Framing disease: An underappreciated mechanism for the social patterning of health

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Available online 1 April 2008

Abstract

The emerging fields of social epidemiology and population health seek to understand the social determinants of health. Especially, with regards to how income inequality causes health disparities, attention has been focused on material and psychosocial mechanisms. I use examples from the epidemiological and social science literature to argue for a third broad etiological framework: the role played by the ways we generally recognize, define, name, and categorize disease states and attribute them to a cause or set of causes. These framing effects shape population health by influencing: health and illness beliefs; patterns of consumption and other behaviors; perceptions of what interventions and policies work; class, ethnic, and other social dynamics; and clinical and public health practices. Important characteristics of many framing phenomena are their capacity to be self-perpetuating and their performative power. A better understanding of framing effects can lead to deploying them more deliberatively and flexibly to improve individual and population health.

Keywords: Social epidemiology; Population health; Social determinants of health; History of medicine; Social construction of disease

Introduction

Clinicians, researchers, and policy makers have rediscovered the social determinants of health and health inequalities in the United States and other modern, industrialized countries. In parallel, new disciplines have emerged — most prominently, social epidemiology and population health — whose core definition is not being risk factor epidemiology and rejecting the emphasis on individual-centered public health practices. These reconfigured fields aim to find new means of improving societal health outside the provision of health care services, for example by influencing social and economic policies, mass communication, and the built environment.

One contentious issue in establishing this emerging paradigm is the mechanism by which social factors lead to health and disease in the bodies of individuals. Frequently, the shorthand for this issue is “how does culture get under the skin?” Most everyone recognizes that individual behavior, environmental exposures, and differential access to health care make a difference, but what these new fields want to understand and influence

* This paper benefited from the thoughtful comments of David Asch, Charles Bosk, Charles Rosenberg, Jason Schnittker, Steve Feierman, and the Robert Wood Johnson Foundation (RWJF) Health and Society Scholars at Penn. This work was supported in part by a RWJF Investigator Award in Health Policy.

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doi:10.1016/j.socscimed.2008.02.017
are **contextual** factors “above” the level of the individual. Two broad causal conceptions have emerged, especially salient in debates over the health consequences of income inequality. One emphasizes material mechanisms over the lifecourse — such as the availability and quality of schools, housing, and health care (Lynch, Davey Smith, Kaplan, & House, 2000). The competing conception emphasizes the important role of psychosocial mechanisms, such as perception of one’s position on the socio-economic ladder, in shaping societal health (Wilkinson, 1996). Psychosocial proponents point to research on stress and heart disease and the role played by relative not simply absolute income on health outcomes.

I want to make the case for a third broad etiological framework for understanding the social patterning of health and disease. These are ways that the health of individuals and populations results from how societies generally recognize, define, name, and categorize disease states and attribute them to a cause or set of causes. These **framing** ideas and practices can have profound effects by influencing individual and group behavior, clinical and public health practices, and societal responses to health problems. Many of these causal pathways are frequently uncovered in standard epidemiological studies but are categorized as biases or confounders rather than as causes or mediating influences. This dismissal makes sense if we assume, as most health researchers do, that social and historical variation in health beliefs, diagnostic practices, and what problems are medicalized are mere epiphenomena in relation to “real” health status. But I will argue below that these framing phenomena are sometimes the very mechanisms by which the social patterning of health and illness emerges.

I use **framing** somewhat euphemistically for what is often referred to as the social construction of disease (Rosenberg, 1992). This usage may avoid a few unwanted connotations sometimes associated with constructionist arguments — a style of dated cultural relativism, a lack of common sense, and a reflexive opposition to biomedicine. In recent socio-historical scholarship, not only have there been terminological shifts but also a substantive one from case studies of obvious borderline diagnostic categories (e.g. homosexuality as a disease in psychiatric nosology) to more complex and systematic research into normative medical categories and classifications (cancer, heart disease, etc.). Although there is no inconsistency between attributing a causal role to framing phenomena and also to materialist and psychosocial ones, they are rarely combined and often sort by discipline (e.g. historians focus on framing phenomena while social epidemiologists focus on materialist and psychosocial ones). Yet, knowledge and insights stored in these different silos might profitably borrow from one another, resulting in more complete accounts of the social patterning of health.

