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## Population Health & Genetic vs. Social Causes of Disease: Matters of Relative Priority

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**Abstract:**

This article critiques the effort to disentangle genetic from social causes of disease, but also argues that rough assessments of the relative effect each set of factors makes in shaping patterns of disease and inequities is both possible and is ethically recommended. The essay is divided into two main sections. The first provides the theoretical critique of the genetic-social causal dichotomy as to disease. The second offers the empirical critique of the same dichotomy, and moves on to consider the implications of this empirical evidence for priority-setting in public health policy. Ultimately, because theoretical and empirical considerations suggest that social causes are of much greater significance than genetic causes in causing disease and inequities in populations, even where measures intended to address both sets of causes should be supported, greater resources and attention should be directed to influencing social causes than genetic causes.

**Keywords:** genetics, social, causes, disease, priority, population health, inequities.

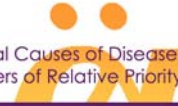
**Resumen:****La salud de la población y las causas genéticas versus causas sociales de la enfermedad: asuntos de prioridad relativa**

Este artículo realiza una crítica al esfuerzo de desenmarañar las causas genéticas de las causas sociales de la enfermedad, pero a su vez sostiene que las evaluaciones aproximadas del efecto relativo que cada conjunto de factores ejerce en la formación de patrones de enfermedad y desigualdades es posible y éticamente recomendado. El ensayo se divide en dos sesiones principales. La primera proporciona la crítica teórica de la dicotomía causal genético-social con respecto a la enfermedad. La segunda ofrece la crítica empírica de la misma dicotomía, y procede a considerar las implicancias de esta evidencia empírica para el establecimiento de prioridades en la política de la salud pública. En última instancia, debido a que las consideraciones teóricas y empíricas sugieren que las causas sociales son mucho más significativas que las causas genéticas en las causas de enfermedad y desigualdades dentro de las poblaciones, inclusive cuando se deberían apoyar medidas cuyo objetivo es tener en cuenta ambas causas, se deberían dirigir recursos y atención que tengan mayor influencia en las causas sociales que en las causas genéticas.

**Palabras clave:** genética, social, causas, enfermedad, prioridad, salud de la población, desigualdades.

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## I. Introduction

In a 2007 article, diabetologist and sociologist Claudia Chaufan (2007a) pointed out that proper attributions of disease causality for phenylketonuria ("PKU") have changed over time. Prior to understanding the effects of dietary manipulation on the genetic defect that produces the disease, PKU was caused 100 percent of the time by the defect itself. After such knowledge became widely known, argues Chaufan, PKU causality is properly attributed 100 percent of the time to the environment, for the simple reason that if diets free of phenylalanine were universally available, no cases of PKU could possibly arise (Chaufan, 2007).

This perspective is, of course, provocative, but the key insight for purposes of the current article is not so much whether Chaufan is correct, but rather the way in which her analysis blurs demarcations between genetic and social causes of disease. Thus the central point is not that PKU causality is in fact 100 percent attributable to social and environmental factors, but rather that attempts to precisely disentangle genetic and social causes of disease is a fool's errand.

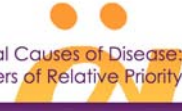
The underlying claim of this article is that genetic and social causes of disease are not meaningfully distinguishable, but that entanglement does not negate the possibility and the significance of assigning relative priority to each set of causes. The argument proceeds in two parts. First, I address the theoretical and empirical evidence that significantly undermines the genetic-social causal dichotomy as to health and illness in human societies. The theoretical level of analysis addresses the very idea of genetic causation of disease in context of what Jeremy Freese has termed the "phenotypic bottleneck" (2008, p. S4), and challenges the general idea that genetic entities in and of themselves cause disease. The empirical level of analysis utilizes the copious and compelling evidence regarding the social determinants of health ("SDOH") and their causal contributions to shaping patterns of disease and inequities as a means of undermining the distinction between genetic and social causes. Second, again using what I will term the "SDOH evidence base," I argue that considerations of relative priority in public health policy justify a greater share of attention and resources be paid to social causes of disease than to genetic causes. This holds true even where it is impossible to neatly distinguish genetic from social causes of disease, and even where both sets of entities exist.

