Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis

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Summary

Background Fast-food consumption has increased greatly in the USA during the past three decades. However, the effect of fast food on risk of obesity and type 2 diabetes has received little attention. We aimed to investigate the association between reported fast-food habits and changes in bodyweight and insulin resistance over a 15-year period in the USA.

Methods Participants for the CARDIA study included 3031 young (age 18–30 years in 1985–86) black and white adults who were followed up with repeated dietary assessment. We used multiple linear regression models to investigate the association of frequency of fast-food restaurant visits (fast-food frequency) at baseline and follow-up with 15-year changes in bodyweight and the homoeostasis model (HOMA) for insulin resistance.

Findings Fast-food frequency was lowest for white women (about 1·3 times per week) compared with the other ethnic-sex groups (about twice a week). After adjustment for lifestyle factors, baseline fast-food frequency was directly associated with changes in bodyweight in both black (p=0·0050) and white people (p=0·0013). Change in fast-food frequency over 15 years was directly associated with changes in bodyweight in white individuals (p<0·0001), with a weaker association recorded in black people (p=0·1004). Changes were also directly associated with insulin resistance in both ethnic groups (p=0·0015 in black people, p<0·0001 in white people). By comparison with the average 15-year weight gain in participants with infrequent (less than once a week) fast-food restaurant use at baseline and follow-up (n=203), those with frequent (more than twice a week) visits to fast-food restaurants at baseline and follow-up (n=87) gained an extra 4·5 kg of bodyweight (p=0·0054) and had a two-fold greater increase in insulin resistance (p=0·0083).

Interpretation Fast-food consumption has strong positive associations with weight gain and insulin resistance, suggesting that fast food increases the risk of obesity and type 2 diabetes.

Introduction

The frequency of obesity has risen at an alarming rate in all age and ethnic groups in the USA.[1,2] The age-adjusted prevalence of obesity, defined as a body-mass index (BMI) of 30 kg/m² or greater, was 30·5% in 1999–2000 compared with 22·9% in 1988–1994, with even higher rates in ethnic minority groups.[1] About two of every three US adults and four of five African-American women were overweight or obese in 1999–2000.[1] In children and adolescents, the prevalence of being overweight rose by 50% in the past decade to about 15%.[2]

The medical and economic outcomes of excessive bodyweight are great, including an estimated 300 000 excess deaths and at least US$100 billion per year in medical expenditures.[3–6] One particularly ominous public-health issue is the occurrence of glucose intolerance[7] and type 2 diabetes in obese adolescents and young adults.[8]

Because of its rapid development in genetically stable populations, the obesity epidemic can be attributed to environmental factors affecting diet, or physical activity level. One potentially important dietary factor is consumption of fast food, which can be defined as convenience food purchased in self-service or carry-out eating places.[9,10] From its origins in the 1950s, fast food

has grown into a dominant dietary pattern, with a current estimate of about 247 115 restaurants in the USA.[11] Consumption of fast food by children has risen from 2% of total energy in the late 1970s to 10% of energy in the 1990s.[12]

Several factors inherent to fast food as it now exists could promote a positive energy balance[11,13] 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, emphasising primordial taste preferences for sugar, salt, and fat; high energy density;[14] and high glycaemic load. Several dietary factors such as trans-fatty acids[15] and high glycaemic load[16] might also enhance risk for diabetes through energy-independent mechanisms.

Surprisingly few studies have investigated the effects of fast-food consumption on energy balance or body-weight,[17–20] and most of these are of cross-sectional design. To our knowledge, no data for fast-food consumption and diabetes-related endpoints are available. For these reasons, we aimed to investigate the association between reported fast-food habits and changes in bodyweight and insulin resistance over a 15-year period in young black and white adults in the USA.

Methods

The Coronary Artery Risk Development in Young Adults (CARDIA) study is a multicentre, population-based prospective study of cardiovascular disease risk factor evolution in a US cohort of African-American and white young adults. The four study centres are Birmingham, AL, Chicago, IL, Minneapolis, MN, and Oakland, CA.

