Psychosocial epidemiology

Psychosocial factors and public health

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Another point of view (see page 565)

Macleod and Davey Smith state that the aim of their paper is to critically examine the role of psychosocial factors in health.[1] Unfortunately, what could have been an interesting discussion is compromised by the authors implicit assumption that there is a *single* pathway linking social position to health. The authors seem to equate parsimonious causal analysis with a narrow, reductionistic perspective, subsequently devoting most of their paper to a discussion of "psychosocial versus material explanation", while ignoring evidence showing multiple pathways linking social position to health.[2 3]

This commentary widens the debate by considering three issues:

(1) PSYCHOSOCIAL VARIABLES: DEFINITION AND THEIR IMPORTANCE TO HEALTH

Psychosocial variables encompass two categories of variables. The first consists of psychological attributes like hostility, depression, hopelessness, etc, which exist at the individual level, and are likely to be a result of the process of socialisation. The second category is more structural in nature, work conditions for example. These two categories work synergistically at the individual level, as can be seen from social support at work, which is a function of both work conditions and personal social interaction skills. Although the authors start out with a similar definition of psychosocial variables, in fact they interpret them rather narrowly as being the way in which "poor people feel about their poverty". This restrictive view of psychosocial variables negates the importance of the ubiquitous association between social disadvantage and a host of psychosocial variables in the developed world. We still know very little about the mechanisms that create and sustain this link, and when in the lifecourse this link is established.

In considering the importance of psychosocial variables to health Macleod and Davey Smith create a false dichotomy between "objective disease" and "misery". They themselves acknowledge "misery" to be a legitimate public health issue, particularly in the developed world with increasing life expectancies. It may be important to examine the links between "misery" and lifestyle in light of the World Health Organisation claim that "lifestyle-related diseases and conditions are responsible for 70–80% of deaths in developed countries".[4]

The authors also discuss the part played by "reporting bias" (people who report feeling miserable also report feeling ill) in explaining the association between psychosocial exposure and illness. However, one feels that this is a diversion as the authors go on to cite evidence showing psychosocial exposures to be associated with "objective" health outcomes.

(2) SOCIAL STRUCTURE AND HEALTH: ARE PSYCHOSOCIAL VARIABLES ON THE PATHWAY?

The relative importance of different pathways linking social position to health can only be assessed if these pathways are modelled simultaneously, something that has not yet been attempted. The causal sequence would be A (social position) leading to X (various pathways: social, cultural, psychological, and economic) that in turn leads to B (ill health). The authors accept the existence of this causal chain: both the link between A and psychosocial-X, and that between psychosocial-X and health. Attempts to assess the impact of the psychosocial pathway, or any other pathway, on health needs to be carried out within this sequential causal framework. Neglect of temporal order by treating psychosocial variables as another subset of factors along with measures of social position in multiple regression type analysis has been shown to systematically underestimate their role in disease aetiology.[5] It is therefore necessary to envisage new ways of examining the links between social structure and health.

Figure 1 shows a simple example of the way in which the relative importance of different pathways linking social structure to health could be modelled. This causal model respects the sequential relation between the variables, paying heed to the importance of distinguishing between proximal and distal variables in a causal chain.[5 6] Structural equation modelling (SEM) would allow the relative size of each of these pathways to be assessed. SEM has the added advantage of allowing latent constructs to be modelled, enabling a comprehensive assessment of all variables in the model. There are some recent examples of SEM[7 8] and alternative approaches to modelling pathways in the literature,[9 10] demonstrating the way in which complex analytical techniques can be used to answer complex questions.

To assess the "independent effect" of psychosocial variables, Macloed and Davey Smith put their faith in the counterfactual model of causation. The basis of establishing causality here is the probability of disease in the exposed group that would have occurred had they not been exposed. As random assignment of psychosocial variables is not feasible, the authors recommend an examination of the impact of psychosocial interventions. However, psychosocial intervention studies are unlikely to shed any light on the importance of psychosocial variables. This is primarily because the counterfactual contrast being set up is meaningless if social structure is inextricably associated with psychosocial variables.[11] Let us take the example of a "psychosocial intervention" set up to improve social support at work for the socially disadvantaged group. This would involve achieving a minimum of two things: fundamental changes in the structure of work, and instant learning of appropriate