As to the latter point, this article assumes rather than denies that genetic causes of illness exist. This assumption is justified by the significant evidence suggesting that genetic variables exert causal effects as to disease (Freese, 2008, citing studies). The fallacy arises in the oft-accompanying supposition that genetic causal effects meaningfully exist separate and apart from social ones. This is not only an error; it is an error that has significant consequences for understanding and analysis of population health and inequities in human societies.

To begin to understand how and why this might be the case, it is necessary to emphasize the significance of relative priority (Goldberg, 2009). Relative priority means simply that stakeholders should avoid the false choice fallacy in thinking about public health (policy) priorities. In many cases, it is not true that a just social order ought to implement policy or practice A and B instead of C, X, and Z. There frequently are excellent reasons for endorsing all of these policy options, and many others besides. Yet policy is inevitably a contested space, because even where a particular position or platform is uncontested, there is always an opportunity cost paid for expending resources on that position rather than others (Goldberg, 2009; Stone, 2002). The simple fact of scarce social, political, cultural, and economic resources means that stakeholders are forced to consider which of a palette of policy options – all of which should be endorsed – are of higher priority relative to the others, and hence of which policy actions justify greater or lesser attention and resources.

Priority-setting is of particular ethical significance in context of health, in no small part because health policy often involves tragic choices (Powers and Faden, 2006; Ham, 1999). A classic example of this is the prevention paradox, which suggests that interventions targeted at whole populations are likely to be more effective in sustaining and improving population health than those targeted at high-risk subpopulations (Rose, 1992; Rose, 1985; Capewell and Graham, 2010). This epidemiologic fact presents ethical challenges because the most high-risk populations are frequently the most socially disadvantaged, which implicates issues of justice that compel relative priority for ameliorating the accumulated disadvantages affecting those lower on the social gradient (Wolff, 2009).

While the prevention paradox is not the subject of this analysis, it demonstrates the centrality of relative priority for thinking about ethics, health policy, inequities, and population health. Thus, one of the aims of this paper is to interrogate the significance of genetic causes of disease *relative* to social causes in terms of public health policy. To that end, I readily assume that primary goals of just public health policy ought to include the improvement of health across populations and the compression of health inequities. In Benach, Malmusi, Yasui, Martinez, and Muntaner's (2011) recent formulation, the ethically optimal public health policy is one in which the overall curve of population health shifts left, and in which the shape of the curve narrows. The former indicates that overall population health has improved, while the latter indicates that inequities have narrowed. Conceptualizing just public health policy goals as such is important for the central objectives for this article, which are: (1) to demonstrate the incoherence of the genetic-social causal dichotomy as to disease and inequities; (2) to weigh the relative effect size of genetic and social causes of disease and inequities; and (3) to consider the implications of the analysis in (1) and (2) for just public health policy.



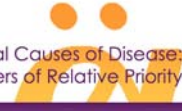
## II. A critique of the genetic-social causal dichotomy as to disease

### A. Theoretical critique

As to health and illness, the producers of the award-winning documentary *Unnatural Causes* (2008) saw the biological-social distinction as significant enough to justify its opening lines. With the opening credits still rolling, Nancy Krieger explains that one view of humans “as biological creatures is that we are determined by our genes (...) because of who we were born to be” (Adelman, 2008). Krieger rejects this view, and in its place argues that the consequences of our social lives are imprinted on our bodies. In her words, “we carry our history in our bodies”. And then, she queries evocatively, “how could we not?” How indeed?

The difficulty of drawing a meaningful distinction between genetic and social causes of disease follows from a relatively simple series of theoretical claims. The genetic factors involved in the etiology of the most prevalent human diseases are polygenic (Weeks and Lathrop, 1995), with untold numbers of genes and gene variants interacting with untold numbers of social and environmental variables to produce system behavior (expression). This iterative process follows from the basic tenets of systems theory, especially complex adaptive systems, which posits that overall system behavior is a product of the myriad interactions between variables and attractors within those systems. Classical mechanistic approaches are quite limited in their explanatory power for such systems, because isolating individual variables for analysis necessarily distorts the iterative processes through which variables interact with each other to determine system behavior (Jayasinghe, 2011). Thus Freese notes the fallacy of the notion that for any given disease,  $x$  % is caused by genetic factors and  $1-x$  % is caused by environmental factors (Freese, 2006).