Participants

We used recruitment stratification to obtain nearly equal numbers of participants who were black and white, young (18–24 years) and old (25–30 years), and with more (high school or more) and less (less than high school) education. Participants were followed up for 15 years and had six clinical examinations: in 1985–86 (baseline or year 0), 1987–88 (year 2), 1990–91 (year 5), 1992–93 (year 7), 1995–96 (year 10), and 2000–01 (year 15). More details of the CARDIA study design and participants have been previously reported.[21]

We excluded participants from our analysis for the following reasons: did not come to the year 15 examination; missing data for fast food, bodyweight, or important covariates at baseline or follow-up; female participants who were pregnant at baseline or within 180 days of year 15, or were breastfeeding; suspected type 1 diabetes based on insulin treatment; and for the insulin resistance analysis only, participants fasting for fewer than 8 h at year 0 or year 15. Some participants belonged to more than one of the above categories.

Procedures

We used standard questionnaires to maintain consistency in the assessment of demographics (age, sex, ethnic origin, and education) and behavioural information across CARDIA examination visits. The CARDIA physical activity history questionnaire[22] queries the amount of time per week spent in leisure, occupational, and household physical activities over the past 12 months. We estimated total physical activity—expressed in exercise units as a product of intensity×frequency—at every clinical examination. Television watching was calculated as hours per week and was reported at the examinations at years 5, 10, and 15. We quantified education as the number of years of school completed at every examination and cigarette smoking status as current smoker, former, or never-smoker at every examination.

For dietary assessment, we did a structured interview at every CARDIA examination and included a series of questions on dietary practices, including food preparation, and where meals were typically eaten. Only the year 2 examination did not include fast-food habits. We quantified fast-food habits based on responses to 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 (fast-food frequency) on a semicontinuous scale and classified as less than 1, 1–2, or greater than 2.

To assess possible confounding or mediation, we also used dietary data obtained at year 0 and 7 from the CARDIA diet history,[23] which queried usual dietary practices and obtained a quantitative food frequency of the past month. Liu and colleagues[24] reported on the reliability and validity of the CARDIA diet history in 128 young adults. As an internal validation of the fast-food frequency question, we identified all foods that might have been obtained at fast-food restaurants—eg, "double cheeseburger", "chicken nuggets", and "french fries". Frequency of consumption for every food item (times per week) was used to estimate relative intake per week for every food. We also considered intake of other food groups and nutrients that could confound associations or serve as mechanisms linking fast-food intake with weight gain and insulin resistance, including intake (times per week) of fruit, non-starchy vegetables, dairy, soda and sugar-sweetened beverages, whole and refined grains, total energy (kcal/day), alcohol (mL/day), fibre (g/1000 kcal per day), trans-fatty acids (g/day), and percentage of energy from animal and vegetable protein and total, saturated, unsaturated, and trans-fatty acids.

As an additional approach to adjusting for the confounding effects of correlated lifestyle habits, we calculated a healthy lifestyle score, modified from our previously used measure.[25] We created binary variables based on smoking status (current smoker [scored 0], non-smoker [scored 1]) and median cutpoints for physical activity (low [0], high [1]), television viewing (low [1], high [0]), saturated fat intake (low [1], high [0]), wholegrain intake (low [0], high [1]), fruit and non-starchy vegetable intake (low [0], high [1]), low-fat dairy intake (low [0], high [1]), and soft drink intake (low [1], high [0]). We summed these binary scores into the healthy lifestyle score with a range from 0, indicating a very unhealthful lifestyle, to 8, indicating a very healthful lifestyle.

We undertook all clinical procedures in accordance with the CARDIA study manual of operations. For anthropometric measures, participants were standing and dressed in light clothing without shoes. We measured bodyweight to the nearest 0·2 kg with a calibrated balance beam scale, height with a vertical ruler to the nearest 0·5 cm, and waist size with a tape in duplicate to the nearest 0·5 cm around the minimum abdominal girth. BMI was calculated as weight in kg divided by height in m^2.

