

Socioeconomic Status and Coronary Heart Disease: A Psychobiological Perspective

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The socioeconomic disparities in health are particularly striking in the case of coronary heart disease, with rates of disease being substantially higher in lower-status individuals as defined by education, occupational position, or income. Both childhood and adult socioeconomic factors contribute, and disparities are maintained in old age after retirement (Marmot and Shipley 1996; Wannamethee et al. 1996). Low socioeconomic status is associated with subclinical levels of the underlying disease (atherosclerosis) as well as with manifest heart disease (Lynch et al. 1995). Coronary heart disease has also been linked with living in deprived neighborhoods independently of individual socioeconomic characteristics (Diez Roux et al. 2001).

Several psychosocial factors are associated with increased risk of coronary heart disease as well. Authoritative reviews have concluded that the strongest evidence is for work stress, lack of social integration, depression, and depressive symptoms, with suggestive but weaker evidence for anger, hostility, and anxiety (Hemingway, Kuper, and Marmot 2003; Krantz and McCeney 2002). Other forms of chronic stress such as caregiver burden have been related to the incidence of coronary heart disease in a more limited set of studies (Lee et al. 2003). It is telling that many of these psychosocial factors are socially graded. For example, low job control, one of the most toxic elements of chronic work stress, is more prevalent among people working in lower-status jobs (Marmot et al. 1997). Social isolation is more common among less educated and less affluent individuals (Turner and Marino 1994), and there is a consistent association between lower socioeconomic position and depressive symptoms (Lorant et al. 2003).

This chapter addresses the issue of how socioeconomic position and other social and psychological factors are reflected in differences at the bio-

logical level. What are the pathways through which socioeconomic factors accelerate or retard the physical processes of coronary atherosclerosis, thrombosis, and impaired cardiac function? We have been working on this problem for the last several years, bringing together perspectives from epidemiology and psychobiology. Here we present a rationale for how to tackle these issues and a summary of what we have learned so far.

Pathways to coronary heart disease

Economic factors, educational attainment, social isolation, and other psychosocial factors are indirect causes of coronary heart disease. They do not affect disease pathology directly, but do so through more proximal processes. An understanding of these pathways requires some background concerning the pathology of coronary heart disease. The underlying problem in coronary heart disease is coronary atherosclerosis, a progressive disease involving the gradual thickening of the walls of the coronary arteries. Coronary atherosclerosis used to be regarded as a largely passive process resulting from the gradual accumulation of lipid (cholesterol) in the arterial wall. The last two decades have witnessed a fundamental change in knowledge about the causes of atherosclerosis and thrombosis, however, and the disease is now known to involve chronic vascular inflammation (Ross 1999). The inflammation begins in the cells lining the vessel wall and leads to progressive accumulation not only of lipid, but also of smooth muscle cells and white blood cells such as macrophages, lymphocytes, and platelets. The process is regulated in part by inflammatory cytokines such as interleukin (IL)-6 and tumor necrosis factor alpha and by molecules called acute phase proteins, notably C-reactive protein. At later stages of the disease process, plaque (clumps of atheromatous material) forms on the internal vessel walls and obtrudes in the interior through which the blood flows. These plaques can rupture, causing thrombosis (internal clot formation) and acute blockage of arteries. Blood clotting or hemostatic factors are therefore involved in this process in conjunction with inflammation of the arterial walls; molecules such as fibrinogen are important, together with small white blood cells called platelets.

Atherosclerosis starts early in life and continues for decades without clinical consequences. The disease typically comes to light at an advanced stage when the coronary arteries become partly or completely blocked and the muscle of the heart fails to be supplied with energy. The person may then experience angina pectoris, a myocardial infarction, or death.

Proximal causes

The observation that low socioeconomic position affects disease risk and is associated with subclinical atherosclerosis indicates that socioeconomic po-

sition has a long-term influence on the disease process. Thus, rather than triggering acute cardiac events in people with advanced disease, socioeconomic factors influence the underlying disease. Similarly, many prospective studies of psychosocial factors such as work stress have demonstrated effects that developed over 10 to 25 years (Kivimaki et al. 2002). These factors must therefore influence the process of vascular inflammation and accumulation of cells in the sub-endothelial layers, and also possibly plaque rupture and thrombus formation. The major determinants of atherosclerosis are the standard risk factors, namely high circulating cholesterol, high blood pressure, and cigarette smoking (Greenland et al. 2003). A second set of risk factors involves disturbed glucose metabolism, leading to insulin resistance and in some cases to diabetes. The new understanding of coronary atherosclerosis has led to the identification of inflammatory markers that also predict coronary heart disease, such as the concentration of fibrinogen and C-reactive protein in the blood (Danesh et al. 1998). Several of the factors involved in blood clotting and the formation of thrombus have also emerged through prospective epidemiological studies as risk factors, including plasma viscosity and a molecule involved in the adhesion of platelets to vessel walls called von Willebrand factor (Danesh et al. 2000).

