Association between adiposity outcomes and residential density: a full-data, cross-sectional analysis of 419 562 UK Biobank adult participants

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Summary

Background Obesity is a major health issue and an important public health target for urban design. However, the evidence for identifying the optimum residential density in relation to obesity has been far from compelling. We examined the association of obesity with residential density in a large and diverse population sample drawn from the UK Biobank to identify healthy-weight-sustaining density environments.

Methods For this full-data, cross-sectional analysis, we used UK Biobank data for adult men and women aged 37–73 years from 22 cities across the UK. Baseline examinations were done between 2006 and 2010. Residential unit density was objectively assessed within a 1 km street catchment of a participant's residence. Other activity-influencing built environment factors were measured in terms of density of retail, public transport, and street-level movement density, which were modelled from network analyses of through movement of street links within the defined catchment. We regressed adiposity indicators of body-mass index (BMI; kg/m²), waist circumference (cm), whole body fat (kg), and obesity (WHO criteria of BMI ≥30 kg/m²) on residential density (units per km²), adjusting for activity-influencing built environment factors and individual covariates. We also investigated effect modification by age, sex, employment status, and physical activity. We used a series of linear continuous and logistic regression models and non-linear restricted cubic spline models as appropriate.

Findings Of 502 649 adults in the prospective cohort, 419 562 (83·5%) participants across 22 UK Biobank assessment centres met baseline data requirements and were included in the analytic sample. The fitted restricted cubic spline adiposity-residential density dose–response curve identified a turning point at a residential density of 1800 residential units per km². Below a residential density of 1800 units per km², an increment of 1000 units per km² was positively related with adiposity, being associated with higher BMI (β 0·19 kg/m², 95% CI 0·14 to 0·24), waist circumference (β 0·41 cm, 0·28 to 0·54), and whole body fat (β 0·40 kg, 0·30 to 0·50), and with increased odds of obesity (odds ratio [OR] 1·10, 1·07 to 1·14). Beyond 1800 units per km², residential density had a protective effect on adiposity and was associated with lower BMI (β –0·22 kg/m², –0·25 to –0·20), waist circumference (β –0·54 cm, –0·61 to –0·48), and whole body fat (β –0·38 kg, –0·43 to –0·33), and with decreased odds of obesity (OR 0·91, 0·90 to 0·93). Subgroup analyses identified more pronounced protective effects of residential density among individuals who were younger, female, in employment, and accumulating higher levels of physical activity, except in the case of whole body fat, for which the protective effects were stronger in men.

Interpretation Housing-level policy related to the optimisation of healthy density in cities might be a potential upstream-level public health intervention towards the minimisation and offsetting of obesity; however, further research based on accumulated prospective data is necessary for evidencing specific pathways. The findings might mean that governments, such as the UK Government, who are attempting to prevent suburban densification by, for example, prohibiting the subdivision of single lot housing and the conversion of domestic gardens to housing lots, will potentially have the effect of inhibiting the conversion of suburbs into more healthy places to live.

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Introduction

Cities are experiencing rapid urban growth driven by land-use changes, demographic shifts, and socio-economic development, resulting in new public health challenges.[1,2] Obesity has emerged as a global pandemic;[3] excessive adiposity being an important risk factor for morbidity and mortality from type 2 diabetes as well as for cardiovascular disease and cancer.[4–6] The residential built environment has been shown to be one of the first causes of obesity.[7–9] A systematic review of 132 articles concluded that neighbourhoods with high residential density, street intersections, and services were associated with lower risks of obesity.[10]

Housing is the fundamental component of a city's built environment and residential density, expressed as the number of residential units per residential acre, constitutes

Research in context

Evidence before this study

We searched PubMed, MEDLINE, EBSCO, Scopus, and Google/Google Scholar databases for studies and reports published in English between Jan 1, 2003, and May 15, 2017, using terms including "residential/housing density", "urban density", "built environment", and "walkability"; and body mass index", "physical activity", "obesity", and "adiposity". There is now an increasing body of evidence that several measures of high urban density, including residential density, retail and service density, street-intersection density, and land-use diversity, are all associated with lower body-mass index and obesity. Housing is the fundamental component of any city's built environment and residential density is a key parameter around which land uses and services that affect health are planned and developed. The evidence for identifying the optimum residential density in relation to obesity has been far from compelling. Very few studies have been done examining the associations between housing unit density and adiposity. Previous studies have mostly used aggregate-level, census-defined data with limited reliability. In some of the studies, residential density only constitutes one component of composite indicators of sprawl and walkability, and a dose–response relationship cannot be established. Most of the studies so far have been small scale, in low-density settings, and within relatively homogeneous environments, generating limited statistical power. As we build and retrofit our cities, there is an increasing necessity to examine the dose–response relationship between residential density and adiposity to identify optimum density environments for sustaining healthy weight.

Added value of this study

The study analysed a high-quality dataset of unprecedented size (>419 000 individuals across 22 UK cities) and diversity, both in terms of population characteristics and environmental exposures. The study included highly characterised built environment data with objectively measured housing unit density, street-level movement density, and density of activity-influencing destinations measured at individual level. The study uses objective measures of body-mass index, waist circumference, and whole body fat as indicators of adiposity. These design features enabled analyses with exceptional detail and statistical rigour. Our study is the first to have systematically investigated non-linear associations across the continuum of residential density and to examine effect modification (by age, sex, employment status, and levels of physical activity). The association of residential density and adiposity has been shown to be curvilinear, with the dose–response curve detecting a turning point at 1800 residential units per km^2 and the findings being consistent across all three measures of adiposity and robust to adjustments. Below 1800 housing units per km^2, higher residential density was associated with higher adiposity, whereas above 1800 units per km^2 the associations were protective.

