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Ethnicity and place: the geography of diabetes inequalities under a strong welfare state

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Background: The Nordic 'health paradox' designates the seemingly puzzling empirical reality in which, despite the presence of strong welfare policies targeting structural inequalities, distinct health disparities persist in Scandinavian societies. In Norway, previous research has shown that inequalities in diabetes prevalence are particularly salient, notably between ethnic groups. These have often been attributed to lifestyle, socioeconomic factors, or genetics. No previous research has sought to investigate the sociospatial mediation of diabetes inequities. **Methods:** In this article, we examine the social geography of diabetes in Oslo to examine whether the link between ethnicity and diabetes is confounded by place. We use data from the 2002 Oslo Health Study (n = 17 325) to fit logistic regression models, assessing whether contextual factors, such as the concentration of fast food outlets, predict self-reported diabetes outcomes after controlling for relevant individual level covariates. We also test for spatial autocorrelation in the geographical distribution of diabetes. **Results:** The findings suggest that the organisation of urban space and the spatial distribution of health-related resources exert an independent effect on diabetes prevalence, controlling for ethnicity and other covariates. Living on the east side of Oslo increases the odds of suffering from diabetes by almost 60%, whilst living in a neighbourhood characterized by a relative concentration of fast food and relative absence of healthy food shops and physical exercise facilities increases the odds by 30%. **Conclusion:** Spatial context and toxic environments contribute to diabetes inequalities in Oslo, Norway. Future research and policy-making should take the geography of health disparities into account.

Introduction

In his account of the *Killing Fields of Inequality*, Göran Therborn identifies a contemporary public health conundrum: why have Scandinavian welfare models failed on 'vital inequality'? Defining the latter as 'socially constructed unequal life-chances of human organisms' (p. 49), the Swedish sociologist suggests that 'the question of why the relatively egalitarian Nordic welfare states have had so little success in reducing vital inequality is more a socio-political than a medical question' (p. 133).[1] By virtue of its commitment to curb the detrimental effects of unbridled commodification, the Scandinavian welfare model has been associated with greater overall equality[2,3]—also when it comes to health. However, as Therborn spotlights, empirical evidence indicates the persistence of significant health disparities even within countries with strong social democratic welfare states, such as Norway.[4–7] Research has shown that in Norway's capital, the gap in life expectancy between the richer districts located on the west side and the poorer districts located on the east side of the city is as high as 10.7 years.[8,9] Thus overall health inequalities in Norway's capital echo those of European nations with starkly contrasting welfare systems and political economies.[10,11]

Below, we examine the case of type 2 diabetes inequalities in Oslo. Not unlike other capital cities in advanced industrial nations, Oslo's social history is marked by a distinct pattern of urban polarisation. At the tail end of the 19th century, Oslo established itself as a dynamic sociospatial microcosm characterised by a socioeconomically dualized city landscape. The advent of industrial capitalism served not only to forge a steeply stratified class structure within a geographically bounded context but also to stamp the mark of the former onto the latter, with the economic elites firmly ensconced in the western parts of the capital—at a safe distance from the eastern working class districts. Although post-war deindustrialisation has certainly resulted in various urban reconfigurations, Oslo, with a population barely exceeding half a million, remains divided between an increasingly segregated 'West', inhabited by a wealthy and ethnically homogeneous community, and a historically disadvantaged and much more densely populated 'East', popularly associated with immigration and social stigma. (See Supplementary Appendix figure SA1). For instance, at the turn of the century, over half of the inhabitants of the western-most districts had studied at university and fewer than 10% were ethnic minorities. In contrast, in the eastern-most areas, <20% had profited from higher education and over 25% were ethnically 'non-Norwegian'.[12,13] Against this sociohistorical backdrop, we take our cues from the 'neighbourhood effects' literature[14–23] by examining how diabetes inequalities may be spatially mediated, i.e. how the organisation of urban space might play an important role in the (re)making of health disparities.

