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Allostatic Load and Health Disparities: A Theoretical Orientation*

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Abstract: Eliminating racial and ethnic health disparities requires restructuring the biomedical models that have focused on the individual as the level of analysis and emphasized the parts rather than the whole. A recently developed understanding of human physiology and adaptive regulation, constructs of allostasis and allostatic load, provides a theoretical orientation that needs to be explored. Thus, the purpose of this article is to present an orientation of allostasis and allostatic load as a theoretical framework for exploring health disparities. This article will (a) present a general background on the evolution of relevant physiologic theories, (b) offer the general theoretical definitions and explanations of allostasis, allostatic load, and mediation processes, (c) examine empirical evidence for the constructs, and (d) discuss the implications of this orientation for health disparities research.

Keywords: allostatic load; health disparities; complexity theory; African American

Eliminating racial and ethnic health disparities has become a national research priority (Department of Health and Human Services, 2005). Common explanations of poverty, access barriers, and lifestyle choices do not sufficiently account for the large disparities in risk, morbidity, or mortality in US populations. As a result, the multidimensional effects of discrimination and racism are increasingly implicated as one of the underlying factors influencing these racial and ethnic health disparities (Jones, 2000; Smedley, Stith, & Nelson, 2002). Consequently, this research agenda requires a restructuring of biomedical models that have focused on the individual as the level of analysis and emphasized the parts rather than the whole (Plsek, 2001).

Furthermore, the historic understandings of homeostasis and stress have not provided a sufficient framework to elucidate the physiological pathways that explain racial and ethnic disparities

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in health (Brunner & Marmot, 2000). One approach to these disparities is the ecological model, which seeks to understand individual morbidity and mortality within the context of the social environment (Kawachi & Berkman, 2003). However, the challenge of the ecological approach is how to think about social factors, psychological factors, and biological factors collectively and simultaneously.

A recently developed understanding of human physiology and adaptive regulation, constructs of allostasis and allostatic load, provides a theoretical orientation that needs to be explored. Thus, the purpose of this article is to present the constructs of allostasis and allostatic load as a theoretical framework for exploring health disparities. The intent is not to provide a detailed description of physiological mechanisms, but to offer an overview of the constructs and orient them toward health disparities research.

We focus on African Americans, as an exemplar of racially mediated health inequities, to examine how health disparities research might shift if oriented from the constructs of allostasis and allostatic load. We focus on African American health disparities for two reasons. First, African Americans carry one of the largest burdens of increased morbidity and mortality of any racial or ethnic group in the United States throughout the life span. Second, African Americans, as a socially defined racial category, hold a unique historical position in the context of American society. This article will (a) present a general background on the evolution of relevant physiologic theories, (b) offer the general theoretical definitions and explanations of allostasis, allostatic load, and mediation processes, (c) examine empirical evidence for the constructs, and (d) discuss the implications of this orientation for health disparities research.

EVOLUTION OF PHYSIOLOGIC THEORIES

At the beginning of the 20th century, Cannon introduced the term *homeostasis* to describe the tight regulatory environment that seemed to capture the essence of adaptation inherent to human physiology (Cannon, 1932). According to this classic concept of physiological control, the role of feedback mechanisms was to reduce variability and maintain constancy as a function of healthy systems. Cannon believed that the failed regulation of these physiological parameters was antecedent to disease. The role of the physician was to restore the physiological alterations to within normal set points. This formulation of human physiology has dominated Western biomedical training since its introduction (Guyton & Hall, 2000; Marieb, 2004).

In the 1940s, general systems theory began to infiltrate physiological thinking (Buchman, 2002). Using general systems theory, biologists were encouraged to consider an alternative model of physiology. This model included dynamic, nonlinear processes that provided homeostatic stability, contrasting with Cannan's depiction of feedback loops to control variability. Yet, even within this alternative model, underlying assumptions of the mechanical paradigm (looking at the parts rather than the whole) and biostatistical modeling (based on linear dynamics) remained inadequate to capture the complexity of systems physiology (Goldberger, Peng, & Lipsitz, 2002). In essence, mathematical models of the time were insufficient to explain complex physiological systems or account for the variability of integrative networks of adaptation to environmental stressors (Kitano, 2002; Wolkenhauer, 2001).

