




# Are neighbourhood restaurants related to frequency of restaurant meals and dietary quality? Prevalence and changes over time in the Multi-Ethnic Study of Atherosclerosis

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## Abstract

**Objective:** To examine whether the density of neighbourhood restaurants affected the frequency of eating restaurant meals and subsequently affected diet quality.

**Design:** Cross-sectional and longitudinal designs. Structural equation models assessed the indirect relationship between restaurant density ( $\leq 3$  miles (4.8 km) of participant addresses) and dietary quality (Healthy Eating Index 2010 (HEI)) via the frequency of eating restaurant meals, after adjustment for sociodemographics, select health conditions, region, residence duration and area-level income.

**Setting:** Urbanised areas in multiple regions of the USA, years 2000–2002 and 2010–2012.

**Participants:** Participants aged 45–84 years were followed for 10 years ( $n$  3567).

**Results:** Median HEI (out of 100) was fifty-nine at baseline and sixty-two at follow-up. The cross-sectional analysis found that residing in areas with a high density of restaurants (highest-ranked quartile) was associated with 52% higher odds of frequently eating restaurant meals ( $\geq 3$  times/week, OR: 1.52, 95% CI 1.18, 1.98) and 3% higher odds of having lower dietary quality (HEI lowest quartile  $< 54$ , OR: 1.03, 95% CI 1.01, 1.06); associations were not sustained in longitudinal analyses. The cross-sectional analysis found 34% higher odds of having lower dietary quality for those who frequently ate at restaurants (OR: 1.34, 95% CI 1.12, 1.61), and more restaurant meals (over time increase  $\geq 1$  time/week) were associated with higher odds of having worse dietary quality at follow-up (OR: 1.21, 95% CI 1.00, 1.46).

**Conclusions:** Restaurant density was associated with frequently eating out in cross-sectional and longitudinal analyses but was associated with the lower dietary quality only in cross-sectional analyses. Frequent restaurant meals were negatively related to dietary quality. Interventions that encourage less frequent eating out may improve population dietary quality.

**Keywords**  
Food environment  
Food away from home  
Restaurant meals  
Diet  
Structural equation models

During the past 30 years, there have been increases in the availability and consumption of prepared foods<sup>(1–4)</sup>. Relative to meals at home, restaurant foods tend to be larger in portion size and higher in Na, saturated fat and cholesterol, and lower in fibre<sup>(3,5,6)</sup>. Among adults, eating fast food has been associated with lower overall dietary quality<sup>(7,8)</sup> and the frequency of fast-food restaurant meals has been directly associated with lower quality diet<sup>(9)</sup>. While most studies of

the effect of restaurant dining on dietary quality have focused on fast foods, the growth of full-service restaurants (AKA sit-down restaurants) has occurred alongside fast-food/fast-casual restaurants<sup>(4,10)</sup>. Chains dominate the full-service restaurant industry – capturing 70% of market share<sup>(11)</sup> – and a number of studies have documented that the dietary quality of most full-service restaurant meals is as low or even lower than fast-food/fast-casual restaurants<sup>(5,6,12)</sup>.

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Research investigating neighbourhood conditions on health posits that environments offering many opportunities for eating out make it more convenient to eat out<sup>(1,13)</sup>. Thus, the density of neighbourhood restaurants may be associated with a higher frequency of eating restaurant meals and subsequently worse dietary outcomes among adults (Fig. 1). Findings that directly link neighbourhood restaurants to dietary quality have been mixed<sup>(14)</sup>, and most studies focused only on youth or young adults<sup>(15)</sup>. Among mid- to older-aged adults, most studies reported no evidence of an overall association<sup>(9,16–19)</sup> but there have been exceptions<sup>(18)</sup>. Work by Burgoine *et al.*<sup>(18)</sup> found that fast-food density within 1 mile of residence was cross-sectionally associated with more consumption of foods that are typically found in fast-food establishments (pizza, burgers and deep-fried foods).

Reasons for null or mixed results in studies of mid- to older-aged adults could be due to a number of factors including measurement issues such as inadequacy in the way neighbourhood restaurant density was defined (only fast food<sup>(9,19)</sup> or only fast-food chains<sup>(17)</sup>, measurement limited to one or two regions<sup>(17,18,20)</sup> and/or limitations in dietary assessment and operationalisation (e.g. only energy and a few macro-nutrients<sup>(16,17,19)</sup> rather than a full dietary score). Importantly, most studies have not explored intervening mechanisms on the pathway from restaurant exposure to dietary quality. For example, frequency of eating restaurant food is presumed to be an intermediary between restaurant environment and diet but is rarely considered.

The current study examined the association between restaurant density, frequency of eating restaurant meals and dietary quality in a multi-ethnic cohort of mid- to older-aged adults. The cross-sectional hypothesis was that participants with higher exposure to restaurants will have more frequent restaurant meals and lower dietary quality; specifically, that restaurant meals will be a mediator between the restaurant environment and dietary quality.

The longitudinal hypothesis was that residing in areas where there were increases in restaurant density would be associated with more frequent eating out and subsequently worse dietary quality over time.

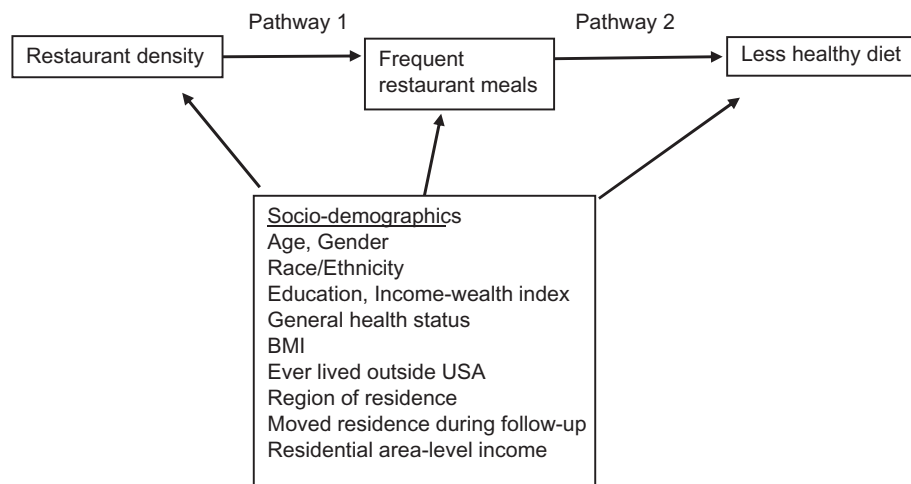
**Methods**

**Data**

Data came from The Multi-Ethnic Study of Atherosclerosis (MESA), a population-based longitudinal cohort study. MESA’s main objective was to determine the characteristics of subclinical CVD and its progression. The study recruited ethnically diverse adults aged 45–84 years with no known presence of CVD. Individuals were recruited from six sites across the USA: Bronx/Upper Manhattan, NY; Baltimore City and Baltimore County, Maryland; Forsyth County, North Carolina; Chicago, Illinois; St. Paul, Minnesota and Los Angeles County, California. Each site recruited participants from locally available sources (lists of residents, list of dwellings, telephone exchanges) as well as publicising the study in local media. Sampling and recruitment procedures have been described in detail elsewhere<sup>(21)</sup>. MESA included a baseline examination (2000–2002) and four follow-up exams. Exam 5 data were collected approximately 10 years after baseline (2010–2012). We limited analyses to baseline and exam 5 data because the dietary questionnaire was only collected at exams 1 and 5. Written informed consent was obtained from the participants, and the study was approved by institutional review boards at each site.

