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Locating gene–environment interaction: at the intersections of genetics and public health

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Abstract

Over the past two decades, the applications of genetic and genomic technologies have begun to transform research questions and practices within epidemiology and toxicology, the "core sciences" of public health (Annu. Rev. Public Health 21 (2000) 1). These technologies provide new models and techniques for studying genetic traits, environmental exposures, and gene–environment *interaction* in the production of human health and illness. This paper explores the consequences of emergent genetic and genomic approaches, their ongoing redefinitions of both genetic and environmental "risks", and their potential implications for public health practice. The central argument of the paper is that the increasing focus on gene–environment interaction directs scientific, biomedical, and public health attention both *inward*, to the gene/genome, and *outward*, to particular places. In so doing, studies of gene–environment interaction create a challenging and productive tension—at the same time that bodies are being geneticized (Am. J. Law Med. 17 (1992) 15), they also are emphatically emplaced, located where social and cultural practices come to matter. This tension, this simultaneous movement outward and inward, towards the gene and towards the environment, into the body and into place, opens up a vista into the processes through which culture and biology form a locally and historically situated dialectic (Encounters With Aging: Mythologies of Menopause in Japan and North America, University of California Press, Berkeley, CA, 1993) and raises important questions about the production of health and illness.

Keywords: Public health; Genetics; Environment; Health policy

> For my flesh is finely meshed with the worlds flesh—and thus with the places presented and sedimented within the world: a place-world in which I can live and move and have my being.
>
> Casey, 1999, p. 238

> …society is inscribed on the expectant canvas of our flesh and bones, blood and guts.
>
> Scheper-Hughes and Lock, 1991, p. 413

Introduction

Contemporary public health research and practice is based, in large part, on the quantification of different types of "risk" and the identification of specific factors which enhance and/or compromise human health. Such factors may be understood as internal to the body, external to the body, or as produced through myriad practices that create exchanges and interfaces between people and the places they inhabit. The location and attributed salience of risk(s) for a particular condition (or for health and illness more generally) are consequential, in part, because they shape both individual and institutional practices aimed at producing, protecting, and restoring health. Different frameworks for understanding bodies and environments suggest and prioritize different types of health promotion, disease prevention, curative practices and public policies.

In academic research centers, government regulatory agencies, and some community-based organizations, there appears to be a "growing consensus" (NIEHS,

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1994) that gene–environment interaction plays a critical role in etiologies of human health and illness. This emerging consensus simultaneously is supported by and provides the rationale for a rapidly broadening scope of knowledge production focused on the processes, mechanisms, and consequences of gene–environment interaction.[1] This paper describes the study of gene–environment interaction,[2] explicates its definitions of risks, and explores the implications of these definitions for public health policy and practice. In particular, I examine what aspects of bodies and environments, people and places are made more and/or less visible by conceptual frameworks and technologies for studying gene–environment interaction. I argue that the promise of the study of gene–environment interaction is in its direction of scientific, biomedical, and public health attention simultaneously *inward*, towards the gene/genome and the interior of the body, and *outward*, towards particular practices, places and the exposures they contain and enable. Insofar as it places the reductionist paradigm of molecular genetics within the context of place,[3] this dual focus helps to elucidate the inextricability of the biological and the social (Clarke, Fishman, Fosket, Mamo, & Shim, 2000; Lock, 1993; Scheper-Hughes and Lock, 1987, 1991) and raises important questions about how people become vulnerable to environmental exposures.

This research derives from a larger research study of the implications of genetic/genomic technologies and conceptual frameworks for the practice of environmental health science and environmental regulation in the United States (Shostak, 2000). This article draws from a review of the literature on gene–environment interaction and human health, 1950–2000. The primary articles considered for this analysis were identified through a Medline search using the keywords "environmental genetics", "environmental genomics" and/or "ecogenetics". I also considered review articles and book chapters in the following fields: genetic epidemiology, molecular epidemiology, pharmacogenetics, and toxicology. In addition, I reviewed the table of contents of the National Institutes of Environmental Health Sciences journal "Environmental Health Perspectives" from its inception in 1972 to January 2001, and collected articles that contained title word references to genetic susceptibility to environmental exposure and/or gene–environment interaction. Finally, the review encompassed reports of relevant symposia and conferences that have been published as special issues of journals or as independent government reports. Articles were coded and analyzed using the general principles of grounded theory, an inductive research methodology (Glaser & Strauss, 1967; Strauss, 1987; Strauss & Corbin, 1990/1998).

Studying genes and the environment—a partial history

Many of the questions that shape contemporary studies of genetics, environmental exposures, and their interaction are not new (Calabrese, 1996). Indeed, scientists speculated about the relationships between genes and the environment in human health and illness as early as 1938, when J.B.S. Haldane suggested that genetic differences might explain the variability in responses of potters to industrial exposures:

> …But while I am sure that our standards on industrial hygiene are shamefully low, it is important to realize that there is a side to this question that has been completely ignored. The majority of potters do not die of bronchitis. It is quite possible that if we really understood the causation of this disease, we should find that only a fraction of potters are of a constitution that rendered them liable to it… There are two sides to most of these questions involving unfavorable environments (Haldane, 1938, p. 102).

In 1957, Motulsky first noted that the individual variations which had begun to appear in both clinical practice (see, for example, Jensen, 1962) and scientific research might be related to susceptibility or resistance to conditions other than "drug idiosyncrasies": "genetically conditioned drug reactions… may be considered pertinent models for demonstrating the interaction of heredity and environment in the pathogenesis of disease" (Motulsky, 1957, p. 836). Brewer (1971) coined the term "ecogenetics" to refer to the study of the "unique biochemical individuality" of humans and argued that genetic variation is not only relevant to drug action, but should be considered in response to any type of environmental agent (1971, p. 92). In 1975, a report by the National Academy of Sciences featured a separate section on the role of genetic metabolic "errors" as predisposing factors in the development of toxicity from occupational and environmental pollutants; an appendix to the report listed 92 genetic disorders that could predispose affected persons to toxic effects (NAS, 1973/1975). In 1983, the Cold Spring Harbor Laboratory hosted a conference on genetic

[1] Consider, for example, the National Institutes of Environmental Health Sciences (NIEHS, 1997) Environmental Genome Project, begun in 1997, and National Center for Toxicogenomics, established in 2000.

[2] The study of gene–environment interaction is referred to, in the literature, by researchers, and in this paper as "environmental genetics", "environmental genomics", and "ecogenetics" (Nebert, 1999, p. 248).

[3] My thanks to one of the anonymous reviewers of my manuscript for this helpful phrasing.

factors enhancing susceptibility to chemical agents (Omenn & Gelboin, 1983).

