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Allostasis, Homeostasis, and the Costs of Physiological Adaptation, edited by Jay Schulkin. Cambridge: University of Cambridge Press, 2004. 384 pp. $100 (hardcover).

For much of the past century the study of physiology has rested on the bedrock assumption that our bodies maintain a constant internal environment through the action of a network of homeostatic negative feedback loops. As one example, the central nervous system monitors onboard energy reserves through adipose-derived hormones, such as leptin, and adjusts energy intake and expenditure in a fashion that tends to defend a stable body weight. Similarly, when a sugary meal is consumed and blood glucose rises, the pancreas produces insulin, helping tissues remove the excess glucose, thereby maintaining blood sugar within narrow functional limits. Through the action of such regulatory processes our bodies preserve a stable internal environment against the backdrop of our ever-changing physical and social surroundings.

Or do they? If our physiologies are indeed self-correcting, as all physiology textbooks would have us believe, then how do we explain the current epidemic of obesity, the rising rates of hypertension or elevated fasting glucose, or the modern epidemic of psychopathologic states such as depression? The lion's share of contemporary health problems in countries such as the United States points to the tendency of our bodies to *change* their target state as environments change. These phenomena are not easily explained by traditional models of homeostasis.

To help account for such trends, the concept of allostasis, or "stability through change," has been proposed as an amendment to the homeostatic model (Sterling and Eyer 1988). In *Allostasis, Homeostasis, and the Costs of Physiological Adaptation,* edited by psychologist Jay Schulkin, the leaders of this emerging field make a strong case for taking the allostasis model seriously. The book's eight full-length chapters, introduction, and concluding commentary cover the history and theory of the allostasis model and its application in such fields as psychology, biomedicine, and aging research. After reading these contributions, one cannot help but realize that traditional models of physiologic regulation require updating and that allostasis holds promise as a way to move the field forward, even though the concept's advocates are not yet consistent in how they define it.

Following Schulkin's brief historical introduction, Peter Sterling—a coauthor of the original allostatic model—provides a wide-ranging discussion of allostasis, including an entertaining firsthand account of its history and a detailed consideration of how allostasis differs from homeostasis. To Sterling, an allostatic system is one in which the set points that govern internal states, such as

the target range of blood pressure that the body attempts to protect, are not static but shift in response to both acute and sustained changes in demand. When systems are forced to operate outside their normal range, this is metabolically costly, but it also reduces the sensitivity of the system by pushing it to the fringes of its detection limits. When this deviation is sustained, the body compensates by recentering the system on this outlier state, a process of "continually rematching outputs to expected inputs" (p. 30). In Sterling's model of allostasis, then, systems are indeed self-correcting, but not in the classic sense of a homeostatic system defending a static internal state. Rather, regulatory systems adjust their structure to maximize their *efficiency* and *sensitivity* as the ranges under which they are forced to operate shift.

The allostatic model further differs from homeostasis in its emphasis on the brain-body interface, exemplified by the capacity of many systems to make adjustments that are predictive of future demand. For instance, the body's stress-induced mobilization of glucose is not a reaction to increased need, but a change made in *anticipation* of the increased demand for energy that predictably follows a stressful challenge. The central monitoring and regulation of many systems makes possible such feed-forward adaptive processes, and this anticipatory brain-body theme is an integral component of the allostatic model that differentiates allostasis from homeostasis.

After Sterling's overview, three chapters explore the health consequences of chronic overstimulation of allostatic systems, described as allostatic load. Bruce McEwan has been a primary proponent of this view, and his chapter provides an extensive introduction to the concept. His model differs somewhat from Sterling's; McEwan identifies a series of true homeostatic systems that strive to maintain constant levels of the most basic physical requirements of life, such as pH and oxygen tension. These systems, in turn, are embedded within and protected by a network of allostatic systems that interface more directly with the external world and thus are required to have a more adaptable operating range. In the allostatic model it is the downstream effects of chronic overstimulation of these higher-level systems, notably the hypothalamic-pituitary-adrenal, the sympathetic-adrenal-medullary, and inflammation, that account for the deleterious health and functional effects of stress.

In a pithy chapter devoted to the merging of homeostasis with the concept of allostatic load, David Goldstein emphasizes the need to consider multiple homeostatic systems regulated in parallel and governed by dynamic rather than static settings. In the final contribution devoted to allostatic load, Pete Singer and colleagues survey strategies available to operationalize allostatic load, conceptualized here as the clustering of multiple risk factors. Drawing from their work on the McArthur Aging Study, they discuss the use of summary indexes derived from dichotomous risk categories, the use of standardized scores and canonical correlation to derive summary indexes, and finally, a method of recursive partitioning that provides a more nuanced picture of how risk factors relate to psychosocial experiences across the life cycle.