**Diet**

Diet was assessed via a FFQ. The FFQ was a modified Block-style, 128-item questionnaire. Participants were asked about their usual eating habits over the past 12 months. For each of the food items on the FFQ, respondents chose their consumption frequency (rare or



**Fig. 1** Illustration of pathways between restaurant density and diet



never, 1/month, 2–3/month, 1/week, 2/week, 3–4/week, 5–6/week, 1/d and 2+/d). Their frequency of consumption was then weighted by a multiplier, according to their reported typical serving size ( $\times 0.5$ ,  $\times 1.0$  and  $\times 1.5$  for small, medium and large, respectively).

The MESA FFQ was adapted from the questionnaire designed for the Insulin Resistance and Atherosclerosis Study<sup>(22)</sup> and has been described elsewhere<sup>(23)</sup>. Modifications to the FFQ included additional items to reflect the multi-ethnic composition of the MESA cohort. Insulin Resistance and Atherosclerosis Study was validated against 24-h dietary recalls<sup>(22)</sup>, and the MESA diet data correlated as expected with HDL-cholesterol and TAG concentrations<sup>(24)</sup>, and cardiometabolic conditions<sup>(25–29)</sup>.

Total energy was calculated for each FFQ line item using the Nutrition Data System for Research (NDS-R database; Nutrition Coordinating Center)<sup>(24)</sup>. Following work by others, we excluded participants whose dietary data were considered unreliable, due to reporting usual energy intake  $<600$  or  $>6000$  kcal<sup>(24)</sup> (approximately 6% of the participants who completed the dietary questionnaire).

## Outcome

### Healthy Eating Index

We used the Healthy Eating Index version 2010, to assess dietary quality. It reflects 2010 U.S. federal Dietary Guidelines, has been used to monitor and assess diet quality in the USA<sup>(30–32)</sup> and has: (1) adequate content validity<sup>(30)</sup>; (2) sufficient construct validity and (3) acceptable reliability<sup>(33)</sup>. It includes twelve components: total fruit, whole fruit, total vegetables, greens and beans, whole grains, dairy, total protein foods, seafood and plant proteins, fatty acid, refined grains, Na and empty energies. Each component contributes a minimum of 0 to a maximum of 5, 10 or 20 points, resulting in a range of 0–100 for the total score; higher scores indicate a healthier diet<sup>(30)</sup>. Linkage of MESA food consumption with HEI food composition was done following the protocol established by the National Cancer Institute<sup>(30,34)</sup>. Each individual's nutritional values were derived by linking the food items from the FFQ to MyPyramid Equivalents Database version 2.0, multiplying by the number of servings reported in the FFQ, summing to obtain a value for each component in the HEI and then calculating the HEI score.

In cross-sectional analyses, the HEI at exam 5 was divided into quartiles of the observed HEI distribution (range 11.67–89.56) with the lowest quartile hereafter referred to as a 'lower quality diet' ( $<54.28$ ). In the longitudinal analysis, each participant's HEI at exam 5 was subtracted from exam 1 (change score range  $-44.50$ – $42.40$ ) and then divided into quintiles with the lowest quintile hereafter referred to as 'worse diet quality over time' ( $<-6.19$ ). (There were only small changes in HEI score over time; thus, we used a lower cut-point – the lowest 20th percentile – in longitudinal analyses in order to

measure a meaningful amount of change.) The rationale for using within-sample ranking of dietary data is that it acknowledges the low precision inherent in dietary self-reports<sup>(35)</sup>. Numerous studies have used ranked values to define unhealthy or healthy diets (e.g. ref. (36–38)) because it differentiates lower and higher values within a sample without relying on an absolute threshold of dietary quality<sup>(39)</sup>.

## Mediator

### Frequency of restaurant meals

'Frequency of restaurant meals' (an intermediate variable in the causal pathway between neighbourhood food environment and healthy eating) was determined by a single question in the FFQ: 'how many times per week do you eat at restaurants for meals?'. In the cross-sectional analysis, *frequency of restaurant meals* was operationalised as a binary indicator: being in the top quartile at exam 5 ( $\geq 3$  times/week) or not. In the longitudinal analysis, *higher frequency of restaurant meals* was a within-person change indicator, operationalised as a binary variable,  $\geq 1$  more time/week relative to exam 1 (note that  $\geq 1$  more time/week was approximately the top 25%).

### Neighbourhood-level exposures

Addresses of MESA participants and addresses of restaurant establishments were used to link participants to the density of restaurants near their residence. Restaurant establishment data originated from Dun and Bradstreet and was compiled/cleaned for the National Establishment Time Series database<sup>(40,41)</sup>. Eating places were first classified as 'fast-food chain' (name search of the top seventy-five chains from Restaurant & Institutions<sup>(42)</sup>) and then 'fast-food non-chain' (limited-service restaurant SIC code 581203 not already identified as a chain). 'Other eating places' were identified (eating place with SIC 5812 not in the fast-food group). 'Other eating places' includes a wide variety of restaurants. We excluded coffee, donut and ice cream shops because those shops generally sell snacks/limited food offerings at the time of this study period. Drinking establishments that only serve alcohol were excluded.

Restaurant density was derived in GIS by computing a three-mile (4.8 km) kernel density of food establishments around each MESA participant's home. Using a kernel density resulted in a distance-weighted density such that restaurants furthest from the participant's residence were weighted less than those closest to the residence<sup>(43)</sup>. A three-mile kernel radius was chosen because it aligns with empirical findings of average distances to food shopping<sup>(44,45)</sup> and roughly aligns with what others have done<sup>(46,47)</sup> thus enabling comparability across studies.

The measure presented in this study represents density to all restaurants. The correlation was very high between



the density of total restaurants and density of subgroups of restaurants (Spearman's rank correlation coefficient which is appropriate for skewed variables  $\geq 0.92$ ); thus, analyses will only be shown for total restaurants. Further, combining all restaurants mitigated misclassification of restaurants by type and reduced the number of participants with zero exposure to restaurants.

In the cross-sectional analysis, *high density of restaurants* was operationalised as the highest-ranked quartile of restaurants at exam 5 ( $\geq 16$  restaurants within 3 miles of each participant's residential address). In the longitudinal analysis, *change in restaurant density* represented a relatively stable value or an increase in density ( $-0.6$  to  $+69.8$  restaurants within 3 miles, top 25 % of the sample). We included relatively stable density in this group because preliminary analyses showed that almost all participants experienced a decrease in restaurant density over time.