Throughout the 1970s, ecogenetic research focused primarily on the effects of genetic conditions (e.g., G-6-PD deficiency, sickle cell anemia, thalassemia) on individuals' ability to metabolize toxic substances (Calabrese, 1996). However, starting in the 1980s, and accelerating in the 1990s, the scope of environmental genetic research began to broaden. New technologies[4] provided researchers with an array of new models and techniques for studying gene–environment *interaction* (Puga, Jana, Ching-yi, Hung-chi, & Nebert, 1996). These technologies have supported the development of molecular biomarkers as indicators of exposure to environmental substances and/or the effects of such exposures, increasing scientific understanding of the mechanisms through which toxic exposures affect human health (Hemminki, Grzybowska, Widlak, & Chorazy, 1996; Nebert, 1999; Perera & Weinstein, 1999). New approaches have also increased the variety of genetic susceptibility that can be studied (Puga et al., 1996).[5] The concept of gene–environment interaction is now a central theme in studies that assess causes of human diseases in populations (Khoury, 1997).

Contemporary research into gene–environment interaction encompasses both the ways in which persons are differentially vulnerable to environmental agents *and* the ways in which individuals have been affected by exposure to such agents (including the acquisition of genetic susceptibilities). Put differently, the current iteration of environmental genetic research attempts to assess two different categories of risk, those posed by characteristics within the body (e.g., individual genetic susceptibilities) and those posed by the environment (e.g., chemicals or ionizing radiation), *and how they interact* in the production of health and illness. In so doing, researchers also study the many biological processes and mechanisms that mediate the outcomes of particular exposures, such as nutrition, age, the physiological effects of prior exposure, and/or the consequences of simultaneous, multiple exposures. This research arena includes a variety of techniques and technologies, including DNA microarrays (Iannaccone, 2001; Lovett, 2000, pp. 536–537; Nuwaysir, Bittner, Barrett, & Afshari, 1999; Society of Toxicology, 2001), molecular biomarker techniques (Christiani, 1996; Hemminki et al., 1996; Ishibe & Kelsey, 1997; Jones, Buckley, Henderson, Ross, & Pike, 1991; McMichael, 1994; Perera & Weinstein, 1982, 1999; Perera, 1997; Schulte & Perera, 1993; Wang et al., 2000; Wogan, 1992), and a wide range of epidemiological approaches (Khoury, Adams, & Flanders, 1988; Khoury, Beaty, & Cohen, 1993; Khoury, Flanders, & Beaty, 1988; Khoury and the Genetics Working Group, 1996; Khoury 1997). Amidst this disciplinary and technological heterogeneity, it is possible to discern several predominant (and increasingly interrelated) lines of research.

Genetic epidemiology, the disciplinary intersection of genetics and epidemiology, is an integration of the research methods of traditional epidemiology with the concerns of human genetics to assess genetic–environmental interactions in the etiology and progression of disease (Khoury et al., 1993). Though the field has existed since 1977, it has been fueled in recent years by rapid advances in molecular biology (Khoury et al., 1993, p. 8). Genetic epidemiology focuses on the interaction between human genetics and environmental factors, emphasizing that most diseases are neither purely genetic nor environmental, but dependent on *interactions* between internal and external factors (Khoury et al., 1993, p. 12). However, in the environmental health sciences, genetic epidemiological research to date has focused on the question of individual susceptibilities to environmental exposures (Calabrese, 1997).

Molecular epidemiology is characterized by the use of molecular biological techniques and biomarkers[6] in epidemiologic research to assess individual exposure, dose, pre-clinical effects, and susceptibility to toxic substances (usually carcinogens) (Perera & Weinstein, 1999, p. 517; Schulte & Perera, 1993, p. 7). Molecular epidemiology was first proposed in 1982 as a framework for combining the techniques and concerns of molecular biology and epidemiology in cancer research (Perera & Weinstein, 1982; Perera & Weinstein, 1999). The identification and assessment of individual susceptibility markers is a primary goal of molecular epidemiology: "Why similarly exposed people do not get the same diseases is a target question for molecular epidemiology. In most disease systems, susceptibility markers are being identified and evaluated" (Schulte & Perera, 1993, p. 7).[7]

[4] Examples include polymerase chain reaction (PCR), differential display reverse transcriptase PCR, and targeted gene disruption approaches (Puga et al., 1996) and DNA microarray techniques (Iannaccone, 2001; Lovett, 2000).

[5] These now include allelic differences in genes encoding proteins involved in the function of receptors, drug metabolism, ion channels, multidrug resistance protein pumps, second messenger pathways, DNA repair, and the chelation of metals (Puga et al., 1996).

[6] Molecular biomarkers are "indicators signaling events in biological systems or samples" at the molecular level, such as DNA adducts, gene mutations, and chromosomal aberrations (Perera & Weinstein, 1999, p. 518).

[7] There are three primary categories of susceptibility markers: (1) markers pertaining to enzymes that increase or decrease the ability of a chemical to interact with DNA, RNA, or proteins; (2) markers of genetic differences in the capacity of cells to repair DNA damage caused by environmental insult; (3) pre-existing inherited genetic conditions that increase the risk of disease (NIEHS, 1994).

However, molecular epidemiology also explicitly focuses on the interactions between individual genetic susceptibility and environmental factors in disease initiation and progression: "…perhaps the greatest contribution of molecular epidemiology has been the insights it has provided into interindividual variation in human cancer risk and *the complex interactions between environmental factors and host susceptibility factors, both inherited and acquired*…" (Perera & Weinstein, 1999, p. 517, emphasis added). Moreover, molecular epidemiology also offers new means of detecting and documenting exposure. This line of research identifies biomarkers that signal events at the molecular level; these visible alterations in molecular structures then can be used to reconstruct environmental exposures (Jones et al., 1991; Kreps, Banzet, Christiani, & Polla, 1997; Mendelsohn, 1991; Vogelstein & Kinzler, 1992; Wogan, 1992).

Toxicogenomics is the study of gene expression and gene products important in adaptive responses to toxic exposures (Iannaccone, 2001). Specifically, toxicogenomics uses microarray techniques called "chips" to examine how genes respond to environmental exposures (Lauerman, 2001; Nuwaysir et al., 1999). At this time, data from microarrays are difficult to interpret and genomic techniques have not replaced more traditional methods of environmental health risk assessment. However, researchers believe that toxicogenomics increasingly may be used to compare and evaluate the effects of different chemicals, to screen new chemicals, and to elucidate the effects of chemical mixtures in the human body (Warhurst, 2000, p. 18; Lovett, 2000, pp. 536–537; Society of Toxicology, 2001).

