



Proximal, Distal, and the Politics of Causation: What's Level Got to Do With It?

Nancy Krieger, PhD

Causal thinking in public health, and especially in the growing literature on social determinants of health, routinely employs the terminology of *proximal* (or *downstream*) and *distal* (or *upstream*).

I argue that the use of these terms is problematic and adversely affects public health research, practice, and causal accountability. At issue are distortions created by conflating measures of space, time, level, and causal strength.

To make this case, I draw on an ecosocial perspective to show how public health got caught in the middle of the problematic proximal–distal divide—surprisingly embraced by both biomedical and social determinist frameworks—and propose replacing the terms *proximal* and *distal* with explicit language about levels, pathways, and power. (*Am J*

Public Health. 2008;98:221–230. doi:10.2105/AJPH.2007.111278)

PROXIMAL. DISTAL. UPSTREAM.

Downstream. Risk factor. Determinant. Level. Multilevel. These terms feature prominently in current discussions of causal pathways and public health, especially in work on the social determinants of health. A central focus is on how “upstream” societal influences—typically referred to as *distal*—shape “downstream,” or *proximal*, exposures, thereby affecting population health.^{1–16} Exemplifying this line of thought are recent reports issued by the World Health Organization Commission on Social Determinants of Health² and the World Health Organization Regional Office for Europe.³ Common assumptions are that (1) diseases

are attributable to many causes, located outside and within the body; (2) the social lies in the realm of the *distal*; (3) the biological belongs to the *proximal*; and (4) the distal and proximal are connected by *levels*, e.g., societal, institutional, household, individual, which can be conceptualized as *near to* or *far from* the causes under consideration. For example, as discussed in both reports, “distal” societal factors drive the risk of smoking; how smoking harms health involves “proximal” biology.^{2,3} What could be more obvious?

Yet what seems clear-cut can be deceiving. I argue that although notions of *proximal*, *distal*, and *level* all matter for elucidating causal pathways, clear thinking—and, hence, public health research, practice, and causal accountability—is distorted

by conflating measures of space, time, level, and causal strength. When it comes to causation, it is one thing to think about *near* and *far* in relation to space and time; it is another matter entirely to do so for levels. To make this intellectual argument, I draw on an ecosocial perspective^{1,17–21} to show how public health got caught in the middle of the problematic proximal–distal divide—surprisingly embraced by both biomedical and social determinist frameworks—and propose replacing the terms *proximal* and *distal* with explicit language about levels, pathways, and power.

PROXIMAL AND DISTAL IN PUBLIC HEALTH THOUGHT

The idea that disease etiology and distribution are attributable to causes deemed “far” from and



“near” (including within) the body is ancient^{22–27}; Hippocratic tradition, in the 5th century BCE, famously invoked both atmosphere and individual constitution as explanations for epidemic disease.^{22,25} By contrast, the idea that there is a causal etiological hierarchy, spanning from distal to proximal, is relatively new. It became a core part of the public health canon only in the mid–20th century CE. How this change happened and its public health implications have been little discussed.

Strand 1. 19th Century Emergence of Proximal and Distal as Scientific Terms for Spatiotemporal Scale

Only in the early 19th century CE did the terms *proximal* and *distal* enter the scientific discourse.²⁸ Invented to describe anatomical location and distance, as measured on a spatial scale, these words were coined by biologists at a time when comparative anatomy occupied a key place in debates over the classification and nature of species.^{29,30} *Proximal*, derived from the Latin noun *proximus* (“nearest”), took on the meaning “situated toward the center of the body, or the point of origin or attachment of a limb, bone, or other structure.”²⁸ Its antonym, *distal*, derived from *distant*, was intended to echo 2 other widely used biological concepts: *ventral* and *dorsal*.²⁸ Soon other natural sciences adopted the terms, albeit with some critical modifications. In geology, for example, the terms took on a temporal as well as a spatial dimension, reflecting how adjacent

geological strata typically are “close” in time as well as in space.²⁸

The moment time entered the picture, however, the terms *proximal* and *distal* were primed to develop new meanings. This is because of the ubiquitous metaphorical linkage of time, space, and causal reasoning.^{31(pp133–138)} In all known languages, temporal events are described in spatial terms: Time moves through space.^{31(p134)} This metaphorical relationship, as argued by the linguist Deutscher, is essential to causal reasoning, because it enables us to “talk freely about one thing coming ‘from’ another, ‘out of’ another, or happening ‘through’ another, to express abstract chains of cause and event.”^{31(p137)}

New European scientific discoveries of powerful physical laws for gravity, electricity, and magnetism^{32–34} further affected scientific thinking about causation. These inverse square laws, expressed as pithy equations, clarified that force depends on distance: The more proximal the mass or the charge of the interacting objects, the greater the force—and the more powerful the effect. It was a short step from here to equate distance with causal strength, in not only the physical but also the life sciences.

Strand 2. From Spatiotemporal Scale to Causal Hierarchies and Levels

Not until the later 19th century, however, did the scientific meanings of *proximal* and *distal*

leap from referring only to *spatiotemporal scale* to also describing *levels* and *causal hierarchies*. In their new usage, the “closeness” or “distance”—of levels defined a new type of proximity, one that could be measured only conceptually, not in meters or minutes.

Initially, this conceptual change occurred within disciplines focused on a different type of body: that of body politic, i.e., the social sciences.^{35–42} In books with such titles as *Social Pathology*⁴¹ and *Organism and Society*,⁴² influential late–19th century sociologists drew parallels between the biologically nested hierarchies of cell–organism–species and the socially nested hierarchies of individuals–families–societies.^{35, 36,37(pp4–8),38(pp231–323),40–42} In their view, just as organs, composed of their constituent cells, must collectively work together for an organism to survive, so too do social groups and their constituent individuals have complementary roles they must perform for society to thrive.^{35,36,41,42} The intent was counter not only to the ruthless competition of Social Darwinism^{30(pp87–90),39, 40(pp196–199),42(p10)} but also to the contending Marxist view that class conflict determined societies’ structure and development.^{35, 37(pp4–8),41,42(pp182–186),43,44(pp178–179)} Borrowing biological terminology, these sociologists newly deployed the terms *proximal* and *distal* to describe societies’ structural “levels.”^{41(xxiii)} Ranging from individual to institutional, these levels and the “distance” between them became defined by their nested relationships:

Adjacent levels were “close,” and nonadjacent levels were “far.”

Meanwhile, biologists likewise expanded the use of the terms *proximal*, *distal*, and *level*, bringing these terms explicitly into their thinking about causal distance. As part of the early 20th century modern evolutionary synthesis, which integrated Darwinian evolutionary biology, paleontology, and Mendelian genetics,^{29,45,46(pp503–591),47} these biologists newly contrasted what they termed “proximate” (physiological) versus “ultimate” or “distal” (evolutionary) causes.^{29(pp313–321), 35,46(pp1340–1343),47} This distinction recognized that asking *how* a biological event occurs (e.g., a muscle contraction) is not the same as asking *why* a biological phenomenon exists (e.g., muscles enable locomotion to find food and flee predators). Drawing on holistic thinking,^{35,48} they argued that valid explanations could coexist across levels (e.g., species, organism, cell, molecule) and involve the distant past (evolution) and the immediate present (current stimulus). In the instant of a muscle contraction, both proximal and distal causes were at play.

