Status Syndrome: A Challenge to Medicine

Michael G. Marmot

JAMA. 2006;295(11):1304-1307 (doi:10.1001/jama.295.11.1304)

http://jama.ama-assn.org/cgi/content/full/295/11/1304

Status Syndrome
A Challenge to Medicine

Michael G. Marmot, MBBS, MPH, PhD, FRCP, FFPHM

THE POOR HAVE POOR HEALTH. AT FIRST BLUSH THAT is neither new nor surprising. Perhaps it should be more surprising than it is. In rich countries, such as the United States, the nature of poverty has changed—people do not die from lack of clean water and sanitary facilities or from famine—and yet, persistently, those at the bottom of the socioeconomic scale have worse health than those above them in the hierarchy. Even more challenging is that socioeconomic differences in health are not confined to poor health for those at the bottom and good health for everyone else. Rather, there is a social gradient in health in individuals who are not poor: the higher the social position, the better the health. I have labeled this "the status syndrome."[1]

When we first drew attention to the phenomenon of the social gradient in coronary heart disease (CHD) in the Whitehall study of British Civil Servants,[2] it seemed a quirky observation that flew in the face of conventional wisdom. In the 1970s, it was widely accepted that CHD was a disease of affluence caused by stress and affluent lifestyle. Yet the Whitehall study showed that individuals second from the top of the occupational hierarchy had higher CHD mortality rates than those above them, and those third from the top had higher rates still. Moreover, this social gradient in mortality was seen not only for CHD, but for most of the major causes of death.

It turned out that civil servants in the London area of Whitehall, far from representing an atypical postimperial backwater, were typical of the developed world. The phenomenon of the social gradient in health has been observed worldwide and has, at last, become a vigorous area of research.[3] This article draws on the research of the London-based group and argues that what researchers have learned about causes of illnesses and death and what society could do about them has great relevance to health in the United States and other rich nations. Conventional explanations for noncommunicable disease—lack of access to medical care, unhealthy lifestyles—at best only partially explain the status syndrome. Rather, the lower individuals are in the social hierarchy, the less likely it is that their fundamental human needs for autonomy and to be integrated into society will be met. Failure to meet these needs leads to metabolic and endocrine changes that in turn lead to increased risk of disease.

The Social Gradient in Health Is Widespread

Perhaps because of the British obsession with class, data by socioeconomic position have been more readily available in Britain than elsewhere. However, the social gradient in mortality is found across Europe.[4] In the United States, income, education, and occupation have all been shown to predict mortality.[5] Data on morbidity are less available, but a comparison of morbidity rates according to both income and education show the social gradient in health to be steeper in the United States than in England (James Banks, PhD, unpublished data, 2006).

The sheer magnitude of the difference in life expectancy between the top and bottom of the hierarchy calls for attention. In the 1990s, Murray et al[6] compared county-level differences in life expectancy. For example, when traveling along the distance of nearly 12 miles on the Washington, DC, Metro from downtown to Montgomery County, Maryland, life expectancy of the local population segment rises about a year and a half for each mile traveled. Poor black men at one end of the journey have a life expectancy of 57 years, and rich white men at the other end have a life expectancy of 76.7 years.[1]

These shocking differences should not be viewed as qualitatively different from the status syndrome, the social gradient in health, but as simply the ends of the spectrum. Although there is much concern in the United States with racial and ethnic disparities in health, this low life expectancy in Washington, DC, is not primarily to do with being black. It may well be that conventional socioeconomic measures do not account for the high mortality of blacks compared with whites.[7] The question is not which socioeconomic measure is the best predictor of mortality,

Author Affiliation: International Institute for Society and Health, Department of Epidemiology and Public Health, University College London, London, England.
Corresponding Author: Michael G. Marmot, MBBS, MPH, PhD, FRCP, FFPHM, International Institute for Society and Health, Department of Epidemiology and Public Health, University College London, 1-19 Torrington Pl, London, England WC1E 6BT (m.marmot@ucl.ac.uk).

but how to understand the causal framework. I propose that the correct causal framework will show that the same fundamental processes, related to the way society is organized, account for socioeconomic and racial disparities in health. As stated, the nature of poverty has changed. This is seen in the diseases that affect those lower in the hierarchy. The principal causes of premature mortality in black men are not diseases traditionally thought of as related to material deprivation, but cardiovascular disease, violence, and HIV/AIDS.