### **Covariates**

Person-level covariates were age, sex, race/ethnicity, education level, household income/wealth (combination of income level and ownership of four assets: car, home, land and investments) and years lived outside the USA (classified into none *v.*  $>0$ ); see variable classifications shown in Table 1. Additional covariates were: self-reported general health status (poor or fair *v.* good to excellent, only available at baseline) and BMI. Additional area-level characteristics corresponding to participant addresses were census region (northeast, mid-west, south and west) and percentage of households with higher incomes (per capita household income  $> \$50\ 000$ ). Census region was included because diet and restaurant outlets are known to vary by region. Longitudinal control variables also included change variables: change in per capita income (exam 5 – exam 1) and change in area income (exam 5 – exam 1); and categorical variables representing region at exam 1, region at exam 5 and moved outside of baseline county. The list of variables is in the regression table footnote.

### **Analytic sample**

Out of a total 6814 participants enrolled at baseline, 4716 participated in exam 5 (69 % of the exam 1 sample). We excluded those without the following data elements: neighbourhood food environment data ( $n\ 13$ ), dietary components at exam 1 and/or exam 5 ( $n\ 851$ ), frequency of restaurant meals ( $n\ 78$ ) and key covariate information ( $n\ 207$ ). Finally, 3567 (53 % of 6814 participants) were retained for analyses.

Sample characteristics for included *v.* excluded participants were similar by age and sex, but included participants had higher income and education, fewer Black/African-American and slightly lower restaurant density around their residence (data not shown).

### **Statistical analyses**

As described above, restaurant density (exposure) and restaurant meals (mediator) were transformed into ranked categorical variables and then a binary variable was derived that represented the top-ranked categories (highest density of restaurants and highest restaurant meals) *v.* not top-ranked. The reasons for this classification were: (1) both variables were skewed, thus classification aided interpretation; and (2) preliminary analyses found non-linearity in the association (e.g. there was only a discernible effect between restaurant density and diet for the upper rank). Further, for the change analyses, on average, there was little change over time in these exposures; thus, we needed to maximise change by selecting the highest increase. We only show binary variables to facilitate interpretation of results in structural equation modelling (the method becomes overly complex to interpret when operationalised with multi-category exposures/mediators).

Cross-sectional analyses limited the data set to exam 5. The rationale for using exam 5 rather than exam 1 in the cross-sectional analysis is that there was more heterogeneity in exposure at exam 5 because participants relocated to other areas during follow-up.

### **Structural equation modelling**

We used a structural equation model (SEM). Our conceptual framework constructed a causal pathway between the density of food environment and poor dietary quality via frequency of restaurant meals (frequency of restaurant meals was the mediator, Fig. 1). There is no plausible reason why density of local restaurants would affect diet directly (not via restaurant meals); thus, we did not model a direct causal effect of density of food environment on poor dietary quality.

Adjusted analysis presents results for pathway 1, the direct effect between high restaurant density and high frequency of restaurant meals; pathway 2: the direct effect between high frequency of restaurant meals and low or worse dietary quality; and the combination of pathways 1 and 2: the total effect of restaurant density on dietary quality. The analyses only had one sequence/pathway, and thus the total effect is also the 'total indirect effect' which tests whether the effect of restaurant density on dietary quality was mediated by frequency of restaurant meals. Standard errors for the test were generated via bootstrapping (based on 1000 resamples, with replacement).

We implemented the SEM in M-plus 8.3<sup>(48)</sup>. Maximum Likelihood Estimation was used to estimate the model parameters. We chose this estimator in M-plus as it can accommodate binary outcomes and binary mediators and permit the evaluation of indirect (mediation) effects via logit regression<sup>(49)</sup>.

Goodness-of-fit statistics assessed whether the structure of the model was appropriate for the data. Logistic

regression has limited options for assessing SEM fit and lacks external target values to indicate acceptable fit. Thus, we used the probit distribution to assess fit because it is able to generate standard fit statistics available for a Gaussian distribution. We employed a group of well-known fit indices to evaluate the model fit:  $\chi^2/df$  ratio, Standardized Root-Mean-Square Residual, Tucker–Lewis Index, Comparative Fit Index and Root-Mean-Square Error of Approximation. Goodness-of-fit in SEM indicates the degree of agreement between the model-implied covariance matrix and the covariance matrix of the observed data<sup>(50)</sup>. If these two covariance matrices are close, then the model fits the data well (see the regression table footnote).

### Adjustment variables

Models adjusted for confounding by socio-demographics: age, sex, race, education, income/wealth categories, general health, BMI, ever having lived outside the USA, region of residence, whether they moved residence during follow-up and area-level income (details are in the regression table footnotes). Adjustment was achieved by allowing for direct paths between sociodemographics and exposure, sociodemographics and mediator, and sociodemographics and outcome.

### Sensitivity analyses

Sensitivity analysis used nested model comparisons (AKA multiple-group analysis<sup>(51)</sup>) to test interactions between restaurant density and the following variables: population density (below median, at or above median), sex (male *v.* female), income/wealth index (low to middle *v.* high), movers (moved since baseline *v.* not) and obesity (obese *v.* not obese).

We examined the sensitivity of the cross-sectional results to operationalising dietary quality as a *continuous* variable. Successful interpretation of mediation results requires consistency in the directionality (signage) of the pathways<sup>(52)</sup>. For this reason, we reverse-coded dietary quality so that higher values would signify a worse diet. (Note that we did not examine continuous variables for restaurant density and frequency of restaurant meals due to these variables being highly skewed. Further, we did not operationalise change in diet as a continuous variable as there was very little longitudinal change in diet; thus, we would not be able to detect a signal in our data.)

Additionally, we used the longitudinal data and tested the *inverse of our main hypothesis*: whether a *decline* in restaurant density was associated with *less* eating out; and *less* eating out was associated with *better* diet. In order to align these analyses with variable operationalisations used in the main analyses, ‘decline in restaurant density’ was defined as the lowest quartile (loss of at least seven restaurants within a 3 mile area), ‘less eating out’ was at least 2 times less/week (relative to exam 1) and ‘improved

dietary quality’ was defined as highest quintile of change in HEI score.

