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Behavioral science at the crossroads in public health: Extending horizons, envisioning the future

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Abstract

The social and behavioral sciences are at a crossroads in public health. In this paper, we attempt to describe a path toward the further integration of the natural and behavioral sciences with respect to the study of behavior and health. Three innovations are proposed. First, we extend and modify the "stream of causation" metaphor along two axes: time, and levels of nested systems of social and biological organization. Second, we address the question of whether 'upstream' features of social context are causes of disease, fundamental or otherwise. Finally, we propose the concept of a *risk regulator* to advance the study of behavior and health in populations. To illustrate the potential of these innovations, we develop a multilevel framework for the study of health behaviors and obesity in social and biological context.

Keywords: Behavior; Causality; Epidemiology-history; Epidemiology-theory; Social class; Social factors

Introduction

> ENOUGH! A century of misunderstanding, the drawn-out Verdun and Somme of Western intellectual history, has run its exhausting course, and the culture wars are an old game turned stale. It is time to call a truce and forge an alliance. Within the broad middle ground between the strong versions of the Standard Social Science Model {*caps in the original*} and genetic determinism, the social sciences are intrinsically compatible with the natural sciences. The two great branches of learning will benefit to the extent that their modes of causal explanation are made consistent.
>
> E. O. Wilson (1998, p. 188)

The social and behavioral sciences are at a crossroads in public health. Decades of behavioral research has culminated in a series of large-scale intervention trials yielding unsatisfactory results (Susser, 1995). Flagship studies like the (Multiple Risk Factor Intervention Trial (MRFIT), 1982; Stallones, 1983), Community Intervention Trial for Smoking Cessation (COMMIT) (Anonymous, 1995) and, more recently, the Enhancing Recovery in Coronary Heart Disease trial (ENRICHD) (Berkman et al., 2003), have yet to demonstrate the expected efficacy of behavioral interventions to modify health outcomes (Glass, 2000; Relman & Angell, 2002). While it is generally accepted that modest changes in health behavior can be achieved with carefully designed, and theoretically informed interventions, the extent to which behavior change is lasting, or translates into health improvements at a population-level is considerably less clear

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(Glasgow, Klesges, Dzewaltowski, Bull, & Estabrooks, 2004). A consistent criticism of these intervention studies is that they largely ignore the social context that shapes behaviors (McKinlay & Marceau, 2000). A second criticism is that the physiological consequences of behavior change are seldom considered and may be more complex than has been assumed (Sheps, Freedland, Golden, & McMahon, 2003). In sum, these studies illustrate the limits of well-intentioned interventions that treat individual health behaviors as separate from social context and from biological influences.

In the context of etiological studies, important behavioral risk factors for many diseases, especially coronary heart disease, have been identified and well-characterized. Behaviors such as cigarette smoking, inactivity, and a high-fat diet, explain a substantial amount of the world's experience with atherosclerosis (Beaglehole & Magnus, 2002; Magnus & Beaglehole, 2001). Long-running studies, including MRFIT, the Framingham Heart Study, and the Chicago Heart Association Detection project, have shown that exposure to these traditional risk factors is common to virtually all individuals who develop coronary artery disease (Greenland, Gidding, & Tracy, 2002); although up to 85% of men and women thusly exposed do not develop clinical atherosclerosis. However, despite considerable progress, two problems remain. First, socioeconomic gradients in nearly all health outcomes persist after adjusting for this long list of individual risk-factors (Davey Smith, Shipley, & Rose, 1990; Harding, 2003; Koskinen, 2003; Mackenbach, 1994; Margellos, Silva, & Whitman, 2004; Marmot, Rose, Shipley, & Hamilton, 1978; Marmot, Shipley, & Rose, 1984; van Rossum, Shipley, van de Mheen, Grobbee, & Marmot, 2000; Woodward, Shewry, Smith, & Tunstall Pedoe, 1990). Secondly, the processes that give rise to the social patterning of risks remain poorly described and understood. A great deal is known about the behaviors that lead to disease, but much less is known about how those behaviors arise, become maintained, and more importantly, can be changed. The two problems are obviously connected. Needed is a vigorous and sustained effort to better understand what differentially places people *at risk for risks* (borrowing language from Link and Phelan, 1995). We need better theory, and better data, to understand how social factors regulate behaviors, or distribute individuals into risk groups, and how those social factors come to be embodied. In this paper, we offer ideas to advance the study of social determinants of disease, which point toward potentially more effective population interventions. First, we expand, modify, and "unpack" the stream of causation metaphor. Second, we address issues related to causal reasoning in light of vexing problems created by the study of complex social phenomenon. Finally, we propose a new type of variable, the *risk regulator*, as a potential solution to several of these vexing problems.

Behavior is contingent

Behavioral science within public health, especially in the US, has focused primarily on individual health-related behaviors (or "life styles"), without due consideration of the social context in which health behaviors occur and become socially patterned (For critiques of life style, see Blaxter, 1990; Coreil, Levin, & Jaco, 1985). Obvious examples include exercise, diet, and tobacco and condom use. From Wade Hampton Frost (Maxcy, 1941) to Geoffrey Rose (1985, 1992), public health scholars have pointed out the weaknesses of this approach, arguing for increased emphasis on population determinants, rather than characteristics of individuals. In the last decade, a flurry of commentaries in US public health journals has appeared. In this Journal, Krieger challenged us to locate the spider responsible for the "web of causation" in risk factor epidemiology (Krieger, 1994). Link and Phelan (1995) argued for increased emphasis on what they termed fundamental (social) causes of disease. McKinlay (1995) called for a "new public health" that treats behavior within a broader social context. Susser and Susser (1996) question the traditional "black box" whereby disease arises mysteriously from unexamined social forces currently beyond the epidemiologic gaze. McMichael (1999) wondered whether we have become "prisoners of the proximate". Ben-Shlomo and Kuh (2002) and Davey Smith (2003) have advanced "life-course epidemiology" to better understand social gradients in health. Beaglehole and Bonita (2004) noted the isolation of epidemiology from theories and methods from social sciences. In short, consensus has grown for the need to stretch the boundaries of the study of behavior and health in order to capture the role of social structure to a greater degree.

Most of these critiques have in common a commitment to an ecological approach (Susser & Susser, 1996) that posits features of the social and

built environment *above* and *before* the individual (at the familial, community, organizational, and societal levels), constrain, limit, reward, and induce the behavior of individuals. For example, social class is a product of labor relations at higher levels of social organization. Black males in the presence of white power structures are exposed to discrimination (at lower levels of organization). The health behaviors of black males are, in turn, shaped, constrained, and induced from above in a complex relational web.[1] This point is by no means new (Macintyre, 1994; Mackenbach, 1998; McMichael, 1999; Susser, 1998; Syme, 1987); however, much of public health continues to treat behaviors such as diet, smoking, violence, drug use, and sex work as if they were voluntary decisions, without regard to social constraints, inducements, or pressures. There are important examples of progress toward contextualizing behaviors (for example research on the role of advertising on smoking initiation), but for the most part, constraining factors on health behavior have been limited to individual psychological factors (such as depression, coping styles, self-efficacy, or "readiness to change"). Variation in rates of smoking, drug use, or violence across time, space, and between social groups are often ignored, in part because explanation of population-level phenomenon requires different theories, data and methods. The question is: how can behavioral and public health scientists move further beyond this emphasis on individual decision making, toward a more ecological perspective? We address this issue next.