There are differences in the histories and practices of each of these fields; however, they share several fundamental assumptions. First, they examine the possible contribution of genetic factors (including changes in gene expression as a response to environmental stressors) in the etiology of a wide variety of human diseases (Khoury, Burke, & Thomson, 2000, p. 7). Second, they posit that disease is neither purely genetic nor environmental, but dependent on *interactions* between genetics and environment (Khoury et al., 1993, p. 12; Perera & Weinstein, 1999, p. 517). Third, they encompass a dual focus on "why some exposed individuals develop adverse events following exposure to environmental agents (e.g. carcinogens, food, insecticides) and how people adapt to the environment in different ways" (McNicholl, Downer, Aidoo, Hodge, & Udhayakumar, 2000, p. 188).

Together these research endeavors, their theories and technologies, form the theoretical, material, and empirical basis for the applied field of *public health genetics*,[8] defined as "the application of advances in genetics and molecular biotechnology to improve public health and prevent disease" (Khoury et al., 2000, p. 5). Public health genetics endeavors to encompass the following traditional public health functions: (1) public health assessment [surveillance]; (2) evaluation [of genetic testing]; (3) development, implementation and evaluation of population interventions; (4) communication and information dissemination (Khoury et al., 2000, p. 8). Advocates of public health genetics see genetic knowledge and technologies as integral to fulfilling the mission of public health[9] and assert that the integration of genetic information into all public health programs is "unavoidable": "All public health professionals… will need an increasing appreciation for integrating genetic research, policy, and program development into their daily work" (Khoury et al., 2000, p. 5). Public health genetics is a growing presence in major public health institutions in the United States.[10]

The implication of the public health genetics framework is that public health practice requires knowledge of both individual (and subpopulation) genetic susceptibilities *and* the risks posed by environmental factors. Individual susceptibilities are not seen as amenable to intervention, so public health practice is directed to "the modifiable risk factors for disease that interact with genetic variation and that may be used to help target prevention" (Khoury et al., 2000). The goal of such practices is "phenotypic prevention", or "the prevention of the physical manifestation of genetic traits", and is achieved by interrupting the interaction of environmental cofactors with human genetic variation or by using gene therapies to correct for deficiencies in gene

[8] I focus here only on public health genetics in the United States, though there is an analogous field in Europe, which is known as "community genetics" (Khoury et al., 2000, p. 5).

[9] The Institute of Medicine defines the mission of public health as follows: "to fulfill society's interest in assuring the conditions in which people can be healthy" (in Khoury et al., 2000). There is great heterogeneity of opinion across the field of public health regarding how this mission is best accomplished.

[10] For example, in 1996, the CDC sponsored a task force on Genetics in Disease Prevention, which published recommendations for integrating genetic concepts and technologies into public health practice in prominent genetics and public health journals (Khoury and the Genetics Working Group, 1996; Khoury, 1997). In 1997, the CDC opened an Office of Genetics and Disease Prevention. There are now specialty programs in public health genetics in the schools of public health at the University of Michigan and the University of Washington. There are also an ever proliferating number of public health genetic conferences and events, including an annual meeting of the Association of State and Territorial Health Officials (ASTHO), the CDC, the Health Resources and Services Administration (HRSA), and the National Human Genome Research Initiative (NHGRI). Advocates of this new approach predict a transformation of public health practice through public health genetics that will "usher in a golden age of genetics and public health sciences and public health practice, particularly for environmental health" (Omenn, 2000, p. 1).

products (Khoury et al., 2000, p. 8).[11] One model for phenotypic prevention comes from largely successful genetic screening and dietary intervention programs for phenylketonuria (PKU), a condition that, if untreated, leads to mental retardation in affected individuals. Approximately 40 years ago, researchers demonstrated that if infants with PKU are diagnosed at birth and "treated" with a phenylalanine-limited diet, then they do not develop mental retardation (Omenn 2000, p. 36). Although PKU is a "genetic condition", mental retardation among people with PKU manifests only as a result of the *interaction* of genetics and environmental exposure (in this case, diet). As such, PKU screening and treatment provides a striking example of a phenotypic prevention that averts an adverse health outcome (mental retardation) by enabling individuals with a genetic condition (PKU) to avoid a particular environmental exposure (dietary phenylalanine). The possibility of phenotypic prevention for individuals with PKU is oft cited by advocates of public health genetics as having "exploded" the myth that nature and nurture are competing explanations, rather than interacting factors, in health and disease (Omenn 2000, p. 36). At the same time, the stringent lifetime dietary modifications necessary to prevent mental retardation for individuals with PKU highlights the challenges of such prevention strategies for individuals who are susceptible to exposures that may prove even less easily avoidable than dietary intake of phenylalanine.

The emerging public health focus on gene–environment interaction has the potential to reshape the definition of both genetic and environmental risks and, concomitantly, the regulatory and legal contests over their meaning and significance. The importance of changing definitions of genetic and environmental risks within public health genetics is, in large part, a result of the prominence of risk discourse within public health.

Risk discourse and public health

Since the end of WWII, public health research has been characterized by efforts to predict and, when possible, modify risk factors for disease. In turn, public health practice was increasingly oriented to identifying discrete individuals and groups in a population which though they may be experientially and clinically "well" are defined as "high risk" for adverse health outcome(s) (Pearce, 1996; Petersen & Lupton, 1996, p. 18; Susser & Susser, 1996a; Taubes, 1995; Wing, 1994). The "risk factor paradigm" has generated intensive debate within public health. Advocates claim that risk-based analyses provide a rational approach to disease prevention and point to its ability to (1) provide a mechanism for surveillance to see whether problems are being prevented and whether interventions are beneficial or otherwise; (2) predict the level of care required by individuals or communities at different levels of risk; (3) provide anticipatory care and allocation of resources to individuals and communities at different levels of risk (discussed in Frankenberg, 1993, p. 233). Social epidemiologists have criticized risk factor analysis for ignoring fundamental causes (e.g., poverty) in favor of proximal ones (e.g., lifestyle) (Krieger, 1994; Link & Phelan, 1995; Pearce, 1996; Susser & Susser, 1996a, b). Molecular epidemiologists are troubled by the "black box" of risk factor analysis, that is, its inability to explain the biological mechanisms that lead to health and illness (Vandenbroucke, 1988). It has also been widely analyzed by social scientists a form of governmentality characteristic of neoliberal states (Castel, 1991; Gordon, 1991; Lupton, 1994; Petersen & Lupton, 1996; Turner, 1997). However, no matter which positions one may take regarding the desirability of risk analyses, "risk" has clearly taken a central role in the organization of contemporary public health research, policy, and practice (Pearce, 1996; Susser & Susser, 1996a, b). Indeed, struggles over health policies, whether lawsuits regarding the effects of tobacco smoke or community-based conflicts over the siting of hazardous waste facilities or the assessment of a particular hazards, are often struggles over the definition of "risk" (Beck, 1992; Brown & Mikkelsen, 1990/1997; Irwin & Wynne, 1996).

