EPIDEMIOLOGY AND THE PEOPLE'S HEALTH

THEORY AND CONTEXT

NANCY KRIEGER

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Nancy Krieger

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Epidemiology at any given time is something more than the total of its established facts. It includes their orderly arrangement into chains of inference, which extend more or less beyond the bounds of direct observation. Such of these chains as are well and truly laid guide investigations to the facts of the future; those that are ill made fetter progress. But it is not easy, when divergent theories are presented, to distinguish between those which are sound and those which are merely plausible.

Wade Hampton Frost
"Introduction" to *Snow on Cholera*
New York: Commonwealth Fund, 1936; ix.

Both thinking and facts are changeable, if only because changes in thinking manifest themselves in changed facts. Conversely, fundamentally new facts can be discovered only through new thinking.

Ludwick Fleck
Genesis and Development of a Scientific Fact
Chicago: University of Chicago Press,
1979 (1935); 50–51.

Once we recognize the state of the art is a social product, we are freer to look critically at the agenda of our science, its conceptual framework, and accepted methodologies, and to make conscious research choices.

Richard Levins and Richard Lewontin
"Conclusion" of *The Dialectical Biologist*
Cambridge, MA: Harvard University Press,
1987; 286.

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Preface

Why a Book on Epidemiologic Theory?

Epidemiologic theory. As a phrase, it sounds at once dry and arcane. Yet, in reality, it is vital and engaging. Epidemiologic theory is about explaining the people's health. It is about life and death. It is about biology and society. It is about ecology and the economy. It is about how the myriad activities and meanings of people's lives—involving work, dignity, desire, love, play, conflict, discrimination, and injustice—become literally incorporated into our bodies—that is, embodied—and manifest in our health status, individually and collectively. It is about why rates of disease and death change over time and vary geographically. It is about why different societies—and within societies, why different societal groups—have better or worse health than others. And it is about essential knowledge critical for improving the people's health and minimizing inequitable burdens of disease, disability, and death.

In other words, epidemiologic theory is about the health status of populations—in societal and ecological context. It is not about why specific individuals become ill or stay healthy. Epidemiologic theory instead seeks to explain extant and changing population distributions of health, disease, and death, within and across societies, over time, space, and place. To fulfill this expansive mandate, epidemiologic theory necessarily must engage with a whole host of other theories relevant to explaining society, biology, population dynamics, the mechanisms of disease causation, and the processes that promote health—along with theories pertaining to probability, statistics, and causal inference. And it does so to tackle epidemiologic theory's defining question: Who and what determines population rates and distributions of morbidity, mortality, and health?

Not that there is one answer to this question—or one theory to tackle it, let alone one disciplinary approach. Instead, a wide array of academic fields, in the "social," "natural," public health, and biomedical sciences have engaged in asking questions about aspects of population health. What distinguishes the epidemiologic theories and approaches is their obligate engagement with defining and measuring health outcomes and exposures in populations and empirically testing hypotheses to explain the observed population rates and risks of the outcomes under study.

Accordingly, epidemiologic theory is a particular *type* of theory, concerned with explaining the distribution and causes of population patterns of health, disease, and well-being–that is, the substantive phenomena that comprise the domain of epidemiologic inquiry (Krieger, 1994; Krieger, 2001). Thus, just as evolutionary *theory* encompasses a variety of complementary and competing theories to explain the *fact* of biological evolution (Mayr, 1982; Eldredge, 1999; Gould, 2002), so too does epidemiologic *theory* include myriad complementary and competing theories to explain the *fact* of differential distributions of population health. Although sharing a common focus on the population patterning of health, the specific explanations nevertheless depend on choice of epidemiologic theory. Are the explanations to be found in individuals' choices? In the actions of institutions? In the interactions of nations? In the characteristics of particular pathogens, toxins, or other biophysical exposures? In the nucleotide sequences of the genome? In how work, the economy, and political systems are organized and families and relationships are constituted? In how people interact with the rest of the ecosystem? Or somewhere else?

One might consider the sorts of questions posed by epidemiologic theory to be compelling. As a practicing epidemiologist, I certainly do. As would, I imagine, many others concerned about people's health—whether in terms of disparate burdens of disease, of premature mortality, or of harmful social, physical, chemical, and biological exposures encountered at home, at school, at work, in the neighborhood, or other contexts.

And yet.

Meaning: given the issues at stake, one would think there would be plenty of books—or even just articles—on the topic of epidemiologic theory. But there aren't.

I provide the evidence for this assertion in Chapter 1, along with my thoughts on why this gap in the literature exists. And throughout I make the case that analysis and development of epidemiologic theory is essential for two reasons: one intellectual, one empirical.

—The *intellectual argument* is that epidemiology, like any science, needs theory to explain the phenomena in its specified domain. For epidemiology, this means theory to explain extant and changing population health profiles, so as to inform efforts to prevent disease, improve population health, and reduce health inequities. Understanding the strengths and limitations of diverse epidemiologic theories, and their origins and applications, is essential for improving the intellectual rigor, moorings, and creativity of the field.

—The *empirical argument* is that without explicit and transparent theory—as is currently the case in most epidemiologic textbooks and articles—we are likely to pose poorly-conceived hypotheses, inadequately interpret our findings, and potentially generate dangerously incomplete or wrong answers.

The overall premise is that theoretical clarity about the substantive questions epidemiology poses can improve the odds of generating valid—and potentially useful—knowledge. Theory is essential for formulating, testing, and assessing competing explanations—in other words, for good science. And good science, in turn, is a precondition for science that can make a difference for the good.

The book begins by arguing, in Chapter 1, that epidemiologic theory is a practical necessity for thinking about and explaining disease distribution. Chapter 2, concerned with theories about disease occurrence in various ancient and also contemporary traditional societies, introduces a range of ways that diverse peoples in various contexts, over time, have sought to explain their society's patterns of health and disease, as influenced by both their societal and ecologic context. Chapter 3 applies this analytic perspective to the emergence of epidemiology as a self-designated discipline and considers the range of competing theories of disease distribution employed between 1600 and 1900, with a focus on poison, filth, class, and race. Chapter 4 extends these analyses to encompass the first half of the twentieth century, whose theories focused on germs, genes, and the (social) environment. Chapter 5 then turns critical attention to the biomedical and lifestyle approaches dominating epidemiologic theorizing and research since the mid-twentieth century. Chapter 6 offers a systematic summary of the main alternatives to the dominant framework, as provided by the theories of the two main trends in social epidemiology: its sociopolitical and psychosocial frameworks. Chapter 7 introduces a newer variant of social epidemiologic theory: ecologically informed approaches, in particular the ecosocial theory of disease distribution that I first proposed in 1994 (Krieger, 1994) and its systematic linking of social and biological processes across levels and in relation to diverse spatiotemporal scales, as informed by both political economy and political ecology. As a final argument for why epidemiologic theory matters, Chapter 8 presents four cases examples illustrating how people's health can be harmed—or aided—depending on choice of epidemiologic theory and concludes by arguing that the science of epidemiology can be improved by consciously embracing, developing, and debating epidemiologic theories of disease distribution.

The impetus for me to write this book is the same as that for the course I first created and taught in 1991 to address the issue of epidemiologic theory in societal context and which I have been teaching, with modification, ever since. Recognizing the strengths and gaps in my own training as an epidemiologist (having obtained my master degree in 1985 and my Ph.D in 1989), and drawing on my background in biochemistry and biology, my interest in the history and philosophy of science, and my commitment to research and activism regarding the profound links between social justice and public health, I wanted to create a course that would address what I perceived as a huge lacuna in my education: a profound silence on the topic of epidemiologic theory. I accordingly designed my course to introduce others to ideas and literatures that I found relevant for my conceptual and empirical work as an epidemiologist. This path of inquiry has been, and continues to be, an ongoing intellectual journey, informed by the stark circumstances of people's lives I have encountered via my work and also the colleagues I have met along the way. The goal throughout has been to attain a better understanding of the realities of—and the possibilities for—the people's health.

While engaged in writing this book, I have of course been conscious that its content inevitably reflects my individual interests, experiences, and limitations as a scholar. In particular, I am acutely aware that my fluency only in English, coupled with my passable ability to read scientific texts in Spanish and French, restricts the primary literature I can analyze, such that I must rely on expert translation of works in all other languages. That said, English currently is, for good or for bad, the dominant language of scientific texts on epidemiologic theory and research. My hope is that my linguistic limitations nevertheless do not unduly restrict the ideas presented or their relevance to the majority of the world's people who speak languages other than English. To help ensure the cited texts speak in their own voice, and also to acquaint readers with the variety of expressions and ideas employed, I frequently use textboxes to accompany the analysis I present in my own words. Any errors in fact or interpretation are my responsibility alone.

Finally, I end this preface by acknowledging my debt to the many whose work, lives, and thoughts inform this text and the research on which it is based. Their contributions only just begin to summarized by the book's bibliography. In particular, I offer deep thanks to my mentors: Ruth Hubbard, who taught me it was not only possible but essential to think critically and historically about science and its inextricable links to concerns about social justice, while still doing—and not only critiquing—the science; Noel S. Weiss, who taught me to be an epidemiologist; and S. Leonard Syme, who gave full support to my becoming a social epidemiologist.

With regard to institutional support, I first thank Lisa Berkman, who served as chair of my department from 1995 to 2008, was enthusiastic about this project from the start, and who permitted me to work on it in lieu of teaching my course during the 2009 to 2010 academic year. I likewise thank the many students who, as my successive literature search assistants, have tracked down many an obscure book and article; they include, in chronological order since 2002, when I began more concretely planning for this project: David Rehkopf, David Chae, Malavkia Subramanyam, Shalini Tendulkar, and Marlene Camacho. I also thank the editors I have worked with at Oxford University Press—Jeffrey House, William J. Lamsback, Regan Hofmann, and Maura Roessner—for their interest and encouragement.

The making of a book, however, is not simply an academic enterprise. I thank my friends and colleagues who over the years have engaged with me on and off about the ideas I present in this book and who at various points have given invaluable support as I worked on it: Mary Bassett, George Davey Smith, Sofia Gruskin, Lisa Moore, Anne-Emanuelle Birn, Rosalyn Baxandall, and Jason Beckfield, and also my core team members, Jarvis T. Chen and Pamela D. Waterman, whose wonderful daily work on our many theoretically-grounded empirical epidemiologic investigations has bolstered, extended, and given space for my thinking. To the extent that my own epidemiologic investigations inform the content of this book, I thank, for the studies that enrolled participants, the individuals who agreed to share information about their lives to inform the public understanding of health and disease, and for the studies that relied on vital statistics and other public health surveillance data, I thank the staff of the agencies involved who diligently transform information from people's medical records, birth and death certificates, and other such resources into usable data for understanding population health.