Revising a metaphor: extending the stream in three directions

Leading theorists in social epidemiology have employed the image of a running stream to describe the chain of causal influences flowing from distal social factors to proximate, individual factors further downstream (Anonymous, 1994; Kaplan, 1995). In this paper, we extend and unpack the metaphor in order to advance the study of behavior and disease in a way that simultaneously accounts for social context and biology, as well as their interactions across the life-course. Our revision (Fig. 1) is based on two primary axes: time and a nested hierarchy of systems from genes, to cells and organs, to social networks and groups, to the global environment. Time is represented by the flow of water across an irregular surface (horizontal axis), while biological and social organization is represented by a vertical axis reflecting nested biological and social hierarchies. The vertical axis begins in bedrock (genes), and rises through biological systems lying beneath the surface (underwater), progressing to the plane of the waterline where individual behavior occurs. Above the water's surface is a landscape of ever larger structures that make up the social, built, and natural environments. Together, these elements form a metaphorical landscape of hierarchically arranged spheres of influence.[2] We suggest that the complex interrelationships among these nested levels (and their multiple feedback loops through time) motivate a powerful approach to thinking about social and biological influences of behavior and disease.

A pictorial representation of a three-dimensional space poses graphical challenges. The metaphor acquires depth upon consideration of populations distributed spatially. Individuals are like buoyant objects floating in a network of tributaries, streams and rivers, each beginning a journey at different points and affected by differing topographical features. When envisioned as a three-dimensional space, rather than a plane, the potential richness of the image can be more fully appreciated. Illness states can be represented by pockets in the river bed into which a person might descend. The watershed might contain bumps, hills, or mountains that parallel barriers to adoption of health promoting behaviors. Areas of depression (valleys or canyons) represent opportunities or inducements that attract the flow of water. Depending on differential distribution of resources, some objects floating in the stream may be more able to scale obstacles against the force of gravity, while others may be destined to take the shortest, easiest course. In short, the proposed metaphor offers a rich vocabulary of images for depicting the multi-layered processes that generate population patterns of health.

What we have proposed is best understood at the meta-theoretical level. That is, we are describing a general approach to theory construction that addresses issues that cut across a broad range of what Robert Merton might have called "theories of

[1] We thank an anonymous reviewer for the suggestion of this particular example.

[2] A third dimension, representing spatial variation, is implied by the landscape perspective, but not elucidated here.

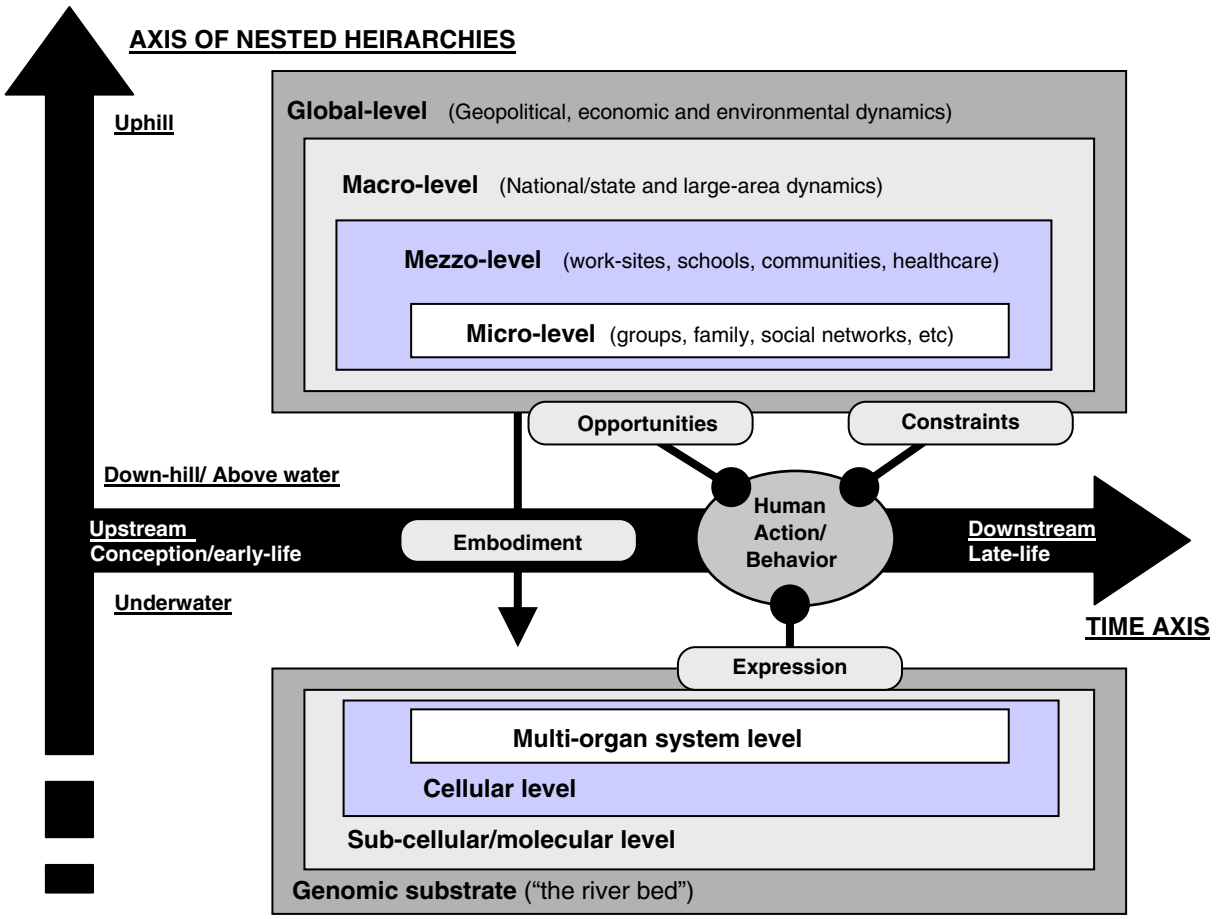

Fig. 1. The society-behavior-biology nexus as depicted in multidimensional space. The large arrows represent the axes of time and nested hierarchical structures. The sphere of health-related behavior and action moves through time from infancy to old age. Behavior is influenced by structured contingencies within the social and physical environment and by biological phenomena. Structural contingencies (opportunities and constraints) are shown by paths ending with nodes, while biological phenomena (embodiment and expression) are shown by paths ending with arrows or nodes.

the middle range.'' We are influenced by the philosopher of science Pepper (1942), who characterized four distinct *world hypotheses,* which he defined as grand-scale conceptual systems: formism, mechanism, contextualism, and organicism. Pepper argued that each world hypothesis is girded by a 'root metaphor' that functions as a cognitive map guiding scientific discourse. By proposing an alternative root metaphor (the 2-axis stream of causation) we hope to influence the way smaller scale theories are built and tested. A three-dimensional root-metaphor, like what is proposed here, requires a shift away from simple linear causal thinking that naturally follows from a two-dimensional (upstream-downstream) root metaphor, toward a more complex, more contingent way of thinking. At a higher level of abstraction then, our topographical metaphor also serves to encourage the transition from a mechanistic world metaphor, toward contextualism. In that sense, we are proposing a framework for generating theories and organizing research, rather than a specific theory of how a particular set of explanatory variables can be systematically organized to account for the observed data gathered about a specific outcome of interest. To illustrate how this might be accomplished, an example will be presented later.

Ecosocial landscapes: gaining elevation/extending upward

One of the limitations of the stream metaphor as used to date, is that the horizontal dimension has typically been used to define two distinct axes: time

and levels organization in social systems. Traditionally, upstream factors are conditions that are temporally and sociospatially distal to disease outcomes. We suggest parsing these two axes. In Fig. 1, the horizontal axis depicts temporal influences over time, which could be conceptualized at the individual level as the life-course from birth to death, or at the population level in terms of historical changes. Social and environmental forces external to individuals are treated along the vertical axis, as rising above the waterline. Gaining elevation implies that social factors induce and constrain health-related behaviors (and various other proximate risk factors) across the entire life-course, just as a landscape influences the course of a river. This implies an analogue to gravity; the pull of gravity is analogous to the trajectory of human development and aging.

