Are exposures to ready-to-eat food environments associated with type 2 diabetes? A cross-sectional study of 347 551 UK Biobank adult participants

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Lancet Planet Health 2018; 2: e438–50

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Summary

Background Rapid urbanisation and associated socioeconomic transformations have modified current lifestyles, shifting dietary preferences towards ready-to-eat, calorie-dense food of poor nutritional quality. The effect of ready-to-eat food environments that sell food for instant consumption on the risk of type 2 diabetes has received scant attention. We therefore aimed to examine the association between exposure to ready-to-eat food environments and type 2 diabetes in a large and diverse population sample.

Methods We conducted a cross-sectional study of adult male and female participants from the baseline phase of the UK Biobank cohort. Participants in this cohort were aged 37–73 years and resided in one of 21 cities in the UK. Ready-to-eat food environments, which we determined from a modelled and linked built environment database, were objectively measured within 1-km catchment areas of the residential streets of participants and were expressed as metrics of density and proximity to the participants' homes. We used logistic regression models to examine the associations between exposure to ready-to-eat food environments and the odds of type 2 diabetes, adjusting for individual covariates such as physical activity. As sensitivity analyses, we investigated the associations between the street distance to the nearest ready-to-eat food outlet and type 2 diabetes. We also tested post hoc for effect modification by sex, income, body-mass index, and location of the UK Biobank collection centre.

Findings Of 502 635 UK Biobank participants enrolled between March 13, 2006, and Oct 1, 2010, the sample analysed included 347 551 (69·1%) participants. The density of ready-to-eat food environments within a 1-km catchment area was associated with higher odds of type 2 diabetes for participants in the groups with highest exposure to restaurants and cafeterias (odss ratio 1·129, 95% CI 1·05–1·21; p=0·0007) and a composite measure of ready-to-eat outlet density (1·112, 1·02–1·21; p=0·0134) compared with those with no exposure. Exposure to hot and cold takeaways was only significantly associated with higher odds of type 2 diabetes at the second highest exposure category that we examined (1·076, 1·01–1·14; p=0·0171), representing a density of 0·75–2·15 units per km². A protective effect with distance decay was observed: participants in the highest quintile of street distance to nearest ready-to-eat food outlet reported lower odds of type 2 diabetes than those in the lowest quintile (0·842, 0·78–0·91; p<0·0001 for restaurants and cafeterias; and 0·913, 0·85–0·98; p=0·0173 for hot and cold takeaways). These effects were most pronounced in overweight participants (p=0·0329), but there was no evidence of interaction by sex, income, or UK Biobank collection centre.

Interpretation Access to ready-to-eat food environments was positively associated with type 2 diabetes. Top-down policies aimed at minimising unhealthy food access could potentially reduce unhealthy consumption and risks of chronic diseases. Further long-term studies are needed to effectively guide such interventions.

Funding University of Hong Kong, UK Biobank, and UK Economic & Social Research Council.

Introduction

Diabetes affects 425 million people worldwide, accounting for US$727 billion (12%) of the global health-care budget.[1] In the UK, the number of adults diagnosed with diabetes was estimated to be approximately 3·7 million in 2017.[2] Type 2 diabetes is a complex chronic disease that is characterised by insulin resistance and relative insulin deficiency and is generally associated with excessive body fat and obesity.[3] Type 2 diabetes has been directly linked to an increased risk of mortality[4] and of cardiovascular diseases.[5,6] A UK-wide study has projected the economic burden of type 2 diabetes for 2035–36 at £15·1 billion accrued in direct costs and £20·5 billion in indirect costs.[7]

Simultaneously, decades of rapid urbanisation and associated socioeconomic transformations have resulted in a shift in lifestyles and dietary preferences.[8] High-income economies have moved from diets consisting of traditional home-cooked meals to those of ready-made food and takeaways of poorer nutritional value, low production costs, longer shelf-life, and considerable retail value-added. Low-income and middle-income countries are currently in the process of this transition. With

Research in context

Evidence before this study

We searched PubMed, MEDLINE, Scopus, Google Scholar, and Web of Science for studies and reports published up to April 20, 2018, with a combination of search terms, including "fast food outlet", "takeaways", "food environment" OR "obesogenic environment" AND "diabetes", and "type 2 diabetes" OR "physical activity". We also manually searched the reference lists of related papers. We limited our search to English language publications. The twin processes of urbanisation and globalisation have led to shifts in lifestyles and dietary preferences and proliferation of unhealthy food environments that supply calorific processed food. There is evidence of an association between access to food environments in urban neighbourhoods, especially categories and accessibility of specific food environments, dietary consumption patterns, and prevalence of chronic diseases. Type 2 diabetes has emerged as a public health challenge and there have only been a few studies that have examined the association between accessibility of unfavourable food environments and prevalence of type 2 diabetes. Evidence on the association between exposure to ready-to-eat food environments and type 2 diabetes has been scarce and ambiguous because most of the studies done thus far have been small scale with homogeneous population-level and exposure characteristics, and they have had an ecological design, which has limited generalisability and reliability.

Added value of this study

To our knowledge, this study is the largest in the UK to investigate the association between exposure to ready-to-eat food environments that sell food for instant consumption (pubs and bars, restaurants and cafeterias, and hot and cold takeaways, and a composite of the three) and the odds of type 2 diabetes. We used high-quality data regarding 347 551 participants who are middle-aged or older (aged 37–73 years) from the UK Biobank, which has substantial population-level and spatial variability. Exposure to ready-to-eat food environments that sell food for instant consumption was objectively assessed with metrics of the density of units selling ready-to-eat food within a catchment area of 1 km from a residential street and the street distance to the nearest ready-to-eat food outlet. The study reported 11% higher odds of type 2 diabetes amongst participants in the highest category of exposure to ready-to-eat food outlets within a 1-km catchment area (ie, those with >10·70 units/km^2) than those with no exposure, after adjustments for all other risk factors and covariates. A protective effect with distance decay was also observed: the odds of type 2 diabetes was approximately 10% lower among participants in the highest quintiles of street distance to the nearest ready-to-eat food outlet than among those in the lowest. The effect sizes were slightly bigger in the higher exposure category among women, those in middle-income groups, and those who were overweight, although a significant interaction was observed only among participants with a higher body-mass index.