Implications of all the available evidence

Housing unit density is independently associated with adiposity. The curvilinear dose–response curve of residential density and adiposity, with an identified turning point at a density of 1800 residential units per km^2, might have important public health consequences—especially in the identification of parameters for residential density associated with low adiposity, thereby guiding evidence-based policy related to densification as an upstream obesity prevention intervention. Further longitudinal evidence is needed to identify potential pathways and thereby effectively guide policy.

one of the simplest proxies of urban densification. Specific residential density profiles have carrying capacities to support particular levels of health-influencing land uses and services in their neighbourhood. As such, optimisation of residential density profiles to make them capable of supporting health-promoting infrastructures and behaviour via the allocation of housing stocks in developing cities and via retrofitting in fully developed cities is intrinsic to healthy urban living. Increased residential density has been hypothesised to support compact mixed-use urban development, enhancing street-level physical accessibility and connectivity to employment and service destinations. Increased residential density is also thought to be associated with reduced private vehicle-miles travelled and an increased frequency of active travel and physical activity.[11–13] The protective effects of optimum urban density on obesity have been reported in previous studies through direct density metrics[14,15] and through more composite indicators of urban sprawl.[16,17]

With the global effect of urban densification, more specifically, the pervasive effect of the built environment on health, the evidence for the identification of the optimum residential density in relation to obesity is a current priority. No studies on the dose–response relationship between residential density and adiposity have been done so far that adjust for all other activity-influencing built environment factors to identify optimum density levels for guiding healthy housing policies. Furthermore, most studies use census-defined aggregate data, are relatively small scale, and are done in low-density settings within relatively homogeneous environments.

To establish robust evidence of the links between residential density and obesity, we assessed a large and diverse population sample for residential density and various indicators of adiposity. We modelled objective measures of residential density and activity-influencing built environment at an individual level of analysis to identify optimum levels of residential density for sustaining healthy weight.

Methods

Study population

For this full-data, cross-sectional analysis, we obtained data from the UK Biobank, a prospective population

cohort of 502 649 adults aged 37–73 years (99·9% aged between 40–69 years) at recruitment. Participants were selected from the National Health Service patient register and resided within a 25 mile radius of one of the 22 assessment centres. The baseline examination (2006–10) included detailed questionnaires on sociodemographics, lifestyle, psychosocial factors, and medical history; anthropometric measurements; biosampling (blood, urine, and saliva); imaging; cognitive function; and hospital-related outcomes.[18] Details of the UK Biobank study protocol, including the scientific rationale and study design, can be found elsewhere.[19]

UK Biobank received ethical approvals from the North West Multi-centre Research Ethics Committee, the Community Health Index Advisory Group, the Patient Information Advisory Group, and the National Health Service National Research Ethics Service.

Data on adiposity outcomes, sociodemographics, and related covariates were collected at baseline (2006–10), whereas individual-level built environment exposures were assessed towards the end of the baseline phase (2010).

Obesity indicators

Conventional measures of body-mass index (BMI) in kg/m², waist circumference in cm, and whole body fat in kg constituted the primary indicators of adiposity. They were assessed as per UK Biobank's standard protocol for anthropometric measurements.[19,20] Additionally, WHO's definition of obesity as BMI of 30 kg/m² or higher was used. Standing height (cm) was measured with a Seca 202 device and waist circumference (cm) with a Wessex non-stretchable sprung tape. Weight and whole body fat mass (kg) were measured using electrical bio-impedance with the Tanita BC-418 MA body-composition analyser. BMI was derived by dividing weight (kg) by the square of standing height (m²).

Exposure assessment: dwelling environment

Built environment data were derived from the UK Biobank Urban Morphometric Platform (UKBUMP). UKBUMP comprises a set of urban objectively assessed metrics of density, design, and accessibility to quantify health-influencing environmental exposures within predefined catchments of each participant's geocoded dwelling address. In reference to each Biobank participant's dwelling location, multiple health-influencing and activity-influencing built environment metrics were developed through a series of Geographic Information System-based spatial and network analyses of the UK Ordnance Survey spatial database and other national-level datasets. The methods are detailed elsewhere.[21] These built environment metrics of UKBUMP were previously piloted in the Caerphilly Prospective study[22] and were used in the obesity-risk–built-environment study[23] within the UK Biobank.

We modelled density metrics from the UK-wide AddressBase Premium data of Ordnance Survey, which comprised approximately 36 million valid address point features of 550 different land-use classifications. Residential density (units per km²) was defined as the number of housing units, including detached, semi-detached, terraced, and self-contained flats within a street catchment of 1 km of a participant's geocoded dwelling. We had tested multiple neighbourhoods as a part of a previous sensitivity analysis with pilot data and chose the criterion of a 1 km network catchment for land-use density variables corresponding to a 10 min walk from the geocoded dwellings.[22] The criterion also corresponds well with a-priori evidence on functional neighbourhoods in previous built-environment–adiposity studies.[14]

Other activity-influencing, built-environment density variables statistically controlled for included exposure to retail outlets and public transport (bus stops and train stations), expressed as the density (number per km²) within the 1 km home neighbourhood. A graphical metric of "betweenness centrality" was used as a proxy for street-level movement density, expressed in terms of the underlying morphology and design of street network, and modelled through network analyses[24] of the Ordnance Survey Integrated Transport Network database, which comprises 5 million street links within a 50 km radius of each of the UK Biobank assessment centres. Movement density within an 800 m street network catchment of a participant's dwelling was expressed as the simulated counts of movement along each street link adjoining a participant's dwelling given its relative position in the network and its topological connectivity with all other links in the network.[21,22] Our choice of 800 m corresponded to a network activity space equivalent to a 10 min walk and was found to be correlated with walking[25] and obesity.[23] Movement density or "betweenness" of any street link (x) in a graph of a number of links (N) can be defined as:

$$Bt\ Wl\ (x) = \sum_{y \epsilon N} \sum_{z \epsilon R_y} L\ (y) L\ (z) P\ (z) OD\ (y, z, x)$$

In this formula, y and z are the geodesic endpoints; R_y is the set of links within a defined radius (800 m in this case) from y; L(y) and L(z) are length of links y and z, respectively; and P_z is the proportion of link z within the defined radius.

The origin-destination (OD) function is defined as:

$$OD = \begin{cases} 1, \textit{ if } x \textit{ is on the geodesics from } y \textit{ to } z \\ \frac{1}{2}, \textit{ if } x \equiv y \not\equiv z \\ \frac{1}{2}, \textit{ if } x \equiv z \not\equiv y \\ \frac{1}{2}, \textit{ if } x \equiv z \equiv y \\ 0, \textit{ otherwise} \end{cases}$$

These built environment variables were categorised into quartiles and modelled as 4-level factors.