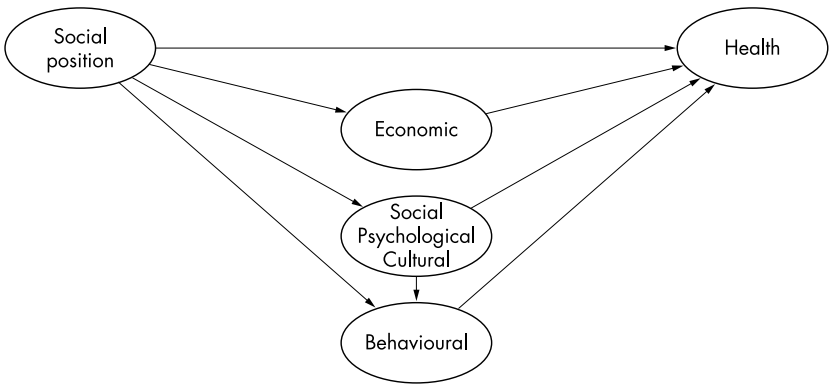

Figure 1 Relation between social position and health.

social skills normally acquired over the lifecourse. The near infeasibility of such an intervention is clear. The way ahead entails choosing appropriate statistical models that reflect advances in conceptual and theoretical models.

(3) PSYCHOSOCIAL VARIABLES AND POLICY IMPLICATIONS

Macleod and Davey Smith are quite right in stating that amelioration of social inequality in health is a priority for public health policy in most economically developed countries. However, they believe that "psychosocial solutions do not necessitate fundamental social change", while accepting the causal link between social disadvantage and psychosocial adversity. It seems difficult to understand how psychosocial change would work without a change in social inequality to which it is causally linked. This commentary calls for a push in social epidemiology towards understanding the mechanisms by which social structure influences psychosocial variables. Socialisation agents may be responsible, and the part played by parents, schools, and other agents needs to be elucidated. Policy should also be directed towards improving the structural aspects of psychosocial variations, in terms of work structure, work-life balance, etc. Psychosocial variables are important both because they affect quality of life ("misery") and are on the causal pathway to somatic disease.[12] As public expenditure on health encompasses both these outcomes, policy implications need to address them both.

In conclusion, any discussion on psychosocial variables is welcome as it is likely to promote development of both theory and method aimed at understanding the links between social structure and health.

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Authors' reply

We are sorry that Dr Singh-Manoux felt our discussion was not as interesting as it could have been, and while we recognise that this is necessarily true, her rejoinder leaves us unclear as to why she feels this way. Most of the points she raises in her commentary relate to areas we discussed in some detail.

Dr Singh-Manoux accuses us of "ignoring evidence showing multiple pathways linking social position to health". On the contrary, we did exactly the opposite. In our view there are multiple but specific pathways between social position and health outcomes, as we have discussed in depth elsewhere.[1] Examples include childhood living conditions that predispose to *Helicobacter pylori* acquisition and (many decades later) adult stomach cancer risk. The current social patterning of adult stomach cancer risk is thus the outcome of material processes acting in the early years of life.[1] Conversely an adult income that allows the purchase of airline tickets to sunny places, thus increasing the risk of melanoma or death in plane crashes, explains why these two causes of death often show a strong positive social gradient.[1] While recognising the fact that psychosocial experiences reflect events in the external world impacting on the micro-processes of brains of individuals, we will refer to "material" causes in this response as those aspects of the world that will influence health independent of the psychological response they engender.

Recognition of these (and many other[1]) specific pathways is explicitly opposed to the "general susceptibility" theories that underlie much of the psychosocial discourse[2]; it is in the psychosocial literature that one reads of how psychoneuroendocrine pathways mediate between the external psychosocial environment and nearly every health problem imaginable.[3–5] In our paper we acknowledge that many factors (including psychosocial factors) are associated with social position, and hence potentially with health, as health is socially patterned. Our central concern, as public health scientists, should be to establish which of these associations are causal, rather than merely correlational. We need to make this distinction because non-causal associations will not form the basis for effective interventions to improve population health and reduce health inequalities. We have made no a priori implicit assumption that only material pathways link social position to health, and have only argued that the evidence should be examined critically. But we make no apology for continuing to emphasise the probable key role for material factors. Across all the different classification schemes what, fundamentally, defines differences in social position?[6] We suggest, differences in the power to access material assets and, linked to this, the power to make healthy choices. Wealth is required to convert knowledge to health.[7]