Social structure can be represented as physical obstacles (rocks, boulders, valleys, canyons, and hills) that shape and constrain the flow of water (representing here health-related behaviors). By tracing the topographical features of the social context, additional explanatory power can be brought to bear in understanding patterns of behavior. This does not imply that topographical obstacles are "natural" features of social context, but rather that human actions and policies manifest themselves as patterns of obstacle placement—the boulders and channels in our metaphorical river. Like the Army Corps of Engineers, policy makers can have dramatic impact on how (and where) rivers flow by altering the placement of obstacles. At the same time, the placement of obstacles in the landscape reflects socially constructed power relations, not the hand of mother nature. This three-dimensional landscape also creates opportunities for explaining the movement of multiple streams within a watershed. Landscape features closer to the river are lower on the organizational hierarchy (e.g., families, social networks, etc.) compared with higher-level features further uphill (e.g., labor markets, systems of inequity, governmental policies). Uphill forces are distal both in terms of time, but also in the extent to which they have greater leverage in altering the flow of water.

Society, behavior, and biology over the life-course: extending horizontally

The impact of social context plays out over time and across space throughout the human life-course. This idea is illustrated by recent work in life-course epidemiology (for a recent summary of this literature, see Davey Smith, 2003). Developmental and life-course perspectives in public health are relatively new (Ben-Shlomo & Kuh, 2002; Brunner, 2000; Lynch et al., 1994; Pearlin & Skaff, 1996). At least four important implications of a life-course perspective have emerged. First, early life exposures (both social and physical) explains trajectories of health in adulthood, decades later (Graham, 2002; Hertzman, Power, Matthews, & Manor, 2001; Power & Hertzman, 1997; Van de Mheen, Stronks, & Mackenbach, 1998). Barker and colleagues have stimulated much of this work with their findings on the fetal origins of cardiovascular disease (Barker, 1991a–c). Secondly, the impact of those exposures varies according to its timing. This suggests windows of heightened sensitivity to the effects of certain exposures. For example, studies of work and health show that employment status and working conditions exert their strongest influence during mid-adulthood (Marmot, Shipley, Brunner, & Hemingway, 2001). Thirdly, the effect of exposure to social conditions appears to be cumulative. Gerontologists have demonstrated increasing heterogeneity over time in health and functional status as a result of differential exposure to social conditions throughout the lifespan (Dannefer, 1988; House et al., 1994; Maddox, 1987). A dose-response association has been consistently observed between the number and duration of episodes of social and economic disadvantage and increased disease risk (Hallqvist, Lynch, Bartley, Lang, & Blane, 2004; Lynch, Kaplan, & Shema, 1997; Ross & Wu, 1996; Singh-Manoux, Ferrie, Chandola, & Marmot, 2004). Late life appears to be a period of increasing vulnerability to the cumulative influence of disadvantage across the life course (House et al., 1994; Lantz et al., 2001). Forth, human development at the population level is influenced by how historically specific events differentially effect the experiences and trajectories of entire birth cohorts. So called 'cohort effects' result in complex patterns as a result of the overlap of early life exposure, differential windows of vulnerability, and cumulative exposure. Moreover, in addition to extending the horizons of our thinking upwards (towards the effect of social context), the proposed model seeks to stretch horizontally, across time, to better understand the cumulative, time-sensitive, historically specific, and duration-dependent effects of social context of human behavior and development.

Biological embodiment below the surface: extending underwater

A central problem in understanding health behavior is explaining how human bodies respond to, and are altered by social conditions over time, and how behaviors are impacted by these biological adaptations. Addiction is an obvious, if somewhat specialized example. As if to anticipate our extended metaphor, Geronimus has coined the term "weathering" to describe the cumulative changes that occur in bodily systems as a function of repeated exposure to social adversity (Geronimus, 1992, 1996, 2001; Wildsmith, 2002). Krieger and Davey Smith (2004) use the term embodiment, explaining that:

> ...biologic beings and species are constituted through their engaged interaction with biotic and abiotic environments they in part construct and embody, in the context of dynamic ecologic systems (p 94).

Embodiment is an important integrating concept in our model. It describes the sculpting of internal biological systems that occurs as a result of prolonged exposure to particular environments. It is how features of social and built environments become internalized, or get "under the skin" (Taylor, Repetti, & Seeman, 1997). A long tradition of research has added to our understanding of how external material and social conditions become embodied below that water's surface. Examples include studies of unemployment (Bartley, 1994), acute stress (Benschop et al., 1998; Goldberg et al., 1996), working conditions (Brunner, 1996), social isolation (Cacioppo et al., 2002), social connectedness (Cohen, Kaplan, & Manuck, 1994; Uchino, Cacioppo, & Kiecolt-Glaser, 1996), and chronic stress (Manuck et al., 1987). This work has been strongest in the area of cardiovascular disease where recent studies of allostatic load (McEwen, 2000, 2001; Seeman, McEwen, Rowe, & Singer, 2001) have documented the embodiment of social conditions.

Recent advances in genetics has led to concerns about whether interest in the social determinants of population health will be eclipsed by the current enthusiasm for genetic explanations of behavior and health (Allen, 2001). These concerns may be exaggerated to the extent that the potential synergies between genetics and social sciences have yet to be fully appreciated. Understanding the behavioral determinants of population health is not likely to be improved solely by studying main effects of genes on behavior. Very few complex human behaviors are probably caused by single genes. This is undoubtedly a consequence of the unique developmental trajectory of the human genome toward what Deacon (2000) calls combinatorial complexity; innovation, flexibility and variation in human behavior results from the decline of fixed genetic determination in highly "domesticated" species (like humans).

The proposed model emphasizes feedback loops and cross-level influences between physiology (including genetics) and social context. Complex human behavior is the outcome of dynamic exchange among factors above and below the water's surface (to return to our metaphor). Research that explores the interaction between genes and socio-environmental exposures will play an especially powerful role in understanding mechanisms. This work is just beginning (examples include Albeck et al., 1997; Caspi et al., 2003; Epel et al., 2004; Lesch et al., 1996). These examples provide a preview of an emerging multilevel perspective whose characteristics can now begin to be discerned.

In summary, we propose a modification and extension of the stream of causation metaphor that specifies two axes of influence (time and levels of social and biological organization) and important integrating phenomena (embodiment and structured contingencies, which are discussed below). The purpose of this expanded metaphor is to draw attention to increasing areas of convergence in the study of health behaviors across multiple disciplines in public health and science, which suggests consilience (Gould, 2003; Wilson, 1998) with respect to causal explanations of behavior and health. The model posits a multidimensional landscape of causal influences that are hypothesized to govern health behavior.

At the core of these ideas is a conceptualization of human behavior as sandwiched inextricably between ecology and biology. Diet, smoking, exercise, alcohol consumption, sexual activity, help-seeking, and seat belt use are not free, unconstrained choices. If they were, individualistic, health education interventions would no doubt have proven to be more effective than they have. Health behaviors occur in patterns because they are shaped by social factors residing at levels of organization above the individual, in conjunction with the consequences of

biological systems within the body. The important scientific challenges of the future will involve understanding the synergies and dynamics that occur between these nested levels of organization (Institute of Medicine of the National Academy of Sciences, 2001; Shonkoff & Phillips, 2000).