Seyle (1956) was the first to propose that chronic stress may have a cumulative damaging effect on human physiology. In his theory of a general adaptation syndrome, he attempted to explain physiological responses to environmental stressors. Since then, there have been many iterations of the stress model related to aging and disease (Sterling & Eyer, 1988; Wykle, Kahana, & Kowal, 1992). These advances have spawned new integrative fields of psychoneuroimmunology and psychoneuroendocrinology (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Lupien et al., 2005). In addition, such advances have helped to elucidate the links between the molecular physiological systems, the structures and function of the brain, and emotional interpretations and coping capabilities (Sapolsky, 2003).

Yet, the promise of all these advances is a greater appreciation for the complexity in physiological systems. As a result, simple homeostatic constructs of stable states and feedback loops are no longer adequate to understand integrative physiological networks that connect molecules, cells, tissue, organs, and organ systems (Goldberger et al., 2002). New physiologic and mathematical models suggest that healthy dynamic stability arises through a combination of specific feedback mechanisms and spontaneous properties of interconnected networks (Buchman, 2002). Physiologic health appears to be a function of both classical concepts of homeostasis and

more recent understandings of complex network integrity and nonlinear interactions.

As such, a defining feature of healthy function appears to be the adaptive capacity to respond to unpredictable stimuli and stresses. As one indication of the shifting physiological paradigm, the National Institutes of Health, National Center for Research Resources has established the Research Resource for Complex Physiologic Signals (Goldberger et al., 2000).

The center has established this web-based resource to stimulate new investigations into the complexity of integrative biological networks. A major feature is the shared access to biostatistical software that is not dependent on assumptions of fixed and linear variables. From this perspective, the loss of variability is proposed as the generic feature of pathologic dynamics that precede disease morbidity and aging (Goldberger et al., 2002).

ALLOSTASIS AND ALLOSTATIC LOAD

Of equal importance is a need to understand how the social environment exerts a cumulative impact on the physical and mental well being of individuals. The construct of allostasis was first developed in an effort to understand the physiological basis for disparate patterns of morbidity and mortality unexplained by socioeconomic status, access issues, or lifestyle choices (Sterling & Eyer, 1988). McEwen and Stellar (1993) later proposed the construct of allostatic load to convey the cumulative impact of progressive physiological wear and tear that could predispose biological organisms to disease. In this section, we offer the general explanations of allostasis, allostatic load, and mediation processes. Readers are referred to original sources for more detailed physiological descriptions.

Allostasis

The construct of allostasis was proposed as the process of achieving stability through change (Sterling & Eyer, 1988). This definition was meant to distinguish the dynamic, nonlinear physiological networks that support homeostatic systems through adaptive variability (Sterling & Eyer, 1988). Allostasis denotes the capacity to adapt to changing environments or stressful challenge in order to support the homeostatic systems that are essential to life. The term homeostasis is then reserved for those physiological systems that necessitate tight physiological regulation to maintain survival of the organism (e.g., body temperature, pH balance, or oxygen tension). Thus, the allostatic state allows the organism to cope physiologically, behaviorally, and emotionally with specific environmental challenges while maintaining regulatory control of the homeostatic systems that operate within narrow parameters (McEwen & Wingfield, 2003).

This distinction between homeostatic and allostatic systems is important for several reasons. First, it provides a theoretical distinction of those physiological systems of homeostasis that are designed to reduce variability to maintain life from those systems where variability is a characteristic of a healthy system. As such, homeostatic systems are those where a narrow physiological range is indicative of health and deviance from this range is an indication of pathology (e.g., an elevated body temperature). In contrast, allostatic systems are those where normal resting points vary according to dynamic biological processes and variability is a healthy adaptive mechanism to environmental challenge (e.g., heart rate). Thus, allostasis is consistent with new conceptualizations of healthy physiology as complex adaptive systems (Goldberger et al., 2002; Plsek, 2001).

Second, the distinction between homeostasis and allostasis addresses the problem of how to define stress. Difficulties understanding the physiological pathways are partly due to the ambiguous ways that stress has been defined in the past. One reason for the difficulty has existed because stress is often viewed as either a subjective experience or an external event. Yet, there are aspects of daily life events that may not be perceived as stressful but, nevertheless, exact a negative physiological toll on the body. Similarly, there are large individual variations in the subjective burden of stress incurred by major life events (Ursin & Eriksen, 2004).

Furthermore, the difficulty understanding the link between stress and physiological pathways occurs because there continues to be a limited appreciation for the nonlinear, integrative network dynamics that mediate these pathways (McEwen & Seeman, 1999). These dynamics included the structure and function of the hypothalamo-pituitary-adrenal (HPA) axis, the autonomic nervous system (ANS), and the immune system. Contrary to Selye's (1956) original emphasis on environmental stressors, researchers have demonstrated that the more powerful predictors of stress activation are individual expectations and interpretations of events (McEwen, 2000).

