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ORIGINAL RESEARCH

# Association of Fast-Food and Full-Service Restaurant Densities With Mortality From Cardiovascular Disease and Stroke, and the Prevalence of Diabetes Mellitus

Mohsen Mazidi, PhD; John R. Speakman, DSc

*Background*—We explored whether higher densities of fast-food restaurants (FFRs) and full-service restaurants are associated with mortality from cardiovascular disease (CVD) and stroke and the prevalence of type 2 diabetes mellitus (T2D) across the mainland United States.

*Methods and Results*—In this cross-sectional study county-level data for CVD and stroke mortality, and prevalence of T2D, were combined with per capita densities of FFRs and full-service restaurants and analyzed using regression. Mortality and diabetes mellitus prevalence were corrected for poverty, ethnicity, education, physical inactivity, and smoking. After adjustment, FFR density was positively associated with CVD ($\beta$=1.104, $R^2$=2.3%), stroke ($\beta$=0.841, $R^2$=1.4%), and T2D ($\beta$=0.578, $R^2$=0.6%) and full-service restaurant density was positively associated with CVD mortality ($\beta$=0.19, $R^2$=0.1%) and negatively related to T2D prevalence ($\beta$=−0.25, $R^2$=0.3%). In a multiple regression analysis (FFRs and full-service restaurants together in same model), only the densities of FFRs were significant (and positive). If we assume these relationships are causal, an impact analysis suggested that opening 10 new FFRs in a county would lead to 1 extra death from CVD every 42 years and 1 extra death from stroke every 55 years. Repeated nationally across all counties, that would be an extra 748 CVD deaths and 567 stroke deaths (and 390 new cases of T2D) over the next 10 years.

*Conclusions*—These results suggest that an increased density of FFRs is associated with increased risk of death from CVD and stroke and increased T2D prevalence, but the maximal impact (assuming the correlations reflect causality) of each individual FFR is small.

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**Key Words:** cardiology • epidemiology • statistical analysis • stroke

Cardiovascular diseases (CVDs) are the main global cause of death, currently accounting for >17.3 million deaths annually, a figure that is predicted to increase to >23.6 million by 2030.[1] Likewise, CVDs were the leading cause of death in the United States in 2015, accounting for 864 000 deaths.[2] There is little controversy over the benefit to cardiovascular health of eating a well-balanced diet and keeping active, as demonstrated in several large cohort studies.[1,3–5] These health behaviors also play an important role in other noncommunicable diseases, such as type 2 diabetes mellitus (T2D).[6,7]

Money spent on food away from home and energy consumed away from home have increased steadily in the United States over the past 50 years, paralleled by an increase in the availability and consumption of both fast foods and foods consumed at full-service establishments.[8–11] Fast foods are quickly served, convenient, relatively inexpensive, and liked by people of most age groups.[12] However, fast foods have high calorie densities; high contents of fat, sugar, and salt; large portion sizes; and high palatability.[12] It has been suggested that consuming fast food leads to weight and adiposity increases, which are a major risk factor for CVDs, noncommunicable diseases, and metabolic abnormalities, such as insulin resistance.[13–15] However, the evidence linking

From the State Key Laboratory of Molecular Developmental Biology, Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, Beijing, China (M.M., J.R.S.); University of the Chinese Academy of Sciences, Beijing, China (M.M.); and Institute of Biological and Environmental Science, University of Aberdeen, Scotland, United Kingdom (J.R.S.).

**Correspondence to:** John R. Speakman, DSc, State Key Laboratory of Molecular Developmental Biology, Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, Chaoyang, Beijing, China. E-mail: j.speakman@abdn.ac.uk

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## Clinical Perspective

### What Is New?

- The spread of fast-food restaurants (FFRs) and full-service restaurants and increased portion sizes are often blamed for the bad health of the nation.
- Yet, objective data supporting these popular notions are sparse.
- We sought associations between densities of FFRs and full-service restaurants and rates of mortality from cardiovascular disease and stroke across the mainland United States.
- Higher densities of FFRs were associated with higher mortality rates for both cardiovascular disease and stroke.
- The effect sizes, however, were small, with the impact of opening 10 new FFRs in a county leading to maximally 1 extra death from cardiovascular disease in 42 years and 1 extra death from stroke in 55 years.

### What Are the Clinical Implications?

- Lifestyle interventions are key to preventative medicine.
- The spread of FFRs is often regarded as a factor driving poor health choices.
- These data suggest that, although there is a positive statistical association between densities of FFRs and both cardiovascular disease and stroke mortality, the impact of opening an individual new FFR is exceedingly small.

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fast foods to obesity is confused.[16] There are some studies that have indicated that availability of fast-food restaurants (FFRs) is not associated with obesity prevalence,[17–19] shows only a weak positive association,[20,21] or is associated only in certain groups (eg, low-income individuals[22]). Yet, other studies have indicated a negative association between FFR availability and body weight status.[23] This confusion could stem from many different causes, such as different definitions of what constitutes an FFR, and unaccounted for confounding factors, such as socioeconomic status, education, poverty, and lifestyle (eg, engagement in physical activity and smoking).

Another scenario by which FFRs could have a deleterious impact on CVDs and noncommunicable diseases is through diet quality. Previous studies have shown fast-food consumption is associated with higher intakes of saturated fatty acids (SFAs), trans-fatty acids (TFAs), cholesterol, and sodium, and lower intakes of vitamin A, carotenes, vitamin C, calcium, and magnesium.[9,24] Fast-food consumption has been suggested to also be associated with decreased dietary intakes of whole grains, legumes, dairy, fruits, and vegetables, which are key sources of essential nutrients.[25] However, the association between neighborhood exposure to FFRs, consumption of fast food, and diet composition is inconsistent across studies.[18,26–28]

Some previous studies have reported the link between FFRs and T2D. For example, a cross-sectional study of 10 461 participants (mean age, 59 years) suggested that increased exposure to fast-food outlets is associated with increased risk of T2D.[29] Moreover, in another cohort consisting of adult respondents (aged 20–84 years) to the Canadian Community Health Survey, the researchers reported that among younger adults (aged 20–65 years), a greater proportion of fast food relative to all restaurants was significantly associated with incident diabetes mellitus.[30] However, we have recently reported a minor impact of both FFRs and full-service restaurants (FSRs) on the prevalence of obesity, which may translate to a low impact on prevalence of T2D, because obesity is the main risk factor for T2D.[31]