This does not imply that behavior is the only route through which upstream/uphill factors effect health. As Marmot's work (among others) demonstrates (Marmot et al., 1978; Marmot et al., 1984; Marmot et al., 1991), accounting for health behaviors does not fully explain the association between social conditions and health. However, it is well-recognized that virtually all the major adverse health states that make up the global disease burden, are to some extent rooted in complex human behavior. Thus, health behavior is our topic in this paper, and we believe, a more contextual understanding of smoking, diet, physical activities and health care utilization (among others) would advance the effectiveness of public health policies and interventions.

The focus on health behaviors in social context is motivated by Anthony Gidden's theories of structuration across time and space. Giddens defines structuration as the active bi-directional process whereby knowledgeable social actors pursue goals within the constraints and opportunities of local environments that are historically and spatially rooted (Giddens, 1979, 1981, 1987, 1993). The aggregated results of these individual actions reproduce (and often alter) the structural arrangements observed at higher levels of organization. While a full explication of Gidden's ideas is beyond the scope of this paper, his emphasis on human agency is a useful tool to overcome the vexing micro–macro problem (for reviews, see Alexander, Giesen, Münch, & Smelser, 1987; Byrne, 1998). Giddens implies a shift of focus from *health behavior* (often conceptualized as robotic response to external stimuli), to a focus on *health action*, as the reciprocal process from which social structure emerges. An emphasis on action places the knowledgeable, strategic, and intention-driven social actor on center stage. It also implies, in our view, the need to prioritize those aspects of social structure that impinge on the consciousness, knowledge base (and decision making) of social actors; a position that accounts in part for our emphasis on behavior and the mid-level social phenomenon that shape those behaviors in the course of purposeful social action.

The fundamental causes debate

In epidemiology and public health, experimental study designs (chiefly the randomized clinical trial) are the gold-standards for evaluating causal hypotheses. Randomized trials allow us to estimate the average causal effect of a treatment by comparing two groups that, because of random assignment, are similar on average measured *and* unmeasured variables. Differences between these two groups reflect the magnitude of the "causal" association due to treatment. This is because the "control" group estimates the counterfactual condition (what the outcome would have been in treated subject, if those same subjects had not been treated). However, experimental manipulation of social factors like income inequity, racial discrimination, or neighborhood social disorganization, is not politically, ethically, or practically feasible in most cases. The study of social factors is therefore often restricted to observational data or "quasi-experiments". Fundamental thinkers in causal inference, including Holland (1986), make clear that causal models can be tested using observational data, provided that the causal associations hypothesized are not "causally meaningless". Holland does not object to hypothesizing causal factors for social phenomenon that are hypothetically subject to experimental manipulation. However, he rules out "attributes" as possible causes:

> An attribute cannot be a cause in an experiment, because the notion of potential exposability does not apply to it. The only way for an attribute to change its value is for the unit to change in some way and no longer be the same unit. Statements of "causation" that involve attributes as "causes" are always statements of association between the values of an attribute and a response variable across the units in a population. (1986, p 955)

Based on this argument, individual attributes like age, social class, race/ethnicity, and gender are not causally meaningful. Kaufman and Cooper (1999) agree, but argue that personal attributes serve as proxies for complex extra-individual social processes that are (in theory) subject to exposability restrictions, and which could be experimentally altered. While it may not be meaningful to talk about the causal role of being Black, the causal effect of racial discrimination as a social process, with specific practices and history, can be imagined.

In response to this problem, social scientists (House et al., 1990; House et al., 1994; Lieberson, 1985) and public health investigators (Link & Phelan, 1995) have proposed that social factors such as inequity, poverty and racism are "fundamental" or "basic" causes of disease. These arguments are not new (e.g., Stallones, 1980; Susser, 1973) and remind us of classic epidemiologic studies of the social production of diseases such as syphilis (Kark, 2003), cholera (Byrne, 1998, Chapter 6; Frost, 1936; Smith, 2002 for a discussion of the classic work of John Snow), pellagra (Morris, 1975), and tuberculosis (Bradbury, 1933). However, the concept of "fundamental causes" has been met with energetic debate in epidemiology (Krieger, 1994; See Rothman, 1988; Savitz, 1994; Susser, 1998; Vandenbroucke, 1988). Rothman, among others, argues that the real fundamental causes are biological and more proximate to illness, and that the association between social class and disease is a spurious by-product of the correlation between social class and those "real" causes (Rothman, 1986, p. 90). The field has been traumatized by clashes between micro-biologic and the macro-social interpretations of what is fundamental (see for example Shy, 1997; or Vandenbroucke, 1988). These debates have been unhelpful in our view. Competing claims about what is fundamental are ultimately reductionist (in both cases) and block, rather than facilitate new science. Despite the tendency to distill complexity down to false dichotomies (e.g., micro vs. macro, nature vs. nurture, people vs. places), accounting for health behavior requires a more complex idea of causation that includes dynamics occurring *across* rather than *within* levels. Next-generation models of health behavior will focus on how the social environment affects (in the probabilistic, not deterministic sense) the organism, which, in turn, affects intra-organismic levels (the organ, the cell, the sub-cellular and the molecular), and how each of these levels feeds back in return to levels above (See Anderson 1998). As has been well articulated before (Macintyre, Maciver, & Sooman, 1993), people create places and places also create people (Bronfenbrenner, 1979). The emerging subfield of neighborhood, or area-based studies, is one illustration of how multilevel thinking can bear methodological and substantive fruit (Diez Roux, 2001; Duncan, Jones, & Moon, 1998; Earls & Carlson, 2001; Elliott et al., 1996; Mayer & Jencks, 1989; O'Campo, 2003; Ross & Mirowsky, 2001; Sampson, Morenoff, & Gannon Rowley, 2002; Sampson, Raudenbush, & Earls, 1997; Taylor, 1997). This literature also illustrates the need for the continued refinement of emerging conceptual and methodological tools for thinking across (rather than within) levels. What, then, are the implications of this for the issue of causal reasoning and the problem of "meaningless" causal associations noted by Holland?

Causality, confounding and counterfactuals

The goal of observational and intervention studies in public health is to identify causal effects, and to exploit that knowledge in the service of improving population health. This requires a clear set of rules for determining true (as opposed to spurious or confounded) causal effects. While several models of disease causation have risen and fallen in the history of public health research (see for example Greenland & Brumback, 2002), causal reasoning has generally been based, in whole or in part, on a variant of the counterfactual model (or potential outcome model) (Little & Rubin, 2000; Maldonado & Greenland, 2002). According to the counterfactual model of causality, the estimability of causal effects is only feasible and practical when two possible worlds can be imagined: one world that is like ours, and another in which all conditions are identical with the exception of the isolated causal factor under investigation (Greenland & Morgenstern, 2001). The counterfactual model poses potentially insurmountable barriers to identification of uphill (distal) social factors as causes of disease (Kaufman & Cooper, 1999; Kaufman & Kaufman, 2001; Oakes, 2004). In the absence of an experiment, simulating two possible worlds that are identical except for some "treatment", it becomes difficult (or impossible) to identify the causal effect of variables like racial discrimination, poverty, or income inequity. It is impossible to imagine a world that is identical to our world, except that there is no (or less) poverty, or that all those in poverty were suddenly lifted into a higher socioeconomic position. That world, were it to exist, would be quite different in a myriad of ways as a result of this change. Therefore, the causal "effect" of uphill social conditions (or any other causal effect for that matter) are not identifiable in observational studies (Robins & Greenland, 1992) and will remain so because it is either unethical or impossible, to design experimental studies of many social factors.