Implications of all the available evidence

We found that access to ready-to-eat food outlets was associated with higher odds of type 2 diabetes in a nationwide population sample. Top-down interventions at a city scale, such as through constraining, via licensing or planning regulations, the location of outlier fast food outlets within residential areas, confining outlets to regulated cluster, developing nutritional standards, and labelling cooked food and takeaways can all disincentivise unhealthy access and unhealthy consumption.

globalisation, the domestic food industry has become tightly aligned with the global economy. The type of food products available and their accessibility are governed more by economic considerations than by factors such as nutritional value, long-term health effects, and food production sustainability.[9] Cities have been characterised by their areas of so-called food deserts (areas with poor access to healthy food options) and so-called food swamps (areas with a greater provision of unhealthy, calorie-dense foods and drinks).[10,11] There is evidence of an association between the prevalence of chronic diseases[12] and dietary consumption patterns, access to food environments in urban neighbourhoods, in terms of categories of and access to unhealthy food environments.[13,14]

Sedentary lifestyles and consumption of energy-dense and processed food that is rich in fats and sugars both constitute major risk factors of type 2 diabetes.[15] Unhealthy diet has emerged as one of the primary causes in the development of chronic disease, particularly type 2 diabetes.[16] Exposure to the local food environment has been shown to be associated with dietary behaviour and the quality of dietary intake[17] and implicated in the prevalence and sociospatial distribution of obesity,[18] gestational diabetes,[19,20] and type 2 diabetes.[15] Previous studies[21] have relied on geographical information system (GIS)-based measures of accessibility to food environments, expressed in terms of objective measures of proximity and density and as the proportion of unhealthy food environments to the total food supply environments within geographically defined neighbourhoods. Unhealthy food-supply environments generally comprise retail outlets that sell high-calorie and processed foods, soft drinks, alcohol, and other beverages—ie, products rich in saturated fat, salt, sugar, and other ingredients that are harmful to health. Associations between the accessibility of unfavourable food environments and higher prevalence of type 2 diabetes[22–24] and between a healthy food environment and lower risk of type 2 diabetes have previously been established.[25,26]

Despite the evidence generated thus far, some ambiguity remains in the association between exposure to food environments and type 2 diabetes. Evidence is especially

scarce with respect to access to food outlets selling food that can be instantly consumed. Previous studies[27] have been ecological in design, assessing the food outlets within aggregate census-defined neighbourhoods (by use of zip codes, census tracts, or postcodes) and the census bureau population; these studies are limited in their ability to accurately measure exposure, given that a census-defined neighbourhood might not accurately represent an individual's activity space or functional neighbourhood. A few studies have reported null or counterintuitive findings.[28,29] Most studies have been small in population size and geographical scale with relatively homogeneous populations and exposure characteristics, which has limited their statistical power and generalisability, and many were based on self-reported, survey-based measures. Further, most of the studies evaluating the association of food outlet accessibility with chronic disease risks have been done in the USA, and there have been no studies that have examined the associations between accessibility to ready-to-eat food environments selling food that can be instantly consumed and type 2 diabetes at a national scale in the UK.

We aimed to examine the associations between exposure to ready-to-eat food environments and the prevalence of type 2 diabetes in a diverse nationwide population sample, to establish a robust evidence base. We also aimed to examine the effect modification of sex, income, body-mass index (BMI), and location of UK Biobank collection centres.

Methods

Study design and participants

In this cross-sectional observational study, we used data on male and female participants from the baseline phase of the UK Biobank cohort. The UK Biobank is a population-based cohort aged 37–73 years (99·5% of whom are aged between 40 and 69 years) that was set up to study the lifestyle and environmental and genetic determinants of a range of adult chronic diseases. In 2006–10, around 9·2 million invitation letters were sent out to recruit potential participants from the UK National Health Service Register, from which 502 635 participants were recruited.[30]

The participants resided within 25 miles of one of the 22 collection centres across the UK. At baseline, all participants provided electronically signed informed consent; gave verbal interviews; had their eye, physical, and anthropometric measurements taken; and provided blood, urine, and salivary samples. Participants also answered touch-screen questionnaires about a range of sociodemographic, occupational, lifestyle, psychosocial, environmental, and health-related variables. Physical activity measures were determined from a self-reported short-form International Physical Activity Questionnaire. We modelled individual-level exposures to the built environment within functional street neighbourhoods (in the form of residential street catchment areas), and we linked these exposures to all members of the cohort. The cohort protocol, scientific rationale, and study design is described on the UK Biobank website.

For the **UK Biobank protocol** see http://www.ukbiobank.ac.uk/wp-content/uploads/2011/11/UK-Biobank-Protocol.pdf

Procedures

Metrics of exposure to ready-to-eat food environments were derived from the UK Biobank Urban Morphometric Platform (UKBUMP). UKBUMP is a spatial database of built environment metrics, comprising objectively measured indices of density and accessibility of health-affecting land uses and services (including food environments), street network accessibility, and the physical environment (such as greenness and terrain) that were developed to identify environmental determinants of chronic disease.[31] These measures were derived from GIS-based spatial and network analyses on national level spatial databases, including the Ordnance Survey GB (OSGB) dataset.

Given that consumption patterns increasingly reflect a greater reliance on food that can be instantly consumed, we focused on the association of access to ready-to-eat food environments that sell food to be consumed instantly with the prevalence of type 2 diabetes. These environments include fast food or takeaway outlets, pubs and bars, and restaurants and cafeterias. The locations of the homes of UK Biobank participants were geocoded and we subsequently delineated 1-km street catchment areas with the OSGB Integrated Transport Network street centreline data. Ready-to-eat food environments were extracted from the OSGB AddressBase Premium database and were measured as the density (units/km²) of pubs or bars (OS land use code CR06), restaurants or cafeterias (CR07), and fast food outlets or hot and cold takeaways (CR10) within the defined 1-km street catchment area of the geocoded residence of the participant. Previous research[32] that examined the association between food environment and chronic disease have also used 1-km neighbourhood areas. We used 1-km street catchment areas to act as functional neighbourhoods that corresponded to 10 min of walking at moderate pace. As a measure of destination accessibility, street network distances from the geocoded home dwelling location to the nearest ready-to-eat food destinations (pubs and bars, restaurants and cafeterias, and fast food or hot and cold takeaway outlets) were also measured in ArcGIS Network Analyst version 10.3.

In addition to single-type ready-to-eat food environment measures, a composite metric for density of ready-to-eat food environments within the 1-km catchment area was also developed from the individual food destinations, and expressed as the total of the density of pubs and bars, restaurants and cafeterias, and fast food or takeaway outlets within this 1-km radius.

The densities of petrol filling stations (OS land use code CR05) and train and bus stations or terminals (CT08) were measured as a proxy for potential exposures to other food-related retail outlets, given the high likelihood of clustering around them.

Outcomes

The primary outcome was the risk of type 2 diabetes and was defined as in a previous study.[33] All participants who answered "yes" in the self-reported questionnaire to the question "Has a doctor ever told you that you have diabetes?" were considered to have diabetes. To exclude participants who are likely to have type 1 diabetes, participants taking insulin within the first year after diagnosis were excluded. Participants reported to have been diagnosed younger than 35 years were also excluded, to account for those with autoimmune or other monogenic forms of diabetes. Participants who did not report their age of diagnosis were also excluded from the study. Participants diagnosed with type 2 diabetes within 1 year of recruitment were also excluded because we were unable to determine their insulin usage and the consequent likelihood of these participants having a slow onset of type 1 diabetes.

As post-hoc analyses, we adjusted for individual-level covariates on the basis of a priori literature.[34] These covariates were sociodemographic variables (age, sex, ethnicity, highest educational qualification, total household income before tax, and employment status), lifestyle variables and comorbidities (smoking status, alcohol intake frequency, diet, BMI, vascular disease status, and parental diabetes), and activity-related variables and other exposures (television screen time and metabolic equivalent of task [MET]-h/week and a nearby location of petrol-filling stations or train or bus stations or terminals). The role of potential confounders in the associations between exposure to ready-to-eat food environments and type 2 diabetes, determined with a Directed Acyclic Graph,[35] is shown in figure 1.