Below I survey a range of framing mechanisms and consider the plausibility of understanding them as etiological in the social patterning of health. Some of the examples are drawn from the epidemiological literature. Investigators typically explored associations hitherto understood in material or psychosocial terms but upon further and more subtle analysis proved to better explained as reporting and selection biases, misclassifications, misapprehension of changed diagnostic criterion and patterns for true disease change, or misleading representations of efficacy.

Other examples come from the social sciences. Consideration of framing phenomena in disease has a long history within medical history, sociology, and anthropology, albeit under different labels, each with their own (often contested) scope and connotations — social construction, medicalization, labeling, and others. It is much less widely recognized, especially in the emerging field of population health and the policy attention given to health disparities, that framing phenomena (1) in aggregate represent a mechanism causing or mediating some of the social and temporal patterns of health and disease in the contemporary U.S. and (2) that much of the evidence for “framing as mechanism” is right before our eyes in the medical and epidemiological literature and yet is not recognized as such, in large measure because from the perspective of everyday medical and public health rationality, these phenomena are obstacles and diversions from understanding “true” causal mechanisms.

As a final introductory note, I want to point out that while I evoke “how culture gets under the skin” to situate my argument about framing as mechanism, I do so only because it is the most common shorthand that epidemiologists and clinicians use to map social conditions to biological phenomena. Epidemiologists and clinicians use this shorthand because they cannot precisely or even imprecisely define the interface they intuitively want to capture — what exactly is inside and outside the body or culture, or what culture is and is not. In other words, I am using an existing and problematic term of reference. Yet, by arguing for the inclusion of framing phenomena I want to challenge medicine’s and epidemiology’s everyday if poorly articulated assumptions about the location and meaning of this culture/body interface. Again, this is by no
means a unique challenge but I hope to make a contribu-
tion to the burgeoning population health field by
using findings from within the medical and epidemi-
ological literature and the interpretive social sciences,
which are non-trivial in their scope and implic-
ations, and that are not confined to borderland health
conditions.

While I will highlight some beliefs, linguistic and
classificatory norms, and social dynamics not usually
understood in etiological terms, they are not down
payments on a new conceptual model. The expanding
literature on the social determinants of health does
not need another “arrow salad” in which everything
causes everything else.1 I want to focus on a few iden-
tifiable pathways because the very complexity of
multi-causal and multi-level models can and has been
used as a pretext to give up population-based health
interventions as critics argue that it is too difficult to
imagine sensible and effective points of leverage
(Farmer, 1999; Tesh, 1988).

Social and structural framing of diagnoses

It has been widely observed in the United States that
asthma’s incidence, mortality, and hospitalization rates
have been rising and that these trends are dramatically
worse for the urban poor and ethnic minorities. Bio-
medical and social scientists have generally looked to
micro-environmental (e.g. dust mites and cockroaches)
and psycho-biological (stress formulations with im-
une mechanisms) explanations. Yet, there are clues
in the existing clinical and epidemiological literature
that the distribution of asthma diagnoses also reflects
historically conditioned values, interests, and social
structures, many of which might be profitably studied
and understood with the methods of a cultural anthro-
pologist or medical historian. For example, Cunning-
ham, Dockery, and Speizer (1996) have demonstrated
some discordance between the prevalence of wheezing
and rates of asthma diagnoses in an emergency room
setting. These authors cautiously suggested that race
and socio-economic status may be more important to
the acquisition of a diagnosis of asthma than to the prev-
ance of symptoms themselves. In an accompany-
ing editorial, Peter Gergen (1996) interpreted these results
to possibly mean that inadequate primary care (and
emergency physicians’ worries about poor follow-
up) led to more asthma diagnoses among poor chil-
dren who had wheezing episodes. If this type of

1 I thank Bruce Link for this term.

framing effect was widespread, a large amount of
the observed temporal, income, ethnic, and geo-
graphic variation in asthma might be due to differ-
ences in diagnostic and labeling routines (in
addition to the putative role played by more material
aspects of poverty).

In this case, “culture may have gotten under the
skin” by the way interacting socio-economic realities
(lack of insurance, inadequate primary care) and
beliefs about disease (e.g. that people with poor fol-
low-up do better with the asthma diagnosis and imme-
diate treatment rather than watchful waiting) create
social patterns of health and disease that can at some
point also become self-sustaining. The self-sustaining
aspect occurs when the perceived social patterning of
disease itself becomes a part of the diagnostic act, in
this case the fact that asthma is perceived to be more
prevalent among the poor itself favors the diagnosis
of asthma among the poor. Medical diagnosis is a nec-
essarily Bayesian exercise. The “prior probability” of
disease shapes the diagnosis. Such beliefs also influ-
ence individuals’ health-seeking behavior.