The question of whether social conditions can meet the demanding epistemological criteria for causation, operationalized in the counterfactual model, has been met with spirited debate (Rothman, 1986; Rothman, 1988). On one side, traditional views of causation emphasize the Bradford Hill (1965) criteria,[3] whereby a "risk factor" must have strong, specific (if and only if), and unmediated influences to be classified as a cause of disease (Stehbens, 1985). On the other side, social epidemiologists argue that social conditions can meet causal criteria with minor adjustments (Link, Northridge, Phelan, & Ganz, 1998; Link & Phelan, 1995; Morris, 1975). A shouting match between factions fighting the "epidemiology wars" has been the result (Poole & Rothman, 1998).

Our argument is a practical one. Instead of arguing over what factors qualify as causes of disease, the focus should be on identifying potentially powerful levers[4] of behavior change at the population level, regardless of whether those factors are causal in the traditional sense. Thus, we are prepared to concede that social conditions are not disease *causes*. However, public health might be advanced more rapidly and more efficiently, if the focus were on social conditions of life that *regulate* behavioral risk, but do not strictly qualify as causal because their independent influences are weak, nonspecific, subject to temporal variation, and contingent on a series of probabilistic, intermediate processes. Following Rose (1992), Rose and Day (1990) and Stallones (1980), it is more efficient to identify population-level factors that influence rates of disease, and target those for intervention, even if they fail the test of causality at the individual level. So, for example, the cause of death for the victim of a gunshot is the physical damage done by a fast-moving metallic projectile. Had the victim not been shot (counterfactual), she would not have bled to death. However, this statement is independent of whether public health interventions that target the social conditions that make guns plentiful and violence commonplace, may be more effective in reducing firearm fatalities than issuing Kevlar vests to every person.

Having conceded ground in the battle over the definition of disease causes, a partial retraction is required. The purpose of this paper is to nudge the field toward a more complex view of causality than has persisted during the 'biomedical' regime. The problem posed by social conditions is but one of many challenges that point to the limitations of a Newtonian view of causation. Interestingly, medicine and epidemiology are among the last domains of science that have yet to move beyond the 19th century canon of simple deterministic, linear causal effects. Meteorology, physical chemistry, particle physics, systems ecology, and even economics, have adopted more complex understanding of causality. Epidemiology is being dragged, kicking and screaming, toward complexity and contingent causation. This does not imply a negation of the idea of scientific adjudication, or the possibility of future prediction (as the post-modernists would have it), but rather an acceptance of the inherent complexity of human behavior within a hypercomplex social world. Social conditions, to use the language of systems theory, are control parameters that affect the probability of behaviors that are "causes" of obesity, lung disease, HIV infection, and violent death. That is not to demote social factors to a lower priority, but to argue that their influence is second-order, and can only be appreciated (and exploited) in the context of a 21st century concept of disease causation.

Given this, we are left with the problem of how to study variables that are clearly related to health, that exist uphill and upstream, but are not "causes". What should researchers call social conditions, such as racial discrimination, material deprivation, and cultural norms, if they qualify neither as independent risk factors (causes) or confounders? If social class, gender, and race/ethnicity are markers for exposure to uphill/upstream social and structural processes, how do we classify them for study, in order to better appreciate, measure, and exploit their considerable influence on health? Their influence varies over time and place, and their effects are non-specific; they operate probabilistically through a complex chain of intermediate steps that can involve factors at multiple levels or organization. Next, we propose an alternate framework for understanding these kinds of variables.

[3]These include (1) strength, (2) consistency, (3) specificity, (4) temporality, (5) biological gradient, (6) plausibility, (7) coherence, (8) experimental evidence, and (9) analogy.

[4]A reviewer points out that the allusion to powerful levers of behavioral change implies causation. This is true. However, we mean this in the experimental sense. That is, interventions, such as policy changes, that affect behavior may allow us to demonstrate "causal" associations between large-scale social conditions and population health in future decades. The example of cigarette taxation and smoking rates in New York City is an example. The capacity of observational studies to identify levers of change at the macro level remains controversial.

Risk regulators and structured contingencies

As we have argued, social factors such as racial discrimination, social inequity, poverty, neighborhood deprivation and social capital are difficult to study, in part because variables that measure these social processes do not fit the definition of a risk factor. In the absence of an alternative, most researchers treat these variables as potential confounders, and adjust for them in regression models.[5] Where do features of social context such as poverty or inequity fit? Poverty does not "cause" any single disease in everyone similarly exposed. Yet, the association between poverty and adverse health behaviors that cause disease can scarcely be ignored. The concept of an *independent* risk factor, one that causes disease if and only if it is present, and that does so without being contingent on the commingling of other factors, evolved to allow causal inferences within a single level of analysis. How can we begin to construct a multilevel structure for causal inference that is well-suited to explaining health disparities in complex human systems?

We propose an alternative class of variable, one that shapes health outcomes in populations, but in a more indirect way. For this purpose, we propose the concept of a *risk regulator* as a class for variables that capture aspects of social structure that influence individual action. We define a *risk regulator* as a relatively stable feature of a particular patch of the social and built environments, residing at levels of organization above the individual (uphill), but below larger-scale macro-social levels. They are the phenomena that impose constraints and opportunities that shape, channel, motivate and induce behavioral risk factors that cause disease, and the salutary factors that protect against exposure and delay disease progression. Risk regulators index the *structured contingencies* in the social and built environment as experienced by social actors in discrete action settings—structured because they are specific, stable dimensions that exist external to individuals, and contingent in the sense that contexts within each dimension are varied, and likely to affect patterns of risk depending on personal, community, and historical processes. They are not themselves risks, but are the conditions that regulate or control exposure probabilities to those distal behaviors (and non-behavioral risks) that lead to disease.

Structured contingencies can be described at different levels of organization from families, to local communities, to legal jurisdictions, cultures, societies, and to larger global environments. *Risk regulators* are, therefore, determinants of disease rates, as opposed to *risk factors*, which are measures of the specific proximate causes of cases (Schwartz & Diez-Roux, 2001). They function as control parameters that operate at a system level to up- or down-regulate the likelihood of key risk factors (including health behaviors like smoking, inactivity, high-risk sex, and overeating).

In contrast to a causal risk factor, a risk regulator operates through multiple pathways and through complex (and potentially non-linear) causal sequences over time and place. Consider an extreme example: war. In 1998, a 6-year war broke out in the Democratic Republic of Congo (DRC), resulting in perhaps the most devastating humanitarian crisis since World War II. In four comprehensive mortality surveys in the DRC, the International Rescue Committee found that the conflict has been responsible for approximately 3.8 million deaths in excess of what would have been expected in the absence of war (The International Rescue Committee, 2005). Of the estimated 31,000 excess persons dying in Congo every month, the majority are not "caused" by war, but are associated with non-violent causes due to the collapse of the public health and food distribution systems. In all regions of the country and among all ages, violence was responsible for less than 2% of deaths. The vast majority of deaths have been among civilians, and 80–90% have been due to treatable and preventable conditions (infection, and malnutrition). In other words, this horrendous society-wide conflict is undoubtedly to blame for nearly 4 million deaths, but not because war "causes" death, but because war has fundamentally altered the social conditions of life in ways that create a new and lethal regime of risk.[6]

Risk regulators bear resemblance to the strange attractors of chaos theory (Byrne, 1998), or to Link and Phelan's (1995) fundamental causes. They differ

[5]The same objections can be raised about the "causal" role of genes, given that genetic influences on health rarely meet the Bradford Hill criteria.

