Social Epidemiology

Chapter · January 2005

DOI: 10.1007/978-3-540-26577-1_23 · Source: OAI

2 authors:

Tarani Chandola
The University of Manchester
150 PUBLICATIONS **9,938** CITATIONS
SEE PROFILE

Michael G Marmot
University College London
1,333 PUBLICATIONS **127,256** CITATIONS
SEE PROFILE

Social Epidemiology

TARANI CHANDOLA

MICHAEL MARMOT

Department of Epidemiology and Public Health

University College London

1-19 Torrington Place

London WC1E 6BT

e mail: Tarani@public-health.ucl.ac.uk
telephone: +44 20 7679 5629
fax: +44 20 7813 0242

Outline

1. Introduction

2. Research questions
    - 2.1 the social determinants of health
    - 2.2 the social context of behaviour
    - 2.3 economic and political determinants of health and disease
    - 2.4 a developmental and life-course perspective
    - 2.5 advances in social biology
    - 2.6 ecological and contextual perspectives
    - 2.7 generalised susceptibility to disease through psychosocial mechanisms such as
        - 2.7.1 lack of social support and social networks
        - 2.7.2 work stress
        - 2.7.3 social disorganisation and lack of social capital
        - 2.7.4 job loss and unemployment
        - 2.7.5 Depression and affective states

3. Research methods
    - 3.1 Applying a population perspective
    - 3.2 Utilising better measures of social conditions, psychosocial conditions, health related quality of life
    - 3.3 Utilising better statistical methods to analyse longitudinal data, latent unobserved variables, multilevel data
    - 3.4 Analysing longitudinal data, population surveys, birth cohorts

4. Setting government policy agenda
    - 4.1 Black and Acheson reports in the United Kingdom
    - 4.2 Collating evidence for policies through intervention studies and cross national comparative studies

5. Conclusion

1 Introduction

Social Epidemiology has been defined as the branch of epidemiology that studies the social distribution and social determinants of health (Berkman and Kawachi 2000). As all aspects of human life are inextricably bound within the context of social relations, every conceivable epidemiological exposure is related to social factors. In this broad sense, all epidemiology is social epidemiology (Kaufman and Cooper 1999) with perhaps the latter discipline making explicit the analysis of the social determinants of health.

The idea that social conditions influence health is not new. Chadwick (Flinn 1965) wrote about the insanitary conditions of the working classes and how overcrowding, damp and filth contributed to their lower life expectancy. Durkheim (1996) wrote about how social norms and conditions affect risks of suicide in the population. Social epidemiology builds and expands on this literature by posing new research questions, utilising new research methods and influencing government policy agenda. The rest of this chapter will discuss each of these three developments in social epidemiology.

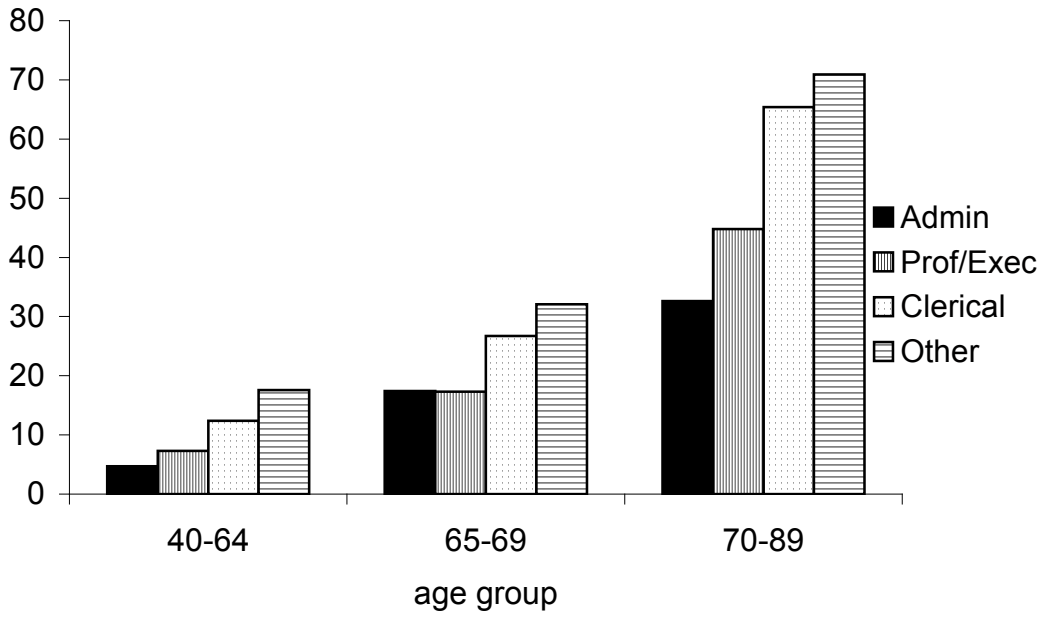
2 Research questions

2.1 The social determinants of health

If the social environment is an important cause of health, this is likely to be manifested as social inequalities in health. People from better social environments with greater access to socio-economic resources are likely to have better health. Supporting this view, social inequalities in health have been documented for most countries, for most causes of deaths and diseases, and in most age-groups. People from lower socio-economic backgrounds are more likely to be unhealthier and have lower life expectancies, even in the richest countries. In Figure 1, from the first Whitehall study on the health of civil servants in the United Kingdom (Marmot and Shipley 1996), men in the lowest, office support employment grades have mortality rates four times that of men in the highest administrative grade in the youngest age-group. This difference in mortality between hierarchies in the civil service remains

even after retirement among men in the oldest age group. What remains unclear are the pathways leading from the social structure to health- or the social determinants of health.

Figure 1



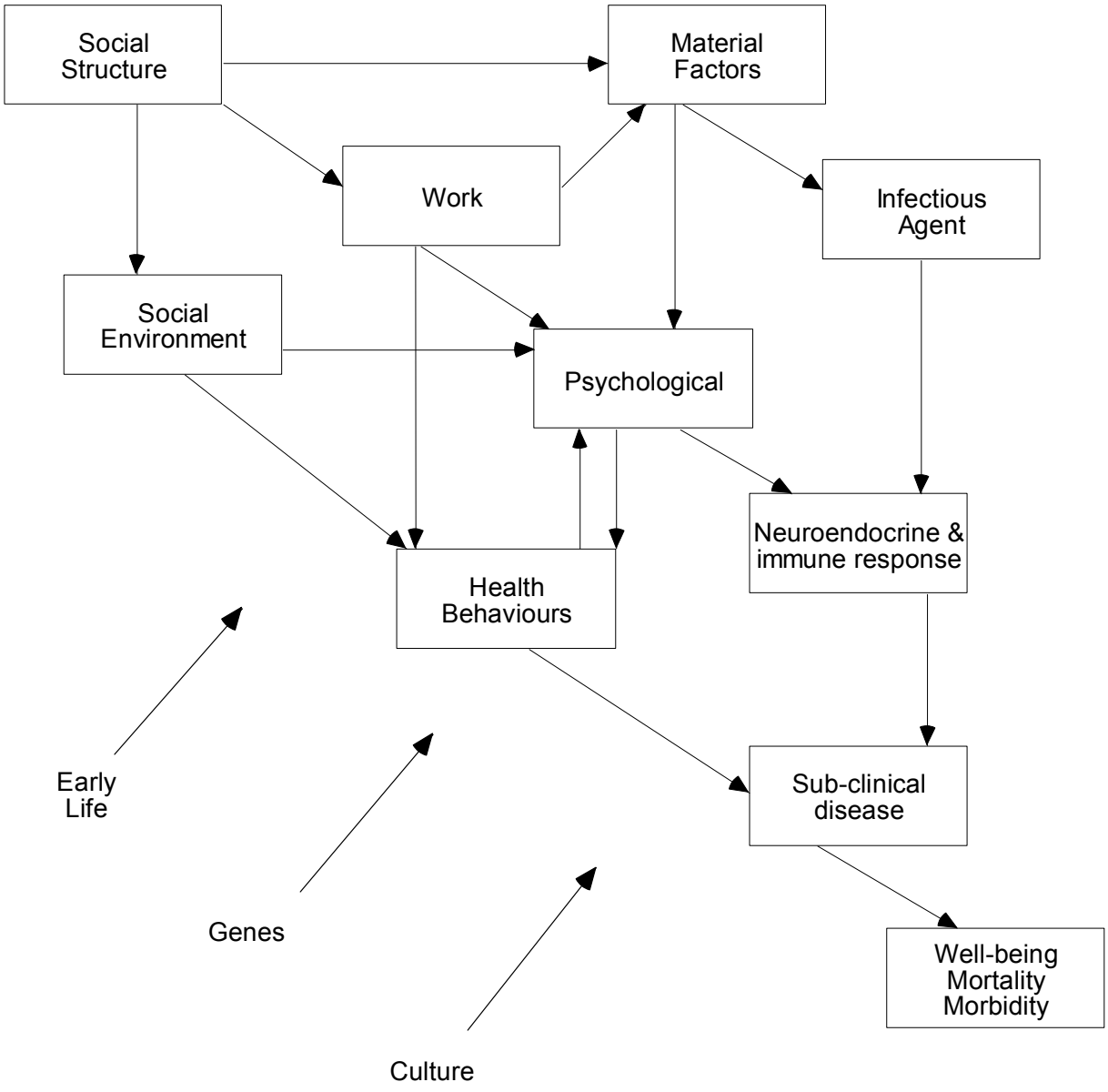
All cause mortality per 1000 person years by employment grade: Whitehall men, 25 year follow up (from Marmot and Shipley 1996)