The Mid–20th Century Public Health Embrace of Proximal and Distal

By mid–20th century, to be close or far could thus refer to space, to time, to lineage, or to location in hierarchical conceptual levels. The terms *proximal* and *distal* thus became widely encompassing terms to express—and contest—causal conceptions in both the social and the natural



sciences. Amid these divergent uses, the terms *proximal* and *distal* finally entered the public health causal lexicon.

Prompting their adoption was growing recognition that the field of public health, still riding the crest of enormous success against infectious diseases in the 19th and 20th centuries CE, had to move beyond a monocausal to a multifactorial account of disease causation, which involved not only the agent but also the host and the environment.^{1,17,24,49–51} As exemplified by the findings of the Framingham study of heart disease, rising rates of chronic disease and cardiovascular mortality seemed to be attributable not to any one single exposure but instead to a variety of factors,^{51–53} leading the Framingham researchers to coin the term *risk factor* to describe these partial—i.e., not sufficient, not always necessary, but nonetheless contributing—component causes.⁵³

It was through the multifactorial perspective that the terms *proximal* and *distal* emerged as terms for the discussion of causality in the public health literature.¹⁷ Unfortunately, however, their new usage drew on shallow understandings of the terms *near* and *far* that impeded rather than deepened multilevel thinking. The essential features of the multifactorial framework remain well-sketched by the still highly influential spiderless¹⁷ “web of causation,” first articulated in the 1960s⁵⁴ and which, as I have previously argued,¹⁷ (1) leveled all exposures to a single plane; (2) defined “proximal” factors to

be those operating directly on or within the body, and relegated all other exposures to the murky realm of “distal”; (3) linked causal potency to distance—i.e., the “closer” the cause, the greater the effect (following the logic of the previously described physical inverse square laws); (4) held that distal causes necessarily exerted their influence through successively more proximal factors; (5) took a studied agnosticism as to what accounted for the array of exposures included in the web and eschewed any discussion of power or injustice; and, hence, (6) adopted a narrow stance of what may best be termed *causal pragmatism*^{55,56} that prioritized focusing on what they considered to be “proximal” factors ostensibly amenable to control by either individuals or by public health or medical professionals (including by health education) rather than what they termed the more “distal” determinants requiring societal change.

The use of the terms *proximal* and *distal* persists to this day. It underlies the 21st century successor to the web of causation—that is, the “gene–environment interaction” framework,^{57–60} which posits that the occurrence of common and complex diseases reflects the interplay of individual genetic variability with an array of exogenous exposures.^{57–60} Work in this area is chiefly engaged^{57–60} (albeit with some exceptions^{61–65}) in the quest to discover genetic determinants of biological susceptibility and to develop pharmacological interventions that can block deleterious gene expression.

The proximal–distal discourse likewise pervades the social determinants of health perspective,^{1–16} which holds that “distal” institutional priorities and practices of government and the private sector shape people’s cumulative exposure, across the life course via intermediary pathways, to the proximal physical, behavioral, psychosocial, and biological exposures that trigger pathogenic processes (including gene expression), thereby causing disease. Secondarily, once illness occurs, the social determinants of health framework asks how prognosis is affected by socially produced inequities in access to needed medical care.^{1–16}

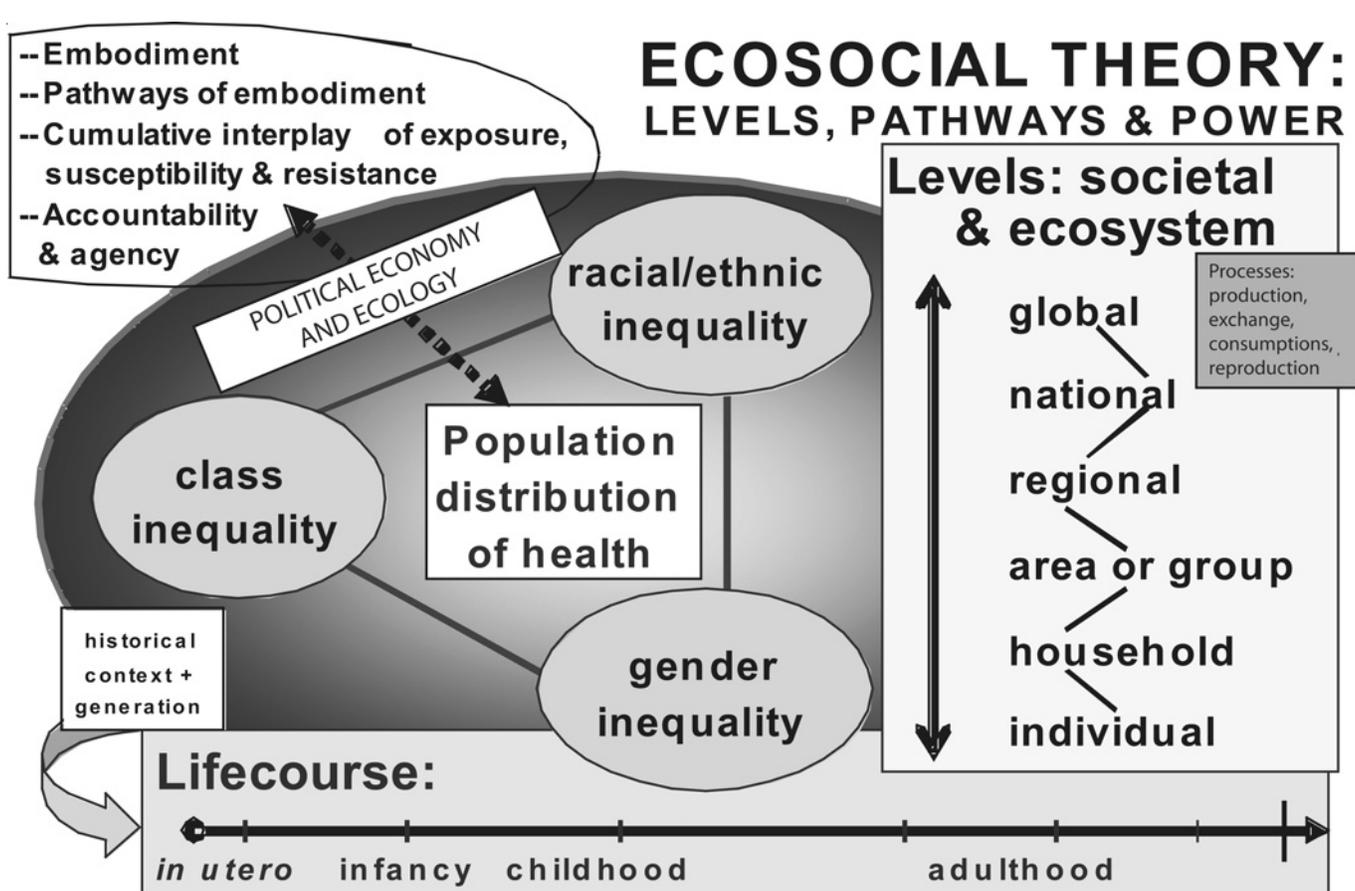
In both cases, causal distance still matters for causal strength: In the gene–environment interaction model, “proximal” causes remain most potent, whereas for the social determinants of health perspective, “distal” causes are decisive. Despite their fundamentally different approaches, both frameworks cling to the proximal–distal divide. This little remarked convergence hints that some causal logic may be askew.