Although much of the focus on the increasing amount of energy consumed away from home as a driver for excess consumption has concerned FFRs, the calorie contents of meals at FSRs are generally larger than those consumed at FFRs.[32] Consequently, although meals are consumed at FSRs less frequently, calculations suggest that these out-of-home calories are contributed to almost equally by both FFRs and FSRs. For example we previously estimated that in the United States, adults, on average, consume 8.6% of their daily calorie intake at FFRs and 7.6% at FSRs.[33] Portion sizes have inflated at both FFRs and FSRs over time, and recent studies have suggested that 32% of FFRs and 22% of FSRs did not meet the guidelines for the nutritional quality of meals served to children.[34] Hence, although FSRs are slightly better than FFRs in this respect, they should not be considered as a "healthy alternative." In addition, although FFRs have generally been quick to publish the energy and nutritional contents of their products, full-service outlets have been much slower to do so.[35] Therefore, customers may be unaware of what they are consuming in such establishments and possible health impacts. This is supported by observations that customers at FSRs that provided menu labels with nutritional information consumed options with lower fat and calorie contents than those that did not.[36] If FFRs and FSRs are associated with elevated risk of CVD mortality, they might be a reasonable target for public health interventions aimed at reducing CVD and related conditions, including T2D. We, therefore, aimed to evaluate the association between both FFR and FSR densities, obtained from the US Department of Agriculture Economic Research Service, with mortality rates from CVD and stroke and the prevalence of T2D (from the Centers for Disease Control and Prevention [CDC]), controlling for the potential confounding effects of poverty, education, ethnicity, physical inactivity, and smoking (from the US census). Furthermore, we aimed to evaluate the impact of putative maximal impact of increasing the number of FFRs on incidence of T2D.

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# Methods

## Data Sources

The data, analytic methods, and study materials have been made available to other researchers for purposes of reproducing the results or replicating the procedure.[37] We used data on mortality rate (per 100 000 individuals) for CVD and stroke (between 2011 and 2013, aged >35 years) from the publicly available CDC web site (http://www.cdc.gov). CVD mortality was defined as the number of deaths per 100 000 person-years because of circulatory causes (*International Statistical Classification of Diseases, Tenth Revision* [*ICD-10*] codes I00–I99). Detailed information on preparation, definition, download, and sorting of data on prevalence of T2D (age adjusted), poverty, and ethnicity has been explained elsewhere.[38] In brief, county-level data on the prevalence of T2D were downloaded from the US CDC web site (http://www.cdc.gov). Data on T2D were estimated using data from the CDC Behavioral Risk Factor Surveillance System, which is a monthly state-based telephone survey of a nationally representative sample of adults aged >20 years. In 2012, the year for which these data were downloaded, the survey included landline telephones only and, hence, excluded individuals living in care homes or those without a landline telephone. More than 400 000 individuals are contacted annually to take part in the survey, which has been running since 1984. Individuals are judged to have diabetes mellitus if they respond "yes" to the question, "Has a doctor ever told you that you have diabetes?," excluding women who indicate in a follow-up question that they only had diabetes mellitus during pregnancy. Previous work indicates that self-report of a physician's prior diagnosis of diabetes mellitus is highly reliable compared with medical records.[39] This question does not separate those with type 1 diabetes mellitus and T2D. In the adult population of the United States, >96% of diabetes mellitus is type 2; we, therefore, called the estimated prevalence that of T2D. Given the magnitudes of the trends described herein, they cannot be attributed to differences in prevalence of type 1 diabetes mellitus. Data on rate of mortality (per 100 000 individuals) from CVD and stroke (between 2011 and 2013, aged >35 years) were also downloaded from the CDC web site (http://www.cdc.gov, National Vital Statistics System and National Center for Health Statistics). A previous variogram analysis has established that counties are an appropriate spatial level at which to explore the associations of factors to T2D prevalence.[38] Institutional review board approval was obtained by CDC, and each subject provided informed consent. All the data are already in the public domain from sites identified in the main text.[37]

## FFR and FSR Definition

We have provided detailed information on preparation, definition, download, and sorting of data on FFRs and FSRs elsewhere.[33] Briefly, density of FFRs and FSRs in 2012 was obtained from US Department of Agriculture Economic Research Service (http://www.ers.usda.gov) and food environment atlas.[33] The aim of the atlas is to provide a spatial overview of a community's ability to access food.[40] Density of the FFR (2012) was defined as the number of FFRs in the county per 1000 county residents. Fast-food or "limited-service" restaurants (defined by North American Industry Classification System code 722211) include establishments primarily engaged in providing food (except snack and nonalcoholic beverage bars), where customers generally order or select items and pay before eating. Food and drink may be consumed on premises, taken out, or delivered to the customer's location. FSRs (2012) were defined as number of FSRs in the county per 1000 county residents. FSRs (defined by North American Industry Classification System code 722110) include establishments primarily engaged in providing food services to customers who order and are served while seated (ie, waiter/waitress service) and pay after eating.

## Adjustment Variables

Data of county-level poverty (percentage in poverty and average income) and ethnicity were obtained from the US Census, 2010 census data (http://www.census.gov/2010census), specifically files PVY01, PVY02, INC01, INCO2, INCO3, IPE01, and RHI02. The distributions of ethnicity data showed heteroscedasticity in the variance. We tried various transformations to remove this, but none were completely successful. The outcome of the final analysis was robust to the type of data transformation used. The analysis presented is based on arcsine transformation of percentage values. Poverty was defined by US census, as follows: poverty status is defined by family (either everyone in the family is in poverty or no one in the family is in poverty). The characteristics of the family used to determine the poverty threshold are as follows: number of people, number of related children <18 years, and whether the primary householder is >65 years. Family income is then compared with the poverty threshold; if that family's income is lower than that threshold, the family is in poverty. Data on smoking (the percentage of adults who responded "yes" to the question, "Do you smoke cigarettes now?") and physical inactivity were obtained from the county health rankings and roadmaps organization (http://www.countyhealthrankings.org). This is a collaboration of the Robert Wood Johnson Foundation and the University of Wisconsin Population Health Institute. The organization compiles data at the county level from several national government sources, including health

behaviors, clinical care, social and economic factors, and aspects of the physical environment.

