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Socioeconomic inequality in health in the British household panel: Tests of the social causation, health selection and the indirect selection hypothesis using dynamic fixed effects panel models

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ABSTRACT

Despite social inequality in health being well documented, it is still debated which causal mechanism best explains the negative association between socioeconomic position (SEP) and health. This paper is concerned with testing the explanatory power of three widely proposed causal explanations for social inequality in health in adulthood: the social causation hypothesis (SEP determines health), the health selection hypothesis (health determines SEP) and the indirect selection hypothesis (no causal relationship). We employ dynamic data of respondents aged 30 to 60 from the last nine waves of the British Household Panel Survey. Household income and location on the Cambridge Scale is included as measures of different dimensions of SEP and health is measured as a latent factor score. The causal hypotheses are tested using a time-based Granger approach by estimating dynamic fixed effects panel regression models following the method suggested by Anderson and Hsiao. We propose using this method to estimate the associations over time since it allows one to control for all unobserved time-invariant factors and hence lower the chances of biased estimates due to unobserved heterogeneity. The results showed no proof of the social causation hypothesis over a one to five year period and limited support for the health selection hypothesis was seen only for men in relation to HH income. These findings were robust in multiple sensitivity analysis. We conclude that the indirect selection hypothesis may be the most important in explaining social inequality in health in adulthood, indicating that the well-known cross-sectional correlations between health and SEP in adulthood seem not to be driven by a causal relationship, but instead by dynamics and influences in place before the respondents turn 30 years old that affect both their health and SEP onwards. The conclusion is limited in that we do not consider the effect of specific diseases and causal relationships in adulthood may be present over a longer timespan than 5 years.

1. Introduction

Studies have repeatedly shown that socioeconomic position (SEP) is negatively associated with both morbidity and mortality rates in adulthood (Marmot and Wilkinson, 2005). Despite the association being well documented it is still unclear which causal mechanisms best explain the social inequality in health. Explaining the association is complicated by the social gradient in health, which demonstrates that social inequality in health cannot only be caused by financial and social deprivation, and the fact that most early deaths are due to chronic conditions such as cardiovascular diseases and cancer with a long and complex aetiology (Adler et al., 1994; Bartley, 2004). This paper is concerned with testing the explanatory power of three widely proposed causal explanations for social inequality in health in adulthood: the social causation hypothesis, the health selection hypothesis and the indirect selection hypothesis (Blane et al., 1993; Marmot et al., 1997).

According to the social causation hypothesis social inequality in health is caused by the negative effect a lower SEP has on health. A lower SEP is theorised to have a negative effect via mediating factors that have a material, cultural-behavioural or psychosocial character (Skalická et al., 2009). Material factors reflect our social positions in society, which may structure health advantages and disadvantages, experienced in relation to e.g. housing, workplace and atmospheric pollution. Further the advantages or disadvantages that material factors produce in one sphere of life are likely to

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be correlated with similar advantages or disadvantages in other spheres both cross-sectional and longitudinal (Blane et al., 1997). Cultural-behavioural mediating factors are explained by the appearance of different lifestyles and preferences in different socioeconomic classes, with lower classes having less healthy behaviours (Bartley, 2004). This claim may be understood in the light of Pierre Bourdieu's theory that taste is not only related to social positioning but also an expression of social positioning (Bourdieu, 1984). Finally psychosocial factors related to SEP such as lack of social support, social insecurity, lack of control over work and effort-reward imbalance at work may influence biological processes that affect our risk of developing stress and chronic diseases (Marmot et al., 2005; Stansfeld, 2005). One biological process thought to be influenced by these psychosocial factors is the excessive frequency and strength of the fight-or-flight response with the consequence of increased risks of elevated blood pressure and blood clotting (Brunner and Marmot, 2005).

The second causal explanation for social inequality in health, the health selection hypothesis, turns the causality around so that health is hypothesised to determine SEP. The theory can be traced back to the late nineteenth century and the focus has in general been on establishing how ill people, and especially the long-term ill, are more prone to be disadvantaged occupationally and penalised financially (Blane et al., 1993). Social inequality in health is therefore thought to be caused by health related social mobility. Taken to its extremes the theory reflects a form of "social Darwinism" where people are assumed to get access to education, jobs and higher salaries in a selection process with "survival of the fittest", or put differently "survival of the healthiest" (Cardano et al., 2004).