Risk discourse in public health implicates a wide variety of sites and practices (Petersen & Lupton, 1996). Social scientists have delineated at least three "types" of risk which constitute risk discourse, each one with its own set of foci (Lupton, 1994). Under one definition, risk is conceptualized as "the composition of impersonal 'factors'" (Castel, 1991, p. 288; Rabinow, 1996, p. 100). Such a construction of risk is seen in the actuarial tables of health insurance underwriting (Hacking, 1991, p. 2). This transformation of individual subjects into agglomerations of various "types of risk" enables the identification and control of "flows of population", defined by "the collation of a range of abstract factors deemed liable to produce risk in general" (Castel, 1991, p. 281). When risk is calculated this way, purely through "the identification of sites statistically locatable in relation to norms and means", a new biomedical subject emerges that is independent of history, place, and experience (Rabinow, 1996, p. 100). The second definition of risk,

[11] In contrast, genotypic prevention is the interruption of genetic trait transmission from one generation to the next through reproductive counseling, carrier testing, prenatal diagnosis, and pregnancy termination. This is a set of strategies that hearkens back to both American and Nazi eugenic programs (Kevles, 1985) and has been critiqued by observers of contemporary genetics (see, for example, Duster, 1990; Rapp, 1999).

traditionally used in the occupational and environmental health sciences, concerns dangers to populations posed by environmental hazards such as pollution, nuclear waste, and toxic chemical residues. Although neither identity nor experience is encompassed by this definition, it does address a place-specific set of risks, insofar as exposure is a socially contingent experience and social practices (such as occupation, diet, etc.) modify exposure and, therefore, risk. The third definition of risk, that most frequently invoked by activists, advocacy groups, and social epidemiologists, focuses on the political processes through which specific social groups are put "at-risk" by virtue of their political-economic position (Lupton, 1994, p. 77). This is an explicitly critical analysis that looks for the production of risk in political and economic structures and processes. Each of these definitions of risk highlights and/or obscures a different set of locations, whether inward (bodily), outward (environmental), or interactive.

Public health practices include efforts to identify high risk individuals and groups, inform them of their "risk status" and to offer interventions for "managing" their risk(s), based, varyingly, on one or more of these definition of risk. Depending on the type of risk, public health practice may include individual risk assessment, counseling and case management, family level interventions, intervention programs placed in schools, work sites, or communities, mass media campaigns, environmental remediation and clean-up, and other policy interventions (Lupton, 1994, p. 82). Risk discourse may support public health and clinical practices that focus both within and beyond the body: "the risk factor exists in a mobile relationship with other risks, appearing and disappearing, aggregating and disaggregating, crossing spaces within and without the corporeal body" (Armstrong, 1995).

Given the importance of risk discourse in public health practice and policy, ongoing redefinitions of genetic and environmental risks have the potential to significantly reshape the practices of this field. There are at least two processes at work herein. First, as discussed below, new technologies for studying gene–environment interaction may make different interior and exterior locations and processes more (and potentially less) visible, mapping pathways between internal and external spaces, the interaction of their contents, and the relevance of these interchanges for human health. Second, these conceptual frameworks, definitions, and technologies are being brought into public health by researchers and policymakers, who are eager to see an active role for genetic knowledge and practices within public health (Khoury et al., 2000; Omenn, 2000).

In the coming pages, I explore key conceptual and practical implications of the sciences of gene–environment interaction for the field of public health. My point of departure is the premise of public health genetics, as articulated by some of its most passionate advocates: "…all human disease is the result of *interactions* between genetic variation and the environment (broadly defined to include dietary, infectious, chemical, physical, and social factors)" (Khoury et al., 2000, p. 7, emphasis added).

Bodies in places: individual susceptibilities, localizing biologies, and public health

The identification of individual variation in response to environmental exposures is a primary goal of the study of gene–environment interaction. Moreover, much of the scope of contemporary public health genetics pertains to genetic testing and evaluation of individual susceptibilities. The process of using genetic technologies to identify "individuals at risk" has been the subject of much concern and contention. This section of the paper describes predominant themes in the debate about research and testing for individual genetic susceptibilities, focusing particularly on issues of genetic reductionism and determinism (for a discussion of the debates about privacy and discrimination, see Greely, 1992; Rothstein, 1997). It also discusses the implications of studying gene–environment interaction, both in terms of this ongoing debate and for the field of public health.

Genetics' risks

The tendency towards reductionism has been the subject of multiple social scientific critiques of genetic framings of "risk". Such critiques of genetic reductionism are aimed not at the heuristic methodological reductionism of the biological sciences, but to the extension of such methodological approaches to matters of ontology (Sloan, 2000, pp. 16–17; Opitz, 2000, p. 446). Genetic reductionism refers to an interpretation of "the gene" as the material entity that is the singular underlying "true cause" of expressed bodily traits and/or "human nature" (Sloan, 2000, p. 17). For example, in her pioneering analysis of "geneticization", Lippman described the process by which "priority is given to searching for variations in DNA sequences that differentiate people from each other and to attributing some hereditary basis to most disorders, behaviors and physiological variations" (Lippman, 1992, p. 13). To the extent that this "single conceptual model… is increasingly elicited to reveal and explain health and disease, normality and abnormality, and that is directing the application of intellectual and financial resources for resolving health problems, profoundly affecting our values and attitudes", Lippman predicted a concomitant

lack of attention to the environmental context of disease:

> Recently, a genetic variation said to be associated with increased susceptibility to lead poisoning was described in the literature, with the authors implying this might be a useful objective for a screening program. Do we really want to screen for genes rather than clean out lead to prevent the avoidable damage known to affect the millions of children unnecessarily exposed annually to this toxic agent? (1992, p. 27)

Sociologist Troy Duster levied a similar critique, arguing that the tremendous expenditure on genetic research would disproportionately and negatively impact Blacks by diverting attention and resources away from the environmental factors which are most likely responsible for the increasing rates of lung cancer and cardiovascular morbidity and mortality in the African American population (Duster, 1990, p. 116). His arguments highlighted the consequences of genetic framings of disease: "To conceptualize problems as primarily genetic is to chart a range of actions set in motion by the purported explanatory power of the genes… it is hardly neutral, unpackaged scientific "facts" that are influencing whether one heads down one path or another" (Duster, 1990, p. 55). These critics contend that to focus on genetic contributions to disease is to obfuscate, and thereby fail to address, the social and environmental causes of illness (Di Chiro, 2002; Draper, 1991).

An associated critique pertains to genetic determinism, that is, the assumption that a genetic association is sufficient for explaining phenotypic expression, independent of any other factors (Lippman, 1992; Alper & Beckwith, 1993). Genetic determinism is common among advocates of genetics, though many scientists, both biological and social, have tried to curb the rhetorical excesses of their colleagues (Cunningham-Burley & Kerr, 1999, pp. 156–7). Concern about genetic determinism has been particularly pronounced in the area of behavioral genetics (Billings, Beckwith, & Alper, 1992; Conrad, 1999). Conrad notes that the "one gene one disease" (OGOD) model predominates popular discourse about genetics, despite the fact that single-gene disorders are relatively rare (Conrad 1999, p. 231). This tendency is also manifest in discourse about finding "the gene for" a particular trait, as if our genes are our destiny (Billings et al., 1992; Conrad, 1999).