There have been a number of attempts to delineate the pathways underlying the social determinants of health. One such example is illustrated in Figure 2 (from (Marmot and Wilkinson 1999). Social structure, top left of Figure 2, influences well-being and health, at the bottom right. The influences of the social structure operate via three main pathways- material factors, work and the social environment. While material factors such as poverty and unhygienic circumstances may be directly related to disease through infectious agents, the social and work environments may affect health though psychological and behavioural pathways, which in turn have biological consequences for well-being, morbidity and mortality. Work environments may also affect health through hazardous material working conditions such as radiation or

chemical/biological hazards. There has been relatively little testing of the pathways between social structure and health, primarily because to date, there have been few data available to test these pathways. However, a few studies have examined some of these pathways and their contribution towards understanding social inequalities in health. The rest of this section of the book chapter will highlight some of the research on the search for the social determinants of health.

Figure 2

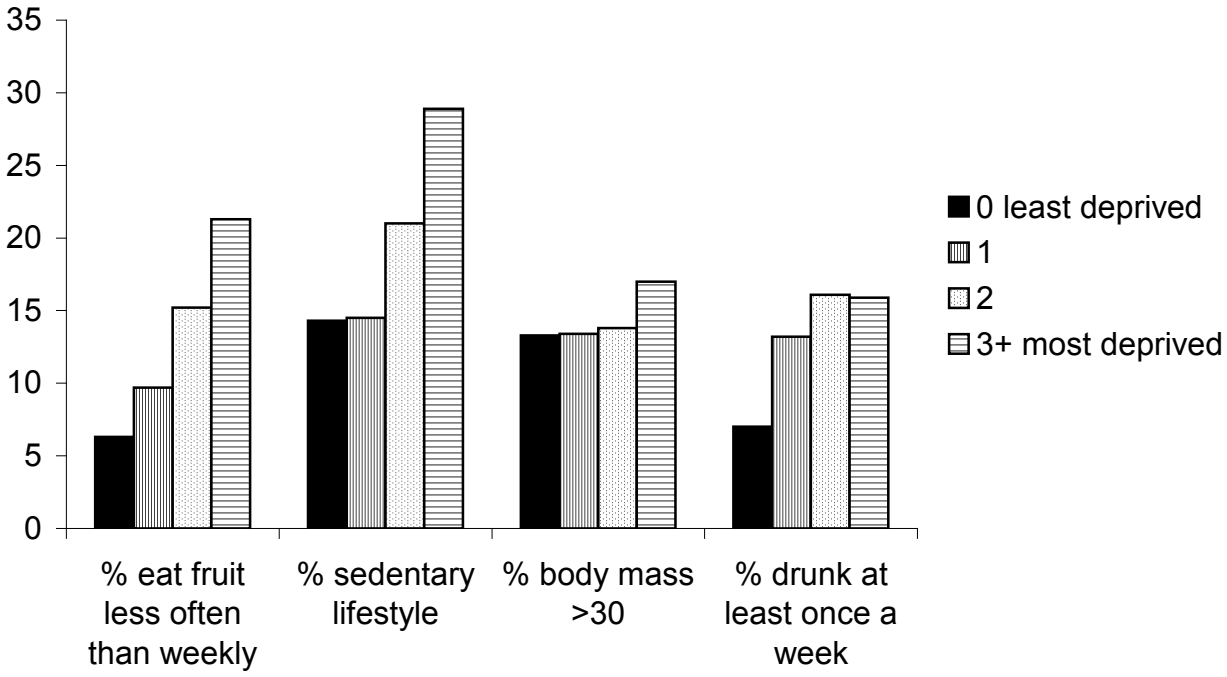
The Social Determinants of Health



2.2 Health behaviours

People from lower socio-economic groups are more likely to smoke, drink alcohol excessively, have less physical exercise and unhealthier diets. It is likely that such unhealthy behaviours form part of the pathways underlying social inequalities in health. Poor people in the UK are less likely than those who are well off to eat a good diet, more likely to have a sedentary lifestyle, more likely to be obese, and more likely to be regularly drunk (Figure 3, from (Colhoun and Prescott-Clarke 1996). Some studies have analysed the contribution of such health behaviours to explaining the social gradient in health and have found that a substantial social gradient in health still remains even after adjusting for such (un)healthy lifestyles (Marmot *et al* 1978). So there may be other social determinants of health not directly related to health behaviours, as suggested by the pathways through work and material factors in Figure 1. Some evidence of these other pathways (shown in Figure 2) is discussed in sections 2.3 to 2.7.

Figure 3



Distribution of some health behaviours among men by level of soio-economic deprivation
(from Colhoun and Prescott-Clarke: Health Survey for England 1994)

Lynch et al. (Lynch *et al* 1997) argue that we still need to understand why poor people behave poorly. Without some understanding of how the social environment influences behaviour (through, for example, social norms or environments which may be health damaging or health promoting such as workplace restrictions on smoking or stressful environments for which smoking may be an effective, albeit temporary coping strategy), interventions to modify behavioural risk factors may not be successful.

2.3 Material, economic and political determinants of health

The link between health and material or socio-economic circumstances has been observed at least since mid 19th century Britain, if not earlier. Chadwick (Flinn 1965) wrote about how overcrowding, damp and filthy living conditions contributed to the lower life expectancy of working class men. In 1848, partly through fear of cholera and partly through pressure from Chadwick, the British parliament passed the first Public Health Act. This, in addition to the pioneering work of the epidemiologist John Snow (Snow 1855), set in motion the public health movement in 19th century Britain which saw improvements in housing, sewage and drainage, water supply and contagious diseases and provided Britain with the most extensive public health system in the world.

It has also been argued that much of the decrease in the mortality rate in the 19th and early 20th century was primarily due to better nutrition in the population which led to increased host resistance to opportunistic infections (McKeown 1979). The driver behind better diets in the general population can be traced to economic growth which made nutritious foods more easily affordable by most of the population. Others, like Szreter (Szreter 1988), argue that the public health movement of the mid 19th century in the UK also played an important role in combating deaths due to infectious disease. It is likely that a combination of macro-economic factors (economic growth) and public policies (public health measures) led to the overall decreases in mortality rates due to infectious diseases and increases in life expectancy.

