



Research article

Geospatial predictors of vitamin D deficiency across London involve complex interactions between the social and built environment

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ABSTRACT

Vitamin D deficiency is a global public health concern, causing musculoskeletal complications and linked with COVID-19, cancer and other morbidities. Global prevalence is high, even in countries with abundant sunshine, suggesting a complex aetiology. Vitamin D deficiency disproportionately impacts people with higher skin melanin content, thus potentially exacerbating health inequalities amongst ethnically minoritised communities. Whilst vitamin D is acquired most efficiently through sunlight, this can be restricted in urban areas with high population and building density. Furthermore, air pollution may interfere with vitamin D synthesis by blocking UVB photons. Given that over 68% of the global population are expected to live in cities by 2050, it is essential to explore environmental barriers and opportunities to prevent vitamin D deficiency.

Drawing upon data from the UK Biobank and UK Data Service, we conducted a geographically weighted regression (GWR) analysis to predict features of the built environment that may contribute to vitamin D deficiency across London. Our GWR model is the first to identify heterogeneity in the relationship between air pollution and vitamin D across London and suggests this can be explained by the hyperlocal social and built environment. Our results describe several significant modifiable predictors of vitamin D at the Middle Layer Super Output Area (MSOA), including greenspace, air pollution and road network variables ($p < .05$).

Our findings highlight that public health interventions must address the complexity of the hyperlocal social and built environment to effectively tackle vitamin D deficiency and improve health outcomes across divides.

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1. Background

Vitamin D is a steroid hormone with a primary role in calcium homeostasis and subsequently, is well-known for regulating musculoskeletal health. However, vitamin D receptors are distributed widely, indicating the diverse roles of vitamin D including the maintenance of genomic integrity, regulating cell proliferation and initiating DNA repair [1]. The National Institute for Clinical Excellence (NICE) suggests serum vitamin D levels should be above 50 nmol/L for optimal bone health [2]. Levels between 25 and 50 nmol/L are classed as 'insufficient' and levels below 25 nmol/L as 'deficient'. Consequences of deficient vitamin D have been studied extensively, first being linked in the early 1600s with rickets in children, and osteoporosis in adults [3]. More recent studies link vitamin D deficiency with an increased risk of infections, including COVID-19, cancers of the colorectum and bladder, as well as chronic metabolic diseases including cardiovascular disease, and degenerative diseases including dementia [4–7]. Low vitamin D levels are also found in adolescents with obesity, and linked to increased oxidative DNA damage, thus exacerbating the risk of chronic conditions in later life [8]. Maternal vitamin D deficiency can also have serious consequences for neonates; linked to a higher risk of spontaneous pre-term births, gestational diabetes, toxæmia of pregnancy and small-for-dates births [9].

The most efficient way to acquire vitamin D is from exposure to sunlight. 80% of Vitamin D is derived from ultraviolet B (UVB) induced photoconversion in the skin of 7-dehydrocholesterol to previtamin D₃. Only 20% is acquired from dietary sources including oily fish, cod liver oil, fortified foods or supplementation. For this reason, vitamin D deficiency is strongly linked to the availability of UVB and is more prevalent during winter months across Europe [10]. In the European region, the British population presents with the highest rates of vitamin D deficiency. Over half the population (55.3%) possess insufficient vitamin D, and over a quarter (25.9%) are deficient [11]. Vitamin D deficiency is significantly more prevalent amongst ethnic minority groups in the UK - winter levels were 5.9% in White Britons, 30.8% in Black Britons and 50.8% in South Asians [12]. This can be partially explained by higher levels of skin pigmentation amongst ethnic minorities, as melanin absorbs UV rays and reduces the rate of vitamin D synthesis. The risk of vitamin D deficiency also increases with age and increasing levels of body fat [13,14].

It is also important to note that low vitamin D levels are not restricted to people living in countries with less sunshine, they are a prominent global concern. It is estimated that 15.7% of the global population have vitamin D deficiency whilst nearly half the global population (47.9%) have insufficient levels [11]. The highest prevalence of vitamin D deficiency is in the Middle East. Evidence on the high prevalence of vitamin D deficiency in countries with abundant sunshine suggests that there are wider public health concerns to be considered [15]. One important factor is behavioural practices. Low vitamin D levels have been linked to skin covering, use of sunscreen and reduced outdoor exposure [13]. The local environment may play a key role in determining behavioural practices and engagement with local outdoor spaces and exposure to UV rays.

Environmental determinants of vitamin D levels are potentially vast. Interestingly, vitamin D deficiency has persisted in the UK ever since the increase of urbanisation during the post-medieval period [16]. There are several potential mechanisms that link urbanisation with vitamin D deficiency. Firstly, reduced access to greenspace may limit time in outdoor spaces and thus reduce sun exposure [17]. The British Research Establishment (BRE) guidelines recommend that 50% of the garden or open space should receive at least 2 h of sunlight on March 21 [18]. However, not all households may have access to gardens or other adequate open spaces, indicating a complex interplay between the social and built environment as predictors of vitamin D status. Further, there can be substantial barriers for ethnic minorities groups to engage with green spaces [19]. Notably, green canopy cover may restrict sunlight in outdoor spaces [20], highlighting further complexities for urban planning and public health initiatives aimed at mitigating vitamin D deficiency in urban populations.

Furthermore, several studies link increased exposure to air pollution with low vitamin D levels, suggesting that air pollutants may block UVB rays from reaching the skin surface [21]. A recent review of the literature identified a negative link between air pollution and vitamin D levels across several populations including adults, children and pregnant women [22]. Poor air quality and increased exposure to nitrogen dioxide (NO₂), particulate matter (PM₁₀) and ozone were found across several studies to be associated with lower serum vitamin D levels. However, results were inconsistent across NO₂, PM_{2.5} and black carbon exposure. For example, maternal NO₂ exposure was associated with low vitamin D amongst newborns in the city of Poitiers, and with adults in the UK [23,24]. However, no link was found between NO₂ exposure and vitamin D deficiency in Adults in Tanzania [25]. As these studies are conducted across different urban forms and environmental conditions, it is likely that there are local factors which modify the relationship between air pollutants and vitamin D levels.

Furthermore, vitamin D deficiency has been described as a causative factor for poor health and premature death amongst disadvantaged groups [26]. Several studies exploring the urban determinants of vitamin D deficiency have demonstrated a link with the social and built environment, using large-scale UK Biobank data [12,17,24]. The complex interplay between the social and built environment, and heterogeneity in the makeup of local neighbourhoods, call for investigations that assess spatial relationships at hyperlocal level. This is even more important in ethnically and socially diverse cities such as London. Furthermore, given that compliance with dietary supplementation of vitamin D is poor amongst vulnerable communities [27], localised studies can potentially aid the identification of modifiable environmental factors to develop targeted public health interventions.

The aim of this research was to identify geospatial predictors of vitamin D status at Middle Super Output Area (MSOA) level across London.