However, as we clearly stated, the main purpose of our paper was not to consider the evidence for a material causal hypothesis in relation to social health inequalities. Rather it was to consider the evidence for the psychosocial causal hypothesis. Most of this evidence is observational, and is therefore subject to considerable problems of interpretation.[8] One of these is reporting

bias. It is interesting that Dr Singh-Manoux feels that this is a "diversion". In fact, much of the evidence in this area of research, such as that from the Whitehall II study, has been based on links between psychosocial factors and physical health outcomes assessed from self reports.[9] A comparison of these relations with those involving the few objective physical health measures thus far reported from Whitehall shows the latter to be considerably weaker.[10] We reproduce these data in table 1, alongside our own from the West of Scotland Collaborative Study[11]—which provide clear evidence of reporting bias—to allow readers to make their own assessment. Other than as a reflection of reporting bias, how should we explain these findings?

Table 1 Associations between perceived stress and job control and subjective and objective outcomes in the West of Scotland Collaborative Study and the Whitehall II Study

Outcome type	Effects in Collaborative Study[11]	Effects in Whitehall II Study[9 10]
Fully subjective*		
High exposure	2.66 (1.61 to 4.41)	2.02 (1.22 to 2.34)
Medium exposure	1.37 (0.91 to 2.08)	1.44 (0.86 to 2.39)
Low exposure	1.00	1.00
Fully objective**		
High exposure	0.67 (0.36 to 1.26)	1.17 (0.8 to 1.8)
Medium exposure	1.03 (0.71 to 1.49)	1.16 (0.8 to 1.7)
Low exposure	1.00	1.00‡

*Rose angina in both studies; †ECG abnormalities (Minnesota coding system) in both studies. All estimates adjusted for age, social position, and cardiovascular risk factors other than ‡ (only unadjusted estimates were reported in the paper).

Perhaps even more important, is the issue of confounding—are psychosocial exposures themselves health damaging or are they merely markers for other factors that are causally related to physical health?[12] As Dr Singh-Manoux notes, there currently appears an almost ubiquitous association between general social disadvantage and a host of psychosocial variables in the developed world. She then seems to chastise us for our neglect of the question as to why such factors may be linked to social position. Are we the only readers of the *JECH* who feel that it is scarcely mysterious that a lifetime of social disadvantage and disenfranchisement may be associated with negative feelings in the individual experiencing such hardship? However, simply because the basis of the relation between disadvantage and bad feeling is self evident it does not follow that bad feeling self evidently *causes* objective physical disease. Bad feelings are clearly a bad thing, but they may not be on the pathway between social disadvantage and objective physical disease as Dr Singh-Manoux claims.

Dr Singh-Manoux then raises the issue of the behavioural or "lifestyle" pathway between negative feelings and poorer health. We are far more accustomed to hearing the argument that neuroendocrine pathways represent the main mechanism by which psychosocial factors "get under the skin".[13] Social gradients in heart disease in Whitehall were, after all, equally apparent among lifelong non-smokers.[14] Furthermore, adjustment for lifestyle measures only partly attenuated most of the social inequalities in physical health reported from Whitehall.[14 15] In our own data from Scotland, higher stress was indeed associated with less healthy lifestyle but not with poorer health.[11 12] So we agree with Dr Singh-Manoux, that negative feelings may, depending upon context, feed into unhealthy lifestyles. However the coincidence of some unhealthy behaviours with social disadvantage is comparatively recent: in 1950 53% of physicians in the US smoked, compared with 40% of all adults.[16] Lifestyle thus seems an incomplete explanation, as suggested by the above evidence, for current social health gradients. The determinants of behaviour are complex and the generally unimpressive effects of individually targeted interventions aimed at modifying behaviour should remind us of this.