I conclude with a final set of thanks that transcend words. I begin with thanks to my parents, Dorothy T. Krieger (1927–1985) and Howard P. Krieger (1918–1992), who taught me to value learning and apply that knowledge to making the world a better place; to my brother, Jim Krieger, who is simultaneously friend, family, and a public health inspiration, connecting social justice and public health through his tangible work to reduce health inequities and promote the public's health; to Mrs. Montez Davis (1914–1997), who helped raise me; and to my three cats, who have been my constant companions since I first conceived this project: Emma (1981–1996), Samudra (b. 1996), and her brother Bhu (1996–2010). And to Lis Ellison-Loschmann: thank you.

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Table of Contents

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Epidemiology and the People's Health

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1

Does Epidemiologic Theory Exist?

On Science, Data, and Explaining Disease Distribution

Theory. Traced to its Greek roots, "theory" means to see inwards; to theorize is to use our mind's eye systematically, following articulated principles, to discern meaningful patterns among observations and ideas (Oxford English Dictionary [OED] 2008). The implication is that without theory, observation is blind and explanation is impossible.

In this chapter, I will make the argument that epidemiologic theory is a practical necessity for thinking about and explaining disease distribution. What could be more obvious?

Yet, apparently refuting what ought to be this simple self-evident claim is the curious fact that epidemiologic textbooks have, for the past several decades—as I discuss below—offered little or no guidance on what an epidemiologic theory is, let alone why such theory is important or how it can be used (Krieger, 1994). Sorting out this conundrum requires considering what scientific theory is—and what place it might have in epidemiologic thinking and research.

Figuring Out the People's Health: Theory and the Stories (About) Data (that People) Tell

First: Why even posit that epidemiologic theory is a practical necessity? Consider the epidemiologic data shown in **Figures 1–1** through **1–7**. Together, they illustrate population distributions of disease—over time, space, and social group—in the United States and globally.

Figure 1–1 presents data from a study titled "The fall and rise of US inequities in premature mortality: 1960–2002" (Krieger et al., 2008). These data show that between 1960 and 2002, as rates of U.S. premature mortality (**Figure 1–1a**, deaths before age 65 years) and infant death (**Figure 1–1b**, deaths before age 1 year) declined in all county income quintiles, socioeconomic and racial/ethnic inequities in premature mortality and infant death (both relative and absolute) *shrank* between 1966 and 1980, especially for U.S. populations of color, but from 1980 onward, the relative health inequities *widened* and the absolute differences barely changed. Why?

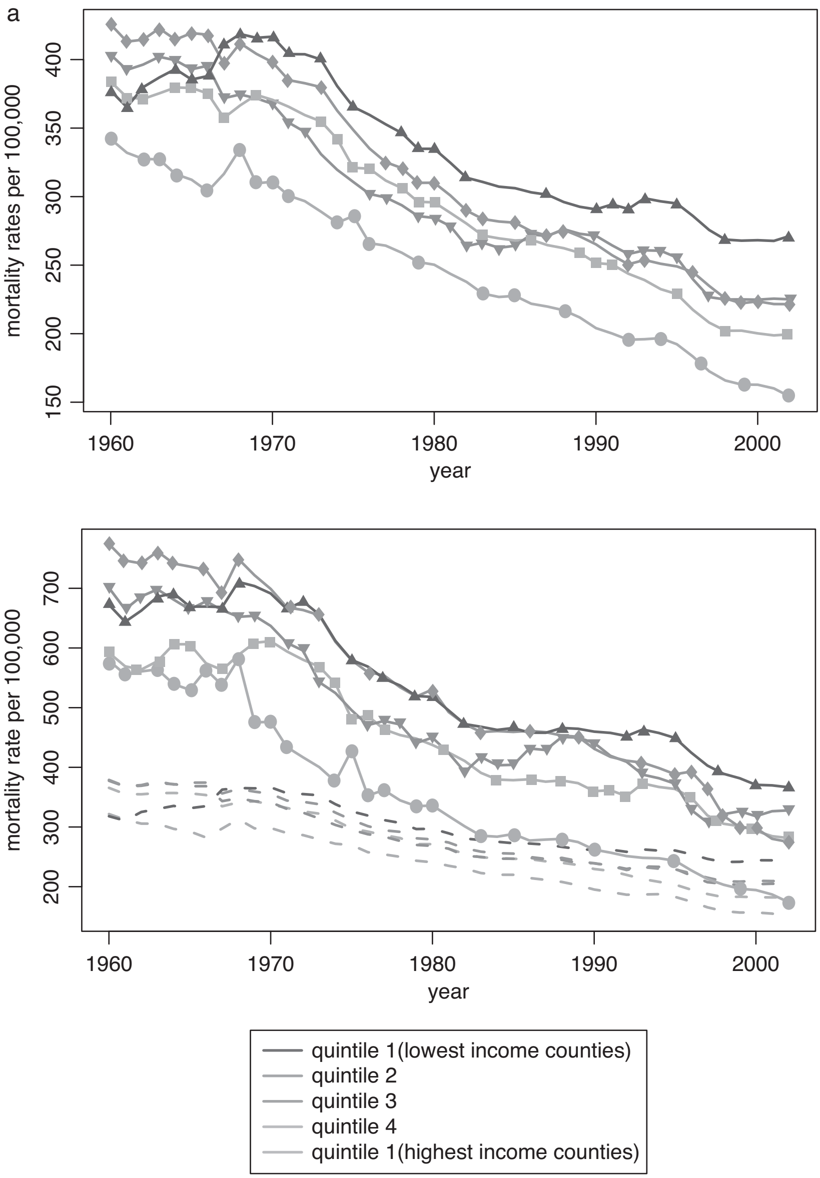

Figure 1–1. The fall and rise of U.S. inequities in premature mortality, 1960–2002, by county median income quintile. (Krieger et al., 2008)

Figure 1–1a. The fall and rise of U.S. inequities in premature mortality: deaths before age 65 years, 1960–2002, by county median income quintile, for: (A) total population by county median income quintile, and (B) the US White population (dashed lines) and populations of color (solid lines).

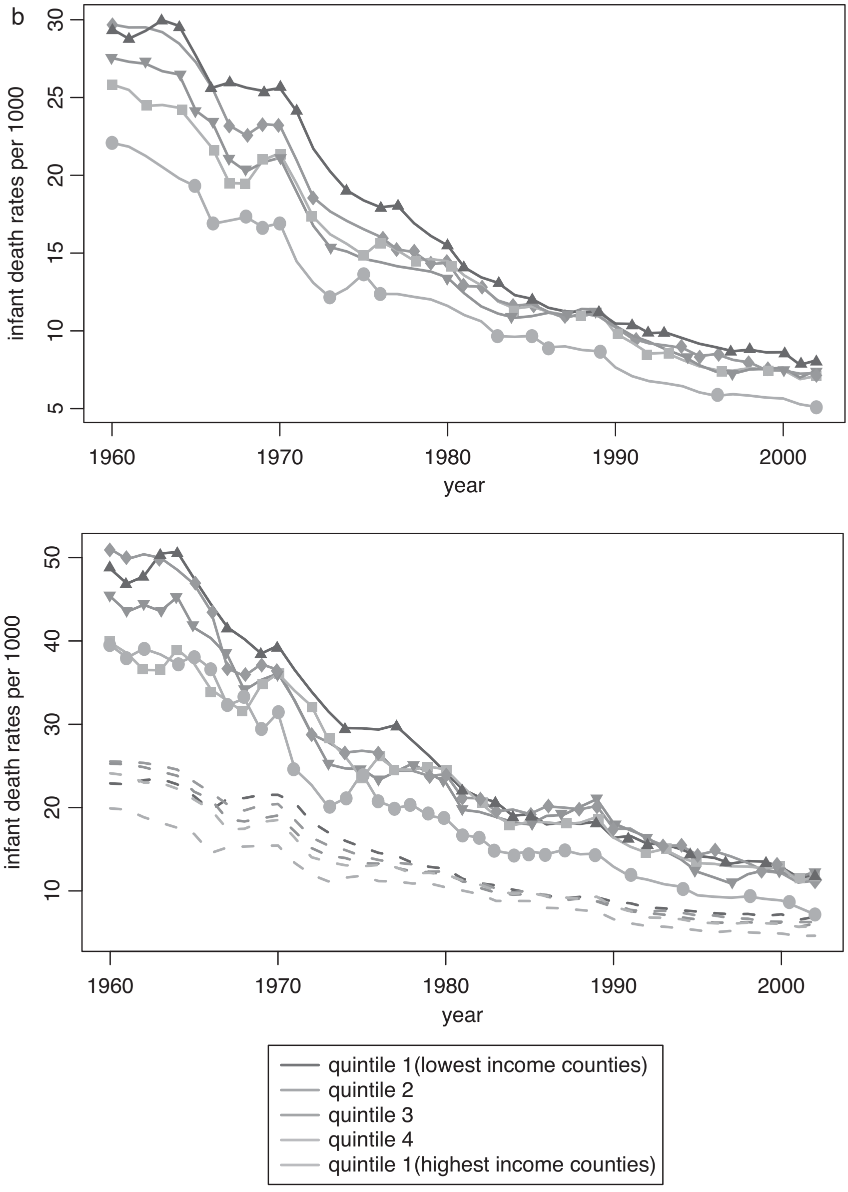

Figure 1–1b. The fall and rise of U.S. inequities in premature mortality: infant deaths, 1960–2002, by county median income quintile, for: (A) total population, and (B) the US White population (dashed lines) and populations of color (solid lines).

Figure 1–1 Technical Considerations

1. U.S. counties are politically defined geographic areas within United States. As explained by the U.S. Census, "states and counties are the major legally defined political and administrative units of the United States" (U.S. Census Geographic Areas Reference Manual 2008, p. 4-1). More specifically, counties are "a type of governmental unit that is the primary legal subdivision of every State except Alaska and Louisiana" (the former uses "borough" and the latter uses "parish" instead of "county") (U.S. Census Geographic Areas Reference Manual 2008, p. G-17).
2. The county income quintiles are based on county median family income, referring to the income level at which half the families in the county are below this level and half are above. The lowest income county quintile (the darkest line) contains the bottom fifth of counties, and the highest income county quintile (the lightest line) contains the top fifth of counties, as ranked by their county median family income.
3. In Figure 1–1 the death rates are age-standardized to permit meaningful comparison of death rates by county income level over time and across counties (Anderson & Rosenberg 1998; Krieger & Williams 2001). In this approach, each and every county in each and every year is treated as if it had the exact same age distribution, such that any county differences in the age-standardized death rates are not simply a result of the population of one county being younger or older than another but, rather, because the county mortality rates differ within specific age groups. For example, a county with many retirees would be expected to have a higher death rate than a county consisting chiefly of young families with young children, simply because older people are more likely to die than younger people. Age-standardization avoids this problem by taking into account whether, at each and every age, from young to old, the deaths rates are similar or different. Stated more technically, the age-standardized death rate is computed by applying each county's age-specific mortality rates (e.g., for persons ages 0–4, 5–9, 10–19,…, 65–69, 70–75, 75–78, and 80+ years) to a specified "standard population," determining the number of persons in each age group who would have died (given the county age-specific mortality rates), summing them up, and dividing by the total size of the "standard population." If the age-standardized rate for the counties differs, then by definition it differs because of something other than the counties' age structure. In the hypothetical example below, two populations have similar crude death rates ([total of deaths divided by total population]*100,000) but the age-standardized death rate of Population 2 is 1.3 times higher than that of Population 1, because in every age strata, Population 2 has higher age-specific death rates than Population 1, which is masked by the crude death rate, given the younger age structure of Population 2 compared to Population 1 combined with lower mortality rates at younger ages.