2. Methods

A cross-sectional study was conducted drawing upon data from the UK Biobank and UK Data Service to predict features of the built environment that may contribute to vitamin D deficiency across London. The UK Biobank consented participants and obtained ethical

approval from the Human Research Authority, North West - Haydock Research Ethics Committee (IRAS ID 299116 – 29/06/2021). Our study used fully anonymised data from the UK Biobank for which ethical approval was obtained from the University of Westminster Research Ethics Committee (ETH2021-0440- 10/11/2020).

2.1. Study population

This study used data from the UK Biobank, a population-based study that collected data from 500,000 adults aged 37 to 73 years between 2006 and 2010 across the UK. Participants provided consent and completed information collection through touch-screen questionnaires, anthropometric measurements, and biological sampling. The study protocol and population characteristics are available on the UK Biobank website (www.ukbiobank.ac.uk).

London borough shapefile data were imported from the UK Data Service and coordinates from the Biobank data for participants within the London area were extracted and joined to 2011 Middle Layer Super Output Area (MSOA) boundaries using a spatial join. Cases with missing MSOA codes were removed. This procedure led to a final sample of 43235 participants (Fig. 1). Fig. 2 below shows the spatial distribution of data points for the London Biobank subset.

2.2. Vitamin D measurement

The measurement of serum 25-hydroxyvitamin D (25OHD) levels, a proxy indicator of Vitamin D status, was performed using chemiluminescence technology on a DiaSorin Ltd. LIAISON XL instrument. To ensure the accuracy and precision of the results, internal and external quality control procedures were implemented by the UK Biobank. The measurement precision was determined by assessing the coefficients of variation (CV) of the 25OHD measurements, which were found to be between 5.0 and 6.1%. These results indicate that the measurements were reliable and met acceptable standards. Vitamin D deficiency was defined as the levels of serum 25OHD lower than 25 nmol/L, and a level of 25–50 nmol/L was considered insufficient [2]. Further details about serum biomarker measurements and assay performances have been described in the online UK Biobank Showcase.

2.3. Particulate matter assessment

Air pollution data were obtained from the Small Area Health Statistics Unit and the BioShaRE-EU Environmental Determinants of

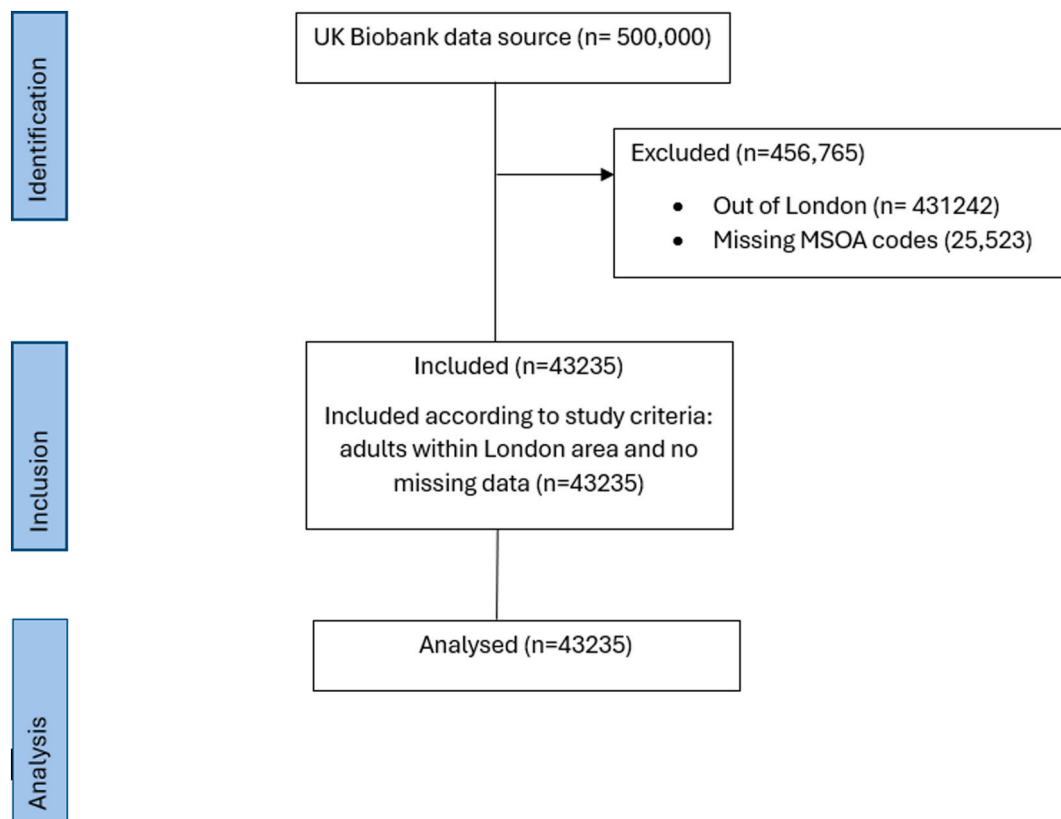


Fig. 1. STROBE flowchart of identification, inclusion and analysis of participants.

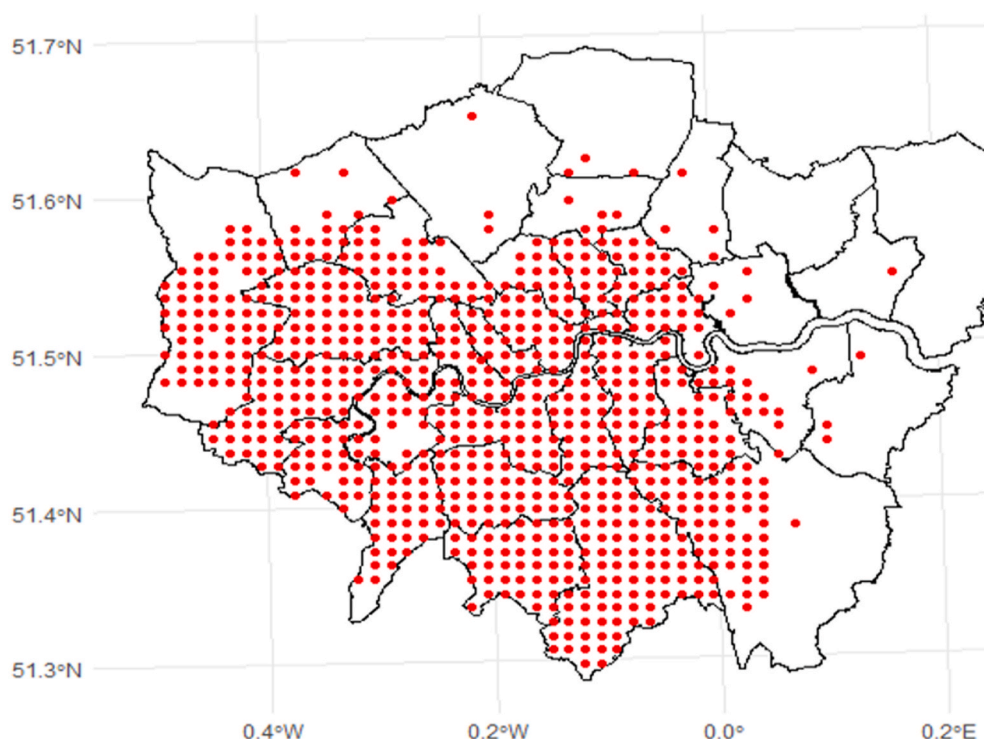


Fig. 2. Spatial distribution of data for the London Biobank subset.