Egregious examples of social and economic inequality are not needed to see inequalities in health. In egalitarian Sweden, Erikson showed that individuals with a PhD have lower mortality than those with a master's degree, who have lower mortality than those with a bachelor's degree, and so on down the educational hierarchy.[8] "Greater poverty," or material deprivation, is not a helpful answer to the question of why someone with a master's degree should have higher mortality than someone with a PhD.

Conventional Explanations

In the United States it is difficult to discuss the issue of disparities in health without consideration of the intrusive fact that more than 40 million individuals do not have health insurance. But the United States is unique among rich countries in not providing universal access to medical care; and the social gradient is seen in these other countries. In Britain and Sweden it is hard to argue that lack of medical care accounts for the higher mortality of those second from the top of the social hierarchy compared with those at the top. The Whitehall II study of British Civil Servants[9] showed that the lower the position in the occupational hierarchy, the more cardiac investigations and the more interventional procedures for coronary artery disease were performed.[9] The higher rate of interventions in lower grades matched, proportionately, the higher rate of disease. There was no evidence of undertreatment of the less privileged.

Much is known, and much demands action, about the causes of noncommunicable disease. Were there not unhealthy diets, smoking, and sedentary lifestyles, there probably would be no CHD epidemic. But social gradients in mortality are seen everywhere. Where CHD rates are low, there are gradients in communicable disease.[10] And where these coronary risk factors are highly prevalent, there is a social gradient in CHD occurrence that is not, for the most part, explained by these risk factors. In the first Whitehall study, a combination of smoking, serum cholesterol level, blood pressure, height (as a marker of early life), and blood glucose level accounted for less than a third of the social gradient in CHD mortality.[11]

A reflection on these causal hypotheses from a different direction comes from animal studies: there are social gradients in atherosclerosis and markers of cardiovascular risk in nonhuman primates.[12] It is difficult to argue that either differential access to medical care or differences in health behaviors account for these differences. Sapolsky hypothesizes that stress, linked to low social status, is to blame.[12]

Relative Differences in Status Translate Into Absolute Differences in Life Chances

If the social gradient in health is not due to differences in medical care, or primarily to differences in health behaviors, or to differences in material circumstances, then what is the cause? Is it a pure status effect?

Status is a relative, not an absolute, concept. Therefore, a link between status and health could be taken as showing the importance of relative position for health. But relative position, in its turn, must imply some sort of "exposure" if it is to be a powerful cause of disease. Sen has pointed out that relative position on the scale of incomes may translate into absolute position on the scale of, what he calls, "capabilities."[13] In other words, it is not what a person has that is important, it is what he or she can do with what he or she has. For example, one way of comparing income among countries is to adjust gross national product to purchasing power. By that scale, blacks in the United States have about 4 times the income of men in Costa Rica or Cuba, but about 9 years' shorter life expectancy.[1]

This is not to argue that blacks are not worse off than whites in the United States. However, the ways that blacks in the United States are worse off have little to do with absolute income. They have much more to do with 2 fundamental human needs: autonomy and full social participation. Deprived of a clean, safe neighborhood, meaningful work, opportunities for quality children's education, freedom from police harassment and arrest, and freedom from violence and aggression, it is harder to have control over one's life or be a full social participant.