Methods

Our research draws on data from three population-based, cross-sectional studies conducted between 2000 and 2002 in Oslo under the aegis of the Norwegian Institute of Public Health (n = 17 325, overall participation rate = 45.4%, informed consent obtained from all participants). In the first study, 46% of all Oslo residents born in 1924, 1925, 1940, 1941, 1955, 1960 and 1970 took part in the survey (n = 18 770).[24] The second study targeted residents born in Sri Lanka, Pakistan, Iran, Vietnam or Turkey between 1942 and 1971, and 39.7% (n = 3019) participated.[25] The final study included 2960 (48%) of all residents born between 1933 and 1969 in two socioeconomically disadvantaged and ethnically heterogeneous districts.[26] Henceforth, the pooled version of these data will be referred to as the Oslo Health Study. Our outcome variable is self-reported diabetes

Table 1 Descriptive statistics of individual and neighbourhood characteristics

		n
Ethnicity	Sri Lanka	1186 (6.8%)
	Iran	748 (4.3%)
	Norway	13 213 (76.3%)
	Pakistan	894 (5.2%)
	Turkey	581 (3.4%)
	Vietnam	703 (4.1%)
Age	31–40 years	6389 (36.9%)
	41–50 years	5956 (34.4%)
	51–60 years	4218 (24.3%)
	61+ years	762 (4.4%)
District	Outer west	3371 (19.5%)
	Inner west	1687 (9.7%)
	Inner east	2272 (13.1%)
	Outer east	9995 (57.7%)
Sex	Male	8144 (47%)
	Female	9184 (53%)
Education	≤9 years	2594 (15.0%)
	10–15 years	8864 (51.2%)
	16+ years	5867 (33.9%)
Paid employment	Yes (full-time)	11 883 (68.6%)
	Part-time	2157 (12.5%)
	No	3285 (19.0%)
Economic insecurity	Yes	3249 (18.8%)
	No	14 076 (81.2%)
Organization memberships		Mean = 1.02 (SD = 1.91)
BMI > 25	Yes	9137 (52.7%)
	No	8188 (47.3%)
Physical exercise	Active	12 286 (70.9%)
	Inactive	5039 (29.1%)
Mental health problems	Yes	3654 (21.1%)
	No	13 671 (78.9%)
Perceived unavailability of food	Yes	3488 (20.1%)
	No	13 837 (79.9%)
Neighbourhood	Fast food shops	Mean = 26.38 (SD = 38.94)
	Healthy food shops	Mean = 3.81 (SD = 3.91)
	Physical exercise facilities	Mean = 8.23 (SD = 5.78)

status. Summary statistics of participant and neighbourhood characteristics are displayed in table 1, and complete variable definitions and further information about the data are provided in the Supplementary Appendix. Multiple imputation techniques were employed to account for missing values.[27] The nested data structure was preserved using multilevel multiple imputation by chained equations.[28] In selecting the set of variables to be included in the imputation process, 30% was chosen as a cut-off point for the acceptable proportion of missingness (although most imputed variables had <10% missing values), and post-imputation diagnostics revealed smooth convergence and reliable outcomes that only marginally altered the analytic findings. In addition, we utilise geographical data generated by and purchased from Statistics Norway on the spatial distribution of health-related economic activities in Oslo at the time of the Oslo Health Study, i.e. the geographical location of firm establishments engaged in the supply of selected types of food or physical exercise facilities around year 2001. This dataset was put together following a request made by the authors. By examining the locations of food stores and physical exercise facilities, our aim was to relate the social logics of health inequality to the spatial logics of urban life. We thus create a binary index of 'toxic environment', defined by the relative combined presence and/or absence of fast food chains, healthy food shops, and physical exercise facilities using principal components analysis. We define healthy outlets as food stores explicitly designed to sell high quality fruit and (green) vegetables. Physical exercise facilities are not only gyms or sports arenas, but also places associated with physical exercise or a broader promotion of good health (e.g. physiotherapies with gym facilities or yoga studios). We also categorise neighbourhoods according to their level of socioeconomic deprivation using a pre-existing composite index.[29] How our neighbourhood variables were constructed is described in detail in the Supplementary Appendix.