Whereas the health selection hypothesis argues that there exists a direct causal selection effect flowing from health to SEP the last explanation, the indirect selection hypothesis, does not assume a causal relationship between health and SEP, but argues some third factors are responsible for their association (Blane et al., 1993). These third factors are theorised to be biological and psychological attributes linked to the adverse influence of poor social circumstances in early life. Parents social circumstances may for example be correlated with maternal health behaviours, which can effect the biological development of the foetus, and also with the development of the child's cognitive, social and emotional skills (Bosma et al., 1999; Wadsworth and Butterworth, 2005; West, 1991). These factors can in turn influence both educational attainment and health behaviours and thus be third factors that cause variation in both health and SEP.

The three causal hypotheses are not mutually exclusive and recent literature has stressed the importance of a life course perspective, acknowledging the interplay of different causal processes that reinforce one another, so an accumulation of advantage or disadvantage is created over the life course (Bartley, 2004; Dannefer, 2003; Elder, 1998). Even though social inequality in health may be caused by interplay between the causal mechanisms, knowing the relative importance of the different mechanisms in different periods of life is valuable to enable effective political interventions. In this study we are interested in the relative importance of the causal mechanisms in shaping social inequality in health among adults aged 30–60, in general, those in the workforce, post education and not yet retired. Despite extensive research on the associations between health and SEP in adulthood, studies examining the causal mechanisms are less frequent. A growing number of studies are assessing the importance of either the health selection or the social causation hypothesis. In the economic literature, studies have used exogenous shocks to identify the effect of one of the causal hypothesis (Anderson and Marmot, 2012; Meer et al., 2003; Smith, 2007). However, only a handful of other longitudinal studies have tested the relative importance of the causal hypotheses in adulthood, and with one exception (Case and Paxson, 2011), all evidence comes from studies that have relied on random effects models (Aittomäki et al., 2012; Chandola et al., 2003; Elovainio et al., 2011; Mulatu and Schooler, 2002; Warren, 2009). While random effects models mix cross sectional variation with longitudinal variation, fixed effects models only uses longitudinal or within individual variation (Halaby, 2004). Whereas the possible effect of indirect selection is ruled out when estimating fixed effects models, because all time-invariant factors that may affect both health and SEP in adulthood are accounted for, random effects models allow limited control for indirect selection as only observed measures of individual's health and environment in early-life can be controlled for. Warren (2009) adjusts for retrospective measures of childhood family SEP, health in childhood and education in the random effects structural equation models (SEM) he estimates while the other studies apply random effects regression or SEM models without any covariates for childhood SEP or health. Results from these studies may therefore be biased due to unobserved heterogeneity. Interestingly, the studies applying random effects models consistently conclude that the social causation hypothesis is most important in explaining social inequality in health (Aittomäki et al., 2012; Chandola et al., 2003; Elovainio et al., 2011; Mulatu and Schooler, 2002; Warren, 2009) as opposed to Case and Paxson (2011) who, on the basis of first difference models adjusting for individual fixed effects, conclude that their findings are only consistent with the health selection hypothesis. Considering this discrepancy, more studies applying fixed effects models are required to understand whether the studies applying random effects models are producing a flawed picture of the causal connections between SEP and health in adulthood.

In this study we use dynamic data from the last nine waves of the British Household Panel Survey (BHPS) collected between 2000 and 2009, which to our knowledge, has not earlier been used to test the relative importance of the causal mechanisms between health and SEP. Although there are examples of studies using the BHPS to examine either the social causation hypothesis or the health selection hypothesis (Contoyannis and Rice, 2001; Contoyannis et al., 2004). A time-based Granger approach to causality (Granger, 1969, 2004) is studied in which two regression models with lagged variables are estimated in order to investigate (1) the influence of former health on current SEP – controlling for former SEP and all time-invariant covariates, and (2) the influence of former SEP on current health – controlling for former health and all time-invariant covariates. The effect of the health selection hypothesis is hereby tested in (1) and the social causation hypothesis in (2). If proof of neither of the two causal hypotheses is found we consider the results to support the indirect selection hypothesis in explaining the observed correlation between SEP and health. The approach is similar to the one applied by Case and Paxson (2011), but we expand on their analytical framework by using the instrument variable approach suggested by Anderson and Hsiao (1982) to take account of the difference term in the lagged dependent variable being correlated with the difference term in the disturbance and by exploiting the long panel to test if the causal mechanisms can be traced over a one to five year period. Furthermore we consider measures of different dimensions of SEP (income and occupational status) and use a latent variable modelling approach to construct a general health measure which may be considered a more reliable estimate of the true health status of the population compared with any indicator used on its own. These analytical characteristics also present an expansion of earlier studies that have used BHPS to test either the social causation or the health selection hypothesis (Contoyannis and Rice, 2001; Contoyannis et al., 2004). In addition, it is worth mentioning the much-