Age group (years)	Population 1			Population 2			US 2000 standard million population	Population 1	Population 2
	Deaths(N)	**Population(N)**	**Age-specific death rate per 100,000**	**Deaths (N)**	**Population(N)**	**Age-specific death rate, per 100,000**		**Number of deaths if apply their death rates to the same standard population**	
	(A)	(B)	(C) = ((A)/(B)) ∗ 100,000	(D)	(E)	(F) = ((D)/(E)) ∗ 100,000		(C) ∗ standard population	(F) ∗ standard population
<1	99	17,150	577.3	202	15,343	1,316.6	13,818	79.8	181.9
1–4	22	67,265	32.7	27	64,718	41.7	55,317	18.1	23.1
5–14	32	200,511	16.0	51	170,355	29.9	145,565	23.3	43.5
15–24	134	174,405	76.8	200	181,677	110.1	138,646	106.5	152.6
25–34	118	122,567	96.3	296	162,066	182.6	135,573	130.6	247.6
35–44	210	113,616	184.8	421	139,237	302.4	162,613	300.5	491.7
45–54	426	114,265	372.8	895	117,811	759.7	134,834	502.7	1,024.3
55–64	784	91,481	857.0	1,196	80,294	1,489.5	87,247	747.7	1,299.5
65–74	1,374	61,192	2,245.4	1,471	48,426	3,037.6	66,037	1,482.8	2,005.9
75–84	1,766	30,112	5,864.8	1,117	17,303	6,455.5	44,842	2,629.9	2,894.8
85+	1,042	7,436	14,012.9	360	2,770	12,996.4	15,508	2,173.1	2,015.5
Total	6,007	1,000,000		6,236	1,000,000		1,000,000	8,195.0	10308.04

Population	Death rates (per 100,000)		Ratio of death rates: Population 2/Population 1	
	Crude	**Age-standardized**	**Crude**	**Age-standardized**
Population 1	600.7	819.5	1.04	1.27
Population 2	623.6	1,038.0		

Figure 1–2 depicts age-specific trends in U.S. breast cancer incidence rates among U.S. White women from 1937 to 2003 (Krieger, 2008). It reveals a marked jump in incidence among women age 55 years and older starting in 1980, with rates then falling after 2002. Why?

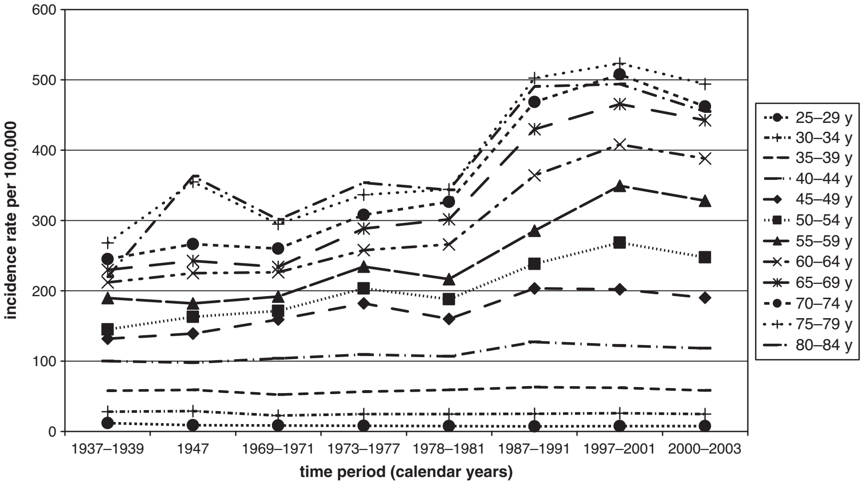

Figure 1–2. The rise and perhaps fall of U.S. breast cancer incidence rates. (Krieger, 2008)

Figure 1–3 is the graph of changing trends in mortality among women and men ages 55 to 64 years in England and Wales from 1850 to 1950 that Jerry Morris (1910–2009) used in his classic 1955 article on "Uses of Epidemiology" (Morris, 1955) and with which he opened his pathbreaking 1957 textbook by the same name (Morris, 1957). During this time period, mortality rates fell in both groups, but not evenly so: whereas the male:female mortality ratio was approximately 1.1 in 1850, it was 1.3 in 1920, and 1.9 in 1950. The growing divergence, Morris noted, resulted chiefly from the "emergence of three diseases from obscurity to become exceedingly common, disease which particularly affect men and are very frequent in middle-age: duodenal ulcer, cancer of the bronchus and coronary thrombosis" (Morris, 1957, pp. 1–2). As Morris also wondered: Why?

Figure 1–4 shows maps from the "Worldmapper" project, in which the size of countries is scaled to the size of the outcome depicted: population size, economic resources, and health status (Worldmapper, 2008). **Figure 1–4a** provides the conventional map of the world, with countries scaled to land size; in **Figure 1–4b,** the countries are scaled to the size of their population. **Figure 1–4c** shows the data for "absolute poverty," defined by the World Bank as living on an income of at most $2 per day; **Figure 1–4d** displays the data for wealth, as measured by the gross domestic product (GDP). In the former, the African continent and Asian subcontinent loom large; in the latter, the United States, Europe, and Japan are bloated, and the Asian subcontinent shrinks and the African continent dwindles to the merest strand. **Figure 1–4e** presents data on infant mortality; **Figure 1–4f** provides data on lung cancer deaths; **Figure 1–4g** shows data on "often preventable deaths," defined

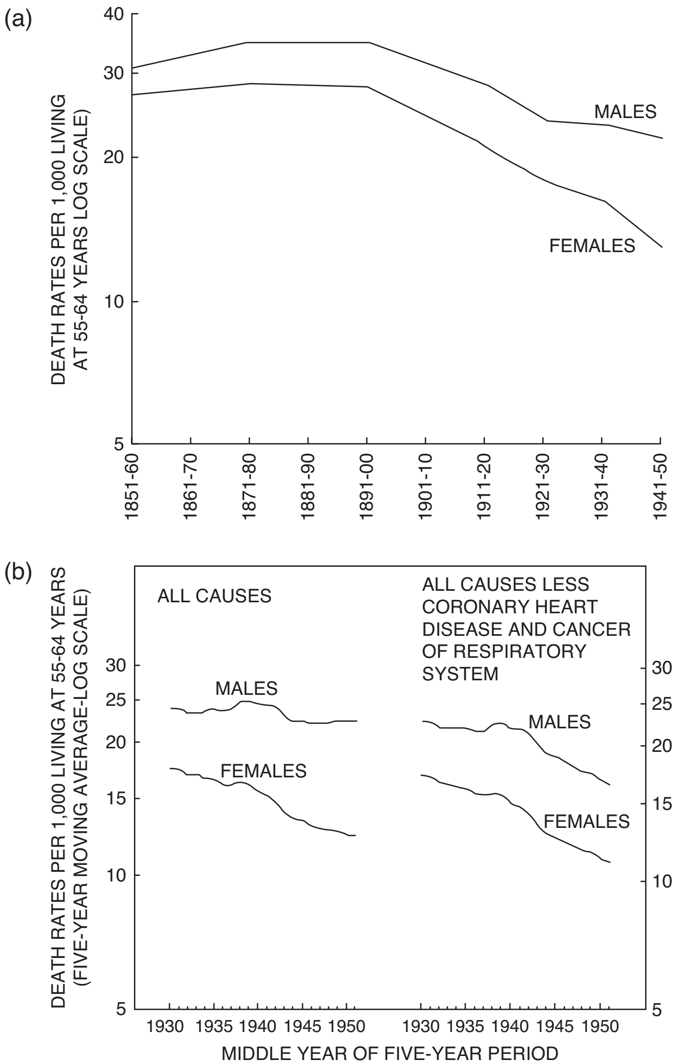

Figure 1–3. Trends in mortality by gender and cause of death in England and Wales, 1850–1950, as presented in Morris's 1955 article on "Uses of Epidemiology" (Morris, 1955) and incorporated into his 1957 pathbreaking textbook *Uses of Epidemiology* (Morris, 1957, pp. 1–2).

in relation to communicable infections and maternal, perinatal, and nutritional conditions and accounting for one-third of the world's deaths in 2002; and **Figure 1–4h** depicts data on sewerage sanitation. In **Figures 1–4e** and **1–4g**, the African continent and Asian subcontinent again loom large, whereas the United States, Europe, and Japan are massively shrunk. In **Figures 1–4f** and **1–4h**, the reverse occurs. Why?