AN ALTERNATIVE ECOSOCIAL APPROACH TO LEVELS, EMBODIMENT, AND ACCOUNTABILITY

I suggest that one reason the proximal–distal terminology can be so readily used by such totally disparate frameworks is their now deeply entrenched conflation of relationships among space, time, distance, levels, and causal potency. Three examples, based on arguments offered from

an ecosocial perspective (Figure 1, Table 1),^{1,17–21} supplemented by the conceptual clarifications provided in Box 2, illustrate the problems that can arise when logics of scale are confused with analysis of levels and when distance is conflated with power.

The basic point is that societal patterns of disease represent the biological consequences of the ways of living and working differentially afforded to the social groups produced by each society’s economy and political priorities.^{1,17–21} Class and racial inequality, for example, differentially affect the living standards, working conditions, and environmental exposures of the dominant and subordinated classes and racial/ethnic groups, thereby creating class and racial/ethnic health disparities. Stated more generally, a society’s economic, political, and social relationships affect both how people live and their ecologic context, and, in doing so, shape patterns of disease distribution. The understanding of the societal distributions of health thus cannot be divorced from considerations of political economy and political ecology.^{1,17–21} Driving health inequities are how power—both *power over* and *power to do*,^{66–68} including constraints on and possibilities for exercising each type—structures people’s engagement with the world and their exposures to material and psychosocial health hazards. Notably, neither type of power readily maps onto a metric of proximal or distal. Nor do they neatly partition across levels. A critical



Note. To explain current and changing population distributions of disease, including health inequities, and who and what is accountable for the societal patterning of health, it is necessary to consider causal pathways operating at multiple levels and spatiotemporal scales, in historical context and as shaped by the societal power relations, material conditions, and social and biological processes inherent in the political economy and ecology of the populations being analyzed. The embodied consequences of societal and ecologic context are what manifest as population distributions of and inequities in health, disease, and well-being.

Source. See references 1,17-21.

FIGURE 1—A heuristic diagram for guiding ecosocial analyses of disease distribution, population health, and health inequities.

corollary is that, contrary to the logic of the proximal–distal divide, within the very phenomena of disease occurrence and distribution—just as in a muscle contraction—the distal and the proximal are conjoined.

Example 1. Why Spatiotemporal Scale Is Not the Same as Level

The first example, drawn from ecology, the original multilevel

science, clarifies why population sciences cannot afford to confuse metrics of spatiotemporal scale with the phenomena of levels. The example concerns, literally, the forest and the trees. Forests are levels within ecosystems, which involve not only trees but also the other plants and animals that inhabit them.^{69–75} Notably, forests can be large or small (a spatial metric), as well as old or young (a temporal metric). Indeed,

one key issue in conservation ecology today, spurred by intensified commercially driven logging and deforestation, forest fragmentation, habitat degradation, and spread of zoonoses (e.g., Lyme disease), is just what size, spatially, an expanse of woods needs to be—and how close it needs to be to other such expanses—to function as a particular type of forest.^{69–75} Too small, with the ratio of edge-to-interior too high, or too spatially

isolated, without connecting corridors, and its species composition will change, often losing diversity, including to the point of outright extinction.^{69–75} The phenomenon of a forest (a level), and interactions among both the entities that constitute it and also between the forest and its environs, is affected by, but not identical to, the forest's size (spatiotemporal scale). Similarly, for measles to become endemic



TABLE 1—Core Constructs of Ecosocial Theory—an Epidemiological Theory of Disease Distribution—and Some Predictions

Construct	Elaboration
Embodiment	A concept that refers to how we literally incorporate, biologically, the material and social world in which we live, from in utero to death; a corollary is that no aspect of our biology can be understood absent knowledge of history and individual and societal ways of living. Epidemiologically, “embodiment” is thus best understood: (1) As a construct, process, and reality, contingent upon bodily existence; (2) As a multilevel phenomenon, integrating soma, psyche, and society, within historical and ecological context, and, hence, an antonym to disembodied genes, minds, and behaviors; (3) As a clue to life histories, hidden and revealed; and (4) As a reminder of entangled consequences of diverse forms of social inequality.
Pathways of embodiment	Causal pathways that involve exposure, susceptibility, and resistance (as both social and biological phenomena), structured simultaneously by (1) societal arrangements of power, property, and contingent patterns of production, consumption, and reproduction, and (2) constraints and possibilities of our biology, as shaped by our species’ evolutionary history, our ecologic context, and individual histories, that is, trajectories of biological and social development, and that involve gene expression, not just gene frequency.
Cumulative interplay among exposure, susceptibility, and resistance	Expressed in pathways of embodiment, with each factor and its distribution conceptualized at multiple levels (individual, neighborhood, regional or political jurisdiction, national, international, or supranational) and in multiple domains (e.g., home, work, school, other public settings), in relation to relevant ecologic niches, and manifested in processes at multiple scales of time and space.
Accountability and agency	Refers to who and what is responsible for social inequalities in health and for rectifying them, as well as for the overall current and changing contours of population health, as expressed in pathways of and knowledge about embodiment. At issue are the accountability and agency of not only institutions (government, business, and public sector), communities, households, and individuals, but also of epidemiologists and other scientists for theories used and ignored to explain social inequalities in health. A corollary is that, given likely complementary causal explanations at different scales and levels, epidemiological studies should explicitly name and consider the benefits and limitations of their particular scale and level of analysis.
Analytic implications and predictions	Determinants of disease distribution (a population-level phenomenon) presume but are not reducible to mechanisms of disease causation (which occur within individuals’ bodies). Key contingent hypotheses are: (1) population patterns of health and disease constitute the embodied biological expression of ways of living and working differentially afforded by each society’s political economy and political ecology, and (2) policies and practices that benefit and preserve the economic and social privileges of dominant groups simultaneously structure and constrain the living and working conditions they impose on everyone else, thereby shaping particular pathways of embodiment.

Source. See references 1,17–21.

Consider, for example, the 1973 US Supreme Court ruling that legalized abortion, on the grounds of individuals’ rights to privacy.⁸⁰ Here, the levels at issue were defined jurisdictionally, with the federal judicial ruling on individual constitutional rights overturning federal and state laws that interfered with individual privacy by prohibiting abortion. In this case, the so-called distal determinant (1) directly affected individual girls’ and women’s reproductive rights and (2) reverberated up to other levels, by requiring changes in state laws and by expanding the permitted range of services that could be provided by health professionals and health facilities.

