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Preface to *The Biology of Disadvantage: Socioeconomic Status and Health*

This volume is the product of the John D. and Catherine T. MacArthur Network on Socioeconomic Status (SES) and Health. For the last 12 years the network has provided a structure through which scientists from a wide range of disciplines jointly addressed the question: How does SES get under the skin to affect health? In 1999, early in our life as a network, we organized a conference held at the National Institutes of Health on SES and health in industrialized nations. The conference presentations were published as a special volume of the *Annals of the New York Academy of Sciences.*[1] Since that time, network members have worked together to provide answers to the central questions about the relationship of SES and health. In the process, numerous articles and books reflecting our work have been published.[2] The current volume builds on these findings and stands as a bookend to the former *Annals* volume, presenting what we have learned in our decade of work since the 1999 conference, and our thoughts on the current state of knowledge about the pathways by which SES affects health.

What have we learned?

Our work on SES and health began with epidemiological findings linking SES (defined in some studies by education, in others by income, and in yet others by occupational status or occupational grade within an organization) to morbidity and mortality. We now know that the picture is far more complex. As with many phenomena, the more closely one looks at any one aspect, the more complicated the picture becomes. For example, we lack consensus on how best to measure SES components. Most studies of SES use a single indicator of SES. Although a number of composite scales have been developed they do not solve the SES measurement problem, and may introduce their own difficulties in interpretation.[3] Another complexity is that the meaning and measurement of SES differs among sociodemographic groups. For example, the same level of income may have different implications for African Americans than for Whites, since a given level of income provides relatively fewer resources and is associated with fewer health benefits for African Americans.[4]

Social disadvantage and its consequences

SES differences in health are embedded in the larger problem of health disparities associated with social disadvantage. In the decade in which the network has been functioning, there has been an explosion of interest in health disparities and in empirical work focused on discovering the mechanisms responsible for creating and maintaining such disparities. In the introductory chapter of the volume, Adler and Stewart identify the evolution across five eras of research of questions about the causes of health disparities and the development of methods for studying these associations. Disparities in health can be observed not only in relation to SES, but also among racial/ethnic groups, between men and women, and among those living in different locales in the United States. Dow and Rehkopf place work on health disparities in an historical context in the United States and in comparison to other countries, and show how patterns

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of morbidity and mortality change in relation to social conditions. Health disparities emerge from social disadvantage inherent in different bases of social stratification and exclusion. Most studies examine only one aspect of status and stratification, but as Williams, Leavell, Collins, and Mohammed discuss in their paper, the greatest, most persistent impact occurs at the intersection of these different bases of disadvantage.

Causal direction

While the MacArthur network uses a model linking SES and health in which the causal pathway goes from SES (and other bases of social disadvantage) to health, the actual causal pathways are more complex. Health affects social status as well as vice versa. Kawachi, Adler, and Dow discuss some of the methodological challenges in establishing causal associations between SES and health and the current state of the evidence.

Stress

The network started by exploring biological pathways through which social disadvantage enters the body to create differential rates of disease. We noted that the specific components of SES—most generally education, income, and occupational status—were only moderately related to one another and provided different types of resources. But despite this finding, all of the specific measures were related to health status in a similar fashion. One explanation for this pattern is that the different indicators share common properties. One common element in the different components of SES is social disadvantage that results in greater exposure to stress. We conjectured that the lower individuals are on any given SES indicator, the more likely they are to be exposed to social and physical stressors.

Having identified the stress pathway as central to the SES effect on health, we looked at research on the biological effects of stress. Most of the existing human and animal research on stress examined acute exposures. However, low SES not only exposes individuals to greater acute stress but importantly it also increases exposure to chronic stress. When we began to investigate this relationship there were few models or methods for assessing chronic stress and its biological reverberations. Bruce McEwen's prior seminal work using animal models had shown hypothalamic-pituitary-adrenal (HPA) axis involvement in stress responses and demonstrated the central role of the brain in this process. Building on this work on acute stress, McEwen extended the concept of allostasis, a protective process in the short run, to identify the adverse effects of allostatic load that can emerge in the face of ongoing stress. Allostatic load provides a potential explanation for how chronic stress associated with social disadvantage could result in poorer health. In a productive collaboration, McEwen worked with Teresa Seeman and other colleagues to translate knowledge from animal studies to measurement in humans.

The concept of allostatic load has had great heuristic value. Since l997, over l50 articles using "allostatic load" as a keyword have appeared in PubMed. The underpinnings for the concept are discussed by McEwen and Gianaros who depict how the brain responds to and regulates the stresses associated with social disadvantage and the resulting impact on other biological processes. Seeman, Epel, Gruenewald, Karlamangla, and McEwen further extend this analysis to peripheral biology and describe how allostatic load accelerates the normal aging process. They discuss the measurement of allostatic load and its role in linking SES to disease (specifically cardiovascular disease) and mortality.