In 20th century industrialised societies, infectious diseases played an increasingly smaller role in causing deaths while chronic diseases such as heart disease and cancers caused the majority of deaths. Although people from poorer social classes are more susceptible to such chronic diseases (repeating the patterns of infectious diseases like cholera in 19th century Britain), the mechanisms underlying this social patterning of chronic diseases are not easy to specify. A single infectious agent such as a bacterial agent which thrives in unhygienic circumstances is unlikely to account for why poorer, less advantaged people have more heart attacks.

Some authors argue that, even today, economic and political processes are the fundamental determinants of health and disease (Coburn 2000, Navarro and Shi 2001). Determinants of health can be analysed in terms of who benefits from specific government policies and practices. Economic and political institutions and decisions that create, enforce and perpetuate social inequality also create and maintain social inequalities in health. For example, neo-liberal (market oriented) policies which favour the dismantling of the welfare state may help to widen existing social inequalities in health. Navarro and Shi (2001) found that countries with more economic and social redistributive policies (Sweden, Finland, Norway, Denmark and Austria) were more successful in improving the health of their populations (reducing their infant mortality rate). In contrast, neo-liberal countries (Canada, United States, United Kingdom, Ireland) where the market reigns supreme and the welfare state is the weakest had the lowest rates of improvements in the infant mortality rates. The substantial decline in life expectancy in Russia in the 1990s has been linked to its transition to a neo-liberal economy (Walberg *et al* 1998).

2.4 Life course

The idea that a person's experiences over a life time can have a cumulative effect on their health is a central idea within social epidemiology. The study of long term effects of physical and social exposures during gestation, childhood, adolescence, young adulthood and later adult life on the risk of chronic disease has been defined as a life course approach to chronic disease epidemiology (Ben-Shlomo and Kuh 2002).

Such studies include biological, behavioural and psychosocial pathways that operate over an individual's life course, as well as across generations, to influence the course of chronic disease. However, it is only in fairly recent years that adequate data and appropriate statistical methods have been made available to test the hypotheses associated with a developmental and life course perspective.

There are three different ways in which factors from early life might influence subsequent disease risk (Power and Hertzman 1997). The first is a latency model of early life experiences which hypothesises that experiences in utero and early life affect cardiovascular disease in adulthood (Barker 1991, Barker 1997). Barker found evidence that birthweight and other indicators of fetal growth in the newborn are related to fibrinogen and insulin resistance fifty years later. He also found that birthweight is related to functioning of the hypothalamic-pituitary-adrenal axis. Low birthweight is associated with poorer childhood health which some researchers have linked to lower social position in adulthood (Illsley 1986). This evidence suggests that a short term exposure in utero can have a long latency period with adverse health and social consequences in adulthood.

Another theory of the life course suggests that the accumulation of social advantage and disadvantage throughout the life course affects adult health (Ross and Wu 1995). Studies that have examined social circumstances in childhood and beyond do show an effect of social advantage throughout the lifetime (in childhood, early adulthood and later adulthood) on blood pressure, obesity (Wadsworth 1997) and measures of health status. A third life-course pathway is one in which childhood circumstances may not affect adult risk of ill health and disease directly. It is possible that parental social class and educational qualifications are important because they help to determine the social circumstances in which the offspring lives and works in adult life, and it is these circumstances that give rise to social inequalities in disease. Some studies have found the relationship of education on adult health can be explained in terms of occupational class and income((Dahl 1994, Davey Smith *et al* 1998). Although other studies have found the relationship of education to health remains strong even after controlling for occupational class and income (Winkleby *et al* 1992).

2.5 Social biology

Human beings are both social and biological and understanding the interaction between the two is crucial to understanding the social determinants of health. The biological processes that underlie the social determinants of health makes explicit the pathways from psychosocial factors to biological responses. Psychosocial factors may affect health in two distinct ways- they may directly cause biological changes which predispose to disease, or they may, indirectly, influence behaviours such as smoking and diet, which in turn affects health (Brunner 2000).

The direct effect of psychosocial factors on biology may be through the experience of chronic stresses which in turn modify neuroendocrine and physiological functioning (Selye 1956). Humans are adapated to meet the challenge of short-term threats. However, frequent and prolonged activation of the fight-or-flight, or defence, reaction appears to be maladapted (Sapolsky 1993). The main axes of the neuroendocrine response appears to be the sympatho-adrenal and hypothalamic-pituitary-adrenal (HPA) systems (Brunner 2000). The former, the sympatho-adrenal system is characterised by the rapid release of adrenaline from the adrenal medulla and noradrenaline from the sympathetic nerve endings, which produces among other things, cognitive arousal, raised blood pressure and glucose mobilisation. There is evidence of wide variations between individuals in the size and duration of these endocrine responses attributed to individual differences in psychological coping resources (Grossman 1991). The HPA system involves cortisol release from the adrenal cortex . Like the sympatho-adrenal system, functioning of the HPA axis also appears to be conditioned by psychosocial factors (Hellhammer *et al* 1997). Lower social position is associated with prolonged elevations or cortisol release or blunted responses from a raised baseline (Suomi 1997). These patterns of cortisol secretion differ from the normal sharp response and rapid return to a low baseline. A comparison of Swedish and Lithuanian men given a stress test revealed higher morning cortisols and blunted reactivity among the low-income group drawn from the higher coronary risk Lithuanian population (Kristenson *et al* 1998).

There is some evidence for the hypothesis that psychosocial factors directly affect neuroendocrine mechanisms which result in social inequalities in coronary heart

disease. Hostility and anxiety have been linked with reduced heart rate variability (HRV) which refers to the beat-to-beat alterations in heart rate (Hemingway *et al* 1998). HRV appears to be sensitive and responsive to acute stress as well as a marker of cumulative wear and tear. HRV has been shown to decline with the aging process which has been attributed to a decrease in efferent vagal tone and reduced beta-adrenergic responsiveness. By contrast, regular physical activity (which slows down the aging process) has been shown to raise HRV, presumably by increasing vagal tone.

The metabolic syndrome is a well-known precursor state to coronary heart disease (CHD) and is linked with increased risk of type-2 diabetes. The main components of the metabolic syndrome are impaired glucose tolerance, insulin resistance, and disturbances of lipoprotein metabolism characterised by raised serum triglycerides and low HDL cholesterol.(Folsom *et al* 1989, Seidell *et al* 1990) Although the link between the metabolic syndrome and CHD is well-established, the association between psychosocial factors and the metabolic syndrome is less certain. Central obesity and other components of the metabolic syndrome are consistently related to low socio-economic position in industrialised countries (Brunner *et al* 1993, Kaplan and Keil 1993) It is possible that chronic psychosocial stresses result (directly) in the metabolic syndrome pattern of abnormalities through the activation of the HPA axis. Increased HPA activity results in redistribution of body fact leading to central obesity, hypertension and type 2 diabetes as found in Cushing's syndrome (Howlet *et al* 1985). The alternative explanation is that psychosocial stresses lead to unhealthy behaviours (smoking, inappropriate diets). However, in the Whitehall II cohort, adjusting for health behaviours did not change the social gradient in the metabolic syndrome, suggesting a direct neuroendocrine effect (Brunner *et al* 1997).

Infectious disease may also contribute to social differences in morbidity. Helicobater pylori infection, acquired in childhood, is linked with deprivation and over-crowded housing, and may produce long-term low level systemic inflammatory responses which enhance atherogenesis. In Whitehall II, employment grade and chronic low control at work are linked to raised fibrinogen (Brunner *et al* 1995) raising the possibility that inflammatory processes may mediate the effect of psychosocial circumstances on CHD.