## Statistical Analysis

All variables were tested for normality and, where necessary, were normalized by log or arcsine transformation. Because it was impossible to adequately normalize the restaurant data when counties where no FFR and FSR were located were included, we excluded these counties (no FFR, n=51; or no FSR, n=8) from the analysis, and then took the natural logarithm of FFR and FSR (both +1) to normalize the data. Each county was identified by the Federal Information Processing Standard code, which is a 5-digit code that allows counties and county equivalent units to be uniquely identified. Apart from determining the crude association between the dependent factors and the county-level densities of FFRs and FSRs, we corrected the mortality from CVD and stroke and the age-adjusted prevalence of T2D for county-level poverty (percentage in poverty), ethnicity (percentage black), and education (percentage with bachelor's degree or higher), which we have previously established are significant confounding factors for the FFR and FSR densities.[33] We performed stepwise multiple linear regression analyses, including all the potential confounding factors as independent variables (poverty, ethnicity, education, physical inactivity, and smoking) and CVD mortality, stroke mortality, or prevalence of T2D as the dependent variables. We then derived the standardized residuals from these regressions and used these as the dependent variables in simple linear regressions with the densities of FFRs or FSRs. We have previously run clustering analysis for the prevalence of obesity and diabetes mellitus; however, there was no sign of clustering at the level of county.

The correlation of incidence to prevalence for T2D was 0.98. Herein, we report only the relationships for prevalence, because the relationships for incidence were identical. We have also performed multiple linear regression to better reflect the food environment (simultaneously evaluating impact of FFRs and FSRs) on our outcomes of interest (CVD mortality, stroke mortality, and T2D prevalence). To run the multiple linear regressions, we had 3 models: crude, partially adjusted (poverty, ethnicity, and education), and fully adjusted (poverty, ethnicity, education, smoking, and physical inactivity). We performed the analysis in this stepwise manner because we had previously shown that poverty, ethnicity, and education are associated with the densities of FFRs and FSRs. However, for physical inactivity and smoking, such associations were unknown. We investigated the relationships between the densities of the 2 restaurant types and the levels of physical inactivity and smoking using simple linear regression. We then included these variables into the third

"fully adjusted" model. In the partially and fully adjusted models, we derived the residuals for each of the health parameters in relation to the stated variables, and then we ran multiple linear regressions, including both FFRs and FSRs simultaneously in the model as independent predictor variables. The densities of FFRs and FSRs were weakly correlated across counties ($R^2$=0.059) but not to an extent that would create major issues of collinearity (variance inflation ratio, 1.15) in such a multiple regression analysis. In the database, there were 2 counties with much higher numbers of restaurants per capita. We performed the analysis including or excluding these counties, and it had no impact on the findings, so we have included them into the final analysis. All the analyses were performed using SPSS, version 11.5 (SPSS, Chicago, IL). $P \leq 0.05$ was considered statistically significant.

## Putative Impact Analysis

To evaluate the putative maximal impact of increasing the numbers of FFRs on the basis of the β coefficients of the fitted regressions, we calculated the expected impact of opening an additional 10 FFRs in a given county on the numbers of deaths from CVD and stroke and the number of new cases of T2D (assuming the relationships we observed are causal). Because the average number of FFRs per county is currently 60, this increase represented an increase of 17% in the numbers of FFRs. To calculate the impact of opening these restaurants, we used the effect size of the FFRs on CVD, stroke, and diabetes mellitus (β value). If 10 new restaurants are opened in a county, then given the average county population of adults >35 years old is 65 725, this would be 0.153 new restaurants per 1000 people in that age class. The current mean number of restaurants per 1000 people is 0.954 (62 per county). Hence, opening the 10 new restaurants elevates this to 1.106. Logging these values and inserting them into the fully adjusted model equation {$y$=1.1034 [Log ($x$+1)]−0.514} gives a residual mortality rate of −0.193 deaths per year per 100 000 population currently, which increases to −0.157 after the new restaurants have opened. We have performed the same analysis for the mortality from stroke and T2D.

## Results

There was a significant negative relationship between both unadjusted CVD and stroke mortality with the density of FFRs (Table 1). Age-adjusted T2D prevalence also had a significant negative relationship with FFRs (Table 1). Densities of FSRs were also significantly negatively associated with CVD mortality (Table 1). In addition, we observed a strong highly

**Table 1.** Details of Association Between FFRs and FSRs With Mortality From CVD and Stroke and T2D Prevalence Analysis on the Basis of Different Models

| Models | | β Value | 95% CI | P Value |
|---|---|---|---|---|
| Simple linear regressions | | | | |
| 1 | CVD mortality vs FFR | −75.83 | −94.57 to −57.33 | <0.001 |
| 2 | Stroke mortality vs FFR | −9.30 | −12.93 to −5.62 | <0.001 |
| 3 | T2D corrected for age vs FFR | −2.07 | −2.53 to −1.60 | <0.001 |
| 4 | CVD mortality vs FSR | −126.5 | −137.77 to −115.40 | <0.001 |
| 5 | Stroke mortality vs FSR | −18.85 | −21.14 to −16.61 | <0.001 |
| 6 | T2D corrected for age vs FSR | −4.20 | −4.47 to −3.94 | <0.001 |
| 7 | CVD mortality corrected for poverty, ethnicity, and education vs FFR | 0.26 | 0.03 to 0.48 | 0.022 |
| 8 | Stroke mortality corrected for poverty, ethnicity, and education vs FFR | 0.28 | 0.05 to 0.51 | 0.013 |
| 9 | T2D corrected for age, poverty, ethnicity, and education vs FFR | −0.09 | −0.31 to 0.12 | 0.403 |
| 10 | CVD mortality corrected for poverty, ethnicity, and education vs FSR | −0.46 | 0.60 to −0.32 | <0.001 |
| 11 | Stroke mortality corrected for poverty, ethnicity, and education vs FSR | −0.19 | −0.33 to −0.04 | 0.010 |
| 12 | T2D corrected for age, poverty, ethnicity, and education vs FSR | −0.87 | −1.01 to −0.73 | <0.001 |
| 13 | CVD mortality corrected for poverty, ethnicity, education, physical inactivity, and smoking vs FFR | 1.10 | 0.81 to 1.39 | <0.001 |
| 14 | Stroke mortality corrected for poverty, ethnicity, education, physical inactivity, and smoking vs FFR | 0.83 | 0.55 to 1.30 | <0.001 |
| 15 | T2D corrected for age, poverty, ethnicity, education, physical inactivity, and smoking vs FFR | 0.57 | 0.29 to 0.86 | <0.001 |
| 16 | CVD mortality corrected for poverty, ethnicity, education, physical inactivity, and smoking vs FSR | 0.19 | 0.01 to 0.37 | 0.036 |
| 17 | Stroke mortality corrected for poverty, ethnicity, education, physical inactivity, and smoking vs FSR | 0.13 | −0.05 to 0.31 | 0.157 |
| 18 | T2D corrected for age, poverty, ethnicity, education, physical inactivity, and smoking vs FSR | −0.25 | −0.43 to −0.07 | 0.006 |

Linear regressions were conducted. Both FFR and FSR were log transformed. Standardized residual techniques were used for the multivariable regressions. CI indicates confidence interval; CVD, cardiovascular disease; FFR, number of fast-food restaurants per 1000 population; FSR, number of full-service restaurants per 1000 population; T2D, type 2 diabetes mellitus.

significant negative association between the density of FSRs with age-adjusted T2D prevalence (Table 1).