An extreme form of this kind of self-sustaining pat-
ttern exists when the social shaping of disease classifi-
cation has resulted in a small set of stable, highly
culturally framed diagnostic options. Individual diag-
nostic acts are constrained by this nosology, while at
the same time their cumulative effect can reinforce
the stability of the nosological system. Consider the
highly gendered framing of headache diagnoses in
the United States today. As a result of a complicated
if characteristic social history for gendered diagnoses
(see Kempner, 2006), many American neurologists
came to believe that most headache syndromes could
be classified as either a primarily female “migraine”
or a primarily male “cluster headache.” Aside from
the contribution played by any putative biologically
based sex differences in the etiology of different head-
ache patterns, once these gendered diagnoses were
made available and legitimated, individual diagnostic
acts would be constrained by these clinical and epide-
miological “facts.”

Technological change affects frame

In a widely cited 1985 article, epidemiologist Alvan
Feinstein et al. coined the term the Will Rogers phe-
nomenon to explain an apparent paradox in cancer
statistics (Feinstein, Sosin, & Wells, 1985). Feinstein
recalled that humorist Will Rogers had once quipped
that when the Okies migrated from Oklahoma to Cali-
foriia during the Depression, the average IQs of both
states went up. This could only happen if the migrating Okies were on average less intelligent than the average Oklahoman but more intelligent than the average Californian. Feinstein observed that a similar phenomenon might explain the apparent progress in stage-specific, cancer survival rates in previous decades. As diagnostic technology improved (e.g., the use of CT scans to diagnose previously unapparent metastasises), there was a systematic reclassification downward of the “worst prognosis” cancers to less favorable stages. Analogous to the assumptions underlying Will Rogers’ joke, this reclassification had the effect of improving the average survival of both stages, because these “downshifted” cases were on average the worst of the original stage but the best of their new stage.

The Will Rogers phenomenon (and related framing effects discussed below such as apparent, improved case fatality rates that have resulted from screening, changed thresholds for pathological diagnosis, and new pre-cancer diagnoses) has undoubtedly contributed to the impression that there has been widespread medical progress in cancer survival. Most people within and outside of medicine reflexively attribute such progress to technological improvements in the way cancer has been diagnosed and treated. This belief has then been used to justify further investments in cancer diagnostic and therapeutic technology and practices, leading to further apparent progress and so on. These types of interactions among technological change, clinical practices, and perceptions of progress are widespread and constitute important mechanisms by which social and temporal patterns of disease get established. Like other framing mechanisms considered here, an important characteristic of these interactions is their capacity to be self-perpetuating. Another key insight is the way that some beliefs and perceptions about causality and efficacy have performative power. They can be actors in, not simply reflections of, the health of individuals and populations. While Feinstein et al. understood the Will Rogers phenomenon as a bias and illusion to be factored out of better and more accurate epidemiological observations, it nevertheless may have had a “real” effect on individual and collective ideas about what works and where future investments should be made.

Risk, fear, and demand for control

In some other interactions, a cycle of perceived risk, fear, and demand for control has created a self-sustaining process similar in dynamics to the Will Rogers phenomenon, but perhaps even more difficult to recognize, understand, and modify. The history of breast cancer risk (Aronowitz, 2001, 2007) has shed some light on how this worked earlier in the century. The American Society for the Control of Cancer (later the American Cancer Society) began in the early 20th century a public education campaign to get women to “not delay” seeking medical attention for different deadly cancer signs. This campaign was based on assumptions about time and cancer, especially that early, “complete” cancer surgery was effective in treating cancer. Its efficacy was hardly doubted by the cancer establishment and the general public. The campaign lasted over 50 years and was not so much disproved or discarded as eclipsed by more technologically based screening programs such as PAP smears and screening mammograms.