Some of these cardiovascular risk factors are socially graded, and as such must be candidates for mediating socioeconomic disparities. Smoking has a strong social gradient in the United States and northern Europe at least, with higher rates in less affluent groups. For other risk factors, social gradation seems to be more pronounced for the newly identified inflammatory and metabolic markers than it is for the older established indexes such as high blood pressure and high cholesterol level. Thus high blood pressure has a small and inconsistent association with socioeconomic position, and social gradients in cholesterol levels are small and variable across studies (Colhoun, Hemingway, and Poulter 1998; Marmot et al. 1991; Wamala et al. 1997). By contrast, insulin resistance and fibrinogen have shown consistent socioeconomic gradients, and differences have also been recorded for C-reactive protein, IL-6, and von Willebrand factor (Brunner et al. 1996; Brunner et al. 1997; Kumari, Marmot, and Brunner 2000; Owen et al. 2003).

The issue of the mediation of socioeconomic disparities can therefore be reframed as a specific question about how social and psychological experience can induce changes in vascular, metabolic, and inflammatory processes. Our conceptual model is that low socioeconomic position is associated with greater exposure to adversity in life, coupled with lower protective resources such as social support and effective coping responses (Stephoe and Marmot 2003). These in turn affect biological responses that increase cardiovascular disease risk. The translational process is a problem of psychobiology.

Psychobiological processes

Psychobiological processes are the pathways through which psychosocial factors stimulate biological systems via central nervous system activation of autonomic, neuroendocrine, and immunological responses (Steptoe 1998). The physiological responses that are relevant to coronary heart disease are organized through central nervous stimulation of two neurobiological pathways: the hypothalamic–pituitary–adrenocortical (HPA) axis leading to release of steroid hormones such as cortisol, and the sympatho-adrenal axis, involving activation of the sympathetic branch of the autonomic nervous system in conjunction with hormones such as epinephrine (adrenaline) and norepinephrine. These pathways have a range of effects on biological systems that are adapted for vigorous physical activity and for fight-or-flight responses. For example, cortisol stimulates the production of glucose in the liver, helps release free fatty acids from fat stores, and is involved in the regulation of water balance and control of the immune system (McEwen et al. 1997). Sympathetic nervous system activation leads to increases in blood pressure and heart rate, stimulation of blood clotting processes, upregulation of immune function, and release of stored free fatty acids. These responses become maladaptive under conditions of repeated or chronic stimulation, or when they are elicited under inappropriate conditions. When this happens, either excessively high or low levels of HPA and sympatho-adrenal function occur, with adverse effects on health. This can be illustrated by rare clinical conditions such as Cushing’s disease, which is characterized by prolonged heightened secretion of cortisol; victims of Cushing’s disease are prone to high blood pressure (hypertension), insulin resistance, abdominal obesity, osteoporosis, gonadal dysfunction, depression, irritability, and fatigue. These factors in turn contribute to type 2 diabetes and coronary heart disease.

Psychobiological processes are studied extensively in animal experiments, where many of the specific responses leading to physical pathology have been worked out. For example, studies in nonhuman primates have demonstrated that exposure to chronic social stress can accelerate coronary artery blockage or stenosis (Kaplan et al. 1982). Furthermore, individual differences in cardiovascular stress reactivity predict the impact of social stress on stenosis (Manuck, Kaplan, and Clarkson 1983). The challenge is how to investigate the psychobiological processes related to socioeconomic disparities in humans. Epidemiological techniques provide evidence for associations between biological factors and socioeconomic status. But to understand dynamic influences of life experience on biological responses, we are limited to two principal methods: acute mental stress testing and naturalistic monitoring of biological responses in everyday life.

Mental stress testing and socioeconomic disparities

Mental stress testing involves the standardized measurement of biological responses when people are exposed to demanding situations. Participants are typically assigned tasks that they find difficult and stressful. These might include problem-solving tasks, emotionally stressful interviews, and simulated public speaking. The biological responses are recorded at baseline, during tasks, and then in the post-task recovery period.