The positive health consequences were both immediate and long-term: US girls and women alike no longer were forced, by law, to face the risk of having an unsafe illegal abortion and they were also less likely to bear unwanted children, thereby reducing risks of adverse maternal and birth outcomes.^{81–84} More recent US Supreme Court decisions restricting the right to abortion likewise illustrate this principle of skipping levels, with contrary effects.^{85,86}

Analogous examples can readily be drawn from the health and human rights literature, whereby state obligations to respect, protect, and fulfill individuals’ human rights affect policies and interventions at multiple levels.⁸⁷ The implication, argument 2, is that non-adjacent levels can have direct causal relationships, an insight obscured by the proximal–distal logic.

with a community (a level), community size (a scale) must exceed 250 000 people.^{76,77} Hence, argument 1: Confuse scale and level—or consider only one, not both—and understanding of population phenomena will be undermined.

Example 2. On Nonlinear Causal Pathways, With Immediate and Long-Term Effects

The second example illustrates that levels need not play by the proximal–distal schema that the path from what is

considered “far” to “near” necessarily travels through what is termed “intermediate.” This is because events at one level can directly and profoundly affect nonadjacent levels, instantly and persistently, without intermediaries.^{29,46–48,66,78,79}



TABLE 2—Proximal and Distal, Spatiotemporal Scale Versus Level—Meanings, Contrasts, and Causal Implications

Category	Spatiotemporal Scale		Level
	Space	Time	
Metric of distance	Units of spatial distance, measured in nested increments; examples include: kilometer-meter-millimeter-micron; or mile-foot-inch	Units of temporal distance, measured in nested increments; examples include: millennium-century-year-day-hour-minute-second-millisecond	Adjacency of levels, which can be organized—theoretically, conceptually, or structurally—as nested or nonnested hierarchies; examples include: (1) nested: nation-region-city-neighborhood-household; or ecosystem-species-organism-organ system-organ-cell; (2) nonnested: school workplace neighborhood-individual
“Near”	Proximal, near in space, close	Proximal, near in time, recent	Conceptual or structural nonscalar relationship: adjacent levels
“Far”	Distal, distant in space, far away	Distal, distant in time, long ago	Conceptual or structural nonscalar relationship: nonadjacent levels
Strength of effect	Usually inverse relationship of spatial distance and force: closer = stronger, hence proximal = powerful; farther = weaker, hence distal = dilute	Usually inverse relationship of temporal distance and force: closer = stronger, hence proximal = powerful; farther = weaker, hence distal = dilute	Cannot predict “strength” of “effect” based solely on level: a given phenomenon at any given level potentially can powerfully or weakly affect or be affected by phenomena at the same level, adjacent levels, and nonadjacent levels
Typical causal inference	Proximal = stronger cause Distal = weaker cause	Proximal = stronger cause Distal = weaker cause	Causal inference depends on level of question being asked: There may be different explanations for phenomena at different levels, and explanations for events observed within any given level may involve solely phenomena within that level or also interactions between levels; adjacency of levels may or may not predict causal strength of cause-effect relationship
Relationship to space and time	Physical distance is a spatial dimension distinct from time, but space and time can be related mathematically, e.g., distance = speed × time (and the length of a meter is now defined in relation to time and the speed of light ^{32(pp537)})	Chronological distance is a temporal dimension distinct from space, but time and space can be related mathematically, e.g., time = distance/speed (and initial time units were based on the earth’s rotation, involving spatial distance ^{32(pp3-5)})	Level is not a spatiotemporal phenomenon. It is, instead, a conceptual nonspatiotemporal relational construct that organizes and distinguishes (conceptually or structurally) different orders of hierarchically linked systems and processes (including both nested and nonnested hierarchies). “Distance” for levels does not involve spatiotemporal separation: For any phenomenon at any given point in space and time, all levels co-occur simultaneously, even though some levels may be more causally relevant than others to phenomena occurring at any given level. Space and time nevertheless do matter for levels in the case of nested hierarchies, whereby units within lower-order levels typically are smaller and involve faster processes than units in higher-order levels.

Example 3. On Levels and the Perils of Commodity Fetishism—the Simultaneity of Material Properties and Social Relations

The third example involves a key problem that permeates

the proximal–distal divide: its incompatibility with truly multi-level thinking. This problem can be likened to the old-fashioned error of “commodity fetishism,” albeit multiplied. In its original usage, this concept, introduced

by Karl Marx (1818–1883), referred to how the value of commodities was mistakenly assumed to be an intrinsic property, rather than a consequence of the complex relationships of ownership, labor, and exchange

inherently involved in their production, sale, and consumption.^{43(pp35–41,71–83),88} Erring, however, in both directions, whether looking up or down levels, the proximal–distal divide simultaneously does the following:



(1) It promotes analysis of specific exposures and their biological embodiment stripped from the political economy, social relations, actual labor, and engagement with the material world that set the basis for their existence (the error of biomedical individualism and decontextualized “lifestyle” analyses^{1–21,89–91}) and

(2) It encourages analysis of population health as if all that matters are social hierarchies, and not also the tangible properties of the commodities, i.e., goods and services, at issue (the error of public health nihilism^{5,92–94}).

Thus, on the proximal side, official conventional reports^{95,96} urge individuals to avoid specific risk factors without mention of the societal changes needed to curtail these factors’ production, distribution, and consumption (precisely what the social determinant of health framework appropriately criticizes),^{1–21}

whereas on the distal side, some contend that public health initiatives that focus on specific risk factors or diseases are futile as long as “distal” or “fundamental” causes are at play.^{13,14,97–99}

But insofar as health is concerned, the material substances and the social relations inherent in any given product or process both matter, precisely because of the physical and social exposures involved. To focus on only one or the other misses the fact we embody both.^{1,17–21,94,100–103} To take but one example, consider the political economy and ecology of tobacco products and their embodied health consequences. A

cigarette (or Freud’s infamous cigar¹⁰⁴) is *simultaneously*:

(1) A combustible mass of tobacco leaves and additives whose burning smoke transports psychoactive and addictive chemicals (e.g., nicotine) and carcinogens deep down the respiratory tract to the innermost parts of the lung and its alveolar capillaries, thereby increasing risk of cancer, cardiovascular and pulmonary disease, and other smoking-related ailments, and

(2) A highly profitable product whose production, distribution, advertisement, and consumption involves relentless corporate marketing (including manipulation of ideologies involving freedom, class, gender, sexuality, and race/ethnicity and targeting of marginalized groups), government regulation and taxation, tobacco farmers and workers, land ownership, trade agreements, and international treaties.^{105–109}

Consequently, as recognized by several new sophisticated multilevel initiatives (e.g., Sweden’s 2003 new public health policy,¹¹⁰ the American Legacy Foundation’s Truth Campaign,¹¹¹ and the Corporations and Health Watch project¹¹²), effective action to curb tobacco use and social disparities in tobacco-related diseases requires integrated, multifaceted, multilevel campaigns that are relentlessly honest about who gains and who loses from the status quo. The same could be said for any other public health concerns deemed “proximal” or “downstream,” whether about environmental and

occupational hazards,^{6,8,9,113–116} access to safe water,^{117,118} access to affordable nutritious food,^{119,120} or violence,^{121–123}—just as could be said for efforts focused on such ostensibly “distal” or “upstream” social determinants as economic poverty.^{124–130}

Hence, argument 3: Unlike distal and proximal events separated by space or time, levels coexist simultaneously, not sequentially, and exert influence accordingly. The proximal–distal divide, however, inherently cleaves levels rather than connects them, thereby obscuring the intermingling of ecosystems, economics, politics, history, and specific exposures and processes at *every* level, macro to micro, from societal to inside the body. As William Blake (1757–1827) famously put it, the challenge instead is “to see a world in a grain of sand”¹³¹—because it is there.