Health Project for the UK Biobank study. Annual concentrations of particulate matter with diameters up to $10\ \mu\text{m}$ (PM_{10} and $\text{PM}_{2.5}$), nitrogen dioxide (NO_2), and nitrogen oxides (NO_x) were modelled for the year 2010 using a Land Use Regression (LUR) model developed as a part of the European Study of Cohorts for Air Pollution Effects.

The LUR model was applied to determine the spatial variations of annual average air pollution concentrations using geographic predictor variables obtained from a geographical information system. These variables included traffic, land use, topography, and population density [28]. The model performance was evaluated using leave-one-out cross-validation, in which each site was left out sequentially while the included variables of the models were left unchanged. The results of this evaluation indicated a good model performance for $\text{PM}_{2.5}$, PM_{10} , and NO_2 , with cross-validation R^2 values of 77%, 88%, and 87% respectively [29].

2.4. Greenspace exposure and built environment assessment

The proportion of land classified as greenspace or domestic garden, relative to all land use types, was modelled using data from the 2005 Generalized Land Use Database for England (GLUD) for 2001 Census Output Areas. The lowest geographical scale for UK administrative boundary data, lower layer super output areas (LSOAs) were used for data extraction. These geographical units were defined as areas with populations between 1000 and 3000 people, and between 400 and 1200 households. For each residential address included in the baseline survey, a circular distance buffer of 1000 m was applied, and the proportion of public greenspace (public greenspace %) and domestic garden (domestic garden%) were included in the analysis.

The GLUD classified greenspace as green land cover designated for public spaces such as parks, communal gardens, and cemeteries and agricultural land. Domestic gardens were defined as parcels of land annexed to residential buildings [30].

The proximity of residential addresses to major roads was determined by indicating whether the address was located within 50 m of a class 1 or 2 type road and/or within 100 m of a class 0 road based on the central road network. The central road network was obtained from Eurostreets version 3.1 digital road network (scale 1:10000), derived from the TeleAtlas MultiNet TM dataset for the year 2008. All roads of class 0, 1 and 2 (motorways, main roads of major importance and other main roads) were classified as major roads. Additionally, based on local knowledge, classes 3 and 4 (secondary roads and local connecting roads) were also classified as major roads.

The traffic intensity on the nearest major road was also considered, it was defined as the average total number of motor vehicles per 24 h on the nearest major road based on a local road network. The traffic count data was obtained from the Road Traffic Statistics Branch at the Department for Transport and was attached to the local road network. For unmonitored links, traffic data were estimated based on the surrounding monitored links. The local road network was obtained from the Ordnance Survey Meridian 2 road network 2009 (scale 1:50000, 1 m accuracy). A major road on the local road network was defined as a road with traffic intensity greater than 5000 motor vehicles per 24 h.

The study also considered the proportion of the home location buffer classified as "Water" within a 1000-m buffer radius, relative to all land-use types. Additionally, the Euclidean distance from each participant's residence location to the coast was also measured in kilometres (km) in the analysis.

2.5. Covariates

To control for potential confounding factors, we included several covariates in our analysis. Gender was coded as binary (women and men), and ethnicity was coded as a six-factor variable (White; Mixed; Asian; Black; Chinese; and Other). Season of blood collection was also considered (spring: March–May; summer: June–August; autumn: September–November; and winter: December–February). Age was derived from date of birth and date of attending assessment centre and refers to the age of the participant on the day they attended an assessment centre, truncated to the whole year part. We also controlled for the type of accommodation lived in, the number of vehicles in the household, oily fish intake and time spent outdoors in summer.

2.6. Statistical analysis

Spatial dependence in model residuals was first assessed at the MSOA level. A global ordinary least squares (OLS) model was estimated using MSOA-aggregated vitamin D means. Moran's I test of OLS residuals indicated significant positive spatial autocorrelation ($I = 0.25$, $p < .001$), suggesting clustering of unexplained spatial variation.

To determine whether this clustering reflected spatial dependence or structural non-stationarity, spatial lag and spatial error models were estimated. Both models demonstrated improved fit relative to the global OLS specification (lower AIC values), and the spatial autoregressive parameters (ρ and λ) were statistically significant, confirming the presence of spatial structure. However, these global spatial models assume spatial stationarity of regression coefficients across the study area. Given the study objective of examining whether associations between vitamin D and environmental predictors vary geographically across London, geographically weighted regression (GWR) was employed to allow regression coefficients to vary spatially. GWR therefore complements global spatial models by explicitly modelling spatial non-stationarity rather than solely correcting for spatial dependence. Local R^2 values and spatially varying coefficients were examined to characterise heterogeneity in associations across MSOAs.

The GWR model specified serum vitamin D level as the dependent variable and included sex, assessment centre month, deprivation index, accommodation type, vehicle ownership, time spent outdoors, dietary intake, ethnicity, BMI, age, air pollution exposures, road network variables, and greenspace indicators as predictors. Adaptive bandwidth selection was performed using the `gwr.sel` function to identify the optimal neighbourhood size. Both fixed and adaptive bandwidth models were explored, and model diagnostics, including local R^2 values, were examined. Results were visualised by mapping local R^2 values and spatially varying coefficients. Statistical significance was defined as $p < .05$.

To address potential temporal misalignment between vitamin D assessment (2006–2010) and environmental exposure datasets, sensitivity analyses incorporating assessment year were conducted. Models including year as a linear covariate and as a flexible spline term yielded negligible changes in adjusted R^2 (0.177–0.178) and did not materially alter effect estimates. Restricting analyses to

Table 1
Descriptive statistics for continuous predictor variables.

Variable	Mean	SD	Minimum	Maximum
Age	56	8	38	72
BMI	26.8	4.8	14.1	68.1
TDI	0.8	3.2	−6.1	9.4
Time at Current Address (Years)	17	11	1	70
Time Spent Outdoors in Summer (Hours)	3.6	2.3	0	16
NO	55	18	20	266
NO ₂	34.2	7.3	12.9	108.5
PM2.5	10.5	1.1	8.2	20.1
PM2.5 Absorption	1.49	0.28	0.83	4.57
PM2.5–10	6.8	0.8	5.6	11.2
PM10	17.3	1.6	11.8	30.7
Traffic Intensity – Nearest Road	2000	6100	500	117,000
Traffic Intensity – Major Road	24,000	18,000	5000	174,000
Traffic Load – Major Road	660,000	1,510,000	0	9,970,000
Road Length – Major Road (100m)	41	91	0	1200
Greenspace % (300m)	19.0	13.5	0.2	95.0
Greenspace % (1000m)	25.2	11.5	4.4	89.1
Domestic Garden % (300m)	33.7	14.3	0.1	75.6
Domestic Garden % (1000m)	28.9	10.8	0.5	66.1
Natural Environment % (300m)	9.3	13.8	0.0	100.0
Natural Environment % (1000m)	17.0	13.9	0.0	98.8
Water % (300m)	0.6	2.1	0.0	34.1
Water % (1000m)	1.0	1.6	0.0	14.5
Distance to Coast	41	8	19	62
C-Reactive Protein	2.4	4.1	0.1	77.6

participants assessed in 2009–2010 produced comparable or slightly higher explanatory power (adjusted $R^2 = 0.183$), with consistent directional associations. These findings suggest that temporal misalignment did not substantially bias results.