We reported previously that both FFRs and FSRs were found more frequently in areas where the population was richer ($R^2$=12.2% and $R^2$=8.9%, respectively; both $P$<0.001) and were more highly educated ($R^2$=13.3% and $R^2$=10.3%, respectively; both $P$<0.001); for FSRs, there was a lower proportion of blacks ($R^2$=12.6%, $P$<0.001).[33] Once we adjusted the mortality rate of CVD and stroke for poverty, ethnicity, and education, a significant positive association with the density of FFRs was observed for both CVD and stroke mortality (Table 1).With respect to T2D, adjusting for the same confounding factors removed the significant association between FFRs and T2D (Table 1). We obtained significant negative, but much weaker, associations between the densities of FSRs with mortality from CVD and stroke and the prevalence of T2D after adjustment for poverty, ethnicity, and education (Table 1).

Once we adjusted the mortality rate of CVD and stroke for these 3 factors (poverty, ethnicity, and education), FFRs and FSRs together in a multiple regression analysis explained 2.2% and 1.0%, respectively, of the variation in CVD and stroke

mortality (CVD mortality: FFR: β=0.63 [95% confidence interval (CI), 0.39–0.88]; $P$<0.001; FSR: β=−0.65 [95% CI, −0.82 to −0.49]; $P$<0.001; overall $P$<0.001; and stroke mortality: FFR: β=0.53 [95% CI, 0.29–0.77]; $P$<0.001; FSR: β=−0.38 [95% CI, −0.55 to −0.22]; $P$<0.001; Table 2). In these cases, both FFR and FSR densities had independent and significant effects. In a similar multiple regression analysis, FFRs and FSRs together explained 5.1% of the variation in prevalence of T2D after correction for age, poverty, ethnicity, and education (FFR: β=0.48 [95% CI, 0.25–0.72]; $P$<0.001; FSR: β=−1.04 [95% CI, −1.21 to −0.88]; $P$<0.001; overall $P$<0.001; Table 2). Densities of both restaurants were significant.

Both FFRs and FSRs were more frequently found in areas where the population was less physically inactive (FFR: β=−0.07 [95% CI, −0.008 to −0.006]; FSR: β=−0.019 [95% CI, −0.020 to −0.017]; both $P$<0.001). Moreover, there was a greater percentage of smokers in locations with lower densities of both establishments (FFR: β=−0.03 [95% CI, −0.04 to −0.02]; FSR: β=−0.012 [95% CI, −0.013 to −0.011]; both $P$<0.001). Consequently, we further adjusted the disease mortalities and prevalence for physical inactively and smoking. Once these factors were also taken into

**Table 2.** Details of Multiple Linear Regressions for FFR and FSR With Dependent Variables (Mortality From CVD and Stroke and T2D Prevalence)

| Multiple Linear Regressions | β Value | 95% CI | P Value |
|---|---|---|---|
| Crude* | | | |
| FFR and FSR with CVD mortality | FFR: β=−1.45 | −20.40 to 17.49 | 0.880 |
| | FSR: β=−134.54 | −147.52 to −4121.56 | <0.001 |
| FFR and FSR with stroke mortality | FFR: β=2.89 | −0.91 to 6.69 | 0.136 |
| | FSR: β=−21.30 | −23.91 to −18.69 | <0.001 |
| FFR and FSR with T2D prevalence | FFR: β=0.42 | −0.02 to 0.86 | 0.064 |
| | FSR: β=−4.52 | −4.82 to −4.21 | 0.001 |
| Adjusted[†] | | | |
| FFR and FSR with CVD mortality | FFR: β=0.63 | 0.39 to 0.88 | <0.001 |
| | FSR: β=−0.65 | −0.82 to −0.49 | <0.001 |
| FFR and FSR with stroke mortality | FFR: β=0.53 | 0.29 to 0.77 | <0.001 |
| | FSR: β=−0.38 | −0.55 to −0.22 | <0.001 |
| FFR and FSR with T2D prevalence | FFR: β=0.48 | 0.25 to 0.72 | <0.001 |
| | FSR: β=−1.04 | −1.21 to −0.88 | <0.001 |
| Adjusted[‡] | | | |
| FFR and FSR with CVD mortality | FFR: β=1.10 | 0.80 to 1.40 | <0.001 |
| | FSR: β=0.03 | −0.16 to 0.23 | 0.733 |
| FFR and FSR with stroke mortality | FFR: β=0.89 | 0.58 to 1.19 | <0.001 |
| | FSR: β=−0.05 | −0.25 to 0.14 | 0.595 |
| FFR and FSR with T2D prevalence | FFR: β=0.76 | 0.46 to 1.06 | <0.001 |
| | FSR: β=−0.35 | −0.55 to −0.15 | 0.733 |

Multiple linear regressions were conducted. Both FFR and FSR were log transformed. CI indicates confidence interval; CVD, cardiovascular disease; FFR, number of fast-food restaurants per 1000 population; FSR, number of full-service restaurants per 1000 population; T2D, type 2 diabetes mellitus.
*T2D prevalence is adjusted for age in all the models.
[†]CVD, stroke, and T2D adjusted for poverty, ethnicity, and education.
[‡]CVD, stroke, and T2D adjusted for poverty, ethnicity, education, physical inactivity, and smoking.