## Results

### Descriptive results

Participant socio-demographics at baseline (exam 1) and exam 5 (approximately 10 years later) are reported in Table 1. At baseline, mean age was 60.2 (STD 9.6) years,

**Table 1** Participant characteristics, *n* 3567\*

	Exam 1 (2000–2003)	Exam 5 (2010–2011)
	%	%
Sociodemographic characteristics		
Age (years)		
Mean	60.2	69.6
STD	9.6	9.5
Gender		
Male	47.6	–
Race/ethnicity		
White (Caucasian)	44.4	–
Chinese-American	10.7	–
Black, African-American	24.3	–
Hispanic or Latino	20.6	–
Education		
Completed HS/GED or less	29.2	–
Some college, Technical or Associate degree	29.1	–
Bachelor's degree or higher	41.7	–
Income–wealth index†		
Low	9.5	7.9
Middle	39.1	43.0
High	51.4	49.1
General health status		
Fair or poor	7.4	–
BMI (weight, kg/height, m <sup>2</sup> )		
Normal (<25)	29.3	29.1
Overweight (25–<30)	39.3	37.5
Obesity (≥30)	31.4	33.4
Ever lived outside USA		
Yes	26.2	–
Region of residence		
Midwest	37.7	36.4
Northeast	16.7	16.5
South	30.4	31.4
West	15.2	15.7
Moved residence during follow-up		
Did not move	–	69.9
Moved within the same county	–	21.2
Moved out of the county	–	8.9
Residential area-level income		
Percentage of household living in areas at or above US median income (≥\$50 000)		
Mean	42.7	51.2
STD	17.6	18

\*The analytical sample includes 3567 participants. Out of a total 6814 participants enrolled at baseline, 4716 participants were retained in exam 5. We further excluded: (a) thirteen participants with missing neighbourhood food environment data; (b) 851 participants with missing dietary information in both exams; (c) seventy-eight participants with missing eating out information in both exam 1 and exam 5; and (d) 207 participants with missing covariates.

†Income–wealth index is participant's inflation adjusted annual per capita inflation-adjusted household income (5-levels) + wealth index. Wealth is home ownership + car ownership + land ownership + investments. In preliminary analyses, generalised additive models were used to assess non-linearity and data were subsequently classified into low <2, medium 2–<6 and high ≥6.

**Table 2** Distribution of the Healthy Eating Index, frequency of restaurant meals and restaurant density, at baseline and follow-up, *n* 3567\*

	Exam 1 (2000–03)	Exam 5 (2010–2011)
Baseline and follow-up values		
Healthy Eating Index (HEI-2010)		
Mean	58.9	60.4
STD	9.3	10
Median value	59.3	61.6
25th–75th percentile	52.8–65.6	54.4–67.2
Frequency of restaurant meals, number of times/week		
Median value	2	1
25th–75th percentile	1–4	1–3
0–<2 times/week, %	43.5	51.8
≥2 times/week, %	56.5	48.2
Restaurant density within 3-mile buffer (all restaurants includes fast food and other eating places)		
Median value	9	6.8
25th–75th percentile	4.8–23.3	3.4–16
Change variables (continuous)		
Change Exam 5 – Exam 1		
Change in Healthy Eating Index		
Range (minimum–maximum)	–44.5 to 42.4	
Median value	1.39	
25th–75th percentile	–4.57 to 8.03	
Change in frequency of restaurant meals, per week		
Range (minimum–maximum)	–9 to 9	
Median value	0	
25th–75th percentile	–1 to 0	
Change in restaurant density in 3 mile buffer		
Range (minimum–maximum)	–226.5 to 69.85	
Median value	–1.8	
25th–75th percentile	–6.7 to –0.658	

\*The analytical sample includes 3567 participants. Out of a total 6814 participants enrolled at baseline, 4716 participants were retained in exam 5. We further excluded: (a) thirteen participants with missing neighbourhood food environment data; (b) 851 participants with missing dietary information in both exams; (c) seventy-eight participants with missing eating out information in both exam 1 and exam 5; and (d) 207 participants with missing covariates.

slightly more than one-half sample was non-White, 58.3% had less than a college degree and 31.4% had obesity. Approximately one-half of participants lived in areas where median per capita income was at or above the US median ( $\geq \$50\,000$ <sup>(53)</sup>).

### **Diet (Healthy Eating Index 2010 and restaurant meals)**

Median HEI was 59.3 at baseline (similar to the US average<sup>(54)</sup>) and rose slightly by exam 5 (median 61.6, 25th–75th percentile, 54.4–67.2) (Table 2). Participants ate out approximately 2 times/week at baseline (median 2, 25th–75th percentile, 1–4), and the frequency declined slightly by exam 5 to 1 time/week (median 1, 25th–75th percentile, 1–3).

### **Restaurants**

At baseline, participants lived in areas with a median of nine restaurants in their area (25th–75th percentile, 4.8–23.3 restaurants in the 3 miles surrounding their home). Median (25th–75th percentile) in 3 miles was 2.10 (1.29–4.00) for fast food and was 4.71 (25th–75th percentile, 1.03–12.03) for non-fast food. At follow-up,

residents lived nearby slightly fewer restaurants (median –1.8 fewer restaurants). Over the follow-up period, 30% moved residence. Most of the movers stayed within the same region/county but moved to less densely populated areas (where there were fewer restaurants). Population density was highly correlated with restaurant density (spearman rank correlation 0.85, data not shown).

### **Adjusted results**

Table 3 displays cross-sectional and longitudinal adjusted results (Panel A and Panel B, respectively). In cross-sectional analyses, high restaurant density was associated with more eating out and worse dietary quality. Relative to areas with fewer restaurants, residing in an area with many restaurants (top quartile,  $\geq 16$  restaurants within 3 miles) was directly associated with 52% higher odds of eating out frequently ( $\geq 3$  times/week, OR 1.52, 95% CI 1.18, 1.98). In turn, frequent eating out was directly associated with 34% higher odds of lower dietary quality (OR 1.34, 95% CI 1.12, 1.61). Cross-sectional results suggest that frequency of eating out was a mediator in the pathway between restaurant density and diet (total indirect effect *P*-value 0.02). Relative to areas with fewer restaurants,



**Table 3** Regression table. Adjusted odds ratios for having worse dietary quality and frequently consuming restaurant meals, in response to residing in areas with more restaurants (*n* 3567)

	Pathway 1			Pathway 2			Pathway 1 and Pathway 2		
	Direct effect			Direct effect			Total indirect effect*		
	Odds ratio	95 % CI	<i>P</i>	Odds ratio	95 % CI	<i>P</i>	Odds ratio	95 % CI	<i>P</i>
<b>PANEL A† Cross-sectional results, exam 5</b>									
	<b>Outcome A-1</b>			<b>Outcome A-2</b>					
	Frequent restaurant meals (4th quartile, ≥3 times/week)			Lower dietary quality (1st quartile of Healthy Eating Index)					
High restaurant density (top quartile, ≥16 restaurants within 3 miles v. fewer restaurants)	1.52	1.18, 1.98	0.009	–	–	–	1.031	1.01, 1.06	0.02
High frequency of restaurant meals (top quartile, ≥3 times/week v. less eating out)	–	–	–	1.34	1.12, 1.61	0.007	–	–	–
<b>PANEL B‡. Change results, exams 1–5</b>									
	<b>Outcome B-1</b>			<b>Outcome B-2</b>					
	Increased frequency of restaurant meals (approximately 4th quartile, ≥1 times/week)			Worse diet quality at follow-up (lowest change quintile 1 indicating worsening dietary quality)					
Stable or increase in restaurant density (top quartile of change, –0.6 to +69.8 restaurants within 3 miles v. fewer restaurants over time)	0.99	0.81, 1.22	0.94	–	–	–	1.00	0.99, 1.01	0.87
Increase in restaurant meals (top quintile, ≥1 more time/week relative to exam 1 v. stayed same or less eating out over time)	–	–	–	1.21	1.00, 1.46	0.08	–	–	–

\*The ‘total indirect effect’ *P*-value tested whether the effect of restaurant density on dietary quality was mediated by frequency of restaurant meals. The analysis only had one sequence/pathway, and thus the ‘total indirect effect’ is also the ‘total effect’. Standard errors for the test were generated via bootstrapping (based on 1000 resamples, with replacement).