As Richard Lewontin (2002, p. 141) puts it, in and of itself DNA “makes nothing” and “organisms are not determined by it”. What matters for health and illness, of course, is gene expression. But gene expression is best characterized by the “phenotypic bottleneck: the strict mediation of genetic causes by the phenotype” (Freese, 2008, p. S4). Moreover, the point is not merely that genes and environments interact to express, but rather that if drawing causal attributions to patterns of health and illness in populations is the goal, even a conceptual dichotomy between genetic and social causation of disease simply cannot get off the ground. Note that this claim is entirely compatible with the recognition that genetic factors exert causal effects on health and illness. However, whatever causal effects genes exert in shaping patterns of disease only follow from a complex series of interactions with an untold but likely large number of exogenous social and environmental variables. And again, attempts to isolate and



abstract these variables from each other fundamentally distort the causal processes themselves. As Lewontin puts it,

“[a] living organism at any moment in its life is the unique consequence of a developmental history that results from the interaction of and determination by internal and external forces. The external forces, what we usually think of as ‘environment’, are themselves partly a consequence of the activities of the organism itself as it produces and consumes the conditions of its own existence. Organisms do not find the world in which they develop. They make it. Reciprocally, the internal forces are not autonomous, but act in response to the external. Part of the internal chemical machinery of a cell is only manufactured when external conditions demand it.” (Lewontin, 2002, p. 148)

Indeed, it is not even true that genes interact with environments to express because, as Krieger and George Davey Smith (2004, p. 94) observe, it is organisms that interact with environments. This interaction has “consequences for gene regulation and expression” (Krieger & Smith, 2004, p. 94), but the key point underlying the analysis is that humans are necessarily embodied beings, which implies the fruitlessness of attempts to isolate strictly a particular molecular variable that exerts causal effects on illness.

Although the question of whether monogenic diseases such as Huntington’s chorea are caused solely by genetic entities is debatable (Braun, 2002), time and space preclude analysis, and hence I will assume the affirmative for the sake of argument. That is to say, the argument assumes that in cases that are rare across populations, such as those cases of Huntington’s chorea (prevalence of 1/10,000), the causal properties of the genetic factors relevant to the phenotypic expression of the disease can be meaningfully separated from the social factors relevant to its expression.<sup>1</sup> Although this is contestable, the assumption here is that social causes of disease do not alter the expression of Huntington’s chorea in those who have the requisite genetic composition.

But for what Chaufan refers to as the “common diseases” that account for the overwhelming majority of disease burden in the U.S., the argument holds (Chaufan, 2007, p. 19-21). There is simply no way of drawing any *a priori* distinction between genetic and social contributions to causality of any of the most epidemiologically significant diseases in American society (e.g., type II diabetes, coronary artery disease, asthma, pain). Therefore, if understanding causality of patterns of common diseases in American society is the primary goal, attempts to demarcate precisely genetic and social causes is chimerical. It is, to borrow a

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<sup>1</sup> In the U.S., the Rare Diseases Act defines a rare disease as any disease with a prevalence of 200,000 cases or less, or roughly 1/1500.

Wittgensteinian metaphor I return to time and again, an attempt to open doors that are painted onto walls (Goldberg, 2011; McGinn, 1997).

As it has since antiquity, causation remains one of the most closely scrutinized subjects in contemporary Western philosophy, and I follow Freese in conceding that the crude theoretical analysis above is utterly inadequate as a substitute for a thorough philosophical analysis of genetic and/or social disease causality (Freese, 2008). Yet while the simple account sketched above is both incomplete and insufficient, it is not, I submit, inaccurate in the salient respects. Philosophers who have closely examined notions of genetic causation largely concur. Carl Cranor (1994, p. 133), for example, observes that

“[t]he concern about ascribing too much causal influence to genes appears to have even greater credence in relation to polygenic diseases, diseases that are by definition the outcome of a complex set of conditions, including the interaction among several or many genes as well as between those genes and the surrounding biological context.”

Cranor’s analysis tracks the same claims I have advanced thus far: that, as to disease, genes almost certainly exert causal effects, but that attempts to ascribe causal force to genes separate and apart from the phenotypic bottleneck is even in principle dubious.

### *B. Empirical critique*

The empirical complement to this theoretical account is almost certainly more important, because the evidence base it rests on supports each of the central claims in this article: (1) that, as to health and illness, genetic causes are not meaningfully distinguishable from social causes; and yet (2) that difficulty negates neither the need nor the possibility of evaluating the relative priority of each set of causes as to just public health policies.