To confirm the spatial clustering of diabetes prevalence across districts in Oslo, we employ the Moran's I statistic. This utilises areal data to test for spatial autocorrelation, defined as the presence of non-zero covariance between values on a random variable for neighbouring locations. The Moran's I statistic measures the presence of correlation between an outcome variable (diabetes prevalence, calculated from the raw data) and its spatially lagged values. Consequently, a positive value for the global measure indicates a positive spatial autocorrelation, i.e. the clustering of similar values on the random variable amongst neighbouring observations.[30,31] Below, we use Moran's I to identify patterns of geographical clustering. We then use our data to fit regular and multilevel logistic regression models that estimate the relative importance of individual and neighbourhood-level properties in the distribution of diabetes in Oslo.

Results

Calculations reveal that out of the 17 325 individuals who completed the Oslo Health Study questionnaire, 741 reported diabetes (4.3%). However, the distribution of cases seems to be highly unequal across spatial contexts (see Supplementary Appendix figure SA1): diabetes seems to cluster in the eastern regions of Oslo, with the eastern districts counting 659 total cases (calculated prevalence = 5.4%), as opposed to only 82 cases for the western districts (calculated prevalence = 1.6%). The spatial autocorrelation test, which simply assesses the degree of clustering of diabetes prevalence across neighbourhoods, reveals that the test statistic is highly significant: Moran's I is equal to 0.58 (P value = 4.34×10^{-7}), suggesting that the probability of suffering from diabetes is not randomly distributed across Oslo's capital. We therefore proceed with regression models designed to capture this spatial variation.

Table 2 shows the results for a standard logistic regression model (Model 1). The first thing to note is that, even when adjusted for a wide range of covariates at the individual level, *the spatial component of the analysis remains highly significant*, with a shift from the western to the eastern part of Oslo being associated with an almost 60% increase in the odds of diabetes prevalence [exp(0.45) = 1.58]. This does not diminish the fact that *ethnicity also remains a strong predictor*, where being of non-Western origins increases the odds by a factor of almost 5 [exp(1.54) = 4.68]. In addition, diabetes susceptibility seems to increase with age (as expected), with those in the 61+ years of age category being almost four times as likely to suffer from the condition compared with those aged below 40 [exp(1.33) = 3.78], whilst men are less susceptible than women [exp(−0.27) = 0.76].[32] Furthermore, those with medium to low levels of education are more at risk than those with the highest levels of education, with odds ratios equal to 1.68 [exp(0.52)] and 1.78 [exp(0.57)], respectively. Interestingly, an alternative model (not included here for lack of space), which included a variable stratified by specific types of occupations (modelled on the Goldthorpe class schema), showed that socially relegated occupations, associated with routine or manual labour, do *not* seem to have a statistically significant impact. However, Model 1 shows that *complete absence or exclusion* from the labour market increases the odds of suffering from diabetes by 80% [exp(0.59) = 1.80]. This may be partly due to pensioners being included in the count in the Oslo Health Study, thus making age a key confounder. However, diagnostics checks reveal that the bias induced by this overlap between active and inactive populations in the data is minimal. As expected, high BMI is a highly significant predictor, whilst, unexpectedly, low

Table 2 Logistic regression model for diabetes with covariates (Model 1)

Intercept	−6.002*** (0.182)
Part of town (ref: West)	0.455***(0.129)
Non-Western (ref: Norwegian)	1.545*** (0.095)
Age 41–50 years (ref: 31–40 years)	0.633*** (0.108)
Age 51–60 years	1.378*** (0.109)
Age 61+ years	1.332*** (0.205)
Sex (ref: female)	−0.277*** (0.084)
Education ≤ 9 years (ref: 16+ years)	0.520*** (0.137)
Education 10–15 years	0.578*** (0.119)
Paid employment no (ref: Yes)	0.592*** (0.095)
Paid employment part-time	0.189 (0.135)
Economic insecurity (ref: No)	0.099 (0.095)
Organisation memberships	−0.0006 (0.014)
BMI > 25 (ref: Normal)	0.855*** (0.094)
Physically inactive (ref: Active)	0.081 (0.084)
Mental health problems (ref: none)	−0.041 (0.082)
Perceived unavailability of food (ref: No)	0.300*** (0.092)
Observations	17,325
Log likelihood	−2563.357
AIC	5158.714

Note: Log-odds reported. SEs in parentheses.
*$P < 0.1$; **$P < 0.05$; ***$P < 0.01$.