Before every CARDIA examination, we asked participants to fast and to avoid smoking and heavy physical activity for the final 2 h. For measurement of insulin and glucose concentrations, we drew blood into vacuum tubes containing no preservative. We separated serum by centrifugation at 4°C within 60 min, stored it in cryovials, and froze it at –70°C within 90 min until laboratory analysis. The radioimmunoassay for insulin

required an overnight, equilibrium incubation and used a unique antibody that has less than 0·2% crossreactivity to human proinsulin and its primary circulating split form Des 31,32 proinsulin (Linco Research, St Louis, MO, USA). Masked analysis of split serum samples resulted in a technical error of 16·6% of the mean ($r=0·98$). We measured fasting glucose by the hexokinase method at every examination. The homoeostasis model (HOMA) for insulin resistance was calculated as glucose (mmol/L)×insulin (mU/L)/22·5.[26]

Statistical analysis

We did all analyses with SAS statistical software version 9 (SAS, Cary, NC, USA). To assess changes in fast-food intake over time, and the effects of age, time, and secular trends on fast-food intake, we used repeated measures regression analysis (PROC MIXED). We used general linear models (PROC GLM) to calculate adjusted means of demographic and lifestyle factors according to category of fast-food intake and to analyse the relation between the independent variables (baseline fast-food frequency and 15-year change in fast-food frequency) with changes in the dependent variables (bodyweight and HOMA insulin resistance) over the 15-year follow-up period. For these analyses, 15-year change in fast food was calculated as the baseline fast-food frequency subtracted from the value of fast-food frequency at the final follow-up examination at year 15. We modelled many potential confounding or mediating factors as their baseline value and their change over time, when available.

Ethnic origin-specific multivariable linear regression models were constructed as follows. Model 1 included demographic covariates (sex, age [continuous years], centre, and education [continuous years]) and the respective baseline value [continuous] of the dependent variable (baseline bodyweight and height when modelling weight change, and baseline HOMA insulin resistance when modelling HOMA). Model 2 further included non-dietary lifestyle covariates (alcohol consumption [mL/day at baseline and year 15], smoking status [never, former, current at baseline and year 15], physical activity [units per day at baseline and 15-year change], and television viewing [h/day at year 10 and change between year 10 and year 15]). In model 3, we added baseline values and changes in dietary factors, including total caloric intake, dietary fibre (g/1000 kcal at year 7 only), percentage of

calories from saturated fatty acids, unsaturated fatty acids, trans-fatty acids (g/day), and daily intake of soft drinks, refined grains, wholegrains, low-fat and high-fat dairy products, fruits, non-starchy vegetables, meat, and fish. We also examined interactions between fast-food frequency and ethnic origin, sex, and baseline overweight status (BMI ≥25 kg/m²).

Role of the funding source

The sponsors of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

5115 people attended the baseline examination, with 74% retention of the surviving cohort in year 15. Those who were not in the 15-year cohort were younger (mean 24·2 years [SD 3·7] vs 25·1 years [3·6], p<0·0001) and more likely to be black (62·9% vs 47·1%, p<0·0001), men (49·1% vs 44·1%, p=0·0014), and have less education (61·0% vs 80·9%, p<0·0001) at baseline, but they did not differ by BMI (mean 24·4 kg/m² [SD 5·2] vs 24·5 kg/m² [5·0], p=0·2287) or frequency of fast-food visits (2·1 times a week [2·3] vs 1·9 times a week [2·2], p=0·1450) at baseline. After exclusion of 1443 people who did not come to the year 15 examination, 492 who had missing data, 223 who were pregnant or breastfeeding, 17 with suspected type 1 diabetes, and 419 who fasted fewer than 8 h, 3031 participants could be included in the bodyweight analysis and 2767 in the insulin resistance analysis.