Social context

Stress is a central concept for understanding how social disadvantage produces ill health; it is posited to underlie the development of allostatic load, which in turn increases morbidity and morality. However, there is no standard measure of stress. Most stress researchers focus on an individual's experience of stressors, with differential impact on physiological responses depending

on the person's evaluation of the stressor's significance and degree of threat.[5] However, a full understanding of SES mechanisms requires attention to the context in which individuals encounter these events. Individuals of different socioeconomic levels inhabit social environments that differ markedly in the types of threats encountered, their frequency of exposure, and the availability of resources that can counter these threats. Network members have studied the environments associated with SES, demonstrating contextual influences, including greater challenges in neighborhoods and communities with fewer socioeconomic resources. In an early paper Taylor, Repetti, and Seeman reviewed the evidence linking unhealthy environments associated with SES and health,[6] and Diez Roux, and Mair in this volume extend our understanding of neighborhood effects on health and suggest new directions for research linking place to biology.

While most studies of context investigate the impact of neighborhoods where people reside, the work environment also plays a large role in the lives of most adults (as schools do for children). The original Whitehall study demonstrated the powerful effects of the occupational grade of British civil servants on their health and longevity. In addition to using the remarkably rich data available in the Whitehall II study, the network also built a novel data set to study the health effects of work environments among blue- and white-collar workers in a U.S. manufacturing industry. Clougherty, Souza, and Cullen examine a number of ways the work environment shapes the social gradient, including illustrations from this new industrial data set.

Psychological mediators

Psychological responses to environmental context are central to a stress response. Higher SES helps build psychological resources that allow individuals to cope more effectively with stressors. These resources provide people with greater capacity to avoid stress exposure in the first place and also enable more effective coping responses that diminish adverse physiological responses to those stressors that are encountered. Karen Matthews with Linda Gallo developed the concept of "reserve capacity" to reflect the whole set of psychological resources that accrue to those with more socioeconomic resources. Like money in the bank, these psychological resources can buffer individuals when they encounter adversity. Matthews, Gallo, and Taylor review the evidence that reserve capacity and other psychological factors account for some of the observed health effects associated with SES.

Cumulative risk across systems and the life course

The allostatic load model highlights the health risk associated with even small degrees of biological dysregulation when cumulated across systems. The theory posits that such cumulated risk creates a common pathway to the onset and progression of disparate diseases. This way of framing risk runs counter to the conventional focus on disease-specific pathways and the study of singular risk factors analyzed independently.

Our discussions of the pathways by which SES gets into the body highlight the cascade of adverse exposures and experiences that are associated with social disadvantage. This cascade of effects resulting from social disadvantage begins early in life and cumulates over one's lifetime. Early experiences can have residual effects on health many years later. Cohen, Janicki-Deverts, Chen, and Matthews demonstrate the importance of socioeconomic factors early in life and the different patterns by which childhood SES relates to health in adulthood. Evans and Kim show how cumulative risk, spanning physical and psychosocial factors, mediates the association of SES and health.

What do we do about it?

Although our network was organized around a scientific question, the research is providing answers that are relevant to policy. All members of the network were drawn to this work out

of a concern for equity and hope that our findings will contribute to the elimination of health disparities. Our aim is to allow all people to experience the health status enjoyed by the most advantaged groups. We were initially challenged to think about policy when network member Sir Michael Marmot asked for our input to the Acheson Commission, the commission charged with suggesting policies that would eliminate health disparities in Great Britain. Our thinking about policy was further stimulated by a joint meeting with the World Health Organization Commission on Social Determinants of Health, which Sir Michael chaired.

In this spirit, in addition to our academic papers, we jointly produced a booklet, *Reaching for a Healthier Life: Facts on Socioeconomic Status and Health in the United States.*[7] This booklet has been widely distributed and is on the Web;[8] it is being used by public health officials, nonprofits, and legislatures in their work tackling health disparities. The current volume provides scientific underpinning for that booklet and for policy recommendations for eliminating health disparities. Because of the crisis in health care and the plight of the uninsured, U.S. health policy has primarily been focused on the financing and problems of health care. In the final paper, Dow, Schoeni, Adler, and Stewart note the importance of nonhealth sector inputs to health. They describe the benefits of doing policy analysis from a Bayesian perspective when evaluating the impacts of nonhealth policies on health outcomes, and present a taxonomy of policies and interventions aimed at reducing SES gradients in population health.

The work in this volume emerged from a group process facilitated by the support and structure of the MacArthur Foundation program, which created a number of research networks since its inception. Adler and Stewart end the volume with a description of our network's process and evolution, and reflect on the challenges and rewards of conducting team science; we conclude, in brief, that "the juice is worth the squeeze."

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