Third, the distinction between homeostasis and allostasis allows for the measurement of predisease mediators of pathophysiology. McEwen (2002) defined an allostatic state as the activation and altered, sustained level of the primary endogenous molecules mediating stress activity. These substances reflect engagement and activity of the adrenal cortex (glucocorticoids), the adrenal medulla (adrenalin), and the immune system (inflammatory cytokines). Thus, the distinction provides a theoretical framework of physiological processes that might be leveraged to prevent or delay the trajectory of early morbidity and mortality. This physiological trajectory underlies the allostatic load hypothesis proposed by McEwen and Seeman (1999).

Allostatic Load

While the term allostasis describes the process of adaptation, the term allostatic load is used to denote the accumulation of wear and tear on physiological systems from the adaptation process (McEwen & Seeman, 1999). Physiological wear and tear is a natural consequence of environmental adaptation. An underlying assumption is that the allostatic load of any biological organism will increase over time. However, a resilient organism with adaptive plasticity (ability to be enhanced by environmental challenges) will be able to minimize physiological wear and tear.

In terms of an individual, it is expected that the allostatic load created by person-environment interactions will increase as a function of aging (Crimmins, Johnston, Hayward, & Seeman, 2003). Yet, within any given age cohort there is a range of variation that is based on genetic predisposition, early life predisposing influences, adaptation to daily life events, and the effects of major life stressors (Lupien et al., 2005; McEwen, 2003). Excessive wear and tear on physiological systems (allostatic load) accumulates from either an overactive or inefficiently managed allostatic response to challenge. Thus, while activation of primary stress mediators is adaptive within the context of environmental challenge, excessive wear and tear is a function of atypical response patterns.

McEwen (2002) suggested four adaptive response patterns that are indicative of excessive wear and tear: (1) a response pattern with excessive and repeated insults over time, (2) a response pattern where the organism is not able to habituate to stressful stimuli, (3) a response pattern that is activated but remains at a heightened level of activation without sufficient recovery, and (4) a response pattern where the primary mechanisms are inadequate to the challenge, resulting in the activation of compensatory mechanisms. These four types of overactive or inefficiently managed allostatic responses may occur alone or in combination.

Allostatic Mediation

What these four response patterns all have in common is the activation or the inefficient management of the primary molecular mediators of the allostatic response. Thus, the distinction between genotypically induced effects and environmentally induced effects (nature vs. nurture) become less relevant, since both mediate cognitive processing and physiological response to challenge. As a result, individual interpretations of reality play a pivotal role in eliciting these physiological and behavioral responses to challenge (Lupien et al., 2005; McEwen, 2002).

Individual interpretations of reality are mediated by the hippocampus, the region of the brain responsible for the regulation of the primary stress mediators of an allostatic state. As the mediator, the hippocampus plays a crucial role in learning and memory. From a survival perspective, the hippocampus determines which sensory stimuli are important enough to encode and remember (Crawford & Cacioppo, 2002; Sapolsky, 2003). Consequently, the hippocampus is particularly important to the memory of context (the environment or circumstances where the stimulus took place) when the emotional interpretation of events have elicited an allostatic response (McEwen & Seeman, 1999).

The memory of context has important implications for the theoretical understanding of the wear and tear of allostatic load. For instance, the ability to anticipate needed physiological adaptations is based on the memory of similarly arousing stimulation. This encoded memory allows the physiological process of allostasis to be made prior to the actual occurrence of the stimuli. One type of reaction to a potentially challenging situation is an increased state of anxiety or vigilance. This is particularly true when the challenge is ill defined or when there is no alternative behavioral response to eliminate the challenge (McEwen, 2002).

Therefore, anticipation of a stressful context is enough to elicit an allostatic response. Individual differences in the interpretation of events, and the interpretation of coping capabilities, will result in

variations of physiological reactivity to the same or similar stressors (McEwen, 2003). Thus, within the allostatic model, stress becomes a function of eliciting a physiological response to challenge versus a subjective evaluation or an externally defined event. Within limits, the allostatic response is adaptive but when it occurs in excess of effective coping capabilities, such responses result in physiological overload (McEwen & Wingfield, 2003).