Age-adjusted fast-food frequency was relatively stable over time in black people but fell in those who were white (p<0·0001 for ethnic origin-time interaction; table 1). Fast-food frequency was higher in black than in white people (p<0·0001) and in men than in women (p<0·0001) for every examination year. The reported fast-food frequency in white women was notably low (table 1).

In both ethnic groups, fast-food frequency rose over time in those with a low baseline frequency (<1 time per week), whereas in individuals with high baseline frequency it fell over time (table 2). The strong positive association between fast-food frequency and consumption of individual fast-food items, as reported during the diet history interview ("Foods typical of fast food"), lends support to the internal validity of the fast-food frequency data. Individuals with high fast-food frequency were younger than those with low frequency (table 2). No association was seen between year 0 fast-food frequency and year 0 bodyweight or HOMA insulin resistance in either black or white people. Year 0 associations noted between fast-food habits and lifestyle factors seemed to be stronger for white people than for black individuals, with the exception of energy intake, which was directly associated with fast-food frequency in both populations.

	1985–86	2000–01
Black men (n=647)	2·4 (0·09)	2·3 (0·09)
Black women (n=797)	1·8 (0·08)	2·0 (0·08)
White men (n=804)	2·4 (0·07)	1·9 (0·08)
White women (n=783)	1·6 (0·08)	1·3 (0·08)

Data are mean (SE) and are adjusted for age and study centre. Time, race, sex, and time×race×sex differences are significant (p<0·0001).

Table 1: Reported frequency of fast food restaurant visits (times per week) by race, sex, and time in the CARDIA study

	Black people			p*	White people			p*
	<1 (n=450)	1–2 (n=508)	>2 (n=486)		<1 (n=625)	1–2 (n=517)	>2 (n=445)	
Year 0 fast-food frequency (times per week)	0·4	1·4	4·6	<0·0001	0·4	1·4	4·8	<0·0001
15-year change in fast food (times per week)	1·4	0·7	–1·8	<0·0001	0·7	0·1	–2·5	<0·0001
Foods typical of fast food (times per week)†	1·5	2·4	4·3	<0·0001	0·8	1·7	3·6	<0·0001
Age (years)	25·0	24·4	24·0	<0·0001	26·1	25·5	25·1	<0·0001
Women (%)	61·7	56·6	47·7	<0·0001	59·5	46·8	37·9	<0·0001
Bodyweight (kg)‡	72·4	73·5	72·9	0·6506	69·8	71·5	70·8	0·1598
HOMA insulin resistance score	2·4	2·4	2·6	0·1387	1·8	2·0	1·9	0·3277
Healthy lifestyle score	3·6	3·7	3·5	0·2257	4·9	4·6	4·1	<0·0001
Education (years)	13·8	13·9	14·0	0·1723	16·0	15·5	15·5	0·0011
Physical activity (exercise units)	360	389	394	0·0713	480	454	440	0·0298
Television (h/day)§	3·2	3·1	3·1	0·5334	1·6	1·7	1·9	0·0003
Current smokers (%)	36·2	27·7	29·5	0·0256	20·1	23·6	30·7	0·0002
Alcohol intake (mL/day)	12·3	9·9	11·7	0·7304	12·1	11·9	17·6	<0·0001
Total energy intake (kcal/day)	2829	3169	3485	<0·0001	2451	2694	2978	<0·0001
Total fat (g/day)	110·1	123·2	138·7	<0·0001	91·5	103·9	117·7	<0·0001
Total fat (% energy)	34·3	34·7	35·6	0·0006	33·2	34·5	35·4	<0·0001
Saturated fat (% energy)	13·8	14·1	14·4	0·0011	13·6	14·1	14·5	<0·0001
Trans fat (g/day)	4·2	4·3	4·8	0·0192	3·3	3·5	3·5	0·1316
Soft drink intake (times per week)	6·2	6·9	8·6	<0·0001	2·5	4·3	6·4	<0·0001
Meat intake (times per week)	10·6	12·3	13·8	<0·0001	6·8	8·9	10·8	<0·0001
Refined grain intake (times per week)	23·4	26·9	26·5	0·0114	18·9	20·0	22·1	<0·0001
Wholegrain intake (times per week)	7·7	8·3	8·7	0·1242	11·9	10·5	8·6	<0·0001
Fruit and non-starchy vegetable intake (times per week)	14·2	15·1	15·2	0·9386	22·5	19·1	16·3	<0·0001
Fibre intake (g/1000 kcal per day)¶	7·6	7·5	6·8	<0·0001	10·2	8·7	8·2	<0·0001
Reduced-fat dairy intake (times per week)	3·6	3·9	3·5	0·8452	11·2	10·5	9·6	0·0286