2.6 Ecological perspectives

In the UK and elsewhere, there are marked differences in health between areas. People living in areas with higher levels of poverty have poorer health on average and lower average life expectancy. However, explanations for these area differences in health remain debatable. Some argue that excess mortality in deprived areas can be wholly explained by the concentration of poorer people in those areas (Slogett and Joshi 1994, Duncan *et al* 1993). In other words, the compositional or aggregate effect of poor individuals (each of whom has lower than average life expectancy) in an area explains the lower average life expectancy for the area. Others argue that such compositional effects cannot entirely explain area differences in health (Diez-Roux *et al* 1997). They point out that even after adjusting for the composition of individuals living in an area (such as their income and wealth levels), significant area differences in health remain. They argue that there may be contextual or ecological reasons for area differences in health. There may be particular characteristics of an area such as its pollution levels or its lack of medical services which may have an impact on the health of everyone living in that area (Macintyre *et al* 1993). Research from the United States has found that states with lower levels of trust have higher rates of violent crime, including homicide (Kawachi *et al* 1999a). Such contextual effects may also interact with an individual's characteristics and this combined interaction may alter their risk of disease. For example, the lack of medical services may have a greater impact on the health of poor people living in an area compared to richer people who may have the resources to travel or access medical services outside their local area Such ecological or contextual characteristics clearly form part of the social determinants of health and may play some role in explaining social inequalities in (individual) health.

Ecological approaches were disfavoured for many years in social epidemiology (Macintyre and Ellaway 2000). Although public health practioners in the 19th and early 20th centuries focused on dealing with health damaging and promoting environments such as sewage, clean water, housing and physical working conditions, the decline in infectious diseases led to less emphasis being placed on such ecological

factors. The rediscovery of social inequalities in health towards the end of the 20th century focussed primarily on the role of individual health-risk factors such as behaviours, low income, lack of employment and education and a relative neglect of contextual or environmental determinants of health. This neglect has been explicitly addressed in the most recent literature with multilevel analyses that explicitly take into account compositional and contextual social factors that affect health (Macintyre and Ellaway 2000).

2.7 General susceptibility to disease

According to the general susceptibility hypothesis (Syme and Berkman 1976), social factors influence disease by creating a vulnerability or susceptibility to disease in general rather than to any specific disorder. This idea was built on the observation that many social conditions are linked to a broad range of diseases. While behavioural, environmental, biological and genetic factors influence specific diseases, these factors may interact with socially stressful conditions in the development of these diseases resulting in illness and early mortality.

As discussed above, research from social biology shows that some stressful experiences activate multiple hormones, affecting multiple systems and potentially producing wide-ranging organ damage. The cumulative experience of stress may affect a variety of chronic and infectious diseases through neuroendocrine-mediated biological pathways. There are a number of different sources of stressful experiences, some of which are discussed below. The linking of such stressful experiences (often measured using psychological concepts) to wider social circumstances has been called a psychosocial approach to understanding the social determinants of health.

2.7.1 Social support

The effect of social support and social networks on health has been researched (at least) since the late 19th century when Durkheim investigated the links between social integration and suicide. He explained suicide in terms of social dynamics, arguing that suicide is not an isolated individual tragedy but a reflection of social conditions

such as the lack of attachment and regulation in society. Attachment is also a core concept for Bowlby (Bowlby 1969) who argued that marriage is the adult equivalent of childhood attachment between mother and child. Secure attachment, whether in terms of parent-child or marital relationships, provides for successful and healthy development. Men who have never married or have recently divorced have a significantly greater risk of dying from both cardiovascular and non-cardiovascular diseases than married men (Ebrahim *et al* 1995). Married women are generally healthier than unmarried women as well, although the health benefits of marriage may not be particularly strong for employed women (Waldron *et al* 1996).

Throughout the 1970s and 1980s, a series of studies appeared which consistently showed that the lack of social ties or social networks predicted mortality from almost every cause of death (Berkman 1995). Social ties and networks were measured in terms of numbers of close friends and relatives, marital status, and membership in religious or voluntary associations. Since then, studies have gone on to focus on the provision of social support rather than on the elaboration of the structural aspects of social networks. Not all social ties or networks are supportive and there is variation in the type, frequency and extent of support provided. Social support, in theory, can be divided into emotional support (usually provided by a confidant or intimate other), instrumental support (or help in kind, money or labour), appraisal support (help in decision making) and informational support (provision of advice or information). Lack of emotional support has been linked to early cardiovascular disease mortality among both men and women, younger and older people (Berkman *et al* 1992). Other studies have found that social integration, particularly operating though emotional support, influence recovery from strokes (Berkman and Glass 2000).

2.7.2 Social disorganisation

Social scientists have puzzled over why some societies seem to prosper, possess effective political institutions and have better health outcomes compared to other societies. One of the hypotheses that has been proposed to explain this difference between societies is the amount of social capital or cohesion (and its converse- social disorganisation) in a particular society (Coleman 1988, Putnam 2000). Social

cohesion refers to the extent of connectedness and solidarity among groups in society. A cohesive society has greater amounts of social capital (higher levels of interpersonal trust, reciprocity and mutual aid) than a disorganised society. There is emerging evidence that greater social capital is linked to lower mortality rates as well as better self-rated health (Kawachi and Berkman 2000). As mentioned in section 2.6, states in the United States with lower levels of trust have higher homicide rates (Kawachi *et al* 1999a). Even after adjusting for individual risk factors for poor self-rated health (e.g. low income, low education, smoking, obesity, lack of access to health care), individuals living in US states with low social capital were at increased risk of poor self-rated health (Kawachi *et al* 1999b). Such results suggest that there are contextual explanations for area differences in health as discussed in the section on ecological perspectives.

Social capital may be linked to health through a number of different mechanisms. We have already discussed two types of explanations for understanding area differences in health- compositional and contextual explanations. Socially isolated individuals (not having contacts with friends or relatives, not belonging to any groups) are more likely to be living in communities with lower social capital so the association between social capital and health may be the compositional effect of the aggregation of socially isolated individuals. However, there may be other pathways by which social capital affects health (Kawachi and Berkman 2000):

1. Through health related behaviours. Social capital may influence the health behaviours of neighbourhood residents by exerting social control over deviant behaviours such as adolescent smoking, drinking and drug abuse.
2. Through access to services. Socially cohesive neighbourhoods are more successful at organisation access to services such as transport, health services and recreational facilities
3. Through psychosocial processes. Socially disorganised neighbourhoods with low social capital could have higher levels of fear of crime and other stressors which could negatively impact on the residents' health.

2.7.3 Work stress

One of the more established results in epidemiology has been the link between physical working conditions and health. Reports on occupational health have highlighted the link between emphysema and other lung disease with coal mining, musculoskeletal disorders and accidents with certain types of manual work. In recent years, there has been increased research on work related stress and how that affects both physical and mental health.

There are two dominant models of work stress in the literature. The first, the job strain model is based on the concepts of job control and demands (Karasek *et al* 1981). Workers with low levels of job control and high levels of demand are said to have high levels of job strain (or work stress). Job control (or decision latitude) consists of whether or not workers are able to utilise and develop skills (skill discretion) and their authority over decisions. Job demands consist of qualitative emotional demands as well as quantitative demands specifying output per unit of time. Prolonged and repeated exposure to job strain is hypothesised to increase sympathadrenal arousal and decrease the body's ability to restore and repair tissues which in turn affects health. Civil servants in the UK with greater exposure to job strain and lower job control have higher levels of fibrinogen (Brunner *et al* 1996), which may result in their higher risks of coronary heart disease (Bosma *et al* 1997).