Prior to model interpretation, multicollinearity among predictors was assessed using Generalized Variance Inflation Factors (GVIFs) derived from the fully adjusted multiple linear regression model. Because the model included multi-level categorical variables (e.g., ethnicity, accommodation type, dietary intake), GVIF values were adjusted for degrees of freedom to ensure comparability with standard VIF thresholds. Adjusted GVIF values greater than 5 were considered indicative of potentially problematic multicollinearity. Although moderate correlations were observed among certain air pollution indicators (e.g., NO_2 , NO_x , $\text{PM}_{2.5}$), all adjusted GVIF values remained below conventional thresholds, indicating that multicollinearity was unlikely to meaningfully distort coefficient estimates.

All analyses were conducted in R version 4.1.2 (R Core Team, 2021) using the *sf*, *dplyr*, *ggplot2*, *mgcv*, *stargazer*, *spdep*, *rgdal*, *GWmodel*, and *spgwr* packages.

3. Results

The descriptive analysis of vitamin D levels across London revealed an average (mean) value of 46.44 (20.69) nmol/L, indicating that mean vitamin D levels in London fall in the insufficient category and highlight a moderate level of variability in vitamin D status among the studied population. Additionally, according to NHS classification, 6803 (15.73%) participants are considered vitamin D

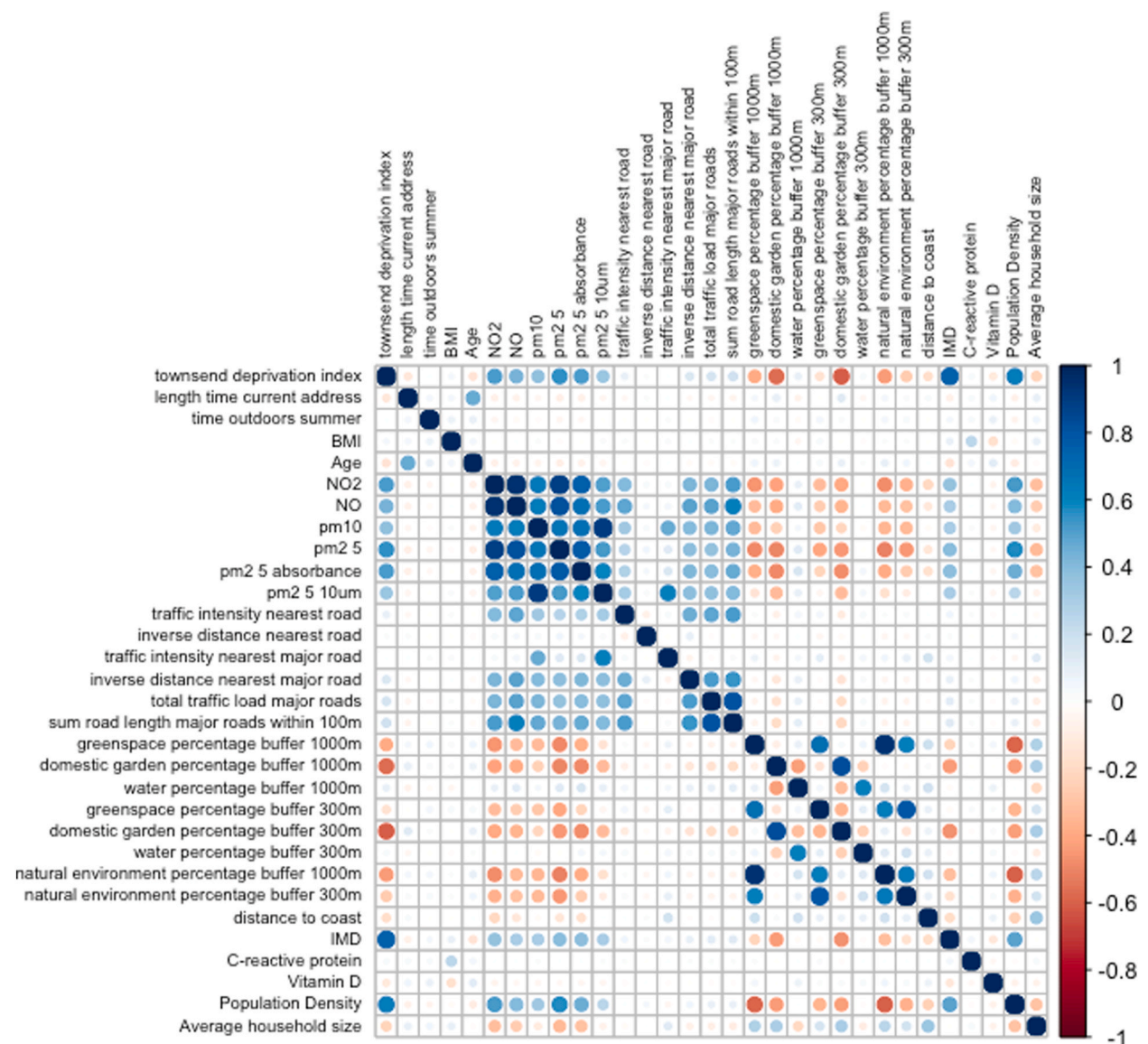


Fig. 3. Correlation coefficients of variables in the MLR model.

deficient, 18989 (43.92%) vitamin D insufficient, 13357 (30.89%) vitamin D adequate and only 4086 (9.45%) would be considered optimal Vitamin D levels. Table 1 below highlights the descriptive statistics for continuous variables that are predictors of Vitamin D and Fig. 3 presents the correlation matrix of the variables.

The results indicated that the model was a significant predictor of Vitamin D levels, $F(33, 43202) = 223.02, p < .001$ (Table 2). The model explained 14% of the variance (adjusted R-squared = 0.14). The significant predictors of vitamin D status are BMI (Body Mass Index), Index of Multiple Deprivation, Nitrogen Dioxide, PM_{10} (Particulate Matter 10 μm), $PM_{2.5}$ (Particulate Matter 2.5 μm), $PM_{2.5}$ Absorbance, Traffic Intensity on the Nearest Road, Proximity to Major Roads (Inverse Distance), Greenspace Percentage Buffer (300m and 1000m), Natural Environment Percentage Buffer (300m and 1000m), Distance to Coast, Population Density and Average Household Size. The regression coefficients are presented in Table 3.

Furthermore, the Akaike Information Criterion (AIC) for this model stands at 365,832. In summary, model 1 presents a modest explanation of the variance of the dependent variable, and with an AIC of 365,832, it sets a standard for comparing the relative fit of different predictive models for the dataset at hand.