SCALE, LEVEL, AND THE POLITICS OF CAUSATION

In summary, efforts to advance public health thinking and work about the causes of disease distribution, including health inequities, would do well to abandon the deeply confused language of the terms *proximal* and *distal*. The point is not simply semantic. Clear action requires clear thinking. By deleting the terms *proximal* and *distal* from the public health lexicon, we would have to expose our causal assumptions and also promote greater accountability for the public’s health, both within our field and more broadly.

A final example suffices. In recent years, the Bill and Melinda Gates Foundation has become an enormous presence in work on global health,^{132–135} funding technically oriented¹³⁶ research and medical interventions to address malaria, tuberculosis, HIV/AIDS and other diseases that disproportionately burden poorer regions of the world. In January 2007, however, the Los Angeles Times published a 2-part exposé,^{137,138} “showing that the foundation reaps vast financial gains every year from investments that contravene its good works.”¹³⁹ The foundation’s response¹⁴⁰:

“The stories you told of people who are suffering touched us all. But it is naive to suggest that an individual stockholder can stop that suffering. Changes in our investment practices would have little or no impact on these issues. While shareholder activism has worthwhile goals, we believe a much more *direct* [italics added] way to help people is by making grants and working with other donors to improve health, reduce poverty and strengthen education.”¹⁴¹

The foundation’s view that its real-world health portfolio somehow includes only its explicit biomedical research and health intervention projects and not also the health impacts of its financial investment strategies is the mind-set fostered by the proximal–distal divide. The distance and contradictions created by the proximal–distal discourse—in conceptual understanding and in professional and political accountability—are unacceptable. The extensive reach of this



flawed logic is made only the more manifest by its equal use among those who profess a narrow biomedical vantage and those who articulate a more expansive social determinants of health framework. I accordingly propose that we banish the terms *proximal* and *distal* from the public health lexicon and refer instead explicitly to levels, pathways, and power, as one small but needed step toward developing better thinking and strategies for leveling health inequities. ■

About the Author

Nancy Krieger is with the Department of Society, Human Development, and Health at the Harvard School of Public Health, Boston, Mass.

Requests for reprints should be sent to Nancy Krieger, PhD, Professor, Department of Society, Human Development, and Health, Harvard School of Public Health, Kresge 717, 677 Huntington Ave, Boston, MA 02115 (e-mail: nkrieger@hsph.harvard.edu).

This article was accepted June 7, 2007.

Acknowledgments

The author thanks the following colleagues for useful discussions about the ideas in this article: George Davey Smith, Mary Bassett, Madeline Drexler, Sofia Gruskin, and Elizabeth Barbeau.

Human Participant Protection

No research on human subjects was performed.