Lack of control and low social participation have a powerful influence on disease risk. Low control at work is associated with increased risk of CHD,[14] absence due to sickness, and mental illness. One way of conceptualizing "full social participation" is by examining participation in social networks and receipt of social support. Low participation in social networks is linked to increased risk of a range of diseases[15] and increased mortality rates.[16] Another aspect of full social participation is provided by the notion of reciprocity. If a person expends effort on behalf of others, there is a reasonable expectation of reward. In the workplace, imbalance between effort and reward is associated with increased risk of disease.[17]

Social participation should not be seen as simply a characteristic that high-status individuals are fortunate enough to have in abundance and of which low-status individuals are deprived. It is fostered by society and removed by it. In this sense, it has resonance with the concept of social capital.[18] The way Western societies are organized leads high-status individuals to be able to reap more of the health benefits that social participation brings than are individuals lower in the hierarchy.

Both low control or lack of autonomy and low social participation are likely to be important contributors to inequality in health.[1] There is biological plausibility to this proposition. Low social position is linked to activity of the 2 main biological stress pathways: the sympatho-adrenomedullary axis and the hypothalamic-pituitary-adrenal axis. The Whitehall II study of British civil servants[9] examined heart rate variability as an indicator of sympathetic tone and showed that individuals in the lower grades of the civil service had less heart rate variability than those in the higher grades. Low control at work was also linked to low heart rate variability.[19] Similarly, low social position is associated with indicators of raised cortisol levels.[20] Low social position has also been linked to delayed heart rate recovery after exercise, which is related to autonomic activity, and to low exercise functional capacity.[21] One likely mechanism of action of these stress pathways is through an effect on the metabolic syndrome, which shows a clear social gradient and is linked both to autonomic function and activity of the hypothalamic-pituitary-adrenal axis. Stress at work is related to the metabolic syndrome: the more occasions on which job strain was reported, the greater the likelihood of having the metabolic syndrome.[22]

Not Simply Relative Position

Arguing that it is not simply position in the hierarchy that accounts for worse health among individuals of lower status resolves problems both of explanation and of action. All societies, even those of nonhuman primates, have hierarchies. Yet the magnitude of the social gradient in health varies among societies and within a society over time. It is not position in the hierarchy per se that is the culprit, but what position in the hierarchy means for what one can do in a given society: the degree of autonomy and social participation. Consider the example of education both as a mediator of the relationship between social position and health and a place to intervene to reduce the ill health effects of low status. In the United States, for example, there is a steep social gradient in literacy levels among young adults according to parents' level of education. Lower parental education is associated with lower literacy levels among offspring. There is a social gradient, too, in Sweden, but it is much shallower[23]; ie, children of parents with low education have less literacy disadvantage in Sweden than in the United States. Put another way: in Sweden there is a weaker link between low status and a potential cause of the status syndrome, literacy, than there is in the United States.

An Urgent Social and Medical Problem

The United States spends more on health care than any other country—around 15% of a large gross domestic product. Yet the United States ranks 29th in the world in life expectancy.[24] Something is not right. And that something is the gross inequalities in health seen within US society. These inequalities are in turn related to features of society that meet basic human needs to vastly varying degrees depending on socioeconomic position or degree of social exclusion. Research on inequalities in health suggests that there is much that can be done from early life, childhood, among adults of working age, and older individuals that would reduce these inequalities.[25] More research is needed, but much could be done with current knowledge. To this end, the World Health Organization has set up a Commission on Social Determinants of Health that will marshal the evidence and promote action across the whole of society to reduce inequalities in health.[26]

The medical profession should take a lead in promoting such action. Using one analogy, smoking rates declined in countries such as the United States and the United Kingdom not simply because physicians told patients not to smoke, but because the medical profession took the lead in bringing governments to see that action was necessary across a broad front: taxation to increase price, restrictions on advertising, restrictions on availability in public places, labeling, and more. Reductions in the gradient in health and disease will require action across a broad front, starting with women of childbearing age, early child development, education, skills training, better working and living conditions, and support for older individuals. Who will be the agents to bring to the attention of policy makers the need for such action on the social determinants of health? Why not the medical profession? Who cares more about the tragedy of lives blighted by premature ill health than do we in the medical profession? If we care, we should be leading the charge for action across a broad front to reduce inequalities in health.[26]