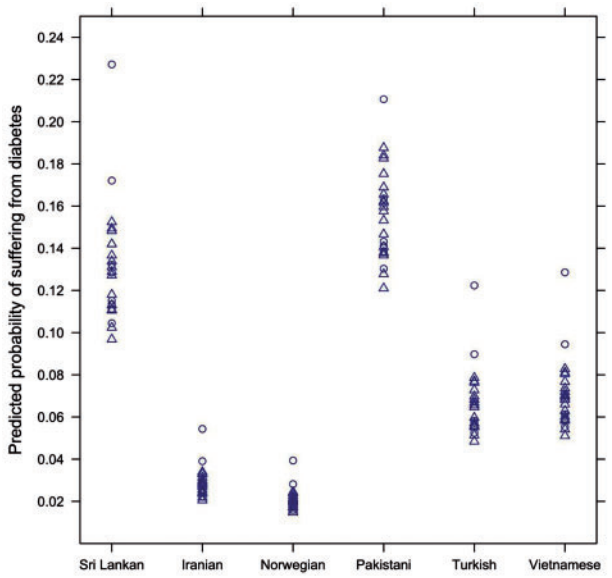

Figure 1 Spatially mediated variation in predicted probabilities of suffering from diabetes for the six largest ethnic groups in Oslo. Each point represents a separate neighbourhood. Circles designate eastern neighbourhoods, whilst triangles designate western neighbourhoods

levels of physical exercise are insignificant. This is most probably due to the fact that levels of physical exercise on Oslo's east side and amongst ethnic minorities are low in general, leading to insufficient variance within this variable to make it a statistically meaningful predictor of diabetes.[32] Moreover, neither economic insecurity, social capital, nor mental health problems are significantly associated with diabetes. Of particular interest is the final predictor, perceived unavailability of food, which encapsulates the interface between individual and neighbourhood-level variance in the form of the lived experience of urban space. The model suggests that the subjective perception of neighbourhood characteristics is a significant factor that impacts on health-related behaviours: viewing the residential context as deprived of adequate food provisions increases the odds of suffering from diabetes by 35% [exp(0.30) = 1.35].

Model 1 relies on a rather crude measure of spatial variation. The rationale for using multilevel modelling is that it facilitates the exploration of hierarchical data structures, where individuals are nested within neighbourhoods. Whilst violation of the assumption of error independence tends to lead the conventional regression model to yield incorrect standard errors and biased parameter estimates, multilevel models integrate the clustering effects of ecological contexts. As such, multilevel analysis furnishes researchers with a powerful means of jointly exploring micro- and macro-level phenomena.

An 'empty' multilevel model (not displayed here) in which diabetes is allowed to vary randomly across neighbourhoods indicates that the log-odds of suffering from diabetes in the 'average' neighbourhood are −3.41 (OR = 0.033). The intercept for neighbourhood j is $-3.41 + U_{0j}$, where the variance of U_{0j} is estimated to be 0.3231. In order to test the null hypothesis that this variance is equal to zero, the log-likelihood ratio statistic was calculated as a means of comparing the multilevel null model to the corresponding single-level model without random effects. The test statistic is equal to 129.85 on 1 degree of freedom, where the critical value for statistical significance at the 99% level of confidence is 10.83. There is thus strong evidence that between-neighbourhood variance is non-zero.

In order to further quantify the importance of residential context in the making of inequalities in diabetes, the null model was used to calculate the intraclass correlation (ICC), also known as the variance partition coefficient. This was equal to 8.9%, suggesting that ∼9% of the residual variation in diabetes prevalence is attributable to neighbourhood-level characteristics. This is a standard result in the existing literature on the contextual determinants of health. In light of various methodological issues linked to the ICC, the median odds ratio, defined as 'the median value of the odds ratio between the area at highest risk and the area at lowest risk when randomly picking out two areas',[33] was also calculated. Its value was equal to 1.72, indicating that on average, when randomly selecting two individuals from different neighbourhoods, the residual variation pertaining to space increases the individual odds of suffering from diabetes by 72%.