Similar to other feedback cycles between aggregate perception, beliefs, and routines discussed above, the “do not delay” campaign was sustained by a cycle of fear, selling of control, behavior change, and the perception of efficacy. To summarize briefly the campaign’s dynamics vis a vis breast cancer, women were implored to come to medical attention if they noticed any suspicious changes in their breasts. This educational message was designed and delivered in ways that appealed to and increased fears of cancer. At the same time in which this campaign took hold, pathologists and clinicians were defining new and earlier types of pre-cancers and were also lowering their threshold for diagnosing cancer. As more women sought medical attention as a result of the campaign, these changes in diagnostic practices interacted with the greater pool of women with breast problems seeking medical attention to result in an ever increasing number of breast cancer diagnoses. Since mortality from breast cancer was largely unchanged during the period of the “do not delay” campaign, these increasing numbers of diagnoses meant that the ratio of deaths to diagnosed cases was rapidly decreasing. From an epidemiological perspective, the case fatality rate associated with breast cancer declined and survival rates increased.

These favorable aggregate statistics were widely touted and perceived. They contributed to lay and medical belief in the efficacy of early detection and treatment. This perception motivated even greater compliance with the “do not delay” campaign — resulting in a cascade effect that lasted until medical skepticism about the unchanged mortality became more vocal and new means of prevention (screening mammography) were promoted. In the meantime, breast cancer’s impact on American society had been
transformed without necessarily changing the deadliness of the disease.

The situation today with breast and other site-specific cancer prevention practices echoes this earlier history of cascade-like interactions among mass interventions, perceived changes in aggregate impact and the efficacy of interventions, fear, and the resulting high demand for control. To take one recent example (Schwartz, Woloshin, Fowler, & Welch, 2004), 38% of a sample of 500 U.S. adults reported they had experienced at least one false positive result on one of three cancer screening tests (mammography, Pap smear, or PSA; 35% of the female subjects had experienced a false positive screening mammogram). Some of those subjects with false positives waited over a month before finding out they did not have cancer. Many subjects reported that this was “very scary” or the “scariest time” of their lives. But it does not seem that the experience of false positives in any way derailed subjects’ enthusiasm for screening. Instead, some 98% of the subjects who experienced false positive results reported that they were very satisfied that they had been screened. The experience of a cancer diagnosis and its subsequent removal appeared to only strengthen already positive attitudes about cancer screening, providing a partial explanation for the autonomous and self-sustaining quality of some contemporary disease prevention interventions (Aronowitz, 2007).

**Economic and structural contributions to rising number of newly defined health risks and how efficacy is framed**

In cancer screening and many other contemporary health risk interventions, we have created a momentum from which it has been difficult to pull back, change direction, or even question. Large scale public health or marketing campaigns have changed the routines of not only ordinary men and women but of general practitioners, radiologists, pathologists, and other important players in the modern “risk factor” rapid reaction force. These interactions among changing epidemiological perceptions, beliefs, and behavior have been sustained by a massive and rapid diffusion of screening technologies, with their embedded suppliers, managers, and interpreters. But these technological and manpower investments are only one part of a larger system in which economic interests have shaped new conceptions of health and disease.

The New York Times recently gave prominent coverage to a health risk development that illustrates the economic contribution to the cascade-like interactions described above. The Times reported that a new pharmaceutical company-sponsored study showed that people at high risk of developing diabetes (study details were unspecified, but some 54 million Americans were estimated to have “prediabetes”) had reduced their risk by two-thirds by taking the drug rosiglitazone for three years (Rosenthal, 2006). The article discussed the enthusiasm of some observers as well as the marketing plans of the pharmaceutical companies. But it also raised questions about costs (noting that the analogous “statin” class of drugs to reduce risk of heart disease was now a $40 billion industry, “consuming more than 10% of health spending in some European countries”) and efficacy in terms other than individual risk of diabetes (a diagnosis that has features of both a symptomatic chronic disease and a state of being at risk for future disease). When framed in arguably more clinical and population-relevant terms, the same study showed that 544 people would have to be treated for three years to save one life. While it evidently matters whether efficacy is framed in terms of individual relative risk or in terms of population impact, there has been little explicit societal debate or negotiation among the affected parties to how disease and health should be framed. Instead, there is a kind of free market in which champions of particular risks and interventions, pharmaceutical companies, and other actors work to change behavior and routines and expand the kinds of variation labeled as health risks (see Becker, 1963 for a full discussion of the role played by such “moral entrepreneurs”).