Fig. 4 is a thematic map of local R^2 values of vitamin D across London, reflecting the heterogeneity in the relationship between social and environmental predictors, and vitamin D status. Hot (high vitamin D) and cold spot (low vitamin D) analysis identified highest levels of vitamin D to be distributed across the Richmond area (Fig. 5). Within Croydon, both hot and cold spots are identified, demonstrating the hyperlocal spatial clustering of vitamin D levels. There are significant differences across the hot and cold spots with respect to IMD and greenspace. As predicted, cold spots/lower vitamin D levels were associated with higher IMD (Fig. 6) and reduced access to greenspace (Fig. 7).

4. Discussion

To the best of our knowledge, this GWR model is the first to identify heterogeneity in the relationship between air pollution and vitamin D across London and suggests this may be explained by social and built environment characteristics described at the MSOA level.

Firstly, our model identifies several contributing factors to vitamin D that are concurrent with the literature. A higher BMI is associated with lower levels of vitamin D. Previous studies highlight a causative role for adiposity in vitamin D deficiency [31]. As adipose tissue is one of the storage sites for vitamin D it has been suggested that Vitamin D may be increasingly sequestered into adipose tissue, reducing its bioavailability [14,32]. However, a more plausible explanation is simply volumetric dilution of vitamin D [33]. Furthermore, exposure to high levels of air pollution may exacerbate vitamin D deficiency in people with a high BMI [34,35]. This suggests that individuals with obesity should be considered vulnerable to vitamin D deficiency and prioritised in public health interventions. Strategies such as dietary supplementation, food fortification, and promoting safe sun exposure could be particularly beneficial for this group. Additionally, addressing environmental factors such as air pollution may further help reduce the risk of deficiency in individuals with a higher BMI.

We found several air pollutants to be contextual predictors of vitamin D status, including hyperlocal levels of NO_2 , PM_{10} and $PM_{2.5}$, similar to a previous study with data from the UK Biobank, a meta-analysis of vitamin D levels in Brazil, and the findings of recent literature review providing a global context [24,36]. We also found traffic intensity to be related to vitamin D status, such that high traffic intensity on the nearest road is associated with lower vitamin D levels. Similarly, closer proximity to major roads is also associated with lower vitamin D in our model. High levels of PM_{10} are a significant negative predictor of UVB radiation intensity on the ground, supporting the theory that air pollutants may absorb UVB rays, reducing the availability for vitamin D synthesis [37]. However, Rahman and Elmi found NO_2 levels to be positively associated with UVB intensity [37]. This contrasts with a study conducted in Tehran, which found vitamin D deficiency to be more prevalent in areas that were 1.5x more polluted with NO_2 [38]. Given that we observed heterogeneity between air pollutants and vitamin D status across different MSOAs, behavioral factors and other environmental variables at hyperlocal level may influence these patterns in different urban settings. For example, we found that proximity to natural environments (within 300m and 1000m buffers) is positively associated with vitamin D levels. Though causality cannot be inferred, this association may be linked to increased opportunities to engage in outdoor activities [39] and reduced exposure to air pollutants by residing further away from major roads and high traffic areas. In a similar vein, distance to the coast is also associated with higher vitamin D levels, previously explained by higher solar irradiance [40]. Furthermore, we found higher population density correlates with low vitamin D levels, this pattern may reflect factors such as increased air pollution and limited access to outdoor space. Other potential geospatial modifiers that were not included in this study but may be significant include wind speed and the quality and greenness of outdoor spaces, particularly as PM_{10} flux has been associated with increasing population density and wind pressure [41].

The comparative analysis of predictors between hot and cold spots provides further valuable insights into potential factors that may contribute to spatial clustering of vitamin D levels. We found that people with lower vitamin D levels tend to reside in more socio-

Table 2
Model fit measures.

Model	R	R^2	Adjusted R^2	Overall Model Test			
				F	df1	df2	p
1	0.38	0.15	0.14	223.02	33	43202	<0.001

Table 3
Model coefficients – vitamin D.

Predictor	Estimate	SE	95% CI Lower	95% CI Upper	t	p	Std. Estimate	95% CI Lower	95% CI Upper
Intercept^a	35.48	2.59	30.41	40.55	13.71	<0.001			
Sex									
Male – Female	−0.92	0.19	−1.28	−0.55	−4.90	<0.001	−0.04	−0.06	−0.03
Birth Month	0.06	0.03	0.01	0.11	2.18	0.03	0.01	0.00	0.02
TDI	−0.32	0.05	−0.41	−0.23	−7.03	<0.001	−0.05	−0.06	−0.04
Accommodation Type									
Flat – House	−1.30	0.26	−1.81	−0.79	−4.97	<0.001	−0.06	−0.09	−0.04
Mobile/Temp – House	2.42	4.09	−5.59	10.43	0.59	0.55	0.12	−0.27	0.50
Number of Vehicles									
1 – None	2.06	0.28	1.52	2.60	7.48	<0.001	0.10	0.07	0.13
2 – None	3.69	0.32	3.06	4.32	11.45	<0.001	0.18	0.15	0.21
3 – None	4.07	0.48	3.14	5.01	8.51	<0.001	0.20	0.15	0.24
4+ – None	5.12	0.75	3.65	6.59	6.82	<0.001	0.25	0.18	0.32
Time Outdoors (Summer)	0.91	0.04	0.83	0.99	22.03	<0.001	0.10	0.09	0.11
Oily Fish Intake (per week)									
<1 – Never	1.05	0.35	0.36	1.74	2.97	0.003	0.05	0.02	0.08
Once – Never	2.81	0.35	2.13	3.49	8.08	<0.001	0.14	0.10	0.17
2–4 – Never	5.13	0.38	4.39	5.88	13.53	<0.001	0.25	0.21	0.28
5–6 – Never	7.51	0.91	5.73	9.29	8.27	<0.001	0.36	0.28	0.45
Daily – Never	8.95	1.50	6.00	11.89	5.96	<0.001	0.43	0.29	0.57
BMI	−0.66	0.02	−0.70	−0.62	−33.11	<0.001	−0.15	−0.16	−0.14
Age	0.16	0.01	0.14	0.19	14.12	<0.001	0.07	0.06	0.07
NO	−0.04	0.01	−0.06	−0.01	−3.16	0.002	−0.03	−0.05	−0.01
PM10	0.47	0.17	0.14	0.80	2.80	0.005	0.04	0.01	0.06
PM2.5	0.76	0.22	0.33	1.18	3.50	<0.001	0.04	0.02	0.06
PM2.5–10	−1.02	0.35	−1.70	−0.34	−2.95	0.003	−0.04	−0.06	−0.01
Traffic Intensity (Nearest Major Road)	−0.00	0.00	−0.00	−0.00	−2.06	0.04	−0.01	−0.03	0.00
Close to Major Road									
Yes – No	1.15	0.49	0.20	2.10	2.38	0.02	0.06	0.01	0.10
Greenspace % (1000m)	0.07	0.03	0.01	0.12	2.51	0.01	0.04	0.01	0.07
Domestic Garden % (1000m)	−0.07	0.02	−0.10	−0.04	−4.43	<0.001	−0.04	−0.05	−0.02
Water % (1000m)	0.49	0.07	0.35	0.63	6.91	<0.001	0.04	0.03	0.05
Natural Environment % (1000m)	−0.06	0.02	−0.10	−0.01	−2.64	0.008	−0.04	−0.07	−0.01
Distance to Coast	0.20	0.01	0.17	0.22	16.45	<0.001	0.08	0.07	0.09
Ethnicity									
Mixed – White	−7.14	0.76	−8.64	−5.65	−9.37	<0.001	−0.35	−0.42	−0.27
Asian – White	−18.76	0.40	−19.54	−17.98	−46.94	<0.001	−0.91	−0.94	−0.87
Black – White	−10.77	0.41	−11.58	−9.96	−26.17	<0.001	−0.52	−0.56	−0.48
Chinese – White	−13.05	1.17	−15.34	−10.77	−11.19	<0.001	−0.63	−0.74	−0.52
Other – White	−10.98	0.55	−12.06	−9.90	−19.93	<0.001	−0.53	−0.58	−0.48