†PANEL A Cross-sectional results, exam 5. Outcome A-1 shows the odds of frequent restaurant meals (4th quartile, ≥3 times/week). Outcome A-2 shows the odds of worse dietary quality (1st quartile of Healthy Eating Index). Adjustment variables were: linear splines for age (younger, and older), gender, race/ethnicity, education, income–wealth, ever lived outside USA, general health status, BMI categories, region, area income is high. Model fit indices from a Probit model were:  $\chi^2 = 1.25$ , *df* = 1, *P*-value = 0.26, Comparative Fit Index (CFI) = 1, Tucker–Lewis Index (TLI) = 0.987, Root-Mean-Square Error of Approximation (RMSEA) = 0.008, Standardized Root-Mean-Square Residual (SRMR) = 0.004.

‡PANEL B Change results, exams 1–5. Outcome B-1 shows the odds of increase in restaurant meals (approximately 4th quartile, ≥1 more time/week relative to exam 1). Outcome B-2 shows the odds of a having worse diet quality at follow-up (lowest change quintile 1 indicating worsening dietary quality). Adjustment variables were: linear splines for age (younger, and older), gender, race/ethnicity, education, income–wealth at exam 1, change in per capita income (exam 5 – exam 1), region at exam 1, region at exam 5, area income is high at exam 5, change in area income is high, ever lived outside USA and moved outside of baseline county. Model fit indices from a Probit model were:  $\chi^2 = 1.606$ , *df* = 1, *P*-value = 0.205, RMSEA = 0.013, CFI = 0.998, TLI = 0.884, SRMR = 0.004.



residing in an area with many restaurants was associated with 3% higher odds of lower dietary quality (HEI 1st quartile, OR 1.03, 95% CI 1.01, 1.06).

In the longitudinal analysis, relative to exam 1, residing in areas with stable or increased restaurant density was not associated with more restaurant meals and was not associated with worsening of dietary quality. In longitudinal analyses, there was no evidence that frequency of eating out was a mediator between restaurant density and diet (total indirect effect  $P$ -value 0.87). Nonetheless, after approximately 10 years of follow-up, results suggested that more restaurant meals over time (increase of  $\geq 1$  times/week) were associated with 21% higher odds of having worse dietary quality, although the CI included the null value (OR 1.21, 95% CI 1.00, 1.46).

### Sensitivity analyses

There was no evidence of cross-sectional interactions between restaurant density and population density (below median, at or above median), sex (male *v.* female), income/wealth index, movers (moved since baseline *v.* not) and obesity (obese *v.* not obese); all  $P$  for interaction  $\geq 0.2$ .

The cross-sectional inference was unchanged when dietary quality was operationalised as a continuous variable. Frequent eating out was directly associated with 1.48 lower (worse) HEI score ( $\beta$  1.48, 95% CI 0.77, 2.20), results not shown in tables. Results suggested that frequent eating out was a mediator in the pathway between restaurant density and worse diet (total indirect effect  $P$ -value 0.007). Relative to areas with fewer restaurants, residing in an area with many restaurants was associated with 0.15 lower HEI score ( $\beta$  0.15, 95% CI 0.04, 0.27).

Changes were very small in restaurant density, eating out and diet; thus, longitudinal interactions were not tested; and continuous dietary change was not examined. However, we used the longitudinal data to test the *inverse* of our main hypothesis: whether a *decline* in restaurant density was associated with *less* eating out; and *less* eating out was associated with *better* diet. Under this hypothesis, longitudinal inference was largely unchanged except that now pathway 2 was also null ('is less eating out associated with better diet?'). We conjecture that pathway 2 was null because the *inverse* hypothesis followed the overall temporal trend of the data (on average, participants ate out less and dietary quality improved over time) thus making it harder to detect a signal in our data set.

## Discussion

### Summary

This study of mid-aged/older adults living in select urbanised areas across the USA found that living in an area with many restaurants was associated with more restaurant meals and lower dietary quality. However, those findings

were only apparent in cross-sectional data. When we examined changes in restaurant environment and changes in diet quality, there was no association between restaurant density and restaurant meals or between restaurant density and dietary quality. The impacts of frequent restaurant meals on dietary quality were more robust. Frequent restaurant meals were associated with much higher odds of having lower dietary quality in cross-sectional data and the relationship persisted in longitudinal analyses (despite CI including the null value).

Distinct advantages of this study are described here: (1) We included cross-sectional and longitudinal data and participants who resided in many urbanised areas across the USA. Almost all prior studies used cross-sectional data, and many were limited to a single state/province which limits generalisability of the findings (examples here (17,19)); (2) While aggregating restaurants into all restaurant types presented some limitations to our analyses (discussed in Limitations section), there were also strengths in this approach. By combining all restaurants, restaurant-type misclassification was not an issue. Further, prior research on the effect of restaurant density on dietary outcomes among mid-older adults has almost exclusively focused on fast-food restaurants and reported null findings<sup>(9,17,19)</sup>. Full-service restaurants have been overlooked even though the dietary quality and obesogenic potential of most full-service restaurant meals are roughly equivalent or worse than fast-food/fast-casual restaurants<sup>(5,6,12)</sup> and (3) We incorporated two causal pathways into the same model: (i) the pathway between restaurant density and restaurant meals and (ii) the pathway between restaurant meals and dietary quality. The method we used simultaneously modelled these pathways and adjusted for potential socio-demographic confounding of both pathways. Below, we discuss our findings in the context of the literature.

### Pathway 1 + 2

#### Restaurant density and diet

Prior studies that aimed to quantify the direct association between the restaurant density and diet focused mostly on youth or young adults and reported mixed results<sup>(15)</sup>. Among mid- to older-aged adults, cross-sectional data reported no evidence of an overall association between GIS-assessed fast-food density and dietary intake<sup>(9,16–19)</sup>. The exception was a study conducted in one UK county that found a positive association between fast-food outlet density and total grams of foods commonly associated with fast-food establishments (pizza, burgers and deep-fried foods)<sup>(18)</sup>. The UK study used different measures from ours making comparisons difficult. Nevertheless, our cross-sectional findings aligned with the UK study: that restaurant density could promote an unhealthy diet. However, the magnitude of the association found in our sample was small: the top quartile of restaurant density was associated





with 3 % higher odds of having a lower quality diet. Further, the association did not persist when we examined changes in restaurant density and changes in diet over a 10-year period. The small magnitude of the cross-sectional association and lack of longitudinal results suggest that restaurant density may not have a notable influence on dietary quality among mid-aged/older adults.