The rosiglitazone diabetes prevention trial is part of a much larger shift in the framing of health, catalyzed and promoted by the economic interests of pharmaceutical companies, but also having its roots in other aspects of Western culture. Shifts in body metaphors, clinical practice, and perceptions of efficacy have resulted in and interacted with profound changes in lay demand for risk interventions and what individuals consume and do with their bodies (Aronowitz, 2006).

Greene (2006) tells this emergence of risk story from the perspective of the pharmaceutical industry and with special emphasis on the industry’s skillful use of new means of establishing efficacy (especially the randomized clinical trial). Greene begins his narrative at a 1957 meeting of the American Drug Manufacturer’s Association in which an industry representative reflected on the paradoxical market impact of the industry’s most obvious success — the production and marketing of antibiotics. The net result of this success — effectively curing infectious disease — was to limit the industry’s market. No normal industry would be based
on a business model in which the industry’s products completely consumed the demand for its products. The representative warned of the dire consequences for the industry if they did not develop a new paradigm for drug development and use. In 1957, this representative could only imagine the outlines of another paradigm in which drugs would grow rather than shrink their market. In subsequent decades, the industry would develop and promote risk-reducing drugs and a new probabilistic concept of efficacy as risk-reduction. Unlike antibiotics, the market for risk-reducing drugs was the whole population and the duration of use could potentially span an individual’s entire life.

**Dynamics of consumption — feedback loops between consumers and producers**

A related and relatively unstudied framing mechanism which has contributed to the emergence and social patterning of many conditions, is the interaction between patterns of consumption and disorders of consumption. Let me illustrate with an example from a recent case study (see also the historical production of the medical consumer by Nancy Tomes, 2001, the rise and fall of the cigarette (Brandt, 2007)).

Schull (2005) carried out a largely ethnographic study of relationship between the producers and consumers of video poker games. She showed a complex interaction among the makers and users of game technologies that has led to subtle, often individualized manipulation of design elements that accommodates and creates complex and diverse consumer needs. As a result of these interactions, consumers spend more money on gambling and more consumers have, or are understood to have, a gaming addiction. While addiction to almost anything can be framed as a health problem, gaming addiction may have negative effects on one’s resources and personal relations and excessive machine use may have direct, negative effects on the body itself, especially one’s mental health. Framed as a health problem, video poker addiction can then elicit a variety of health care responses, from 12 step programs to interventions by health professionals. These responses can themselves be understood as a form of medical consumption, as they have their own economy shaped by the perceived needs of the addicted as well as the actions of the promoters of different programs, health care professionals, and third party payers.

What I want to call attention to here is the interaction between the culturally specific intelligent design and marketing of consumer goods and the coincident framing of addiction and excessive consumption as a disease, which may itself elicit consumer-oriented health restoring responses. The intense research and design efforts of the producers of such technologies to create an experience appealing to the individual consumer’s needs and vulnerabilities is arguably as or more manipulative than the design and marketing strategies of addictive substances now widely condemned (like manipulating nicotine content in cigarettes) while at the same time not necessarily involving an easily isolatable unethical practice. The net effect is something akin to addiction without a specific biological agent to blame or vulnerable target (children, biologically predisposed) to avoid.

I imagine the perplexed early 21st-century Martian arriving at her motel room in the United States after her long trip and turning on the television. How would she make sense of the marketing of calorie dense, super-sized, highly processed food stuffs along the direct-to-consumer medical advertisements pitching heart disease and obesity preventive medications, as well as commercials for weight loss programs, gym memberships, and exercise machines?

This pattern repeats itself in other types of consumption and societal response, and leads to a more expansive and complicated conception of diseases of affluence or civilization. It is not adequate to explain these diseases solely by using evolutionary models of “cave men in the fast lane” or evoking changed material conditions of modern life (Rosenberg, 1998). The ill health associated with affluence does not always result from environmental dangers like radon or collateral damage from economic and social advances. Some of this particular social patterning also results from an increasingly effective and individualized product development and marketing capacity that creates new members for an ever enlarging class of disorders framed as addictions, excessive consumption, and victims of unfair or deceptive marketing. The social patterning arises from the interaction between the changing mechanisms of consumption and new ways diseases and health problems are legitimated and defined.