To further investigate the degree to which the impact of ethnicity on diabetes is mediated by spatial context, predicted probabilities of diabetes outcome were calculated for each neighbourhood and plotted for each ethnicity. Figure 1 shows that, for an individual of Pakistani origins (to take a high-risk group), the probability of suffering from diabetes ranges from around 12 to over 20% between areas. For ethnic Norwegians, whose prevalence rates are the lowest, probabilities range from 1.5 to around 4%. Moreover, as indicated by the circles, the highest prevalence rates all seem to be located in the eastern neighbourhoods. Conversely, the lowest prevalence rates all seem to be located in the western neighbourhoods, as indicated by the triangles. This once again suggests that the relationship between ethnicity and diabetes is at least partly confounded with neighbourhood context.

Table 3 compares two different models. The first multilevel model (Model 2) regresses diabetes on a set of covariates, all located at the level of the individual. Model 3 introduces two additional predictors, both located at the area level: the first is a dichotomous variable identifying socioeconomically disadvantaged neighbourhoods, whilst the second identifies areas that can be characterised as 'toxic' environments. In this model, practically all the neighbourhood-level variance is explained. The results reveal that living in a toxic environment increases the odds of having diabetes by almost 30% [exp(0.25) = 1.29]. The log-likelihood ratio test was applied to all models, revealing they are all highly significant. We thus conclude that there is strong evidence that the relationship between ethnicity and diabetes is at least partly mediated by ecological factors.

Table 3 Multilevel logistic regression models for diabetes with individual-level predictors only (Model 2), and individual and neighbourhood-level predictors (Model 3)

	Model 2	Model 3
Intercept	–5.733*** (0.165)	–5.912*** (0.174)
Non-Western (ref: Norwegian)	1.626*** (0.096)	1.589*** (0.095)
Age 41–50 years (ref: 31–40 years)	0.619*** (0.108)	0.625*** (0.108)
Age 51–60 years	1.360*** (0.109)	1.367*** (0.109)
Age 61+ years	1.314*** (0.205)	1.329*** (0.205)
Sex (ref: female)	–0.289*** (0.084)	–0.283*** (0.084)
Education $\leq$ 9 years (ref: 16+ years)	0.572*** (0.137)	0.533*** (0.137)
Education 10–15 years	0.613*** (0.119)	0.591*** (0.119)
Paid employment no (ref: Yes)	0.599*** (0.095)	0.599*** (0.095)
Paid employment part-time	0.182 (0.135)	0.189 (0.135)
Economic insecurity (ref: No)	0.075 (0.098)	0.061 (0.098)
Organization memberships	–0.003 (0.014)	–0.002 (0.014)
BMI > 25 (ref: Normal)	0.865*** (0.094)	0.864*** (0.094)
Physically inactive (ref: Active)	0.082 (0.084)	0.081 (0.084)
Mental health problems (ref: none)	–0.029 (0.082)	–0.031
Perceived unavailability of food (ref: No)	0.274*** (0.096)	0.273*** (0.096)
Neighbourhood socioeconomic disadvantage (ref: No)		0.126 (0.113)
Toxic environment (ref: No)		0.254** (0.116)
Observations	17 325	17 325
Log Likelihood	–2567.907	–2563.468
AIC	5167.814	5162.937
BIC	5291.972	5302.615

Note: Log-odds reported. SEs in parentheses.
*$P < 0.1$; **$P < 0.05$; ***$P < 0.01$.

