Background

Although the influence of wealth, status and power on health has been documented across different cultures for centuries (Liberatos, Link & Kelsey, 1988), it was not until the nineteenth century that more systematic scientific evidence emerged showing that those who were more affluent lived longer and healthier (e.g. by Villermé (1840) in France, Chadwick (1842) in Britain and Virchow (1848) in Germany). However, with the advance of bacteriology in the late nineteenth century and the ensuing dominance of the biomedical paradigm of health and illness, considerations of socioeconomic status (SES) in relation to health were largely put aside and confined to its role as a control variable (House, 2002).

With the realization of the limits of modern medicine, interest in social epidemiology and medical sociology grew again during the second half of the twentieth century (Bloom, 2002) and so did the output of research looking at SES, in particular poverty, and health. These early studies assumed a threshold effect of SES on health (Adler & Ostrove, 1999, see Figure 1); increases in income were thought to improve health only beneath, not above, a given ‘poverty line’. As discussed below, however, emerging evidence showed the picture to be far more complex than this.

Main observations

Socioeconomic status as used in research is a conglomeration of various concepts which centre around indicators of desirable social and material attributes. How it is operationalized depends both on theoretical orientations as well as practical limitations and most studies tend to estimate SES either by educational attainment, income, occupation or a combination of these (Kaplan & Keil, 1993). Irrespective of the actual SES measure used, the last 50 years have produced a number of consistent ‘big’ findings about the association between SES and health (Young, 2004).

The most ubiquitous feature of this association is its shape. What has been found in virtually all studies is a ‘gradient’ effect (Adler et al., 1994) rather than a threshold effect (Figure 1), which holds across various countries (Braveman & Tarimo, 2002), different ethnic groups within countries (e.g. Davey-Smith et al., 1996) and gender (e.g. Bosma et al., 1997). Stepwise increase in SES is accompanied by:

- Stepwise improvement of standardized mortality rates (e.g. Marmot, Shipley & Rose, 1984), disease progression (Lynch et al., 1998) and life expectancy (e.g. Guralnik et al., 1993)
- Stepwise reduction of infant mortality (e.g. Maher & Macfarlane, 2004), chronic disease (e.g. Townsend, 1974) and psychiatric disorder morbidity (e.g. Dohrenwend & Schwartz, 1995)
- Stepwise increase in self-reported good physical (Figure 2) and mental (Figure 3) health (e.g. Hemingway et al., 1997).

The only notable exception to this rule is the differential incidence of neoplastic diseases, some of which follow the social gradient (e.g. lung cancer, Mao et al., 2001) whereas others do not (e.g. breast cancer, Devesa & Diamond, 1980).

Additional features of the SES–health relationship include the existence of ethnic differences that hold within each different social class; non-white populations tend to have worse health indices than white populations along the social spectrum (Pamuk et al., 1998).
Relationship between SES and health.

Fair or poor health by family income and sex, United States 1995.

Adapted from: Pamuk et al., 1998.
There is considerable evidence for the influence of SES on health at all stages of the life cycle; the social gradient is found from childhood (e.g. Case, Lubotsky & Paxon, 2002) through to old age (e.g. House et al., 1994). Of particular importance for health across the lifespan is childhood SES. Although health is modified by changes in SES throughout life (Blane, 1999), the social class of origin influences health risks independent of risks associated with the class of destination (e.g. Bartley & Plewis, 1997).

It is worth noting that the health gap between the rich and poor is widening. Both in the UK (Macintyre, 1997) and in the USA (Pappas, Queen, Hadden & Fisher, 1993), there is ample evidence for an increase in health disparities between different socioeconomic groups despite overall improvement in general health. A similar widening of health inequalities has also been observed between countries. For instance, over the last 30 years life expectancy rose in Western Europe whereas it declined in Eastern Europe (Bobak & Marmot, 1996).