Given these critiques, what, then, are the implications of reorganizing public health policy and practice around genetic knowledge and technologies? The extension of a genetic framework to all forms of disease, that is, the geneticization of human health and illness, is a central aspect of public health genetics. Advocates of the integration of genetics into public health argue that "genetic factors play a role in the etiology of all human diseases, even those that are not traditionally thought of as 'genetic'" (Khoury et al., 2000, p. 7). Is there any reason to believe that environmental genetics offers a reframing of health and illness that may overcome the reductionist and deterministic framings of earlier iterations of human genetics? Might the focus of environmental genetics on epigenetic (i.e., focusing on gene–environment interaction) traits and multifactorial etiologies redefine dominant notions of risk?

Inherited tendencies

Early environmental genetic definitions of "individual susceptibility" shared the reductionist and deterministic limitations of the wider genetic discourse. These tendencies derive, in part, from the disciplinary origins of studies of gene–environment interaction. Specifically, ecogenetics began as an extension of pharmacogenetics, "the study of variability in drug response due to heredity" (Nebert, 1999, p. 245). Such research and writing created the first analytic spaces and institutional resources for investigating gene–environment interactions. For example, in a 1963 paper entitled "Detecting Hypersusceptibility to Toxic Substances", Mountain described his research on genetic susceptibility to the air pollutants ozone and nitrogen dioxide as based on a model developed in pharmacogenetic studies of G-6-PD deficiency and hemolytic anemia (Mountain, 1963, p. 360).

The goal of early pharmacogenetic research was to make individual genetic traits/risks visible through drug studies. Indeed, in the first review of pharmacogenetic research in the *Journal of the American Medical Association*, pharmacogenetics was defined as "the *study of genetically determined variations* that are *revealed* solely by the effects of drugs" (Price-Evans, 1963, p. 639, emphasis added). The "environment" was not an independent variable in pharmacogenetics. The focus was on the genetically determined responses of individuals (either alone or aggregated into family or other "subpopulations") to specific drugs.

Environmental genetics inherited these reductionist and deterministic tendencies from its disciplinary predecessor. The history of environmental genetics is replete with examples of situations in which genetic susceptibility (whether or not clearly identified) has been sought and/or claimed in order to minimize or obscure corporate, environmental, or occupational factors related to disease in individuals and populations. For example, the tobacco industry has a long-standing interest in genetic research that might identify a gene that both leads one to smoke and predisposes one towards cancer. Such a gene would be of enormous legal and public relations value to the tobacco industry: "if

differential cancer susceptibilities to tobacco could ever be established, one could plausibly argue that people who come down with the disease have at least partly their own heredity to blame'' (Proctor, 1995, p. 107). Genetic susceptibility has also been invoked to ''explain away'' cancer clusters of possible environmental origin. When high rates of breast cancer (15% above the national average) were found on Long Island, the CDC reported that the excess was due primarily to the high percentage of Jewish residents, noting that Jews are known to have higher rates of breast cancer than other Americans (Proctor, 1995, p. 241).

Environmental genetic knowledge also has implications for public health practices in occupational settings (Rothstein, 1997; Schulte, Lomax, Ward, & Colligan, 1999; Soskolne, 1997) and the history of such interventions raises the specters of genetic determinism and discrimination. For example, genetic studies have been used in the efforts of various government scientists and regulators and business owners to mandate genetic screening for workers with ''hypersusceptibility'' to occupational exposures, defined as ''a condition of inordinate or abnormally increased susceptibility to chemicals, infective agents, or other agents which in the normal individual are entirely innocuous'' (Draper, 1991, p. 43; Nelkin, 1989). Many advocates of such screening are clearly interested not only in protecting the health of workers, but in protecting companies from workers' compensation claims. Very early in the history of genetic testing, government scientists argued: ''The industrial physician could employ to advantage such tests to distinguish heredity based disease from job claimed disability'' (Stokinger, Mountain, & Scheel, 1968, p. 973). Such initiatives have been criticized by labor and environmental activists and academic observers for obfuscating the hazards of workplaces by focusing instead on internal, individual genetic risks. For example, critics point out that the assumption that the chemicals to which ''susceptible'' workers are considered vulnerable are otherwise innocuous ''reflect[s] management's interest in finding individual rather than industrial causes of problems'' (Draper, 1991, p. 39–43).[12] In the occupational setting, as in the larger field of environmental health, history leaves no doubt that a focus on genetic susceptibilities may be used to shift the focus of public health intervention and policy to the individual level and away from larger social, economic, and political factors that are fundamental to the production of human health and illness (Link & Phelan, 1995).

A critical issue, then, will be how public health genetics enables and enacts intervention approaches. Does responsibility for ameliorating risk fall squarely on the individual? For example, are individuals who are identified as particularly susceptible to developing adverse outcomes following exposure to toxic substances simply barred from participating in workplaces wherein such substances are present? (Draper, 1991) Or, will public health advocates use the vulnerability of some proportion of the population to what have been considered ''safe'' levels of such substances to demand a reevaluation of environmental health regulations? Likewise, will public health educators urge that individuals identified as ''at risk'' make lifestyle modifications to minimize their exposures—for example, encouraging people who are ''hypersusceptible'' to carcinogens in the soil (e.g., polynuclear aromatic hydrocarbons (PAHs)) not to garden in communities with high PAH soil concentrations? Or will they urge that the soil of these communities be made safe for even the most sensitive person to enjoy his/her garden?

Critics of public health, who conceptualize it as a disciplinary practice (Foucault, 1979), predict that genetic knowledge will be used primarily (if not exclusively) as a rationale for individualizing strategies that govern the life practices of individuals (and create a productive and docile population) by forcing them to assume ''responsibility for their health'' (Petersen & Lupton, 1996) and obscuring the larger social and economic forces that shape the life chances of individuals and communities (Pearce, 1996). Certainly, this is a possible application of knowledge of individual genetic susceptibilities to environmental exposure. Genetic screening is already promoted as a means of identifying individuals who might be ''motivated to take special steps, beyond those taken to protect everyone'' (Omenn, 1991), a construction of ''risk'' that has the potential elide a critical examination of the social, political, and economic processes that create environmental exposures. For example, a recent book chapter on genetic screening promotes chemoprevention for women who may have an increased susceptibility to breast cancer but never addresses the environmental cofactors that interact with this susceptibility in disease etiology (Benkendorf, Peshkin, & Lerman, 2000, p. 371). Similarly, Conrad has noted that environmental exposures are often absent from the discourse surrounding the BRCA1 gene and breast and ovarian cancer risk:

> BRCA1 is a tumor suppressor gene. Most women are born with two copies of the gene and have no problem. Those with the inherited BRCA1 mutation have only a single copy. A single copy is sufficient for tumor suppression until, for some reason, it is

[12] At the same time, there are some conditions, such as susceptibility to occupational chronic beryllium disease, for which this assumption seems to be true and for which susceptibility screening might prove to be an important aspect of protecting worker's health (Bartell et al., 2000; Rossman, 2001).