Covariates

The addition of study covariates was theoretically informed from a-priori research evidence on the links between obesity and sociodemographic, lifestyle, and environmental factors. Sociodemographic covariates comprised age, sex, employment status, and education. Employment status was expressed as a 3-level factor (employed; retired; and unemployed, home maker, or other). Self-report education was coded as a 5-level factor (no qualification; O levels, GCSEs, or CSEs [school certificate]; A levels or AS levels [high school certificate]; NVQ, HND, HNC, or other professional qualification; and college or university degree). Among the lifestyle-level factors, vehicle ownership was categorised as none, one, two, and more than two, whereas housing tenure was modelled as a 3-level factor (own outright, own with mortgage, and rented). Processed meat intake was included as a dietary variable expressed as never, up to once a week, two to four times a week, more than four times a week. In a subsample of 60 694 participants, we obtained data on dietary energy intake (in kJ) from the 24 h diet recall questionnaire.[26] Intake was calculated from food and beverage consumption yesterday, excluding any supplements. Smoking was coded as a 3-level factor (never, previous, or current). Family history of disease was derived from history of mother's illness, coded as a 3-level variable (none; heart or cerebrovascular disease; or cancer). Self-reported medication use for cholesterol, blood pressure, or diabetes, or use of exogenous hormones was coded as a 4-level variable (none; use of medication for cholesterol; use of medication for hypertension; insulin-use, hormone replacement, or contraceptive pills; and medicine for multiple morbidities). Neighbourhood deprivation (expressed in terms of Townsend score) was derived from postcode of residence and categorised into quintiles. Physical activity behaviour, expressed in metabolic equivalent of task (MET) h/week, was derived from the self-reported International Physical Activity Questionnaire short form and was computed as the sum of weekly walking, moderate physical activity, and vigorous physical activity components.[27] Physical activity data across all the three activity components were available for 331 814 participants.

See Online for appendix

Statistical analysis

For this cross-sectional analysis, a systematic multilayered analysis protocol was pursued. Separate linear models were fitted for each of the adiposity outcomes using the full data. BMI, waist circumference, and whole body fat mass were treated as continuous outcomes, and odds of obesity (in reference to non-obese: BMI <30 kg/m²) was modelled as a binary outcome. Residential density acted as a continuous linear predictor in our models. We estimated average effects of 1000 units per km² (10 units per ha) increments in residential density (which is also equivalent to approximately 1 IQR) on adiposity outcomes. The initial model building exercise involved sequential introduction of covariates and assessment of collinearity and fit statistic. Model 1 included sociodemographic and lifestyle level variables of age, sex, education, employment status, vehicle ownership, housing tenureship, smoking status, processed meat intake, mother's illness, and medication use, and health-influencing built environment density variables of density of retail outlets, public transport, and street-level walking density. In model 2, neighbourhood-level deprivation was introduced. Model 3 further controlled for physical activity.

As a second step, we repeated these analyses using restricted cubic spline (RCS) models to examine non-linearity in the associations between residential density and adiposity, with a restricted spline basis for residential density. Harrell's knots were placed at equal percentiles of the data on the basis of a series of prior iterations, with 3, 4, 5, 6, and 7 knots along the residential density spline. Respective goodness-of-fit by Akaike information criteria were compared to produce the most parsimonious model.[28] The fitted RCS curve showed the variation of adiposity in relation to the residential density continuum. The mfxrcspline command in Stata was further used to generate marginal effect plots of first derivative or slope of the adiposity function with respect to residential density. The marginal effect plots determined the so-called turning point in the association. Linear models were then fitted piecewise on either side of the identified turning point.

Finally, effect modification was examined by sequentially introducing interaction terms between the residential density spline basis and four variables (age, sex, employment status, and physical activity behaviour), adjusting for all other factors.

For each point estimate, two-tailed 95% CIs estimated by bootstrapping are presented. Stata 14 was used for all analyses.

Role of the funding source

The study was done using UK Biobank resources. The funders and UK Biobank participants did not participate in developing the research questions, outcome measures, and environmental measures of the present study. They had no role in the study design, modelling, data analysis, data interpretation, or writing of the report. CS, CW, and JG had full access to all the data in the study, take responsibility for the integrity of the data and the accuracy of the data analysis, and had final responsibility for the decision to submit for publication.

Results

Of 502 649 adults in the prospective cohort, 419 562 (83·5%) participants across 22 UK Biobank assessment centres had valid baseline data on BMI, waist circumference, body fat, residential density, and other built environment densities, and were included in the analytic sample (table 1). The three markers of adiposity (BMI, waist circumference, and whole body fat) used as outcome measures in respective

models were positively correlated with Pearson's correlation coefficient ranging from 0·65 to 0·88. A summary of residential density and other built environment exposures is presented in table 2. The mean duration of residence was 17·4 years.

Overall, an inverse linear association between residential density and obesity was found (table 3). Collinearity remained low, with the variance inflation factor lying in the range of 1·2 and 1·9. For the fully controlled model, a 1000 dwelling units per km² increment within a 1 km dwelling catchment was associated with lower adiposity across the four indicators of BMI, waist circumference, whole body fat, and obesity (table 3).

Goodness-of fit tests identified models with three knots as parsimonious fits for the adiposity outcomes. The variance inflation factor remained between 1·2 and 2·2. The fitted RCS models of adiposity-residential density dose–response are shown in figure 1. A clear obesogenic asymptote corresponding to the turning point (change in direction of the slope) was consistently seen at a residential density of about 1800 units per km² for all the three measures of adiposity. Fully adjusted piece-wise models developed on either side of the detected turning point are presented in table 4 (see the appendix for full results). Below a residential density of 1800 units per km², an increment of 1000 units per km² was associated with higher BMI (β 0·19 kg/m², 95% CI 0·14 to 0·24), waist circumference (β 0·41 cm, 0·28 to 0·54), and whole body fat (β 0·40 kg, 0·30 to 0·50), and with increased odds of obesity (odds ratio [OR] 1·10, 1·07 to 1·14). Above 1800 units per km², residential density had a protective effect across all the markers of adiposity, being beneficially associated with BMI (β –0·22 kg/m², –0·25 to –0·20), waist circumference (β –0·54 cm, –0·61 to –0·48), and whole body fat (β –0·38 kg, –0·43 to –0·33), and with decreased odds of obesity (OR 0·91, 0·90 to 0·93).