There are two components to the psychobiological response: the size of the response and the speed of post-task recovery. One individual or group might produce larger blood pressure increases than another in response to the same standardized task. Variations in acute blood pressure reactions have been shown to predict future hypertension and the progression of subclinical atherosclerosis (Lynch et al. 1998; Treiber et al. 2003). Alternatively, two individuals might produce the same size of reaction, but differ in the rate at which they recover or return to baseline blood pressure levels. Future cardiovascular disease can also be predicted by the speed of recovery (Schuler and O'Brien 1997). The recovery component is particularly relevant to the concept of allostatic load developed by McEwen and colleagues (McEwen 1998). They argue that chronic or repeated attempts at adaptation to life's demands result in wear and tear on biological regulatory systems so that they no longer remain within optimal operating ranges, resulting in reduced ability to adapt over time (McEwen and Wingfield 2003).

Before we began our studies, the influence of socioeconomic status on psychobiological responses during mental stress testing had been investigated by a number of researchers with inconsistent results. Studies of children and adolescents showed that individuals from poorer families or those who lived in deprived neighborhoods experienced greater blood pressure reactions than more advantaged participants, but findings in adults had been variable. We have reviewed this literature elsewhere (Step toe and Marmot 2002). Two factors might be important in explaining these inconsistencies. The first is the nature of the tasks given to people during mental stress testing. A strength of the laboratory method is that conditions are standardized, so that the same challenges are administered to everyone; any differences in response are therefore due to variations in how people react, not to what they experience. But many of the tasks used in mental stress testing might be appraised differently across the social gradient. Some tasks are intelligence tests, so that people of different intellectual capability are not challenged to the same extent. One large study of socioeconomic status used portions of an intelligence test to stimulate cardiovascular responses (Carroll et al. 1997). Simulated public speaking tasks are also popular for eliciting

stress responses, but public speaking is much more familiar to people in higher socioeconomic positions who may speak to audiences as part of their regular work. Most of these tasks do not provide a fair test for comparison of socioeconomic groups, and we therefore thought it important to use stimuli that were appraised similarly by people across the social gradient. Second, the previous literature had focused exclusively on blood pressure and heart rate responses. Although these are critical, they need to be supplemented by more-direct measures of the inflammatory and hemostatic factors involved in atherosclerosis.

The Whitehall psychobiological studies

Over the last five years, we have carried out psychobiological studies to test the hypothesis that heightened biological responsivity is associated with lower socioeconomic position. The main study involved 238 middle-aged men and women who were members of the Whitehall II epidemiological cohort (Stephens et al. 2002). Whitehall II is a sample of 10,308 London-based civil servants originally recruited in 1985–88 to investigate demographic, psychosocial, and biological risk factors for coronary heart disease (Marmot et al. 1991). The reason for using this sample is that socioeconomic status defined by occupational grade in the British civil service is known to be related to coronary heart disease, so that by systematically sampling from different grades we identified groups varying on socioeconomic characteristics that are definitely relevant to cardiovascular health. We compared individuals from higher, intermediate, and lower grades of employment. None of the participants had manifest heart disease or other cardiovascular disorders such as diabetes. Although participants were selected on the basis of occupational grade, they also varied on other common markers of socioeconomic status. For example, at the time of testing, the median personal income for the lower, intermediate, and higher grade participants was equivalent to US\$38,000, \$50,000, and \$71,000 respectively. Three-quarters of the lower grade group had only elementary education, compared with 45 percent of the intermediate and 18 percent of the higher grade groups.

We measured blood pressure, heart rate, cortisol, and several blood analytes during and following the performance of two brief tasks that we had pre-tested for being appraised similarly across groups. These tasks were a color/word interference task and a mirror tracing task. Ratings obtained after each task confirmed that men and women in the three occupational grades found the tasks equally stressful, difficult, and uncontrollable.