account (full model adjusted for poverty, ethnicity, education, physical inactivity, and smoking), the density of FFRs was positively associated with mortality from CVD (β=1.10 [95% CI, 0.81–1.39]; $R^2$=2.3%; P<0.001; Figure 1A and Table 1) and stroke (β=0.84 [95% CI, 0.55–1.30]; $R^2$=1.4%; P<0.001; Figure 2A and Table 1) and age-adjusted prevalence of T2D (β=0.57 [95% CI, 0.29–0.86]; $R^2$=0.6%; P<0.001; Figure 3A and Table 1). By this model, a unit increase in the density of FFRs per 1000 people would be anticipated to lead to 1.10 more deaths per 100 000 people per annum from CVD and 0.84 more deaths per 100 000 populations per annum from stroke. In addition, a unit increase in the density of FFRs per 1000 people would be anticipated to lead to a 0.57% higher prevalence of age-adjusted T2D. In the model that adjusted for poverty, ethnicity, education, physical inactivity, and smoking, CVD mortality was positively associated with density of FSRs (β=0.19 [95% CI, 0.01–0.37]; $R^2$=0.1%; P=0.036; Figure 1B and Table 1), whereas mortality from stroke was no longer

associated with the density of FSRs (P=0.157; Figure 2B and Table 1). Prevalence of age-adjusted T2D was still weakly and negatively associated with the density of FSRs (β=−0.25 [95% CI, −0.43 to −0.07]; $R^2$=0.3%; P=0.006; Figure 3B and Table 1) after adjustment for age, poverty, ethnicity, education, physical inactivity, and smoking. Because of the large sample size, this was still statistically significant. By this model, a unit increase in the density of FSRs per 1000 people would be anticipated to lead to 0.19 more per 100 000 people per annum from CVD and a 0.25% reduction in the prevalence of age-adjusted T2D.

In a multiple regression analysis, FFRs and FSRs together explained 14.0% of the variation of CVD mortality (FFR: β=−1.45 [95% CI, −20.40 to 17.49]; P=0.880; FSR: β=−134.54 [95% CI, −147.52 to −4121.56]; P<0.001; overall P<0.001; Table 2). They (FFRs+FSRs) also explained 8.6% of the variation in mortality from stroke (FFR: β=2.89 [95% CI, −0.91 to 6.69]; P=0.136; FSR: β=−21.30 [95% CI, −23.91 to −18.69]; P<0.001; overall P<0.001; Table 2).
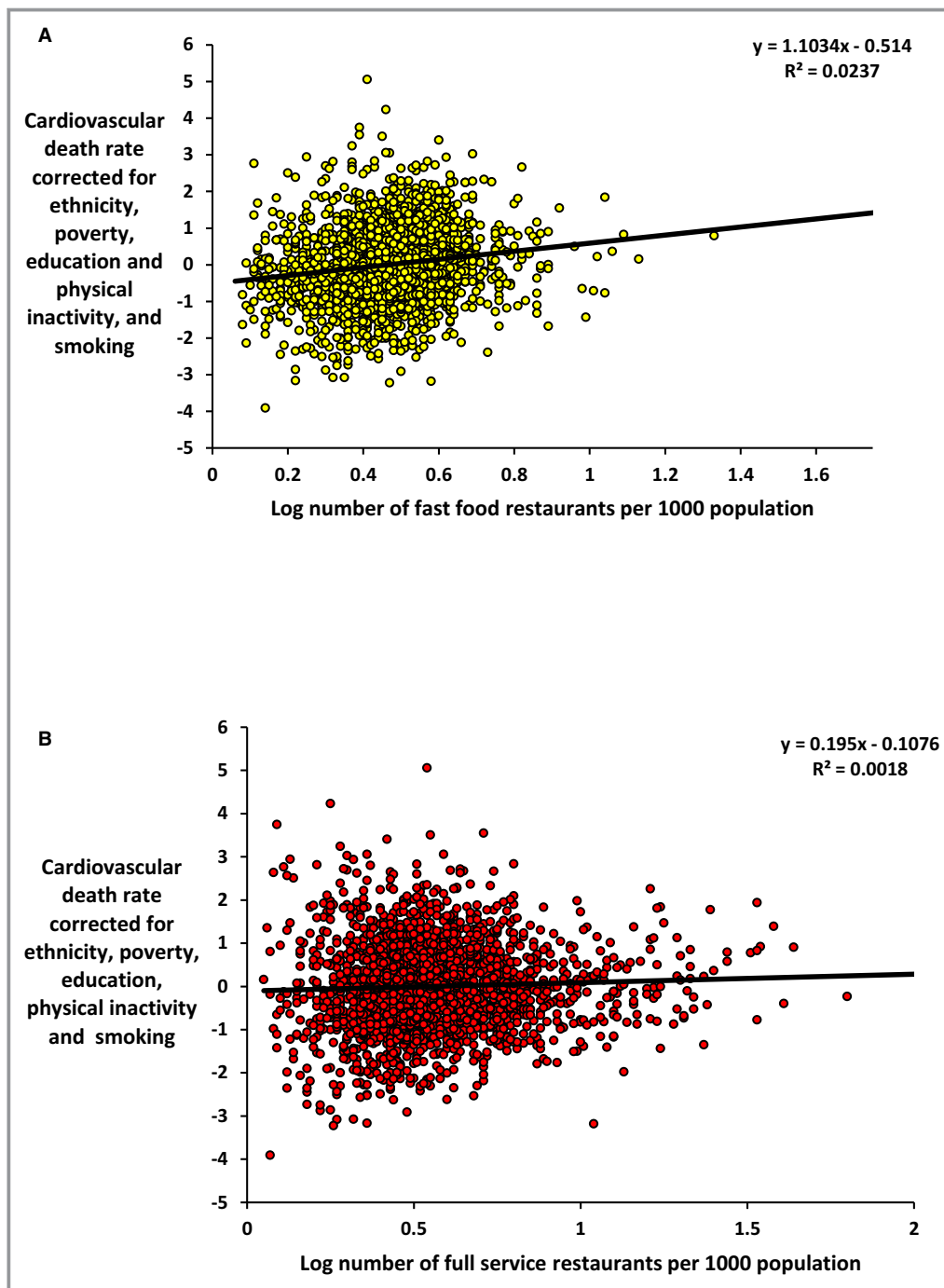
**Figure 1.** Association between density of fast-food restaurants (FFRs) and full-service restaurants (FSRs) with corrected mortality of cardiovascular disease (CVD) across the mainland United States. A, CVD mortality corrected for ethnicity, poverty, education, percentage physical inactivity, and smoking against log-transformed number of FFRs per 1000 population (+1). B, CVD mortality corrected for ethnicity, poverty, education, percentage physical inactivity, and smoking against log-transformed number of FSRs per 1000 population (+1).