Data are mean values, and are adjusted for age, sex, and study centre. Data are from year 0 except for those noted. *Highest compared with lowest fast food category. †Sum of french fries, hamburgers, breakfast items, and chicken items reported during the CARDIA diet history interview, which may have been obtained at fast-food restaurants. ‡Bodyweight was also adjusted for height. §Measured at year 5. ¶Measured at year 7.

Table 2: **Adjusted demographic and dietary factors by frequency of fast-food restaurant visits at year 0 (1985–86)**

The healthy lifestyle score was strongly inversely associated with fast-food intake in white but not black people (table 2). By comparison with individuals in the low fast-food category, those in the high category had higher year 0 intakes of total energy, total fat, saturated fatty acids, soft drinks, refined grains, and meat, and lower intakes of dietary fibre. An inverse association between fast-food frequency and cigarette smoking was noted in black people, whereas a direct association was recorded in white individuals (table 2). For black, but not white, people, high fast-food frequency was associated with high intake of trans-fatty acids. For white but not black individuals, by comparison with those in the low fast-food category, those in the high category had fewer years of education, had lower physical activity levels, watched more hours of television, consumed more alcohol, and had a lower intake of wholegrains, fruit and non-starchy vegetables, and reduced fat dairy products.

Table 3 includes the adjusted mean 15-year changes in bodyweight associated with differences in year 0 fast-food frequency and 15-year change in fast-food frequency of three times per week. Year 0 and change in fast-food frequency were modeled simultaneously. A difference of three times per week was chosen because, as table 2 shows, this difference was close to the mean between individuals with low versus high fast-food frequency at baseline and also approximated the mean range in change over time in these two baseline

categories. Year 0 fast-food frequency was associated with a rise in bodyweight in both ethnic groups, independent of many other lifestyle factors. Even after thorough adjustment for many possibly confounding or mediating dietary factors in model 3, a difference in year 0 fast-food frequency of three times per week was associated with mean weight gains of 2·2 kg (SE 0·72) in black people (p=0·0014) and 1·6 kg (0·55) in white people (p=0·0064). Change in fast-food frequency over

	Fast-food variable	Black people		White people	
		b (SE)	p	b (SE)	p
Model 1	Baseline	1·44 (0·58)	0·0126	2·68 (0·47)	<0·0001
	Change	0·70 (0·40)	0·0774	2·67 (0·41)	<0·0001
Model 2	Baseline	1·72 (0·61)	0·0050	1·84 (0·50)	0·0013
	Change	0·63 (0·42)	0·1004	1·97 (0·42)	<0·0001
Model 3	Baseline	2·22 (0·72)	0·0014	1·56 (0·55)	0·0064
	Change	0·74 (0·45)	0·1053	1·84 (0·44)	<0·0001

Model 1 includes the following covariates: age (continuous years), study centre, sex, education (highest attained, continuous years), baseline bodyweight (continuous), and height (continuous). Model 2 contains the extra covariates: cigarette smoking status (never, former, current, at baseline and year change), alcohol intake (mL/day, at baseline and 15-year change), physical activity (units per day, at baseline and 15-year change), television viewing (h/day, at year 10 and change between years 10 and 15). Model 3 contains the covariates in model 2 and dietary intake (at baseline and change between baseline and year 7) of total energy (kcal/day), saturated fatty acids (% kcal), unsaturated fatty acids (% kcal), and trans-fatty acids (g/day), fibre (g/1000 kcal at year 7 only), and daily servings of fruits, non-starchy vegetables, wholegrains, refined grains, soft drinks, meat, dairy, and fish.