Stonington, Holmes, and the editors of the journal *PLoS Medicine* put the point succinctly in 2006: “the stark fact is that most disease on the planet is the result of the conditions in which people work and live.” The evidence base overwhelmingly suggests that social and economic conditions are the prime determinants of patterns of disease within and across human populations (which especially includes the inequitable distribution of disease burden in both the developing and the developed world). In Link and Phelan’s influential formulation, social conditions are fundamental causes of disease (Phelan, Link, and Tehranifar, 2010; Chang and Lauderdale, 2009; Lutfey and Freese, 2005; Link and Phelan, 1995). The evidence regarding the effect of social and economic conditions on health is copious, and is



supported by demographic and historical analyses extending from at least 150 years ago to the present (for the historical evidence, see Szreter, 2004).

These conclusions regarding the SDOH evidence base are not intended to paint a false picture of uniformity; there are vigorous disputes within social epidemiologic and affiliated literatures regarding any number of particular concerns and issues, both methodological and substantive. Nevertheless, the existence of these disputes in no way undermines the quantity and robustness of the evidence suggesting that social and economic conditions like income, housing, occupation, stigmatization, education, and environmental exposures are by a significant margin the prime determinants of health and illness in human societies. Moreover, the same evidence base demonstrates that it is likely the social gradients, the hierarchies according to which societies and communities organize themselves, that to a very large extent determine patterns of health and illness in populations. Hence the intentionally provocative opening salvo of the recent Final Report of the World Health Organization's Commission on the Social Determinants of Health: "Social injustice is killing on a grand scale" (2008; Birn, 2009, p. 31).

For purposes of this article, the evidence regarding the SDOH provides the empirical complement to the theoretical account offered above regarding the incoherence of an *a priori* distinction between genetic and social causes of disease. Even if the theoretical account is assumed to be incorrect, the SDOH evidence base offers powerful *a posteriori* reasons for denying the distinction. This evidence base demonstrates that our social lives are deeply imprinted on our biological selves, in ways consonant with the feedback loop concept that is a key metaphor for understanding any complex adaptive system.

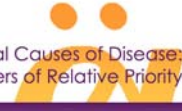
The following three examples highlight this relationship between the biological and the social. The first of these is one of the most promising causal models for the SDOH evidence base: the allostatic load theory (Borrell, Dallo & Nguyen, 2010; Mattei, Demissie, Falcon, Ordovas & Tucker, 2010; Brunner & Marmot, 2006). The SDOH evidence base generally documents robust and persistent correlations between indices of social, economic, and political status and health outcomes. But of course some causal models must be offered to explain the mechanisms by which deleterious social and economic conditions produce poor health. One of the most studied of these is the allostatic load theory, which posits that human stress responses are typically characterized by relatively short bursts of activity (usually but not always measured by increased levels of stress hormones like cortisol) with a return to a resting state (Borrell, Dallo, & Nguyen, 2010; Mattei, 2010; Brunner & Marmot, 2006). However, in individuals who are constantly subjected to profoundly noxious stimuli – like those exposures the most disadvantaged and most stigmatized experience constantly – the neuroendocrine system is essentially always running at a high level.

An accumulation of stress hormones like cortisol has been linked with all manner of diseases and negative health outcomes, especially in context of childhood development, where researchers have shown that persistently high levels of stress hormones can be neurotoxic (Lupien et al., 2009; McEwen, 2008; Lupien et al., 2000). This finding provides a molecular explanation for the overwhelming evidence that very early childhood is uniquely important in shaping and predicting health in later life.<sup>2</sup> This idea, typically referred to as the life course perspective, indicates that influences and exposures in childhood are critical and exert significant effects in shaping health over the life-span, even many decades later (Kuh and Smith, 2004). Moreover, the life course perspective is not limited to a single person's lifespan, because there is also excellent evidence that the social and economic conditions in which members of a community's parents and grandparents lived are powerful predictors of the health of persons in the current generation (Graham, 2010; Modin and Fritzell, 2009; Kuh and Smith, 2004). Accordingly, as Hilary Graham noted recently, "we urgently need perspectives that explicitly incorporate future generations, making future publics and future health integral to the concept of public health" (Graham, 2010, p. 154).