As a sensitivity analysis, we further examined the relation between physical activity and residential density by rerunning the RCS model with log-transformed physical activity (figure 2), adjusting for all other variables. We also estimated the odds of reporting less than the 7·5 MET h/week activity (cutoff corresponding to evidenced health benefits[29]) either side of the identified turning point in the adiposity–density relationship. The OR of reporting low physical activity per 1000 units per km² increment in residential density was 1·14 (95% CI 1·09–1·19) for a density of 1800 units per km² or less, and 0·95 (0·94–0·97) for a density greater than 1800 units per km².

To examine the effects of residential density among participants who are not obese but at risk of obesity, we excluded obese participants from the analyses and modelled the odds of being overweight (BMI ≥25 kg/m² and <30 kg/m²) in reference to the healthy weight (BMI <25 kg/m²) category. We found a slight attenuation in the effects per 1000 units per km² increment in residential density. The OR for overweight was 1·04 (95% CI 1·01–1·07) for residential densities of 1800 units per km² or less, and 0·95 (0·93–0·96) for densities greater than 1800 units per km².

We also tested the effects of additionally controlling for dietary energy intake in a subsample (n=49 981) for whom calorific data were available. Effect estimates remained

	Not obese (n=319 151)	Obese (n=100 411)	Overall (n=419 562)
BMI (kg/m²)	25·3 (2·7)	33·9 (3·8)	27·4 (4·7)
Waist circumference (cm)	85·3 (10·3)	104·7 (11·0)	90·0 (13·4)
Whole body fat mass (kg)	21·1 (6·0)	36·6 (9·1)	24·8 (9·5)
Physical activity (MET h/week)*	48·2 (50·6)	40·7 (48·7)	46·5 (50·3)
Age (years)	56·4 (8·1)	56·8 (7·8)	56·5 (8·0)
Sex			
Female	179 710 (56·3%)	53 953 (53·7%)	233 663 (55·7%)
Male	139 441 (43·7%)	46 458 (46·3%)	185 899 (44·3%)
Education			
None	46 307 (14·5%)	20 897 (20·8%)	67 204 (16·0%)
College or university degree	114 516 (35·9%)	25 588 (25·5%)	140 104 (33·4%)
O levels/GCSEs/CSEs	85 185 (26·7%)	29 427 (29·3%)	114 612 (27·3%)
A levels/AS levels	37 424 (11·7%)	10 735 (10·7%)	48 159 (11·5%)
NVQ/HND/HNC/other professional qualification	35 719 (11·2%)	13 764 (13·7%)	49 483 (11·8%)
Employment status			
Employed	190 147 (59·6%)	56 831 (56·6%)	246 978 (58·9%)
Retired	106 396 (33·3%)	33 578 (33·4%)	139 974 (33·4%)
Unemployed/home maker/ other	22 608 (7·1%)	10 002 (10·0%)	32 610 (7·8%)
Vehicle ownership			
None	22 173 (6·9%)	9365 (9·3%)	31 538 (7·5%)
One	130 946 (41%)	42 936 (42·8%)	173 882 (41·4%)
Two	128 681 (40·3%)	36 093 (35·9%)	164 774 (39·3%)
More than two	37 351 (11·7%)	12 017 (12%)	49 368 (11·8%)
Tenureship			
Own outright	174 728 (54·7%)	48 589 (48·4%)	223 317 (53·2%)
Mortgage	120 477 (37·7%)	39 074 (38·9%)	159 551 (38·0%)
Rent	23 946 (7·5%)	12 748 (12·7%)	36 694 (8·7%)
Smoking status			
Non-smoker	180 969 (56·7%)	51 715 (51·5%)	232 684 (55·5%)
Previous smoker	105 859 (33·2%)	39 446 (39·3%)	145 305 (34·6%)
Current smoker	32 323 (10·1%)	9250 (9·2%)	41 573 (9·9%)
Processed meat intake			
Never	32 325 (10·1%)	5999 (6%)	38 324 (9·1%)
Up to once per week	194 449 (60·9%)	57 951 (57·7%)	252 400 (60·2%)
Two to four times per week	80 999 (25·4%)	31 863 (31·7%)	112 862 (26·9%)
More than four times per week	11 378 (3·6%)	4598 (4·6%)	15 976 (3·8%)
Calorific energy intake (KJ)†	8794·42 (3100·9)	8943·34 (3416·5)	8827·86 (3175·1)
Medication use			
None	231 974 (72·7%)	55 014 (54·8%)	286 988 (68·4%)
Cholesterol lowering	18 535 (5·8%)	7248 (7·2%)	25 783 (6·1%)
Blood pressure medication	24 877 (7·8%)	14 686 (14·6%)	39 563 (9·4%)
Insulin, hormone replacement, and contraceptive pills	16 453 (5·2%)	3080 (3·1%)	19 533 (4·7%)

(Table 1 continues on next page)

	Not obese (n=319 151)	Obese (n=100 411)	Overall (n=419 562)
(Continued from previous page)			
Multiple morbidities	27 312 (8·6%)	20 383 (20·3%)	47 695 (11·4%)
Mother's illness			
None	229 414 (71·9%)	66 913 (66·6%)	296 327 (70·6%)
Heart or cerobrovascular disease	74 471 (23·3%)	28 241 (28·1%)	102 712 (24·5%)
Cancer	15 266 (4·8%)	5257 (5·2%)	20 523 (4·9%)

Data are mean (SD) or n (%). Not obese is defined as BMI of less than 30 kg/m² and obese is defined as BMI of 30 kg/m² or higher. BMI=body-mass index. MET=metabolic equivalent of task. *331 814 participants with valid MET data across the three physical activity categories. †60 694 participants with valid MET data on energy intake.

Table 1: **Participant characteristics**

consistently significant, being slightly modified in the case of waist circumference and whole body fat (in reference to the models over the full data; appendix).

From an urban planning perspective, UK policy guidelines consider densities below 3000 units per km² as inefficient use of land and encourage housing development to have densities of 3000–5000 units per km².[30] As a sensitivity analysis to further interpret our findings in relation to prevailing residential densification scenario, we created an additional cutoff point at 3200 units per km² (32 units per ha) and obtained piecewise effect estimates in the density ranges 1800–3200 and more than 3200 units per km². This cutoff point represents the mean residential density surrounding newly developed residential units over the period 2014–16.[31] The associations were protective within the density range 1800–3200 units per km² for both BMI (β–0·13 kg/m², 95% CI –0·21 to –0·05) and obesity (OR 0·98, 0·94 to 1·02; table 5). However, a more pronounced beneficial effect was seen beyond the density of 3200 units per km² for BMI (β –0·17 kg/m², –0·21 to –0·12) and obesity (OR 0·92, 0·90 to 0·94). Similar trends were seen in the cases of waist circumference and whole body fat (table 5).