The other model of work stress, the effort-reward imbalance model (Siegrist 1996), hypothesises that the degree to which workers are rewarded for their efforts is crucial for their health. When a high degree of effort does not meet a high degree of reward, emotional tensions arise and the risk of illness increases. Effort is the individual's response to their job demands and this response may be extrinsic effort (referring to the individual's effort to cope with external job demands) and intrinsic effort (referring to the individual's drive to fulfil their goals). Reward can be measured through financial rewards, self-esteem and social control. While there is some overlap between the job strain and effort reward imbalance models, the former is entirely focussed on the organisation of the structure of work while the latter includes the individual's way or coping methods of handling difficulties (through the concept of intrinsic effort).

There is some evidence that both models of work stress contribute independently of one another to predicting coronary heart disease events (Bosma *et al* 1998). The cumulative adverse health impact of low job control and effort-reward imbalance indicates that both job stress factors provide supplementary information on the relevant stressors in the psychosocial work environment.

2.7.4 Unemployment and job loss

There has been considerable research into the effects of unemployment and job loss on health. However, this is an area of research that is particularly sensitive to the claims of 'health selection', that the reason why unemployment is associated with ill health is because ill health selects people out of employment. The reverse argument is that a disadvantaged socio-economic position has an effect on a stable job career (and the risk of unemployment) as well as health. It is therefore important to disentangle the causal narrative in studies about unemployment and health and find out which comes first.

The evidence on unemployment and health supports both the social causation and health selection interpretations. In a review of the effect of unemployment on health, Kasl and Jones (Kasl and Jones 2000) summarised the evidence as follows:

1. Unemployment is associated with a 20-30% excess in all cause mortality in most studies
2. There is some evidence of the impact of unemployment on physical morbidity but with results that are more difficult to interpret
3. Unemployment is linked to biological indicators of stress reactivity
4. Unemployment is associated with behavioural and lifestyle risk factors although the direction of causality is hard to disentangle
5. Unemployment clearly increases psychological distress
6. Threatened job loss (job insecurity) is associated with physical and psychological morbidity and cardiovascular risk. The anticipation of job loss affects health even before changes in employment status (Ferrie *et al* 1995)

2.7.5 Depression and affective states

Depression is one of the most common psychiatric problems and is also common in patients with chronic medical conditions. Some depressive episodes are brought upon by physical illness, but many depressive patients have depressive episodes long before they develop any physical symptoms of illness. Furthermore, depression may alter the course and outcome of physical illness (Carney and Feedland 2000).

Depression has been associated with immunological dysfunction. Patients with major depression have been found to have blunted natural killer cell activity (Maes *et al* 1994) increasing their risk for many acute and chronic illnesses. There is also some evidence that depression may play a causal role in the development of heart disease. There is some evidence of a social gradient in depression in a healthy, working population- it appears to be more common among those from poorer, more disadvantaged social positions (Stansfeld *et al* 1998) and may originate from their lower control over aspects of their work and home environment (Griffin *et al* 2002).

Another set of psychological pathways by which social conditions may affect health is through emotions and the physiological, cognitive and behavioural responses they evoke. Emotions may be transitory states brought on by specific situations, or traits, i.e. stable and general dispositions to experience particular emotions (Spielberger and Krasner 1988). Much of the research on emotion and health has been carried out in relation to coronary heart disease. Much of this literature has focussed on type A behaviour, (which includes a free-floating but well rationalised hostility, hyperaggressiveness and a sense of time urgency), chronic anger and hostility, anxiety and a mixture of emotions associated with depression including hopelessness, loneliness, guilt and shame (Kubzansky and Kawachi 2000).

There is some evidence of a social patterning of emotions (Bradburn 1969, Mroczek and Kolarz 1998). Kemper (1993) suggests that many emotions are responses to power and status differentials embedded within social situations. Potentially stressful events can be associated with a variety of different emotions. Emotions can be considered as products of stress as well as mediators of its effects thus representing a crucial link in the chain of causation from social stressors to individual biological

responses (Spielberger and Krasner 1988). Evidence from animal studies suggest that additional to hypothalamic control of the stress response, areas of the brain involved with emotional or affective responses such as the limbic system also play a major role in stress responses (Menzhaghi *et al* 1993) and adaptation to the stress response (Sapolsky *et al* 1986).

3 Research methods

3.1 Applying a population perspective

Rose (1992) proposed that an individual's risk of illness cannot be considered in isolation from the risk of disease of the population to which they belong. For example, the distribution of cholesterol levels in the Finnish population is shifted to the right of the Japanese distribution- on average, Finnish people have higher cholesterol levels than Japanese people. The level of 'normal' cholesterol for the Finnish population would be 'abnormal' for the Japanese population and would be a risk factor for CHD in the latter population. Applying the population perspective into epidemiological research means asking "why does this population have this particular distribution of risk factors", in addition to asking "why did a particular individual get sick?" (Berkman and Kawachi 2000). Answering the second question has been the focus of clinical medicine while answering the first question is the key to the largest improvements in the health of the population as it focuses attention on the majority of cases of illness within the bulk of the population. Medical care can prolong survival after some serious diseases, but the social and economic conditions that affect whether people become ill are more important for health gains in the population as a whole.

3.2 Better measures of exposures

There is no simple relationship between social-structural conditions such as income distribution and welfare state regimes on the one hand and health inequalities in the population on the other. The different pathways by which different social factors can have an effect on different health outcomes implies that there is no single measure of

social factors, health outcomes or single pathway between the two that can adequately represent the complexity of the associations between the social structure and health. One of the ways of advancing our knowledge of the social determinants of health is by utilising better measures of the social structure, the health outcomes as well as the pathways that link exposures and outcomes. One of the defining characteristics of research in social epidemiology has been the constant refinement of such measures and improvements in the methodology of measuring complex concepts and associations.

In the UK, the standard epidemiological measure of social class since the start of the 20th century has been the Registrar General's social class (RGSC). However, the RGSC as been heavily criticised by being atheoretical- the basis for classifying people into different social classes has never been made explicit (Szreter 1984). The changeover to a more theoretically based measure of social class- the National Statistics Socio-Economic Classification- based on differences in employment relations and conditions, was prompted in part by research in social epidemiology which found that the RGSC was not useful in understanding the social determinants of health (Bartley *et al* 1996). Other research (Chandola 2001) has similarly argued that the standard epidemiological technique of controlling for social class does not have much meaning, especially when the measure of social class does not adequately represent the different dimensions of the social structure that affect health (such as housing and neighbourhood conditions, labour market conditions, employment relations, household income and social status). In attempting to understand the social determinants of health, research into social epidemiology has pioneered the use of better measures of the social structure.

3.3 Better measures of health

The concept of health is multidimensional (the WHO definition states that health is a state of complete physical, mental and social well-being), including hard to measure concepts like quality of life. Research in social epidemiology does not just focus on clinically measured disease outcomes because the absence of disease is not sufficient for health. Rather, one of the main focuses of social epidemiological research is the

use of health related quality of life measures as valid measures of health outcomes (Fitzpatrick *et al* 1992). Population mortality statistics tell us little about the health of general populations in developed countries. The use of standardised health related quality of life measures in different countries (Ware and Gandek 1998) enable international comparisons of physical, mental and social well-being.

Subjective health status covers a wide variety of areas, including role functioning (e.g. the ability to perform domestic and work tasks), the degree of social and community interaction, psychological well being, pain, tiredness and satisfaction with life (Bowling 1997). Health related quality of life has come to mean a combination of subjectively assessed measures of health, including physical function, social function, emotional or mental state, burden of symptoms and sense of well being (Coulter 1993). The development and use of such subjective measures of health status and health related quality of life, have been one of the defining aspects of social epidemiology.