Although the allostatic load theory foregrounds the life course hypothesis, it is not alone in doing so. The life course perspective is also integral to a second example of the feedback-loop-relationship between the biological and the social: that related to epigenetics, or "the regulatory processes that control the transcription of information encoded in the DNA sequence into RNA before their translation into proteins" (Relton & Smith, 2010). In a very recent article, Relton AND Davey Smith (2010) point out the "increasing body of evidence to demonstrate that epigenetic patterns are altered by environmental factors known to be associated with disease risk (e.g., diet, smoking, alcohol intake, environmental toxicants, stress)...". The fact that Davey Smith is a co-author on this paper is significant, as he has been a key figure in developing the research base supporting the life course hypothesis and in addressing its policy implications. Accordingly, it is unsurprising that Relton and Davey Smith (2010) highlight the intergenerational nature of epigenetic effects: "Evidence that environmental exposures can act across generations to influence epigenetic patterns in offspring exist, with maternal exposure to famine during the perinatal period influencing offspring DNA methylation in adulthood." Thus not only do genetic factors exert causal effects on phenotypic disease expression, but exogenous social and environmental factors can alter DNA structure itself, in ways that very likely have effects on health.

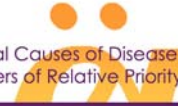
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<sup>2</sup> However, it is important to avoid the "biologization" of illness here, since such a reduction is exactly what analysis of the SDOH evidence base undermines. Understanding the molecular levels by which deleterious conditions determine poor health is important, but not at cost of the reduction of myriad complex social and upstream factors that shape health at the structural level. I am indebted to Melissa Flinders for emphasizing this point.



As a third example of the ways in which the biological and the social amalgamate, Krieger and Davey Smith (2004) have argued that social conditions cause illness by altering physio-anatomic structures themselves. They argue for an embodied understanding of health and illness in human societies, one that posits the image of the human as a jointly biologic organism and a social being. Krieger and Davey Smith do not dispute the existence of either the biologic organism or the social being, but argue that both recursively create and recreate the other. They document a number of ways in which “social influences become literally embodied into physio-anatomic characteristics that influence health and become expressed in societal disparities in health” (Krieger & Smith, 2004, p. 92). Thus, for example, Krieger and Davey Smith (2004, p. 95) note that birth weight is a “macroscopic characteristic that has long been associated with socioeconomic conditions”, and in turn document the evidence regarding the significant correlations between low birth weight and intergenerational health outcomes. Chaufan and Weitz argue for a similar causal pathway between deleterious social conditions and type II diabetes, because “[w]hen a pregnant woman is malnourished, her fetus also receives inadequate nourishment. This affects the development of the fetal pancreas leading to largely irreversible glucose intolerance, i.e., an in-born (but not genetic) biological predisposition to diabetes” (Chaufan & Weitz, 2009, p. 79). Krieger and Davey Smith (2004) cite other examples regarding the effect of deprivation and “socially patterned exposures” on age of menarche, height, *H. pylori* infection, and early childhood abuse, all intended to support the notion of embodiment in social epidemiology. “Embodiment” in this context means that so-called biological facts like height and birth weight – which are almost certainly affected by genetic factors – are substantially caused by social and environmental conditions and exposures.

Accordingly, the allostatic load theory, epigenetic causation, and the notion of embodied inequality in health and illness supply *a posteriori* reasons for doubting the meaning of a distinction between genetic and social causes of illness. Not only our social lives, but the social lives of what anthropologists term our “local moral worlds”, of the lives and worlds of our parents and our grandparents, shape our own lived experiences of health and illness. They are imprinted on our own bodies. It makes no sense to speak of the pathophysiology of illness or of the role the neuroendocrine system plays in disease states and illness experiences without understanding the fact that those mechanisms of illness and those systems are necessarily embodied, and, in being embodied, are substantially determined by our lived lives as social creatures. We are our physiologies and our neuroendocrine systems and our brains, but we are not merely any of these entities; we are embodied, which literally means that our social experiences shape and are shaped by those entities (Glannon, 2009; Krieger & Smith, 2004). Again, to paraphrase Krieger’s fundamental question (Adelman, 2008), how could it be any other way?