Interaction terms were introduced to examine modification by age, sex, employment status, and physical activity (figure 3, appendix). Effects on adiposity of increasing residential density were greater in younger age groups, women, employed people, and people who are more physically active (≥50 MET h/week), with a $p_{interaction}$ of less than 0·01 in each case. The results in the case of whole body fat suggested a more pronounced protective effect in men than in women. The mean whole body fat was also higher in women (26·9 kg [SD 10·0]) than in men (22·3 kg [SD 8·2]).

High adiposity might be associated with self-selection and migration to low density areas. As a further sensitivity analysis, adiposity was correlated with density according to high (>10 years), medium (3–10 years), and low (<3 years) duration of residence. No differences in the association were found between these subgroups.

Discussion

Using objective measures of the built environment, in a large and diverse population sample, we have shown the association of residential density and adiposity to be curvilinear, suggesting a turning point at a residential density of 1800 units per km². This finding was consistent across all three measures of adiposity, with stronger associations being found among people who are younger, female, employed, and accumulating higher levels of physical activity.

Overall, our results suggest that high residential density was significantly and independently associated with lower adiposity outcomes. These results are consistent with a

	Mean (SD)	Minimum	P_{25}	P_{50}	P_{75}	Maximum
Residential density (units per km²)	1877·65 (1115·62)	0	1235·94	1755·89	2267·88	10 513·24
Retail density (units per km²)	43·22 (64·70)	0	7·32	21·09	52·85	1752·07
Public transport density (units per km²)	22·61 (10·86)	0	15·51	21·72	28·45	207·23
Street-level movement density	$4{\cdot}58\times10^6$ ($5{\cdot}46\times10^6$)	5076·23	$8{\cdot}48\times10^5$	$2{\cdot}63\times10^6$	$6{\cdot}38\times10^6$	$8{\cdot}75\times10^7$
Townsend deprivation index	–1·50 (2·95)	–6·26	–3·71	–2·28	0·20	11·0

P_{25}, P_{50} and P_{50} represent the 25th, 50th, and 75th data percentiles.

Table 2: **Descriptive statistics of environmental exposure variables**

	BMI (kg/m²; β [95% CI])	Waist circumference (cm; β [95% CI])	Whole body fat (kg; β [95% CI])	Obesity (BMI>30 kg/m²; OR [95% CI])
Model 1 (n=419 562)*	–0·05 (–0·07 to –0·03)	–0·11 (–0·15 to 0·07)	–0·08 (–0·11 to –0·05)	0·99 (0·98 to 1·00)
Model 2 (n=419 562)†	–0·13 (–0·15 to –0·11)	–0·29 (–0·33 to –0·24)	–0·21 (–0·24 to –0·18)	0·96 (0·95 to 0·97)
Model 3 (n=331 814)‡	–0·14 (–0·15 to –0·12)	–0·30 (–0·35 to –0·26)	–0·22 (–0·26 to –0·19)	0·95 (0·94 to 0·96)

Models contain UK Biobank data for 419 562 participants. Residential density was per 1000 units per km². BMI=body-mass index. OR=odds ratio. *Effect estimates after controlling for age, sex, education, employment, car ownership, housing tenureship, smoking status, processed meat intake, mother's illness, and medication use, and health-influencing built environment densities variables (density of retail outlets, public transport, and street-level movement density). †Effect estimates after further controlling for neighbourhood-level deprivation. ‡Fully adjusted effect estimates after additionally controlling for physical activity (metabolic equivalent of task h/week).

Table 3: **Linear models of association of adiposity with residential density**

few mostly small-scale studies on the topic. In a study[14] of 10 878 participants residing in a low-density environment in Atlanta, GA, USA, significant negative correlation was found between residential density and BMI and distance walked in men. Density was expressed in people per acre within census block group. Another large-scale Canadian study[15] involving 3·78 million participants from Toronto and 1·63 million from Vancouver[15] found a protective association between residential density and BMI. Their density measure was similar to the present study's and the reported effect estimates for BMI were slightly lower for Toronto (at –0·05 kg/m²) but higher for Vancouver (at –0·30 kg/m²). Another small-scale ecological study[32] of 2375 adolescents from Nanjing, China, found that participants residing in the highest density tertile had 2·17 times higher odds of being overweight compared with participants in the lowest tertile. However, this was an ecological study with a poorer resolution-density measure (expressed in terms of number of residents per km² within districts) for children. With respect to physical activity, a 2016 multicountry study[13] involving 6822 adults across 14 cities reported a consistent positive association between net residential density and moderate-to-vigorous-intensity physical activity.

None of the previous residential density–adiposity studies investigated non-linearity and associations stratified across density profiles. In this regard, our findings are important in principle and in practice. In principle, the findings show the value of a large, high-quality health dataset and detailed environmental metrics to identify environmental determinants of chronic disease. In practice, the curvilinear association of residential density with adiposity identifies a turning point with public health consequences, providing a metric for the estimation of the effect of housing policy and urban planning on adiposity.

In a low-residential-density setting of 1800 units per km² or less, the positive association between density and adiposity might point to a low density suburban sprawl effect. Typically, the left end of the curve reflects semi-urban neighbourhoods that facilitate walkability and physical activity via access to private outdoor spaces in the form of larger residential gardens[33] and enhanced exposures to salutogenic green and open amenities.[34] The density nearing the turning point of the RCS curve (at 1800 units per km²) might be a proxy of areas experiencing suburban sprawl. The positive associations of suburban sprawl with obesity[16,17] and inadequate physical activity[35] are well established, primarily on account of sedentary lifestyles, including increased vehicle-miles travelled to employment centres and decreased activity levels. Our physical activity analysis supports this finding with higher odds of reporting less than 7·5 MET h/week of physical activity. Above 1800 units per km², increasing density had a protective effect on adiposity. This density range corresponds to urban suburbs, compact inner suburbs, and city centres,