Table 3: **Mean adjusted 15-year changes in bodyweight by three times per week year 0 differences in fast food frequency between participants, and by three times per week 15-year change in fast food frequency within participants**

	Fast-food variable	Black people		White people	
		b (SE)	p	b (SE)	p
Model 1	Baseline	0·09 (0·14)	0·5011	0·32 (0·10)	0·0008
	Change	0·26 (0·10)	0·0061	0·37 (0·08)	<0·0001
Model 2	Baseline	0·22 (0·14)	0·0688	0·17 (0·10)	0·0056
	Change	0·29 (0·09)	0·0015	0·28 (0·09)	<0·0001
Model 3	Baseline	0·13 (0·16)	0·4112	0·04 (0·11)	0·7596
	Change	0·32 (0·10)	0·0021	0·23 (0·09)	0·0127

Model 1 includes the following covariates: age (continuous years), study centre, sex, education (highest attained, continuous years), baseline HOMA insulin resistance (continuous). Model 2 contains the extra covariates: cigarette smoking status (never, former, current, at baseline and year 15), alcohol intake (mL/day, at baseline and 15-year change), physical activity (units per day, at baseline and 15-year change), television viewing (h/day, at year 10 and change between years 10 and 15). Model 3 contains the covariates in model 2 and dietary intake (at baseline and change between baseline and year 7) of total energy (kcal/day), saturated fatty acids (% kcal), unsaturated fatty acids (% kcal), and trans-fatty acids (g/day), fibre (g/1000 kcal at year 7 only), and daily servings of fruits, non-starchy vegetables, wholegrains, refined grains, soft drinks, meat, dairy, and fish.

Table 4: Mean adjusted 15-year changes in HOMA insulin resistance by three times per week year 0 differences in fast food frequency between participants, and by three times per week 15-year change in fast food frequency within participants

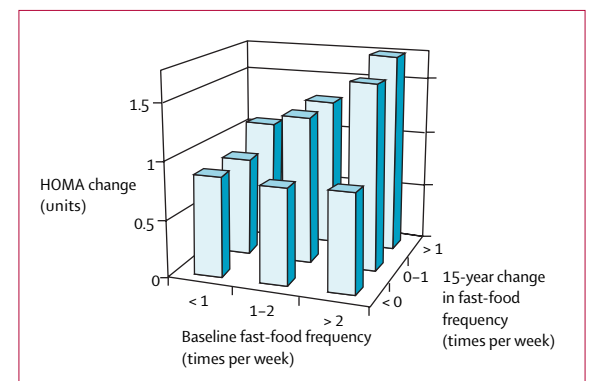*Figure 1:* Joint association of year 0 fast-food frequency and 15-year changes in fast-food frequency with 15-year changes in bodyweight
The model is adjusted for the same covariates as in model 2 of table 3, with ethnic origin as an additional covariate. Cell-specific sample sizes range from 87 (>2 times per week at baseline and >1 times per week change) to 730 (>2 times per week at baseline and <0 times per week change).

15 years was also independently associated with changes in bodyweight in white people (1·8 kg [SE 0·44], p<0·0001), with a weaker association recorded in black individuals (0·7 kg [0·45], p=0·1053). The association between fast-food change and weight change was stronger for white than black people (p=0·0075 for interaction). Interaction terms between fast-food frequency and year 0 BMI or sex were not significant.

