Spatial structures of health outcomes and health behaviours in Scotland: Evidence from the Scottish Health Survey

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Spatial structures of health outcomes and health behaviours in Scotland: Evidence from the Scottish Health Survey

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“Obesity, poor diet and excessive alcohol consumption continue to be a cause of unacceptable levels of ill health which are inequitably distributed across society... Health promotion campaigns usually have a positive effect on some people but often those in most need of changing their behaviour are least likely to take notice of such campaigns.” (Annual Report of the Chief Medical Officer, Scotland, 2010).

“Tobacco smoking remains the biggest single preventable cause of ill health, disability and early death in Lanarkshire and Scotland as a whole. ... Three out of every 10 adults in Lanarkshire smoke however prevalence is significantly higher in areas of deprivation. ... Smoking behaviour is primarily driven by addiction to nicotine however a wide range of personal, social and environmental factors interplay with the addiction to influence who starts smoking, who continues to smoke and who gives up. Tackling smoking within Lanarkshire will therefore require a comprehensive approach which incorporates a range of public health interventions at different levels to tackle the individual, social and cultural influences on smoking behaviour. ... The Scottish Government has recognised tobacco control as a key public health priority and has invested nationally and locally” (Lanarkshire Tobacco Control Strategy 2012-2015, April 2012).

Abstract
Socioeconomic characteristics, health behaviours, and the utilisation and quality of healthcare are prime examples of socioeconomic, cultural and demographic phenomena that are inherently spatial in nature. Understanding the spatial structure of these factors is particularly relevant in order to efficiently allocate resources. This paper explores the general equilibrium spatial structure of health outcomes and health behaviours across Scottish health boards using a variant of the spatial Durbin model which allows for an a priori unknown spatial weights matrix. The results suggest that there is substantial spatial dynamics in behaviours across Health Boards and that these spillovers are, as expected, asymmetric. We then demonstrate how the model can be used to estimate the behavioural and health impact of a targeted education policy within each health board taking into account both the direct effect on the particular health board itself and the indirect effect in terms of spillovers. The results illustrate how the dynamic effects play a large role in designing place based policies that maximise the overall effectiveness of health interventions. Taking into account the spatial dynamics allows policy makers to better target resources and interventions on particular clusters where the direct and indirect spillover benefits are likely to be the greatest in terms of improving health.

Keywords: spatial econometrics, spatial weights matrix, spatial Durbin model, health outcomes, health behaviours, health care utilisation.

JEL: I12, I18, C33.

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1. Introduction

Health outcomes and its potential determinants – socioeconomic characteristics, access to healthcare and health behaviours – are all characterised by substantial spatial clustering and dynamics, driven partly by spatial fixity (neighbourhood effects), spatial sorting by income, class and race (residential location choices), spatial health investments (health interventions implemented at specific locations), plus positive and negative interactions over space, time, and across socioeconomic subpopulations (spatial and cross-cluster spillovers). Moreover, the health economics and disease mapping literatures point to the concentration of adverse health risks on small areas classified by geographical space, age, race, gender, and other socioeconomic characters (health inequalities); see, for example, Gray and Leyland (2009), Dibben and Popham (2011), Popham et al. (2011) and Dass et al. (2012). This leads to clustering, as well as spillovers, across multidimensional socioeconomic and geographical features. Evidently, understanding the role of such spatial clusters and dynamics is crucial for policies aimed at minimising healthcare costs while improving health outcomes and reducing health inequalities.

Understanding the spatial structure of socioeconomic characteristics, health behaviours, healthcare and health outcomes are particularly relevant for Scotland. This is because Scotland is characterised by poor health behaviours and substantial health inequalities, both across communities and over space (Exeter et al., 2009; Shelton, 2009; Brown and Leyland, 2010; Evans et al., 2011; and Norman et al., 2011). Further, there is an increasing evidence base for a 'Scottish Effect', and more specifically a 'Glasgow Effect', with higher observed levels of morbidity and mortality in Scotland and Glasgow beyond what can be explained by socioeconomic factors; see, for example, Hanlon et al. (2005), Gray and Leyland (2009), Popham et al. (2011) and Walsh et al. (2010).

Recent literature also finds a strong connection between spatial health inequalities and deprivation (Maclennan, 2000; Exeter et al., 2005; Brown and Leyland, 2010; Norman et al., 2011; and Bhattacharjee and Maclennan, 2012), as well as spatial variation in access to healthcare (Gravelle et al., 2003; Goyder et al., 2006; Macintyre et al., 2008; and Walsh et al., 2010). In addition, using spatial data analysis on Scotland, Sridharan et al. (2007) find evidence of spatial relationships where the impact of deprivation on mortality is affected by the deprivation in neighbouring regions.

While much of the research around spatial aspects of health in Scotland has focussed on the variation in health outcomes across geographical space and deprivation categories, little work has considered the spatial general equilibrium nature of these relationships: what supply side or demand side conditions have led to spatial spillovers, the casual mechanisms through which these spatial relationships exist, and spatial or cross-cluster spillovers. For example, there is little research to investigate whether these spatial effects in health outcomes exist due to the spatial externalities in health behaviours, healthcare utilisation or the impact of these on health outcomes; see Gravelle et al. (2003) and Macintyre et al. (2008).

Accounting for spatial dynamics in the variation of health outcomes and their determinants in Scotland will provide important information for policy makers about the complex patterns of determinants which are partly determined by spatial geography and other clustering mechanisms within society. Such information can be used to understand health and healthcare behaviour and behavioural change and thus how and why risk factors and the impact of risk factors differ across spatial networks. This allows policy makers to better target resources and interventions on particular clusters where the direct and indirect spillover benefits in terms of improving health and reducing health inequalities are maximised. Such complex interventions are often used in practice; see, for example, Maclennan and More (1999) for further discussion...
on place based public policy. In 2006 Scotland launched a population-based intervention programme called Keep Well (http://www.keepwellscotland.org.uk/keepwell/index.aspx), targeting the 15% most deprived areas within each Health Board, and aiming to tackle inequalities in cardiovascular and diabetes related outcomes. However, the evidence base and evaluation processes underlying such targeting are limited (Mackenzie et al., 2010) and potential spatial spillovers for investments are ignored.

Spatial mechanisms can emerge from several sources. Learning and peer effects may lead to spatial clustering of health outcomes and healthcare behaviours. Observed or unobserved determinants may not only affect the healthcare behaviour within any particular health board, but also in surrounding health boards. For example, relative prices for healthy versus unhealthy consumption goods or the provision of services at the health board level may partly induce or prevent hospitalisations in other health boards. Likewise, hospitalisations may be affected by economic, demographic, social, labour market and urbanisation structures of the neighbouring regions. Most importantly, spatial clustering of healthcare behaviour together with spatial clustering of observed as well as unobserved determinants would typically lead to spurious regressions, where non-existent relationships appear statistically significant because of common spatial clustering between observed and unobserved determinates. Not correctly accounting for spatial dynamics leads to biased and inefficient estimates of the impact of risk factors, rendering evidence and evaluation inadequate; see Anselin (2002), Fingleton (2003) and Baltagi et al. (2007) for further discussion. By placing spatial and socioeconomic clustering and dynamics at the centre of investigation, the current line of research aids credible evidence based policy in public health.

Numerous factors influence health outcomes – mortality, morbidity and hospital admissions – including socioeconomic characteristics, health behaviours, individual behaviour regarding seeking and managing healthcare, doctors’ decisions regarding referral and accessibility of healthcare resources, such as, hospitals. These determinants are unlikely to be randomly distributed, but will instead have underlying spatial patterns both across geographies and other socioeconomic and ethnic clusters within society. If these underlying spatial patterns are not accounted for this is likely to bias research findings and thus invalidate conclusions (Bech and Lauridsen, 2008). Chin et al. (2011) and Barufi et al. (2012) found evidence of spatial correlation in Nepal and Brazil respectively, after controlling for individual and community-level covariates. Such spatial pattern in unexplained variation indicates that place based targeting of health policies could be effective.