Finally, together, they explained 23.9% of the variation in T2D (age-adjusted) prevalence (FFR: $\beta$=0.42 [95% CI, −0.02 to 0.86]; $P$=0.064; FSR: $\beta$=−4.52 [95% CI, −4.82 to −4.21]; $P$<0.001; overall $P$<0.001; Table 2). In all cases, the effect of FSRs was negative and highly significant, but the effect of FFRs was not significant, suggesting the negative effect of the FFRs was mostly because of the weak correlation between numbers of FFRs and FSRs across counties ($r^2$=0.059).
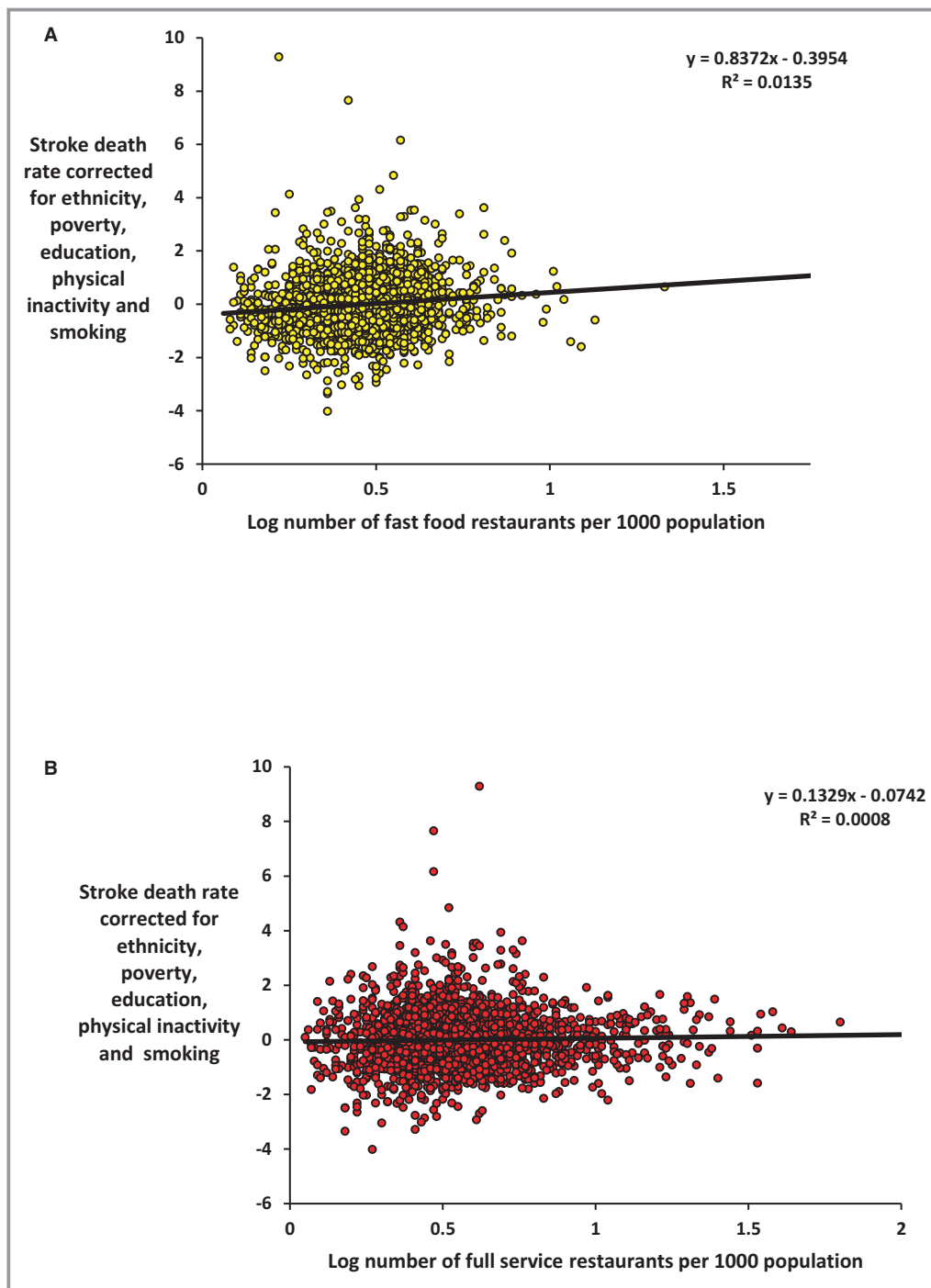
**Figure 2.** Association between density of fast-food restaurants (FFRs) and full-service restaurants (FSRs) with corrected mortality of stroke across the mainland United States. A, Stroke mortality corrected for ethnicity, poverty, education, percentage physical inactivity, and smoking against log-transformed number of FFRs per 1000 population (+1). B, Stroke mortality corrected for ethnicity, poverty, education, percentage physical inactivity, and smoking against log-transformed number of FSRs per 1000 population (+1).