References

- Krieger N. Theories for social epidemiology in the 21st century: an ecosocial perspective. *Int J Epidemiol*. 2001;30:668–677.
- World Health Organization Commission on Social Determinants of Health. Towards a conceptual framework for analysis and action on the social determinants of health. Discussion paper for the Commission on the Social Determinants of Health [draft]. May 5, 2005. Available at: http://www.who.int/social_determinants/knowledge_networks/en. Accessed March 2, 2006.
- Whitehead M, Dahlgren G. Leveling up (part 1): a discussion paper on concepts and principles for tackling social inequities in health. Studies on social and economic determinants of population health, No. 2, WHO Europe. Copenhagen, Denmark: World Health Organization Regional Office for Europe; 2006. Available at: <http://www.euro.who.int>. Accessed November 29, 2006.
- Black D, Morris JN, Smith C, Townsend P. *The Black Report (Report of the Working Group on Inequalities in Health)*. London, England: Penguin; 1982.
- Kunitz S. *The Health of Populations: General Theories and Particular Realities*. Oxford, England: Oxford University Press; 2006.
- Levy BS, Sidel VW, eds. *Social Injustice and Public Health*. New York, NY: Oxford University Press; 2006.
- Wilkinson R, Marmot M, eds. *Social Determinants of Health: The Solid Facts*. 2nd ed. Oxford, England: Oxford University Press; 2006.
- Navarro V, Muntaner C, eds. *Political and Economic Determinants of Population Health and Well-Being: Controversies and Developments*. Amityville, NY: Baywood Publishing Co; 2004.
- Hofrichter R, ed. *Health and Social Justice: Politics, Ideology, and Inequity in the Distribution of Disease*. San Francisco, Calif: Jossey-Bass; 2003.
- Evans T, Whitehead M, Diderichsen F, Bhuiya A, Wirth M, eds. *Challenging Inequities in Health: From Ethics to Action*. Oxford, England: Oxford University Press; 2001.
- Berkman L, Kawachi I, eds. *Social Epidemiology*. Oxford, England: Oxford University Press; 2000.
- Shaw M, Dorling D, Gordon D, Davey Smith G. *The Widening Gap: Health Inequalities and Policies in Britain*. Bristol, England: Policy Press; 1999.
- McKinlay JB, Marceau LD. To boldly go . . . *Am J Public Health*. 2000;90:25–33.
- Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav*. 1995;35(extra issue): 80–94.
- Glass TA, McAttee MJ. Behavioral science at the crossroads in public health: extending horizons, envisioning the future. *Soc Sci Med*. 2006;62: 1650–1671.
- Richard L, Potvin L, Kishchuk N, Pric H, Green LW. Assessment of the integration of the ecological approach in health promotion programs. *Am J Health Promot*. 1996;3:318–328.
- Krieger N. Epidemiology and the web of causation: has anyone seen the spider? *Soc Sci Med*. 1994;39: 887–903.
- Krieger N. Ecosocial theory. In: Anderson N, ed. *Encyclopedia of Health and Behavior*. Thousand Oaks, Calif: Sage; 2004:292–294.
- Krieger N. Embodiment: a conceptual glossary for epidemiology. *J Epidemiol Community Health*. 2005;59: 350–355.
- Krieger N, ed. *Embodying Inequality: Epidemiologic Perspectives*. Amityville, NY: Baywood Publishers; 2004.
- Krieger N. Stormy weather: race, gene expression, and the science of health disparities. *Am J Public Health*. 2005;95:2155–2160.
- Sigerist HE. *History of Medicine. Vol. 1: Primitive and Archaic Medicine; Vol. 2: Early Greek, Hindu, and Persian Medicine*. New York, NY: Oxford University Press; 1955–1961.
- Rosen G. *A History of Public Health* [1958]. [Introduction by Elizabeth Fee; bibliographical essay and new bibliography by Edward T. Morman.] Expanded ed. Baltimore, Md: Johns Hopkins University Press; 1993.
- Porter D. *Health, Civilization and the State: A History of Public Health From Ancient to Modern Times*. London, England: Routledge; 1999.
- Lloyd GER, ed. *Hippocratic Writings*. London, England: Penguin; 1983.
- Temkin O. *Galenism: Rise and Decline of a Medical Philosophy*. Ithaca, NY: Cornell University Press; 1973.
- Greenwood M. *Epidemiology: Historical and Experimental*. Baltimore, Md: Johns Hopkins; 1931;1–26.
- Oxford English Dictionary*. OED online. Available at: <http://dictionary.oed.com.ezp2.harvard.edu> (password required). Accessed January 8, 2006.
- Grene M, Depew D. *The Philosophy of Biology*. Cambridge, England: Cambridge University Press; 2004.
- Coleman W. *Biology in the Nineteenth Century: Problems of Form, Function, and Transformation*. New York, NY: John Wiley & Sons Inc; 1971.
- Deutscher G. *The Unfolding of Language*. London, England: William Heneemann; 2005.
- Holton G, Brush SG. *Physics, the Human Adventure: From Copernicus to Einstein and Beyond*. 3rd ed. New Brunswick, NJ: Rutgers University Press; 2001.
- Gleick J. *Isaac Newton*. New York, NY: Pantheon Books; 2003.
- Gillmor CT. *Coulomb and the Evolution of Physics and Engineering in Eighteenth-Century France*. Princeton, NJ: Princeton University Press; 1971.
- Cohen IB. An analysis of interactions between the natural sciences and the social sciences. In: Cohen IB, ed. *The Natural Sciences and the Social Sciences: Some Critical and Historical Perspectives*. Dordrecht, The Netherlands: Kluwer Academic Publishers; 1994: 1–100.
- Maasen S, Mendelsohn E, Weingart P, eds. *Biology as Society, Society as Biology: Metaphors*. Dordrecht, The Netherlands: Kluwer Academic Publishers; 1995.
- Harris JG. *Foreign Bodies and the Body Politic: Discourses of Social Pathology in Early Modern England*. Cambridge, England: Cambridge University Press; 1998.
- Maclay GR. *The Social Organism: A Short History of the Idea That a Human Society May Be Regarded as a Gigantic Living Creature*. Croton-on-Hudson, NY: North River Press; 1990.
- Chase A. *The Legacy of Malthus: The Social Costs of the New Scientific Racism*. New York, NY: Knopf; 1977.
- Durkheim E. *The Division of Labor in Society* [1893]. Halls WD, trans-ed. [Introduction by Lewis A. Coser.] New York, NY: Free Press; 1984.
- Lilienfeld P. *La Pathologie Sociale*. [Avec une préface de René Worms.] Paris, France: V. Giard, and EBrière; 1896.
- Worms R. *Organisme et Société*. Paris, France: V. Giard, and EBrière; 1896.
- Marx K. *Capital: A Critique of Political Economy. Vol. 1. The Process of Capitalist Production* [1867]. Engels F, ed;



Moore S, Aveling E, trans-ed. New York, NY: International Publishers; 1967.

44. Levitas R. *The Inclusive Society?: Social Exclusion and New Labour*. 2nd ed. Basingstoke, England: Macmillan; 2005.

45. Mayr E. Prologue: some thoughts on the history of the evolutionary synthesis. In: Mayr E, Provine WB. *The Evolutionary Synthesis: Perspectives on the Unification of Biology*. Cambridge, Mass: Harvard University Press; 1998: 1–48.

46. Gould SJ. *The Structure of Evolutionary Theory*. Cambridge, Mass: The Belknap Press of Harvard University Press; 2002.

47. Eldredge N. *The Pattern of Evolution*. New York, NY: W.H. Freeman & Co; 1999.

48. Lawrence C, Weisz G, eds. *Greater Than the Parts: Holism in Biomedicine, 1920–1950*. New York, NY: Oxford University Press; 1998.

49. Gordon JE. The world, the flesh and the devil as environment, host, and agent of disease. In: Galdston I, ed. *The Epidemiology of Health*. New York, NY: Health Education Council; 1953: 60–73.

50. Terris M. The epidemiologic tradition. The Wade Hampton Frost Lecture. *Public Health Rep*. 1979;94:203–209.

51. Susser M. *Causal Thinking in the Health Sciences: Concepts and Strategies of Epidemiology*. New York, NY: Oxford University Press; 1973.

52. Marmot M, Elliott P, eds. *Coronary Heart Disease Epidemiology: From Aetiology to Public Health*. Oxford, England: Oxford University Press; 1992.

53. Rothstein WG. *Public Health and the Risk Factor: A History of an Uneven Medical Revolution*. Rochester, NY: University of Rochester Press; 2003.

54. MacMahon B, Pugh TF, Ipsen J. *Epidemiologic Methods*. Boston, Mass: Little, Brown and Company; 1960.

55. Gannett L. What's in a cause? The pragmatic dimensions of genetic explanations. *Biol Philos*. 1999;14:349–374.

56. Gillies D. An action-related theory of causality. *Br J Philos Sci*. 2005;85: 823–842.

57. National Human Genome Research Institute, National Institutes of Health. The Genes, Environment and Health Initiative (GEI). Available at:

<http://www.genome.gov/19518663>. Accessed January 10, 2007.

58. *Genomics 2006 Program Review Book*. Atlanta, Ga: Centers for Disease Control and Prevention; 2006. Available at: <http://www.cdc.gov/genomics/activities/ogdp/2006.htm>. Accessed January 20, 2007.

59. Costa LG, Eaton DL, eds. *Gene-Environment Interactions: Fundamentals of Ecogenetics*. Hoboken, NJ: John Wiley & Sons; 2006.

60. Lewontin R. *The Triple Helix: Gene, Organism, and Environment*. Cambridge, Mass: Harvard University Press; 2000.

61. Smith GD, Gwinn M, Ebrahim S, Palmer LJ, Khoury MJ. Make it HuGE: human genome epidemiology reviews, population health, and the IJE. *Int J Epidemiol* 2006;35:507–510.

62. Keavney B, Danesh J, Parish S, et al. Fibrinogen and coronary heart disease: test of causality by “Mendelian randomization.” *Int J Epidemiol*. 2006; 35:935–943.

63. Nitsch D, Molokhia M, Smeeth L, DeStavola BL, Whittaker JC, Leon DA. Limits to causal inference based on Mendelian randomization: a comparison with randomized controlled trials. *Am J Epidemiol*. 2006;163:397–403.