Statistical analysis

We examined the associations between exposure to ready-to-eat food environments and type 2 diabetes by use of logistic regression models with robust SEs. The prevalence of type 2 diabetes was coded as one of two categories (case *vs* no case). Among the sociodemographic variables, age was expressed as a three-category variable (<50 years, 50–60 years, or ≥60 years). Ethnicity was coded as a four-category variable (white, Asian, black, and mixed or other). The highest educational qualification was expressed as a five-category variable (no qualification; O-levels, General Certificate of Secondary Education, Certificate of Secondary Education, AS-levels, or A-levels; National Vocational Qualification, Higher National Diploma, Higher National Certificate, or another professional qualification; or college or university degree). Household income was expressed as a four-category variable (<£18 000, £18 000–30 999, £31 000–51 999, or ≥£52 000), and employment status was coded as a three-category variable (employed; retired; and unemployed, home maker, or other). Among the lifestyle covariates, smoking status was coded as a three-category variable (never smoked, previous smoker, or current smoker), and alcohol intake frequency was coded as a four-category variable (never drinks or on special occasions, twice a week or less, three to four times a week, or daily or almost daily). Comorbidities included BMI as a three-category variable (<25 kg/m², 25–29·9 kg/m², or ≥30 kg/m²), and vascular disease status as a two-category variable (none *vs* yes). The prevalence of parental diabetes was expressed as a three-category variable (none, one parent, or both parents). The dietary variables comprised processed meat intake as a four-category variable (never, once a week, two to four times per week, or more than four times per week) and salad or raw vegetable intake as a five-category variable (none, one heaped tablespoon per day or less, two heaped tablespoons per day, three heaped tablespoons per day, or more than three heaped tablespoons per day). For the activity-related variables, television screen time was coded as a five-category variable (none to less than 1 h per day, 1 h per day, 2 h per day, 3 h per day, or more than 3 h per day). Physical activity was calculated as the sum of weekly walking, moderate physical activity, and vigorous physical activity components, expressed in MET-h/week.[36] Other exposures (which were used to indicate a proxy for supermarkets, retail outlets, and convenience stores that sold food) included the density of petrol-filling stations and train and bus stations and terminals within a buffer of 1 km in street distance from the home address, which was combined into a binary variable (absent *vs* present) in both cases.

Initial modelling comprised covariate-adjusted models that involved each single food environment variable (SFEV) models to test separate associations with the three categories of food environments—ie, pubs and bars, restaurants and cafes, and hot and cold takeaways. The models sequentially adjusted for sociodemographic variables (model 1), lifestyle variables and comorbidities (model 2), and activity-related and other covariates (model 3). On the basis of the existing distribution of exposures, each of the SFEVs was first divided into quintiles. The first quintile comprised participants with no exposures and acted as a reference category in our analyses. Given the very small sizes of the second quintile, it was combined with the third, thereby creating a four-category SFEV model (appendix).

See Online for appendix

We then re-ran the models with the composite food environment variable (CFEV), an additive index comprising densities of all three ready-to-eat food outlet types (pubs and bars, restaurants and cafes, and hot and cold takeaways). CFEV was first divided into quintiles. These were rearranged into a new five-category CFEV, in which the first category indicated no exposure, the second category was formed by adding together the original second quintile plus the non-zero exposures within the first quintile, whereas the third to fifth categories were the same as the third to fifth quintiles.

For a sample subset with non-zero CFEV, we further re-ran models with log-transformed CFEV and also tested

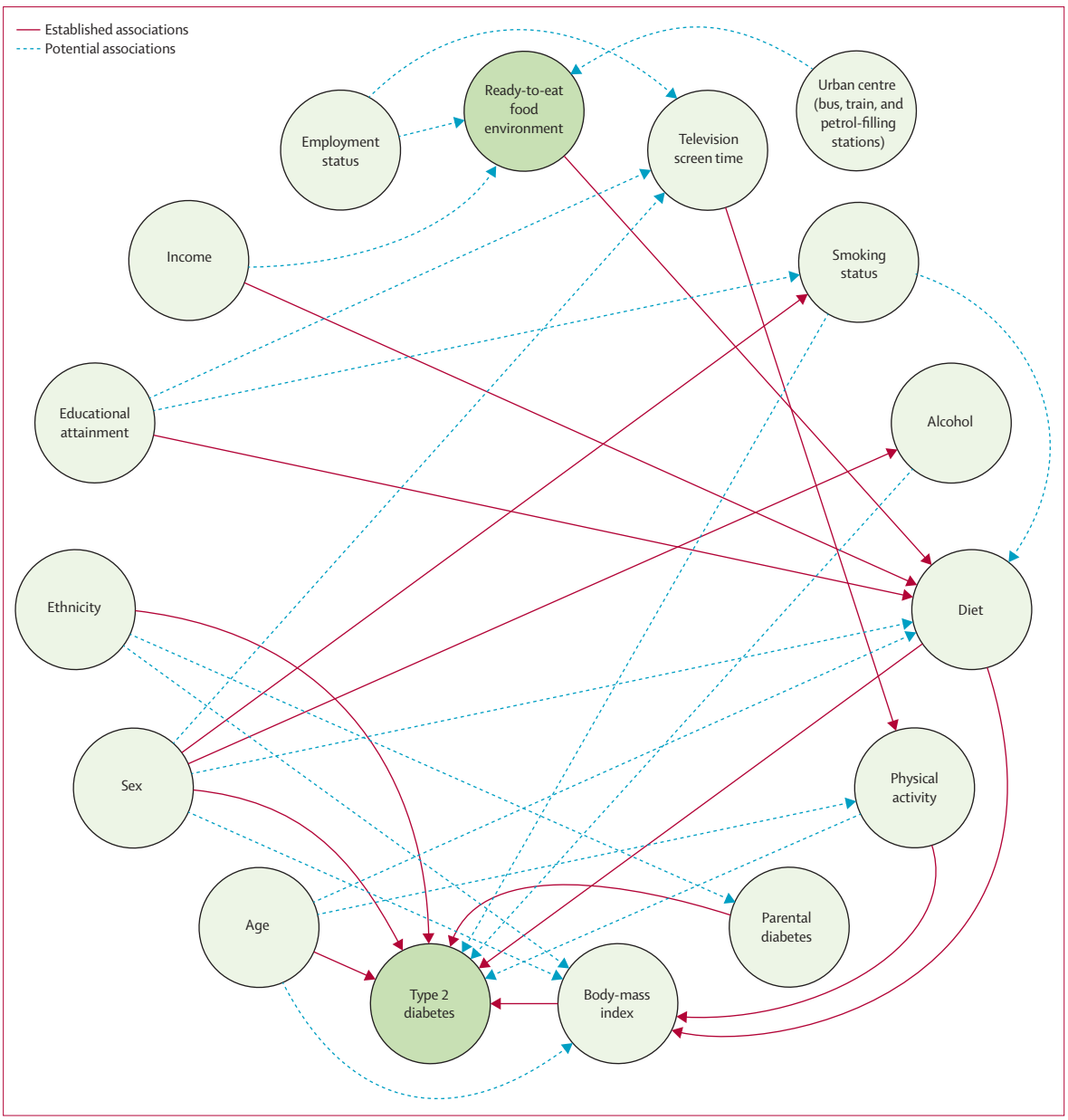


Figure 1: **Directed Acyclic Graph of the association between exposure to ready-to-eat food environments and prevalence of type 2 diabetes, including potential confounders**

for between-city and within-city effects. For this analysis, log-transformed composite food environment exposures assigned to all participants within a city were first averaged to estimate a city-mean exposure. We then subtracted the individual CFEV exposure assigned to each participant from their city-mean exposure to estimate the deviation of every participant from the city mean, and we included both the variables in our regression models, thereby disaggregating effects into between-cities and within-cities effects.[37] As an additional sensitivity analysis, we tested for potential non-linearity in the associations between CFEV and odds of type 2 diabetes. We used the log-transformed CFEV and ran restricted cubic spline models with three, four, five, and six Harrel's knots at equal intervals. We also designed negative control analyses to account for potential residual confounding and other biases that are found in most large-scale observational studies that involve diverse outcomes.[38,39] We posited weight at birth to be unrelated to the current neighbourhood food exposures; however, in type 2 diabetes, birthweight is correlated with individual sociodemographic variables. We re-ran our primary analyses with weight at birth (kg) as a continuous variable and low birthweight (<2·5 kg) as a binary negative control