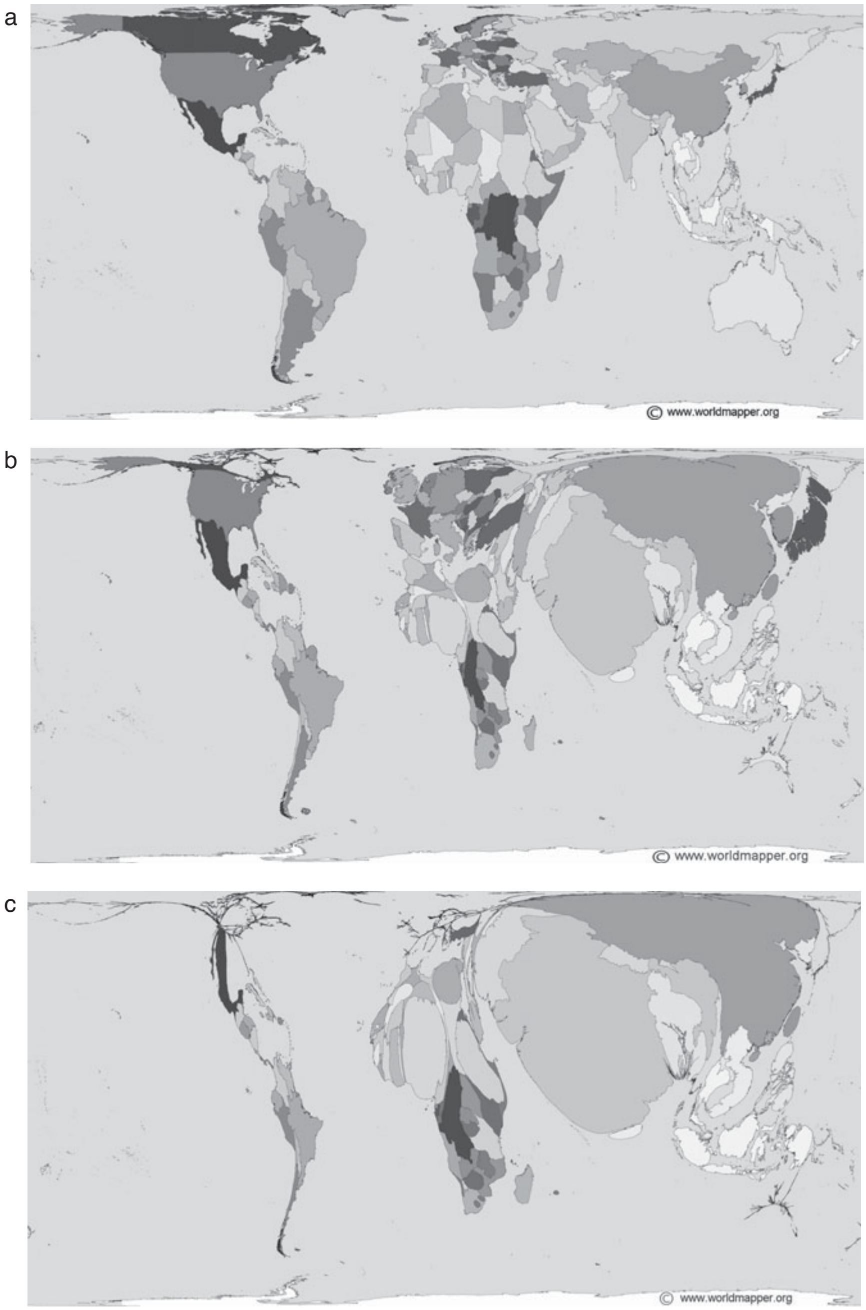

Figure 1–4. Maps from the "Worldmapper" Project (Worldmapper, 2008), in which country size is scaled in relation to the outcome depicted. © Copyright 2006 SASI Group (University of Sheffield) and Mark Newman (University of Michigan).

Figure 1–4a. Countries scaled to land size.

Figure 1–4b. Countries scaled to population size (2002).

Figure 1–4c. Absolute poverty (up to $2 per day) (2002).

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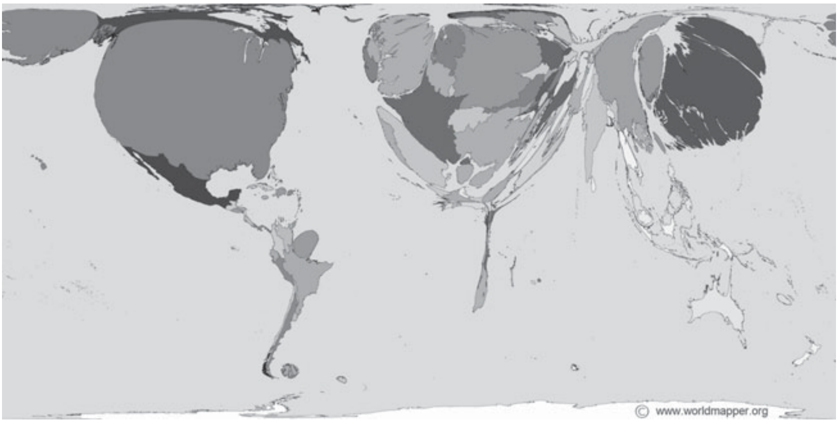

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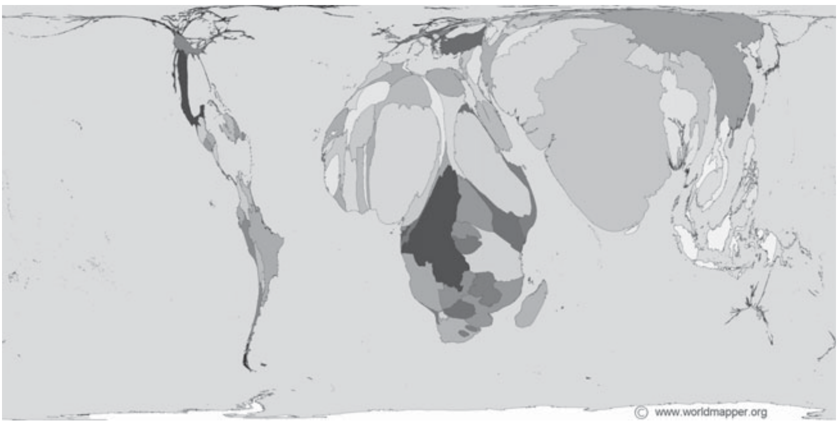

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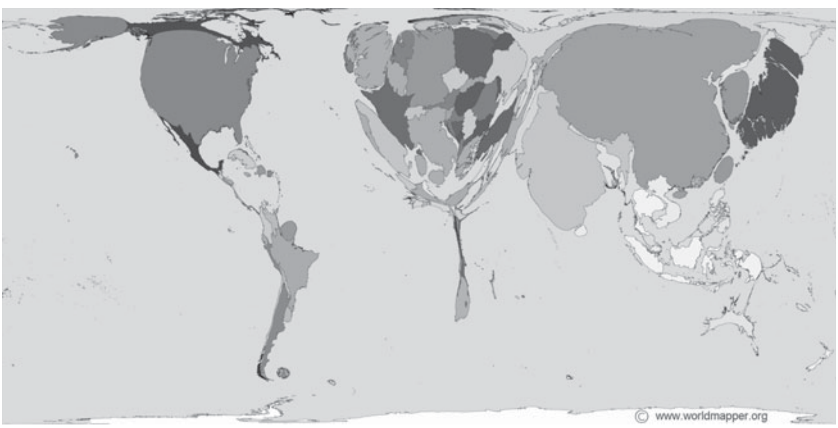
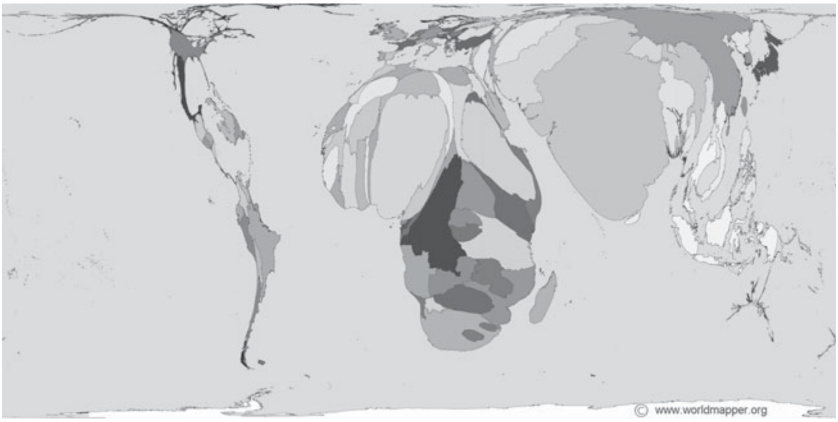


Figure 1–4d. Wealth (gross domestic product) (2002).

Figure 1–4e. Infant mortality (2002).

Figure 1–4f. Lung cancer deaths (2002).

g



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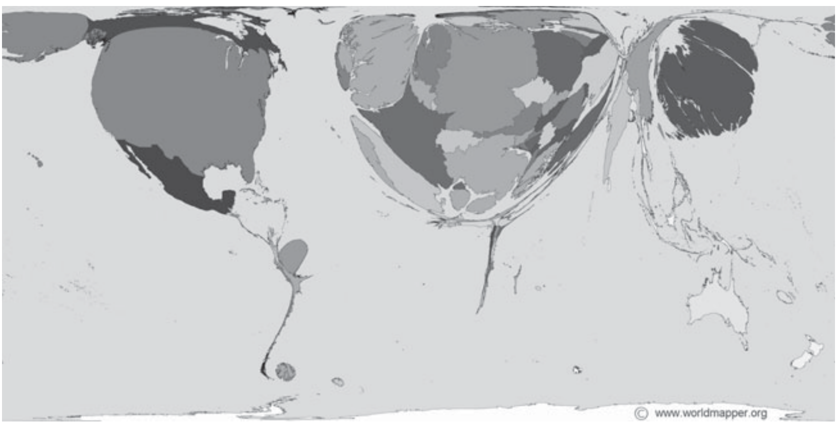


Figure 1–4g. Often preventable deaths (communicable infections, maternal, perinatal, and nutritional conditions) (2002)

Figure 1–4h. Sewerage sanitation (1999).

Finally, **Figures 1–5**, **1–6**, and **1–7** display data from the "Gapminder" project regarding associations between child survival (children dying before age 5 years per 1000 live births) and Gross National Income per capita (Gapminder, 2008). **Figure 1–5** depicts these country-level associations for 2006, with the size of each country's data point scaled to population size, and countries within the same global region shaded the same color. Although it shows an overall robust direct association between child survival and income (the lower the income, the poorer the survival), as underscored in **Figure 1–6**, at any given level of per capita income, countries vary considerably in their rates of child survival (e.g., South Africa fares worse than Malaysia, despite similar per capita income), and at any given level of child survival, countries vary considerably in their per capita income (e.g., Malaysia fares as well as the United States, despite its lower per capita income). **Figure 1–7** in turn presents data on within-region distributions of income in 2000, along with data on within-countries inequities in child survival and income in 2003, for India, Bangladesh, Peru, Guatemala, Yemen, South Africa, and Vietnam. Illuminating