Cross-cultural research into population health has unearthed another aspect of the social gradient. Although it is clear that a nation's health depends on its wealth, what may be equally important is how this wealth is distributed within socio-economic hierarchies. More egalitarian countries such as Sweden tend to have better overall health indices than other wealthy countries with greater social inequalities such as the USA (Daniels, Kennedy & Kawachi, 2000 but see Mackenbach et al., 1997) (see ‘Cultural and ethnic factors in health’).

Lastly, the association between SES and health seems to persist even after controlling for a wide range of possible determinants (Lantz et al., 1998). This implies that there may be direct causal pathways from SES to health. How then, can one explain the link between socio-economic status and health?

**Causal explanations**

The reasons offered for socioeconomic inequalities in health are diverse and multifaceted. The seminal Black Report, produced by the UK Department of Health's Working Group on Health Inequalities in 1980, suggested three possible explanations for the observed association.
**Artefact**

First of all, the association may be an artefact of the way that both SES and health are assessed. In fact, despite the suggestion that findings may be affected by the methods used to estimate SES (e.g. Oakes & Rossi, 2003), the consensus is that the consistency of findings across different measures strongly implies that the relationship cannot be artefactual (e.g. Blane, 1997).

**Social selection**

Secondly, the report proposed that social selection may explain differentials in health by SES. This view implies that health determines SES and not vice versa (see next section). Whether people change their socio-economic position with reference to their parents (intergenerational) or to themselves at an earlier point in life (intragenerational) depends on their health status. While there is evidence for the effect of ill health on downward and good health on upward inter- and intragenerational social mobility, this effect is estimated to be only very moderate in size and therefore not sufficient to explain the SES–health associations (van de Mheen *et al.*, 1999).

Yet it is also possible that a less direct selection pathway could account for socioeconomically determined health differences. One could argue that some individual qualities, such as cognitive ability, coping styles, physical and mental fitness etc. influence success in life and health simultaneously, thereby making social mobility dependent on determinants of health rather than health itself (Goldman, 2001). This approach may indeed have more explanatory scope than the direct selection hypothesis, especially with regards to the possible contribution of genetic factors to the SES–health relationship through personal attributes (Mackenbach, 2005) (see ‘Personality and health’).

**Social causation**

It is, however, the third explanation provided by the Black Report, which accounts for most (40–70%, Marmot *et al.*, 1991) of the observed social gradient – the impact of socioeconomically conditioned determinants on health. In particular, material, behavioural, psychosocial and biological factors have been identified as providing distal and more proximate causal pathways from SES to health (e.g. see Figure 4 for a comprehensive theoretical model of the various determinants of health).
Material factors
The main thrust of health inequalities is likely to derive from material and economic inequalities. People in lower socio-economic positions have to put up with worse living conditions; they are more often exposed to toxic waste, air and water pollution, crowding, ambient noise and generally poor housing quality, all of which are linked to ill health (Evans & Kantrowitz, 2002). Furthermore, these living environments are more conducive to unhealthy lifestyles, featuring a higher concentration of alcohol outlets and a lack of stores selling healthy foods (e.g. Macintyre, Maciver & Sooman, 1993). People with lower income also experience more risky and physically demanding working environments (Lucas, 1974).

In addition, there is evidence of restricted access to, and less effective utilization of, medical care by patients from lower SES (e.g. Field & Briggs, 2001), in part due to the physical environment (e.g. lack of transportation, Takano & Nakamura, 2001).

The influence of economically determined access to health care alone, however, cannot explain the social gradient (Adler et al., 1993), as material differences do not explain the subtle graduation of health indices detected even at the higher end of SES.

Behavioural factors
Behavioural factors related to medical care provide additional insights into this association. Differential health outcomes across different socioeconomic groups may be a result of both differences in doctor–patient communication (Willems et al., 2005) and varying compliance with medical advice. For instance, unsatisfactory adherence to a prescribed medication regime predicts health deterioration and is more...
common in less well educated people likely to be lower down the socioeconomic scale (Goldman & Smith, 2002) (see ‘Adherence to treatment’). However, although possibly explaining some of the SES–health relationship, this is unlikely to be the whole story since the social gradient is evident even before the need to seek medical treatment.

