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Does neighbourhood deprivation affect the genetic influence on body mass?

Gwilym Owen*, Kelvyn Jones, Richard Harris

School of Geographical Sciences, University of Bristol, UK

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ABSTRACT

Most research into the role of gene-environment interactions in the etiology of obesity has taken environment to mean behaviours such as exercise and diet. While interesting, this is somewhat at odds with research into the social determinants of obesity, in which the focus has shifted away from individuals and behaviours to the types of wider obesogenic environments in which individuals live, which influence and produce these behaviours. This study combines these two strands of research by investigating how the genetic influence on body mass index (BMI), used as a proxy for obesity, changes across different neighbourhood environments measured by levels of deprivation. Genetics are incorporated using a classical twin design with data from Twins UK, a longitudinal study of UK twins running since 1992. A multilevel modelling approach is taken to decompose variation between individuals into genetic, shared environmental, and non-shared environmental components. Neighbourhood deprivation is found to be a statistically significant predictor of BMI after conditioning on individual characteristics, and a heritability of 0.75 is estimated for the entire sample. This heritability estimate is shown, however, to be higher in more deprived neighbourhoods and lower in less deprived ones, and this relationship is statistically significant. While this research cannot say anything directly about the mechanisms behind the relationship, it does highlight how the relative importance of genetic factors can vary across different social environments, and therefore the value of considering both genetic and social determinants of health simultaneously.

1. Introduction

Obesity is an important public health issue due to its links to chronic diseases such as type 2 diabetes and hypertension (Kopelman, 2007), and association with increased mortality generally (Flegal et al., 2013). The worldwide prevalence of obesity has more than doubled since 1980, with the World Health Organisation (2015) describing the problem as an "escalating global epidemic". In recent years, lack of success with interventions at the individual level in reversing this trend has led to a substantial amount of research aimed at understanding the wider food and built environments in which people live and work, which promote obesity's proximate causes of poor diet and sedentary behaviour (Lake and Townshend, 2006). These environments are often labelled 'obesogenic' (Egger and Swinburn, 1997). While there has been a growing body of literature on how these types of environments affect obesity, one area that has so far been unexplored is how these environments may affect the genetic influence on obesity.

Obesity has been shown to have a genetic influence. Studies exploiting the genetic relatedness of twins have estimated the heritability of body mass index (BMI), defined as the proportion of variation in a trait attributable to variation in genetics, as anywhere between 0.47 and 0.9 depending on the population studied and the method used (Elks et al., 2012). It has become increasingly acknowledged, however, that the heritability of many human traits, such as BMI, is not a constant and is dependent on the social environment (Turkheimer et al., 2003; Tuvblad et al., 2006). In other words, social factors and genetics may interact in producing health outcomes, and as such cannot be assumed to be independent. Despite this, there has been little research into how wider social contexts, such as neighbourhoods, may moderate genetic

* Corresponding author. University of Bristol, School of Geographical Sciences, University Road, Bristol BS81SS, UK.
E-mail address: gwilym.owen@bristol.ac.uk (G. Owen).

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influence on obesity. Previous gene-environment interaction studies have mostly treated the environment as behaviours such as exercise (e.g. Ahmad et al. (2013)) and diet (e.g. Qi et al. (2014)), rather than the wider obesogenic environments which produce these behaviours and which are currently the focus of much social research (Boardman et al., 2013). Notable exceptions include a study by Boardman et al. (2012), which shows that school context moderates heritability of body mass index (BMI), and Rosenquist et al. (2015), who use measured genetic data to show that the relationship between variants of the fat mass and obesity-associated FTO gene and body mass index varies with cohort of birth.

Understanding the relative importance of genetic and social risk factors and how they interact can be important for future health policy. For example, it may be the case that in the most extreme obesogenic environments, the environment 'outweighs' the effect of an individual's genetics, having such a large effect that differences between people due to genetics are small in comparison. This may not be a causal interaction in the biological sense as the change in heritability would be due to an increase in environmental variation rather than changes in the effect of specific genes. If this were the case, it would suggest that more resources should be put into focussing on understanding social environments.

Alternatively, it may be that there are aspects of obesogenic environments that moderate genetic vulnerability to obesity and cause changes in the relationship between specific genes and obesity. If this was the case, policies could be designed that not only reduce the effect of the environment as a whole, but can also reduce the genetic influences on weight gain (Boardman et al., 2012). Another possibility could be that social policies are only effective for certain types of individuals due to their genotype. Whatever the underlying relationship between social environments and genetics, these possibilities highlight the need for research which considers both genetics and the social environment together.