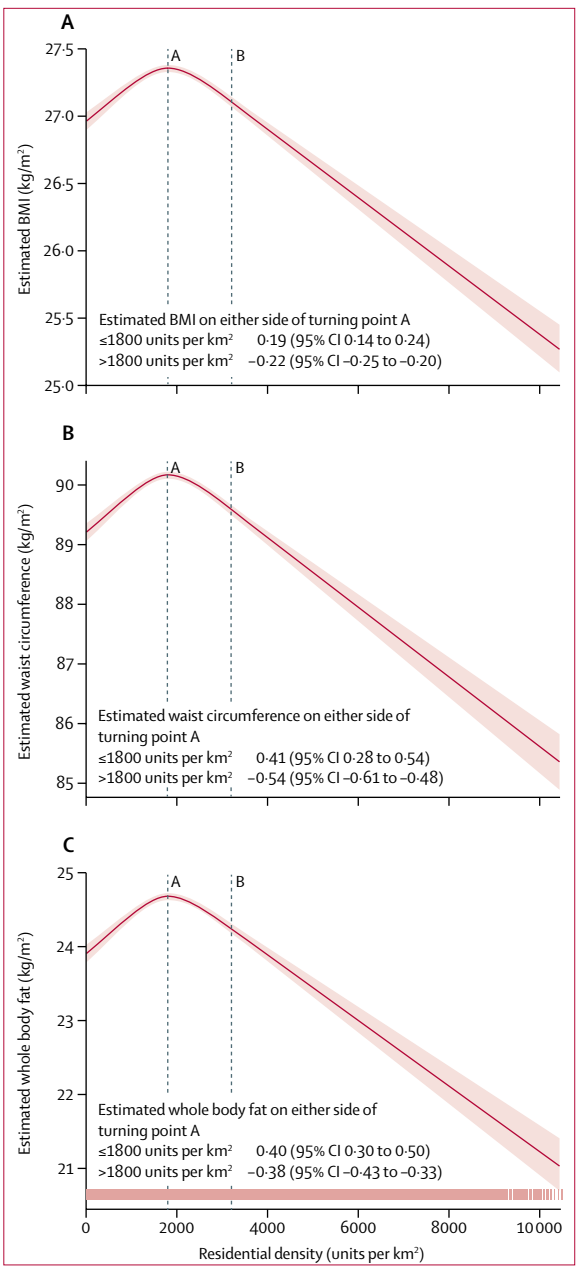

Figure 1: **Association between adiposity and housing density, allowing for non-linear effects**

The continuous line represents the estimated mean adiposity outcome and shaded areas represent 95% CIs. The barcode shows the distribution of the analytic sample across the residential density continuum. Separate models were fitted for BMI, waist circumference, and whole body fat, with restricted cubic splines with Harrell's knots, adjusting for age, sex, education, employment, car ownership, housing tenureship, smoking, processed meat intake, mother's illness, medication use, physical activity, retail, public transport, street-level movement density, and neighbourhood deprivation. Point A indicates the detected turning point of the curve (at which the first derivative or slope changes sign), observed at a density of 1800 units per km². Point B represents the point in the curve corresponding to the residential density of 3200 units per km², which is the density of newly developed housing in the UK over the past 2 years. Effect estimates (β) were measured per 1000 units per km² increase in residential density from piecewise linear models, fitted on either side of the turning point A, of the best fitting restricted cubic spline (chosen on the basis of goodness-of-fit parameter; Akaike information criterion). BMI=body-mass index.

	BMI (kg/m²; β [95% CI])	Waist circumference (cm; β [95% CI])	Whole body fat (kg; β [95% CI])	Obesity (BMI>30 kg/m²; OR [95% CI])
≤1800 units per km²	0·19 (0·14 to 0·24)	0·41 (0·28 to 0·54)	0·40 (0·30 to 0·50)	1·10 (1·07 to 1·14)
>1800 units per km²	−0·22 (−0·25 to −0·20)	−0·54 (−0·61 to −0·48)	−0·38 (−0·43 to −0·33)	0·91 (0·90 to 0·93)

Models used fully adjusted effect estimates controlling for age, sex, education, employment, car ownership, housing tenureship, smoking, processed meat intake, mother's illness, medication use, physical activity, retail density, public transport, street-level movement density, and neighbourhood deprivation (n=331 814). The turning point (point A in figure 1) in the restricted cubic spline curves was detected at 1800 residential units per km². Residential density was per 1000 units per km². BMI=body-mass index. OR=odds ratio.

Table 4: **Fully adjusted piece-wise linear models fitted on either side of the detected turning point in the adiposity–residential density curve**

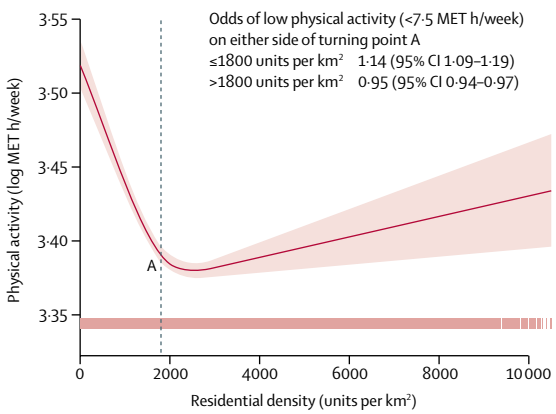

Figure 2: **Association between physical activity (log-transformed MET h/week) and residential density, allowing for non-linear effects**
Shaded areas represent 95% CIs. The barcode shows the distribution of the analytic sample across the residential density continuum. Restricted cubic splines with Harrell's knots were fitted, adjusting for age, sex, education, employment, car ownership, housing tenureship, smoking, processed meat intake, mother's illness, medication use, physical activity, retail, public transport, street-level movement density, and neighbourhood deprivation. Point A indicates the detected turning point of the adiposity–residential density curve (figure 1), observed at a density of 1800 units per km². The odds ratios for doing low physical activity (<7·5 MET h/week) are reported on either side of point A. MET=metabolic equivalent of task.

which are well evolved and designed to accommodate densification with additional supporting infrastructures in the form of retail, employment centres, pedestrian facilities, and other attractive destinations. These urban design features could promote increased walkability and physical activity. The physical activity model corroborated this hypothesis, finding lower odds of reporting less than 7·5 MET h/week of physical activity in this density range. The observed associations in these medium-to-high density regions might point to a physical activity-related mechanism, with the residential exposures acting as a proxy of the degree of walkability and activity friendliness of a neighbourhood. Further evidence based on accumulated longitudinal data are needed to conclusively verify such a causal assumption. We also note that fitted RCS curves can be applied with confidence to the UK context but might not be valid in other countries. These models, when calibrated in the USA and Australia would probably lie along the left of our turning point of 1800 units per km² because, apart from the most densely populated parts, densities tend to be below this figure in these countries (the USA has an average residential density of 1200 units per km² and Australia of 1000 residential units per km²).