3.4 Better measures of the association between the social structure and health

As different measures of the social structure may have different pathways to different health outcomes, the reduction of such differences into a single regression model may obscure rather than elucidate the pathways underlying the social determinants of health. Furthermore, different dimensions of the social structure may influence people's health at different time points of the life-course. For example, in industrialised societies, the period of the life-course when compulsory education is completed may be a crucial time for the health of the population, not because young adults are at a particular high risk of disease or illness at that stage in life, but because educational qualifications are a strong determinant of social position in later adult life which in turn appear to be strongly linked to health outcomes later on in life. It is important to take account of the temporal and causal ordering of the various measures of social position and use methods that make explicit the various underlying causal pathways between different measures of social position and health. There are a number of such causal modelling methods being used in social epidemiological

research (Greenland and Brumback 2002). Failure to take account of the different pathways between the social structure and health outcomes could result in biased results (Singh-Manoux *et al* 2002).

3.5 Analysing population surveys, birth cohorts

One of the defining characteristics of research methods in social epidemiology is the use of population representative sample surveys in analysing the social determinants of health. Research in social epidemiology tends to use non-experimental observational studies, both cross sectional and longitudinal. All observational studies suffer from problems of causality- it is hard to determine and separate out cause from effect (see the chapter by Rothman in this book). This drawback has necessitated the use and development of complex study designs and analytical methods to disentangle the causal pathways underlying the social determinants of health.

Studies with good methodological designs (for example, (Ferri *et al* 2003)) in social epidemiology tend to rely on data from large scale population representative sample surveys because of the complexity of the social structure and the different pathways to health. The representivenss of data is crucial in order to apply a population perspective in social epidemiological research. Smaller scale samples may not be representative of the broader population.

Birth cohort studies are a special type of such large scale population representative samples which incorporates a life course approach to epidemiology. The UK has taken a prominent role in the development of such longitudinal studies. The British Birth Cohort Studies of those born in one week of 1946, 1958 and 1970 link data from one part of the life course (from birth onwards) to another (childhood, adolescence, adulthood) for a large number of individuals. Comparisons between different birth cohort studies enable the disentangling of age, period and cohort effects, which could be problematic when analysing most cross-sectional and even longitudinal sample surveys. For example, in the book ‚Changing Lives, Changing Britain' (Ferri *et al* 2003), *cohort* effects that might be attributed to socio-economic change impacting differentially on people born at different times, can be differentiated from *age*

differences reflecting the different changes between the stages of life, which in turn can be differentiated from the prevailing socio-economic context at the time of data collection - the *period* effect. Such analyses of this unique set of longitudinal data, incorporating a life-course perspective, is very promising for future research into social epidemiology.

4 Setting government policy agenda

One of the goals of epidemiology has always been to use what we learn to improve public health. The science of social epidemiology has repeatedly shown evidence that social conditions are a major determinant of health. However, the translation of the research findings of social epidemiology into public policy has not been straightforward. Unlike results from some branches of epidemiology which can be more easily implemented into government guidelines (such as recommended alcohol intake) or public policy (reduction in smoking prevalence), programs to implement findings from social epidemiology need to take into the account the complexity of the pathways from the social structure to health.

Some authors argue that policy interventions are most effective when they are closest to the root causes of disease (Rothman *et al* 1998). Interventions at the upstream, social level may not be as efficient as interventions closer to disease occurrence. So, for example, policy interventions on reducing the social gap in smoking-related diseases, should focus on interventions on smoking cessation rather than interventions on the social causes of smoking. Others argue (such as (Coburn 2000), mentioned in the section 2.3) that interventions need to be upstream, at the societal and macro-economic level, in order to successfully reduce health inequalities.

4.1 The Black and Acheson reports

The Black report (DHSS 1980) into inequalities in health in the UK had a number of wide-ranging policy recommendations for reducing such inequalities. However, the lack of implementation of these policies by the British government in the 1980s and

early 1990's was due, in part, to a lack of political will and the high cost of these policy recommendations.

The change in government in Britain in 1996 (from Conservative to Labour) paved the way for the publication of the Acheson Report (Acheson 1998) on inequalities in health in 1998 with another list of recommendations for reducing health inequalities. What makes this publication unique is the acceptance by the UK government that some action was needed to reduce inequalities in health. For example, the UK government department of health has subsequently adopted targets on reducing inequalities in health (such as closing the social class gap in infant mortality rates). Here is some evidence that research in social epidemiology is being translated into government policy.

However, the policies that have been developed to reduce such health inequalities focus on reducing social inequalities in general (through income redistribution policies for example). The very fact that social epidemiology deals with the social structure necessitates policies aimed at changing the social structure. Such policies are not always easy to specify and detail. Furthermore, the diffuse ownership of such policies between government departments (such as education, health and treasury departments) makes their implementation harder. In recognition of the complexities of policies aimed at reducing health inequalities, the UK government set up a cross cutting spending review (across various government departments) on tackling health inequalities. This report (Department of Health 2002) explicitly acknowledges that policies on reducing health inequalities need to be co-ordinated across a wide range of government departments and bodies (not just the national health service), including local government and health organisations and sets in process the institutional framework for such co-ordination.

4.2 Collating evidence for policies through intervention studies and cross national comparative studies

One of the ways of ensuring appropriate policies for reducing inequalities in health are implemented is by studying the results from intervention studies. However, social epidemiological research does not easily lend itself to intervention studies, mainly

because the complexity of the social structure makes it hard to disentangle the pathways to reductions in health inequalities. For example, it is hard to disentangle changes from behavioural change interventions from secular trends in society. (Susser 1995). It is also difficult to separate out the influence of secondary support (from support groups organised around behavioural interventions) from the intended influence of the behavioural intervention (Spiegel *et al* 1989). Social support interventions have had mixed results partly because as relationships develop and change slowly, the benefits of support interventions may be missed in the short-term (Glass 2000).

Another method of analysing policy recommendations for health inequalities is through international and longitudinal comparisons of health inequalities. Changes in taxation and income redistribution policies within a country may be hypothesised to have an effect on health inequalities. Furthermore, cross national longitudinal comparisons of different tax policies and their effect on health inequalities may be another way of analysing the effect of policies on health inequalities (Navarro and Shi 2001). However, to date, there has been little research in this area which means that current policies on reducing inequalities in health may not be entirely appropriate or well targeted.

5 Conclusion

Perhaps, the major contribution of social epidemiology to epidemiology in general has been in rediscovering and analysing the role of social factors in producing health and illness. This has primarily come about by the literature on social inequalities in health and consequently, research into the social determinants of health. The search for the pathways between the social structure and health has led to innovations in longitudinal research methodology. While social epidemiology shares common epidemiological problems of reliance on observational studies and problems in interpreting causality, the incorporation of a life-course perspective by analysing and comparing birth-cohort studies holds great promise for future studies. Research into social epidemiology has influenced wide ranging government social policies, because

of the macro-societal level interventions that are needed to reduce inequalities in health.

Although there is some debate over the usefulness of the specialisation of social epidemiology within the medical sciences (Zielhus and Kiemeney 2001), others have argued that the overall contribution of social epidemiology towards understanding current and changing distributions of population health have been striking (Krieger 2001, Muntaner 2001). The interdisciplinary nature of social epidemiology has led to the incorporation of research questions, methods and policy agendas that have enriched our understanding of the social determinants of health.