Further, financial constraints necessitate that healthcare investments increasingly focus on specific policy instruments and small area clusters. For example, a policy maker must decide whether scarce resources are devoted to investments in healthcare provision or the promotion of healthy lifestyles. In the first case, one has to further decide whether these investments are placed in hospitals or local health centres. Likewise, in the second case, informed decisions need to be made as to which is likely to be more cost-effective – investments in education, exercise facilities, or the promotion of a healthy diet? Further, one has to decide which spatial clusters would generate the largest benefits, not only locally but also considering possible spillovers over space, time, and across socioeconomic subpopulations.

In this paper we explore spatial dynamics in health behaviours (smoking, risky alcohol consumption and obesity) across the Scottish Health Boards. We then estimate the impact of these behaviours on individuals’ self-reported health. A key focus of our analyses lies on spatial heterogeneity and spatial externalities in health behaviours and health outcomes. This focus allows us propose new methodology to evaluate policy scenarios, where place based
policy located in different health boards can have potentially different effects locally, and
different spatial general equilibrium spillover effects on other health boards.

This paper improves our understanding of the spatial determinants of health, the complex
patterns of determinants and outcomes which are partly determined by spatial geography and
other clustering mechanisms within society. Thereby, it allows effective targeted policy on
the health of the Scottish population to be planned and evaluated. Policy makers are thus
enabled to better target resources and interventions on particular clusters where the direct and
indirect spillover benefits are likely to be the greatest in terms of improving health and
reducing health inequalities. Our model and methodology are presented in section 2, followed
by results and policy simulations (section 3); section 4 collects conclusions.

2. Models, Data and Methodology

We start with a discussion of the economic model of health behaviours and outcomes, and the
data used in our analyses, followed by discussion of econometric models and methodology.

2.1 The Economic Model

The objective of our analysis is to identify the causal links between health behaviours,
healthcare and health outcomes relating to self-reported health, as well as positive and
negative spillovers over space. Figure 1 outlines a simple model in terms of the determinants
of health behaviours, utilisation and outcomes. As can be seen there are complex
relationships between these outcomes. Socioeconomic and other individual characteristics,
such as income and education, are likely to influence both health behaviours and utilisation of
healthcare, which then in turn impact on health outcomes. In addition to these complex
relationships there are also likely to be dynamic spillover effects across health boards and for
groups within health boards.

Figure 1. Relationship between socioeconomic factors, healthcare,
health behaviour and health outcomes

Our model and analyses also attempts to address issues relating to endogeneity. First, both
health behaviours and health outcomes are subject to endogenous spatial and cross-cluster
dynamics captured in appropriate spatial econometric models. In a significant departure from
the standard literature, the corresponding spatial weights matrices are not held fixed a priori.
This is important for our objective of evaluating policy on the basis of the spatial general
equilibrium effects – direct local effects together with indirect spillover effects – of policy interventions placed at different locations in space. Second, as indicated in Figure 1, the effect of healthcare utilisation on health outcomes is likely to be endogenous. Better utilisation is expected to improve health outcomes, at the same time as poor health outcomes may encourage greater utilisation. Likewise, the effect of health behaviours on outcomes is potentially endogenous as well. We find evidence of endogeneity from all the above sources, each of which is explicitly addressed in our analyses.

One may expect spillovers in behaviours across regions where the causal channels could derive from both the demand and supply sides of the market for substance abuse. For example, on the demand side there may be peer effects from interactions between individuals from different regions, either for work, study, migration or travel. Here, one region’s behavioural norms may influence the behaviour of a visitor.\footnote{Starting from the so-called reflection problem in Manski (1993), there is a large literature in this area, both theoretical and empirical; see, for example, Bramoulle et al. (2009) and Calvó-Armengol et al. (2009).} Further, the influence of drinking or smoking from one region to another need not be symmetric, where one may expect an individual travelling to another region for work may be more influenced by their destination’s behaviours rather than them extending influence on their destination’s behaviour.

Likewise, core-periphery relations would imply asymmetry, where the impact of the core regions (comprising, for example, major cities) on peripheral regions is likely, in general equilibrium, to dominate the impact of the periphery on the core. In addition, though prices (for tobacco/alcohol) do not vary much by locality, the opportunity cost of time spent in substance use does. This, for example, could be through labour market linkages, where localities with higher labour demand would therefore have a higher opportunity cost for time spent in unhealthy behaviours. These patterns would therefore emanate radially from major urban centres, such as Glasgow or Edinburgh.

On the supply side, the availability of alcohol such as a large cluster of nightclubs may influence drinking patterns in other regions where individuals from other regions travel to this cluster for entertainment, which in turn is closely related to substance abuse. Such agglomeration may concentrate on the entertainment areas of major cities (for example, Glasgow and Edinburgh) or in the urban periphery. This, in turn can generate negative spillovers because of spatial competition; see Bhattacharjee and Jensen-Butler (2013) for an example from housing markets.

2.2 Scottish Health Survey (SHeS) data

The following analysis uses pooled cross-sectional data from three rounds of the SHeS from 2008 to 2010. The SHeS provides detailed data on health behaviours, some forms of healthcare utilisation such as visits to the GP (general practitioner, or family physician), treatments for certain conditions and information on self-reported health status. It also included detailed data on socioeconomic and demographic factors. For the current analysis we only consider individuals aged 25 years and older as the impact of health behaviours on self-reported health are unlikely to have presented before the age of 25 years.

Given the nature of the data – pooled rather than longitudinal panel data – we address issues of unobserved heterogeneity at the health board level using fixed effects. However, at the individual level, heterogeneity is modelled directly using measures of socioeconomic and neighbourhood characteristics. While lack of longitudinal data places empirical challenges on our inferences, these are the most comprehensive data currently available for Scotland, covering health outcomes, behaviour, utilisation and socioeconomic features. Given the
policy relevance of the questions addressed here, we therefore view these inferences as the best possible. However, the methodology developed here can be applied to other data rich contexts and can be further extended when more comprehensive longitudinal data for Scotland become available.

2.3 Econometric models and methodology

We employ moment assumption and structural constraint based frequentist inferences on spatial weights using a variant of the spatial Durbin model (Anselin, 1988; LeSage and Pace, 2009) applied to the SHeS data. An important challenge in spatial econometrics is that the reduced forms of standard spatial econometric models (the spatial lag, spatial error and spatial Durbin models, for example) are remarkably similar. Our inferences are informed by careful consideration of the correspondence between alternate models. We focus on a purely geographical cluster given by the 14 NHS Health Boards in Scotland. In particular we examine the relationship between health outcomes \((Y)\) and its determinants \((X)\) – health behaviours \((B)\), healthcare utilisation \((U)\) and socioeconomic factors \((Z)\).

Consider the following spatial error model for the relation between \(Y\) and \(X\):

\[
Y = X\beta + \epsilon, \\
\epsilon = \rho W\epsilon + u. \\
\Rightarrow Y = \rho Y + \rho W\epsilon + WX\theta + u, \quad \theta = -\rho \beta.
\]

The above reduced unrestricted form is called the spatial Durbin model. If the spatial weights matrix, \(W\), is known, this reduced form can be estimated like a spatial autoregressive model, using either maximum likelihood (LeSage and Pace, 2009) or GMM (Kelejian and Prucha, 1998; Conley, 1999) methods. On the other hand, if \(W\) is not known, the underlying structural equation can be estimated using OLS, under the maintained assumption that \(X\) is strictly exogenous. Then, using the OLS residuals, the underlying spatial weights matrix \(W\) can itself be estimated from the autoregressive model for the spatial errors \((\epsilon)\).