economically deprived areas than people with high vitamin D. These findings are similar to those from three studies that have geo-mapped vitamin D levels in relation to demographic factors in Dublin [42–44], and identified vitamin D deficiency to be 2.5x more prevalent in children living in deprived areas of Ireland compared with more affluent areas. In Saudi Arabia, vitamin D deficiency was also found to be more prevalent amongst children and adolescents from low-income households and was linked to household size [45]. Comparably, our GWR model identified a negative association between household size and vitamin D status in adults, possibly indicating lower socio-economic status. Similar links between socioeconomic status and vitamin D have been identified across several different population groups, conducted across France, China, Turkey and Spain [46–49]. However, one study conducted in Spain found contrasting evidence whereby high affluence was linked to lower vitamin D levels amongst pregnant women [50], suggesting that the link between social deprivation and vitamin D may be modulated by other environmental or behavioural factors including dietary intake and sun exposure. Areas with higher deprivation often have limited access to nutritious food, particularly vitamin D fortified food, outdoor spaces, and healthcare resources [26]. Deprived areas in England also have less accessible public green spaces than affluent areas, an inequality described as the ‘greenspace gap’ [51]. Air quality is also disproportionately poorer in socially deprived areas, presenting potential risks of vitamin D deficiency and poor health outcomes amongst people with low socioeconomic backgrounds [52].

Furthermore, our GWR model found a significant link between access to greenspace and vitamin D status. Areas where people have higher vitamin D levels also tend to have more residential greenness. This is concurrent with other studies that used UK Biobank data to assess the link between greenspace and vitamin D levels [17,53]. Similarly, a longitudinal study of 309 women in the UK found that women were more likely to have higher levels of vitamin D living in greener spaces [54]. Living in greener spaces may facilitate more outdoor activities, and thus sun exposure. Green spaces may also be linked with higher levels of vitamin D indirectly by mitigating the effects of air pollution on UVB, as observed in other studies, vegetation can absorb and delay the dispersion of air pollutants [55]. However, one study reports that at least 27% cover is needed to see any potential effects on PM concentrations [41]. They also suggest

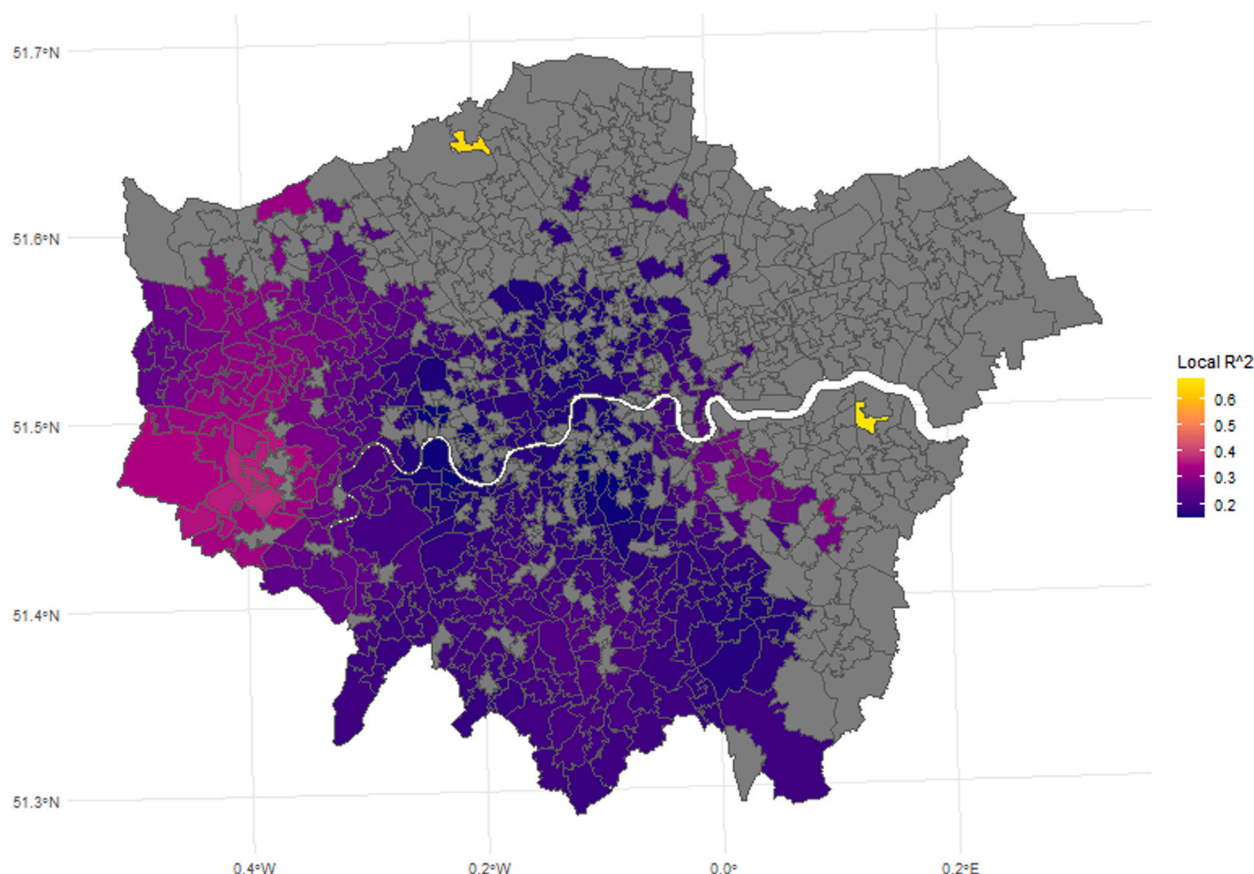


Fig. 4. Thematic Map of Local R^2 values (variation explained) of Vitamin D levels in London.

that to improve air quality, green spaces need to be strategically located near major sources of air pollution, and that this is more important than how much green space there is overall. Other studies suggest that only some types of vegetation increase air pollution. This is because vegetation emits volatile organic compounds that, in already polluted areas, can add to the pollution and affects the dispersion and distribution of polluted air near pollution sources [56,57]. In the UK, the single largest source of urban outdoor air pollution is road transport. Whilst a hedge between vehicles and people may partially block the flow of polluted air from one to the other, an extended tree canopy over a road can partially trap vehicular emissions and thereby increase public exposure [58]. Although we cannot assume causality from our study, it is theoretically plausible that the heterogeneity in vitamin D levels seen across MSOAs may also be related to the location of green spaces, their density and type of vegetation in the micro-environment.