References

Bowling, A (1997), *Measuring Health: A Review of Quality of Life Measurement Scales*, Open University Press, Milton Keynes

Acheson D (1998), *Inequalities in Health: Report of an independent inquiry*, HMSO, London

Barker DJP (1991), The Foetal and infant origins of inequalities in health in Britain, *Journal of Public Health Medicine* 12 Number 2: 64-68

Barker DJP (1997), Fetal nutrition and cardiovascular disease in later life, *British Medical Bulletin* 53: 96-108

Bartley M, Carpenter L, Dunnell K, Fitzpatrick R (1996), Measuring Inequalities in Health: An Analysis of Mortality Patterns from Two Social Classifications, *Sociology of Health and Illness* 18: 455-475

Ben-Shlomo Y, Kuh D (2002), A life course approach to chronic disease epidemiology: conceptual models, empirical challenges, and interdisciplinary perspectives, *International Journal of Epidemiology* 31: 293

Berkman LF (1995), The role of social relations in health promotion, *Psychosomatic Medicine* 57: 245-254

Berkman LF, Glass T (2000), Social integration, networks and health, in *Social Epidemiology*, ed. Berkman LF and Kawachi I, OUP, New York p 137-173

Berkman LF, Kawachi I (2000), A historical framework for social epidemiology, in *Social Epidemiology*, ed. Berkman LF and Kawachi I, Oxford University Press, New York p 3-12

Berkman LF, Leo-Summers L, Horwitz RI (1992), Emotional support and survival after myocardial infarction. A prospective, population-based study of the elderly, *Annals of Internal Medicine* 117: 1003-1009

Bosma H, Marmot MG, Hemingway H, Nicholson A, Brunner EJ, Stansfeld S (1997), Low job control and risk of coronary heart disease in the Whitehall II (prospective cohort) study, *British Medical Journal* 314: 558-565

Bosma H, Peter R, Siegrist J, Marmot MG (1998), Two alternative job stress models and the risk of coronary heart disease., *American Journal of Public Health* 88: 68-74

Bowlby J (1969), *Attachment and Loss vol. 1 Attachment*, London

Bradburn NM (1969), *The Structure of Psychological Well-Being*, ALDINE, Chicago

Brunner EJ (2000), Toward a new social biology, in *Social Epidemiology*, ed. Berkman LF and Kawachi I, Oxford University Press, New York

Brunner EJ, Davey Smith G, Marmot MG, Canner R, Beksinska M, O'Brien J (1996), Childhood social circumstances and psychosocial and behavioural factors as determinants of plasma fibrinogen, *Lancet* 347: 1008-1013

Brunner EJ, Marmot MG, Nanchahal K, Shipley MJ, Stansfeld SA, Juneja M, Alberti KGMM (1997), Social inequality in coronary risk: central obesity and the metabolic syndrome. Evidence from the WII study, *Diabetologia* 40: 1341-1349

Brunner EJ, Mendall MA, Marmot MG (1995), Past or present Helicobacter pylori infection and fibrinogen - a possible link between social class and coronary risk?, *Journal of Epidemiology and Community Health* 49: 545

Brunner EJ, Nicholson A, Marmot MG (1993), Trends in central obesity and insulin resistance across employment grades: the WII Study. *Journal of Epidemiology and Community Health* 47, 404-405.

Carney RM, Feedland KE (2000), Depression and mental illness, in *Social Epidemiology*, ed. Berkman LF and Kawachi I, OUP, New York p 191-212

Chandola T (2001), Ethnic and class differences in health in relation to British South Asians : using the new National Statistics Socio-Economic Classification, *Social Science & Medicine* 52: 1285-1296

Coburn D (2000), Income inequality, social cohesion and the health status of populations: the role of neo-liberalism, *Social Science and Medicine* 51: 135-146

Coleman JS (1988), Social capital in the creation of human capital, *American Journal of Sociology* 94: S95-S120

Colhoun H, Prescott-Clarke P (1996), *Health Survey for England 1994*, HMSO, London

Coulter A (1993), Measuring Quality of Life, in *Critical Reading in General Practice*, ed. Kinmouth A and Jones R, OUP, Oxford

Dahl E (1994), Social inequalities in ill health: the significance of occupational status, education and income- results from a Norwegian survey, *Sociology of Health and Illness* 16: 644-667

Davey Smith G, Hart CL, Hole DJ, MacKinnon P, Gillis C, Watt G, Blane D, Hawthorne VM (1998), Education and occupational social class: which is the more important indicator of mortality risk?, *Journal of Epidemiology and Community Health* 52: 153-160

Department of Health (2002), *Tackling Health Inequalities: Summary of the 2002 Cross Cutting Review*, Department of Health, London

DHSS (1980), *Inequalities in Health: Report of a Research Working Group Chaired by Sir Douglass Black*, DHSS,

Diez-Roux AV, Nieto FJ, Muntaner C, Tyroler HA, Comstock GW, Shahar E, Cooper LS, Watson RL, Szklo M (1997), Neighborhood environments and coronary heart disease: a multilevel analysis, *American Journal of Epidemiology* 146: 48-63

Duncan C, Jones K, Moon G (1993), Do places matter: a multilevel analysis of regional variations in health related behaviour in Britain, *Social Science and Medicine* 42: 817-830

Durkheim E (1996), *Suicide*, Free Press, New York

Ebrahim S, Wannamethee G, McCallum A, Walker M, Shaper A (1995), Marital status, change in marital status, and mortality in middle-aged British men, *American Journal of Epidemiology* 142: 834-842

Ferri E, Brynner J, Wadsworth M (2003), *Changing Britain, Changing Lives*, Education Press, London

Ferrie JE, Shipley MJ, Marmot MG, Stansfeld S, Davey Smith G (1995), Health effects of anticipation of job change and non-employment: longitudinal data from the Whitehall II study, *British Medical Journal* 311: 1264-1269

Fitzpatrick R, Fletcher A, Gore S, Jones D, Speigelhalter D, Cox D (1992), Quality of life measures in health care. I: Applications and issues in assessment, *British Medical Journal* 305: 1074-1077

Flinn MW (1965), *Chadwick, Edwin. Report on the Sanitary Condition of the Labouring Population of Great Britain. 1842 Ed. & Intro.*, Edinburgh

Folsom AR, Burke GL, Ballew C, Jacobs DR, Haskell WL, Donahue RP (1989), Relation of body fatness and its distribution to cardiovascular risk factors in young blacks and whites, *American Journal of Epidemiology* 130: 911-924

Glass TA (2000), Psychosocial intervention, in *Social Epidemiology*, ed. Berkman LF and Kawachi I, OUP, New York p 267-305

Greenland S, Brumback B (2002), An overview of relations among causal modelling methods, *International Journal of Epidemiology* 31: 1030-1037

Griffin J, Fuhrer R, Stansfeld SA, Marmot MG (2002), The importance of low control at work and home on depression and anxiety: do these effects vary by gender and social class?, *Social Science & Medicine* 54: 738-798

Grossman AB (1991), Regulation of human pituitary responses to stress, in *Stress: neurobiology and neuroendocrinology*, ed. Brown MB, Koob GF, and Rivier C, Marcel Dekker, New York p 151-171

Hellhammer DH, Buchtal J, Gutberlet I, Kirschbaum C (1997), Social hierarchy and adrenocortical stress reactivity in men, *Psychoneuroendocrinology* 22: 643-650

Hemingway H, Shipley MJ, Christie D, Marmot M (1998), Cardiothoracic ratio and relative heart volume as predictors of coronary heart disease mortality: the Whitehall study 25 year follow up, *European Heart Journal* 19: 859-869

Howlet T, Rees L, Besser G (1985), Cushing's syndrome, *Clinics in endocrinology and metabolism* 14: 911-945

Illsley R (1986), Occupational Class, Selection and the Production of Inequalities in Health, *The Quarterly Journal of Social Affairs* 2: 151-165

Kaplan GA, Keil JE (1993), Socioeconomic factors and cardiovascular disease: a review of the literature., *Circulation* 88: 1973-1998

Karasek R, Baker D, Marxer F, Ahlbom A, Theorell T (1981), Job decision latitude, job demands and cardiovascular disease: a prospective study of Swedish men., *American Journal of Public Health* 71: 694-705

Kasl SV, Jones BA (2000), The impact of job loss and retirement on health, in *Social Epidemiology*, ed. Berkman LA and Kawachi I, OUP, New York p 118-136