	All participants (n=347 551)	Type 2 diabetes status	
		No type 2 diabetes (n=336 873)	Type 2 diabetes (n=10 678)
Age, years	56·0 (8·0)	55·9 (8·0)	60·6 (6·4)
Density of outlets (units/km²)			
Pubs and bars	2·4 (4·0)	2·4 (4·1)	2·5 (3·8)
Restaurants and cafeterias	3·5 (7·8)	3·5 (7·9)	3·5 (7·3)
Fast food outlets or takeaways	1·3 (2·6)	1·3 (2·6)	1·4 (2·7)
Composite ready-to-eat food environment	7·2 (12·5)	7·2 (12·5)	7·4 (11·5)
Street distance to food outlets, m			
Pubs and bars	943 (878·5)	944·0 (880·1)	909·8 (825·4)
Restaurants and cafeterias	1138·6 (1148·2)	1141·1 (1151·7)	1062·1 (1030·2)
Fast food outlets or takeaways	1886·5 (2177·2)	1891·5 (2184·0)	1731·0 (1943·3)
Density of petrol-filling stations	0·34 (0·47)	0·34 (0·47)	0·36 (0·48)
Density of train and bus stations or terminals	0·15 (0·36)	0·15 (0·36)	0·15 (0·36)
Physical activity, MET h/week*	45·9 (49·5)	46·1 (49·6)	38·4 (46·5)
Sex			
Female	187 366 (53·9%)	183 792 (54·6%)	3574 (33·5%)
Male	160 185 (46·1%)	153 081 (45·4%)	7104 (66·5%)
Ethnicity			
White	333 623 (96·0%)	323 990 (96·2%)	9633 (90·2%)
Asian	5245 (1·5%)	4643 (1·4%)	602 (5·6%)
Black	3844 (1·1%)	3609 (1·1%)	235 (2·2%)
Mixed or others	4839 (1·4%)	4631 (1·4%)	208 (1·9%)
Education			
None	46 605 (13·4%)	44 080 (13·1%)	2525 (23·6%)
College or university degree	126 238 (36·3%)	123 442 (36·6%)	2796 (26·2%)
O-levels, GCSEs, or CSEs	93 216 (26·8%)	90 577 (26·9%)	2639 (24·7%)
A-levels or AS-levels	41 468 (11·9%)	40 396 (12·0%)	1072 (10·0%)
NVQ, HND, HNC, or another professional qualification	40 024 (11·5%)	38 378 (11·4%)	1646 (15·4%)
Annual household income			
<£18 000	71 976 (20·7%)	68 190 (20·2%)	3786 (35·5%)
£18 000–30 999	87 490 (25·2%)	84 395 (25·1%)	3095 (29·0%)
£31 000–51 999	93 349 (26·9%)	91 127 (27·1%)	2222 (20·8%)
>£52 000	94 736 (27·3%)	93 161 (27·7%)	1575 (14·7%)
Employment status			
Employed	215 598 (62·0%)	211 219 (62·7%)	4379 (41·0%)
Retired	107 286 (30·9%)	102 103 (30·3%)	5183 (48·5%)
Unemployed, home maker, or other	24 667 (7·1%)	23 551 (7·0%)	1116 (10·5%)
Smoking status			
Non-smoker	193 288 (55·6%)	188 642 (56·0%)	4646 (43·5%)
Previous smoker	120 025 (34·5%)	115 062 (34·2%)	4963 (46·5%)
Current smoker	34 238 (9·9%)	33 169 (9·8%)	1069 (10·0%)
Frequency of alcohol intake			
Never or special occasions	59 848 (17·2%)	56 498 (16·8 %)	3350 (31·4%)
≤2 times a week	128 551 (37·0%)	124 708 (37·0%)	3843 (36%)
3–4 times a week	84 834 (24·4%)	83 067 (24·7%)	1767 (16·5%)
Daily or almost daily	74 318 (21·4%)	72 600 (21·6%)	1718 (16·1%)

(Table 1 continues on next page)

outcome (yes *vs* no). If our primary analyses of association between CFEV and type 2 diabetes were not residually confounded, we would expect no association between CFEV and weight at birth and odds of low birth-weight.

As a secondary analysis, we also examined the associations between quintiles of street distance to the nearest single ready-to-eat food outlet and type 2 diabetes. We also examined whether the reported associations between density of ready-to-eat food outlets and type 2 diabetes varied by sex, income, BMI, and UK Biobank collection centre location by introducing interaction terms in the adjusted models.

All spatial analyses were done with GIS software ArcGIS version 10.3 and statistical analysis was done with Stata version 14 and R statistical package.

Role of the funding source

UK Biobank, a funder of this study, was involved in data collection. The funders of the study had no role in study design, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

Complete data on the prevalence of type 2 diabetes and food environment exposure were available for 493 821 (98·2%) participants of 502 635 participants who had been enrolled in the UK Biobank cohort between March 13, 2006, and Oct 1, 2010. After excluding participants with missing data on sociodemographic variables (80 310 [16·0%] participants); lifestyle, diet, and co-morbidities (53 471 [10·6%] participants); and food-related built environment (12 489 [2·5%] participants), the sample was restricted to 347 551 (69·1%) participants. We therefore assessed 347 551 participants from 21 UK Biobank assessment centres for our primary outcome. The baseline characteristics of this sample remained representative of the UK Biobank cohort (appendix). Physical activity data across all three activity components were available for a subset of 285 990 participants, and television screen time was available for 346 462 participants. The mean age at baseline of the participants included in the analysis was 56·0 years (SD 8·0) and 187 366 (53·9%) were female (table 1). 10 678 (3·1%) participants of those included in the analysis of the primary endpoint had type 2 diabetes, but 5973 (4·1%) of the 146 270 participants excluded from our analyses because of missing data had type 2 diabetes. 69 208 (19·9%) participants included in the analysis were exposed to the highest category of composite density of ready-to-eat food environment (appendix), compared with 27 027 (20·2%) participants in the excluded subsample. Within the 1-km street catchment areas of the participants' living locations, the mean density of pubs and bars was 2·4 units/km² (SD 4·0), the mean density of restaurants and cafeterias was 3·5 units/km² (7·8), and the mean density of hot and cold takeaways was

1·3 units/km² (2·6); whereas the mean street distances to these food environments were 943·0 m (878·5) to pubs and bars, 1138·6 m (1148·2) to restaurants and cafeterias, and 1886·5 m (2177·2) to hot and cold takeaways.