### III. Implications for public health policy

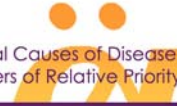
I referred earlier to a Wittgensteinian metaphor regarding quixotic attempts to open doors painted onto walls. Gasking and Jackson note that in a closely related passage, Wittgenstein observes that a man will be trapped in a room with a door that is unlocked so long as it does not occur to him to push rather than pull (Gasking & Jackson, 1978). I turn now to explaining the significance of this metaphor for thinking about the false dichotomy between genetic and social causation of health, and its implications for public health policy.

The first concern is methodological. Consider, for example, the results of a highly publicized clinical trial reported in the *Journal of the National Cancer Institute* in July 2009 (Albain et al., 2009). The authors note the inequalities in outcomes between African-American and Caucasian persons suffering from various cancers. They aver that the rationale for the study was to determine whether race was independently associated with these differences after controlling for variables that are known to produce inequalities such as socioeconomic factors, access to care both before and after diagnosis, and differences in tumor biology (Albain et al., 2009, p. 984). The study showed significant differences in outcomes after the investigators adjusted for confounding variables, and even after additional adjustments for SES. In addition, investigators found that while sex-specific cancers were correlated with worse survival along racial categories, the other tumor types studied showed no such differences. The authors accordingly speculated that perhaps “inherited genetic differences across races, especially in single-nucleotide polymorphisms, exist in the way resistance to therapy develops in these tumors and/or in how standard doses of drugs are activated and metabolized” (Albain et al., 2009, p. 991).

One of the lead investigators was quoted in the comprehensive cancer group’s official press release as concluding that the control for treatment and socioeconomic factors<sup>3</sup> “implicates biology (...) There may be differences in genetic factors by race that alter the metabolism of chemotherapy drugs or that make cancers more resistant or more aggressive” (Southwest Oncology Group, 2009). Similar explanations were proffered in a parallel study published two months prior to the *JNCI* article (Hershman et al., 2009). The latter study showed inequalities in survival between African-American and Caucasian women with breast cancer even after differences in discontinuation and treatment delay were adjusted for. The

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<sup>3</sup> The study dichotomy between medical treatment and socioeconomic factors is odd because medical treatment is almost certainly an effect of socioeconomic factors and may also in some cases be a cause of the latter. This is not to deny that the variables can be adequately operationalized according to the aims of this particular study, but simply to suggest that it is not generally safe to presume that medical treatment and socioeconomic status are independent variables.



authors suggest that “racial or ethnic differences in genes responsible for the metabolism of either chemotherapeutic agents or hormonal treatments may contribute to these findings, and this variability may affect both toxicity and effectiveness of the treatment” (Hershman et al., 2009, p. 2161).

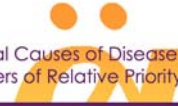
Studies that attempt to isolate genetic contributions to health are not unusual, as scholars concerned with the geneticization of disease have been quick to document (Phelan, 2005; Duster, 2003; Hedgecoe, 2003; Braun, 2002; Keller, 2002; Kerr, 2000). That many of these contributions use race as a proxy for genetic factors is also a source of significant criticism and concern, especially because of the fact that race is an exceedingly crude proxy (Duster, 2003; Braun, 2002).<sup>4</sup> However, the methodological concern is that such studies do not sufficiently control for the effects of social and economic conditions on health. The reason for this is the life course feature of the SDOH evidence base. Indeed, in an editorial accompanying the *JNCI* article, the editor-in-chief of the journal (and the chief medical officer of the American Cancer Society), Otis Brawley (2009, p. 970), explains the problem:

“Albain et al. tried to adjust for SES by using zip code data. This method adjusts for SES at time of treatment but does not take into account the fact that SES can change over a lifetime. Poverty in childhood and early adulthood may influence the tumor and its biology later in life.”

Brawley’s point underscores the fact that controlling for SES at the point of medical treatment does not control for the effect of social and economic conditions on health. As the robust evidence for the life course perspective suggests, it is the accumulated effect of deleterious social and economic conditions, especially those operating extremely early in the lifespan, that over time seem to result in what scholars have termed a “cascade” of health problems and difficulties later in life (Williams, 2003; Siegrist, 2000). SES is of course a powerful predictor of health, but when measured simply as a snapshot at a particular point in time, relatively late in life, does not and cannot account for the effects of low SES from early childhood (let alone from generations prior to conception). Indeed, Brawley’s observation that SES can change over a lifetime, while undeniably true, does not quite support the point he seems to be driving at, because there is excellent evidence that the negative health effects associated with the accumulation of risk in early life are not significantly mediated even by drastically improved SES later in life (Hayward & Gorman, 2004). What such an improvement does mediate is the health of ensuing generations (Freedman, Martin, Schoeni, and Cornman,

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<sup>4</sup> Indeed, the well-known fact that in-group variation exceeds between group-variation means that the social category of race cannot be mapped along genetic-biological lines.