Kaufman JS, Cooper RS (1999), Seeking causal explanations in social epidemiology, *American Journal of Epidemiology* 150: 113-120

Kawachi I, Berkman LA (2000), Social cohesion, social capital and health, in *Social Epidemiology*, ed. Berkman LF and Kawachi I, OUP, New York p 174-190

Kawachi I, Kennedy B, WIlkinson R (1999a), Crime: social disorganization and relative deprivation, *Social Science and Medicine* 48: 731

Kawachi I, Kennedy BP, Glass R (1999b), Social capital and self-rated health: a contextual analysis, *American Journal of Public Health* 89: 1187-1193

Kemper TD (1993), Sociological models in the explanation of emotions, in *Handbook of Emotions*, ed. Lewis M and Haviland JM, The Guildford Press, New York p 41-52

Krieger N (2001), Commentary: Society, biology and the logic of social epidemiology, *International Journal of Epidemiology* 30: 44-46

Kristenson M, Orth-Gomer K, Kucinskiene Z, Bergdahl B, Calkauskas H, Balinkyiene I, Olsson AG (1998), Attenuated cortisol response to a standardised stress test in Lithuanian vs. Swedish men, *International Journal of Behavioural Medicine* 5: 17-30

Kubzansky LD, Kawachi I (2000), Affective states and health, in *Social Epidemiology*, ed. Berkman LF and Kawachi I, OUP, New York p 213-241

Lynch JW, Kaplan GA, Salonen JT (1997), Why do poor people behave badly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse, *Social Science & Medicine* 44: 809-819

Macintyre S, Ellaway A (2000), Ecological apporaches: rediscovering the role of the physical and social environment, in *Social Epidemiology*, ed. Berkman LF and Kawachi I, OUP, New York p 332-348

Macintyre S, Maciver S, Sooman A (1993), Area, class and health: should we be focusing on places or people?, *J Social Policy* 22: 213-234

Maes M, Meltzer H, Stevens W, Calabrese J, Cosyns P (1994), Immuendocrine aspects of major depression. Relationships between plasma interleukin-1 and soluble interleukin-2 receptor, prolactin and cortisol, *Progress in Neuro-Psychopharmacology & Biological Psychiatry* 18: 717-730

Marmot MG, Rose G, Shipley M, Hamilton PJS (1978), Employment grade and coronary heart disease in British civil servants., *Journal of Epidemiology and Community Health* 32: 244-249

Marmot MG, Shipley MJ (1996), Do socioeconomic differences in mortality persist after retirement? 25 year follow up of civil servants from the first Whitehall study, *British Medical Journal* 313: 1177-1180

Marmot MG, Wilkinson RG (1999), *Social determinants of health*, Oxford University Press, Oxford p 1-291

McKeown T (1979), *The role of medicine: dream, mirage or nemesis?*, Basil Blackwell, Oxford

Menzhaghi F, Heinrichs S, Pich E, Weiss F, Koob G (1993), The role of limbic and hypothalamic corticotropin releasing factor in behavioural response to stress, *Annals of the New York Academy of Sciences* 697: 142-154

Mroczek D, Kolarz C (1998), The effect of age on positive and negative affect: a developmental perspective on happiness, *Journal of Personality and Social Psychology* 75: 1333-1349

Muntaner C (2001), Social epidemiology: no way back. A response to Zielhus and Kiemeney, *International Journal of Epidemiology* 30: 625-626

Navarro V, Shi L (2001), The political context of social inequalities in health, *Social Science and Medicine* 52: 481-491

Power C, Hertzman C (1997), Social and biological pathways linking early life and adult disease, in *British Medical Bulletin. Fetal and early childhood environment:long-term health implications. Vol.53. No.1.*, ed. Marmot M and Wadsworth MEJ, Royal Society of Medicine Press Limited, London p 210-222

Putnam R (2000), *Bowling Alone: The Collapse and Revival of American Community*, Simon and Schuster,

Rose G (1992), *The strategy of preventive medicine*, Oxford University Press, Oxford

Ross C, Wu C (1995), The links between education and health, *American Sociological Review* 60: 719-745

Rothman K, Adami H, Trichopoulos D (1998), Should the mission of epidemiology include the eradication of poverty?, *Lancet* 352: 810-813

Sapolsky R, Krey L, McEwen B (1986), The neuroendocrinology of stress and aging: the glucocorticoid cascase hypothesis, *Endocrine Reviews* 3: 301

Sapolsky RM (1993), Endocrinology alfresco: psychoendocrine studies of wild baboons, *Recent Progress in Hormore Research* 48: 437-468

Seidell JC, Bjorntorp P, Sjostrom L, Kvist H, Sannerstedt R (1990), Visceral fat accumulation in men is positively associated with insulin, glucose, and C-peptide levels, but negatively with testosterone levels, *Metabolism* 39: 897-901

Selye H (1956), *The stress of life.*, McGraw-Hill, New York

Siegrist J (1996), Adverse health effects of high-effort/low-reward conditions, *Journal of Occupational Health Psychology* 1: 27-41

Singh-Manoux A, Clarke P, Marmort M (2002), Multiple measures of socioeconomic position and psychosocial health: proximal and distal effects, *International Journal of Epidemiology*

Slogett A, Joshi H (1994), Higher mortality in deprived areas: community or personal disadvantage?, *British Medical Journal* 309: 1470-1474

Snow J (1855), *On the mode of communication of cholera*, Churchill, London

Spiegel D, Bloom J, Kraemer H, Gottheil E (1989), Effect of psychosocial treatment on survival of patients with metastatic breast cancer, *Lancet* 14: 888-891

Spielberger CD, Krasner SS (1988), The assessment of state and trait anxiety, in *Enological factors and associated disturbances*, ed. Noyes RJr, Roth M, and Burrows GD, Elsevier Science Publishers B.V., Holland p 31-50

Stansfeld SA, Head J, Marmot MG (1998), Explaining social class differences in depression and well-being, *Social Psychiatry and Psychiatric Epidemiology* 33: 1-9

Suomi SJ (1997), Early determinants of behaviour: evidence from primate studies, *British Medical Bulletin* 53: 170-184

Susser M (1995), The tribulations of trials-interventions in communities, *American Journal of Public Health* 85: 156-158

Syme SL, Berkman LF (1976), Social class, susceptibility, and sickness, *American Journal of Epidemiology* 104: 1-8

Szreter S (1988), The importance of social intervention in Britain's mortality decline c.1850-1914: a re-interpretation of the role of public health, *Social History of Medicine* 1: 1-37

Szreter SRS (1984), The genesis of the Registrar-General's social classification of occupations, *British Journal of Sociology* 35: 522-545

Wadsworth MEJ (1997), Changing social factors and their long-term implications for health, in *British Medical Bulletin. Fetal and early childhood environment:long-term health implications. Vol.53. No.1.*, ed. Marmot M and Wadsworth MEJ, Royal Society of Medicine Press Limited, London p 198-209

Walberg P, McKee M, Shkolnikov V, Chenet L, Leon D (1998), Economic change, crime and mortality crisis in Russia: regional analysis, *British Medical Journal* 317: 312-318

Waldron I, Hughes M, Brooks T (1996), Marriage protection and marriage selection - Prospective evidence for reciprocal effects of marital status and health, *Social Science and Medicine* 43: 113-123

Ware J, Gandek B (1998), Overview of the SF-36 Health Survey and the International Quality of Life (IQOLA) Project, *Journal of Clinical Epidemiology* 51: 900-912

Winkleby MA, Jatulis DE, Frank E, Fortmann SP (1992), Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease, *American Journal of Public Health* 82: 1-6

Zielhus GA, Kiemeney LA (2001), Social epidemiology? No way, *International Journal of Epidemiology* 30: 43-44