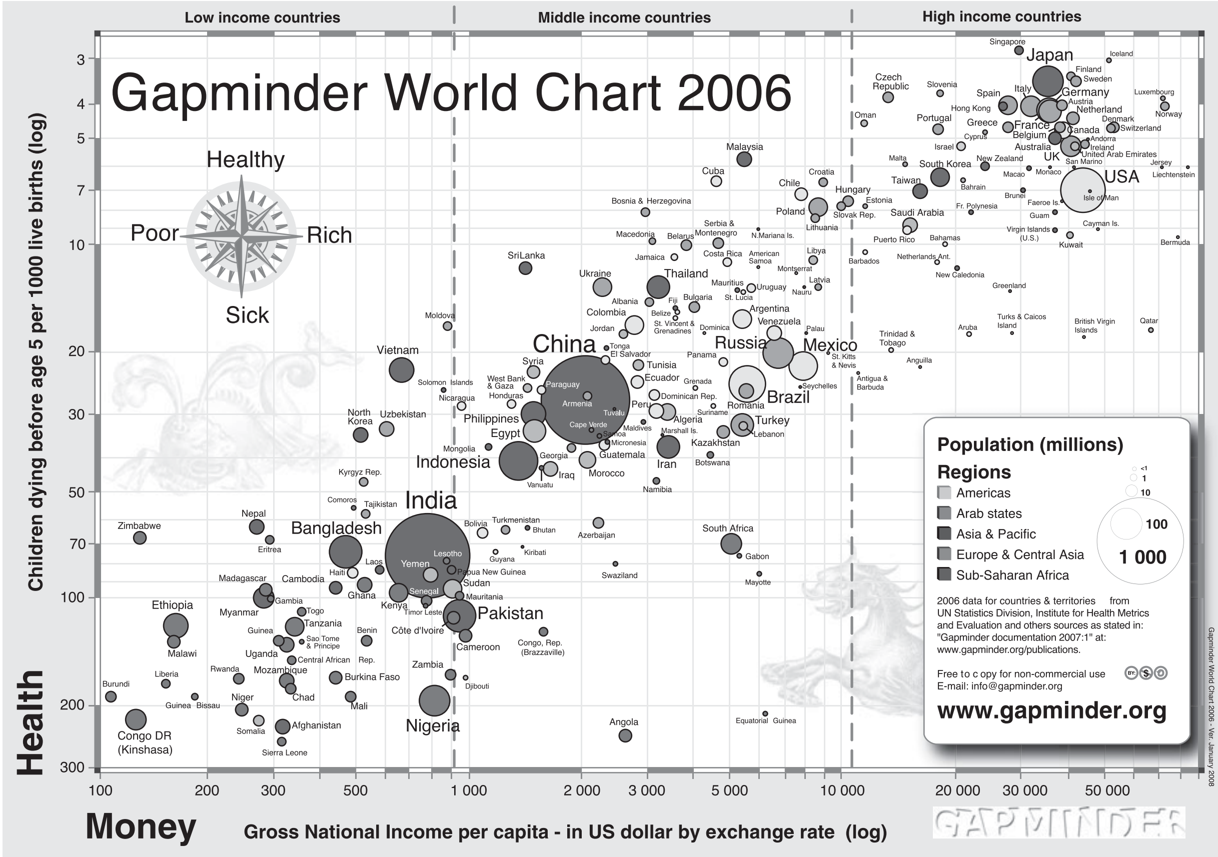
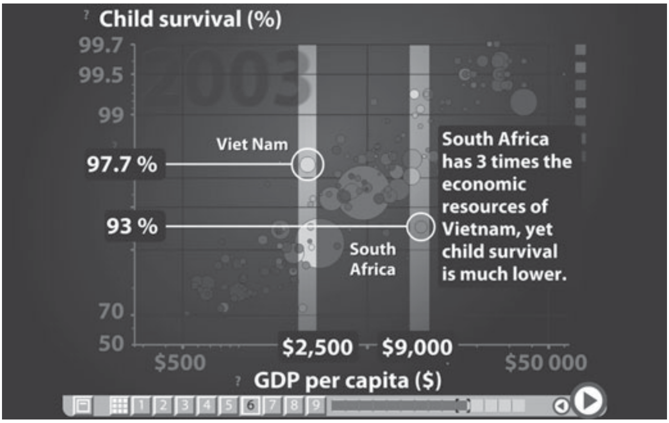


Figure 1–5. Gapminder World Chart 2006: Child survival (children dying before age 5 per 1000 live births) in relation to Gross National Income per capita.

a



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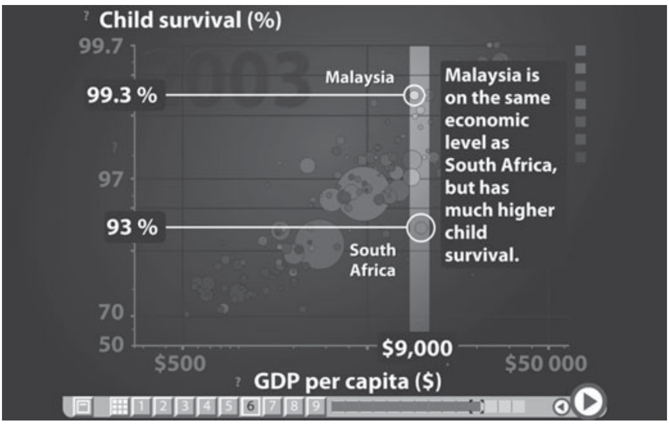

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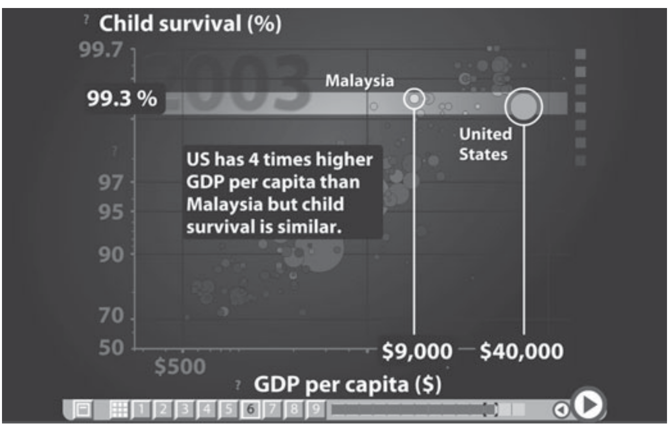


Figure 1–6. Between-country comparisons of child survival and per capita income, by level of child survival and by per capita income, excerpted from the Gapminder Human Development 2005 presentation (Gapminder, 2008).

Figure 1–6a. Income and child survival inequities: South Africa and Vietnam (2003)

Figure 1–6b. Income and child survival inequities: South Africa and Malaysia (2003)

Figure 1–6c. Income and child survival inequities: Malaysia and the United States (2003)

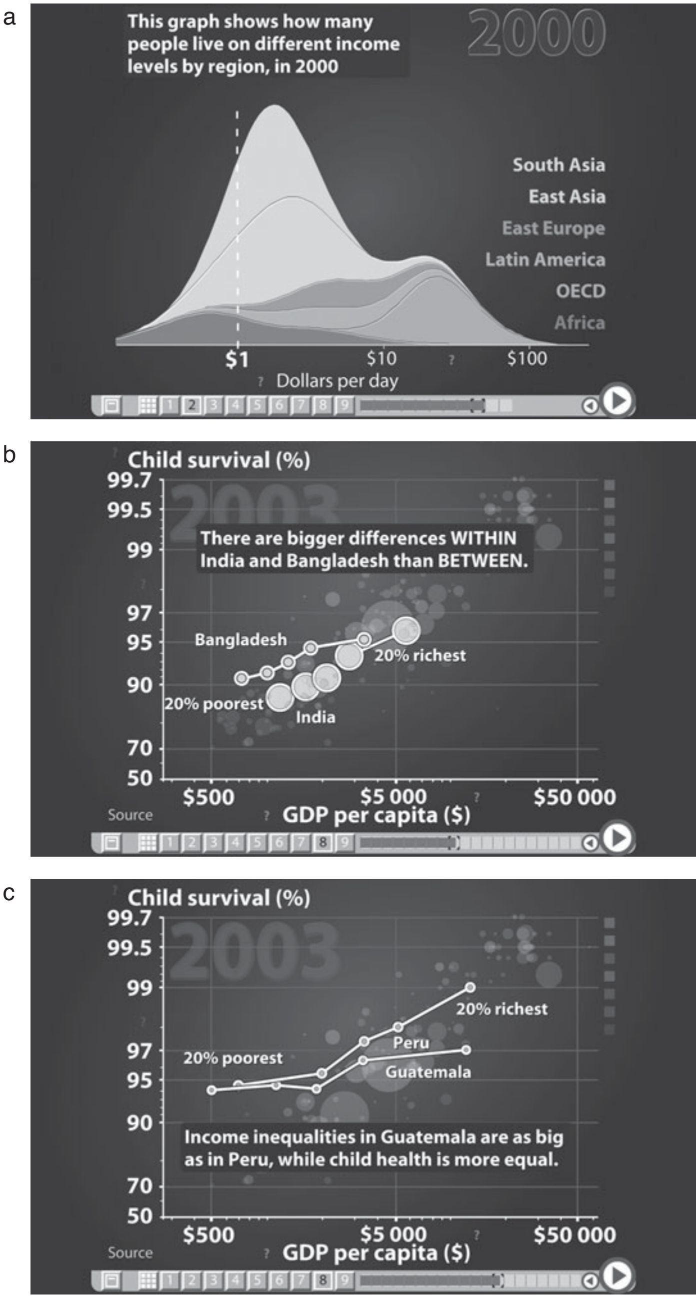
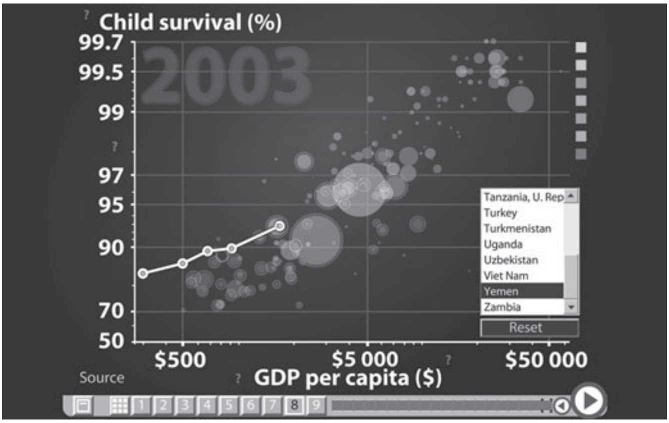


Figure 1–7. Within-country inequities in child survival and per capita income, excerpted from the Gapminder Human Development 2005 presentation (Gapminder, 2008)

Figure 1–7a. Income distribution by global region

Figure 1–7b. Income and child survival inequities: within Bangladesh and India (2003)

Figure 1–7c. Income and child survival inequities: within Peru and Guatemala (2003) d



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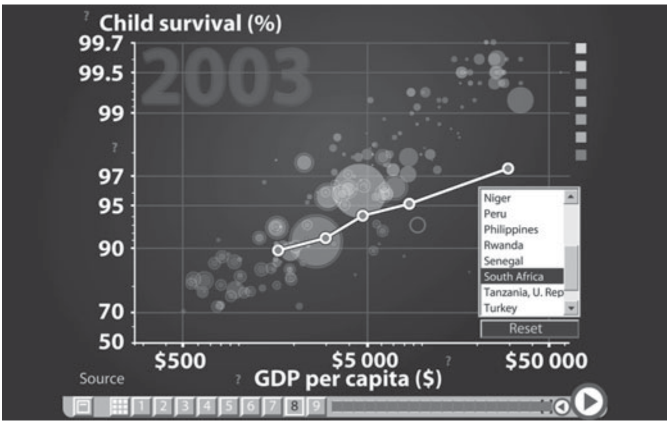

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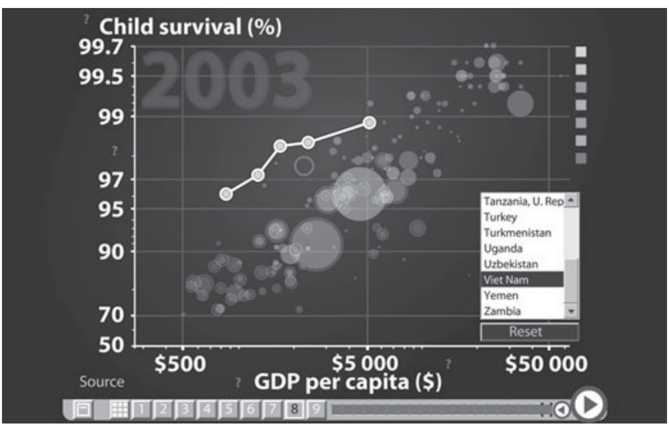

Figure 1–7d. Income and child survival inequities: Yemen (2003)

Figure 1–7e. Income and child survival inequities: South Africa (2003)

Figure 1–7f. Income and child survival inequities: Vietnam (2003) the variability behind the on-average values, these figures show that within-country differences in income and child survival can dwarf between-country differences. Once again: Why?