The framework with an unknown \(W\) is particularly useful in our spatial context, where the structure of spatial weights determine the magnitude and strength of spillovers across health boards. However, this unknown \(W\) setting presents some challenges for identification. First, \(\rho\) and \(W\) are not separately identified; without loss of generality, we need to set \(\rho=1\). Second, even under the above normalisation, \(W\) is only partially identified. Therefore one needs further assumptions for estimation. In fact, the above model can be estimated either under structural constraints (Bhattacharjee and Jensen-Butler, 2013) or moment conditions (Bhattacharjee and Holly, 2013; Conley, 1999); for further discussion, see Bhattacharjee and Holly (2011). An assumption of a symmetric spatial weights matrix constitutes a valid structural constraint for identification. Alternatively, one can use moment conditions for identification. Typically, these moment conditions are based on spatial units at further spatial lags or subunits obtained by drilling down in space, that is, peripheral units or units with weak connections (Bhattacharjee and Holly, 2013).

Alternatively, consider a mixed-regressive spatial autoregressive model (Anselin, 1988):

\[
Y = \rho Y + X\beta + \epsilon, \\
\epsilon = \lambda M\epsilon + u. \\
\Rightarrow Y = \left[ (I - \rho W)(I - \lambda M) - I \right] Y + X\beta + MX\theta + u, \quad \theta = -\lambda \beta.
\]

Like the spatial Durbin model (1), the mixed-regressive model (2) looks similar to a spatial autoregressive model, but with an additional spatial lag for \(X\) as well. The spatial lag in \(X, WX\)
in (1) or $MX$ in (2), can arise as above from a spatial error structure, as in model (1) above. However, it can also arise from a model where $X$ itself has spatial effects through a spatial lag or spatial error structure. All the above variants of the model are observationally almost equivalent, and the reduced forms are therefore closely related. Then, even if inferences based on the reduced form are relatively similar, structural interpretation of the results rely on the assumptions within the underlying theoretical model.

In this paper, we propose a spatial econometric model of health behaviours and outcomes as:

$$
Y = f(WY, \hat{B}, \hat{U}, Z), \\
B = g(MB, Z),
$$

where there are potentially different spatial lag dynamics in health behaviours ($B$) and health outcomes ($Y$), described by spatial weights matrices $M$ and $W$ respectively. In the model for health outcomes ($Y$), health behaviours ($B$) and healthcare utilisation ($U$) are potentially endogenous, as represented by $\hat{B}$ and $\hat{U}$ respectively. The model offers an extremely rich framework for policy analyses. A place based policy induced change in some socioeconomic factor included in $Z$ within a specific health board will induce a direct local change in health behaviours, which in turn will lead to endogenous dynamic general equilibrium spillover effects to health behaviours in other health boards through the spatial lag $MB$. Thus, in turn, there will be effects on health outcomes arising from three distinct channels: (a) a direct effect locally through $Z$, (b) an indirect local effect through $B$ that includes both local and spillover effects on health behaviours discussed above, and (c) spillover effects through $WY$. An important feature of our approach is that the spatial weights matrices $W$ and $M$ are not fixed a priori, but are inferred from the data.

While our model (3) has the structural interpretation of a multi-level spatial lag model, it is estimated in the spatial error form. This is because our observation units are individuals who lie within a specific health board. If the spatial weights matrix (say, $W$) were assumed known, one could construct a spatial lag term $WY$, and include this endogenous lag within a spatial regression model. However, in our case $W$ is unknown, and hence one has to find an alternative method to identify the counterfactual of this individual being hypothetically placed in some other spatial location (health board). Bhattacharjee et al. (2012) have proposed an approach in a similar setting, where each individual within a specific health board can be matched to another individual in a different health board by paired matching along the dimensions of orthogonal factors that contribute to spatial strong dependence. In our case, such matched pairs could be constructed by closest alignment on orthogonal factors derived from the included regressors. In this paper, we propose a different approach. We estimate a one-way fixed effects spatial error model where the endogenous spatial lag remains resident in the spatial fixed effects and the spatial weights matrix $W$ is then estimated in the second stage from the estimated fixed effects. Then, our estimation model is represented as follows:

$$
Y = γ'\hat{B} + δ\hat{U} + Z\theta + (f_y + ε_y); (f_y + ε_y) = W(f_y + ε_y) + η; \\
B = Z\beta + (f_b + ε_b); (f_b + ε_b) = M(f_b + ε_b) + ν;
$$

where $Y$, $B$, $U$ and $Z$ denote health outcomes (self assessed health), health behaviours (obesity, smoking, and high alcohol consumption), healthcare utilisation (time since last visit to the doctor) and socioeconomic behaviour determinants (age, gender, income and education), respectively, and $f_y$ and $f_b$ denote location specific fixed effects. These fixed effects are estimated at the health board × deprivation category × year level, while spatial dynamic spillover (lag) effects act at the health board level on the fixed effects. 

\(^2\)In our policy experiments considered later, we use education as an example of such a socioeconomic factor.
Estimation of the model is discussed later. But, for interpretation, we link the above spatial error model (4) with our base model (3). To draw the correspondence between the two, we express the health behaviour equation in (3) as:

$$B = VB + Z\beta + \left(f_b^* + \xi_b^*\right)$$  \hspace{1cm} (5)

As discussed above, the conduct of policy experiments under model (5) is relatively straightforward. Consider a place-based policy that raises $Z$ by a certain percentage points within a specific health board, say 5% (potentially inverse weighted by population size to reflect the costs of the policy), and then use the estimated $\beta$ to evaluate its direct effect on health behaviour ($B$) within the same health board, and the estimated spatial weights matrix $V$ to evaluate its dynamic effects on behaviour in other health boards.

A corresponding strategy under (4) is more complex. If the increment in $Z$ is uniform over the cross-section of individual units (economic agents), it will not have any distributional effect, and therefore not change the fixed effect $f_b$. Therefore, there will be no spatial spillover effects. However, in our setting the true model is (5), and model (4) is estimated only as an approximation that has the same reduced form representation. As discussed earlier, different spatial models are often observationally equivalent in reduced form, at least approximately, and the same holds in this case for the spatial lag (5) and spatial error (4) models.

To see this, consider the reduced form for the spatial lag model (5):

$$B = VB + Z\beta + \left(f_b^* + \xi_b^*\right)$$
$$\Rightarrow B = (I-V)^{-1}Z\beta + (I-V)^{-1}f_b^* + (I-V)^{-1}\xi_b^*$$
$$= Z\beta + [VZ\beta + (I+V)f_b^*] + (I-V)^{-1}\xi_b^*,$$  \hspace{1cm} (6)

where the approximation holds in the usual scenario when the elements of $V$ are small compared to unity, and therefore $(I-V)^{-1} = (I+V)$.\(^3\) The reduced form errors, $(I-V)^{-1}\xi_b^*$, have spatial dependence but do not contain any systematic variation across the spatial units. From (4), we have:

$$B = Z\beta + f_b^* + \epsilon_b,$$

where, similar to (6), the errors in the main equation, $\epsilon_b$, do not contain systematic variation across the spatial units. Then, comparing (4) with (6), we have:

$$VZ\beta + (I+V)f_b^* = f_b^*.$$  \hspace{1cm} (7)

In other words, systematic changes in $Z$ are then translated to changes in the fixed effects $f_b$.