In this research, neighbourhood deprivation is used as the proxy for the obesogenic environment as, according to a systematic review from Giskes et al. (2011), it is the only measure of neighbourhood environment consistently associated with obesogenic dietary intakes, even after controlling for possible confounding individual level variables. The theory is that deprived neighbourhoods may be poorer environments for food and physical activity, though the empirical evidence for the pathways through which this happens is mixed, and may differ across countries (Cummins and Macintyre, 2006; Townshend and Lake, 2009). One possibility is that access to unhealthy food is easier in deprived neighbourhoods. For example Cummins et al. (2005) show that in the UK, neighbourhood deprivation is correlated with the number of McDonalds restaurants. Another possibility is that more deprived neighbourhoods have poorer quality recreational facilities and greenspaces which may discourage physical activity (van Lenthe et al., 2005), although other research suggests access to facilities for physical activity can actually be better in deprived neighbourhoods (Pearce et al., 2007).

Additionally, as well as the built and physical environments, it may be that the social environments of neighbourhoods matter too. Neighbourhoods may have an influence on health behaviours through their influence in forming social norms and through social networks (Galster, 2012). Living in deprived neighbourhoods may also contribute to increased stress, which has been suggested to have an influence on increasing unhealthy behaviours (Pampel et al., 2010). Finally, although neighbourhoods are the focus of this study, it is important to remember that neighbourhoods themselves are only one aspect of obesogenic environments. Larger scale political and economic contexts and other small scale contexts such schools may also play an important role (Swinburn et al., 1999).

The aim of this research is to examine whether the genetic influence on BMI varies as a function of neighbourhood deprivation and if so, what the nature of this relationship is. The research is based upon data from the UK and genetic influence is included latently as heritability using data from twin pairs. The paper will proceed by first introducing the data, then outlining the statistical methodology before presenting the results and discussing their implications.

2. Data

The study uses data from Twins UK, an ongoing study of twins aged 16 and over from across the UK which began in 1992. Twins chose to become part of the study, meaning that the sample is not representative of the UK population. For historical reasons the sample is 90% female. Additionally, the sample is more highly educated and has proportionally fewer ethnic minorities than the UK population as whole. The dataset consists of measurements of height and weight, from which BMI can be calculated. These measurements were taken at various points between 1992 and 2007, as different individuals joined the study at different times and there were different waves of measurement. Some individuals were measured more than once and if this was the case the most recent measurement was used for the analysis shown in the results.

Geographical identifiers for the twins were available as postcode sectors, but as there are few statistics calculated at this spatial scale, deprivation data was taken from 2001 Carstairs scores calculated at the ward level. Wards contain on average around 6600 individuals while postcode sectors contain approximately 5000 and often coincide with parts of multiple wards. Wards therefore had to be matched to postcode sectors. This was carried out using the UK data service *geoconvert* tool (http://geoconvert.mimas.ac.uk/) which additionally gave the proportion of each postcode sector by area that belonged to each ward. From this, a deprivation score was calculated for each postcode sector using a weighted average by area of all the wards that overlapped with that postcode sector. For 19% of the twin pairs, the two twins lived in the same postcode sector, although this was more common among younger twins. The correlation in terms of neighbourhood deprivation between co-twins was 0.45.

BMI is included as the dependent variable, however, the natural logarithm is used as residual diagnostics of preliminary analyses showed that the residuals were not normally distributed. Age, sex, ethnic origin, and education as a proxy for socioeconomic status were used to control for individual level confounding as these variables may have an effect on both selection into neighbourhoods and BMI (Diez Roux and Mair, 2010). Additionally, a control was added for year of measurement as measurements were taken at different points in time, and during the time of the study there was an increase in obesity prevalence at the population level.

The original dataset contained data on 7629 individuals. Twin pairs in which one or both did not have a single valid BMI reading were excluded, along with pairs with uncertain zygosity and twins who had been raised separately. Zygosity refers to whether the twin pair are monozygotic (identical) or dizygotic (fraternal). Twin pairs who had missing data on residential location or on one of the control variables were also dropped from the analysis. This left 3128 individual observations consisting of 830 pairs of monozygotic twins and 734 pairs of dizygotic twins. As far as it was possible to assess there appeared to be no large systematic differences between the cases with complete data and the cases that had missing data. Summary statistics and information about the variables are presented in Table 1.