Before even considering the role of theory in answering these "whys," it is important to step back and ask: What is the thinking that leads to data allowing these questions even to be posed? And where does theory fit into this process?

One place to begin is to realize that **Figures 1–1** through **1–7** are premised on a host of assumptions. What ideas are built into these figures? To start: population rates of disease—a phrase that requires understanding *population*, *rate*, and *disease*. Other ideas at play include: changing incidence rates over time; geographic variation in disease occurrence; and differences in disease rates by social group. None of these ideas are intuitively obvious. They make sense only if one already has a theoretical orientation that finds it compelling and reasonable to think abstractly about populations, about individuals in numerators and denominators, about averages and distributions, about disease occurrence in space and time, and about disease as a definable entity apart from (as opposed to uniquely residing in) the individual persons in whom it is experienced—and hence diseased persons as countable cases.

Prelude, then, to **Figures 1–1** through **1–7** are the ideas that would compel someone to collect and display their data. Also notable is who and what is omitted, not simply who and what is included—for example, if the data are or are not separately shown by such social categories as social class, gender, race/ethnicity, sexuality, or by subtypes of disease.

In other words, data are not simply "observed": there is active thinking behind the act of data acquisition. Not to mention the active thinking that guides data analysis, display, and interpretation.

And this active thinking is the stuff of theory.

Meaning: contrary to its etymologic origins, data are not a "given" ("datum" is the past participle of the Latin verb "dare," "to give" [OED, 2008; Krieger, 1992]). Nor do data tell stories. People do. An important caveat, however, is that the stories that people who are scientists tell are not simply or simple "stories": they are (or are supposed to be) transparent accounts, informed by theory, and premised on the public testing of ideas and explanations, using explicitly defined concepts and methods.

So What Is a Scientific Theory?

To appreciate what an epidemiologic theory is (or ought to be), it helps first to have a sense of what counts as a scientific theory—and also: what counts as science. The literature on these topics is vast, contentious, and complex (Mendelsohn et al., 1997; Archer et al., 1998; Ziman, 2000; Collins, 2001; Gould, 2002; Grene & Depew, 2004; Daston & Gallison, 2007; Sober, 2008). That said, some common contemporary criteria for science and scientific theories do exist (*see* **Textboxes 1–1** and **1–2**).

To begin, most current scholarship would agree that scientific theories, in contemporary terms, are coherent and presumptively testable sets of inter-related ideas that enable scientists to describe, explain, and predict features of a commonly shared biophysical reality in which cause-and-effect exists (Mendelsohn et al., 1997; Ziman, 2000; Krieger, 2001a). Science, in turn, is both a human activity and a body of knowledge premised on the thinking and action of people to describe and to test their explanations and predictions about features of their commonly shared reality. Particular fields of scientific inquiry are, in turn, distinguished by the domains they seek to understand, the substantive and explanatory

Textbox 1–1. Contested Definitions: Science, Theory, and Hypothesis

Term	Definition
science	***Oxford English Dictionary* (OED, 2008):** —etymology: "(a. F. *science* = Pr. *sciensa*, Sp. *ciencia*, Pg. *sciencia*, It. *scienza*, ad. L. *scientia* knowledge, f. *scient-em*, pr. pple. of *sc re* to know.)" —definition: 4a. In a more restricted sense: A branch of study which is concerned either with a connected body of demonstrated truths or with observed facts systematically classified and more or less colligated by being brought under general laws, and which includes trustworthy methods for the discovery of new truth within its own domain. 5. …In mod. use chiefly: The sciences (in sense 4) as distinguished from other departments of learning; scientific doctrine or investigation. 5b. In modern use, often treated as synonymous with "Natural and Physical Science," and thus restricted to those branches of study that relate to the phenomena of the material universe and their laws, sometimes with implied exclusion of pure mathematics. This is now the dominant sense in ordinary use. ***Oxford Dictionary of Science* (2005) (Daintith, 2005):** no definition (!) —and worth considering what it means the term is considered to be self-evident… ***Oxford Dictionary of Sociology* (2005) (Scott & Marshall,** 2005): no definition (!)—ditto… ***Keywords* (1983) (Williams, 1983):** (*italics* and **bold** in the original; C = century (e = early, m = mid)) p. 277: "**Science** came into English in C14, from fw *science*, F., *scientia*, L–knowledge. Its earliest uses were very general… often interchangeably with art, to describe a particular body of knowledge or skill…" p. 278: "The key distinction was not a first in **science** but in the crucial C18 distinction between *experience* and *experiment*. This supported a distinction between *practical* and *theoretical* knowledge, which was then expressed as a distinction between *art* and **science** in their C17 and C18 general senses… The distinction hardened in eC19 and mC19… we can find by 1867 the significantly confident, yet also significantly conscious, statement: 'we shall… use the word 'science' in the sense which Englishmen so commonly give to it .. as expressing physical and experimental science, to the exclusion of theological and metaphysical.'" ***New Keywords* (2005) (Shapin, 2005)** (*italics* and **bold** in the original; C = century) p. 314: "In the early modern period, the L scientia just mean knowledge, usually in the sense of a systematically organized body of knowledge, acquired through a course of study… During the course of the C19 and C20, 'science' came overwhelmingly to pick out those practices proceeding by observation and experiment, thus jettisoning history and philosophy and leaving the social sciences a courtesy title, with limited credibility in the general cultural or among natural scientists 'proper.'" p. 315: "Linguistically, this more restrictive sense of 'science' was an artifact of the way English usage developed and changed in recent centuries… by the C19 'science' did not usually need the qualifying

'natural' to summon up the idea of organized methodological research into the things, phenomena, and capacities belonging to nature as opposed to culture. How this shift occurred is still little understood…"
p. 317: "Talk about the 'scientific method' is predicated upon some version of the 'unity' of science… Disunity theorists doubt that there are any methodological procedures held in common by invertebrate zoology, seismology, microbial genetics, and any of the varieties of particle physics, which are *not* to be found in non-scientific forms of culture. How can the human sciences coherently either embrace or reject "the natural science model" when the natural sciences themselves display such conceptual and methodological heterogeneity?"

theory ***Oxford English Dictionary*** **(OED, 2008):**
—etymology: "(ad. late L. theōria (Jerome in Ezech. XII. xl. 4), a. Gr. θεωρία a looking at, viewing, contemplation, speculation, theory, also a sight, a spectacle, abstr. n. f. spectator, looker on, f. stem of to look on, view, contemplate. In mod. use prob. from med.L. transl. of Aristotle. Cf. It. *teoria* (Florio 1598 *theoría*), F. *théorie* (15. in Godef. *Compl.*).)"
—definition:
3. A conception or mental scheme of something to be done, or of the method of doing it; a systematic statement of rules or principles to be followed.
4a. A scheme or system of ideas or statements held as an explanation or account of a group of facts or phenomena; a hypothesis that has been confirmed or established by observation or experiment, and is propounded or accepted as accounting for the known facts; a statement of what are held to be the general laws, principles, or causes of something known or observed.
4b. That department of an art or technical subject which consists in the knowledge or statement of the facts on which it depends, or of its principles or methods, as distinguished from the practice of it.

Oxford Dictionary of Science **(2005) (Daintith, 2005):**
p. 464: "A description of nature that encompasses more than one law but has not achieved the uncontrovertible status of a law is sometimes called a **theory**."

Oxford Dictionary of Sociology **(2005) (Scott & Marshall, 2005):**
p. 662: "A theory is an account of the world which goes beyond what we can see and measure. It embraces a set of interrelated definitions and relationships that organizes our concepts and understanding of the empirical world in a systematic way, Generally speaking, there are three different conceptions of theory in sociology. Some think of theory as generalization about, and classification of, the social world. The scope of generalization varies from theorizing about a particular range of phenomena to more abstract and general theories about society and history as a whole. Others believe that theoretical statements should be translated into empirical, measurable, or observable propositions, and systematically tested… Finally, yet others argue that theory should explain phenomena, identifying causal mechanisms and processes which, although they cannot be observed directly, can be seen in their effects."

***Keywords* (1983) (Williams, 1983):** (*italics* and **bold** in the original; C = century [l = late])
p. 316: "**Theory** has an interesting development and range of meanings, and a significant distinction from (later an opposition to) practice.

The earliest English form was *theorique* (C14), followed by *theory* (C16), from fw *theoria*, lL, *theoria*, Gk–contemplation, spectacle, mental conception (from *theoros*, Gk–spectator, rw *thea*, Gk–sight; cf *theatre*)… A distinction between **theory** and *practice* was widely made in C17, as in Bacon (1626)."
p. 317: "But **theory** in this important sense is always in active relation to *practice*: an interaction between things done, things observed and (systematic) explanation of these. This allows a necessary distinction between **theory** and *practice*, but does not require their opposition."

***New Keywords* (2005) (Frow, 2005)** (*italics* and **bold** in the original; C = century)
p. 347: "In its modern sense the word **theory** probably entered English from medieval translations of Aristotle. Etymologically it has the same root (*theoros*, spectator, from rw *thea*, sight) as the word *theatre*; Gk *theorie* is a sight of spectacle, and the literal sense of looking has then been metamorphosized to that of contemplating or speculating… . In a more general philosophical and scientific sense, a theory is:

> a scheme or system of ideas or statements held as an explanation or account of a group of facts or phenomena; a hypothesis that has been confirmed or established by observation or experiment, and is propounded or accepted as accounting for the known facts; a statement of what are held to be the general laws, principles, or causes of something known or observed.
> Central to this definition is the notion of the systematic relations holding between the components of an explanatory model, and the differentiation of theory from the more tentative conception of a hypothesis."

p. 348: "The account of **scientific theorization** in the C20, dominated by the logical positivism of Rudolf Carnap, Karl Popper, and others, attempts to reduce the speculative dimension of theorization by requiring the use of rigorous correspondence rules between observation statements and theoretical meta-languages. A more positive view of theory particularly in the social sciences, however, has stressed that observation statements in the natural sciences are always theory-laden and are meaningful in relation to a particular theoretical framework . . . In contemporary usage in the humanities and social sciences, 'theory' designates less any particular set of systematic ideas than a politically contested attitude toward the use of abstract explanatory models in humanistic and social inquiry."

hypothesis ***Oxford English Dictionary* (OED, 2008):**
—etymology: (a. Gr. ὕπόθεσις foundation, base; hence, basis of an argument, supposition, also, subject-matter, etc., f. ὕπό under + θέσις placing.)