Now, to quantify these changes, we need to know the link between $M$ and $V$. This can be done by comparing the error covariance matrix in both models, since all spatial dependence rests there. Assume that the idiosyncratic errors in both models (4) and (5) have the same covariance matrix:

$$Var(\epsilon_b) = Var(\xi_b) = \Sigma.$$

Then, the error covariance matrix under (4) is

$$(I-M)^{-1}\Sigma(I-M)^{-1},$$

and that under model (5) is

$$(I-V)^{-1}\Sigma(I-V)^{-1},$$

which implies the structural equivalence:

$$V = M. \hspace{1cm} (8)$$

\(^3\) Strictly speaking, this approximation is not necessary and is as such not imposed in our computations. It is used here solely for an illustrative purpose.
That is, the spatial weights matrix for the two competing models is exactly identical. Of course, the structural interpretation of the spatial weights matrix in the two models remains quite different.

Now, we have the basic ingredients to conduct our policy experiments under the one-way fixed effects spatial error model (4). As $Z$ changes to $Z + \Delta Z$, the estimated increments in the fixed effects $f_b$ are:

$$
\left( I - \hat{M} \right)^{-1} \left( \Delta Z \right) \hat{\beta},
$$

that is

$$
f_b \rightarrow f_b + \left( I - \hat{M} \right)^{-1} \left( \Delta Z \right) \hat{\beta}.
$$

This corresponds to a change in the vector of one-way fixed effect errors (shocks), $\Delta (f_b + \epsilon_b)$, which then transmit spatially in general equilibrium as (4). Then, we use the reduced form to compute final changes in $f_b$:

$$
f_b \rightarrow f_b + \left( I - \hat{M} \right)^{-1} \left( I - \hat{M} \right) \left( \Delta Z \right) \hat{\beta}.
$$

Policy experiments on health outcomes ($Y$) are exactly similar to those for health behaviours ($B$) above.

Finally, what remains is estimating the reduced unrestricted form of (4) using data from the SHeS. We first estimate the base model, for health behaviours or health outcomes, and estimate the fixed effects for each health board × deprivation category × year level. Then, we proceed to estimate the corresponding spatial weights matrix. As discussed earlier, estimation of spatial weights would require further assumptions – structural constraints or moment conditions. However, given the nature of demand and supply side spatial linkages in our application, we anticipate core-periphery relationships between the health boards. Thus, the assumption of a symmetric spatial weights matrix may be tenuous. Instead, we initially make assumptions on moment conditions, and proceed by GMM using as instruments for $B$ (or, $Y$), data for peripheral units at higher spatial lags, together with temporal lags and lags of $Z$, and any other covariates that can be assumed exogenous. The validity of the instruments can then be verified by the Sargan-Hansen $J$-test for overidentifying restrictions (Hansen, 1982) and the Kleibergen-Paap underidentification ($r_k$ LM) and weak identification ($r_k$ Wald) tests (Kleibergen and Paap, 2006).

Alternatively, where evidence and theory suggests that the assumption of a symmetric spatial weights matrix may be reasonable, we can estimate the spatial autocovariance matrix using the above estimated fixed effects, and then estimate the corresponding symmetric spatial weights matrix using the methodology developed in Bhattacharjee and Jensen-Butler (2013). In this paper, we employ this methodology to estimate the general equilibrium spatial structure for health outcomes.

2.3 Applying the model to SHeS data

Using data from the SHeS, we illustrate the above conceptual framework and methodology based on model (5). Specifically, we assume a spatial lag model (5), which is estimated in reduced form from the corresponding spatial error model (4). The analysis presents some methodological challenges. Bhattacharjee and Holly (2013) have developed GMM-based methods for estimation of the spatial weights matrix using panel data on a collection of “core” spatial units, and using for instruments unbalanced panel data on a collection of “peripheral” spatial units. Here, however, because of concern for data consistency over time, we have restricted ourselves to three rounds of the SHeS: 2008, 2009 and 2010. Hence, the time
dimension is rather limited, so panel methods are not entirely satisfactory here. However, the key conceptual elements of the Bhattacharjee and Holly (2013) approach can still be used for GMM estimation in our setting.

An alternative approach was proposed by Bhattacharjee et al. (2012) where, based on pure cross section data, the spatial weights matrix is estimated under the structural constraint of symmetric spatial weights. This method is also not directly applicable to our setting. First, as discussed before, symmetric spatial weights may be a rather strong assumption. A priori, we expect that investments in different health boards may have differing spillover benefits, that is spillover from any given health board \((i)\) to another \((j)\), represented in the spatial weights matrix \((M)\) as the element \(m_{ij}\) may be different to the spillover from \(j\) to \(i\), that is \(m_{ji}\). Therefore, \(M\) may not be symmetric here. Second, the method is based on an orthogonal factor model, which is not directly applicable to our setting. Nevertheless, the key idea in the above approach, that the \(M\) matrix is based on an inherent matching of observations across different spatial units, along the dimension of hidden factors, which in turn generates strong spatial dependence (Pesaran, 2006; Pesaran and Tosetti, 2011) is useful in our framework as well.

We propose a synthesis of the above approaches as follows. First, we estimate a base model for health behaviours (or health outcomes) using a suitable single index model – in our case, probit or ordered probit models. In Bhattacharjee et al. (2012), matching of spatial units is based on latent factors. Here, however, we do not assume a factor structure combining explanatory variables across several dimensions – health behaviours, healthcare utilisation and socioeconomic covariates. Instead, we assume that the best possible a priori estimate for the above outcome would be the average outcome for a unit with similar spatial (geographic and socioeconomic) context. To quantify such a context, we use the cross-classification across health boards and multiple deprivation (SIMD) categories. In other words, we assume that fixed effects at each health board × deprivation category × year level constitute strong latent factors for health behaviours and health outcomes.

**Figure 2. Scottish Health Boards**

At the second stage, we use these fixed effects estimates, cross-classified by deprivation category × year levels to constitute replications. In addition, in the case of health behaviours, we also use replications by different measures (obesity, smoking and risky alcohol
consumption), assuming thereby that the spatial effects, across the health boards, are the same for each kind of health behaviours considered here. Based on these replications, we estimate $M$ using the GMM based method proposed by Bhattacharjee and Holly (2013).

This approach entails a classification of the health boards into “core” and “periphery” based on the validity of instruments (Bhattacharjee and Holly, 2013), but in this case also informed by our domain knowledge on the spatial structure of Scottish regions. The health boards thus chosen as constituting the “core” are Fife, Greater Glasgow & Clyde, Lanarkshire, Lothian, and Tayside, while Ayrshire & Arran, Borders, Dumfries & Galloway, Forth Valley, Grampian, and Highlands & Islands\(^4\) constitute the “periphery” (see Figure 2). The two major urban areas in Scotland, namely Glasgow and Edinburgh, lie within the health boards Greater Glasgow & Clyde and Lothian respectively, which are connected through the urban periphery region Lanarkshire. To the north of Lothian beyond the Forth river lie the predominantly rural health board of Fife and further north lies Tayside, which includes the city of Dundee.

There are a large number of questions in the SHeS pertaining to each of the blocks in our analysis: health outcomes, health behaviours, healthcare utilisation, socioeconomic factors and household demographics. We measure health outcomes and its determinants as follows. For outcomes, we use a measure of “self-assessed general health” included in the SHeS data. This variable is ordinal and recorded in 5 levels: very bad, bad, fair, good and very good. Thus, our primary measure of health outcomes, $Y$, is self-assessed general health. For the purpose of estimating the corresponding spatial weights matrix for health outcomes, we have 30 replications within each health board: 5 vigintiles of deprivation category\(^5\) × 3 years (2008, 2009 and 2010) × 2 levels of urbanity\(^6\). However, the spatial weights matrix, $\hat{W}$, thus estimated turns out to have an approximately symmetric pattern of spatial weights. Hence, we decided to estimate a symmetric spatial weights matrix in this case, using the methodology in Bhattacharjee and Jensen-Butler (2013).

