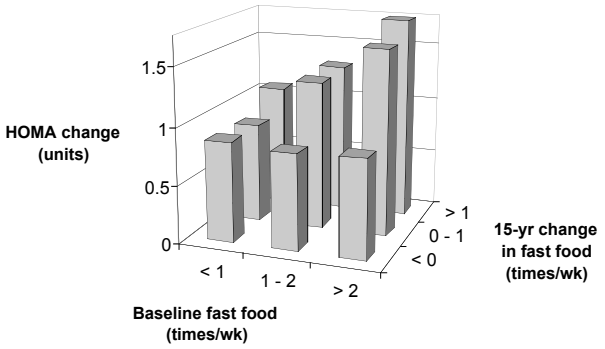


Figure 2. Joint association of year 0 fast food frequency and 15-year changes in fast food frequency with 15-year changes in HOMA insulin resistance in the CARDIA Study, 1985-2000. The model is adjusted for the same covariates as in Model 2 of Table 4, with race as an additional covariate. Cell-specific sample sizes range from 79 (> 2 times per week at baseline and > 1 times / week change) to 672 (> 2 times per week at baseline and < 0 times / week change).

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