[6]An informal reviewer pointed out that this example shows that social conditions can meet epistemological criteria for causation. The counterfactual (had the war not happened) is not difficult to consider. The example here illustrates the non-specificity of social factors.

from fundamental causes, however, in several important ways. First, risk regulators are not causes. They are characteristics of social context that help explain the accumulation and distribution of causes. This is more than a semantic distinction. A weakness of the fundamental cause concept is the difficulty in explaining changes over time in the precise mechanisms and influences of, for example, social inequity. Heart disease, obesity, and lung cancer were associated with higher socioeconomic position in the first half of the 20th century. The direction of that association reversed in the second half as smoking, high-fat diets, and inactivity diffused to lower status groups (Marmot, Kogevinas, & Elston, 1987). This has been used as evidence against the causal role of social class. Secondly, risk regulators are intermediate or mezzo-level variables, midway on the vertical dimension of our causal landscape. They are the bridging tendrils linking larger macro-social processes (such as systems of stratification, labor markets, culture, and systems of production and migration) to the behavioral sphere of human activity and decision making. They reside at levels of organization above the individual, yet below society. In contrast, Link and Phelan define social conditions in surprisingly individualistic terms as "…factors that involve a person's relationships to other people." (p. 81). Risk regulators are measures of external realities that act to promote or constrain behavior in day-to-day reality. Through the risk regulator concept, we can operationalize how social structure reaches down to affect behavior (which, in turn, becomes embodied and has physiologic consequences).

Fig. 2 below is a graphical representation of some key relationships of interest. The figure is not a causal model per se, but a general framework intended to complement Fig. 1 by zooming in on the behavioral sphere. Behavior (or action) is an emergent property of the interplay between a particular set of opportunities and constraints emanating from the environment, and a knowledgeable, goal-seeking actor. The regime of structured opportunities and constraints that exist in a particular patch of space and time, descend from (and are the results of) mediating structures (risk regulators) that lie between larger hierarchically nested systems (depicted in Fig. 1), and the theatre of behavior. Separate from the behavioral realm, these mediating structures act as conduits, through which material exposures and inputs (e.g., air pollution, heavy metals, food, cold temperatures, etc.) challenge the body and must be processed by key regulatory systems. We also show (on the right side) that non-material or symbolic inputs descend from the immediate environment and challenge those same regulatory systems (e.g., threat, support, "peer pressure", advertising, social cues for behavior). The social and environmental conditions that give rise to risk regulators are assumed to be hypercomplex. This complexity is alluded to in the figure but not "unpacked". The same is true for the biological substrate: including cellular, genetic, and molecular processes of equal complexity. We focus on the bridging structures that shape and delimit individual behavior, recognizing that behavior is only one of the ways through which social context impacts health.

Fig. 2 depicts several potentially promising risk regulators including material conditions (Adler et al., 1994; Eachus, Chan, Pearson, Propper, & Davey Smith, 1999; Krieger & Fee, 1994; Macintyre, 1992), discriminatory practices, policies and attitudes (Clark, Anderson, Clark, & Williams, 1999; Krieger & Rowley, 1993; Williams, 1996), neighborhood and community conditions such as housing quality, population density, and fear of crime (Lynch, Smith, Kaplan, & House, 2000; Macintyre, 1994), behavioral norms, rules and expectations (Barabasi & Bonabeau, 2003), conditions of work (Bartley, 1994; Belkic, Landsbergis, Schnall, & Baker, 2004; Bobak, Hertzman, Skodova, & Marmot, 1998; Bobak, Pikhart, Hertzman, Rose, & Marmot, 1998; Bosma et al., 1997; Bosma, Peter, Siegrist, & Marmot, 1998; Hlatky et al., 1995; Karasek et al., 1988; Kuper & Marmot, 2003; Pikhart et al., 2001; Schnall, Landsbergis, & Baker, 1994; Siegrist, 1995; Siegrist, Klein, & Voigt, 1997; Siegrist & Marmot, 2004; Siegrist, Peter, Cremer, & Seidel, 1997), and laws, policies, and regulations (Gostin, 2000; Luepker, 1999; McKinlay & Marceau, 2000; Teret & Wintemute, 1993; Terris, 1980; Wintemute, 1988, 1999). We chose this set of factors because there is an extensive body of literature showing that each is association with health outcomes. Numerous reviews exist that more thoroughly review and explicate the basis for this set of what we are calling risk regulators (see especially Kaplan, Everson, & Lynch, 2000; Lynch et al., 2000; Marmot, 1996; Sampson et al., 2002). An exhaustive listing of potential risk regulators is beyond our scope. What is new is the emphasis on mediating structures (risk regulators), and on health behaviors embedded in social context. What we propose has implications

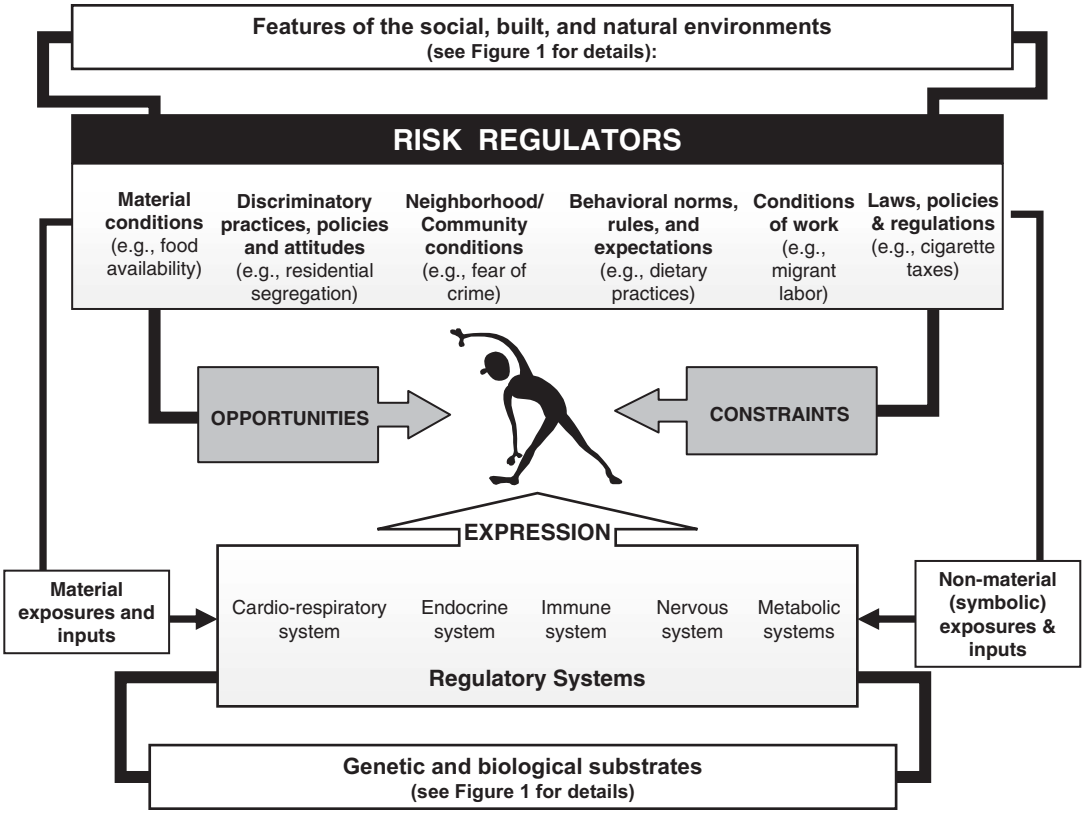

Fig. 2. Detailed view at closer range of the sphere of human behavior/action from Fig. 1. This detail nested within Fig. 1. Risk regulators, as measures of structured contingencies, produce opportunities and constraints on behavior. They also index the set of material exposures, psychosocial experiences, and information that constitute inputs to which the regulatory systems of the body must respond. Thus, risk regulators influence behavior indirectly via structured contingencies (opportunities and constraints) and through effects on biological systems inside the body (through embodiment).

for the way these associations are modeled and understood.