In this condition, the integrative network of physiologic systems produces maladaptive processes and altered systems indicators. These altered system indicators are the potential indices of physiologic dysfunction. They represent the secondary outcomes that are associated with risk of chronic disease and mortality (e.g. cardiovascular activity, adipose tissue deposit, and glucose metabolism; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). As a result, allostatic load is the process whereby the release of primary mediators leads to consequences such as receptor desensitization or tissue damage (McEwen, 2002).

CONSTRUCT VALIDITY

The distinction between allostasis and allostatic load provides two different types of physiological endpoints that can be measured. These endpoints are the primary molecular mediators of an adaptive allostatic response and the secondary outcome measures of allostatic load. Recent researchers have used a summed index of both these primary physiological mediators and secondary outcome measures to test the construct validity of this new theoretical orientation.

MacArthur Studies of Successful Aging were designed to test the construct validity of allostasis and allostatic load (Seeman et al., 1997; Seeman, McEwen, Rowe, & Singer, 2001). This research project represents a longitudinal study of three community cohorts of high-functioning, older adults (age 70–79 at baseline). To test the construct validity, a 10-item index of allostatic load was constructed based on both primary mediators of allostasis and secondary outcome measures of allostatic load (Table 1). It should be noted that these parameters represent a limited number of the potential indicators that could be used as an index of allostatic load.

In the MacArthur Studies, allostatic load was determined by summing the number of parameters for which an individual fell into the highest-risk quartile based on the distribution of scores. As

Table 1. Index of Allostatic Load Used in the MacArthur Studies of Successful Aging

Physiologic indicator	Physiologic system	Quartile parameter cutoff
Systolic blood pressure	Index of cardiovascular activity	(greater or equal to 148 mm/Hg)
Diastolic blood pressure	Index of cardiovascular activity	(greater or equal to 83 mm/Hg)
Waist-hip ratio	Index of long-term metabolism and adipose tissue deposit	(greater or equal to 0.94)
Total/HDL cholesterol ratio	Index of long-term atherosclerotic risk	(greater or equal to 5.9)
Serum HDL	Index of long-term atherosclerotic risk	(greater or equal to 7.1%)
Serum total cholesterol	Index of long-term atherosclerotic risk	(less than or equal to 1.45 mmol/L)
Serum DHEA-S	Index of functional HPA axis antagonist	(less than or equal to 910 ng/mL)
12-hour urinary cortisol	Index of HPA axis activity	(greater or equal to 25.7 mg/g creatinine)
12-hour urinary norepinephrine	Index of sympathetic nervous system activity	(greater or equal to 48 mg/g creatinine)
12-hour urinary epinephrine	Index of sympathetic nervous system activity	(greater or equal to 5 mg/g creatinine)

such, the cutoff point for each parameter held either minimal or no clinical significance when considered individually. Yet, by summing the elevated parameters, higher allostatic load scores were significantly associated with cardiovascular disease incidence at the 2.5-year follow-up and all-cause mortality at 7-year follow-up (Seeman et al., 1997; Seeman et al., 2001). This association was independent of socioeconomic indicators and baseline morbidity. In addition, higher scores on the allostatic load index were significantly associated with declines in both cognitive and physical functioning (Karlamangla, Singer, McEwen, Rowe, & Seeman, 2002; Seeman et al., 1997). The 10-item index was a better predictor than either subset of primary mediators or secondary outcome measures alone. This suggests that both subscales contribute significantly to morbidity and mortality risk index.

Several researchers have used a variant of this 10-item index, often based on convenience of available data or data collection procedures. For example, Kubzansky, Kawachi, and Sparrow (1999) used data from the Normative Aging Study to develop an 8-item index of allostatic load. Schnorpfeil et al. (2003) developed a 14-item index that added body mass index, plasma levels of C-reactive protein, tumor necrosis factor alpha, and urine albumin levels.

Finally, the MacArthur Studies researchers tested a 16-item index that included several markers of inflammation, as well as secondary markers of lung and renal function (Seeman et al., 2004). Again, the total summed index showed predictive validity over and above any individual parameter, subset of parameters, or the shorter 10-item index. This finding further supports the hypothesis that it is the cumulative index of physiological wear and tear that offers advantages in predicting health risks.

Several researchers have shown an association between psychosocial risk factors and the cumulative index of allostatic load. For example, Singer and Ryff (1999) took a life course perspective to investigate the effect that economic circumstances and social relationship patterns had on measures of allostatic load. Relationship pathways produced more discriminatory power than economic pathways to identify high versus low allostatic load scores. However, in those individuals with economic advantage throughout the life course, negative relationships did not significantly influence allostatic load. Similarly, longitudinal studies have demonstrated a link between positive psychosocial experiences and lower allostatic load in adults (Seeman, Singer, Ryff, Love, & Levy-Storms, 2002; Weinstein, Goldman, Hedley, Yu-Hsuan, & Seeman, 2003).