Discussion

Based on the empirical analyses provided in this article, the following may thus be said about inequalities in diabetes in Oslo, Norway. First, the findings reveal a distinct sociospatial pattern: diabetes tends to cluster in the historically disadvantaged eastern regions of the city with high proportions of inhabitants with non-Western ethnic origins. In addition, the logistic regression without random effects showed that, even when adjusting for a range of covariates (such as ethnicity, age or BMI), the residential location of individuals is a strong and highly significant predictor of diabetes. Second, it appears that the well-established association between ethnicity and diabetes is partly mediated by sociospatial context: the multilevel logistic regressions suggested that whilst ethnicity does exert a strong independent effect on diabetes outcome, its overall importance may nevertheless be said to depend on the broader sociospatial situation in which groups and individuals live their everyday lives. Put differently, *the relationship between ethnicity and diabetes is at least partly confounded with environmental setting*. Third, it was revealed that neighbourhood characteristics might play an important role in the making of diabetes inequalities. Insofar as living in a toxic environment *and subjectively perceiving one's food environment as inadequate* increase the probability of suffering from diabetes, it is crucial to take the interaction between individual and group-level attributes into account when investigating health inequalities. Hence, the historically rooted and socially reinforced bisection between 'East' and 'West' is an agent of (re)production of diabetes inequalities in Oslo and must therefore be taken into consideration in future epidemiological research on health disparities under Norwegian capitalism.

The principal policy implication would be that policy-makers move beyond only focusing on conventional individual and disease-specific approaches to health inequalities. Traditional welfare states intervene after market transactions to redistribute income. Social democratic welfare states go further with policies that decommodify labour. This analysis suggests that redistribution and decommodification are not enough to tackle health inequalities: the state must intervene to regulate market activity to combat the construction of toxic environments.

The research suffers from several weaknesses. First of all, sampling issues related to sub-optimal participation rates in the Oslo Health Study may have induced analytic biases. However, evaluations of the data reveal that demographic patterns identified in the study correspond relatively well with other official statistics.[24–26] On the other hand, the employment of cross-sectional secondary data offers ready-made and thus unalterable measurements of quintessential variables, including the outcome variable. The very measurement of diabetes, albeit common in observational studies, is methodologically flawed, as it ignores undiagnosed cases, which, in the Norwegian context, are known to constitute a numerically significant group.[34] Nevertheless, the estimated magnitude of *inequalities* between various groups is likely to be accurate.[24,35,36] That being said, the most reliable clinical measure of diabetes is an oral glucose tolerance test with a 2-h glucose concentration $\geq$11.1 mmol/l.

Moreover, when dealing with ethno-migrational groups, the importance of taking into account the heterogeneity introduced by singular experiences across socially fragmented, discontinuous, and potentially traumatic biographical trajectories cannot be underestimated, and all the more so in research on health and illness. This highlights the failure of this article to explain the strong independent impact of ethnicity on diabetes. Merely assuming that either purely genetic or lifestyle factors are at work is too facile a move in the absence of actual genetic data or detailed knowledge of lifestyle and consumption patterns, the transcriptional embedding of lived experiences (e.g. via epigenetic processes), or cumulative impacts of life course adversity (such as childhood abuse, discrimination or stigma).

A central methodological concern in spatial analysis is the issue of how spatial estimates are influenced by the areal units that are employed by the researcher. This relates to how areal units most readily available to researchers, such as the administrative boundaries employed in this article, are arbitrary in nature and do not necessarily reflect the actual experience of empirical reality.[37] We began by testing for spatial autocorrelation to confirm the underlying hypothesis guiding our research. However, due to limited data, a full-scale spatial econometric model could not be developed and the explicitly spatial part of the analysis was confined to an elementary autocorrelation test based on unalterable geographical units.

Furthermore, the concentration of (say) fast food in a given neighbourhood does not *necessarily* entail increased unhealthy consumption behaviour in that spatial context. Similarly, the presence of organic vegetable stores or physical exercise facilities in another area does not always mean that individuals in the vicinity automatically seize those immediate opportunities. It thus remains imperative to jointly examine the complex and interlocking trajectories of individuals *and* the places they inhabit.[38–40] Future research should seek to analyse the interwoven nature of individual and ecological risk factors in the (re)making of health inequalities.

Supplementary data

Supplementary data are available at *EURPUB* online.

Conflicts of interest: None declared.

Key points

- Urban space contributes to diabetes inequalities in Oslo, Norway.

- The relationship between ethnicity and diabetes is at least partly mediated by ecological factors, such as the spatial distribution of health-related resources.
- Policy-makers should seek to combat the creation of toxic environments.

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