2008), again emphasizing the critical importance of thinking about the SDOH evidence base from an intergenerational, life course perspective.

These arguments are not intended to minimize the significance of this particular study or others like it, but are rather intended to highlight the profound methodological difficulties that attend efforts to disentangle genetic and social causes of disease. If our social lives are imprinted on our biological selves, it is entirely unsurprising that even the most sophisticated modalities for teasing out disease causality encounter great difficulty in demarcating genetic from social causes in shaping inequitable patterns of disease.

Moreover, even assuming for the sake of argument that there exist superior techniques that would permit some kind of longitudinal control of deleterious social and economic conditions on health from an intergenerational life course perspective; the pressing question is what would be the social benefit to deploying them? Even if we could disentangle with any useful degree of precision genetic from social causes of disease, why would we want to?

Chaufan has supplied the most powerful recent answer to this question. In a series of articles that make extensive use of the SDOH evidence base, Chaufan criticizes the quest for “biological” and genetic explanations of the alarming prevalence and growth trajectories of type II diabetes, a quest that in its American rendition largely deemphasizes the relative priority of social and economic conditions as prime determinants of those disease patterns (Chaufan and Weitz, 2009; Chaufan, 2008; Chaufan, 2007a; Chaufan, 2007b). She argues that the social and economic conditions that cause patterns of type II diabetes are more significant from a public health policy perspective than genetic predispositions. Thus, even where disease is jointly caused *inter alia* by genetic and social factors, the empirical evidence strongly suggests that it is the social and economic conditions in which people live and work that exert the largest effects in shaping patterns of disease and inequities.

Of course, the relationship between health priorities and ensuing public policy is anything but a simple correspondence. For example, even if the relative effect size of social and economic conditions’ causal contributions to disease is large, if there are no interventions capable of ameliorating those conditions over a relatively short time horizon, its relative policy priority might be lower than one would imagine given the variables’ effect on health. Fortunately, the SDOH evidence base demonstrates quite well that amelioration of such conditions can have rapid effects in improving population health and compressing health inequities (i.e., significant changes are measurable in <10 years) (Williams et al., 2008, citing studies). The contrapositive proves the rule: negative changes in social and economic conditions can have rapid effects in diminishing population health and increasing inequities, as the evidence for the so-called healthy immigrant effect demonstrates (Malmusi, Borrell & Benach, 2010).

In any event, two physician-scientists issued a response to one of Chaufan's articles, arguing that intensive population-wide genetic studies are important to population health and inequities (Robertson & Poulton, 2008). For example, they reasoned, studies have shown that persons possessing a certain composition of alleles and who suffered maltreatment as children are more likely to suffer depression than persons who were similarly maltreated as children but who lacked the relevant genetic composition. Chaufan's reply (2008, p. 678) illustrates the point:

"I fail to see ... the public health value of a 'lens' that might allow us to identify, even prophylactically, prospective victims genetically. And why target the child at all for prophylaxis rather than maltreatment for elimination? The proper thing to do, it seems to me, if not for moral at least for prudential reasons, is to put societal resources into eliminating childhood maltreatment, while devoting the rest of our time, energy, and moneys to caring for those who have been maltreated, whatever their genotypes."