Equally important in the figure (although less well developed here) is the depiction of bridging structures linking the biological substrate to the expression of behavior, and, by extension, larger structures overhead. Of particular interest are the biological systems that process or metabolize both symbolic and material inputs from the external world (including for example sensory stimuli, symbolic messages, and chemical exposures). The systems that regulate heart rate, hunger and feeding, immune response, and sensory inputs are clear examples. These systems negotiate the organism's boundaries with the local physical and social environment. They regulate essential bodily subsystems in response to various inputs, and when necessary, mount countermeasures to rebuff invasion or threat. It is through these biological systems that the limits and characteristics of the human body feed back on behavior, and in turn, on the nature and shape of social structures higher in the systemic hierarchy. Ground-breaking work on the nature of these internal regulatory systems can be found in classical biology including Bernard (1927), whose ideas have recently re-emerged in systems biology (see the excellent discussion by Cziko, 2000). This set of systems (or something similar) perform an equivalent bridging role at another key location on the society—behavior—biology nexus.

Obesity, an example

An example may be useful. In light of surging interest among social epidemiologists, obesity will serve in this capacity. Obesity is a global epidemic with undeniable roots in complex human behavior,

with obvious, but as yet unspecified environmental antecedents. Several attempts have been made to explain the 'obesogenic' environment (Ball, Mishra, & Crawford, 2003; Brownell & Horgen, 2004; Drewnowski, 2004; Egger & Swinburn, 1997; French, Story, & Jeffery, 2001; Hill & Peters, 1998; Liu, Cunningham, Downs, Marrero, & Fineberg, 2002; Stunkard, 1977; Swinburn, Egger, & Raza, 1999), although to date, few of these models have been empirically tested with any degree of rigor.

Obesity is an ideal example for three reasons. First, individually focused behavioral treatments are costly and, to date, of limited effectiveness (Doak, 2002). Secondly, uphill environmental, social and cultural factors appear to play an important role in shaping the more proximate behavioral patterns that give rise to body weight gain. Studies have shown associations between diet and activity and a range of environmental factors that fit into our framework as mediating risk regulators. Examples include community level deprivation (Brunner et al., 1997; Drewnowski, 2003; Drewnowski & Specter, 2004; Robert & Reither, 2004; van Lenthe & Mackenbach, 2002), the spatial distribution of food availability (French, Story, Neumark-Sztainer, Fulkerson, & Hannan, 2001; Morland, Wing, & Diez Roux, 2002; Morland, Wing, Diez Roux, & Poole, 2002), workplace conditions (Rosmond & Bjorntorp, 2000) and food preference and body image norms (Bowman, Gortmaker, Ebbeling, Pereira, & Ludwig, 2004; Dressler, 1983; Green et al., 2003). Thirdly, explaining the obesity epidemic must incorporate the biological substrate. Whatever has changed in the environment that has led to exponential expansion in population body weight, must be conspiring with epigenetic and psychophysiological factors. Eating behavior is an example of a phenomenon that results from synergistic interactions among biological (hunger) and social (eating cues) levels (Cornell, Rodin, & Weingarten, 1989). Human and animal studies showing that environmental stress alters core features of the appetite regulation system, and the metabolic parameters underlying it, offer convincing evidence of this view (Bjorntorp & Rosmond, 2000; Chrousos, 2000; Epel et al., 2004; Spiegel et al., 2004; Tataranni et al., 1996). So, what would a study look like that exemplified our modified stream of causation metaphor, and put risk regulators to work?

The arguments above have implications for study design, theory building, and analytic methods. The data needed for our imaginary study would be longitudinal and multilevel. Individual-level and contextual-level data could be collected before, during and after, critical developmental windows during which behavioral patterns are established and body weight changes occur. This would require data at multiple levels including behaviors at water level (physical activity and diet), uphill factors in the environment (food availability, transportation patterns, psychosocial stressors, behavioral norms and other cultural factors) and the biology at work below the surface (basil metabolic rates, biomarkers of pubertal changes, and corticosteroid release). Integrative, multilevel theories would be required to generate testable hypotheses about cross-level interactions and non-linearites. Careful attention to the specification of variable types (risk regulators, risk factors, confounders, mediators, moderators, and outcomes) would be required. The basic anatomy of such a study would include hypotheses about how risk regulators in the social and built environments exert a cascading influence on body weight through cross-level mediation. Analytic approaches for the study of multiple types of variables can be found in Kraemer, Stice, Kazdin, Offord, and Kupfer (2001). Approaches to modeling multilevel mediation are provided by Raudenbush and Sampson (1999) and by Muthen (1997). What follows reflects this previous work.

We would hypothesize that ground-level social conditions existing in schools, neighborhoods, and homes (such as cultural norms, area deprivation, laws and policies, and the local food environment) act as risk regulators that influence two key health behaviors, feeding and physical activity, dynamically and over the life course. Changes in these behaviors and the relative balance of energy intake (feeding) and output (activity) are the primary causes of change in body weight. They are also the primary mediators through which uphill factors (risk regulators) exert contingent effects on body weight. We would further hypothesize cross-level interactions, whereby risk regulators alter biological factors underwater (HPA axis response, mood, appetite, metabolism, gene expression), which in turn directly effect those health behaviors (Fig. 3) Several important feedback loops are hypothesized, which imply that body weight change (embodiment) alters the influence of biological control parameters.

There are important differences between what we propose in Fig. 3 and the existing literature. Our approach is similar to studies that examine, for