Alternatively, Evans (2003) combines a cumulative risk model of childhood development and a physiological assessment of allostatic load. In this study, a model of cumulative risk was used as an index of the dynamic, proximal, and distal multilevel factors associated with developmental outcomes in children. The risk model was used to develop a summary index of personal (poverty, single parenthood, maternal high school dropout status), psychosocial (child separation, turmoil, violence), and environmental (crowding, noise, and housing quality) risk. Allostatic load was measured with a 6-item index of physiological dysregulation.

Higher cumulative risk exposures were significantly associated with higher allostatic load scores. In addition, higher cumulative risk exposures were associated with higher scores of learned helplessness and lower scores in the ability to delay gratification. The processes inherent in the cumulative risk index appear to set in motion a cascade of altered physiological, cognitive, and behavioral states that are antecedent to physical and mental health over time. Although the sample population for this study was predominantly White, this research suggests that an integration of cumulative risk is associated with cumulative physiological dysfunction, a finding that has implications for health disparities research.

IMPLICATIONS FOR HEALTH DISPARITIES

To review, the construct of allostasis provides a scientific framework for recognizing that health requires adaptive capabilities. The construct of allostatic load provides a theoretical orientation directed at capturing the physiological wear and tear of adaptive challenge. Further, physiological responses to adaptive challenge depend on memory formation in the hippocampus. These memories depend on cognitive interpretations and, in turn, these cognitive interpretations regulate the molecular mediators of the allostatic response. Yet, within any given age cohort there is a range of variation of physiologic dysregulation that are antecedent to morbidity and mortality risk.

However, this physiological variation favors higher socioeconomic and racially predominant populations. The ecological approach seeks to understand these variations of individual physiology within the context of the social environment. The challenge is how to think about social factors,

psychological factors, and biological factors collectively and simultaneously. The constructs of allostasis and allostatic load hold potential to address this challenge by providing a framework to investigate both bottom-up (effects of glucocorticoids on cognitive processing) and top-down (effects of cognitive processing on glucocorticoid secretion) consequences to adaptation (Lupien et al., 2005). Our intent is to suggest how health disparities research might shift if oriented from a theoretical perspective of allostasis and allostatic load.

We examined the implications of these constructs for health disparities research using the racial category of African Americans as an exemplar. We did this because African Americans continue to carry one of the largest burdens of increased morbidity and mortality of any racial or ethnic group in the United States throughout the life span. For instance, African American infants have significantly worse birth outcomes than White infants, despite socioeconomic status, maternal alcohol or cigarette consumption, or access to early prenatal care (Lu & Halfon, 2003). Similarly, African American adults have significantly worse cancer outcomes than White adults for almost all types of cancer and at all stages of disease progression (Ward et al., 2004).

Recent Institute of Medicine reports contend that socioeconomic position alone does not account for the significant disparities in African American health (Haynes & Smedley, 1999; Smedley et al., 2002). African Americans, as a socially defined racial category, hold a unique historical position in the context of American society shaped by the legacy of slavery. Until recently, the effects of racism and discrimination from the perspective of the recipient have received little attention in public health research. We suggest that there are several reasons for this oversight that might be productively addressed from a theoretical orientation of physiological wear and tear to adaptive challenge.

First, epidemiological research has tended to focus on racial disparities as either a biological genetic attribute, a greater propensity to engage in health-damaging lifestyle choices, or as a proxy measure for low socioeconomic status (Kneipp & Drevdahl, 2003). Nevertheless, it is increasingly clear that African American health disparities reflect a form of institutionalized racism (Smedley et al., 2002; Smelser, Wilson, & Mitchell, 2001). This institutionalized racism links education, employment, socioeconomic status, housing segregation, and insurance coverage into the lived reality of African Americans. Viewed from an orientation of allostatic load, race might more accurately be conceptualized as a multidimensional social system exposure to a hostile environment (Forbes & Wainwright, 2001).