The critical social and ethical issue is assuredly not whether genetic entities exert causal effects on depression, but rather, for those interested in ameliorating the crushing and inequitable distribution of depression in the U.S. (Muntaner et al., 2004; Lorant et al., 2003), identifying what the best evidence suggests are the most important levers and levels of interventions. Granting for the sake of argument that there is some nontrivial quantum of benefit that could be obtained from the kind of genetic prophylaxis Robertson and Poulton posit, under even the most charitable account that quantum pales in comparison to the ameliorative impact the evidence suggests could be obtained by improving the deleterious conditions in which depressed people disproportionately live and work. Similarly, in a very recent article, Hall, Matthews and Morley (2010) scrutinize and reject the claims that genomic medicine is likely to have large effects on population health. They conclude that "[u]ntil we have a much stronger evidence base, and more data on interactions between genotypes and common environmental exposures, advocates of genomic medicine should be much more modest than some have been in the claims they make about its likely impacts upon population health." Ultimately, Chaufan's analysis underscores the point drawn by Lundy Braun: "Disease prevention policies differ dramatically depending on whether genetic factors or environmental and social conditions are invoked to explain racial inequities in causes and the natural history of disease" (Braun, 2002, p. 170).

This conclusion obviously generates significant ethical implications regarding social justice, full exploration of which is left for future work. Yet so compelling is the SDOH evidence base in terms of its ethical implications for public health policy, there is ample grounds for

questioning the apparent ubiquity of the attempts to control for the effects of social and economic conditions on health. Thus, regarding the fact that many of the studies seeking to control for social and economic contributions to health involve African-Americans, the significant question is why there exists so much interest in controlling for the lived experience of being black in the U.S.? (T. Duster, personal communication, December 8, 2010; J. Glenn, personal communication, 2009).

In their analysis of epidemiologic causation, Parascandola and Weed (2001) concur, concluding that there is no compelling reason for privileging molecular causation over variables that exert macro-level causal effects. Thus, if it is the case that the distinction between genetic and social causes of health and illness is dubious, and if social causes by a large margin are of most significance in shaping patterns of disease and inequities in human societies, the relevant question is what is the justification for the apparent commonality in research on health and illness of attempts to control for such social causes in the interests of investigating *molecular* causes?

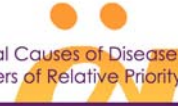
None of the arguments above compel an argument that research and funding for genetics should be abandoned. It is entirely possible to support public health policies that allocate resources and attention both to genetic science and to amelioration of the (social) fundamental causes of disease. But scarce resources and opportunity cost trade-offs require stakeholders to prioritize public health policy choices relative to each other, even while myriad policies can and likely should be simultaneously endorsed. Thus the claim here is not that resources and attention for genetic science should be eschewed, but rather that significantly larger shares of such attention should be directed to the social and economic factors that fundamentally determine patterns of the common diseases and inequities in American society (Goldberg, 2009).

#### IV. Conclusion

Harold Varmus is the current Director of the National Cancer Institute as well as the former Director of the National Institutes of Health. He was awarded the Nobel Prize at least in part for his contributions to the understanding of oncogenes, genes that when mutated or expressed at abnormally-high levels seem to pose significant risk factors for certain kinds of cancer. Yet Varmus himself noted recently that “genomics is a way to do science, not medicine” (Wade, 2010). This does not mean that public support for such an endeavor should be abandoned, but it does underscore the argument that in terms of its contribution to population health and inequities, genetics more closely resembles astrophysics than occupational health,

and occupies a different constellation of ethical significance in relation to health than expansive models of social medicine and public health.

Wittgenstein's remark about the man who is trapped by his own judgments even while the path to freedom is tantalizingly open is of course reminiscent of the allegory of the cave. This article does not suggest any answers to the important question of why the trapped man is so insistent on pulling rather than pushing the door, of why the attention and resources paid to genetic causes of disease seem to be greater than that paid to social causes of disease (Freese, 2008; Braun, 2002). This question is itself critical, and while work has been adduced that suggests plausible answers, there is a pressing need for further work on the social, cultural, and political factors that shape any given community's causal attributions as to disease and the public health policy responses that do or do not follow (Fairchild et al., 2010; Räsänen et al., 2006; Tesh, 1995; Hamlin, 1992). In terms of genetic causes, the significance of the latter body of work is inextricably linked to the merit of the critique offered above. If it is the case that the causal properties of genetic and social causes of disease cannot fairly be disentangled, and if it is also the case that social factors are much more significant causes of disease and inequities than genetic factors, then it becomes all the important to understand why so much attention and resources directed to health, illness, and inequities in American society focuses on genetics.

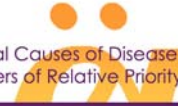


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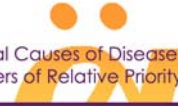
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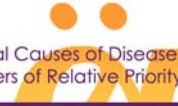
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