As table 4 shows, the association between year 0 fast-food intake and change in insulin resistance in black and white individuals that was noted in model 2 was no longer apparent in model 3 after adjustment for many dietary factors that may be either confounders or on the causal pathway. However, change in fast food was directly associated with changes in insulin resistance in both black and white people even after thorough adjustment in model 3. Interaction terms between fast-food frequency and ethnic origin or sex were not significant. In white people only, we noted somewhat stronger associations between fast-food habits and changes in insulin resistance among those who were overweight at baseline compared with those who were normal weight at baseline (p=0·0121 for interaction between baseline fast food and baseline overweight status, and p=0·0042 for interaction between change in fast food and baseline overweight status).

Adjustment for total, rather than saturated, fat intake did not materially change these findings for either endpoint. Percentage of energy from total fat and saturated fat was not associated with weight gain or insulin resistance in model 3 (p value range 0·14–0·88). Further adjustment for the healthy lifestyle index did not materially alter the findings (data not shown).

Figures 1 and 2 depict the joint associations of year 0 fast-food frequency (p=0·0389 for weight change and p=0·0031 for insulin resistance) and 15-year changes in fast-food frequency (p=0·0030 for weight change and p=0·0001 for insulin resistance) with weight gain and
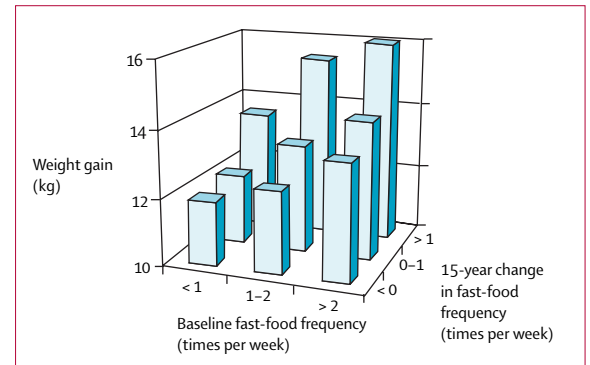
changes in insulin resistance. We recorded direct and independent monotonic associations between both year 0 and 15-year change in fast-food frequency and 15-year weight gain and changes in insulin resistance. The interaction between year 0 fast food and change in fast food was not significant for bodyweight (p=0·9340) or insulin resistance (p=0·1461). By comparison with the average 15-year weight gain in 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 year 0 and follow-up (n=87) gained an extra 4·5 kg of bodyweight (p=0·0054; figure 1) and had a 104% greater increase in insulin resistance (p=0·0083, figure 2).

To further assess possible residual confounding by physical activity and television viewing, we stratified our models by five levels of a composite index of these two behaviours, ranging from low activity and high television viewing to high activity and low television

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
The model is adjusted for the same covariates as in model 2 of table 4, with ethnic origin as an additional covariate. Cell-specific sample sizes range from 79 (>2 times per week at baseline and >1 times per week change) to 672 (>2 times per week at baseline and <0 times per week change).

viewing. These stratified models were tested in the full cohort with adjustment as in model 3 of tables 3 and 4 (evaluated for both year 0 fast food and change in fast food). The interaction terms between fast-food frequency (year 0 and 15-year change) and the activity index were not significant for weight change or for insulin resistance (p values range 0·26–0·51). Thus, there was a direct, albeit underpowered, association between fast-food frequency and 15-year changes in bodyweight and insulin resistance among all activity categories (data not shown). In fact, there seemed to be an especially strong association between fast-food frequency and changes in bodyweight and insulin resistance among the least physically active participants—eg, a change in fast-food frequency of three times per week was associated with a mean increase in bodyweight of 3·9 kg (SE 0·9, n=386; p<0·0001).

Discussion

In young adult black and white men and women living in the USA between 1985–86 and 2000–01, we recorded strong positive associations between frequency of visits to fast-food restaurants and increases in bodyweight and insulin resistance, the two major risk factors for type 2 diabetes.[27] By comparison with the average 15-year weight gain in participants with infrequent fast-food restaurant use at year 0 and follow-up, those with frequent fast-food restaurant use at both baseline and follow-up gained an extra 4·5 kg bodyweight and had a two-fold greater increase in insulin resistance. The associations seemed to be largely independent of other potentially confounding lifestyle factors, such as physical activity and television viewing.