The results of the logistic regression models used to examine the associations between SFEVs or CFEV within 1-km residential street catchment areas and type 2 diabetes are shown in tables 2 and 3. Individual models of SFEV showed increased odds of type 2 diabetes across the higher categories of density of restaurants and cafeterias (table 2). Compared with participants with no exposure to restaurants and cafeterias, those in the highest exposure category (>4·76 units/km²) reported significantly higher odds of type 2 diabetes (odds ratio [OR] 1·129, 95% CI 1·05–1·21; p=0·0007 in the fully adjusted model). No significant association was observed among participants in the category with the highest exposure (>2·15 units/km²) to hot and cold takeaways (1·062, 1·00–1·13; p=0·0566); however, exposure to hot and cold takeaways at the second highest category of density (0·75–2·15 units/km²) was associated with increased odds of type 2 diabetes (1·076, 1·01–1·14; p=0·0171). For our fully-adjusted model of CFEV, participants in the highest exposure category (>10·70 units/km²) reported higher odds of type 2 diabetes than those in the reference category who had no exposure to ready-to-eat food outlets (1·112, 1·02–1·21; p=0·0134; table 3). The effect sizes were attenuated with additional adjustments for confounding across models 1–3.

Rerunning the models with log-transformed CFEV for a subset of participants with non-zero exposures produced similar effects, showing higher odds of type 2 diabetes in the fully adjusted analysis (OR 1·031, 1·00–1·06; p=0·0237; appendix). In our models with disaggregated effects, the within-city effects remained significant (1·028, 1·00–1·06; p=0·0439), but the between-cities effects were not (1·053, 0·98–1·14; p=0·1831). Sensitivity analyses to test non-linearity in the associations were not significant, returning p values of 0·6096 for three Harrel's knots, 0·1120 for four Harrel's knots, 0·1879 for five Harrel's knots, and 0·2007 for six Harrel's knots.

The results of the negative control analyses that assessed the association of birthweight and odds of low birthweight with food environment variables, after adjusting for risk factors and covariates, produced the expected null effects for the models with both SFEVs and CFEV. The effect sizes in our continuous models remained very small (appendix).

The results of our secondary analysis indicated a protective effect of increasing street distance to nearest ready-to-eat food environment on odds of type 2 diabetes that remained consistent for models with each SFEV (table 4). Participants in the highest quintile of street distance to nearest ready-to-eat food outlet (ie, those further away) had lower odds of type 2 diabetes (for restaurants and cafeterias, OR 0·842, 95% CI 0·78–0·91; p<0·0001; and for hot and cold takeaways, 0·913, 0·85–0·98; p=0·0173) than those in the lowest quintile. However, the associations between distance to nearest pubs and bars and the odds of type 2 diabetes were not significant.

We also did a causal mediation analysis to explore whether the association between our composite ready-to-eat environment metric and type 2 diabetes was mediated by the presence of obesity after adjusting for all other factors by use of the mediation package in R.[40] This analysis involved fitting a mediator model in which obesity was modelled as a function of the composite ready-to-eat environment metric (treatment) and all other pretreatment covariates and then fitting the outcome model with odds of type 2 diabetes as a function of treatment, mediator, and pretreatment covariates. 10·5% of the total effects were attributed to the mediation effects of obesity (appendix).

We also tested the effects of additional adjustments for sugar intake and food weight (ie, the estimated intake based on food and beverage consumption the previous day,

	All participants (n=347 551)	Type 2 diabetes status	
		No type 2 diabetes (n=336 873)	Type 2 diabetes (n=10 678)
(Continued from previous page)			
Processed meat intake			
None	31 870 (9·2%)	31 207 (9·3%)	663 (6·2%)
Once a week	207 973 (59·8%)	202 175 (60·0%)	5798 (54·3%)
2–4 times a week	94 252 (27·1%)	90 591 (26·9%)	3661 (34·3%)
>4 times a week	13 456 (3·9%)	12 900 (3·8%)	556 (5·2%)
Salad and raw vegetable intake			
None	31 587 (9·1%)	30 180 (9·0%)	1407 (13·2%)
≤1 tablespoon	123 567 (35·6%)	119 931 (35·6%)	3636 (34·1%)
2 tablespoons	85 270 (24·5%)	82 697 (24·5%)	2573 (24·1%)
≥3 tablespoons	107 127 (30·8%)	104 065 (30·9%)	3062 (28·7%)
Television screen time†			
<1 h	28 739 (8·3%)	28 335 (8·5%)	404 (3·8%)
1 h	47 032 (13·6%)	46 244 (13·9%)	788 (7·4%)
2 h	96 932 (28·0%)	94 780 (28·4%)	2152 (20·3%)
3 h	81 273 (23·5%)	78 769 (23·6%)	2504 (23·6%)
≥4 h	92 486 (26·7%)	87 725 (26·3%)	4761 (44·9%)
Body-mass index			
Normal weight	118 411 (34·1%)	117 486 (34·9%)	925 (8·7%)
Overweight	148 538 (42·7%)	144 910 (43·0%)	3628 (34·0%)
Obese	80 602 (23·2%)	74 477 (22·1%)	6125 (57·4%)
Vascular problems			
None	251 333 (72·3%)	248 413 (73·7%)	2920 (27·3%)
Yes	96 218 (27·7%)	88 460 (26·3%)	7758 (72·7%)
Parental diabetes			
None	285 401 (82·1%)	278 904 (82·8%)	6497 (60·8%)
One parent	57 849 (16·6%)	54 202 (16·1%)	3647 (34·2%)
Both parents	4301 (1·2%)	3767 (1·1%)	534 (5·0%)

Data are mean (SD) or number (%). Density of food environments was measured within a 1-km street catchment area. MET=metabolic equivalent of task. GCSE=General Certificate of Secondary Education. CSE=Certificate of Secondary Education. NVQ=National Vocational Qualification. HND=Higher National Diploma. HNC=Higher National Certificate. *For 285 990 participants. †For 346 462 participants.

Table 1: **Characteristics of the participants assessed in the primary analysis**

	Model 1* (n=347 551)	Model 2† (n=347 551)	Model 3‡ (n=285 990)
Pubs and bars			
0 (ref)	..	..	..
0·40–1·87	1·039 (0·99–1·09); p=0·1425	1·045 (0·99–1·10); p=0·1064	1·025 (0·96–1·09); p=0·4201
1·87–3·67	1·049 (0·99–1·11); p=0·0985	1·034 (0·97–1·10); p=0·2694	1·026 (0·96–1·10); p=0·4495
>3·67	1·066 (1·01–1·13); p=0·0271	1·063 (1·00–1·13); p=0·0452	1·018 (0·95–1·09); p=0·6162
Restaurants and cafeterias			
0 (ref)	..	..	..
0·45–1·81	1·069 (1·01–1·13); p=0·0147	1·051 (0·99–1·11); p=0·0786	1·092 (1·02–1·16); p=0·0065
1·81–4·76	1·099 (1·04–1·16); p=0·0006	1·076 (1·02–1·14); p=0·0108	1·075 (1·01–1·15); p=0·0311
>4·76	1·117 (1·06–1·18); p=0·0001	1·147 (1·08–1·21); p<0·0001	1·129 (1·05–1·21); p=0·0007
Hot and cold takeaways			
0 (ref)	..	..	..
0·30–0·75	1·085 (0·99–1·19); p=0·0697	1·057 (0·96–1·16); p=0·245	1·005 (0·90–1·12); p=0·9216
0·75–2·15	1·104 (1·05–1·16); p=0·0001	1·083 (1·03–1·14); p=0·0031	1·076 (1·01–1·14); p=0·0171
>2·15	1·102 (1·05–1·16); p=0·0002	1·073 (1·02–1·13); p=0·0102	1·062 (1·00–1·13); p=0·0566

Data are odds ratio (95% CI); p value. Density of food environments is presented as units/km² within a 1-km street catchment area. *Adjusted for age, sex, ethnicity, highest educational qualification, employment status, and income. †Also adjusted for smoking status, frequency of alcohol intake, processed meat intake, salad and raw vegetable intake, body-mass index, vascular disease status, and parental diabetes. ‡The fully adjusted model after also controlling for activity-related and other variables: television screen time, presence of bus or train stations, presence of petrol-filling stations, and physical activity (MET-h/week).