There are a few limitations of our study, firstly, vitamin D status was classified using the ≥ 50 nmol/L threshold recommended in UK clinical guidance for bone health. Alternative cut-offs related to optimal concentrations for non-skeletal outcomes could lead to different estimates of vitamin D adequacy and interpretation of population risk. Secondly, there are more individual behavioural practices that could have been incorporated into the study to strengthen our predictive model for vitamin D levels. This includes the use of sunscreen and further dietary analyses beyond fish intake. Another informative variable to include in future work is occupational exposure to sunshine. Other environmental variables which were not included in our study but have shown links with vitamin D levels include humidity, precipitation and latitude [50,59]. Whilst latitude may not be significantly different across London and therefore less relevant for this study, future work on a larger geographical scale would warrant inclusion of this variable.

In addition to the individual-level limitations discussed above, there are a few methodological considerations related to environmental exposures and modelling that should be acknowledged. Firstly, spatial and temporal mismatches between some environmental exposures and health outcomes. The environmental datasets used represent the best available high-resolution exposures compatible with the UK Biobank framework and are commonly employed in Biobank-based environmental epidemiology. While some misclassification is possible, it is mitigated by the structural stability of London's environment, for example, greenspace distribution and major road networks change relatively slowly, particularly at the MSOA scale. For air pollution, the 2010 LUR estimates capture long-term spatial contrasts rather than short-term fluctuations, which aligns with the cross-sectional study design. It is also important to note that some model coefficients exhibit directions that may seem unexpected or inconsistent. These patterns likely arise from a combination of factors, including spatial heterogeneity, model sensitivity, and potential multicollinearity. Rather than viewing individual estimates in isolation, our results highlight the broader spatial patterns and complexity inherent in urban systems,

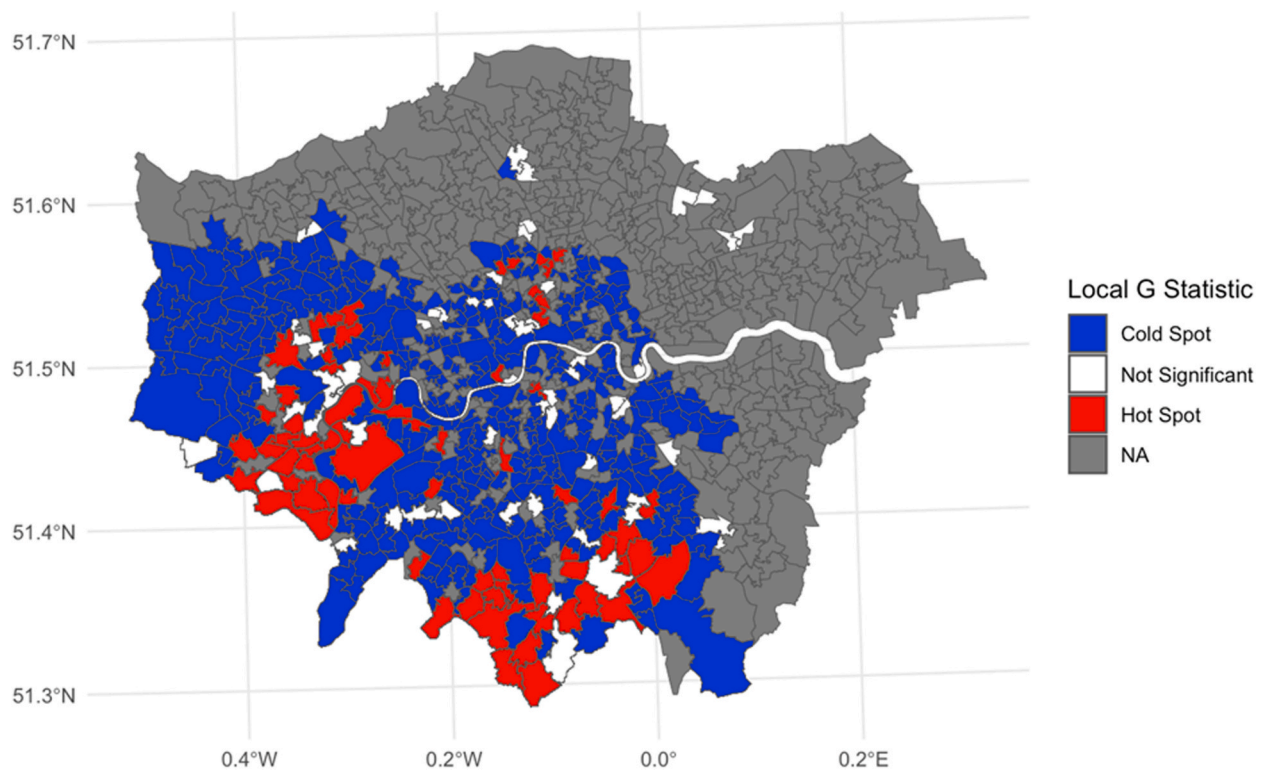


Fig. 5. Hot (high vitamin D) and Cold (low vitamin D) Spots across London.