In our paper we discuss general strategies for drawing causal inference in health science. We are happy to agree with Dr Singh-Manoux that, when prospective observational data are all that are available, there may well be a place for greater use of the graphical approaches, including structural equation modelling, that she suggests. However we reiterate our points regarding the limitations of analytical sophistication in resolving these issues, as exemplified by the recent cases of antioxidant vitamins and hormone replacement therapy, where strong observational evidence of protective effects against heart disease has been overturned by randomised controlled trial evidence.[17] We disagree with Dr Singh-Manoux's dismissal of the role of experimental studies in this regard, and with her interpretation of the work of Weitkunat and Wildner, who basically develop the ongoing argument as to whether it is appropriate to adjust for covariates that may be causal intermediates—rather than confounders—in statistical models. [18] They show that such adjustment will tend to accentuate apparent effects of factors more proximal to the outcome. In other words in the case of psychosocial factors that may mediate the relation between social position and health adjustment will tend to lead to the psychosocial measure appearing to have an effect "independent" of that of the more distal (and perhaps determining) social position measure. Psychosocial exposures *are* amenable to experimental manipulation.[19–21] If they weren't how could they form the basis for useful health interventions? Experiment remains the most powerful means of reducing the risk of being misled by confounding and selection bias (with "Mendelian randomisation"—in essence a natural experiment—a close second).[22] We doubt that Dr Singh-Manoux is really suggesting that we abandon randomised controlled trials in favour of observational studies analysed using structural

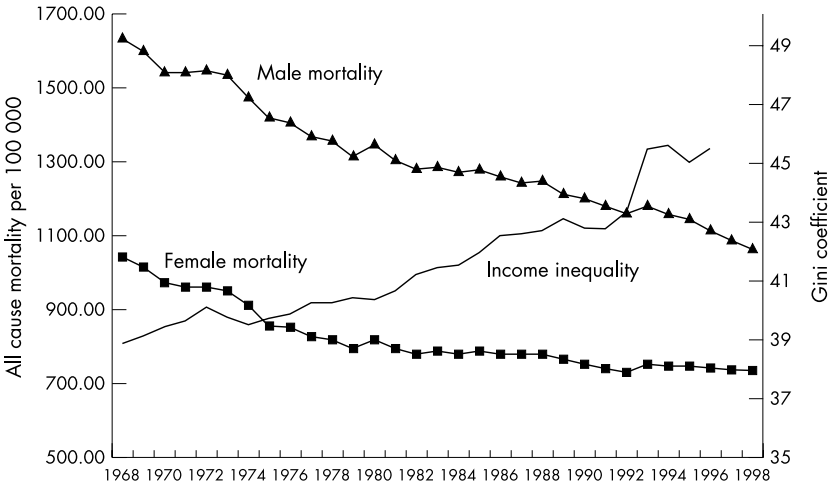

Figure 1 Income inequality (Gini) and sex specific, age adjusted, all cause mortality USA, 1968–1998.

equation modelling as an approach to determining causation and the loci for health interventions. It seems unlikely that the methods she proposes, if applied to observational data on, say, hormone replacement therapy and heart disease risk would have led to reaching the right conclusions either.

Dr Singh-Manoux states that, "psychosocial intervention studies are unlikely to shed any light on the importance of psychosocial variables." We think this is an ill considered assertion based on her idea that changing psychosocial exposure, without changing the social structure that it is imbedded within, will be difficult. This is of course true; in fact we made this very point in our paper and elsewhere.[23] But such difficulty notwithstanding, we agree with Kuper, Marmot, and others, that intervention studies are the bullet that psychosocial epidemiology has to bite if it is to influence policy.[24–26] Experimental studies in this area will provide better evidence on true causality than observational studies, however cleverly the latter are analysed. More importantly they will tell us how, if at all, these causal relations might lead to effective public health policy.

We did not touch upon population health in our paper, but one of the key issues with respect to viewpoints that see a primary psychosocial determination of health is that it makes little sense in regard to trends in overall population health. Factors such as income inequality (and presumably the feelings associated with it), and indices of "social capital" such as rates of participation in the electoral process have all deteriorated over a period when mortality rates have declined (fig 1).[27–31] Of course the contribution of psychosocial factors may differ by particular outcomes and may be complicated by differing time lags between exposure and disease. Nevertheless, perspectives that take into account the life course influences of particular material factors on specific health outcomes are largely congruent with population health trends.[1 32]

We argued for the need to critically examine the evidence supporting a causal role for psychosocial exposures on objective disease and raised issues of reporting bias and confounding in that regard. Considering these issues is standard practice in epidemiology, we ask nothing more from the study of psychosocial exposure than is asked in other areas of population science. And to reiterate, the human misery generated by unfair and unequal societies is unquestionably a bad thing. However, whether it is also a significant cause of physical disease seems unclear; clarifying this issue is important because it has implications for how policy might effectively improve peoples' health in both relative and absolute terms.

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