Table 2: **Models with single food environment variables of the associations between the density of different categories of retail food outlets and the odds of type 2 diabetes**

	Model 1* (n=347 551)	Model 2† (n=347 551)	Model 3‡ (n=285 990)
0 (ref)	..	..	..
0·40–2·40	1·014 (0·95–1·08); p=0·6732	0·997 (0·93–1·07); p=0·9289	0·995 (0·92–1·07); p=0·9003
2·40–4·98	1·101 (1·03–1·18); p=0·0043	1·060 (0·99–1·14); p=0·0991	1·045 (0·97–1·13); p=0·2729
4·98–10·70	1·142 (1·07–1·22); p=0·0001	1·094 (1·02–1·17); p=0·0112	1·057 (0·98–1·15); p=0·1765
>10·70	1·139 (1·07–1·22); p=0·0001	1·135 (1·06–1·22); p=0·0004	1·112 (1·02–1·21); p=0·0134

Data are odds ratio (95% CI); p value. The composite density of ready-to-eat food environments is the density of pubs and bars, restaurants and cafeterias, and hot and cold takeaways, presented as units per km² within a 1-km street catchment area. *Adjusted for age, sex, ethnicity, highest educational qualification, employment status, and income. †Also adjusted for smoking status, frequency of alcohol intake, processed meat intake, salad and raw vegetable intake, body-mass index, vascular disease status, and parental diabetes. ‡The fully adjusted model after also controlling for activity-related and other variables: television screen time, presence of bus or train stations, presence of petrol-filling stations, and physical activity (MET-h/week).

Table 3: **Associations between the composite density of ready-to-eat food environments and type 2 diabetes**

excluding any supplements) in a subsample of participants (n=135 073) for whom these data were available from the 24-h diet recall questionnaire (appendix). The effects of density of ready-to-eat food environments remained consistent; participants in the highest category of composite density of ready-to-eat food environments (>10·70 units/km²) reported higher odds of type 2 diabetes (OR 1·148, 95% CI 1·00–1·31; p=0·0450) compared with those with no exposure.

The results of subgroup level analyses are shown in figure 2 and the appendix. Effect sizes were bigger in the higher exposure category among women, those earning £18 000–32 000, and those in the overweight category (as determined by BMI). There was no significant interaction by sex (p=0·0640) or income (p=0·7439), but BMI was significantly associated with effect size (p=0·0329). The effect sizes were comparatively higher in the highest exposure category among participants of the UK Biobank collection centres of Manchester, Bury, Birmingham, Nottingham, and Wales; however, overall, there was no significant interaction ($p_{interaction}$=0·3032).

Discussion

In a large and diverse population cohort, we have shown that exposure to ready-to-eat food environments was associated with higher odds of type 2 diabetes. These results remained robust after adjustments for risk factors and covariates. Adjustments for activity-related outcomes, including MET-h/week of physical activity, slightly attenuated the effect estimates. To our knowledge, this is the first large-scale analysis of the association between exposure to ready-to-eat food environments that sell food for instant consumption and the risk of type 2 diabetes in the UK.

We showed an 11% higher odds of type 2 diabetes among participants in the category with the highest exposure to ready-to-eat food within a 1-km street catchment area versus those with null exposure, after adjustments for all other risk factors and covariates. Our findings corroborate previous findings on the topic. A UK study[24] that used primary care data regarding 10 461 participants from three diabetes screening studies reported significantly increased odds of type 2 diabetes with a higher number of fast food outlets within a 500-m circular area (OR 1·02, 95% CI 1·00–1·04; p=0·02). However, this was an ecological study in which participants' areas were only geocoded up to a postcode-level. The Jackson Heart study,[22] an African-American population cohort of 3670 participants in the USA found that the density of unhealthy food stores within a 1-mile area of participants' residential addresses was associated with a 34% higher incidence of type 2 diabetes (1·34, 1·12–1·60). A Swedish study[23] based on 2·8 million adults from several national registries reported that the net negative food environment (measured as the ratio of health-harming food outlets to the food outlets per km²) was associated with higher odds of type 2 diabetes (2·11, 1·57–2·82), with relatively higher effect estimates in participants who moved to neighbourhoods with greater relative access to health-harming food outlets. However, the Swedish study was an aggregate-level analysis, since the participants' dwelling could only be geocoded at the level of small geographical units (that averaged 1000 participants per unit) and failed to adjust for key confounders, such as physical activity. Another county-level ecological study[29] that used self-reported data in South Carolina, USA, reported no association between density of food outlets and county-level type 2 diabetes, subsequent to all adjustments.

Our composite exposure measure of ready-to-eat food environments was validated from the results of fully

adjusted models run with SFEVs (table 2), which reported harmful effects of the highest category of exposure to restaurants and cafeterias. Models that used density of SFEVs reported higher odds of type 2 diabetes across higher categories of SFEVs of restaurants and cafeterias. Models that evaluated the association of street distance to the nearest food outlet with prevalence of type 2 diabetes found a 7–16% lower odds of type 2 diabetes among participants in the quintile representing the farthest distance versus those in the closest quintile. Previous studies[32,41] have consistently reported similar protective effects upon obesity with distance decay.

Our findings might indicate an unhealthy access–unhealthy consumption mechanism. Previous research[42] has established positive associations between access to food environments and frequency of dining. Another study[43] found an association between mean daily energy intake and frequency of consumption of out-of-home meals, reporting a greater energy intake of 75–104 kcal/day among participants who ate meals out at least weekly compared with those who rarely ate out. Higher odds of type 2 diabetes among populations with greater access to ready-to-eat food environments might be a result of consumption behaviour, although we did not have the data on eating behaviour to confirm whether such behaviour is supply-side induced.

Our models that explored interaction effects were significant only in the case of BMI; we found significantly greater effects across higher categories of composite ready-to-eat food exposure in overweight participants. This finding could be attributed to the fact that being overweight or obese is a risk factor for type 2 diabetes. The finding that the effect estimates were higher in female participants than male participants could indicate that women have a greater usage of residential neighbourhoods and a higher likelihood of being affected by ready-to-eat food environments within them.