### **Pathway 1**

#### *Restaurant density and frequency of restaurant meals*

Most studies that assessed the association between restaurant density and frequency of eating restaurant food among adults used cross-sectional data from a single province/state, and results were mixed. Some found expected associations between the higher density of restaurants and frequency of restaurant meals<sup>(9,55)</sup> or higher relative spending on away-from-home foods<sup>(56)</sup>. However, other studies did not find evidence of an association<sup>(20,46,57)</sup>. Literature that relied on a single study site/region and focused only on fast food tended to show null results<sup>(20,46,57)</sup>, whereas multi-site/multi-region studies<sup>(9)</sup> and/or including non-fast-food restaurants<sup>(55)</sup> tended to report expected results. Results from our cross-sectional adjusted analyses aligned with studies that found positive associations. We found residing in an area with many restaurants ( $\geq 16$  restaurants of all types within 3 miles) was associated with 52 % higher odds of frequent restaurant meals ( $\geq 3$  times/week) relative to residing in areas with fewer restaurants. However, no association was observed when longitudinal data were used. It is difficult to draw conclusions from the absence of a longitudinal effect in our study because changes in restaurant density were small and changes in the frequency of restaurant meals were small, which hampered the quantification of longitudinal change. Nevertheless, our null longitudinal results aligned with overall null results reported in the only longitudinal study to date<sup>(47)</sup>.

### **Pathway 2**

#### *Restaurant meals and diet quality*

Prior work reported that fast-food and full-service restaurant food consumption among adults was associated with significant increases in lower overall dietary quality<sup>(7–9)</sup> and nutritional biomarkers<sup>(58)</sup>. Our study aligned with those results. Frequent restaurant meals were cross-sectionally associated with 34 % higher odds of having lower dietary quality; and relative to exam 1, on average, those who increased their frequency of restaurant meals (increase of  $\geq 1$  time/week) had 21 % higher odds of worse dietary quality. This pathway had the strongest signal among the pathways examined likely due to being most proximal to dietary decision-making.

### **Limitations**

Below, we note a few study limitations and steps taken to reduce their impact: (1) The limitations of FFQ data are well-known<sup>(59)</sup>; and FFQ are not well-suited for looking at individual dietary components; thus, we only used an overall index for dietary quality (HEI). Strengths of the measures we used are that the face validity of the FFQ used in this study has been documented<sup>(22,24)</sup> and the instrument was designed to include many foods that reflect the diversity of a multi-ethnic population. Further, we confirmed that the sample distribution of dietary measures calculated for our study (HEI and frequency of restaurant meals) roughly aligned with distributions reported in external data sets (surveillance data sets and other research<sup>(54,60,61)</sup>). Additionally, we utilised within-sample ranking of dietary data (quartile or quintile) which differentiated lower and higher values within a sample without relying on an absolute threshold of dietary quality<sup>(39)</sup>; (2) It is unknown whether MESA participants utilised restaurants within 3 miles of their residences. However, 3 miles aligned with the average distance individuals travel for food<sup>(62,63)</sup> and distances associated with dietary outcomes examined in another restaurant study<sup>(47)</sup>. We did not have information on the work location of participants; however, our cohort is older and most were not employed by exam 5; (3) In our sample, the correlation was very high between total restaurants and subgroups of restaurants; thus, we were not able to determine if results differed by restaurant type or diversity of restaurant types; (4) Some of the analyses were cross-sectional which are subject to temporal biases. Further, there were only two exam periods for the diet data which limited our options for longitudinal analyses. Our older sample was quite stable in their residences and showed only small changes in diet and residential exposures over 10-year period; this hampered our ability to detect hypothesised signals from the longitudinal data; (5) General health status was not available at the follow-up exam. Controlling for age will account for some of the changes in health over time; nevertheless, residual confounding could remain; and (6) Finally, results are not likely generalisable to younger populations who tend to have higher frequency of restaurant meals and worse overall dietary quality<sup>(15,64)</sup>.

### **Conclusions**

With a large proportion of the US population not meeting national dietary guidelines, it is important to understand distal and proximal risk factors for low-quality diet. This study affirmed that eating frequent restaurant meals had a negative association with dietary quality, thus reiterating an important public health message that is poorly understood by consumers<sup>(65)</sup>: in general, restaurant meals are not healthier than preparing food at home and



can be associated with worse dietary quality<sup>(64,66,67)</sup>. Our cross-sectional findings also suggested that restaurant density may encourage eating more restaurant meals likely due to residents' having many opportunities for eating out, thus making it more convenient to eat out<sup>(1,13)</sup>. Those findings suggested that restaurant density linkages to dietary quality may occur via frequency of restaurant meals; thus, interventions aimed at consumers to limit the frequency of eating out may be a strategy for improving dietary quality. Despite 10 years of follow-up data, dietary change in our older-aged cohort was minimal thus constraining our ability to detect associations with change in diet. Future work could focus on younger cohorts whose dietary behaviours/habits are less static, as well as cohorts experiencing increases in restaurant density such as those living in rapidly changing urban environments in the developing world<sup>(68)</sup>.

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from the participants, and the study was approved by institutional review boards at each recruitment site for the Multi-Ethnic Study of Atherosclerosis (according to the guidelines laid down in the Declaration of Helsinki and all procedures involving research study participants).