We also used statistical factor analysis to aggregate questions pertaining to health outcomes into three leading factors, respectively accounting for 61, 22 and 17 percent of total variation in the above questions, computed by the iterated principle factor method, followed by orthogonal varimax rotation; see, for example, Rencher (2002). The leading factor aligns itself very closely to the question “had cardiovascular diseases (angina, heart attack and stroke)” with a correlation coefficient of 0.88. The second factor aligns itself closely to “doctor diagnosed high blood pressure (excluding when pregnant)” and the third factor to “receiving treatment for diabetes”. Results using these factor analysis based measures are similar and not reported here.

We follow a similar procedure based on statistical factor analysis for the other blocks. The three leading factors for health behaviour cover 59, 24 and 16 percent of total variation, and correspond to questions on risky alcohol consumption (“drinking over weekly limits”), obesity (“BMI above 30”) and smoking (“average consumption of at least 1 cigarette per day”) respectively. These constitute our three measures of health behaviours. Likewise, the leading healthcare utilisation factor aligns to “frequency of visits to doctors in the last two weeks”, with a correlation coefficient of 0.90. Several other socioeconomic and demographic variables are used as controls – age, gender and family income – and higher education is used

\(^4\) Combining the health boards of Highlands, Orkney, Shetland and Western Isles.

\(^5\) The vigintiles (20 percentile categories) are based on the Scottish Index of Multiple Deprivation (SIMD), classified as the lowest 20 percentile most deprived area through to the highest 20 percentile least deprived area; see for example, Noble et al. (2006) and Maclennan et al. (2011).

\(^6\) One level of urbanity comprises large cities and accessible towns, and the other constitutes rural areas and inaccessible/remote towns.
as our policy variable. Other demographic variables do not contribute significant explanatory power towards either health behaviours or health outcomes.

Two important components of spatiality constitute our characterisation of spatial general equilibrium. First, we pay special attention to spatial fixity which in turn leads to the notion of spatial heterogeneity. In our case, this implies key variations in the character of individual spatial units (health boards) that imply variation in the functional relationships between health outcomes and its determinants. Spatial heterogeneity constitutes the leading explanation for spatial structure in most applications (see, for example, McMillen, 2010) and is usually modelled by locally weighted regressions; see McMillen and Redfearn (2010) for an excellent review. Since in our data, location is recorded only at the level of health boards, the spatial regime model (Anselin, 1988) is adequate in capturing spatial heterogeneity. Hence, we estimate separate regression models for each health board, therefore allowing for both spatial fixed effects and full slope heterogeneity.

The second important spatial aspect is endogenous spatial dynamics – a spatially dependent dynamic process in which health outcomes and its determinants at one health board is dependent on the same states at other locations (health boards). This type of endogenous spatial dependence may arise, for example, from local interactions among spatially distributed agents or cumulative spatial spillovers generated by the decisions of many individual agents over time and space (Irwin, 2010). As our policy experiments will demonstrate, the larger general equilibrium effects of investments in specific locations are generated through endogenous spatial dynamics. Paying special attention to correct specification of the spatial weights matrix is, therefore, of key significance.

There is substantial endogeneity in our estimated models and these arise from two main sources. First, endogenous dynamic spatial lags characterise the nature of spatial spillovers across health boards. These endogenous spatial effects are addressed by estimating spatial weights matrices $M$ and $W$, using identifying moment restrictions in the first case and a structural constraint of symmetry in the second.

Second, we anticipate that healthcare utilisation is endogenously determined with health outcomes, and health behaviours may also be endogenous. We address such endogeneity using a collection of potential instruments. For healthcare utilisation, we use as instruments measures of accessibility and urbanity of the place of residence, in addition to the included health boards, deprivation categories, socioeconomic and demographic variables that we assume to be exogenous. The rationale is that, all other factors held constant, ease of access should improve healthcare utilisation beyond the effect of poor health itself. The categories for urbanisation and access provided in the SHoS are: large urban area (population 125,000 plus), other urban area (10,000-125,000), accessible small town (3,000-10,000), remote small town (3,000-10,000), very remote small town (3,000-10,000), accessible rural (population less than 3,000), remote rural (population less than 3,000) and very remote rural (population less than 3,000); the corresponding indicators are used as instruments. For health behaviours, we take a similar approach. Conditional on included exogenous variables, we treat father’s economic status when the respondent was 14 years old as a potential instrument. These instruments prove adequate in cases where issues of endogeneity are evident.

Finally, the spatial lag model (3) is estimated for health behaviours based on SHoS data for 2008, 2009 and 2010, using the GMM methodology in Bhattacharjee and Holly (2013), where peripheral units are used as potential instruments for the endogenous outcomes for the “core” Health Boards. Further, appropriate structural interpretation of spatial spillovers requires careful delineation of spatial dependence as arising from latent factors (spatial strong dependence) or the geographical ordering of spatial units (spatial weak dependence); see
Pesaran and Tosetti (2011) for the theoretical developments and Bhattacharjee and Holly (2011) for discussion on the nuances of structural interpretation. Following insights from the common correlated effects methodology (Pesaran, 2006), we account for strong dependence in our estimated models by including a full set of year fixed effects together with one annual temporal lag of the dependent variable.

Following Bhattacharjee and Holly (2013), the model was estimated row-wise by GMM, using the Stata program *ivreg2* (Baum et al., 2007). Instruments were selected separately for each row, with a view to satisfy instrument adequacy conditions. These conditions were verified using the underidentification and weak identification tests in Kleibergen and Paap (2006); see Bhattacharjee and Holly (2013) for further technical details. Likewise, for health outcomes, we estimate the spatial weights matrix under the assumption of symmetric spatial weights using the methodology in Bhattacharjee and Jensen-Butler (2013).

### 3. Results

We present our empirical results: first, for health behaviours; second, health outcomes; third, spatial weights matrices for health behaviours and health outcomes; and finally, evaluation of place based policy in enhancement of higher education.

#### 3.1 Probit models for smoking, obesity and risky alcohol consumption

We estimate probit models for whether the individual is a current smoker, whether they have a Body Mass Index (BMI) greater than 30 (that is, whether they are obese), and whether they drink over the UK limits in terms of weekly alcohol intake, 21 units for males and 14 units for females. For all models, we allow for full spatial heterogeneity, including fixed effects for each SIMD quintile within a health board and time fixed effects, together with slope heterogeneity across health boards in all the slope parameters. In the interest of space, we do not report all these results, but only the slopes using data pooled across all health boards, and indicate the degree of spatial heterogeneity by reporting the range of estimates for each slope parameter across the health boards.

#### Table 1. Probit models for smoking status, obesity and risky alcohol consumption

<table>
<thead>
<tr>
<th></th>
<th>Smoking</th>
<th>Risky Alcohol</th>
<th>Obesity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0.0293**</td>
<td>0.0377**</td>
<td>0.0585**</td>
</tr>
<tr>
<td></td>
<td>(.0010,.0062)</td>
<td>(.015,.041)</td>
<td>(.021,.089)</td>
</tr>
<tr>
<td>Age$^2$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-0.00046**</td>
<td>-0.00042**</td>
<td>0.00050**</td>
<td></td>
</tr>
<tr>
<td>(.48e-5)</td>
<td>(.46e-5)</td>
<td>(.45e-5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>[-.0008,-.0003]</td>
<td>[-.0005,-.0001]</td>
<td>[-.0008,-.0002]</td>
</tr>
<tr>
<td><strong>Male</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>0.0589</td>
<td>0.3238</td>
<td>0.0120</td>
</tr>
<tr>
<td></td>
<td>(.023)</td>
<td>(.339,.384)</td>
<td>(.084,.148)</td>
</tr>
<tr>
<td><strong>Eqv. Income</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eqv. Income (000's)</td>
<td>-0.00057**</td>
<td>0.00448**</td>
<td>-0.00081**</td>
</tr>
<tr>
<td></td>
<td>(.0008,-.0001)</td>
<td>(.0017,.0076)</td>
<td>(.0005,.0005)</td>
</tr>
<tr>
<td><strong>High Education</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High Education</td>
<td>-0.3986**</td>
<td>-0.1034**</td>
<td>-0.1411**</td>
</tr>
<tr>
<td></td>
<td>(-.399,-.582)</td>
<td>(-.173,.132)</td>
<td>(-.336,-.137)</td>
</tr>
<tr>
<td><strong>Psuedo R$^2$</strong></td>
<td>0.111</td>
<td>0.054</td>
<td>0.032</td>
</tr>
<tr>
<td>N</td>
<td>16,410</td>
<td>16,469</td>
<td>14,310</td>
</tr>
</tbody>
</table>

| *, **: Statistically significant at 1 percent and 5 percent levels, respectively. Range of estimated slope parameters across the health boards in squared brackets. |