> "knocked out" and a woman is left without the gene. Then she becomes extremely vulnerable to breast or ovarian cancer. The lack of the BRCA1 allele is seen as the cause of breast cancer. Yet, could we not ask, what caused the second copy of the gene to get knocked out? Could not environmental risk factors like a woman's past exposure to radiation be critical in understanding how the mutation leads to cancer? (Conrad, 1999, p. 235)

To focus only on the "internal environment" (Conrad, 1999, p. 233) as if there is a standard external environment in which all genes express themselves, is to risk obscuring environmental conditions and practices that are critical to human health and illness.

Localizing biologies

However, there is also another set of potentialities within contemporary studies of gene–environment interaction. First, the particular foci of studies of gene–environment interaction suggest that, for many susceptibilities, individualized approaches will not provide a feasible basis for public health policy. Unlike many of the relatively rare genetic conditions that have made headlines in the past 10 years (e.g., those associated with Huntington's Disease or the BRCA genes), many of the individual genetic susceptibilities studied by environmental geneticists are common; some are estimated to be present in 20–60% of the US population (Perera, 1997; Perera & Weinstein, 1999; Weincke, 1999). That is to say, in relation to certain chemicals, it is likely that a large proportion, or even a majority, of the population is "high risk" or "hypersusceptible".

Second, environmental genetics tends to focus on susceptibility traits that have *low penetrance*, that is, the presence of an allele itself confers a relatively low statistical risk of developing a condition:

> Alleles of high penetrance are associated with clear and direct phenotypic implications, in many different individuals and in a wide variety of environments… genetic polymorphisms [of low penetrance] are believed to play some role in disease or disease susceptibility, but only in conjunction with other genetic components and/or environmental exposures" (Sharp & Barrett, 1999, p. 177)

Identifying genes with low penetrance therefore necessarily leads to a complicated set of questions about environmental cofactors and gene–environment interactions in disease etiology and progression. The incorporation of epidemiologic models has enabled an expansion of environmental genetic research beyond the pharmacogenetic model, as case-control studies allow the simultaneous examination of both gene–gene and gene–environment interaction (NIEHS, 1993).

Third, the etiologies of the multi-factorial diseases that comprise the growing focus of environmental genetics are complex and involve multiple genes and environmental cofactors, often over long periods of time: "An individual who possesses a polymorphism in an allele of low penetrance may not develop the associated illness, though he or she may be substantially more likely to develop the disease *if particular environmental exposures are present*" (Sharp & Barrett, 1999, p. 177, emphasis added; Sharp & Barrett, 2000). For example, genetic factors themselves are thought to explain only about 5% of all cancers: "the remainder can be attributed to external, 'environmental' factors that act in conjunction with both genetic and acquired susceptibility" (Perera, 1997, p. 1068). This affirms the importance of the focus of contemporary public health genetics on gene–environment *interaction:* "it has become apparent that networks of genes—perhaps hundreds of genes and their products interacting with environmental stressors—are required for disease manifestation" (Singh-Gasson et al., 1999). A focus on interaction, in turn, necessarily directs attention to the historical and geographical situatedness of the production of health and illness.

Within an environmental genetic framework, individuals and subpopulations—whether they be of greater or lesser or "average" susceptibility—are susceptible *in places*. This suggests that if public health genetics maintains a focus on gene–environment *interaction*, its conceptualizations of susceptibility *may* transcend the reductionism and individualism of other instantiations of genetic discourse. Indeed, environmental genetic knowledge and technologies could be used to increase the visibility of the lives and practices of individuals, families, and communities (e.g., income, discrimination, nutrition, work, gardening, recreation, etc.) that constitute both the fundamental and the proximal causes of health status (Link & Phelan, 1995). The study of gene–environment interaction locates risk both outside of the body, as well as within the genes. Indeed, in the terminology of the "epidemiological triangle" of "agent, host, and environment" (Lillienfield & Stolley, 1994, pp. 36–37), analyses of gene–environment interactions reaffirm the importance of considering the "host" within the context of the environment (at all times and over time). For example, there are specific genetic variations—NAT1 and NAT2—which have been associated with increased rates of urinary bladder cancer in men and also with post-menopausal breast cancer in Taiwan. However, *these polymorphisms are only consequential in specific environments*: "Individual risks associated with NAT1 and NAT2 genotypes are small, but they increase when considered in conjunction with other susceptibility genes and/or… exposures" (Hein et al., 2000, p. 68). Studies of the susceptibilities associated with the NAT1 and NAT2 genotypes have

highlighted the interaction between genetic and environmental "risks".

There is some evidence that public health research and practice is increasingly attentive to place-specific aspects of susceptibility. For example, studies of the effects of lead exposure on children now examine both the physical characteristics of the environment and the ways in which children live and play in particular places where exposure may occur (Landrigan & Carlson, 1995). Cancer increasingly is seen as an environmentally dependent condition, even in the presence of genetic predisposition: "Most cancers result from the variable interaction of environmental influences acting on a broad range of host susceptibilities" (Mulvihill & Tulinius, 1987, p. 337). For example, people who carry a genetic polymorphism known as CYP2D6 appear to have an increased susceptibility to lung cancer, *if they are exposed to tobacco smoke* (Christiani, 1996). Contemporary research also focuses on wide range of differences in response to environmental agents: "differences in responsiveness, or susceptibility, differences in past and present exposures to related and unrelated toxicants, and differences related to age, gender, lifestyle, or genetic predisposition" (Albers, 1997). Some environmental genetic researchers conceptualize identifying susceptibility genes and including them in epidemiologic studies as one means to produce powerful new tools for detecting environmental hazards, thereby inverting the emphasis on the internal environment (NIEHS, 1993). The environment thereby may be made visible even in environmental genetic studies of individual susceptibility, insofar as these susceptibilities are located in particular places.

Again, these considerations necessarily focus public health research and practice in the places where people live, work, and play (Di Chiro, 1995) and emphasize that "to understand the causation of disease in a population, it is essential to understand the historical and social context and to emphasize the importance of diversity and local knowledge…" (Pearce 1996, p. 682). Focusing on such gene–environment interactions emplaces environmental health research, increasing its potential to address the lived conditions of real individuals and allowing examination of the processes through which culture and biology form a locally and historically situated dialectic (Lock, 1993). Observers have long noted that universal or "placeless" "safety standards" or "safety procedures" that are insensitive to local conditions may create and/or may legitimate unjust and unhealthy outcomes (Yearley, 1995, p. 469). Place-sensitive analyses are critical to efforts to protect human health, as research that addresses local conditions and practices is more likely to identify risk management practices that will work in local settings. Given such possibilities, it is particularly interesting that research into gene–environment interaction may be used *both* to make a growing proportion of the population aware of their vulnerabilities to toxic substances and to provide a growing proportion of the population with information about the types of hazardous substances to which they have been exposed.