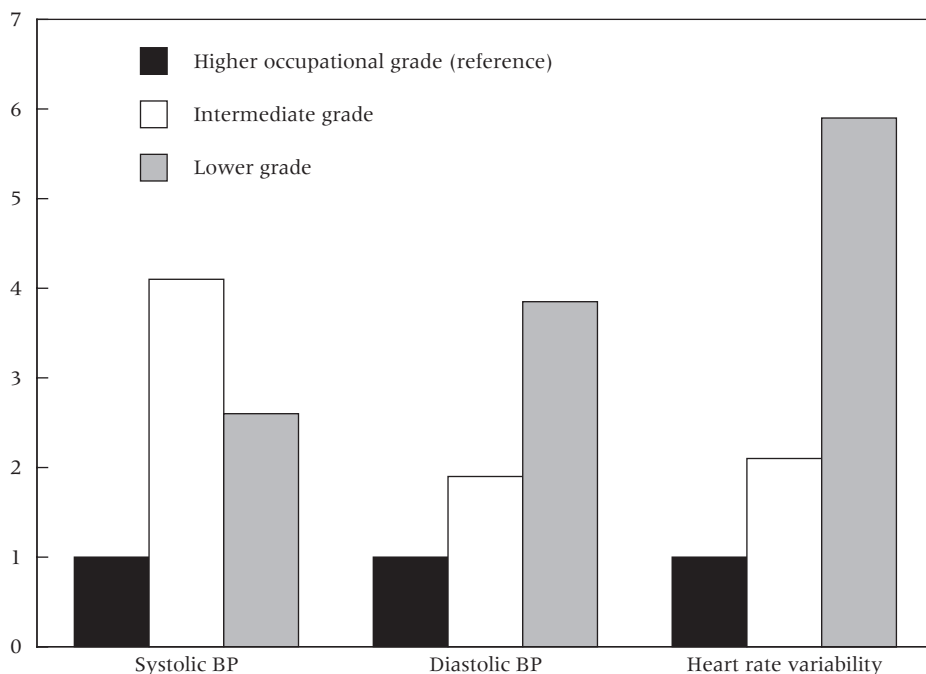
One of the first fruits of this work was our discovery that many of the factors involved in vascular inflammation and in hemostatic control are sensitive to psychological stress. Thus we observed that in response to stress,

increases occur in the concentration of fibrinogen and C-reactive protein, in proinflammatory cytokines such as IL-6 and tumor necrosis factor alpha, in hemostatic factors such as von Willebrand factor and plasma viscosity, and in platelet activation. These responses have quite variable time courses. For example, fibrinogen and von Willebrand factor increase acutely during tasks, and decrease in the post-task period (Steptoe et al. 2003a; Steptoe et al. 2003c). By contrast, C-reactive protein and IL-6 respond slowly, so marked changes are not observed until two hours following the tasks (Brydon et al. 2004; Steptoe et al. 2003d). These biological processes are therefore sensitive to psychosocial influence. The question is whether response patterns vary with socioeconomic position.

The analyses of blood pressure responses indicated that the tasks elicited substantial increases, but that reactivity did not vary markedly by socioeconomic status. Systolic blood pressure increased by an average of 22.5 mmHg, while diastolic pressure rose by 13.6 mmHg during tasks. But important differences emerged during the post-stress recovery period. We continued to measure blood pressure for up to 45 minutes after tasks had been completed. Blood pressure failed to return to baseline levels in a substantial proportion of participants, even though they were relaxing without any further tasks. The failure of blood pressure to return to baseline was associated with socioeconomic status. As shown in Figure 1, the likelihood of incomplete post-task recovery was greater in men and women of lower socioeconomic position. Compared with the higher occupational grades (the reference group), the odds of incomplete recovery in the lower grade group were 2.60 (95 percent C.I. 1.20–5.65) for systolic pressure and 3.85 (1.48–10.0) for diastolic pressure, adjusted for sex, age, baseline values, and the magnitude of reactions to tasks. Figure 1 also shows that there was impairment in heart rate variability recovery in the lower grade group. Heart rate variability is relevant to the autonomic control of cardiac function, and low heart rate variability is a predictor of future cardiovascular disease (La Rovere et al. 1998; Schroeder et al. 2003).

Our data suggest that socioeconomic status is associated not so much with the magnitude of cardiovascular reactions to a challenge as with the rate of recovery or duration of responses. Similar patterns were observed for some other biological measures. For example, we assessed variables involved in the processes through which blood clots and thromboses are formed, including Factor VIII and plasma viscosity. The grade of employment groups did not differ in the magnitude of reactions to tasks, but values remained significantly more elevated 45 minutes following the tasks in the lower grade compared with the high and intermediate grade participants (Steptoe et al. 2003c). These effects were independent of age, sex, and other factors that might be influential such as body mass and smoking. We also observed socioeconomic differences in the post-task increases in

FIGURE 1 Odds of incomplete recovery to baseline by 45 minutes post-stress in systolic blood pressure, diastolic blood pressure, and heart rate variability