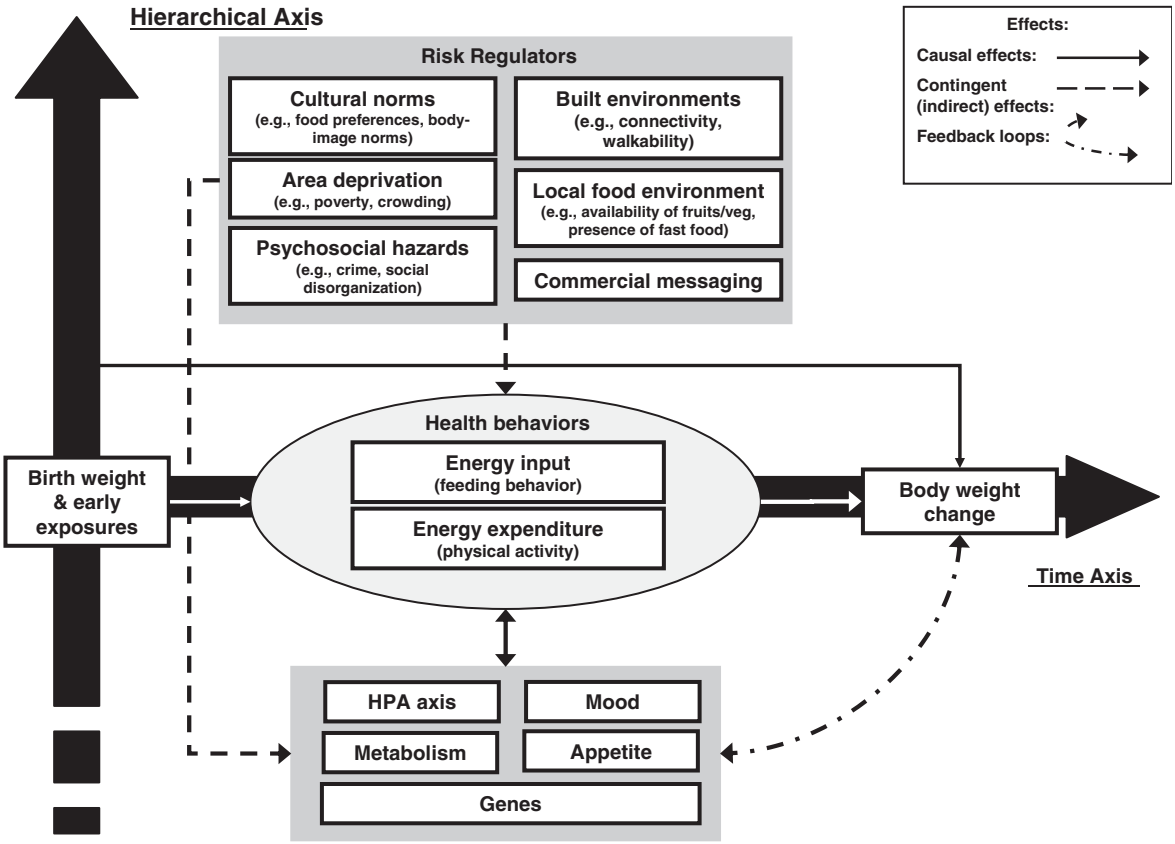

Fig. 3. . The modified stream of causation metaphor applied to the study of obesity. The contingent effects of risk regulators (i.e., embodiment, opportunity, and constraint) are shown with dotted arrows. "Causal" effects of biological and behavioral variables are shown with solid arrows. Feedback loops existing within grouped variables are not shown. Specific effects and multiple, time-ordered feedback loops between variables are not shown in order to reduce diagram complexity.

example, how dietary behavior and physical activity are influenced by broader socioeconomic factors (Jeffery, French, Forster, & Spry, 1991; Reijneveld, 1998; Robert & Reither, 2004). In most cases, however, these investigators model environmental factors while "adjusting" for individual behavioral risk factors in regression models, rather than explicitly modeling more complex causal pathways from risk regulator, to behavior, to body weight (important exceptions include Bobak, Skodova, Pisa, Poledne, & Marmot, 1997; Brunner et al., 1997; Drewnowski & Specter, 2004; Epel et al., 2004). Also different is our emphasis on risk regulators external to individuals. There are many papers that treat individual socioeconomic position as a proxy for differential exposure to risk regulators (Rosmond & Bjorntorp, 2000; Wamala, Wolk, & Orth-Gomer, 1997). Our approach requires that we measure the characteristics of the social and built environments for which individual socioeconomic position is only a poor proxy. Finally, studies that examine the role of biology in modifying behavior-obesity relationships are generally confined to laboratory or animal studies, and do not connect to broader social factors (exceptions include Brunner, 1996; Harburg et al., 1973; Rosmond & Bjorntorp, 2000). The approach outlined here would require careful attention to the role of developmental factors (puberty) and the differential functioning of regulatory systems (appetite, stress response, etc.).

Social factors, including culturally embedded dietary practices, commercial messaging, and economic constraints, exacerbate or dampen the influence of accepted risk factors for obesity. Testing these candidate risk regulators would be analytically challenging. Given the complex, multilevel processes involved, single-equation regression

models would be insufficient. Treating risk regulators as confounders in regression is unreasonable and biases estimates of the role of environmental factors. For example, we might be interested in testing the hypothesis that neighborhood disadvantage impacted obesity risk by altering the probability of proximate behavioral risk factors (eating a high-fat diet, physical inactivity). This cannot be addressed through the standard use of interaction terms; the theory does not suggest that the "effect" of diet is different in those living in neighborhoods high in disadvantage, but rather that poor diets result from conditions of living in those neighborhoods. This problem becomes more complex when considering other individual-level factors such as household income that may limit the types and nutritional values of foods that are available. Given that household income is not independent of neighborhood disadvantage (Sampson & Morenoff, 1997), adjusting for individual socioeconomic status may also lead to biased estimates.

Potential analytic methods for assessing the cross-level effects suggested by this model are available but challenging. Given space constraints, we will limit this discussion to two promising approaches. Harding (2003) has demonstrated the utility of propensity score matching with sensitivity analysis to estimate "causal" effects of neighborhood-level factors. He argues that this method has advantages over other methods by taking account of the selection of persons into different neighborhood exposures. Disadvantages of his method include the need for binary exposure status (living in a good vs. bad neighborhood) and inability to estimate cross-level interaction effects (cross-level main effects could be estimated). A second promising approach is the use of instrumental variables. Economists have used instrumental variables to model the impact of policy changes on the probability of persons receiving a given treatment (Heckman & Hotz, 1989; Heckman & Vytlacil, 1999). In many ways, this can be seen as analogous to our idea that risk regulators alter the probability that deleterious (or salutary) behaviors (as in treatments) will be "chosen" by subjects. These examples highlight how modeling individual trajectories of change in body weight might be approached. However, the ideas here also motivate a shift of focus away from modeling individual trajectories of change, to one that focuses on modeling dynamic systems comprised on interlocking and nested subsystems (for further discussion, see Koopman & Lynch, 1999 or; Prigogine & Stemgers, 1984). Few well-developed examples exist of how to model rates of obesity as system outputs. This will be the subject of a subsequent paper.

Conclusion

Rose (1985) argued that while distal social conditions are more difficult to observe, they are ultimately more important in determining disease rates in populations because they facilitate the expression of individual susceptibility (such as genetic predispositions, personality characteristics, or individual behaviors). The implication is that the control, and manipulation of these structured contingencies (laws, norms, rules, life conditions) may have greater impact on the public's health than the control of proximate causes. Following Rose, we have argued that the study of behavior and health is at a crossroads. The study of health behavior in isolation from the broader social and environmental context is incomplete, and has contributed to disappointing results from experiments in behavior change. The solution requires a shift in emphasis, a reorientation of theories, and new methods. Evidence for these changes is mounting steadily. Several labels have been proposed including the *biosocial model* (Eaton, 2001), *biopsychosocial model* (Anderson & Armstead, 1995; Levi, 1997), *ecosocial* theory (Krieger, 2001), *eco-epidemiology* (Susser, 1998; Susser & Susser, 1996), *epigenesis* theory (Koopman, 1996; Koopman & Weed, 1990), and *multilevel science* (Anderson, 1998; Diez-Roux, 2000). None of these labels has yet been widely accepted. Debate and cross-disciplinary fertilization is likely to continue for some time before any label finally "sticks". We have attempted to contribute to this trend by proposing ideas designed to extend the horizons of behavioral science in public health in three ways: (a) by gaining altitude (to understand causal forces across the topography of social structure), (b) by looking "upstream" at the interactions of environments and biology across the life course, and (c) by looking below the water's surface at how bodies metabolize (embody) social context.

Our model contains some of the elements of similar proposals on which we hope to build, including, for example, Susser (1996), Kaplan et al. (2000) and Lynch et al. (2000). Our approach is different, we believe in several important respects. First, we propose a three-dimensional

root-metaphor that emphasizes a contextual rather than mechanistic orientation. Second, we address (in a new way) conceptual problems in causal inference as it relates to social phenomenon. Third, we focus on health behavior and the mediating structures that lie between the behavioral sphere and the macro-social context. We propose a novel class of variables to represent those mediating structures. We have been careful to insist that behaviors like eating, smoking, seat-belt use, violence, sex work, exercise, medication compliance, and condom use are not the only avenues through which social factors "get under the skin." They may, however, be among the most important. This paper has been an attempt to articulate the broad outlines of what the next-generation approach to the study of behavior and health might look like, without the burden of having to name it.

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