Places in bodies: towards a molecular archeology of exposure and effect

If the first major focus of the study of gene–environment interaction is on individual and subpopulation susceptibility, the second major focus of the study of gene–environment interaction genetics is the assessment and explication of the effects of environmental exposures within human bodies. This knowledge is produced primarily in the fields of molecular epidemiology and toxicogenomics. The conceptual frameworks and technologies of these types of inquiry are being designed for use in environmental health risk assessment and policy (Christiani, 1996), making them of critical import to public health.

Opening the black box of the human body

Molecular epidemiology and toxicogenomics both offer means for establishing the occurrence of past exposures, reconstructing the doses received from past exposures through the identification of changes at the molecular level, and, in some cases, assessing their preclinical effects (Schulte & Perera, 1993, p. 6; Kreps et al., 1997). Within molecular epidemiology, molecular biomarkers are used to identify the effects of exposures prior to the manifestation of clinical symptoms and to assess any genetic susceptibilities that interact with the exposure (Perera & Weinstein, 1999). Toxicogenomic research uses DNA microarray technology to elucidate how the genome of an organism is involved in biological responses to environmental toxicants (Iannaccone, 2001).

Molecular epidemiology and toxicogenomics have the potential to transform public health regulation, policy-making and litigation (Christiani, 1996; Christiani, Sharp, Collman, & Suk, 2001; Warhurst, 2000; Tesh, 2000). Indeed, from the earliest days of these research practices, researchers have been aware of the potential applications of their work far beyond the walls of the laboratory (Shostak, 2001). For example, molecular epidemiologists point to the potential of biomarkers to provide a more reliable and valid empirical basis for environmental health regulation, priority-setting, and policy making: "the goal of such research is practical: to apply relevant and valid biomarkers… and to incorporate the resulting information into public health actions" (Christiani, 1996, p. 921). Similarly, a primary goal of toxicogenomic research is to identify toxicant

class-specific profiles or "signatures" of expressed genes that correspond to particular types of environmental exposures. The signature of a new chemical could then be compared to those of other chemicals known to be either "safe" or "dangerous" to human health (Iannaccone, 2001). Advocates stress that toxicogenomics may be more precise than other forms of assessment, as it does not involve extrapolation from laboratory animal data (Lovett, 2000; NIEHS, 2000a; Society of Toxicology, 2001). Moreover, the introduction of toxicogenomics may also contribute to the rapid assessment of greater numbers of chemicals, as it is faster than traditional toxicological techniques (Lovett, 2000).

In addition to accelerating the risk assessment process, such "signatures" could serve as molecular biomarkers (NIEHS, 2000b) and could be used to establish new scientific and juridical norms for ascertaining an environmental exposure and its effects. In toxic torts, a plaintiff's claim is scientifically strongest when an illness is a "signature disease", that is, when it is uniquely associated with a particular toxic substance (Jasanoff, 1995, p. 119).[13] The identification of "signature" molecular events could similarly become the norm for evaluating claims regarding the effects of exposure. For example, some tumor suppressor genes, such as the "p53" gene, appear to mutate in a predictable fashion, depending on the particular carcinogen to which they have been exposed:

> …chemicals and physical carcinogens leave footprints of their activities because of the base changes they induce… knowledge of the pattern mutations found in genes commonly mutated in human cancer, such as the p53 tumor suppressor gene, allows for predictions to be made on the likelihood of an exogenous DNA-damaging agent being involved" (Jones et al., 1991)

Analysis of how p53 has mutated may eventually allow one to tell whether a given cancer resulted from an environmental carcinogen or an endogenous mutation (Hussain & Harris, 1998).

Some environmental advocacy groups are optimistic about these possibilities. For example, in their position paper "The Crisis in Chemicals", the UK-based environmental group Friends of the Earth predicts that "the biomedical revolution" will result in an increased ability to identify and measure the damaging effects of exposures and, as a result, affected individuals "will be better able to mount liability cases" (Warhurst, 2000, p. 42). They also warn that "industry will be faced by people, armed with scientific evidence… who can show damage from exposure to chemicals" (Warhurst, 2000, p. 9). Clearly, this technology could also be useful to "industry", insofar as the absence of such "fingerprints" or "signatures" functioned to discredit the claims of exposed individuals and communities.

Molecular biomarkers are already being used in local struggles over the effects of low-level, chronic exposures to toxic substances. In January 2000, the Agency for Toxic Substances and Disease Registry (ATSDR) reported that it had found a higher than "normal" level of chromosomal abnormalities and genetic polymorphisms among 58 residents of Midway Village, CA (Pence, 2000a, b). The federal study was undertaken after a more than a decade of activism by residents of the Midway Village who allege that PAHs in the soil beneath their housing project are responsible for the nosebleeds, respiratory illnesses, nausea, rashes, infertility, memory loss, miscarriages, and cancers they experience. The Midway Village residents who submitted samples for analysis hoped that it would link the PAH exposure to their maladies, thereby strengthening their claim that they've been damaged and made sick by the exposures and should be relocated and compensated for their medical expenses (Pence, 2000a). Scientific assessment of the report's findings was cautious (not in the least because a "normal" level of chromosomal abnormalities and genetic polymorphisms was never defined in the report (Pence, 2000b)). However, the initial political response to the report was less equivocal. The US EPA urged the state regulators to retest the soil around Midway Village (Pence, 2000b). The district's legislator requested the state Environmental Protection Agency's Department of Toxic Substances Control convene to take testimony from toxicologists and Midway Village Experts and stated that his staff would investigate the possibility of relocating residents (Pence, 2000b). At the time of this writing, few relocations have occurred. Nonetheless, the Midway Village controversy marks the introduction of molecular biomarker testing into environmental health controversies.