Second, until recently the majority of research on racial discrimination has focused on the White perpetuator and tended to ignore the experience of the African American recipient (Shelton, 2000). Recent work is focused on the subtle, contemporary forms of racial biases that unconsciously mediate behavior (Dovidio, Gaertner, Kawakami, & Hodson, 2002; Lowery & Hardin, 2001). This type of unconscious bias on the part of White individuals is associated with significant consequences for African Americans. Researchers have yet to explore how this type of unconscious bias on the part of African American individuals influences health or health care decision making (Smedley et al., 2002). Viewed from an orientation of allostatic load, this suggests a need to take a life course perspective of racially mediated insults to healthy physiology (Lu & Halfon, 2003).

The third reason for the relative lack of attention to the influences of racism is a substantial perception gap between African Americans and Whites when it comes to the social reality of African American lives (Carlson & Chamberlain, 2004). Most African Americans see or experience at least some type of discrimination when they come into contact with White individuals (Cose, 1993; McWhorter, 2000; White & Cones, 1999). On the other hand, White individuals tend to think that acts of racial discrimination are a rare event in contemporary society, despite consistent evidence to the contrary (Smedley et al., 2002; Smelser et al., 2001; Steinhorn & Diggs-Brown, 1999). Viewed from an orientation of allostatic load, the exploration of this perception gap might provide useful information about racially mediated health disparities (Kagawa-Singer & Lassim-Lakha, 2003). For instance, recognition might facilitate investigation into cross-cultural mis-interpretations that influence physiologic and behavioral outcomes. Further, recognition might shift the locus of intervention away from behavioral change strategies targeted at African Americans to interventions targeted at the dominant, mainstream society.

Thus, health disparities research might benefit from the conceptual shifts necessitated by the theoretical framework of allostasis and allostatic load. The constructs are oriented toward a life course perspective and a multi-system view of adaptive physiological responses. Conversely, the ecological approach seeks to understand individual morbidity and mortality within the context of

the social environment. We suggest that these two orientations can be integrated to align with the new advances from systems biology and the science of complex adaptive systems (Buchman, 2002; Kitano, 2002; Plsek, 2001).

Situated in this way, the ecological model becomes an extension of hierarchical dynamic systems. This orients the approach toward the quality of complex, nonlinear, adaptive interactions rather than simply the variables or agents involved in the interactions. Evans (2003) has attempted to do this using a cumulative index of developmental risk factors and a physiological index of allostatic load. In doing so, he demonstrates that there is a link between the multitude of personal, psychosocial, and environmental stressors and the physiological dysregulation of allostatic load even in childhood.

Similarly, Kendall and Hatton (2002) used the allostatic load framework to suggest that there are many unexplored stressors on Black children, and their families, when the clinical problem is attention deficit hyperactivity disorder. These stressors include not only those encountered in the client–service interface of the health care arena, but the sum of encounters with social systems and environmental stressors that add layers of adaptive challenge to both child and family. As a result, these multiple layers of complex relationships can now be situated into a physiological orientation of cumulative wear and tear.

CONCLUSION

We have explored the theoretical constructs of allostasis and allostatic load and related this physiological orientation to African American inequalities in health. The constructs of allostasis and allostatic load provide a productive theoretical orientation to explore, understand, and intervene to eliminate health disparities. This orientation requires a conceptual model of health that integrates a life course perspective with the multilevel biological and environmental dimensions of the social hierarchies.

We believe that the boundaries of these social hierarchies are a pivotal point of discovery. At these boundaries is where diverse cultural relationships, affected by historically created racial assumptions, interpretations, and behavioral responses, are played out. It is these underlying assumptions, interpretations, and relationship patterns that need to be the focus of research and interventions.

> Sickness is not just an isolated event, nor an unfortunate brush with nature. It is a form of communication—the language of the organs—through which nature, society, and culture speak simultaneously. The individual body should be seen as the most immediate, the proximate terrain where social truths and social contradictions are played out.
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> Scheper-Hughes & Lock (1987, 41p)

REFERENCES

Brunner, E., & Marmot, M. (2000). Social organization, stress, and health. In M. Marmot & R.G. Wilkinson (Eds.), Social determinants of health (pp. 17–43). New York: Oxford University Press.

Buchman, T.G. (2002) The community of self. Nature, 420, 246–251.

Cannon, W.B. (1932). The wisdom of the body. New York: W.W. Norton.

Carlson, E.D., & Chamberlain, R.M. (2004). The Black-White perception gap and health disparities research. Public Health Nursing, 21, 372–379.

Crawford, L.E., & Cacioppo, J.T. (2002). Learning where to look for danger: Integrating affective and spatial information. Psychological Science, 13, 449–453.

Crimmins, E.M., Johnston, M., Hayward, M., & Seeman, T. (2003). Age differences in allostatic load: An index of physiological dysregulation. Experimental Gerontology, 38, 731–734.