### References

1. Swinburn BA, Sacks G, Hall KD *et al.* (2011) The global obesity pandemic: shaped by global drivers and local environments. *Lancet* **378**, 804–814.
2. Ni Mhurchu C, Vandevijvere S, Waterlander W *et al.* (2013) Monitoring the availability of healthy and unhealthy foods and non-alcoholic beverages in community and consumer retail food environments globally. *Obes Rev* **14**, Suppl. 1, 108–119.
3. Kant AK & Graubard BI (2004) Eating out in America, 1987–2000: trends and nutritional correlates. *Prev Med* **38**, 243–249.
4. James P, Seward MW, James O'Malley A *et al.* (2017) Changes in the food environment over time: examining 40 years of data in the Framingham Heart Study. *Int J Behav Nutr Phys Act* **14**, 84.
5. Auchincloss AH, Leonberg BL, Glanz K *et al.* (2014) Nutritional value of meals at full-service restaurant chains. *J Nutr Educ Behav* **46**, 75–81.
6. Wu HW & Sturm R (2012) What's on the menu? A review of the energy and nutritional content of US chain restaurant menus. *Public Health Nutr*. Published online: 11 May 2012. doi: 10.1017/S136898001200122X.
7. Schroder H, Fito M, Covas MI *et al.* (2007) Association of fast food consumption with energy intake, diet quality, body mass index and the risk of obesity in a representative Mediterranean population. *Br J Nutr* **98**, 1274–1280.
8. Barnes TL, French SA, Mitchell NR *et al.* (2016) Fast-food consumption, diet quality and body weight: cross-sectional and prospective associations in a community sample of working adults. *Public Health Nutr* **19**, 885–892.
9. Moore LV, Diez Roux AV, Nettleton JA *et al.* (2009) Fast-food consumption, diet quality, and neighborhood exposure to fast food: the Multi-Ethnic Study of Atherosclerosis. *Am J Epidemiol* **170**, 29–36.
10. Rummo PE, Guilkey DK, Ng SW *et al.* (2017) Beyond supermarkets: food outlet location selection in four U.S. cities over time. *Am J Prev Med* **52**, 300–310.
11. The NPD Group (2012) *Independent Restaurants Account for 87 Percent of Industry Traffic Losses Since 2008*. Port Washington, NY: NPD Group.
12. An R (2016) Fast-food and full-service restaurant consumption and daily energy and nutrient intakes in US adults. *Eur J Clin Nutr* **70**, 97–103.
13. Giskes K, van Lenthe F, Avendano-Pabon M *et al.* (2011) A systematic review of environmental factors and obesogenic dietary intakes among adults: are we getting closer to understanding obesogenic environments? *Obes Rev* **12**, e95–e106.
14. Fleischhacker SE, Evenson KR, Rodriguez DA *et al.* (2011) A systematic review of fast food access studies. *Obes Rev* **12**, e460–e471.
15. Mackenbach JD, Nelissen KGM, Dijkstra SC *et al.* (2019) A systematic review on socioeconomic differences in the association between the food environment and dietary behaviors. *Nutrients* **11**, 2215.
16. Morland K, Wing S, Diez Roux A *et al.* (2002) The contextual effect of the local food environment on residents' diets: the Atherosclerosis Risk in Communities Study. *Am J Public Health* **92**, 1761–1767.



17. Hickson DA, Diez Roux AV, Smith AE *et al.* (2011) Associations of fast food restaurant availability with dietary intake and weight among African Americans in the Jackson Heart Study, 2000–2004. *Am J Public Health* **101**, S301–S309.
18. Burgoine T, Forouhi NG, Griffin SJ *et al.* (2016) Does neighborhood fast-food outlet exposure amplify inequalities in diet and obesity? A cross-sectional study. *Am J Clin Nutr* **103**, 1540–1547.
19. Madrigal JM, Cedillo-Couvert E, Ricardo AC *et al.* (2020) Neighborhood food outlet access and dietary intake among adults with chronic kidney disease: results from the chronic renal insufficiency cohort study. *J Acad Nutr Diet* **120**, 1151–1162.
20. Oexle N, Barnes TL, Blake CE *et al.* (2015) Neighborhood fast food availability and fast food consumption. *Appetite* **92**, 227–232.
21. Bild DE, Bluemke DA, Burke GL *et al.* (2002) Multi-Ethnic Study of Atherosclerosis: objectives and design. *Am J Epidemiol* **156**, 871–881.
22. Mayer-Davis EJ, Vitolins MZ, Carmichael SL *et al.* (1999) Validity and reproducibility of a food frequency interview in a multi-cultural epidemiology study. *Ann Epidemiol* **9**, 314–324.
23. Nettleton JA, Steffen LM, Mayer-Davis EJ *et al.* (2006) Dietary patterns are associated with biochemical markers of inflammation and endothelial activation in the Multi-Ethnic Study of Atherosclerosis (MESA). *Am J Clin Nutr* **83**, 1369–1379.
24. Nettleton JA, Rock CL, Wang Y *et al.* (2009) Associations between dietary macronutrient intake and plasma lipids demonstrate criterion performance of the Multi-Ethnic Study of Atherosclerosis (MESA) food-frequency questionnaire. *Br J Nutr* **102**, 1220–1227.
25. Abiemo EE, Alonso A, Nettleton JA *et al.* (2013) Relationships of the Mediterranean dietary pattern with insulin resistance and diabetes incidence in the Multi-Ethnic Study of Atherosclerosis (MESA). *Br J Nutr* **109**, 1490–1497.
26. Gao SK, Beresford SA, Frank LL *et al.* (2008) Modifications to the healthy eating index and its ability to predict obesity: the Multi-Ethnic Study of Atherosclerosis. *Am J Clin Nutr* **88**, 64–69.
27. Levitan EB, Ahmed A, Arnett DK *et al.* (2016) Mediterranean diet score and left ventricular structure and function: the Multi-Ethnic Study of Atherosclerosis. *Am J Clin Nutr* **104**, 595–602.
28. Nettleton JA, Schulze MB, Jiang R *et al.* (2008) A priori-defined dietary patterns and markers of cardiovascular disease risk in the Multi-Ethnic Study of Atherosclerosis (MESA). *Am J Clin Nutr* **88**, 185–194.
29. Nettleton JA, Steffen LM, Ni H *et al.* (2008) Dietary patterns and risk of incident type 2 diabetes in the Multi-Ethnic Study of Atherosclerosis (MESA). *Diabetes care* **31**, 1777–1782.
30. Guenther PM, Casavale KO, Reedy J *et al.* (2013) Update of the Healthy Eating Index: HEI-2010. *J Acad Nutr Diet* **113**, 569–580.
31. Kennedy ET, Ohls J, Carlson S *et al.* (1995) The Healthy Eating Index: design and applications. *J Am Diet Assoc* **95**, 1103–1108.
32. United States Department of Agriculture & United States Department of Health and Human Services (2010) *Dietary Guidelines For Americans, 2010*, 7th ed. Washington, D.C.: United States Department of Agriculture & United States Department of Health and Human Services.
33. Guenther PM, Kirkpatrick SI, Reedy J *et al.* (2014) The Healthy Eating Index-2010 is a valid and reliable measure of diet quality according to the 2010 Dietary Guidelines for Americans. *J Nutr* **144**, 399–407.
34. NCI (2014) The Healthy Eating Index (HEI): Tools for Researchers – Healthy Eating Index-2010. National Cancer Institute, Applied Research Program, Risk Factor Monitoring and Methods. <https://www.cnpp.usda.gov/healthyeatingindex> (accessed March 2015).
35. NCI (2016) Food Frequency Questionnaire at a Glance. National Cancer Institute; available at <https://dietaassessmentprimer.cancer.gov/profiles/questionnaire/> (accessed December 2016).
36. Arem H, Reedy J, Sampson J *et al.* (2013) The Healthy Eating Index 2005 and risk for pancreatic cancer in the NIH-AARP study. *J Natl Cancer Inst* **105**, 1298–1305.
37. Harmon BE, Boushey CJ, Shvetsov YB *et al.* (2015) Associations of key diet-quality indexes with mortality in the multiethnic cohort: the dietary patterns methods project. *Am J Clin Nutr* **101**, 587–597.
38. Reedy J, Krebs-Smith SM, Miller PE *et al.* (2014) Higher diet quality is associated with decreased risk of all-cause, cardiovascular disease, and cancer mortality among older adults. *J Nutr* **144**, 881–889.
39. Liese AD, Krebs-Smith SM, Subar AF *et al.* (2015) The dietary patterns methods project: synthesis of findings across cohorts and relevance to dietary guidance. *J Nutr* **145**, 393–402.
40. Auchincloss AH, Moore KA, Moore LV *et al.* (2012) Improving retrospective characterization of the food environment for a large region in the United States during a historic time period. *Health Place* **18**, 1341–1347.
41. Walls & Associates (2010) *National Establishment Time-Series (NETS) Database®: Database Description*. Oakland, CA; available at <https://msbfile03.usc.edu/digitalmeasures/cswenson/intellcont/NETS%20Database%20Description2008-1.pdf> (accessed January 2021).
42. Hume S (2009) Restaurants and Institutions 2009 Top 400 Restaurants Chains. Reed Business Information, a division of Reed Elsevier Inc.; available at <http://www.rimag.com/info/CA6670228.html> (accessed January 2010).
43. ESRI (2020) *ArcGIS Platform: How Kernel Density Works*. Redlands, CA: Environmental Systems Research Institute; available at <https://pro.arcgis.com/en/pro-app/tool-reference/spatial-analyst/how-kernel-density-works.htm> (accessed August 2020).
44. Hillier A, Cannuscio CC, Karpyn A *et al.* (2011) How far do low-income parents travel to shop for food? Empirical evidence from two urban neighborhoods. *Urban Geogr* **32**, 712–729.
45. Hirsch JA & Hillier A (2013) Exploring the role of the food environment on food shopping patterns in Philadelphia, PA, USA: a semiquantitative comparison of two matched neighborhood groups. *Int J Environ Res Public Health* **10**, 295–313.
46. Thornton LE, Bentley RJ & Kavanagh AM (2009) Fast food purchasing and access to fast food restaurants: a multilevel analysis of VicLANES. *Int J Behav Nutr Phys Act* **6**, 28.
47. Boone-Heinonen J, Gordon-Larsen P, Kiefe CI *et al.* (2011) Fast food restaurants and food stores: longitudinal associations with diet in young to middle-aged adults: the CARDIA study. *Arch Intern Med* **171**, 1162–1170.
48. Muthén LK & Muthén BO (1998–2017) *Mplus User's Guide*, 8th ed. Los Angeles, CA: Muthén & Muthén.
49. Feingold A, MacKinnon DP & Capaldi DM (2019) Mediation analysis with binary outcomes: direct and indirect effects of pro-alcohol influences on alcohol use disorders. *Addict Behav* **94**, 26–35.
50. Raykov T & Marcoulides GA (2006) *A First Course in Structural Equation Modeling*, 2nd ed. Mahwah, New Jersey: Lawrence Erlbaum Associates, Inc.
51. Little TD, Card NA, Bovaird JA *et al.* (2007) Structural equation modeling of mediation and moderation with contextual factors. In *Modeling Contextual Effects in Longitudinal Studies*, pp. 207–230 [TD Little, JA Bovaird & NA Card, editors]. Mahwah, NJ: Lawrence Erlbaum Associates.