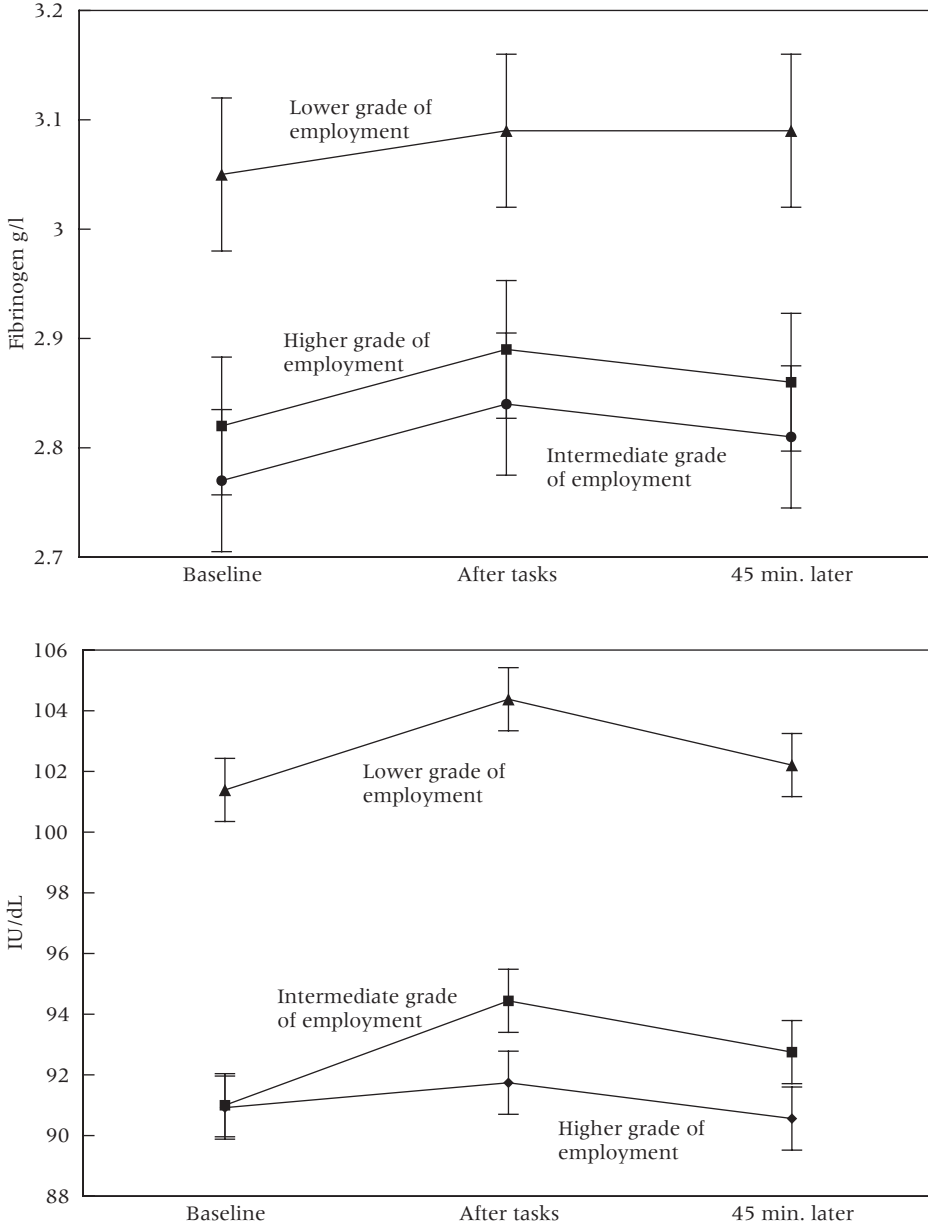
SOURCE: From Steptoe et al. (2002).

the concentration of inflammatory cytokines such as IL-6 and IL-1 receptor antagonist (Brydon et al. 2004; Owen et al. 2003).

However, not all variables show this pattern. Two examples are shown in Figure 2. Higher absolute levels of plasma fibrinogen and von Willebrand factor were recorded in the lower occupational grade groups, suggesting a contribution to heightened cardiovascular disease risk (Steptoe et al. 2003a; Steptoe et al. 2003c). But the magnitude of stress-induced increases did not differ across grades of employment, and the duration of responses was also similar. Figure 2 also indicates that the intermediate employment group was not midway between lower and higher status groups, but closer to the higher grade group in their biological profile. We have observed this pattern in a number of variables, but do not yet have a satisfactory explanation. It could be that acute mental stress testing is not the most accurate way of investigating these particular biological factors.