Financial Disclosures: None reported.
Funding/Support: The Whitehall II study has been supported by grants from the Medical Research Council; Economic and Social Research Council; British Heart Foundation; Health and Safety Executive; Department of Health; National Institutes of Health, National Heart, Lung, and Blood Institute (HL36310); National Institutes of Health, National Institute on Aging (AG13196); Agency for Health Care Policy and Research (HS06516); and the John D. and Catherine T. MacArthur Foundation Research Networks on Successful Midlife Development and Socioeconomic Status and Health. Dr Marmot is supported by an MRC Research Professorship.
Role of the Sponsor: The funding organizations did not participate in the preparation, review, or approval of this article.
Disclaimer: Dr Marmot is Chair of the Commission on Social Determinants of Health set up by the World Health Organization.

REFERENCES

1. Marmot M. *The Status Syndrome.* New York, NY: Henry Holt; 2004.
2. Marmot MG, Rose G, Shipley M, Hamilton PJS. Employment grade and coronary heart disease in British civil servants. *J Epidemiol Community Health*. 1978; 32:244-249.
3. Adler N, Ostrove J. Socioeconomic status and health: what we know and what we don't. In: Adler N, Marmot M, McEwen B, Stewart J, eds. *Socioeconomic Status and Health in Industrial Nations.* New York, NY: New York Academy of Sciences; 1999:3-15.
4. Mackenbach JP, Bos V, Andersen O, et al. Widening socioeconomic inequalities in mortality in six Western European countries. *Int J Epidemiol*. 2003;32:830-837.
5. McDonough P, Duncan GJ, Williams D, House JS. Income dynamics and adult mortality in the United States, 1972 through 1989. *Am J Public Health*. 1997;87: 1476-1483.
6. Murray CJL, Michaud CM, McKenna MT, Marks JS. *US Patterns of Mortality by County and Race: 1965-94.* Cambridge, Mass: Harvard Center for Population and Development Studies; 1998:1-97.
7. Williams DR. Racial variations in adult health status: patterns, paradoxes, and