Although present UK housing density averages 2300 dwellings per km², the mean density around dwelling addresses newly allocated in 2013–14 was 3100 units per km², and 3200 units per km² for 2014–16.[31] From an urban health policy perspective, our models suggest a more pronounced protective effect beyond the range of 3200 units per km² (compared with 1800–3200 units per km²), supporting the policy towards healthy suburban densification. As a recommendation for practice, for dense and compact environments to become active environments, planners need to design them as multifunction urban spaces rather than monofunction suburban spaces.[1,36]

In view of the potentially significant benefits of healthy density environments on weight outcomes, understanding underlying pathways merits further investigation for effective policy making. First, high residential density is synonymous with compactness, greater access to destinations, and walkability, and thus active travel. Positive associations with healthy weight have been reported for walkability[37] and active commuting.[38] A physical activity-based pathway of energy expenditure and corresponding obesity reduction is plausible in view of the evidenced activity-promoting effects of housing density mentioned previously.[13] Physical activity is related to adiposity via its role in influencing levels of individual energy expenditures.[39] Although we were not able to directly test for mediation, our RCS model for physical activity found that higher residential density (more than 2000 units per km²) had beneficial effects on accumulated weekly activity, whereas the interaction models found that the protective effects of density on adiposity were more pronounced among the highly active participants (>50 MET h/week). Second, a highly compact dense residential environment might act as a proxy for enhanced community social capital and support. The intangible stress-relieving potential of centrality, accessibility, and social capital needs to be further examined in view of their protective effects on obesity.[40] Third, high residential density, after adjusting for all other built environment factors, might also represent well designed environments shielding exposures to stressors related to proximity to roads, and having greater provisions for recreational

	BMI (kg/m²; β [95% CI])	Waist circumference (cm; β [95% CI])	Whole body fat (kg; β [95% CI])	Obesity (BMI>30 kg/m²; OR [95% CI])
1800–3200 units per km²	−0·13 (−0·21 to −0·05)	−0·19 (−0·38 to −0·00)	−0·14 (−0·29 to 0·02)	0·98 (0·94 to 1·02)
>3200 units per km²	−0·17 (−0·21 to −0·12)	−0·53 (−0·64 to −0·43)	−0·31 (−0·39 to −0·22)	0·92 (0·90 to 0·94)

The turning point (point A in figure 1) in the restricted cubic spline curve was detected at 1800 residential units per km² and the cutoff point (point B in figure 1) was at 3200 residential units per km² corresponding to the current density surrounding newly developed residential units in the UK. Models used fully adjusted effect estimates controlling for age, sex, education, employment, car ownership, housing tenureship, smoking, processed meat intake, mother's illness, medication use, physical activity, retail density, public transport, street-level movement density, and neighbourhood deprivation. Residential density was per 1000 units per km². BMI=body-mass index. OR=odds ratio.

Table 5: **Sensitivity analyses beyond the detected turning point with piece-wise linear models fitted on either side of the cutoff point in the adiposity–residential density curve**

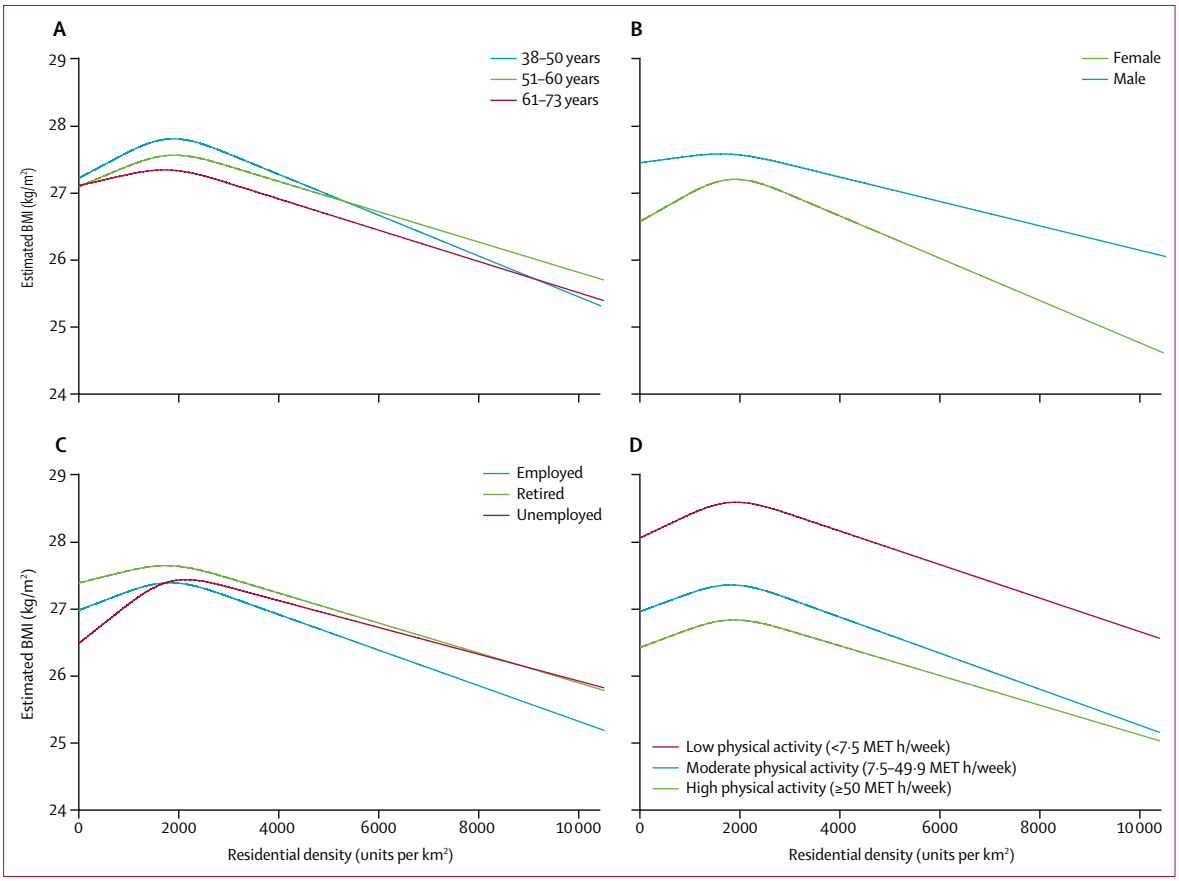

Figure 3: **Association between BMI and housing density with effects modification by age, sex, employment status, and physical activity**
BMI=body-mass index. MET=metabolic equivalent of task.