(Re)Interpreting molecular events

These modes of knowledge production about environmental health present a paradoxical form of biomedicalization (Clarke et al., 2000). On the one hand, molecular epidemiology and toxicogenomics represent the extension of bioscientific means of knowing the body and determining its "norms". The epistemology of environmental genetics is moored in expert knowledge

[13] At the heart of any toxic tort action is the claim that a particular chemical substance or combination of substances has "caused" injury to the plaintiff. To make a persuasive case for compensation for injuries, therefore, the plaintiff must succeed in each of the following actions: (1) identify the harmful substance; (2) trace the pathway of exposure; (3) demonstrate that the exposure occurred at levels at which harm can result; (4) establish that the identified agent can cause injuries of the kind complained of; (5) rule out other possible causes of this injury (Jasanoff, 1995, p. 119).

systems and sophisticated molecular technologies of visibility and normalization (Foucault, 1978/1990, p. 144). The lived experiences of exposed individuals and/or communities may be effaced in such research, as the goal of molecular biological and toxicogenomic techniques is to make visible hazards that are often imperceptible to those who are exposed to them. Indeed, these new techniques for making exposure(s) visible shift the biomedical, legal, and regulatory focus from the lived experience of health or illness of a person to his/her genes, gene products (e.g., proteins and enzymes), and any changes therein. They assume that the knowledge of environmental hazards and their consequences is contingent upon "the 'sensory organs of science'—*theories, experiments, measuring instruments—in order to become visible or interpretable… at all*" (Beck, 1992, p. 27, emphasis in original). These assumptions may entail the minimizing and/or invalidating of human experiences of environments, of embodiment, and of health and illness.

However, at the same time, the focus of molecular biomarker and toxicogenomic research on the *interaction* between genes and environments also provides a means of highlighting the social significance of what the fields of biomedicine and public health might otherwise interpret as purely pathological signs (Scheper-Hughes & Lock, 1991). A vast proportion of the internal conditions made visible by molecular biomarker and toxicogenomic research practices are produced by the interaction between genes and environments; they are the result of the location of people *in particular places*. The location of individuals and communities in these varied places is socially and politically contingent. For example, African-Americans and Latinos have chances of living within a mile of a hazardous waste facility that are approximately four and a half times greater than that of Whites (Mohai & Bryant, 1992). Therefore, the biological products of such emplacements must be understood as simultaneously biological and social, cultural, and political facts (Lock, 1993; Scheper-Hughes & Lock, 1991). Like illness itself, markers of environmental insult are a 'message in the bottle' through which the body registers the effects of "the over determined way we live, work, and respond to or ignore each other's needs" (Scheper-Hughes & Lock, 1991, p. 415). Such marks of environmental exposures make visible the pathways between internal and external environments and remind us of the interpenetration of what is within and outside of our bodies. They also have the potential to call biomedical, public health and popular attention to the unequal distribution of environmental exposures and their effects.

In making exposure(s) visible at the molecular level, environmental genetics may transform both bioscientific and popular understanding of the relationship between bodies and environments. In the absence of relatively anomalous, disastrous and dramatic environmental exposure (e.g., the reactor fire at the Chernobyl power plant), most "illness in Western society is viewed from an 'internalizing perspective'. The relevant causes are immediate and localized within the body…" (Young, 1976, p. 148; Kroll-Smith & Floyd, 1997). However, environmental genetics highlights the interpenetration of bodies and places. The environmental genetic body is porous; it absorbs what it touches in the air, soil, and water and is changed at the molecular and morphological level by these absorptions. The body itself becomes a molecular archeological site revealing the past history of exposures and potential future harms (Proctor, 1995, p. 235), both containing and being transformed by the many places in which it has been situated. These marks left in bodies provide a map of the consequences of geographies of power (Massey & Jess, 1995, p. 69) both inside and outside of the body.

Conclusion

The hybrid, combinatory fields of inquiry that have been made possible by emergent molecular techniques and genetic/genomic knowledge—genetic epidemiology, molecular epidemiology and toxicogenomics—are reshaping definitions of both genetic and environmental risks and highlighting the importance of gene–environment interaction in the production of human health and illness. With these redefinitions comes the possibility of a reorientation of the field of public health towards genetic/genomic knowledge, technologies, and practices.

My analysis suggests that the future of the field of public health will be shaped, in large part, by whether it utilizes emergent genetic/genomic techniques to make more or less *visible* genetic risks, environmental risks, and/or processes of gene–environment interaction in the production of human health and illness. A "geneticization" (Lippman, 1992) of public health could lead to an entrenchment of genetic reductionism and individualized definitions of "risk" as the core orientation of the field. Public health policies and programs, then, would emphasize individual responsibility for health and intervention strategies (e.g., clinical and lifestyle modifications to manage genetic susceptibilities) which obfuscate the varied social, political, cultural, and economic factors that are the prerequisites of many environmental exposures and gene–environment interactions. In contrast, the field of public health could take advantage of the incorporation of genetic knowledge and molecular techniques as an opportunity for the discipline to attend to the production of "local biologies", health, and illness by attending to historically and geographically situated dialectics of biology and culture (Lock, 1993). If this occurs, public health policies and practices would be increasingly oriented to the social, political, and economic relations that enable

environmental exposures, gene–environment interactions, and their effects. While these two cases are somewhat oversimplified for the purposes of comparison, this paper has demonstrated that the growing interest in and institutionalization of genetics and genomics in public health has the potential to shape widely divergent futures for the field.

As such, the field of public health is located at the cusp of an "event": "…[an] emergence of forms… catalyz[ing] previously existing actors, things, temporalities, or spatialities into a new mode of existence, a new assemblage, one that makes things work in a different manner and produces and instantiates new capacities" (Rabinow 1999, p. 180). This is not the first such "event" in the history of public health. Indeed, many observers of the emergence of molecular techniques in public health research have compared it to the advent of "germ theory" during the 19th century (Loomis & Wing, 1990; Susser, 1998; Susser & Susser, 1996a, b; Vandenbroucke, 1988). This historical comparison has been used by some to argue that, like miasma theory of disease, which preceded germ theory and, over time, was largely effaced by it, social and environmental explanations of disease (and associated interventions) will wane as genetic/genomic practices become prominent aspects of public health practice (Vandenbroucke, 1988). However, more commonly, these authors draw on the history of public health to emphasize that neither miasma theory nor germ theory could effectively address the full range of etiologies important to public health policy and practice. Therefore, they suggest, neither a focus solely at the molecular nor at the environmental level will be able to provide an adequate basis for the field of public health (Loomis & Wing, 1990; Susser, 1998; Susser & Susser, 1996a, b).

The introduction of genetic and genomic technologies into the field of public health opens up a wide range of possible futures that will not be determined by the technologies themselves but how they and the knowledge they both produce and reify (Latour & Woolgar, 1979/1987) are used. As I have demonstrated, while the history of environmental genetics bears witness to its potential for genetic reductionism, determinism, and the individualization of risk, it also offers technologies and conceptual frameworks that could enable the field of public health to attend simultaneously to causal pathways located in society, culture, economics and at the molecular level. The new molecular technologies need not obscure the situatedness of bodies (whatever their relative level of susceptibility); they *can* support efforts to better understand the pathways between the interior and exterior of the human body and the ways in which they enable and affect the production of human health and illness. Which of the many potentials of genetic and genomic technologies—and of the field of public health—will be realized in the coming years is a question that deserves continued scholarsip and debate.

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