prospects. In: Smelser NJ, Wilson WJ, Mitchell F, eds. *America Becoming: Racial Trends and Their Consequences, Vol II*. Washington, DC: National Academy Press; 2001:371-410.
8. Erikson R. Why do graduates live longer? In: Jonsson JO, Mills C, eds. *Cradle to Grave: Life-Course Change in Modern Sweden*. Durham, England: Sociology Press; 2001.
9. Britton A, Shipley M, Marmot M, Hemingway H. Does access to cardiac investigation and treatment contribute to social and ethnic differences in coronary heart disease? Whitehall II prospective cohort study. *BMJ*. 2004;329:318.
10. Farmer P. *Infections and Inequalities*. Berkeley: University of California Press; 1999.
11. van Rossum CTM, Shipley MJ, Van de Mheen H, Grobbee DE, Marmot MG. Employment grade differences in cause specific mortality: a 25 year follow up of civil servants from the first Whitehall study. *J Epidemiol Community Health*. 2000; 54:178-184.
12. Sapolsky RM. The influence of social hierarchy on primate health. *Science*. 2005;308:648-652.
13. Sen A. *Inequality Reexamined*. Oxford, England: Oxford University Press; 1992.
14. Kuper H, Marmot M, Hemingway H. Systematic review of prospective cohort studies of psychosocial factors in the etiology and prognosis of coronary heart disease. *Semin Vasc Med*. 2002;2:267-314.
15. Cohen S, Doyle WJ, Skoner DP, Rabin BS, Gwaltney JM. Social ties and susceptibility to the common cold. *JAMA*. 1997;277:1940-1944.
16. Berkman LF, Glass T. Social integration, social networks, social support, and health. In: Berkman LF, Kawachi I, eds. *Social Epidemiology*. New York, NY: Oxford University Press; 2000:137-173.
17. Kuper H, Singh-Manoux A, Siegrist J, Marmot M. When reciprocity fails: effort-reward imbalance in relation to coronary heart disease and health functioning within the Whitehall II Study. *Occup Environ Med*. 2002;59:777-784.
18. Putnam R. *Bowling Alone: The Collapse and Revival of American Community*. New York, NY: Simon & Schuster; 2000.
19. Hemingway H, Shipley M, Brunner E, Britton A, Malik M, Marmot MG. Does autonomic function link social position to coronary risk? the Whitehall II Study. *Circulation*. 2005;111:3071-3077.
20. Brunner EJ, Hemingway H, Walker BR, et al. Adrenocortical, autonomic, and inflammatory causes of the metabolic syndrome. *Circulation*. 2002;106:2659-2665.
21. Shishehbor MH, Litaker D, Lauer CE. Association of socioeconomic status with functional capacity, heart rate recovery, and all-cause mortality *JAMA*. 2006;295: 784-792.
22. Chandola T, Brunner E, Marmot M. Chronic stress at work and the metabolic syndrome: prospective study [published online ahead of print January 20, 2006]. *BMJ*. doi:10.1136/bmj.38693.435301.80**.** Accessed February 17, 2006.
23. Willms JD. Quality and inequality in children's literacy: the effects of families, schools and communities. In: Keating D, Hertzman C, eds. *Developmental Health and the Wealth of Nations: Social, Biological, and Educational Dynamics*. New York, NY: Guilford Press; 1999:72-93.
24. United Nations Development Programme. *Human Development Report 2005: International Cooperation at a Crossroads: Aid, Trade and Security in an Unequal World*. New York, NY: United Nations Development Programme; 2005.
25. Acheson D. *Inequalities in Health: Report of an Independent Inquiry*. London, England: Her Majesty's Stationery Office; 1998.
26. Marmot M. Social determinants of health inequalities. *Lancet*. 2005;365:1099-1104.

EDITORIAL

Editorials represent the opinions of the authors and *JAMA* and not those of the American Medical Association.

Brachytherapy for In-Stent Restenosis

A Distant Second Choice to Drug-Eluting Stent Placement

Debabrata Mukherjee, MD
David J. Moliterno, MD

THE GREATEST RECENT MECHANICAL ADVANCE IN PERcutaneous coronary revascularization (PCR) has been the development of bare-metal stents, which compared with traditional balloon angioplasty substantially reduce angiographic restenosis and the need for repeat target vessel revascularization (TVR). Stents provide a larger arterial lumen diameter immediately postprocedure (acute gain), although their drawback is an increased reparative response of neointimal formation (late loss). Fortunately, the net gain remains greatest with stents compared with other PCR devices. In less complex lesions, the rate of TVR with bare-metal stents is approximately 10% to 15%, although this rate has been reported to be 2- to 3-fold higher in more complex lesions and unique patient subsets.[1,2] In 2003, at a time when the use of bare-metal stents peaked, approximately 1 million coronary stents were placed in patients hospitalized in the United States.[3] Even with a conservative estimate, this means at least 100 000 in-stent restenotic lesions occurred, making this an important clinical problem.

During the last decade, numerous modalities have been used to treat in-stent restenosis (ISR); however, each has provided only modest intermediate-term efficacy. These devices—balloon angioplasty, atherectomy (directional, rotational, and laser), and repeat stenting (stent-in-stent)—provided a high rate of immediate technical success and a low rate of ischemic events, but 30% to 60% of patients required another TVR in the subsequent months.[4-7] The only therapy proven to be uniquely effective in treating ISR has

See also pp 1253 and 1264.

Author Affiliations: The Gill Heart Institute and Division of Cardiovascular Medicine, University of Kentucky, Lexington.
Corresponding Author: Debabrata Mukherjee, MD, Division of Cardiovascular Medicine, University of Kentucky, 900 S Limestone Ave, 317 Wethington Bldg, Lexington, KY 40536-0200 (mukherjee@uky.edu).