activity spaces such as greenspace. The obesogenic potential of air quality and noise pollution[41–43] is well established, and so are the protective effects of green access.[23]

Limitations originate from the use of a cross-sectional and observational study design, designs used by most studies examining associations between health and environmental exposures. The cross-sectional design prevented causal inference. We could not take into account the effect of self-selection, with high adiposity attributed to people moving into areas of low residential density. To address this potential limitation, we examined the association of density with adiposity stratified by duration of residence and detected no differences between duration groups; however, reverse causation cannot be ruled out. Another limitation of cross-sectional data is that they might not reflect the long-term effect of an exposure. However, a mean duration of residence of 17·5 years suggests a cohort with fairly stable residential histories. Future studies should use accumulated longitudinal data to model changes in health in relation to changes in the built environment. Similarly, as accumulated follow-up data become available in the UK Biobank, other designs, such as the effect of migration from one density

environment to another, might also be considered for a small subsample of participants moving houses.

Large-scale data also pose some challenges. Temporal mismatch between assessment of outcomes and covariates and measurement of exposures was inevitable. The model outcomes and covariates were assessed during the study's baseline period (2006–10), whereas the built environment was measured in 2010. This temporal mismatch was unlikely to have significantly affected our findings because the built environment typically changes slowly in the UK. The issue of inferring small effects (often encountered in environment–health studies) with caution in big data statistical analyses must be stated. Our data also deciphered significant differences among population subgroups. We followed a detailed multilayered statistical modelling framework. In addition to preliminary analyses using the full data, subsequent models were developed by stratifying the data by density profiles and meaningful population characteristics, thereby obtaining a range of reliable estimates with respect to different chunks of data.

The risk of residual confounding exists, as in all observational data. Apart from the built environment, the study did not consider other environmental attributes, including land-use heterogeneity, quality of transit services, green quality, and recreation potential. We could not study how these effects might have moderated the associations. The study did not have data on weight-influencing intestinal microbiota and calorific intake data were available for a subset of participants. The intangible effects of prevailing urban and environmental policy and demographic contexts also need to be controlled for to enhance generalisability in future studies.

The range of residential density available to the analysis was representative of the UK's semi-urban and urban environments. However, the findings should not be extrapolated beyond this range for other socially and culturally different contexts. Cities in the UK, as with most European cities, do not have the very high-density developments commonly encountered in Asian population centres, such as in Hong Kong, mainland China, and India, and it's likely that in these high-density areas other turning points would be encountered that are endemic to such density extremes. We emphasise that comparable country-specific evidence from other countries (with different contexts) needs to be reproduced by other research groups and is likely to produce different curves, but it would help us gain insights for policies regarding global healthy densification.

The UK Biobank is an adult cohort (in the age range of 38–73 years) and the reported findings are not generalisable to young adults and children. Additionally, the sampling is associated with a healthy volunteer selection bias and as such might not be representative with respect to the prevalence of obesity and related comorbidities. However, in view of the sufficiently diverse population and heterogeneity of environmental exposures, sample selection is unlikely to have a material effect on the associations reported.[44,45]

Notwithstanding the mentioned limitations, this is the first systematic, large-scale analysis examining the trajectory of adiposity along the residential density continuum after adjusting for a range of other built environment factors and covariates and testing for non-linearity and effect modification. The strengths of the study are owed to the use of a high-quality dataset of unprecedented size (n>419 000) and diversity (22 cities). The UK Biobank data underwent substantial centralised quality control. Standardised measurement protocol ensured uniformity and reliability of the markers of adiposity. In addition to the standard measure of BMI, the use of additional markers[46] provided a more holistic measure of adiposity. A distinctive feature was the use of UKBUMP built environment data, which were characterised with objective indicators.[46] Reliance on the UK-wide Ordnance Survey data meant the density data could be measured at the building-unit level within a street network catchment, and therefore be representative of functional neighbourhoods, rather than at the parcel-level data within census-defined neighbourhoods, as used in all previous studies. Data were analysed at the individual level to avoid the modifiable unit area problem and the ecological fallacy. These design features have provided analyses of exceptional detail and statistical power, enabling a multilayered analysis to detect non-linear associations across the density continuum, and the implementation of informative subgroup analyses. Although longitudinal data are required to confirm these findings, they are not dissimilar to previous reports. The inverse relationships between residential density and obesity in sprawling suburbs has been shown elsewhere,[16,17] as have differential effects according to sex[47] and age[48] on adiposity.

With increasing demand for urban housing, alongside controlling sprawl and enhancing infrastructure, sustaining healthy communities is a major planning challenge. This large-scale study provides evidence that informs the planning equation in terms of identifying parameters for residential density that might sustain healthy weight. Our study consistently found that beyond a threshold of 1800 units per km², residential density had a protective effect on adiposity outcomes, suggesting that the relentless trend towards suburban densification can plausibly be a public health opportunity to be embraced. The inference that residential density is a determinant of adiposity in the general population, and that this association can be specified with sufficient precision to inform policy in support of environments promoting healthy weight, is a conclusion of substantial public health significance that requires careful consideration by urban health planners and policy makers. Further longitudinal studies based on accumulated data are needed to measure changes in adiposity in relation to changes in density, and to infer causality and thereby

characterise obesogenic environments in view of the enduring public health value of reductions in obesity.

Contributors

CS, CW, and JG conceived the study. CS designed the study, developed the built environment metrics, and performed the formal analysis. CS drafted the report. CW and JG commented on the draft and contributed to redrafting. CS produced the final submitted draft.

Declaration of interests

We declare no competing interests.

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