Cose, E. (1993). The rage of the privileged class. New York: Harper Perennial.

Department of Health and Human Services. Healthy people 2010. Retrieved January 29, 2005 from http://www.healthypeople.gov.

Dovidio, J.F., Gaertner, S.L., Kawakami, K., & Hodson, G. (2002). Why can't we just get along? Interpersonal biases and interracial distrust. Cultural Diversity & Ethnic Minority Psychology, 8, 88–102.

Evans, G.W. (2003). A multimethodological analysis of cumulative risk and allostatic load among rural children. Developmental Psychology, 39, 924–933.

Forbes, A., & Wainwright, S.P. (2001). On the methodological, theoretical and philosophical context of health inequalities research: A critique. Social Science & Medicine, 53, 801–816.

Goldberger, A.L., Amaral, L.A., Glass, L., Hausdorff, J.M., Ivanov, P.C., Mark, R.G., et al. (2000). PhysioBank, PhysioToolkit, and PhysioNet: Components of a new research resource for complex physiological signals. Circulation, 101, E215–E220.

Goldberger, A.L., Peng, C.K., & Lipsitz, L.A. (2002). What is physiologic complexity and how does it change with aging and disease? Neurobiology of Aging, 23, 23–26.

Guyton, A.C., & Hall, J.E. (2000). Textbook of medical physiology (10th ed.). Philadelphia: W.B. Saunders Co.

Haynes, M.A., & Smedley, B.D. (1999). The unequal burden of cancer: An assessment of NIH research and progress for ethnic minorities and the medically underserved. Washington, DC: National Academy Press.

Jones, C.P. (2000). Levels of racism: A theoretic framework and a gardener's tale. American Journal of Public Health, 90, 1212–1215.

Kagawa-Singer, M., & Lassim-Lakha, S. (2003). A strategy to reduce cross-cultural miscommunication and increase the likelihood of improving health outcomes. Academic Medicine, 78, 577–587.

Karlamangla, A.S., Singer, B.H., McEwen, B.S., Rowe, J.W., & Seeman, T.E. (2002). Allostatic load as a predictor of functional decline: MacArthur Studies of successful aging. Journal of Clinical Epidemiology, 55, 696–710.

Kawachi, I., & Berkman, L.F. (2003). Neighborhoods and health. New York: Oxford University Press.

Kendall, J., & Hatton, D. (2002). Racism as a source of health disparity in families with children with ADHD. ANS: Advances in Nursing Science, 25, 22–39.

Kiecolt-Glaser, J.K., McGuire, L., Robles, T.E., & Glaser, R. (2002). Psychoneuroimmunology and psychosomatic medicine: Back to the future. Psychosomatic Medicine, 64, 15–28.

Kitano, H. (2002). Systems biology: A brief overview. Science, 295, 1662–1664.

Kneipp, S.M., & Drevdahl, D.J. (2003). Problems with parsimony in research on socioeconomic determinants of health. ANS: Advances in Nursing Science, 26, 162–172.

Kubzansky, L.D., Kawachi, I., & Sparrow, D. (1999). Socioeconomic status, hostility, and risk factor clustering in the Normative Aging Study: Any help from the concept of allostatic load? Annals of Behavioral Medicine, 21, 330–338.

Lowery, B.S., & Hardin, C.D. (2001). Social influence effects on automatic racial prejudice. Journal of Personality & Social Psychology, 81, 842–855.

Lu, M.C., & Halfon, N. (2003). Racial and ethnic disparities in birth outcomes: A life-course perspective. Maternal and Child Health Journal, 7, 13–30.

Lupien, S.J., Fiocco, A., Wan, N., Maheu, F., Lord, C., Schramek, T., et al. (2005). Stress hormones and human memory function across the life span. Psychoneuroendocrinology, 30, 225–242.

Marieb, E.N. (2004). Human anatomy and physiology (6th ed.). San Francisco: Pearson Benjamin Cummings.

McEwen, B.S. (2000). The neurobiology of stress: From serendipity to clinical relevance. Brain Research, 886, 172–189.

McEwen, B.S. (2002) Sex, stress and the hippocampus: Allostasis, allostatic load and the aging process. Neurobiology of Aging, 23:921–939.

McEwen, B.S. (2003). Interacting mediators of allostasis and allostatic load: Towards an understanding of resilience in aging. Metabolism, 52, 10–16.