64. Davey Smith G, Lawlor DA, Harbord R, et al. Association of C-reactive protein with blood pressure and hypertension: life course confounding and mendelian randomization tests of causality [published correction appears in *Arterioscler Thromb Vasc Biol*. 2005; 25:e129]. *Arterioscler Thromb Vasc Biol*. 2005;25:1051–1056.

65. Davey Smith G, Ebrahim S. “Mendelian randomization”: can genetic epidemiology contribute to understanding environmental determinants of disease? *Int J Epidemiol*. 2003;32:1–22.

66. McLennan G. Power. In: Bennett T, Grossberg L, Morris M, eds. *New Keywords: A Revised Vocabulary of Culture and Society*. Malden, Mass: Blackwell Publishing; 2005:274–278.

67. McFarland AS. Power: political. In: *International Encyclopedia of the Social & Behavioral Sciences* [online]. Elsevier; 2004:11936–11939. Available at: <http://www.sciencedirect.com.ezp1.harvard.edu/science> (password required). Accessed May 30, 2007.

68. Clegg SR. Power in society. In: *International Encyclopedia of the Social &*

Behavioral Sciences [online]. Elsevier; 2004:11932–11936. Available at: <http://www.sciencedirect.com.ezp1.harvard.edu/science> (password required). Accessed May 30, 2007.

69. Peterson DL, Parker VT, eds. *Ecological Scale: Theory and Application*. New York, NY: Columbia University Press; 1998.

70. Patz JA, Daszak P, Tabor GM, et al; Working Group on Land Use Change and Disease Emergence. Unhealthy landscapes: policy recommendations on land use change and infectious disease emergence. *Environ Health Perspect*. 2004;112:1092–1098.

71. Rudel TK. Shrinking tropical forests, human agents of change, and conservation policy. *Conserv Biol*. 2006; 20:1604–1609.

72. Laurance WF, Nascimento HE, Laurance SG, et al. Rain forest fragmentation and the proliferation of successional trees. *Ecology*. 2006;87: 469–482.

73. Ferraz G, Russell GJ, Stouffer PC, Bierregaard RO Jr, Pimm SL, Lovejoy TE. Rates of species loss from Amazonian forest fragments. *Proc Natl Acad Sci U S A*. 2003;100:14069–14073.

74. Damschen EI, Haddad NM, Orrock JL, Tewksbury JJ, Levey JJ. Corridors increase plant species richness at large scales. *Science*. 2006;313:1284–1286.

75. Brownstein JS, Skelly DK, Holford TR, Fish D. Forest fragmentation predicts local scale heterogeneity of Lyme disease risk. *Oecologia*. 2005;146: 469–475.

76. Keeling MJ, Grenfell BT. Disease extinction and community size: modeling the persistence of measles. *Science*. 1997;275:65–67.

77. Bartlett MS. Measles periodicity and community size. *J R Stat Soc [Ser A]*. 1957;120:48–60.

78. Ahl V, Allen TFH. *Hierarchy Theory: A Vision, Vocabulary, and Epistemology*. New York, NY: Columbia University Press; 1996.

79. Gunderson L, Holling CS, eds. *Panarchy: Understanding Transformations in Human and Natural Systems*. Washington, DC: Island Press; 2002.

80. Goldstein LF, ed. *Contemporary Cases in Women's Rights*. Madison, Wis: University of Wisconsin Press; 1994: 3–32.

81. Institute of Medicine. *Legalized Abortion and the Public Health: Report of a Study, by a Committee of the Institute of Medicine*. Washington, DC: National Academy of Sciences; 1975.

82. Lee KW, Paneth N, Gartner LM, Pearlman MA, Gruss L. Neonatal mortality: an analysis of the recent improvement in the United States. *Am J Public Health*. 1980;70:15–21.

83. Pakter J, Nelson F. Factors in the unprecedented decline in infant mortality in New York City. *Bull N Y Acad Med*. 1974;50:839–868.

84. Lanham JT, Kohl SG, Bedell JH. Changes in pregnancy outcome after liberalization of the New York State abortion law. *Am J Obstet Gynecol*. 1974;118:485–492.

85. Wright AA, Katz IT. Roe versus reality—abortion and women's health. *New Engl J Med*. 2006;355:1–9.

86. Stout D. Supreme court upholds ban on abortion procedure. *New York Times*, April 18, 2007. Available at: <http://www.nytimes.com/2007/04/18/us/18cnd-scotus.html?ex=1180756800&en=f3466a0d04fb44bb&ei=5070>. Accessed May 31, 2007.

87. Gruskin S, Grodin MA, Annas GJ, Marks SP, eds. *Perspectives on Health and Human Rights*. New York, NY: Routledge; 2005.

88. Frow J. Commodity. In: Bennett T, Grossberg L, Morris M, eds. *New Keywords: A Revised Vocabulary of Culture and Society*. Malden, Mass: Blackwell Publishing; 2005:45–47.

89. Tesh S. *Hidden Arguments: Political Ideology and Disease Prevention Policy*. New Brunswick, NJ: Rutgers University Press; 1988.

90. Lock M, Gordon D, eds. *Biomedicine Examined*. Dordrecht, The Netherlands: Kluwer Academic Publishers; 1988.

91. Fee E, Krieger N. Understanding AIDS: historical interpretations and the limits of biomedical individualism. *Am J Public Health*. 1993;83:1477–1486.

92. Fairchild AL, Oppenheimer GM. Public health nihilism vs pragmatism: history, politics, and the control of tuberculosis. *Am J Public Health*. 1998;88: 1105–1117.