The results of the probit model suggest that after controlling for area deprivation level and health board that males are more likely to engage in risky health behaviours though this is not

---

1 unit contains 10 millilitres of pure alcohol.
significant at conventional levels of significance for obesity. Higher equivalised income is associated with a smaller chance of being a smoker and obese but a larger probability of drinking alcohol at levels higher than the recommended weekly limit. A similar pattern is seen in terms of education with those with high level of education being less likely to be smokers or obese but more likely to be drinking at risky levels. In terms of age, the probability of being; a smoker peaks around 31 years old, a risky drinker peaks around 45 years old and being obese peaks around 58 years old before the probability falls after these ages. There is also substantial slope heterogeneity across the health boards.

3.2 Modelling the impact of health behaviours on self-reported health

In order to explore the extent to which changes in factors such as education would impact on health we estimate a model for self-reported health which takes into account the impact of smoking, obesity and risky alcohol use. Self-reported health is defined on a scale from 1 to 5 where 1 is very bad and 5 is very good. Therefore an ordered probit model is used to model self-reported health where there are four cut points which designate where an individual’s reported health status changes from one category to the next.

After controlling for other factors the old are significantly more likely to report poor health. Those with higher equivalised income and more education report better health. Those involved in any of the three risky health behaviours report worse health though for risky alcohol consumption this is only significant at the 10% level. And the impact of the time since the last GP consult is not as expected with those who have been less recently likely to report better health but this result is not significant at any conventional level of significance.

Table 2. Ordered Probit model for self-reported health

<table>
<thead>
<tr>
<th>Regressors</th>
<th>Coefficient</th>
<th>Std. Err.</th>
<th>Heterogeneity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-0.0127**</td>
<td>0.0012</td>
<td>[-0.071,-0.010]</td>
</tr>
<tr>
<td>Male</td>
<td>-0.0393</td>
<td>0.0373</td>
<td>[-0.550,-0.013]</td>
</tr>
<tr>
<td>Eqv Income</td>
<td>0.00762**</td>
<td>0.000747</td>
<td>[0.0022,0.013]</td>
</tr>
<tr>
<td>High education</td>
<td>0.2430**</td>
<td>0.0235</td>
<td>[0.134,0.637]</td>
</tr>
<tr>
<td>Risky alcohol</td>
<td>-0.9110+</td>
<td>0.5332</td>
<td>[-1.073,0.036]</td>
</tr>
<tr>
<td>Obesity</td>
<td>-0.2998**</td>
<td>0.0382</td>
<td>[-0.448,0.096]</td>
</tr>
<tr>
<td>Smoking</td>
<td>-0.3998**</td>
<td>0.0279</td>
<td>[-0.203,1.052]</td>
</tr>
<tr>
<td>Time since GP consultation</td>
<td>0.2883</td>
<td>0.2154</td>
<td>[0.497,1.478]</td>
</tr>
</tbody>
</table>

| Cut point 1 | -3.0795 | 0.1618 |
| Cut point 2 | -2.2769 | 0.1599 |
| Cut point 3 | -1.3556 | 0.1592 |
| Cut point 4 | -1.5823 | 0.1589 |

Pseudo R² = 0.0784; N = 14,308

1 Fixed effects for each SIMD vigintile within a health board are also included but not reported.
2 Age squared is not included as its coefficient was not statistically significant when both age and its square were included together.
3 This is the predicted probability of being a risky drinker where the predictions are based on a probit model which excludes the SIMD/Health board fixed effects but includes a series of dummy variables for their father’s economic status when they were 14 which acts as an instrument to identify the impact of risky alcohol use on self-reported health in the presence of endogeneity. We assume that both smoking status and obesity are exogenous in relation to changes in self-reported health in the absence of other valid instruments.
4 “Time since GP consultation” is the index derived from the ordered probit of the last time since the individual consulted a GP. The model excludes the SIMD/health board fixed effects but includes dummy variables based on a categorical variable for location and access. The results from this regression can be found in Appendix A.
5 Cut point 1 refers to the point where an individual changes their self-reported health status from “very bad” to “bad” and the subsequent cut points refer the other changes in self-reported health as health improves.
3.3 Modelling spatial dynamics

The fixed effects from the estimated models for health behaviours and health outcomes (sections 3.1 and 3.2 respectively) are then used in the spatial error model with one-way fixed effects (4) to obtain estimates of the spatial weights matrices \((M \text{ and } W)\) between health boards. These estimated matrices are reported in Tables 3 and 4 respectively, and the network is graphically illustrated in Figure 3.

Some qualifications are needed for Tables 3 and 4. First, as discussed earlier, estimation of the spatial weights matrix is a partially identified problem. In the GMM estimation for health behaviours (Table 3), we set some spatial weights to zero. Following Bailey et al. (2013), where the spatial autocorrelation between a pair of health boards was not statistically significant, the corresponding spatial weights are set to zero. Likewise, estimated spatial weights that are not statistically significant are also set to zero. This estimation strategy effectively places structural constraints on the spatial weights matrix. At least 10 such constraints are required, here we have 12 constraints. Thus, estimation under structural constraints could also be undertaken, along the lines of Bhattacharjee and Jensen-Butler (2013). Second, in the model for Lanarkshire, we estimate the two spatial weights (for Glasgow/Clyde and Lothian) separately in two IV regressions, following an estimation strategy developed in Ahrens (2013). Here, this strategy is necessitated by the fact that the set of valid instruments for the two regressors was very different from one another. Third, in all cases, specification tests for instrument validity test \((J\text{ test})\) does not reject the null hypothesis that overidentified restrictions are valid, and the Kleibergen and Paap (2006) tests for underidentification and weak identification reject the null hypothesis in either case. Thus, the choice of instruments is adequate.

Several inferences can be drawn from the estimated matrix \(M\) for health behaviours (Table 3). First, the endogenous spatial spillover effects across the health boards, arising from the spatial weights are, as expected, asymmetric. For example, there are significant spillover effects from the large urban centres of Glasgow/Clyde (G) and Lothian (Lo) to the connecting hinterland Lanarkshire (La), but no significant spillovers the other way round. Likewise, there is no significant spillover from Tayside (T) on Glasgow/Clyde (G), but the spillover from Glasgow/Clyde to Tayside is large and statistically significant.

Second, some health boards are not connected either way. Fife (F) is connected only to Tayside (T) and likewise, Lanarkshire (La) connected only to Glasgow/Clyde (G) and Lothian (Lo). There is no spillover, either way, between Lothian (Lo) and Tayside (T). Both the above observations indicate strong core-periphery relationships.

Third, there is large variation across the health boards in influence over the network. While Glasgow/Clyde (G) has significant (at 5% level) spillover effects on all the health boards except Fife (F). Lanarkshire (La) has the weakest influence, with no significant spillovers to any other health board. It can therefore be hypothesized that investments in Glasgow may have the largest spillover benefits in terms of health behaviours, and investments in Lanarkshire the smallest such benefits.

