

Preface

Within the United States, beginning in the late 1980s, several chronic conditions began epidemic increases of both incidence and prevalence. These conditions included asthma, obesity, obesity-related diseases such as diabetes, certain cancers, and Alzheimer's disease (AD). Scientific papers duly reported the existence of genes that putatively made their carriers more vulnerable to these conditions than the general public. However, the genetic composition of US populations did not change so rapidly as to be a basis for these rapid epidemics. HIV/AIDs and tuberculosis, although infectious, are classed as chronic diseases and also became epidemic in the United States during these years. Mental disorders also form an important set of persistent chronic conditions having "environmental" correlations that challenge simple genetic etiologies.

Furthermore, many of these epidemics had geographic foci at multiple levels of organization: neighborhood, municipality, county, state, and country. Many conditions formed "syndemics"—multiple epidemics with the same geography. Poor neighborhoods of color showed heightened incidence and prevalence within cities and formed the centers from which the epidemics spread. Poor neighborhoods that had experienced residential upheavals and instabilities particularly showed great vulnerability. Cities with residential and economic instability showed higher incidence/prevalence than others and formed the foci of spread between metropolitan regions of the country. The states of the Southeast, characterized by extreme social and economic inequality, rigid socioeconomic hierarchy over time, low educational attainment, and a culture-based ideology of individualism, showed the highest incidence/prevalence of obesity, obesity-related diseases and mortalities, AIDS, and AD mortality.

Maps of prevalence and incidence reflect complex cognitive processes. Socioeconomic and environmental signals impinge on populations and their individual constituents, having varying sensitivity and vulnerability. Sensitivity and vulnerability are determined by the interlinked factors of culture, historical trajectory, material resources, social structure and stability, and, most centrally, public policies that can mitigate or exacerbate these factors.

Individuals and populations are cognitive—able to choose one or a few responses from a larger set of those possible—and act in a multilevel linked process against, or consonant with, these signals. Within individuals, the signals and responses involve internal cognitive processes, for example, the “fight or flight” response to a perceived threat, a response involving the brain, pituitary gland, adrenal gland, circulatory system, and energy regulatory mechanisms. Epigenetic changes triggered by the signals or the initial recognition of the signals induce cognitive genetic processes as well.

The strong influence of cognitive processes at levels of organization from the nation down to the molecular biology of individuals implies that information theory, control theory, and mathematical modeling based on them can describe and predict patterns of chronic disease in cultural and socioeconomic contexts. In this book, we develop statistical models of such processes that are akin in spirit to—but different from—ordinary regression models and report empirical data and analyses to illustrate this paradigm.

The first six chapters use the asymptotic limit theorems of information theory to understand how epigenetic context affects organismal development by invoking a cognitive paradigm for gene expression. A simple argument suggests that epigenetic information sources act as analogs to a tunable catalyst, directing development to different characteristic pathways in a manner similar to ecosystem resilience shifts. The results have significant implications for epigenetic epidemiology, showing how environmental stressors, in a large sense, can induce a spectrum of chronic disorders.

Chapter 7 examines the US obesity epidemic from this viewpoint. Chapters 8 and 9 apply the perspective to heart disease and cancer and, in their later sections, introduce tools from control theory that illuminate the central role of “environmental” context. Chapters 10–12 apply the general model to a number of diseases broadly associated with obesity that are becoming pandemic, using US data at different scales of observation. Chapters 13 and 14 study how “culture” and “environment” affect the onset and progression of mental disorders. Chapters 15–18 provide case histories involving low weight births, “Right-to-work” laws and AD, diabetes and thyroid cancer in Manhattan’s Chinatown, and the etiology of violence and obesity. Chapter 19 explores a composite model of psychopathology, sleep, and culture in the context of environmental insult.

The theory appears to work very well at individual and simple aggregate levels for a number of chronic conditions in stressed populations. Environments that can be characterized as having regularities of “grammar” and “syntax” can interact with organismal development via epigenetic catalysis to literally write distorted images of themselves onto the human life course in a highly pleiotropic and often punctuated manner, producing trajectories to a range of serious dysfunctions. Communities, in which individuals respond collectively, however, display more complicated patterns of chronic disease that may represent another example of a “mesoscale resonance” in which the dynamics of ecological keystone structures entrain phenomena at other scales.

One implication of this work is that pandemic chronic diseases at both the individual, via pleiotropy, and the population scales, via collective modalities

of gene expression, are unlikely to respond to individual-level—i.e., medical—interventions in the face of serious, persistent individual and community stress and may require large-scale changes in public policy and resource allocation for their amelioration. Drugs powerful enough to affect deleterious epigenetic programming are likely to trigger profound iatrogenic “side effects” that, over the life course, would not only obviate the intervention but most likely lead to shortened life spans.

The book, a synthesis across a number of peer-reviewed publications and additional material written for this volume, can be read at several levels. Chapters 1 and 6–20 form a natural introductory unit that can be followed by the more mathematical sections as desired. Mathematical details are collected in an appendix. However, the control theory methods introduced in the latter parts of Chaps. 8 and 9 are used extensively in Chaps. 14 and 19.

In sum, we provide something of a badly needed corrective to simplistic “genetic” explanations of chronic disease that, politically, are inherently useful for blaming the victim and undercutting intervention strategies seen as disruptive to the interests of ruling elites. We focus instead on the generation of disease phenotypes by social forces that can be mitigated or amplified by public policy.

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