Although mental stress testing can help identify biological responses associated with socioeconomic status, it has two limitations. First, the responses are acute and typically subside within two to four hours. Second,

FIGURE 2 Mean concentration of plasma fibrinogen (upper panel) and von Willebrand factor (lower panel) at baseline, immediately after stressful tasks, and 45 minutes later in men and women from higher, intermediate, and lower grades of employment



NOTE: Von Willebrand factor was log transformed before analysis, and geometric means are presented. Error bars represent standard error of the mean.
SOURCE: From Steptoe et al. (2003a and 2003c).

the stimuli used to elicit reactions are artificial and seldom encountered in real life. We therefore need to supplement these findings with investigations of biological responses to everyday experiences in people of differing socioeconomic status.

Naturalistic monitoring of biological markers

Naturalistic monitoring involves the assessment of biological function during everyday life. Clearly it is possible though difficult for health professionals or researchers to visit people in their homes or work places and obtain biological measures, but it is even more informative to study processes repeatedly as participants go about their everyday lives. This field has expanded enormously over recent years because of two advances. The first is the introduction of ambulatory blood pressure monitors. These relatively unobtrusive measurement devices can be programmed to record blood pressure and heart rate repeatedly throughout the day. The instruments are often timed to take measures every 15–20 minutes in the daytime and less frequently at night. This provides information about blood pressure under everyday as opposed to clinical conditions, and is increasingly used in the clinical management of hypertension. It is also possible to relate fluctuations in blood pressure to ongoing activities, events, and experiences during the day, so as to provide an insight into psychobiological processes. The second advance is the development of salivary assays of hormones. The main hormone that has been studied is cortisol, but it is also possible to measure dehydroepiandrosterone (DHEA), testosterone, and other substances. Before salivary measures were introduced, assessments of neuroendocrine function were limited to blood and urine sampling, both of which are invasive and may disrupt the very phenomena that are being investigated. Salivary sampling for cortisol involves having people spit into test tubes, or place dental rolls in their mouths for a few minutes until saturated. These samples are placed in labeled test tubes, which can then be sent to the laboratory for analysis. Fortunately, cortisol is relatively stable in saliva, so it can be stored at room temperature for several days without degradation.

Naturalistic methods have several limitations. One disadvantage is that the range of biological markers that can be assessed is small compared with those recorded in the laboratory, so many of the interesting biological processes involved in cardiovascular pathology cannot be measured. Additionally, the data require sophisticated statistical handling, since it is necessary to adjust for the many other factors that influence biological function in everyday life. For instance, blood pressure is affected by current and recent physical activity, posture, smoking, and drinking tea and coffee, and these need to be accounted for in the analysis.

To date only a handful of published studies have used ambulatory methods to investigate socioeconomic position. Matthews and coworkers (Matthews et al. 2000) carried out ambulatory blood pressure monitoring of 50 higher and 50 lower status men and women as defined by occupation. They found no differences in systolic or diastolic blood pressure over the working day, although heart rate was faster in lower status participants who experienced negative moods over the day. In a study of New York working men, ambulatory blood pressure was positively associated with work stress, but this effect was more marked in participants of lower occupational status (Landsbergis et al. 2003). Cortisol responses to everyday life stressors were inversely related to social status in a sample of 51-year-old Swedish men (Rosmond and Bjorntorp 2000).

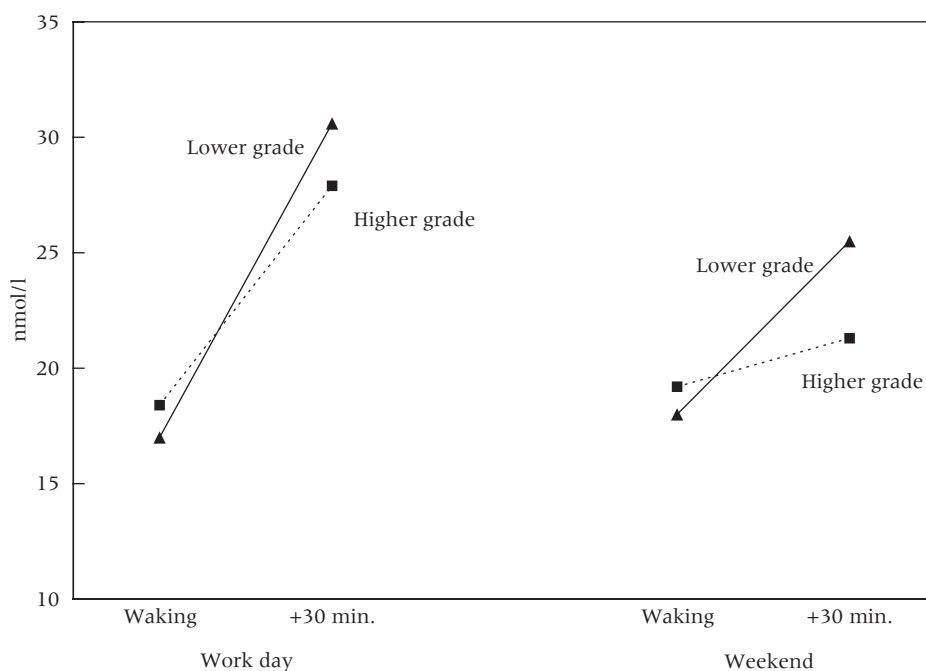
We carried out ambulatory blood pressure monitoring with participants in the main Whitehall psychobiology study and obtained useful data from 199 men and women. We found that systolic blood pressure was higher in the lower occupational grade participants, but only during the morning hours (Stephoe et al. 2003b). Systolic pressure averaged 128.9 ± 15.7 mmHg in the lower grade group, compared with 122.6 ± 12.5 mmHg in the intermediate and 123.3 ± 12.7 mmHg in the higher grade participants. Differences were significant after adjusting for age, gender, smoking, alcohol intake, physical activity, and body weight. The groups did not differ over the rest of the working day or in the evening. Thus associations between socioeconomic status and disturbances of blood pressure control were most apparent early in the day. A similar conclusion is supported by our cortisol data.