93. Szreter S. *Health and Wealth: Studies in History and Policy*. Rochester, NY: University of Rochester Press; 2005.



94. Krieger N. Sticky webs, hungry spiders, buzzing flies, and fractal metaphors: on the misleading juxtaposition of “risk factor” vs “social” epidemiology. *J Epidemiol Community Health*. 1999;53:678–680.
95. US Department of Health and Human Services. *A Healthier You*. Washington, DC: US Government Printing Office; 2005.
96. Bush GW. HealthierUS: The President’s Health and Fitness Initiative. Washington, DC: The White House; 2004. Available at: <http://www.whitehouse.gov/infocus/fitness>. Accessed January 22, 2007.
97. Wilkinson R. *The Impact of Inequality: How to Make Sick Societies Healthier*. New York, NY: The New Press; 2005.
98. Raphael D. Barriers to addressing the societal determinants of health: public health units and poverty in Ontario, Canada. *Health Promot Int*. 2003;18:397–405.
99. Phelan JC, Link BG. Controlling disease and creating disparities: a fundamental cause perspective. *J Gerontol B Psychol Sci Soc Sci*. 2005;60(special issue):27–33.
100. Krieger N, Davey Smith G. “Bodies count,” and body counts: social epidemiology and embodying inequality. *Epidemiol Rev*. 2004;26:92–103.
101. Weiss G, Haber HF, eds. *Perspectives on Embodiment: The Intersections of Nature and Culture*. New York, NY: Routledge; 1999.
102. Schepher-Hughes N, Lock MM. The mindful body: a prolegomenon to future work in medical anthropology. *Med Anthro Q*. 1987;1:6–41.
103. Eldredge N, Grene M. *Interactions: The Biological Context of Social Systems*. New York, NY: Columbia University Press; 1992.
104. The Freud Museum. Frequently asked questions. Available at: <http://www.freud.org.uk/finfaq.htm>. Accessed January 12, 2007.
105. Kluger R. *Ashes to Ashes: America’s Hundred-Year Cigarette War, the Public Health, and the Unabashed Triumph of Philip Morris*. New York, NY: Alfred A. Knopf; 1996.
106. Lock S, Reynolds A, Tansey EM, eds. *Ashes to Ashes: The History of Smoking and Health: Symposium and Witness Seminar Organized by the Wellcome Institute for the History of Medicine and the History of the Twentieth Century Medicine Group on 26–27 April 1995*. Amsterdam, The Netherlands: Rodopi; 1998.
107. Brandt AM. *The Cigarette Century: The Rise, Fall, and Deadly Persistence of the Product That Defined America*. New York, NY: Basic Books; 2007.
108. Mackay J, Eriksen E, Shafey O. *The Tobacco Atlas*. Atlanta, Ga: American Cancer Society; 2006.
109. Washington HA. Burning love: big tobacco takes aim at LGBT youths. *Am J Public Health*. 2002;92:1086–1095.
110. Swedish National Institute of Public Health. *Sweden’s New Public Health Policy: National Public Health Objectives for Sweden*. Rev ed. 2003. Available at: <http://www.fhi.se>. Accessed April 13, 2006.
111. American Legacy Foundation. Programs: overview. Available at: <http://www.americanlegacy.org/18.htm>. Accessed January 21, 2007.
112. Corporations and Health Watch [Web site]. Available at: <http://www.corporationsandhealth.org>. Accessed January 15, 2007.
113. Markowitz G, Rosner D. *Deceit and Denial: The Deadly Politics of Industrial Pollution*. Berkeley, Calif: University of California Press; 2002.
114. Richardson JW. *The Cost of Being Poor: Poverty, Lead Poisoning, and Policy Implementation*. Westport, Conn: Praeger; 2005.
115. McMichael AJ. *Human Frontiers, Environments, and Disease: Past Patterns, Uncertain Futures*. Cambridge, England: Cambridge University Press; 2001.
116. Mooney C. *The Republican War on Science*. New York, NY: Basic Books; 2005.
117. *Human Development Report 2006: Beyond Scarcity: Power, Poverty, and the Global Water Crisis*. New York, NY: United Nations Development Programme; 2006.
118. Whiteford L, Whiteford S, eds. *Globalization, Water, & Health: Resource Management in Times of Scarcity*. Santa Fe, NM: School of American Research Press; and Oxford, England: James Currey; 2005.
119. Nestle M. *Food Politics: How the Food Industry Influences Nutrition and Health*. Berkeley, Calif: University of California Press; 2002.
120. Schlosser E. *Fast Food Nation: The Dark Side of the All-American Meal*. Boston, Mass: Houghton Mifflin; 2001.
121. Krug EG, Dahlberg LL, Mercy JA, Zwi AB, Lozano R, eds. *World Report on Violence and Health*. Geneva, Switzerland: World Health Organization; 2002.
122. Hemenway D. *Private Guns Public Health*. Ann Arbor, Mich: University of Michigan Press; 2004.
123. Sokoloff NJ, Pratt C, eds. *Domestic Violence at the Margins: Readings on Race, Class, Gender, and Culture*. New Brunswick, NJ: Rutgers University Press; 2005.
124. Blank RM. Selecting among anti-poverty policies: can an economist be both critical and caring? *Rev Social Economy*. 2003;61:447–469.
125. Townsend P, Gordon D, eds. *World Poverty: New Policies to Defeat an Old Enemy*. Bristol, England: The Policy Press; 2002.
126. *Human Development Report 2005: International Cooperation at the Crossroads – Aid, Trade and Security in an Unequal World*. New York, NY: United Nations Development Programme; 2005.
127. Katz MB. *In the Shadow of the Poorhouse: A Social History of Welfare in America*. Rev ed. New York, NY: Basic Books; 1996.
128. Sachs J. *The End of Poverty: Economic Possibilities for Our Time*. New York, NY: Penguin Press; 2005.
129. Stiglitz JE. *Making Globalization Work*. New York, NY: WW Norton & Co; 2006.
130. Monbiot G. *Manifesto for a New World Order*. New York, NY: WW Norton; 2004.
131. Blake W. *Auguries of Innocence*. Available at: <http://rpo.library.utoronto.ca/poem/161.html>. Accessed May 31, 2007.
132. Bill & Melinda Gates Foundation. Global health program. Available at: <http://www.gatesfoundation.org/Global-Health>. Accessed January 17, 2007.
133. Okie S. Global health—the Gates-Buffett effect. *New Engl J Med*. 2006;355:1084–1088.
134. Garrett L. The challenge of global health. *Foreign Affairs*. Jan/Feb 2007. Available at: <http://www.foreignaffairs.org/20070101faessay86103-p90/laurie-garrett/the-challenge-of-global-health.html>. Accessed January 22, 2007.
135. [No authors listed.] Governance questions at the Gates Foundation [editorial]. *Lancet*. 2007;369:163.
136. Birn AE. Gates’s grandest challenge: transcending technology as public health ideology. *Lancet*. 2005;366:514–519.
137. Pillar C, Sanders E, Dixon R. Dark cloud over good works of Gates Foundation [Part I]. *Los Angeles Times*, January 7, 2007. Available at: <http://www.latimes.com/news/nationworld/nation/la-na-gatesx07jan07,0,6827615.story>. Accessed January 17, 2007.
138. Pillar C. Money clashes with mission: The Gates Foundation invests heavily in sub-prime lenders and other businesses that undercut its good works [Part II]. *Los Angeles Times*, January 8, 2007. Available at: <http://www.latimes.com/news/nationworld/nation/la-na-gates8jan08,1,6069951.full.story?coll=la-headlines-nation>. Accessed January 17, 2007.
139. Pillar C. Gates Foundation to re-assess investments. *Los Angeles Times*, January 11, 2007. Available at: <http://www.latimes.com/business/la-fi-gates11jan11,1,7943560.story>. Accessed January 17, 2007.
140. Pillar C. Gates Foundation to keep its investment approach. *Los Angeles Times*, January 14, 2007. Available at: <http://www.latimes.com/business/la-na-gates14jan14,1,1844117.story>. Accessed January 17, 2007.
141. Stonesifer P. A foundation states its case [letter]. *Los Angeles Times*, January 14, 2007. Available at: <http://www.latimes.com/news/printedition/opinion/la-le-sunday14.3jan14,1,1775487.story>. Accessed: January 17, 2007.