**Framing health disparities: consequences of or ways to maintain social inequalities?**

Researchers and policy makers have not generally questioned why there has been a recent upsurge in interest in health disparities in the United States. Health disparities are not new phenomena and have been noted before. One reason for the prominence of some health disparities might also be a clue to why
some patterns exist over and above their production by inequalities in the material conditions that produce health and disease and/or the resources available to prevent and treat disease. Could it be that some fraction of observed health disparities are direct consequences of social and cultural dynamics rather than distal consequences of inequalities in the material conditions effecting health? Let me explain what I mean using the example of racial and socio-economic disparities in obesity via an analogy to socially patterned linguistic variation.

Linguist William Labov years ago noted that a historical cycling of “R dropping” in different American dialects. First a feature of upper class speech, it was gradually adopted by the lower classes. Because “R dropping” served as a marker of class distinction, upper class speakers gradually stopped “R dropping” once this pattern became the norm for lower social classes. This is an example of a much needed and socially constructed difference or disparity. Labov also conducted some ingenious experiments in New York City department stores that showed that “R dropping” was patterned by the immediate social context — it was much more likely to happen in conversations between customers and salespersons in less expensive department stores and in bargain basements than in floors that sold more expensive goods in the same store (Labov, 1963).

A similar dynamic to such sociolinguistic variation may lie behind the formation and persistence of some health disparities. In both the social patterning of language and health, difference or disparity may function to signal and maintain class and other social distinctions. Some health disparities and gradients may not result from other social inequalities in any direct sense but serve rather as markers of class, wealth, and/or race difference. These disparities in other words are purposeful and functional in themselves even if there is no single person or groups of people consciously plotting things out.

Some amount of the social patterning of moderate obesity may be an instance of this kind of framing effect. Many observers have called attention to the social patterning of obesity, that it is increasingly a disease of the poor and ethnic minorities. Attention has been focused on cultural norms in dieting, the built environment of inner cities, and the marketing of obesogenic foods to African Americans (e.g. McDonald’s branding as an African American foodstuff) — much of which is ultimately caused by discrimination and prejudice. Obesity has been linked to other poor health outcomes, especially type II diabetes and CHD.

But perhaps some of the racial and socio-economic disparity evident in obesity rates is functional, not only a direct consequence of inequalities in access to healthy foods and other determinants (that clearly are operative). By functional, I mean to call attention to social dynamics, especially important to explain how and why enthusiasm for the current medicalization of obesity has taken root and the class-tinged decisions made by individuals. Like R dropping, higher average weight historically (in a time of more expensive calories) was more prevalent among the better off. It also took on some symbolic value as a marker of socio-economic progress and advancement. But as cheap calories became more widely available the poor also got fatter. What was once a marker of high socio-economic standing reversed its meaning and the social dynamic of differentiation meant that many who had the resources to reduce their weight or evade obesity did so. Supporting this dynamic has been an economy of non-consumption as a commodity — low fat foods, gyms, etc. — in which there has been relatively more participation by affluent people.

In other words, as obesity became a marker for lower prestige and status, people with greater resources had more ability and motivation to avoid the stigma of obesity. The enthusiasm with which moderate obesity has been framed as a risk factor, disease, and public health threat has helped the better off to put additional distance between them and those less fortunate. Thus one can even view the medicalization of moderate obesity in part as a framing mechanism for signaling and maintaining social difference. In our secular society that enshrines health as a transcendent value, there is perhaps no better way to stigmatize a behavioral or other difference than to associate it with a bad health outcome. These are macro-level determinants that do not have to be consciously uttered and negotiated (see Bourdieu, 1984 for a comprehensive sociological analysis of “cultural consumption” and its social functions and role in legitimating social differences). Rather, like R dropping, there is something pre-conscious about these innovations. But one would miss the dynamic that maintains and fuels the “disparity” if we thought of weight gain by different social groups as simply the result of material conditions in which groups lived or cultural difference. Interactions among how ill health is framed, social dynamics, and differential access to resources might also be contributing to the appearance and persistence of disparities in obesity and other health problems (such as smoking) and which suggest seemingly counter-intuitive policy responses like lessening the degree of medicalization and stigmatization of moderate obesity.
Implications

The “framing” mechanisms outlined above may explain only a small fraction of the social patterning of health and disease. They also may act in concert with more direct, material mechanisms. For example, the incidence of breast cancer in the United States and other industrializing countries probably grew considerably in the 19th and early 20th century. The most likely explanations are socio-material ones, that is, a series of demographic and other social factors that together led to greater life time menstrual cycles and estrogen exposure. Better nutrition contributed to lower age of menarche and later age of menopause; greater reproductive freedom, economic advancement, and other changes led to dramatically decreased fertility and later age at first childbirth. Yet, the rising incidence rates later in the 20th century may well have been catalyzed and maintained more by framing mechanisms rather than changes in the material conditions of life: public education and screening campaigns, changes in the definitions of cancer, changed diagnostic thresholds, and increased cancer fears (Aronowitz, 2007).