Cortisol shows a strong diurnal variation, being high early in the morning and falling over the day. Recent work suggests that from the psychobiological perspective, two distinct phenomena are at work. The first is the profile of cortisol over the day itself, in terms of absolute output and pattern of decline into the evening. In our study, cortisol output over the working day was greater in men of lower than higher socioeconomic status, as we might expect from the perspective of the chronic allostatic load model developed by McEwen. But among women, the opposite pattern emerged, with greater cortisol concentrations in higher than lower status participants (Stephoe et al. 2003b). We have no explanation for this pattern, which appears to be independent of marital status, having children, and stress over the day.

The second part of the cortisol profile is the response to waking in the morning. Cortisol typically increases over the first minutes of the day, reaching a peak 20–30 minutes after waking. There is growing evidence that this cortisol awakening response is greater among individuals experiencing chronic stress from work or emotional strain (Pruessner et al. 2003; Steptoe et al. 2000). It appears to be an anticipatory response associated with the realization of the demands of the day ahead.

We persuaded most of the participants in our psychobiology study to take saliva samples on waking and then 30 minutes later both on a work day and a weekend day. We expected the cortisol awakening response to be smaller on the more relaxed weekend day and wondered whether there would be differences by occupational grade and whether these persisted on both days. The relevant results are summarized in Figure 3. Because a number of participants did not produce reliable data on both days, the three occupational grade groups were compressed into two (lower and higher). There are three interesting effects in these results. First, the level of salivary cortisol on waking was stable on the two days, and did not vary with socio-economic position. Thus even though people were waking more than an hour later on the weekend, the cortisol level was unchanged. Second, there were significant increases in cortisol after waking on both days, but the increases were much smaller on the weekend. This is consistent with the notion that the cortisol increase on waking represents an anticipatory response to the coming day, and that this is attenuated when the pressure of commuting and working is eliminated. Third, on both days the lower socio-economic group showed a larger cortisol awakening response than did the

FIGURE 3 Mean concentration of salivary free cortisol sampled on waking and 30 minutes later on a work day and weekend day in men and women from higher and lower grades of employment



SOURCE: From Kunz-Ebrecht et al. 2004.

higher status participants. The differences were statistically highly significant, and were independent of smoking status. They represent real differences in neuroendocrine regulation associated with socioeconomic status.

It is tempting to see parallels between the results that we have obtained in standardized laboratory stress testing and naturalistic monitoring. In both cases, evidence has emerged for disturbances of psychobiological regulation in adults of lower socioeconomic status. In both cases, these disturbances are manifest less in absolute differences in reactivity to events than in the maintenance of healthy regulatory function. In the naturalistic study, the most striking differences were observed not while people were at work, when they might be most exposed to the stresses associated with their occupational status, but earlier as they prepared to face the challenges of the day. In the laboratory, the differences were most apparent during the period after exposure to challenge, as readjustment to normal equilibrium was being established. These effects may therefore be quite insidious, occurring at times of day and in situations where they are not expected.