Entering both restaurant types into a multiple regression model and the partially adjusted mortalities and T2D prevalence (adjusted for poverty, education, and race), FFRs and FSRs together explained 2.4% and 1.5%, respectively, of the spatial variation in mortality rate of CVD and stroke (CVD mortality: FFR: $\beta=1.10$ [95% CI, 0.80–1.40]; $P<0.001$; FSR: $\beta=0.03$ [95% CI, −0.16 to 0.23]; $P=0.733$; overall $P<0.001$; and stroke mortality: FFR: $\beta=0.89$ [95% CI, 0.58–1.19];
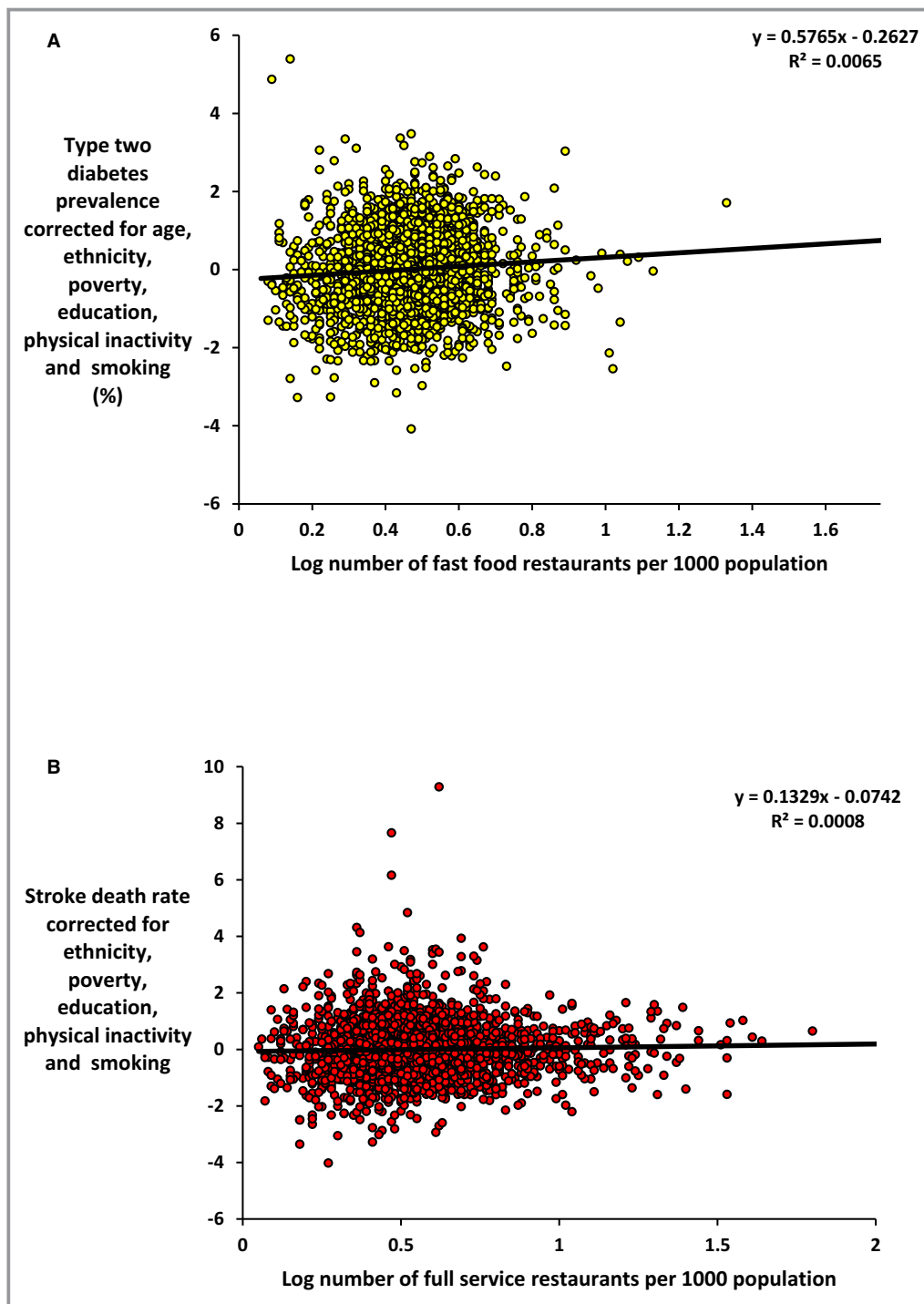
**Figure 3.** Association between density of fast-food restaurants (FFRs) and full-service restaurants (FSRs) with corrected type 2 diabetes mellitus (T2D) prevalence (age adjusted) across the mainland United States. A, Age-adjusted T2D prevalence corrected for ethnicity, poverty, education, percentage physical inactivity, and smoking against log-transformed number of FFRs per 1000 population (+1). B, Age-adjusted T2D prevalence corrected for ethnicity, poverty, education, percentage physical inactivity, and smoking against log-transformed number of FSRs per 1000 population (+1).

*P*<0.001; FSR: β=−0.05 [95% CI, −0.25 to 0.14]; *P*=0.595; overall *P*<0.001; Table 2). In these multiple regression models, it was now FSRs that had no significant effect. After full adjustment for age, poverty, ethnicity, education, physical inactivity, and smoking, FFRs and FSRs together explained 1.1% of variation in T2D prevalence (FFR: β=0.76 [95% CI,

0.46−1.06]; *t*=4.96; *P*<0.001; FSR: β=−0.35 [95% CI, −0.55 to −0.15]; *t*=−3.50; *P*=0.733; overall *P*<0.001; Table 2), with both densities showing significant, but opposing, effects. FFR density was positively, and FSR density was negatively, linked to age-adjusted T2D prevalence.

We evaluated the putative maximal impact of increasing the numbers of FFRs on mortality from CVD and stroke and prevalence of T2D on the basis of the β coefficients of the fully adjusted models. See "Methods" for further calculation details. The increase in mortality from CVD attributable to opening 10 new restaurants was 0.0363 deaths per 100 000 population per annum. Because the average county population is 65 725, then the extra deaths from CVD per annum amounts to 0.024 per year. That is 1 extra death per 42.3 years. The same calculation for stroke gives 1 death every 55.8 years; for T2D, it is 1 extra case per 81.1 years. Repeated nationally, across all counties, involving an additional 31 690 more FFRs would be maximally predicted to lead to 748 more deaths from CVD, 567 more deaths from stroke, and 390 new cases of T2D over the next 10 years.

## Discussion

We have investigated the associations of availability of FFRs and FSRs with mortality from CVD and stroke and the prevalence of age-adjusted T2D. In multiple regression models including both FFRs and FSRs, once we normalized for poverty, ethnicity, education, physical inactivity, and smoking, mortality from CVD and stroke and prevalence of T2D were all significantly positively associated with density of the FFRs. FSR densities had no impact on CVD and stroke mortality, but age-adjusted T2D prevalence was negatively related with availability of FSRs. Why FSRs do not associate with CVD and stroke, when FFRs do, is unclear. The energy contents of meals served in FSRs are generally larger than those consumed at FFRs,[11] but they are consumed less frequently, so their contributions to total calorie intake are approximately equal.[33] This suggests dietary components linked to CVD and stroke (eg, salt, SFA, and TFA) may have greater levels in FFRs than in FSRs.[41] The negative association between diabetes mellitus prevalence and FSRs was also found with respect to obesity[33] and presumably reflects an unaccounted for confounding factor.[33]