Our findings accord with those of cross-sectional and shorter prospective studies of fast food and bodyweight. Binkley and colleagues[19] reported that fast-food consumption was independently associated with bodyweight in a cross-sectional analysis of 16 103 adults. French and co-workers[17] reported that intake of fried potatoes predicted 2-year weight gain in women but not men. In a 3-year prospective observational analysis of 891 women participating in a weight loss study, fast-food restaurant use was directly associated with bodyweight.[18]

The associations between fast food and insulin resistance were not fully accounted for by adjustment for many other lifestyle factors, energy intake, nutrients, and food groups, raising the possibility that some unmeasured factor inherent to fast food might be mediating this effect. The one most obvious aspect of fast food that might lead to weight gain is the large portion size, with certain single-meal calorie levels approaching total daily energy intake requirements.[11,13] We attempted to control for portion size through our adjustment for energy intake; however, energy intake was assessed only at years 0 and 7 and is measured with error. Therefore, residual confounding in energy intake,

and in the other nutrients and foods that we examined, is quite likely. Independent of energy intake, many other aspects of fast food, including direct effects of energy density on passive overconsumption,[14] might also predispose to insulin resistance. Certain fast foods contain large amounts of partially hydrogenated oils, and this class of fatty acids can cause insulin resistance and increase risk of type 2 diabetes.[15] Fast food also contains large amounts of highly refined starchy food and added sugar, carbohydrates that have been characterised as high in glycaemic index.[28] Consumption of a high glycaemic index or high glycaemic load diet has been linked to risk for diabetes, independent of bodyweight changes, in mechanistic studies,[16] short-term human trials,[29,30] and observational studies,[31,32] although this point remains controversial.[33]

The strengths of our study include: long-term prospective study design with high rates of follow-up over 15 years; standardised, repeated, and detailed measurements of dietary practices; direct assessment of specific fast-food habits, rather than proxy measures such as meals away from home or fried foods; use of the Nutrition Coordinating Center (University of Minnesota) archival system, which attempts to keep pace with the food supply, including changes in use of fats in fast food; extensive data for 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 US metropolitan areas who have been examined during a period of life when substantial weight gain occurs and chronic diseases arise.

The limitations of this study relate mainly 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 restricted to frequency of fast-food restaurant use because we were unable to analyse sufficiently the considerable range of available fast-food items and their portion sizes. Nonetheless, our results might underestimate the true magnitude of the effect because of measurement error and thorough adjustment for many covariates that might be on the causal pathway—eg, food groups and nutrients. Furthermore, several analytical issues might have affected our results. Measurement error, if non-differential, would tend to result in attenuated estimates of the strength of association between fast-food intake and the 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 difficulties would also tend to attenuate noted relations compared with the true relation (such as would be observed in a clinical trial of investigator-controlled increase or decrease in fast-food intake). Only data obtained in the first and last CARDIA examination

are included in the reported analyses. Omission of missing data or data from intermediate examination could bias the results. To address these issues data were also analysed by repeated measures regression analysis with SAS PROC MIXED (data not shown), which includes all examinations for which data are available for every individual and properly handles correlated observations within individuals. Because the repeated measures regression analysis did not yield additional insights into the data beyond that presented in the tables, we did not present specific findings using that more complex methodology.

In conclusion, fast-food habits have strong, positive, and independent associations with weight gain and insulin resistance in young black and white adults. Fast-food consumption can be linked to adverse health outcomes through plausible mechanisms, and results from other studies lend support to 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.

Contributors

M Pereira designed the study, directed the statistical analyses, and wrote the first draft of the report. A Kartashov did the statistical analyses. C Ebbeling designed the study and revised the report. L Van Horn and M Slattery supervised collection of dietary data and revised the report. D Jacobs Jr designed the study, provided statistical expertise, and revised the report. D Ludwig designed the study, provided supervision, and wrote the report.

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