Similarly, the spatial weights for dynamics in health outcomes provide interesting inferences (Table 4, Figure 3). Here, the spatial weights matrix \(W\) is assumed to be symmetric and estimated under the structural assumption using the method in Bhattacharjee and Jensen-Butler (2013). The central core of the network is the triad between Glasgow/Clyde (G), Lothian (Lo) and Lanarkshire (La).
Interestingly, all the three connections are negative (depicted by red lines in Figure 3), indicating strong spatial competition in the markets for health and healthcare. If one of these health boards show improvements in health outcomes, it is implied therefore that this comes at the cost of a decline in health outcomes in the other two. Being a close knit triangle of two major cities and their hinterland, such a structure is very important for understanding the spatial general equilibrium. Such negative externalities are pervasive in the social sciences and spatial econometrics literature whenever the spatial weights matrix is not assumed \textit{a priori}. This pattern has close connections with Schelling’s (1969) checkerboard model of spatial segregation, and the analysis in Arbia et al. (2008, 2010) of spatial patterns of industrial agglomeration and dissociation.

![Figure 3: Network diagrams of spatial spillovers across Health Boards](image)

Table 3. GMM Estimates of Spatial Lag Model for Health Behaviours\(^8\)

<table>
<thead>
<tr>
<th>Health Boards</th>
<th>Spatial weights matrix ((M)), spatial lags</th>
<th>Speciation tests</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fife</td>
<td>Glasgow/Clyde</td>
</tr>
<tr>
<td>Fife</td>
<td>0</td>
<td>---</td>
</tr>
<tr>
<td>Glasgow/Clyde</td>
<td>---</td>
<td>0</td>
</tr>
<tr>
<td>Lanarkshire</td>
<td>---</td>
<td>0.6332 (**)</td>
</tr>
<tr>
<td>Lothian</td>
<td>---</td>
<td>0.8957 (**)</td>
</tr>
<tr>
<td>Tayside</td>
<td>0.4754 (*)</td>
<td>0.9101 (*)</td>
</tr>
</tbody>
</table>

\(*\), \(**\), \(+\) : Statistically significant at 1 percent, 5 percent and 10 percent levels, respectively.

Table 4. Estimates of Spatial Lag Model for Health Outcomes, Symmetric

<table>
<thead>
<tr>
<th>Health Boards</th>
<th>Spatial weights matrix ((W)), spatial lags</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fife</td>
</tr>
<tr>
<td>Fife</td>
<td>0</td>
</tr>
<tr>
<td>Glasgow/Clyde</td>
<td>0.1865 (*)</td>
</tr>
<tr>
<td>Lanarkshire</td>
<td>-0.1693 (**)</td>
</tr>
<tr>
<td>Lothian</td>
<td>0.0104 (\times)</td>
</tr>
<tr>
<td>Tayside</td>
<td>0.2248 (*)</td>
</tr>
</tbody>
</table>

\(*\), \(**\), \(+\) : Statistically significant at 1 percent, 5 percent and 10 percent levels, respectively.

\(^8\) A full set of year fixed effects (substituting for temporal latent factors) and temporal lags for the endogenous dependent variable were included to account for spatial strong dependence. Peripheral regions (higher spatial lags) were used as instruments for the endogenous variables. See Bhattacharjee and Holly (2013) for further discussion on the methodology and its implementation.
Like health behaviours, Fife lies on the periphery of the network, being connected only to Tayside. However, unlike health behaviours, Lothian (including Edinburgh) is now the most well connected location to other health boards, connecting the triad to Tayside. There are no direct connection between Glasgow and Tayside.

3.4 Policy evaluation

As discussed earlier, the estimated models (Tables 1 and 2) together with spatial weights matrices (Tables 3 and 4) can be used to conduct policy analysis, towards which we turn to next. Our policy experiments are in the nature of dynamic microsimulation modelling (Harding, 1996; Callan and Sutherland, 1997; Atkinson et al., 2002), with focus on both spatial heterogeneity and spatial dynamics. Specifically, we evaluate the effect of place based policy on education, whereby 100 random individuals surveyed within each health board in turn is moved from the low education to the highly educated category. On average, this would raise the proportion of highly educated individuals within the index health board by about 5 percentage points. The effect of this policy is then evaluated on health behaviour (smoking) and health outcomes (self-assessed health) across all the health board.

Table 5. Policy evaluation – Effect of moving 100 persons to higher education

<table>
<thead>
<tr>
<th>Policy implemented in</th>
<th>Total effects [direct effects], reduction in smokers (%), in</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Fife</td>
<td>Glasgow/Clyde</td>
<td>Lanarkshire</td>
<td>Lothian</td>
<td>Tayside</td>
<td>All 5 HBs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fife</td>
<td>0.08 [0.64]</td>
<td>0.02</td>
<td>0.04</td>
<td>0.04</td>
<td>0.31</td>
<td>0.07</td>
<td></td>
</tr>
<tr>
<td>Glasgow/Clyde</td>
<td>0.13</td>
<td>0.27 [0.36]</td>
<td>0.70</td>
<td>0.51</td>
<td>0.52</td>
<td>0.40</td>
<td></td>
</tr>
<tr>
<td>Lanarkshire</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00 [0.66]</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td></td>
</tr>
<tr>
<td>Lothian</td>
<td>0.08</td>
<td>0.37</td>
<td>0.69</td>
<td>0.32 [0.45]</td>
<td>0.33</td>
<td>0.36</td>
<td></td>
</tr>
<tr>
<td>Tayside</td>
<td>0.34</td>
<td>0.09</td>
<td>0.17</td>
<td>0.18</td>
<td>0.23 [1.31]</td>
<td>0.19</td>
<td></td>
</tr>
</tbody>
</table>

B. Effect on health outcomes at the top end (self-assessed health)

<table>
<thead>
<tr>
<th>Policy implemented in</th>
<th>Total effects [direct effects], increase in good/very good health (%), in</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Fife</td>
<td>Glasgow/Clyde</td>
<td>Lanarkshire</td>
<td>Lothian</td>
<td>Tayside</td>
<td>All 5 HBs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fife</td>
<td>0.00 [0.25]</td>
<td>-58.94</td>
<td>-23.99</td>
<td>-1.26</td>
<td>-25.58</td>
<td>-25.52</td>
<td></td>
</tr>
<tr>
<td>Glasgow/Clyde</td>
<td>27.79</td>
<td>0.08 [0.62]</td>
<td>25.32</td>
<td>22.80</td>
<td>20.25</td>
<td>16.61</td>
<td></td>
</tr>
<tr>
<td>Lanarkshire</td>
<td>17.15</td>
<td>-45.53</td>
<td>0.00 [0.27]</td>
<td>13.67</td>
<td>-2.18</td>
<td>-8.27</td>
<td></td>
</tr>
<tr>
<td>Lothian</td>
<td>1.44</td>
<td>-58.34</td>
<td>-22.61</td>
<td>0.08 [0.52]</td>
<td>-23.80</td>
<td>-24.34</td>
<td></td>
</tr>
<tr>
<td>Tayside</td>
<td>18.46</td>
<td>-43.54</td>
<td>2.32</td>
<td>14.79</td>
<td>0.00 [0.16]</td>
<td>-6.54</td>
<td></td>
</tr>
</tbody>
</table>