The idea of allostatic load is gaining wider acceptance in biomedical and multidisciplinary social science approaches to health and aging. It is unfortunate, therefore, that it is often described as a process of physical damage. As a typical example from this volume, Singer and colleagues define allostatic load as "the cumulative biological burden exacted on the body through attempts to adapt to life's demands," or the "wear and tear" that comes when systems are forced to chronically operate outside their normal range (p. 113). Such definitions suggest a nonreversible ratcheting up of damage through chronic stress-induced overuse. Although appealing for its intuitive simplicity, it is not clear that this mechanical metaphor is a fair descriptor of many of the traits that are used to define allostatic load, including the components of metabolic syndrome that are central to the model.

An example is illustrative. Many Aboriginal Australian populations have what would be defined as a high burden of allostatic load, because hypertension, overweight, and diabetes have become common in the wake of recent lifestyle change. In a classic study a group of overweight adults suffering from several metabolic abnormalities volunteered to temporarily revert to a lifestyle of hunting and gathering in their native countryside. This involved an increase in physical activity and consumption of a leaner diet of gathered foods and wild game (O'Dea 1984). After seven weeks of this intervention, the participants lost weight, and their insulin sensitivity and lipid profiles were markedly improved. Given the reversibility of major components of metabolic syndrome in response to changes in lifestyle and diet, in what sense was this group's former poor metabolic state a reflection of cumulative wear and tear? By shoehorning all risk factors into a poorly defined black box of physical damage, the allostatic load model would explain little in this case while obscuring the complex pathways that contribute to and maintain risk factor clustering in individuals with metabolic syndrome (Wise 2004). Although summary indexes of health and function certainly have value, a more nuanced conceptual model than wear and tear seems appropriate.

Two chapters extend discussion of allostasis to its application in psychology and neuroscience. George Koob and Michel LeMoal provide a concise review of allostasis and its application to the problem of drug addiction, which they characterize as a transition to a chronic allostatic state in the brain's reward centers, compounded by insufficient recovery time between exposures. Building on similar themes, Jeffrey Rosen and Jay Schulkin's contribution provides a more involved discussion of how the adaptive trait of fear can, through a series of progressive feed-forward allostatic resets, spiral into the psychopathology of anxiety and depression.

In a well-written chapter on chronobiology, Ziad Boulos and Alan Rosenwasser note that, for systems such as the hypothalamic-pituitary-adrenal system, "the defended level of a physiological parameter is a 24-hour curve, rather than a flat line" (p. 234). They argue convincingly that changes in the operating ranges of physiologic systems must be decomposed into underlying circadian and other programmed components before deviations resulting from stress or

allostasis can be evaluated. A final contribution by John Wingfield, with the promising title of "Allostatic Load and Life Cycles," introduces a complex model that, in the end, does not articulate well with the volume's other chapters. At the core of Wingfield's proposal is a reframing of allostatic load as resulting from chronically expending more energy on a system than the organism's metabolic budget is capable of sustaining. As noted in the concluding commentary by Michael Power, this model fails to account for the fact that many common expressions of allostatic load, such as hypertension, high cholesterol, and central obesity, are not a result of limited energy but of chronic energy excess.

Not unlike the physiology that it describes, allostasis is still a concept in flux, and it is perhaps fitting that the volume's contributions fail to converge on a single definition or working model. For Sterling, allostasis is simply "predictive regulation," whereas others use the term to describe shifting set points more generally. Several of the contributors seem troubled by the blurry line differentiating allostasis from prior attempts to recast homeostasis in more dynamic terms, such as rheostasis. Michael Powers is refreshingly forthright in his evaluation of the strengths and limitations of the concept in his closing commentary, but a coordinated effort to address differences in viewpoint would have been helpful and might have resulted in a more coherent synthesis. Repetition of core concepts has the benefit of making each chapter self-contained, but it adds to the sense that this book is a collection of independent papers rather than a concerted effort at integration.

But these are minor blemishes in what is otherwise an interesting and thought-provoking set of contributions. The book is well produced and edited and is relatively free of errors. Each chapter includes an abundance of helpful figures and tables, and the index is comprehensive. The contributions are targeted to a technical audience and will primarily be of interest to academics or graduate students working on issues of stress, psychoneuroendocrinology, physiology, endocrinology, aging, and psychology. Although this book may not be the last word on allostasis, it is certain to stimulate fruitful discussion of the fundamental principles of physiologic regulation and, one hopes, a refinement of the important concepts that the field is still grappling to synthesize.

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