McEwen, B.S., & Seeman, T. (1999). Protective and damaging effects of stress mediators: Elaborating and testing the concept of allostasis and allostatic load. Annals of the New York Academy of Sciences, 896, 30–47.

McEwen, B.S., & Stellar, E. (1993). Stress and the individual. Mechanisms leading to disease. Archives of Internal Medicine, 153, 2093–2101.

McEwen, B.S., & Wingfield, J.C. (2003). The concept of allostasis in biology and biomedicine. Hormones & Behavior, 43, 2–15.

McWhorter, J. (2000). Losing the race: Self-sabotage in Black America. New York: Perennial.

Plsek, P. (2001). Redesigning healthcare with insights from the science of complex adaptive systems. In Crossing the quality chasm: A new health system for the 21st Century (pp. 309–322). Washington, DC: National Academy Press.

Sapolsky, R.M. (2003). Stress and plasticity in the limbic system. Neurochemical Research, 28, 1735–1742.

Scheper-Hughes, N., & Lock, M. (1987). The mindful body: A prolegomenon to future work in medical anthropology. Medical Anthropology Quarterly, 1, 6–41.

Schnorpfeil, P., Noll, A., Schulze, R., Ehlert, U., Frey, K., & Fischer, J.E. (2003). Allostatic load and work conditions. Social Science & Medicine, 57, 647–656.

Seeman, T.E., Crimmins, E., Huang, M.-H., Singer, B., Bucur, A., Gruenewald, T., et al. (2004). Cumulative biological risk and socio-economic differences in mortality: MacArthur Studies of Successful Aging. Social Science & Medicine, 58, 1985–1997.

Seeman, T.E., McEwen, B.S., Rowe, J.W., & Singer, B.H. (2001). Allostatic load as a marker of cumulative biological risk: MacArthur Studies of Successful Aging. Proceedings of the National Academy of Sciences of the United States of America, 98, 4770–4775.

Seeman, T.E., Singer, B.H., Rowe, J.W., Horwitz, R.I. & McEwen, B.S. (1997). Price of adaptation–allostatic load and its health consequences: MacArthur Studies of Successful Aging. Archives of Internal Medicine, 157, 2259–2268.

Seeman, T.E., Singer, B.H., Ryff, C.D., Love, G.D., & Levy-Storms, L. (2002). Social relationships, gender, and allostatic load across two age cohorts. Psychosomatic Medicine, 64, 395–406.

Seyle, H. (1956). The stress of life. New York: McGraw-Hill.

Shelton, J.N. (2000). A reconceptualization of how we study issues of racial prejudice. Personality & Social Psychology Review, 4, 374–390.

Singer, B., & Ryff, C.D. (1999). Hierarchies of life histories. Annals of the New York Academy of Sciences, 896, 96–115.

Smedley, B.D., Stith, A.Y., & Nelson, A.R. (2002). Unequal treatment: Confronting racial and ethnic disparities in health care. Washington, DC: National Academy Press.

Smelser, N.J., Wilson, W.J., & Mitchell, F. (2001). America becoming: Racial trends and their consequences, Vol. II. Washington, DC: National Academy Press.

Steinhorn, L., & Diggs-Brown, B. (1999). By the color of our skin: The illusion of integration and the reality of race. New York: Dutton.

Sterling, P., & Eyer, J. (1988). Allostasis: A new paradigm to explain arousal pathology. In S. Fisher & J. Reason (Eds.) Handbook of life stress, cognition and health (pp. 629–649). New York: Wiley.

Ursin, H., & Eriksen, H.R. (2004). The cognitive activation theory of stress. Psychoneuroendocrinology, 29, 567–592.

Ward, E., Jemal, A., Cokkinides, V., Singh, G.K., Cardinex, C.C., Ghafoor, A., et al. (2004). Cancer disparities by race/ethnicity and socioeconomic status. Cancer: A Cancer Journal for Clinicians, 54, 78–93.

Weinstein, M., Goldman, N., Hedley, A., Yu-Hsuan, L., & Seeman, T. (2003). Social linkages to biological markers of health among the elderly. Journal of Biosocial Science, 35, 433–453.

White, J.L., & Cones, J.H. (1999). Black man emerging: Facing the past and seizing a future in America. New York: Routledge.

Wolkenhauer, O. (2001). Systems biology: The reincarnation of systems theory applied in biology? Briefings in Bioinformatics, 2, 258–270.

Wykle, M.L., Kahana, E., & Kowal, J. (1992). Stress and health among the elderly. New York: Springer.