A more obvious explanation relates to behaviours which determine health status directly. For instance, cigarette smoking is strongly linked to socio-economic status and exhibits a gradient very similar to that of health and SES (see Figure 5). What is more, the gap in smoking prevalence between social groups has substantially increased over the last 30 years. Thus health behaviours such as smoking may account not only for health differentials across the whole social spectrum but also explain the observed widening of health differences between socio-economic groups (Jarvis & Wardle, 2005). This proposition is corroborated by evidence of comparable gradient relationships with SES in a variety of other health behaviours. People in lower SES are less likely to be physically active (e.g. Helmert et al., 1989), are more likely to overeat and be obese (e.g. Helmert, Mielck & Classen, 1992), as well as to consume alcohol to excess (e.g. Davey-Smith et al., 1996).

However, even when these various behavioural factors are controlled for in analysis, the social gradient is reduced not eliminated (e.g. Marmot et al., 1984). Perhaps psychosocial factors can further elucidate causal pathways from SES to health.

**Individual psychosocial factors**

One of the most prevalent psychosocial determinants of health is stress. Empirical studies show that people of lower SES are more consistently exposed to chronic stress (Turner et al., 1995), which in turn is associated with poorer physical and mental health (e.g. Avison & Turner, 1988). Variation in chronic stress may also explain some of the ethnic differences in health that are found along the social gradient. For instance, discrimination as a stressor has been related to hypertension, a precursor of cardiovascular disease (Krieger & Sidney, 1996). There is additional evidence that being higher up in the socio-economic hierarchy reduces the risk of exposure to negative life events (Mcleod & Kessler, 1990), which may induce
acute (Theorell, 2005) and chronic illnesses (Cohen & Williamson, 1991).

Social support is a psychosocial factor proposed to moderate the relationship between SES and health. Social support appears to buffer the impact of stress on health, and is associated with socio-economic status both in terms of social network size and participation (Thoits, 1995). Other coping resources important to the social gradient include ‘sense of control’. Higher perceived control over life circumstances is more prevalent in people of higher SES and this is related to improved health outcomes (Lachman & Weaver, 1998) (see ‘Perceived control’). In accordance with the demand-control model (Karasek, 1979), low perceived job control has been associated with both lower SES and increased cardiovascular disease risk in the UK Whitehall II study and, furthermore predicted sickness absence when it was accompanied by high work demands (North et al., 1993). Psychosocial work characteristics may also partially explain the inverse association of depression, itself a risk factor for coronary heart disease (Booth-Kewley & Friedman, 1987), with SES (Stansfeld, Head & Marmot, 1998).

It appears that those exposed to most hardship are also those who have the least resources (both in economic and psychosocial terms) to cope with it. But it is not only at the individual level of explanation that it is possible to discern reasons for the reported observations on health and SES.

Population psychosocial factors
Research shows that residents of low-income neighbourhoods show lower collective efficacy; that is they perceive less social cohesion and social control, which may not only affect physical wellbeing (e.g. reduction in physical activity outside the home) but also mental health (Cohen et al., 2003). These population effects are not only restricted to low-income neighbourhoods; rather they appear to be related to income inequalities in the social environment. Greater inequality is associated with lower collective efficacy but also greater hostility (a risk factor for coronary heart disease, see Dembroski et al., 1989) (see ‘Hostility, Type A behaviour and coronary heart disease’) and violence (Wilkinson, 1999). Indeed, neighbourhood effects account for a large proportion of health outcomes (Pickett & Pearl, 2001).

Wilkinson has argued that social ordering may have a direct effect on health through the effects of social anxiety, which arises from fear of rejection and negative evaluations by others. Shame and social anxiety are intimately linked to social comparison, a process at least partly based on socio-economic position (Sennett & Cobb, 1973). Social anxiety is an innate mechanism that fosters social inclusion by diverting conflict in social relationships (Leary & Kowalski, 1995). However, increased social inequalities lead to intolerable and chronic anxiety which may result in frustration and aggression. Thus in societies with narrower income differences and therefore lower social anxiety, the quality of social interaction is postulated to be better and overall health superior.