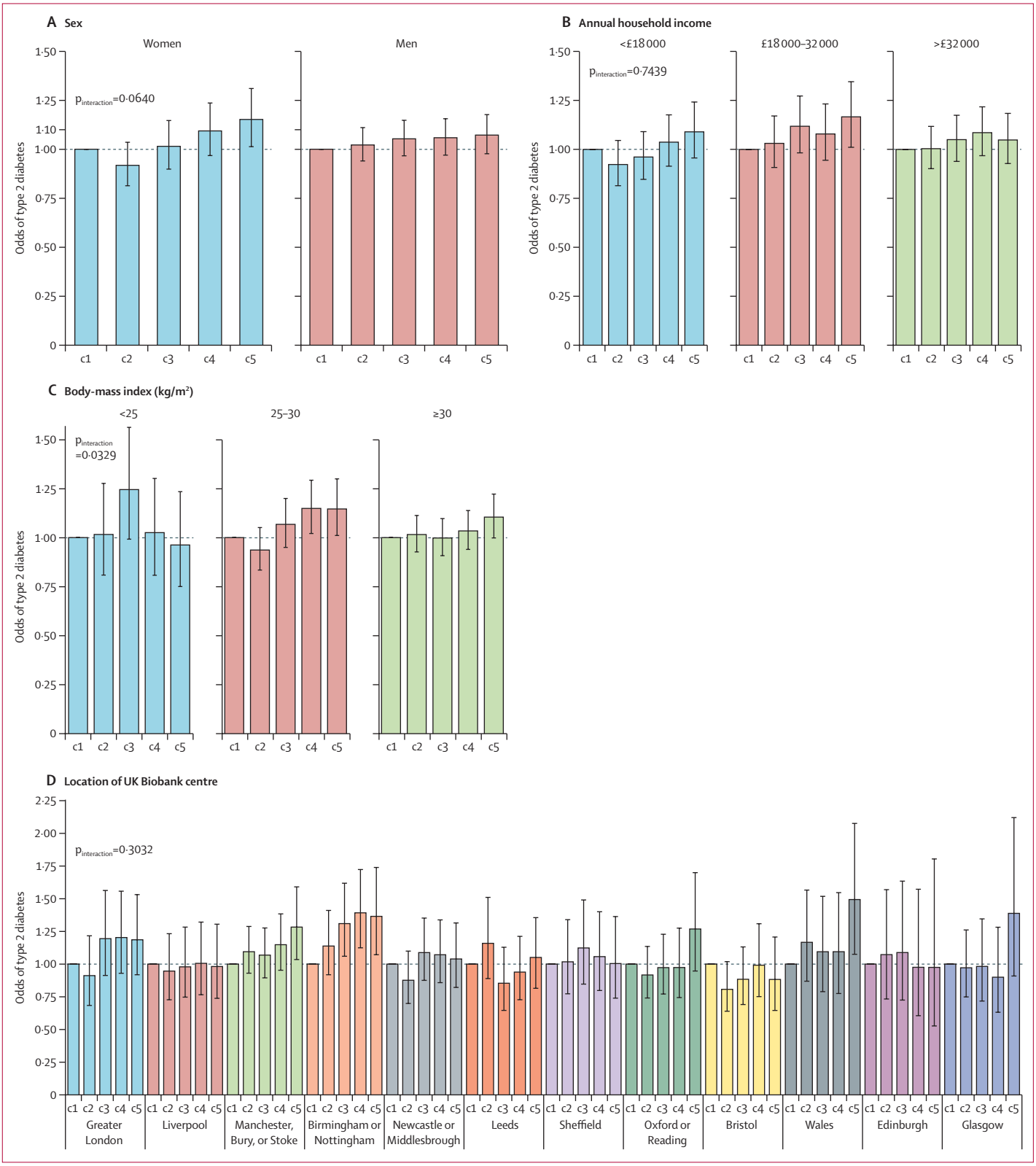
Our study has several strengths. The study relied on a very large and well characterised cohort that represents participants from 21 major cities of the UK with considerable diversity in their population-level characteristics and food environment exposures. Large-scale analyses gave a high level of statistical power to detect effects. In contrast to previous studies, ready-to-eat food environment was measured in a way that included not only fast food outlets but also pubs and bars and restaurants and cafeterias, which also supply food and drinks for instant consumption. Given the shifts in our food consumption behaviour, the health aspects of these neighbourhood food environments are of considerable significance because of their potential to supply food of high calorific value that is rich in saturated fat, salt, sugar, and other components that are detrimental to health. Our results remain consistent across the models of SFEVs and CFEV, and in our continuous models that used log-transformed CFEV. Our measures of density within 1-km individual street catchment areas (which were expressed as outlet units/km²) are more useful than just counts, especially in informing policies aimed at provision of food outlets per unit area of urban space and, thus, optimising access to them (for example, restricting access to health-harming food environments). Density per km² is a measure of opportunity to purchase ready-to-eat food and drink. We were also able to use street distances to the nearest food outlet, another measure of opportunity, as a secondary variable for further sensitivity analyses, to validate our primary findings. In contrast to previous studies,[44] we have modelled outlets within functional catchment areas of geocoded dwellings, and thus we have enhanced the accuracy of exposure assessment (compared with exposures measured within predefined administrative neighbourhoods). Similarly, the application of street network catchment areas (as opposed to Euclidean buffers) enhances the accuracy of exposure assessment.[45] We could measure the association between street distance to the nearest food environment and odds of type 2 diabetes and we found protective trends of increasing distance to restaurants and cafeterias and hot and cold takeaways from dwelling location (representing an increasing opportunity to purchase).

Among the study limitations, our study is an observational study of cross-sectional design, which presents the typical difficulties for causal inference. It is likely that

	Food outlet type
Pubs and bars	
Quintile 1 (0–345·5)	Ref
Quintile 2 (345·5–568·0)	0·953 (0·89–1·02); p=0·1912
Quintile 3 (568·0–839·2)	0·946 (0·88–1·02); p=0·1387
Quintile 4 (839·2–1319·4)	0·932 (0·87–1·00); p=0·0613
Quintile 5 (>1319·4)	0·939 (0·87–1·01); p=0·1032
Restaurants and cafeterias	
Quintile 1 (0–400·8)	Ref
Quintile 2 (400·8–666·7)	0·902 (0·84–0·97); p=0·0052
Quintile 3 (666·7–1002·6)	0·891 (0·83–0·96); p=0·0020
Quintile 4 (1002·6–1576·0)	0·867 (0·80–0·93); p=0·0002
Quintile 5 (>1576·0)	0·842 (0·78–0·91); p<0·0001
Hot and cold takeaways	
Quintile 1 (0–527·3)	Ref
Quintile 2 (527·3–903·2)	0·918 (0·85–0·99); p=0·0200
Quintile 3 (903·2–1434·4)	0·897 (0·83–0·97); p=0·0038
Quintile 4 (1434·4–2640·0)	0·929 (0·86–1·00); p=0·0481
Quintile 5 (>2640·0)	0·913 (0·85–0·98); p=0·0173

Data are odds ratio (95% CI); p value, and are fully adjusted effect estimates. These estimates are adjusted for age, sex, ethnicity, highest educational qualification, employment status and income, smoking status, frequency of alcohol intake, processed meat intake, salad and raw vegetable intake, body-mass index, vascular disease status, parental diabetes, television screen time, presence of bus or train stations, presence of petrol-filling stations, and physical activity (MET-h/week). Street distance is presented in the relevant quintile (m).