—definition:
2. A proposition or principle put forth or stated (without any reference to its correspondence with fact) merely as a basis for reasoning or argument, or as a premiss from which to draw a conclusion; a supposition.
3. A supposition or conjecture put forth to account for known facts; *esp.* in the sciences, a provisional supposition from which to draw conclusions that shall be in accordance with known facts, and which serves as a starting-point for further investigation by which it may be proved or disproved and the true theory arrived at.

***Oxford Dictionary of Science* (2005) (Daintith, 2005):**
p. 464: "A **hypothesis** is a theory or law that retains the suggestion that it may not be universally true."

***Oxford Dictionary of Sociology* (2005) (Scott & Marshall, 2005):**
p. 285: "A hypothesis is an untested statement about the relationship (usually of association or causation) between concepts within a given theory."

***Keywords* (1983) (Williams, 1983):** no entry

***New Keywords* (2005) (Bennett et al., 2005):** no entry

concepts they use, and the metaphors and mechanisms they employ for their causal explanations (*see* **Textbox 1–2**) (Martin & Harré, 1982; Ziman, 2000; Krieger, 2001a). Additionally, those sciences whose domains encompass non-deterministic phenomena (e.g., excluding what are held to be invariant "natural laws," such as the law of thermodynamics) can further be characterized by historical contingency (meaning what occurs depends on context, hence is not universally invariant)—and among these are the subset of reflexive sciences, which are focused on phenomena that can be influenced by human action (e.g., societal characteristics), such that the explanation adduced can be used to transform that which is being explained (Lieberson, 1992; Archer, 1998; Gannett, 1999; Ziman, 2000; Gadenne, 2002; Krieger, 2001a).

Core to the theorizing and conduct of science are a host of assumptions (Lieberson, 1992; Mendelsohn et al., 1997; Archer et al., 1998; Ziman, 2000; Collins, 2001; Gould, 2002; Grene & Depew, 2004; Daston & Gallison, 2007; Sober, 2008). One such assumption is that we humans live in a commonly shared biophysical (including social) world—and, more broadly, universe—which provides the referent for what we term *reality*. Another is that this commonly shared biophysical world encompasses diverse processes, structures, and events that are in principal knowable by humans and amenable to scientific investigation. A third is that the existence of this commonly shared knowable biophysical world can be investigated by—and is independent of—any particular human individual. A fourth is that independent humans (in solo and in groups) can independently formulate and test their ideas about "how the world works" and collectively compare ideas, methods, and results. All four of these assumptions are preconditions for the existence and evaluation of scientific theories. More bluntly, no postulated referent reality shared by and accessible to independent humans, no science.

Equally essential is the assumption that causal processes exist. Whether these processes are "deterministic" or "probabilistic" is another question entirely. I note only in passing that debates have raged for millennia over the meaning of causality—and, more recently, within a variety of scientific disciplines, over connections between "chance" and "necessity," and whether "randomness" is "real" or simply a reflection of ignorance of otherwise deterministic causes (Moyal, 1949; Monod, 1972; Stigler, 1986; Desrosières, 1988; Hacking, 1990; Daston, 1994; Gannett, 1999; Weber, 2001; Gadenne, 2002; Russo & Williamson, 2007; Machamer & Wolters, 2007; Groff, 2008). Regardless of the positions argued in these debates, however, the basic point remains that the scientific work of causal inference necessarily presumes that some sort of underlying causal relationship exists, either of the inevitable or contingent variety. Hence, one key corollary to the assumption about a referent reality: no causal processes, no science—and no scientific explanations.

This is all very abstract. It is supposed to be. Science and scientific theories require abstract thinking: to imagine and discern the causal processes behind the observed and postulated specifics, to derive meaning from pattern, and, as the poet William Blake (1757–1827) put it so well, "[t]o see a world in a grain of sand/And a heaven in a wild flower/Hold infinity in the palm of your hand/And eternity in an hour" (Blake, 1977, p. 506). Or, as stated more prosaically by Stanley Lieberson (b. 1933) in a 1991 presidential address to the American Sociological Association: "[T]heory involves generating principles that explain existing information; but it also goes beyond those observations to integrate and account for a variety of other phenomena in ways that would not otherwise be apparent" and would further "'predict' all sorts of observations not yet made" (Lieberson, 1992, p. 4).

Why bother with these abstract assertions? Because to understand and evaluate epidemiologic theories, it is important to know what science and scientific theories presume—and what they do not.

First, scientific theories are, by definition, conceptual. But they are not about just any set of ideas. They are instead sets of inter-related ideas intended to explain phenomena in specified domains of the commonly shared biophysical world. Additionally, both the ideas and what they refer to are capable of being independently evaluated and employed by different individuals. Accordingly, some of the concepts in scientific theories pertain to the phenomena that are being described and explained. Others pertain to the causal processes that are theorized to explain the selected phenomena. And both kinds of concepts—substantive and explanatory—are essential for scientific theory; neither alone suffices. What is being explained and how it is being explained are constituent and complementary—and often contested—aspects of scientific theory. Within any given discipline, different theories can exist, simultaneously or successively, offering different and debated explanatory accounts; across disciplines, theories additionally differ because of their respective focus on different aspects of what nevertheless is presumed to be a shared referent reality—whether physical, chemical, biological, or social. A theory of biological evolution, for example, needs not only the concepts of organism, environment, reproduction, and heredity (all of which presumably can in some way be studied by independent investigators) but also the causal ideas (which may be convergent, competing, or complementary) that tie these concepts together to explain the occurrence of evolution (Mayr, 1982; Eldredge, 1999; Gould, 2002; Grene & Depew, 2004; Sober, 2008).

Moreover, to express the ideas at play, scientific theories inevitably employ a combination of metaphor and mechanisms—metaphor to convey concepts describing both phenomena and causal processes and mechanisms to explain the pathways between cause and effect (Lakoff, 1980; Osherson et al., 1981; Martin & Harré, 1982; MacCormac, 1985; Young, 1985; Holton, 1988; Krieger, 1994; Keller, 1995; Krieger, 2001a; Keller, 2002). As I have noted in prior essays, this use of metaphor in scientific theories—essential for enabling the "unknown" to be comprehended in terms of the "known"—can simultaneously free and constrain thought (Krieger, 1994). A salient example, relevant to epidemiology, concerns the widespread—and now increasingly contested—metaphor of DNA as the "blueprint" or "master program" for the organism (Watson, 1968). This conceit, as pointed out by the biologist Richard Lewontin (b. 1929) (Lewontin, 2000, pp. 10–11), has dominated the genetics research agenda since the mid-twentieth century. Attesting to its widespread acceptance are the statements of prominent scientists, such as Sydney Brenner (b. 1927; Brenner, 2002), who in 1968 asserted, "The goal of molecular biology is to be able to compute an organism from a knowledge of its genes" (Melnechuk, 1968), and Walter Gilbert (b. 1932; Gilbert, 1980), who in 1992 declared that the complete sequencing of the human genome will enable us to know "what makes us human" (Gilbert, 1992, p. 84). Explicit articulation of the "blueprint" metaphor, moreover, was likewise provided in 1992 by James D. Watson (b. 1928), one of the co-discoverers of the double-helical form of DNA, who declared that the human genome constitutes "the complete genetic blueprint of man (*sic*)," arguing, "if you can study life from the level of DNA, you have a real explanation for its processes" (Watson, 1992, p. 164), a statement echoed in one newspaper account of the first full sequencing of the human genome on June 26, 2000: "The blueprint of humanity, the book of life, the software for existence—whatever you call it, decoding the entire three billion letters of human DNA is a monumental achievement." (Carrington, 2000). Although this architectural/computer programming conceit may initially have fruitfully guided genetic research (with the idea of DNA being "in command"), it is increasingly understood to disregard how DNA—and biological development—is dependent on and subject to myriad exogenous influences on gene regulation and expression (Keller, 1992; Keller, 1995; Gilbert, 2000; Lewontin, 2000; Keller, 2002; Van Speybroeck et al., 2002). The key point is that the concepts employed by scientific theories—whether to describe phenomena or causal processes—are not simply self-evident terms. Instead, they are usually rife with connections to other concepts—which is only to be expected, as theories, by definition, must employ interrelated ideas, and the people who use and develop these theories must employ words and symbols that convey these ideas to others interested in understanding them.

Second, the scientific assumption that there is a commonly shared biophysical world is a precondition for science—even as this assumption does *not* presume this referent reality is commonly perceived or understood by all individuals. Depending on people's specific characteristics and worldviews, individuals within and across different societies and time periods may vary in their perceptions and interpretations of any given biophysical phenomenon. At a fairly trivial level, color-blindness in particular individuals does not mean the absence of reflected light at the frequency at which these individuals are color-blind (Gibson, 1979). At a more profound level, different individuals may agree on the existence of the same set of associations—for example, when the sun passes below the horizon, it gets dark—and yet may have completely different interpretations of why these associations exist (e.g., because the sun is passing through the underworld; because the sun revolves around the Earth and has moved to location where it is not observable by the person on Earth; or because the Earth revolves around the sun and has rotated to a point where the sun is no longer observable by a person on that point of the Earth's surface; Hanson, 1958). Or, more epidemiologically, the shared observation of an association between two variables—say, race/ethnicity and disease—does not mean the variables or their association are comprehended in the same way. Whereas some might deem "race" a biological characteristic that explains the observed association (Burchard et al., 2003), others might argue instead that racism, and its associated socially-constructed categories of race/ethnicity, is what has causal relevance (Krieger, 2005). That said, disputes over the causal ideas at issue—and the substantive phenomena under study—nevertheless presume that there is a common reality to which they refer; otherwise, attempting to elucidate the reasons for disagreement—and testing competing hypotheses—would be impossible.

Third, scientific observation is not a passive phenomenon: what we "see" and apprehend depends on the ideas we have about we expect—and do not expect—to "see" and our technical capacity to do so (Fleck, 1935 [1979]; Hanson, 1958; Daston & Gallison, 2007). In one sense, this means meaningful observation is, at some level, theory-laden: what we "see" depends in part on what our ideas are about what we expect to see and what assumptions underlie the methods used to "observe" the data. If our theoretical ideas do not include micro-organisms, we would not devise methods to see them—and if offered a microscope, we would not know what we are seeing, regardless of the magnification employed. Similarly, if we do not have the idea of birth cohort effects, we will not "see" their impact on a population's age-specific disease incidence rates. For example, whereas Johannes Clemmesen (b. 1908) in the late 1940s (Clemmesen, 1948) saw the slight dip in the period's breast cancer incidence rates after age 50 as evidence that the risk of the disease was lower in women just older than 50 compared to those just younger than 50 and those in their late 50s and older (**Figure 1–8a**), Brian MacMahon (1923–2007) in the late 1950s saw this same pattern as evidence of a change in risk among women who reached age 50 before rather than after the mid-twentieth century (**Figures 1–8b** to **1–8d**; MacMahon, 1957)—and others since have explored the impact of age–period–cohort effects on the observed yearly incidence of breast cancer (Krieger et al., 2003; Chia et al., 2005).