The differences we have observed across the social gradient are quite small in absolute terms both in the laboratory and during everyday life. But the importance of these responses is that they are repeated on a regular basis for months or years of adult life. One can make an analogy with smoking. A single cigarette has an acute effect on biological function that soon dissipates without lasting consequences. But a cigarette every 30 minutes, every day, every month, every year for decades has a profound effect on health. The same may be true of these small psychobiological responses.

Additional pathways

The emphasis that we have placed on psychobiological processes should not be taken to imply that these are the only pathways through which socioeconomic status influences risk of coronary heart disease. Several other mediating mechanisms may be relevant. One that can probably be discounted is mediation through genetic differences. There is a heritable component to much of coronary heart disease and its risk factors (Nabel 2003). Although it is theoretically possible that the genetic determinants of coronary heart disease are socially graded, this is unlikely. Socioeconomic patterns of the disease have changed drastically in Britain and the United States over one or two generations, far more quickly than could be sustained by any inherited process. A more plausible but untested possibility is that exposure to infection is involved. The notion that infection might in part cause coronary heart disease has stemmed from the search for the origins of coronary vascular inflammation. Inflammation of any tissue typically occurs in response to injury or infectious organisms, but studies of specific microorganisms such as *Chlamydia pneumoniae* have been inconclusive (Danesh et al. 2002). An

alternative idea is that the total pathogen burden, or the number of infectious organisms to which people are exposed in their lifetimes, is relevant. Among patients with advanced atherosclerosis, those with antibodies for a larger number of common pathogens were at higher risk for future cardiac events (Espinola-Klein et al. 2002). Exposure to infection might be socially graded, with poorer people living in less hygienic conditions being at higher risk. But the possibility that such exposure accounts for socioeconomic differences in coronary heart disease is highly speculative at present. It is more likely that infectious burden is a marker of socioeconomic status, rather than a mediating pathway.

The pathway that probably makes a large contribution to the translation of socioeconomic disparities into coronary heart disease risk is lifestyle. Lifestyle factors such as smoking, food choice, alcohol consumption, and physical activity are socially graded in many countries. Indeed, the social gradient in cigarette smoking in the UK has increased markedly over recent decades, owing both to differences in uptake and more importantly to greater cessation rates in more affluent sectors (Jarvis and Wardle 1999). There is a relatively consistent social gradient in overweight and obesity, although this is much stronger in women than men (Lantz et al. 1998). Fat consumption shows no clear association with socioeconomic status in the United States and Britain, but fruit, vegetable, and fiber intake is greater in higher status groups (Bennett et al. 1995; Shimakawa et al. 1994). Vigorous leisure-time physical activity is more common in higher status groups, as is moderate alcohol consumption (Marmot 1997; Wardle and Steptoe 2003).

Several studies of social position have demonstrated that gradients in coronary heart disease persist after controlling statistically for factors such as smoking, body mass index, diet, and alcohol, so lifestyle is certainly not the complete explanation (Marmot, Kogevinas, and Elston 1987; Steenland, Henley, and Thun 2002). The question of how much of the variance in coronary heart disease is accounted for by lifestyle is difficult to gauge. In the Finnish Kuopio study, about 50 percent of the income gradient in cardiovascular mortality was accounted for by smoking, alcohol, and physical activity (Lynch et al. 1996), while in the Dutch Globe study, more than half of the educational gradient in acute myocardial infarction was accounted for by smoking, alcohol, body mass, and physical inactivity (Van Lenthe et al. 2002). In the Whitehall II study, we estimate that lifestyle factors account for only about a quarter of the socioeconomic gradient. But in a recent analysis of educational differences in blood pressure in the United States, Stamler and colleagues (Stamler et al. 2003) argued that nutritional factors account for most of the difference between better and less well educated adults. Few studies have assessed all relevant lifestyle factors in a comprehensive way, and it is likely that the importance of these factors varies with the nature of the population (urban/rural, high or low smoking countries,

dietary traditions, etc.) and possibly with the way in which socioeconomic status is measured.

Conclusions

We have argued that psychobiological processes are plausible pathways through which socioeconomic disparities are translated into cardiovascular disease risk and coronary heart disease incidence. The evidence is preliminary. Although variations in psychobiological function have been associated with socioeconomic status, we do not know whether they contribute to disease end points. The quantification of the relative importance of psychobiological, lifestyle, and other mediating pathways has not yet been attempted. Nonetheless, the possibility now exists for identifying mechanisms at the most precise level of pathogenesis that are affected by social forces and psychological characteristics. The elaboration of psychobiological methods so that they can be used in large-scale population studies will further this integration of disciplines and our understanding of how socioeconomic status affects coronary heart disease risk.

Note

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