Table 4: **Sensitivity analysis of the association between the street distance to the nearest ready-to-eat food environment from the residence of UK Biobank participants and the odds of type 2 diabetes**


A Sex
Women
Men
p interaction=0·0640
B Annual household income
<£18 000
£18 000–32 000
>£32 000
p interaction=0·7439
C Body-mass index (kg/m²)
<25
25–30
≥30
p interaction =0·0329
D Location of UK Biobank centre
p interaction=0·3032
Odds of type 2 diabetes
1·50
1·25
1·10
1·00
0·75
0·50
0·25
0
2·25
2·00
1·75
c1 c2 c3 c4 c5
Greater London
Liverpool
Manchester, Bury, or Stoke
Birmingham or Nottingham
Newcastle or Middlesbrough
Leeds
Sheffield
Oxford or Reading
Bristol
Wales
Edinburgh
Glasgow

participants with a preference for specific foods might have self-selected neighbourhoods with high densities of outlets that sell such food (demand-side ecological effect). Alternatively, food retailers might have established themselves in areas of high demand (supply-side ecological effect). The UK Biobank represents a stable population. In our analytic sample, 67·8% of the participants had resided in the current address for 10 years or more, and they had a mean duration of residence of 17·5 years (SD 11·6); we could find no modification of the effects by the duration of residence. Future studies should develop prospective health and exposure data for a more reliable evidence base. As with any study that uses secondary spatial data, error in the measurement of food environment could emerge from factors such as data incompleteness and temporal mismatch. The food environment metrics were derived from secondary spatial data sourced from OSGB. OSGB's food environment data were obtained from Royal Mail, local authorities, and other reliable private agencies and updated at periodic intervals. In our study, the food environment data were obtained during the end of the baseline phase of the UK Biobank, to avoid temporal mismatch. A second source of bias might emerge from potential exposure misclassification within the spatial dataset. A third possibility of bias emerges from the limitation of the food-related spatial data within the OSGB database that are available to this study. We were unable to account for the whole range of food environments; for example, our study did not adjust for variation in access to processed meals, sugary drinks, and alcohol served by supermarkets. We note that, as early as the late 1990s, supermarket supply in the UK was becoming saturated[46] and that fierce competition between the big suppliers has continued the trend. This trend means that, in urban areas of similar population density, access to a supermarket will be roughly constant. The use of density of petrol-filling stations and train and bus stations or terminals could, to a certain extent, act as a proxy for population density and, more generally, it represents urban hubs around which food-related retail and supermarkets tend to cluster. We also note that most large supermarkets invariably have petrol-filling stations adjacent to them. Our study is therefore a focused analysis of the association between accessibility to three distinct separate ready-to-eat food environments (pubs and bars, restaurants and cafeterias, and hot and cold takeaways that sell food for instant consumption) and type 2 diabetes, adjusting for petrol-filling stations and train and bus stations or terminals, which acted as a proxy for ambient availability of processed meals (such as those through supermarkets). Future studies might seek to adjust for a more holistic range of food outlets and for data on frequency of visits and heterogeneity in the type and nutritional value of food sold or served in each food outlet via detailed food audits, which the present national-level study could not do because of inadequate data. Beyond accessibility to retail food outlets, our study could not adjust for other factors that affect market behaviour, such as food pricing.

As with any observational data, the risk of residual confounding cannot be ruled out in our study. We could test for potential residual confounding by running negative control analyses of birthweight, which produced no effects, as expected. Finally, the UK Biobank study had a low response rate (5·5%) and is characterised by a healthy volunteer effect, as is common in any volunteer-based cohort study.[47] Overall, the UK Biobank participants are healthier than the general population and, hence, this cohort might not be the best suitable cohort for estimation of generalised type 2 diabetes prevalence. The overall prevalence of type 2 diabetes in this cohort is lower than the national average but compares well with any other large UK-based cohorts.[6] Nonetheless, given the large sample size and population-level and geographical diversity, this low response rate and healthy volunteer effect does not imply loss of generalisability in the reported findings.[48,49]

Given the rapid pace of urbanisation and globalisation and the associated changes in lifestyles and dietary preferences, the potential effect of access to ready-to-eat food environments that sell food for instant consumption on health requires special attention. Our study that assessed a very large, UK-wide cohort of substantial population-level and geographical diversity found that access to ready-to-eat food outlets was associated with higher odds of type 2 diabetes. The results have important public health implications and make the case for a shift from an exclusively corporate-driven economic model to a public health-driven health economic model of urban food access and consumption. This policy shift might require top-down interventions at a city scale, such as through formulating specific spatial policies to constrain access to unhealthy food environments that sell ready-to-eat food and to reduce their clustering around residential areas to potentially minimise unhealthy dietary consumption. Reducing the number of permits for outlets that sell calorie-dense food, as a policy, can further reduce the availability of unhealthy food. A specific policy suggested by our findings is to discourage the placement of outlier

Figure 2: **Association between categories of composite density of ready-to-eat food environment and type 2 diabetes**
Data are density within 1-km street catchment areas by (A) sex, (B) annual household income, (C) body-mass index, and (D) UK Biobank centre location. All models are adjusted for age, sex, ethnicity, highest educational qualification, employment status and income, smoking status, frequency of alcohol intake, processed meat intake, salad and raw vegetable intake, body-mass index, vascular disease status, parental diabetes, television screen time, presence of bus or train stations, and presence of petrol-filling stations. p values are for the interactions between categories of composite density of ready-to-eat food environments and population subgroups stratified by sex, income, body-mass index, and UK Biobank centre location. c1=0 (ref). c2=0·40–2·40 units/km². c3=2·40–4·98 units/km². c4=4·98–10·70 units/km². c5=more than 10·70 units/km².

fast food outlets within residential areas via licensing or planning regulations. Fast food outlets tend to naturally cluster for economies of scale in attracting customers, especially customers who drive. However, there are still many independent outlets scattered through the UK's residential neighbourhoods. Confining outlets to regulated clusters is likely to reduce proximity-driven consumption. Confining outlets could also increase food quality through the effects of competition. There is an argument for specific nutritional standards and labelling for the food industry, not only for processed food items but also for ready-to-eat meals, fast food takeaways, and beverages to be consumed instantly and mechanisms to ensure compliance through regular auditing. Overall, national-level policies must encourage and incentivise the food industry to transform menus from unhealthy to healthy. By contrast, specific public health interventions at the primary care level could aim to disincentivise unhealthy access and unhealthy consumption in ready-to-eat food environments, especially among vulnerable individuals, including those who are obese and who have type 2 diabetes and cardiometabolic diseases. Additional long-term longitudinal studies are needed to effectively guide such interventions.

Contributors

CS, CW, and JG conceived and designed the study. CS developed the food exposure metrics, did the statistical analysis, and drafted the report. CS, CW, and JG contributed to redrafting and interpretations. All the authors read and approved the final manuscript.

Declaration of interests

We declare no competing interests.

Acknowledgments

The research was done with the UK Biobank resource as a part of the approved Research Application (no. 11730.) The authors thank the Ordnance Survey GB, the UK National Mapping Agency, and MIMAS (University of Manchester) for providing access to its UK-wide spatial data for use in this study. The study was funded by the University of Hong Kong's URC Research Assistant Professorship Grant; UK Biobank seed grant; and UK Economic & Social Research Council's Transformative Research grant (ES/L003201/1).

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