52. MacKinnon DP, Fairchild AJ & Fritz MS (2007) Mediation analysis. *Annu Rev Psychol* **58**, 593–614.
53. Census (2012) Income, Poverty and Health Insurance Coverage in the United States: 2011. Washington, DC: U.S. Department of Commerce & United State Census Bureau; available at <https://www.census.gov/prod/2012pubs/p60-243.pdf> (accessed January 2021).
54. CNPP (2015) Healthy Eating Index 2010 (HEI). Alexandria, VA: United States Department of Agriculture & Center for Nutrition Policy and Promotion; available at <https://www.fns.usda.gov/hei-scores-americans> (accessed December 2015).
55. Jeffery RW, Baxter J, McGuire M *et al.* (2006) Correction: are fast food restaurants an environmental risk factor for obesity? *Int J Behav Nutr Phys Act* **3**, 35–35.
56. Penney TL, Burgoine T & Monsivais P (2018) Relative density of away from home food establishments and food spend for 24,047 households in England: a cross-sectional study. *Int J Environ Res Public Health* **15**, 2821.
57. Paquet C, Daniel M, Knauper B *et al.* (2010) Interactive effects of reward sensitivity and residential fast-food restaurant exposure on fast-food consumption. *Am J Clin Nutr* **91**, 771–776.
58. Nguyen BT & Powell LM (2014) The impact of restaurant consumption among US adults: effects on energy and nutrient intakes. *Public Health Nutr* **17**, 2445–2452.
59. Willett WC & Hu FB (2006) Not the time to abandon the food frequency questionnaire. *Cancer Epidemiol Biomark Prev* **15**, 1757–1758.
60. Pereira MA, Kartashov AI, Ebbeling CB *et al.* (2005) Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. *Lancet* **365**, 36–42.
61. Gallup (2017) *Americans' Dining-Out Frequency Little Changed From 2008*. Washington, D.C; available at <https://news.gallup.com/poll/201710/americans-dining-frequency-little-changed-2008.aspx> (accessed January 2021).
62. Drewnowski A, Aggarwal A, Hurvitz PM *et al.* (2012) Obesity and supermarket access: proximity or price? *Am J Public Health* **102**, e74–e80.
63. Fuller D, Cummins S & Matthews SA (2013) Does transportation mode modify associations between distance to food store, fruit and vegetable consumption, and BMI in low-income neighborhoods? *Am J Clin Nutr* **97**, 167–172.
64. Kant AK, Whitley MI & Graubard BI (2015) Away from home meals: associations with biomarkers of chronic disease and dietary intake in American adults, NHANES 2005–2010. *Int J Obes* **39**, 820–827.
65. Auchincloss AH, Young C, Davis AL *et al.* (2013) Barriers and facilitators of consumer use of nutrition labels at sit-down restaurant chains. *Public Health Nutr* **16**, 2138–2145.
66. Guthrie JF, Lin B-H & Frazao E (2002) Role of food prepared away from home in the American diet, 1977–78 v. 1994–96: changes and consequences. *J Nutr Educ Behav* **34**, 140–150.
67. Kant AK & Graubard BI (2018) A prospective study of frequency of eating restaurant prepared meals and subsequent 9-year risk of all-cause and cardiometabolic mortality in US adults. *PLoS One* **13**, e0191584.
68. Perez-Ferrer C, Auchincloss AH, de Menezes MC *et al.* (2019) The food environment in Latin America: a systematic review with a focus on environments relevant to obesity and related chronic diseases. *Public Health Nutr* **22**, 3447–3464.