C. Effect on health outcomes at the bottom end (self-assessed health)

<table>
<thead>
<tr>
<th>Policy implemented in</th>
<th>Total effects [direct effects], increase in very bad/bad health (%), in</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Fife</td>
<td>Glasgow/Clyde</td>
<td>Lanarkshire</td>
<td>Lothian</td>
<td>Tayside</td>
<td>All 5 HBs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fife</td>
<td>0.00 [-0.12]</td>
<td>58.95</td>
<td>12.10</td>
<td>-0.40</td>
<td>11.38</td>
<td>21.58</td>
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</tr>
<tr>
<td>Glasgow/Clyde</td>
<td>-6.78</td>
<td>-0.04 [-0.39]</td>
<td>-7.62</td>
<td>-6.28</td>
<td>-5.38</td>
<td>-4.51</td>
<td></td>
</tr>
<tr>
<td>Lanarkshire</td>
<td>-4.89</td>
<td>38.82</td>
<td>0.00 [-0.14]</td>
<td>-4.59</td>
<td>0.74</td>
<td>10.29</td>
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</tr>
<tr>
<td>Lothian</td>
<td>0.46</td>
<td>60.12</td>
<td>13.27</td>
<td>-0.03 [-0.26]</td>
<td>12.19</td>
<td>22.39</td>
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</tr>
<tr>
<td>Tayside</td>
<td>-5.17</td>
<td>36.53</td>
<td>-0.88</td>
<td>-4.84</td>
<td>0.00 [-0.07]</td>
<td>9.24</td>
<td></td>
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</table>
The results are reported in Table 5 (panels A, B and C). In each case, we report both the direct effect of policy locally, plus endogenous dynamic spillover effects in all the health boards; in addition, we also report aggregate effect on all the 5 health boards taken together. Panel 5A reports on health behaviours, while 5B and 5C pertain to health outcomes – on good outcomes (good and very good self-assessed health) and poor outcomes (very bad and bad) respectively.

Admittedly, the policy evaluation exercise is highly stylised and intended to be illustrative. Two important issues are ignored. First, whereas the experiment focuses on investments on education within each individual health board in turn, in reality, investments are likely to be placed concurrently in several locations. Such combinations of place based policies are also, in a spatial general equilibrium world, likely to generate the greatest benefits. Second, the exercise does not internalise the fact that socioeconomic indicators are highly correlated. Thus, when an individual is moved into the highly educated category, the incomes generated are also likely to be therefore higher, which is not taken into account. Because of these issues, the results are only indicative. But the methodology is illustrated well, and more realistic policy experiments can be conducted comparatively easily and accurately within this framework.

The largest direct benefits on behaviours are obtained for Tayside, with a 1.31 percentage point reduction in the proportion of smokers. Fife and Lanarkshire follow, with reductions of about 0.65 percentage points, and then Lothian (0.45 percentage points) and finally Glasgow/Clyde (0.36 percentage points). This spatial variation is due to spatial heterogeneity in slopes and spatial fixed effects.

However, the situation is completely reversed, when one considers aggregate effects, combining both direct heterogeneity effects and indirect spatial dynamics effects. Then, in terms of health behaviours, the largest benefits across all the 5 health boards is generated by education investments placed in Glasgow/Clyde, a 0.40 percentage points reduction in smokers, followed closely by Lothian (0.36 percentage points). The overwhelming message is that, general equilibrium spillover effects dominate aggregate benefits and therefore cannot be ignored. However, computing such aggregate benefits requires careful consideration of appropriate spatial weights. Given the structure of the network, one can begin to understand why these major urban centres generate the maximum spillovers.

The message from health outcomes is along similar lines. While investments in all the health boards generate increases in the proportion reporting good/ very good health, the spillover effects dominate. Now, the highest spillovers are generated by investments in Glasgow/Clyde; the aggregate effect is an increase of about 17 percentage points in the proportion of good self-assessed health individuals. This is expected from the spatial weights matrix for health behaviours and health outcomes (Tables 3 and 4, Figure 3) where Glasgow emerged as having the greatest influence within the network. However, perhaps more surprisingly, all other health boards generate negative aggregate returns to investments, which is quite remarkable.

The microsimulation approach employed here also generates some compositional effects. At the other end of the spectrum, investments in Glasgow again generate the highest returns – a 4.5 percentage point reduction in the percentage of individuals reporting very bad or bad health. From a public policy perspective, what is perhaps even more remarkable is the percentage of decline, almost a 60% decrease, from 7.9 percent to 3.4 percent, in the individuals reporting poor health. As before, all other health boards record negative aggregate returns to the investment, though the direct effects are always positive. While the results here are indicative, they point towards important lessons. Most importantly, general equilibrium spatial dynamics effects dominate the direct effects, even when spatial heterogeneity is accounted for.
4. Discussion

In summary, we propose an economic model and corresponding econometric specification and new inference methods to understand the impact of various determinants on the spatial structure of health outcomes. The model and methodology place special emphasis on inferences on an unknown matrix of spatial interactions (spatial weights matrix) between spatial units, and accounts for spatial heterogeneity. Applied to data from the Scottish Health Surveys, this allows analyses of the effect of changes in health investments, health behaviour and healthcare utilisation on health outcomes in Scotland, not only locally but also including spatial dynamics across health boards. Thus, the methods allow policy analyses aimed at targeting geographical and socioeconomic clusters.

Spatial general equilibrium dynamic effects dominate, underscoring the importance of computing such effects in policy work, combined with appropriately constructed spatial weights matrices. Our analyses indicate large variations in returns to policy interventions placed in different health boards within Scotland, with the largest benefits for investments in the Glasgow/Clyde health board. These findings have important policy implications in an age of reduced resources that necessitate effective targeting. Longitudinal data on individuals over a longer timeframe would allow a more robust analysis of the general equilibrium spillovers over space.

References


Dibben, C.J.L. and Popham, F. (2011). Are socio-economic groupings the most appropriate method for judging health equity between countries?. *Journal of Epidemiology and Community Health* 65(1), 4-5.


Dibben, C.J.L. and Popham, F. (2011). Are socio-economic groupings the most appropriate method for judging health equity between countries?. *Journal of Epidemiology and Community Health* 65(1), 4-5.


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Appendix A.

The time since someone consulted with a doctor has a number of possible categories: less than 2 weeks, more than 2 weeks but less than 1 month, more than 1 month but less than 3 months, more than 3 months but less than 6 months, more than 6 months but less than 1 year, more than 1 year and never consulted a doctor.

Table A1. Time since consulted with a doctor

<table>
<thead>
<tr>
<th></th>
<th>Coef.</th>
<th>Std. Err.</th>
<th>P-value</th>
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<tbody>
<tr>
<td>(Large Urban area)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other urban area</td>
<td>0.0810</td>
<td>0.0225</td>
<td>0.000</td>
</tr>
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<td>Accessible small town</td>
<td>0.0533</td>
<td>0.0333</td>
<td>0.109</td>
</tr>
<tr>
<td>Remote small town</td>
<td>0.0711</td>
<td>0.0427</td>
<td>0.096</td>
</tr>
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<td>0.0335</td>
<td>0.000</td>
</tr>
<tr>
<td>Accessible rural</td>
<td>0.0462</td>
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<td>0.130</td>
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<td>Remote rural</td>
<td>0.0841</td>
<td>0.0822</td>
<td>0.307</td>
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<tr>
<td>Very remote rural</td>
<td>0.0325</td>
<td>0.0582</td>
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<tr>
<td>Age</td>
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<td>Age^2</td>
<td>0.0002396</td>
<td>0.0000706</td>
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<tr>
<td>Male</td>
<td>-0.1081</td>
<td>0.0604</td>
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<tr>
<td>Eqv. Income (000’s)</td>
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<td>0.00109</td>
<td>0.001</td>
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<td>High education</td>
<td>-0.0562</td>
<td>0.0288</td>
<td>0.051</td>
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<tr>
<td>Risky Alcohol</td>
<td>3.797</td>
<td>0.6304</td>
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<tr>
<td>Obesity</td>
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<td>0.0193</td>
<td>0.000</td>
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<tr>
<td>Smoking</td>
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