Of course all the factors cited so far, be they material, behavioural or psychosocial, at the individual or population level, are themselves interrelated. For example, adopting a healthier lifestyle like giving up smoking – usually a difficult process – may be particularly difficult for people whose lives are already more prone to stressful episodes due to work strain and neighbourhood effects, which deplete their coping resources. In addition, people of lower SES, as a result of economic constraints, would find themselves in an environment that promotes smoking both physically (access to more stores selling cigarettes) and socially (greater acceptance of smoking habit, e.g. Curry et al., 1993) (see ‘Tobacco use’).

Biological factors
All of the above determinants, at least within a biomedical model (for a more sociological explanation see Young, ibid. for instance), are thought to influence health through proximal biological factors. For example,
as a result of damage to arterial walls and a decrease in HDL cholesterol, smoking leads to atherosclerosis, which is linked to cardiovascular disease morbidity (e.g. Brischetto et al., 1983).

More generally, Seeman and McEwan (1999) suggest that stress (in the broadest sense of the word), which is caused by various material, behavioural and psychosocial health determinants, elevates the activity of physiological systems and over time leads to ‘wear and tear’ of these. Life experiences but also genetic predisposition contribute to ‘allostatic load’, i.e. stress-induced damage. In the short run, the human body is well equipped to deal with stressors through the action of hormonal stress mediators in the hypothalamic–pituitary–adrenal axis and sympathetic adrenal medullary response system. However, constant insults delivered to this system decrease the efficiency with which it is turned on and off (as captured by the concept of ‘reactivity’) and the resultant bodily changes lead to poor health and illness (see ‘Stress and health’). The assessment of allostatic load by secondary outcome measures such as fibrinogen has produced gradients equal to those for disease morbidity and mortality (Markowe et al., 1985) and their inclusion as control variables has even been shown to reduce SES risk differences to non-significance (e.g. Lynch et al., 1996).

**Current and future directions**

This biological perspective lends support to the emerging field of life course epidemiology (Kuh et al., 2003), which proposes the study of long-term effects across the life span. It is evident that no single pathway is responsible for the observed health differentials and this approach is able to integrate different causal descriptions into a coherent whole, forming a conceptual framework for the multitude of explanatory levels (see Figure 5) that bear upon individual and population health. It allows for a better understanding of the temporal influence on health, such as the impact of childhood SES on adult health. As mentioned earlier, numerous studies have found that both the social and physical environment in childhood partly determines a child’s mental and physical ability as well as biological and behavioural patterns later in life (e.g. Rahkonen et al., 1997).

The life course approach can thus account for cumulative risk (as implied by ‘allostatic load’) but also for the independent and interactive influence on health exerted by biological and social factors (Kuh et al., 2003). Lastly, it allows for an exploration of the reciprocal nature of the relation between environmental and genetic determinants on health (Mackenbach, *ibid*).

Others, however, argue for a paradigm shift to a sociological (critical realist) perspective as offering a better approach for explaining socioeconomic health differentials (e.g. Scambler & Higgs, 2001).

Either way, with the development of novel frameworks comes the demand for appropriate methodology to do justice to the investigation of issues as complex as those involved in social epidemiology. More sophisticated multilevel analytical approaches rather than simple regression analyses are required in order to disentangle the various dynamic forces that affect health outcomes (Merlo, 2003). This also highlights the need for better measures of social status in order to preclude the possibility of producing spurious relationships in increasingly intricate analyses (Oakes & Rossi, 2003).

Whatever the outcome of future research, health inequalities are not immutable (Whitehead, 1990). Rather they represent a social injustice that is unlikely to be alleviated by anything other than wide-ranging public policies, which promote a more egalitarian society and aim directly at reducing socioeconomic inequalities (Link & Phelan, 1995).

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