These cross-sectional correlational data on the association of greater densities of FFRs with both CVD and stroke mortality support other correlational data indicating a link between fast-food consumption and risk factors for CVD. For example, impaired metabolism of lipids and lipoproteins is a prominent cardiometabolic risk factor that is considered as an adverse effect of consuming foods in FFRs. The CARDIA (Coronary Artery Risk Development in Young Adults) study suggested that participants who consumed ≥2.5 compared with <0.5 meals/

week of fast-food meals had greater levels of triglycerides and lower high-density lipoprotein cholesterol.[15] Individuals in the third and fourth quartiles, compared with the first quartile, of fast-food intake also had greater odds of metabolic syndrome after 13 years of follow-up. In addition, the homeostatic model assessment of insulin resistance, an indicator of insulin resistance, was also positively associated with fast-food intake.[15] It has also been reported that postprandial lipemia and lipid peroxidation were both augmented after intake of a fast-food meal, compared with a healthy meal.[33] A 15-year follow-up of US women showed that higher fast-food intake (≥2× per week) was correlated with greater insulin resistance.[42] In an intervention study, 4-week consumption of fast-food meals (≥2 meals/day) in healthy subjects increased both insulin resistance index and hepatic triglyceride content.[43]

With respect to T2D, results from NHS (Nurses' Health Study) indicated that increased consumption of burgers, fried chicken meals, sausages, and other processed meat products was associated with an increased risk of developing T2D. Higher intake of French fries increased the 20-year risk of T2D by 21%.[44] The BWHS (Black Women's Health Study) indicated that the 10-year incidence rate of T2D increased by 40% for greater consumption of hamburgers and by 68% for greater consumption of fried chicken (≥2 meals/week compared with none).[45]

Several other studies using similar approaches to that used herein have also shown that accessibility to FFRs could be a risk factor for CVD. For example, in a study including 380 health regions in Ontario, Canada, risk-adjusted outcomes among regions with higher densities of FFRs were more likely to be high outliers for both mortality and acute coronary hospitalizations compared with regions with low FFR density.[46] Moreover, a smaller previous study in the United States covering 64 census tract areas in Texas revealed that residents of census tracts in the highest quartile for FFRs had a 13% greater risk of ischemic stroke than residents of census tracts in the lowest quartile for FFRs, and suggested that the risk of stroke grew 1% with every extra FFR.[47]

FFRs may play a role as a risk factor for noncommunicable diseases via 2 major contrasting mechanisms. First, the establishments may promote obesity, which is a major risk factor for CVD, stroke, and T2D. Alternatively, the diets may promote these disorders directly in an obesity-independent manner. We have previously shown, using the same county data,[33] that there is no significant association between the densities of FFRs and the age-adjusted prevalence of obesity (adjusted for poverty, education, and ethnicity), but not adjusted for physical inactivity, smoking, and density of FSRs. We suggested this is probably because the total energy consumption in FFRs (and FSRs) amounts to <20% of total dietary energy intake. It is much more likely, therefore, that if the associations we observed are causal, they come about

because of the quality of food served in FFRs, rather than the quantity. This could also explain why the effects of FSR density were not significant, if the nutritional quality of food in these establishments is higher.

Fast food is high in total fat and SFA, glycemic load, and glycemic index.[48] High-fat content and the composition of fatty acids (such as high in SFAs, TFAs, and cholesterol) of fast foods is a primary dietary risk factor for CVD. In some of the most popular FFRs, TFAs were up to 24 g/serving.[49] High contents of SFA and TFA are potent health-threatening fats that have been found to increase low-density lipoprotein cholesterol and triglycerides, decrease high-density lipoprotein cholesterol, and accelerate the development of CVD.[50–52] Moreover, SFA and cholesterol, which are components of red and processed meats that often form the basis of FFR-served meals, have been shown to adversely affect not only insulin sensitivity but also β-cell function.[53] Large portion size, a high amount of refined carbohydrates, and added sugar are other characteristics that could explain the negative impacts of FFRs, particularly with respect to the risk of T2D.[50]

This work has several limitations. Importantly, being cross sectional and correlative, the associations we found may not be causal. There may be additional confounding factors that are associated with both the densities of FFRs and mortality attributable to CVD and stroke and the prevalence of T2D that we did not adequately control for. Furthermore, the density of FFRs does not necessarily reflect geographic barriers to access or transport distances and pricing, all of which are factors that probably affect whether people choose to eat at an establishment or not, and thus define actual exposure to the food products available at these establishments. Similarly, we had no actual information on the dietary patterns and lifestyles of individuals living in areas with greater densities of FFRs, which may be important in the case of exposure to cardiometabolic risk factors. Most notably, we did not know the food habits of those individuals who actually died compared with those who did not.

If we assume the associations are causal, this allows us to model the *maximal* impact of FFRs on CVD and stroke mortality. These calculations suggested that opening 10 new FFR establishments in a given county would maximally result in 1 extra death in that county from CVD every 42.3 years and 1 extra death from stroke every 55.8 years. If this level of extra restaurants (an average increase of 17% in the density across all counties) was replicated across the whole country (comprising >30 000 new restaurants), it would amount to maximally 748 extra deaths from CVD and 567 extra deaths from stroke over the following 10 years. This highlights the difficulties facing planning officers who need to make decisions about individual applications to open fast-food establishments that, on their own, have a small effect. Yet, the national result of many such small decisions leads to the avoidable deaths of hundreds of people over the timescale that the restaurants are in business.

## Author Contributions

Mazidi: collection, analysis, and interpretation of data; and drafting of article. Speakman: study conception and design, critical revision, and interpretation of data.

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## Disclosures

Both authors declare no competing financial interests, conflicts of interest, or any direct association with the fast-food or full-service restaurant food industries. Speakman has historically had several links with food industries, none of which are directly involved with fast-food or full-service provision. In the interests of full disclosure, these links are described as follows. He was formerly a member of the Scientific Advisory Board of the Waltham Centre for Pet Nutrition (WCPN), which is a subsidiary of the Mars Corporation, and was remunerated for that role. He spoke twice at Waltham symposia on issues relating to energy balance and was also paid for these presentations. Between 1993 and 2006, he was also recipient of several grants from WCPN and cosupervised several students who worked on issues relating to pet nutrition (grants total, <$500 000). Speakman has made invited presentations at the headquarters of 2 major suppliers of beverages that are supplied in fast-food outlets, Pepsico (Houston, TX) and Coca Cola (Atlanta, GA), both times on the role of energy expenditure in the obesity epidemic, and was paid expenses for both these presentations.

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**Association of Fast–Food and Full–Service Restaurant Densities With Mortality From Cardiovascular Disease and Stroke, and the Prevalence of Diabetes Mellitus**

Mohsen Mazidi and John R. Speakman

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