Lyme disease’s appearance and distribution in the United States in the 1970s probably resulted from a similar complement of materialist, environmental, and framing effects (Aronowitz, 1991). What has often been understood as the discovery of a new disease was on closer inspection more of a re-framing of older knowledge under new and unique conditions. But changed social and environmental conditions permitted the framing of Lyme disease as a new condition. Researchers have speculated on the way that the shift of land use from farms to suburbs led to increased numbers of the intermediate hosts for the ticks that carry the Lyme disease spirochete. New patterns of disease incidence thus arose via material mechanisms, i.e. by the ecological effects of new types of communities. These patterns then influenced the way medical knowledge was constructed, in this case the framing of Lyme disease as new. The fear and attention given to a new disease influenced the pattern of diagnosis (many new diagnoses and the creation of controversial chronic Lyme disease patienthood) and medical response.

Can something useful be made of these parallel and interacting processes? We might reflect more critically on the social and policy attention given to some health disparities. I have suggested that some disparities in highly visible intermediate health outcomes such as moderate obesity might have their own dynamic, independent of and in addition to the material determinants of incidence. In other words, the very disparity is itself important, maintained by different kinds of choices, investments, and attitudes. Understanding disparity in this way can help us better prioritize our limited clinical and public health resources and avoid using medical categories to further stigmatize already disadvantaged groups and individuals.

An example of a “positive” role for emphasizing framing as mechanism is discernable in the social patterning of hypertension in the United States today. Unlike the situation in many other health conditions and despite the evident inequalities in access to — and quality of — health care, African Americans with hypertension are more likely to be correctly diagnosed and treated than European Americans (there remain, nevertheless, significant and disturbing disparities in the underlying incidence of hypertension, due in part to differences in diet and activity and their multiple determinants, as well as in hypertension control; Hertz, Unger, Cornell, & Saunders, 2005). One possible explanation for the “positive” pattern is that earlier public health educational campaigns aimed at the African American community and physicians worked and became self-perpetuating as both medical and lay beliefs about who was at risk changed. The relative lack of stigma attached to hypertension and its invisibility also meant that there would be less chance of a social dynamic emerging similar to the one previously described in maintaining ethnic and class differences in the prevalence of obesity. One implication of this limited reversal of the typical pattern of health inequalities is to avoid unnecessary stigmatization in framing a risk factor or disease as well as to provide wide access to a basic level of health care. At the same time, further progress in reducing disparities in underlying hypertension incidence and control will likely depend on progress in the material conditions and health care of disadvantaged groups.

Conceptualizing some framing phenomena as etiological also suggests new approaches to contentious ecological and population-level associations. A recent prospective observational study of the widely observed although contested association between psychosocial stress and heart disease concluded that the apparent positive association was largely due to a reporting bias: self-reported stress was associated with likelihood of angina (which as a symptom complex, is also a self-report) but not with more objective cardiovascular endpoints (Macleod et al., 2002). Such studies, commonly understood as simply debunking a spurious association, can at the same time be the beginning rather than the end of important population-level etiological study. Are there social, economic,
linguistic, symbolic, or normative influences on men and women’s experience of chest pain and health-seeking behavior that might be contributing to secular trends, social disparities, and clinical presentations in angina? Can we influence health by changing our diagnostic definitions and practice, health beliefs, and aggregate perceptions of disease?

Focusing on both framing effects along with material and psychosocial causes might also provide some help in what might be called the crisis in the epidemiology of everyday life. We are bombarded with often contradictory relative risks of all kinds of environmental exposures, behaviors, dietary regimens, and lifestyles. The roots of this problem have been traced to the inadequacies of epidemiological methods to resolve these questions (Taubes, 1995). But we also need to more systematically ask and understand why some associations but not others are being investigated and labeled systematically as risk factors (Taubes, 1995). But we also need to more systematically ask and understand why some associations but not others are being investigated and labeled systematically as risk factors.

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