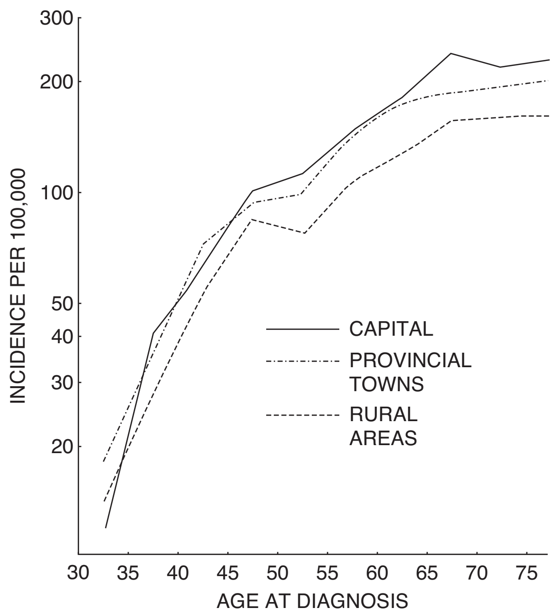

Figure 1–8. Data on breast cancer incidence: differing interpretations by Clemmesen and MacMahon (MacMahon, 1957)

Figure 1–8a. Clemmesen's age-specific breast cancer incidence data for Denmark (1943–1947)

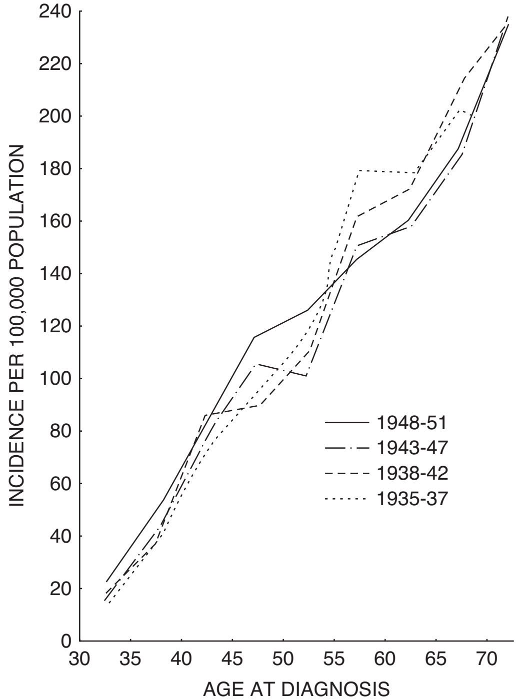

Figure 1–8b. MacMahon's analogous age-specific breast cancer incidence data for Connecticut (1935–1951)

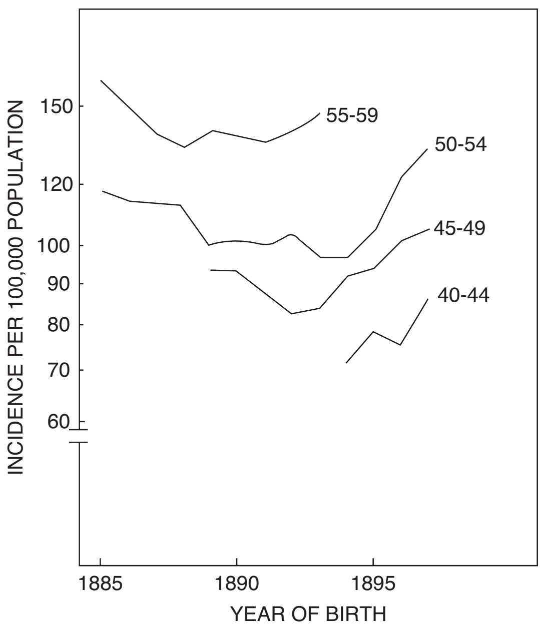

Figure 1–8c. MacMahon's re-expression of the Connecticut data for specific age groups, by birth cohort

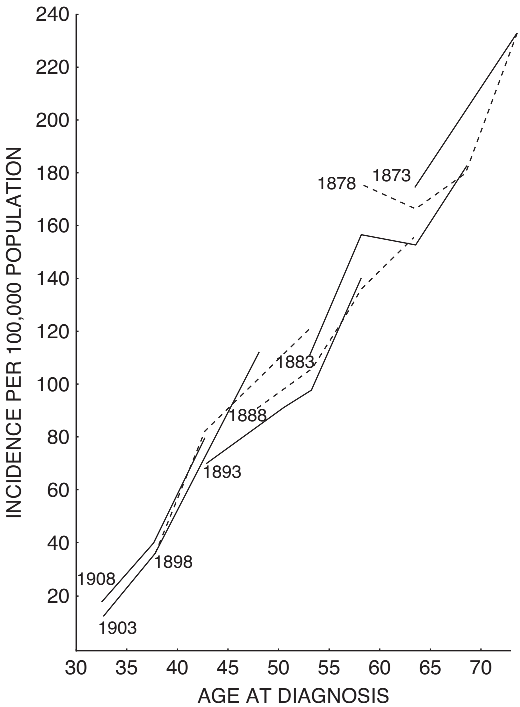

Figure 1–8d. MacMahon's re-expression of the Connecticut age-specific incidence data, by birth cohort

In another sense, meaningful observation is experience-laden: we need familiarity not only with the concepts at issue but also the experience of looking at the data themselves and working with the methods to do so. In other words, trained judgment (Daston & Gallison, 2007). Or, as Ludwig Fleck (1896–1961) wrote in the early twentieth century, even with the expectation that when we look through a microscope we will see cells and micro-organisms, we need to learn to prepare the sample with appropriate methods (e.g., stains) and likewise need to learn to "see," to decipher what is "signal" and what is "noise" (based on theory-laden ideas about what is being observed) (Fleck, 1929; Daston, 2008); the same holds for when we look at epidemiologic data. These statements do not mean that when we do science, we can "see" just anything we please. What counts as scientific evidence is not idiosyncratic; it is instead bound to the assumption of a shared biophysical world and the replicable, contestable, and debatable work of scientists, conducted in the public domain and collectively interpreted and argued.

Fourth, science is by definition fallible—in part because the testing of evidence and ideas, with or without new technologies, can result in the refinement and at times partial (and occasionally wholesale) replacement of explanatory theories, leading to new insights and new predictions as well as new interpretations (or dismissals) of prior observed associations (Fleck, 1935 [1979]; Cohen, 1985; Mendelsohn et al., 1997; Ziman, 2000; Sober, 2008). The recognition that science yields provisional and fallible knowledge, however, does not render all scientific knowledge equally tentative: some theories and their diverse predictions have withstood repeated tests; some hypotheses have been tested only a handful of times. For example, the scientific evidence that biological evolution occurs is rich and robust to the point where scientists concur its existence is a fact—even as lively scientific controversies exist over the causal processes at play (Mayr, 1982; Eldredge, 1999; Gould, 2002; Grene & Depew, 2004; Eldredge, 2005; Sober, 2008).

The testing and evaluation of scientific theories, however, as recognized by an enormous literature, is multifaceted and complex and involves debates over methods as well as substance (Fleck, 1935 [1979]; Lieberson, 1992; Mendelsohn et al., 1997; Ziman, 2000; Gadenne, 2002; Grene & Depew, 2004; Daston & Gallison, 2007; Archer et al., 1998; Sober, 2008). Rarely, if ever, does it simply follow the pristine hypothetico-deductive logic of particular observations refuting entire theories—a stance famously postulated by influential philosopher of science, Sir Karl Popper (1902–1994; Popper, 1959, 1985), and one that has been subjected to serious critique in contemporary philosophy of science (Hacking, 2001; Collins, 2001; Mjøset, 2002; Sober, 2008) (even as it has had its share of adherents in epidemiology [Rothman, 1986; Rothman, 1988; MacClure, 1995]) as well as some epidemiologic critics [Susser, 1986; Pearce & Crawford-Brown, 1989; Krieger, 1994; Greenland, 1998]). The theory of general relativity, for example, does not mean Newtonian mechanics are wrong, but rather that the latter is a subset of the former, applicable only at certain spatiotemporal scales (Hanson, 1958; Holton & Brush, 2001). Moreover, an important asymmetry exists between evaluating results from a particular study to (*1*) decide if they are compatible with a particular theory versus (*2*) determine how much they strengthen or weaken confidence in a theory (Lieberson, 1992). In part, this is because even if the study results are accurate and valid, it is highly implausible a given data set contains enough elements to test all competing hypotheses (especially under alternative sets of conditions). Thus, as noted by Lieberson, in the case of probabilistic theories, "a theory may be correct even if there is negative evidence" (Lieberson, 1992, p. 1)—and understanding why this can occur requires in-depth consideration of the conditions under which certain associations would or would not be expected.

More deeply, however, science is fallible because as historians and other analysts of science have extensively documented (Fleck, 1935 [1979]; Rose & Rose, 1980; Desrosières, 1988; Holton, 1988; Hubbard, 1990; Rosenberg & Golden, 1992; Keller, 1995; Massen et al., 1995; Mendelsohn et al., 1997; Lock & Gordon, 1988; Ziman, 2000; Keller, 2002; Harraway, 2004; Longino, 2006), scientists are part of the societies in which they are raised and work and, consequently, both think with—and sometimes challenge—the ideas and beliefs of their times. The eighteenth to nineteenth century scientific shift from a constrained biblical time-scale to expansive notions of "deep time" not only reflected fundamental changes in theories of geology, cosmology, physics, and biology but also constituted a profound rupture with dominant and deeply held religious views (Mayr, 1982; Gould, 1987; Holton, 1988; Eldredge, 2005). Closer to home for epidemiology are the powerful and painful connected examples of scientific racism and eugenics and their views of innately biologically inferior and superior "races"—which, far from being "crackpot" theories, were widely accepted and promoted by leading scientists in the nineteenth and the first half of the twentieth centuries (Chase, 1977; Harraway, 1989; Harding, 1993; Kevles, 1995; Gould, 1996; Banton, 1998; Harris & Ernst, 1999; Allen, 2001; Proctor, 2003; Lewontin et al., 1984; Jackson & Weidman, 2004; Stern, 2005a). Their lingering influence on how epidemiologists and others analyze racial/ethnic—and also socioeconomic—health inequities remains a topic of considerable concern (Krieger, 1987; Muntaner et al., 1996; Stern, 2005b; Krieger, 2005; Duster, 2006; Braun et al., 2007).

Fifth and finally, science is not the sole arbiter of knowledge, and scientific theories are not the only path to wisdom. It would be hubris to think otherwise (and not just because of