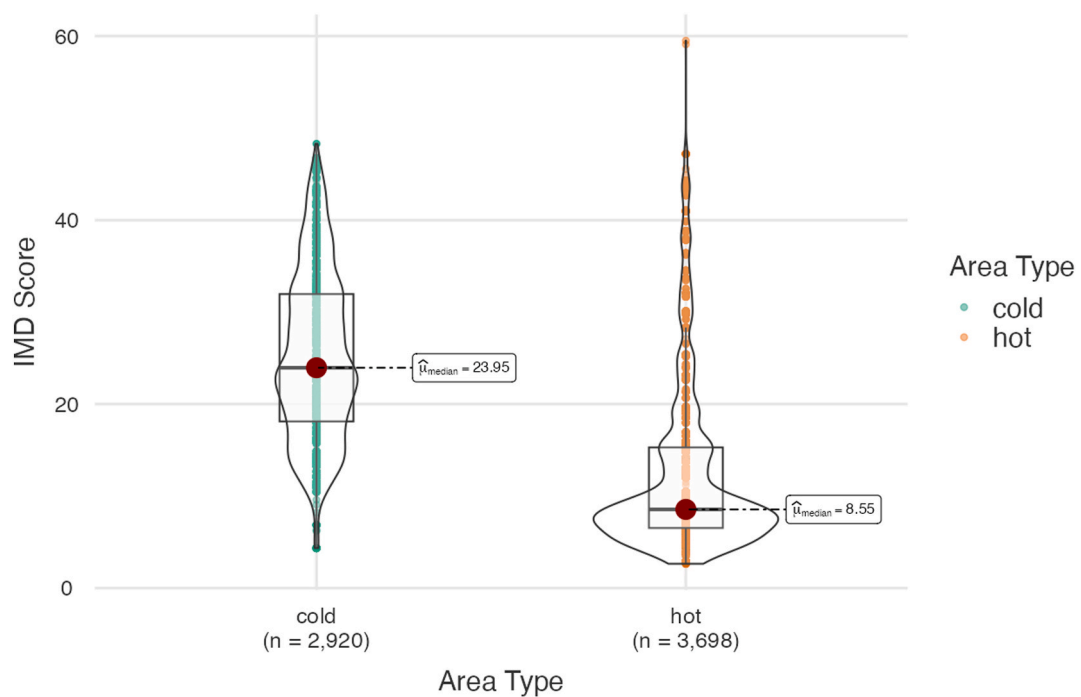


Fig. 6. Index of Multiple Deprivation differences across hot spots. Mann-Whitney U test indicated a significant difference in Index of Multiple Deprivation scores between cold and hot areas ($U = 1,548,264$, $p < .001$).

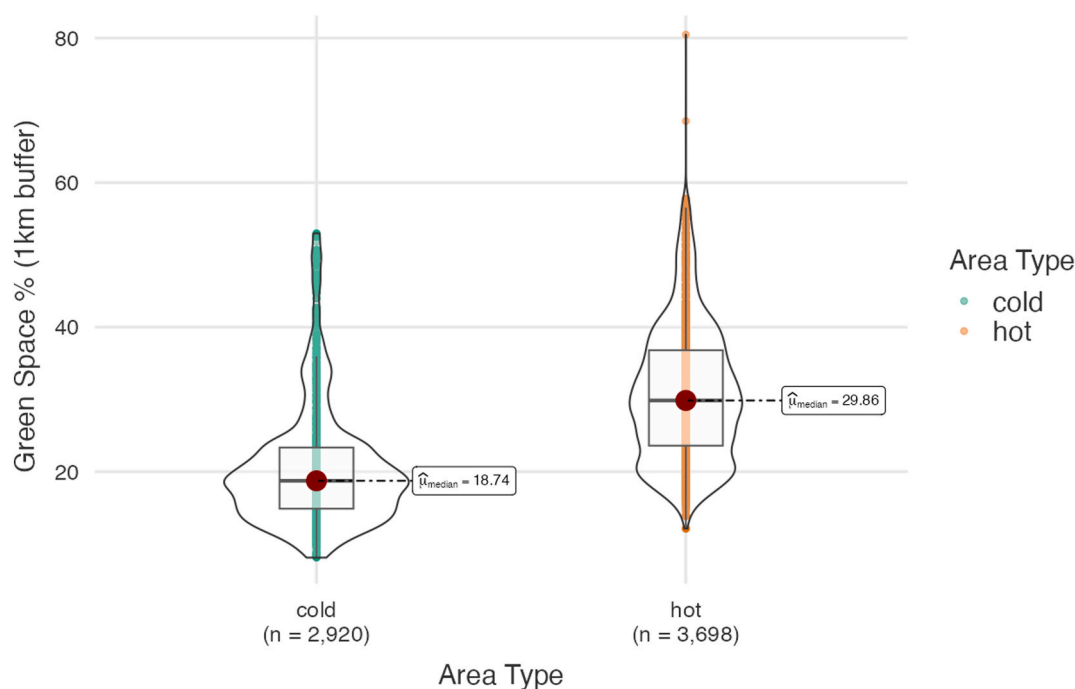


Fig. 7. Greenspace differences across hot spots. A Mann-Whitney U test revealed a significant difference in Greenspace Percentage within a 1000m buffer between cold and hot areas ($U = 1,941,419$, $p < .001$).

emphasizing that heterogeneity itself is an important finding. Finally, potential biases may influence associations. Residual confounding from unobserved lifestyle or socioeconomic variables may remain even after statistical adjustment. Additional sources of bias include limitations of MSOA-level aggregation, where the influence of microenvironmental factors, for example street-level pollution, may not be fully captured. Recognizing these limitations provides context for our findings and highlights areas for further research.

In summary, our results identified heterogeneity in the relationship between air pollution and vitamin D status across London, predicted by several modifiable factors of the hyperlocal local built environment including greenspace and road network variables as well as dietary intake, time spent outdoors and social deprivation (IMD). We acknowledge the modest explanatory power of our GWR model and the heterogeneity in local R^2 values demonstrates that this varies substantially across London, reinforcing the complex interplay of social and built environmental factors influencing vitamin D levels. These modest explanations of variance in vitamin D levels, although small, may be meaningful at population level when exposures are widespread and potentially modifiable. Public health interventions should consider these factors, particularly targeting areas with high pollution and low green space to address vitamin D deficiency. Interventions must also be targeted at vulnerable groups, which include ethnic minorities, people with obesity, and those residing in socially deprived areas. Ethnic minorities in the UK are at enhanced risk due to skin colour but also because they are more likely to reside in socially and greenspace deprived, highly polluted areas [51,52]. Targeted strategies for improving vitamin D levels in such vulnerable populations may include food fortification. An integrated predictive model of serum vitamin D concentration in response to UVB exposure and graded dietary vitamin D intakes provides evidence that food fortification strategies should be encouraged to reduce the disproportionately high prevalence of deficiency in BAME groups [60]. Ultimately, meaningful reductions in vitamin D deficiency also depend on hyperlocal interventions that are tailored to specific communities and explicitly account for social and built environmental factors including housing conditions, access to culturally appropriate foods, availability of fortified products, outdoor space and air quality. Such factors can shape sun exposure, dietary behaviours and our capacity to synthesise and maintain adequate vitamin D levels across the life course.

CRedit authorship contribution statement

Moonisah Usman: Writing – review & editing, Writing – original draft, Visualization, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization. **Evren Raman:** Writing – review & editing, Validation, Software, Resources, Methodology, Investigation, Formal analysis, Data curation. **Maria Woloshynowych:** Writing – review & editing, Writing – original draft, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Mehrdad Borna:** Writing – review & editing, Investigation, Funding acquisition, Data curation, Conceptualization. **Rosa Schiano-Phan:** Writing – review & editing, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization. **Emanuela V. Volpi:** Writing – review & editing, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization.

Ethical statement

This research has been conducted using data from UK Biobank, a major biomedical database. The UK Biobank study received ethical approval from the UK Northwest Multicenter Research Ethics Committee. Our study was approved by the University of Westminster Ethics Committee (ETH2021-0440).

Data availability statement

The data used in this study were obtained from the UK Biobank under an approved research application (Project ID 68906). Due to the terms and conditions governing access to UK Biobank data, the participant-level data cannot be made publicly available by the authors. Researchers may apply for access to the data directly through the UK Biobank Access Management System (<https://www.ukbiobank.ac.uk/enable-your-research/apply-for-access>) subject to approval by UK Biobank. The data underlying this study are available from UK Biobank upon reasonable application and approval.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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