“Bodies Count,” and Body Counts: Social Epidemiology and Embodying Inequality

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INTRODUCTION

Bodies count. In epidemiology, this statement would appear to be a core proposition, for it is by counting people—in varying states of health, disease, and disability, the alive and the dead—that we derive our estimates of population rates and risks of morbidity and mortality. But bodies count for more than this, for, in their manifest form—in height, weight, physique, and overall appearance (including posture and disfigurement)—they provide vivid evidence of how we literally embody the world in which we live, thereby producing population patterns of health, disease, disability, and death (1–5). Readily identifiable to the naked eye, these aspects of our being not only are predictive of future health outcomes but also tell of our conjoined social and biologic origins and trajectories.

In this paper, we argue that the field of social epidemiology has much to gain by considering the construct and reality of “embodiment,” and hence the causes and consequences of bodily constitution (1–5). At issue is how social influences become literally embodied into physio-anatomic characteristics that influence health and become expressed in societal disparities in health. In our view, a concern with differences in macroscopic physiology—or what Laurell has tellingly termed “class physiognomies” (6, p. 1184)—usefully turns attention toward topics often overlooked by much contemporary social epidemiology in the United States and Europe, with its focus on neuroendocrine and behavioral responses to psychosocial stressors, posited both to be directly pathogenic and to increase susceptibility to other exogenous exposures (7–12).

While research mending the “mind/body” rift long characterizing biomedicine is important, it is equally critical to recognize that, in addition to being mindful, bodies also are literally corporeal. Asking the question as to whether bodily being can itself affect health in no way discounts that people’s behavioral, emotional, and cognitive responses to adverse (and beneficial) circumstances may influence somatic health (in ways that vary according to disease outcome); 2) psychosocial adversity impairs the quality of people’s lives, whether or not it harms somatic health; and 3) psychosocial stressors are socially patterned (with some groups being subject to unique stressors, e.g., racial discrimination) (5, 10, 12, 13). Rather, by emphasizing the literal nature of embodiment and refocusing on key aspects of what bodies do, we can potentially generate new insight into the ways societal conditions shape expression of biologic traits, population distributions of disease, and social inequalities in health.

EMBODIMENT AND BODIES: A BEGINNING

In the bones: the long-standing centrality of embodiment to epidemiology

Importantly, a concern with bodily characteristics is not new to epidemiology but rather dates back to the field’s modern origins in the early 19th century (14–16). In the 1820s, for example, Louis René Villermé (1782–1863) conducted pathbreaking research demonstrating that wealth was inversely associated with both risk of mortality in Parisian neighborhoods and short stature, illness, and deformities among young military conscripts (table 1) (14, 17, 18). He further observed that “qu’en generale, ches les personnes d’une petite stature ... les membres inferieurs sont plus courts que chez les personnes d’une grande taille” (“in general, among persons of small stature ... the lower limbs are much shorter than those of persons of great height”) (17, p. 351). The implication, explicitly recognized by Villermé, was that body size, body proportions, and longevity, far from being fixed, bore the imprint of economic conditions and could be affected by government policies (14, 17, 18).
Recognition of links between bodily constitution and the body politic likewise gained attention in England in the 1830s and 1840s, during the throes of the Industrial Revolution (15, 16, 19, 20). In one of the era’s classic texts, The Condition of the Working Class in England in 1844 (21), Friedrich Engels (1820–1895) vividly described the bodily impact of the workers’ abysmal working conditions, food, housing, and inadequate medical care, arguing:

All of these adverse factors combine to undermine the health of the workers. Very few strong, well-built, healthy people are to be found among them ... They are for the most part, weak, thin and pale. ... Their weakened bodies are in no condition to withstand illness and whenever infection is abroad they fall victims to it. Consequently they age prematurely and die young. This is proved by the available statistics of death rates (21, pp. 118–119).

Concerned with the lifelong consequences of inequity, he concisely stated, “Common observation shows how the sufferings of childhood are indelibly stamped on the adults” (21, p. 115). He likewise recounted how the factory hands he met in Manchester were worn out at age 40 and looked 10 years older, while the prosperous classes preserved their appearance well—if they could restrain their desire to drink excessively. Anticipating the conclusions of the late 20th century Nobel-prize-winning economist Robert Fogel (22) but speaking from a different perspective, Engels argued that the capitalists were in fact acting against their interests in allowing the working class to become so enfeebled, since it lessened their productivity.

Despite this early concern with the impact of social disadvantage on bodies, the topic did not become a mainstay of the developing field of epidemiology. Contributing to its marginalization was the rise of germ theory in the 1860s, leading to a focus on specific pathogens rather than broader social reform (15, 19, 23). Also germane was the rise of Social Darwinism, whose influential adherents held that innate inferiority, not social injustice, was the underlying cause of ill health, bodily enfeeblement, and poverty, whether in Europe, the United States, or colonies abroad (15, 24, 25).

With the onset of the economic depression of the 1930s, however, researchers again began investigating links between economic deprivation, bodily characteristics, and health. This was the theme, for example, of the pivotal 1933 US report “Health and Environment,” prepared by Edgar Sydenstricker (1881–1936) for the President’s Research Committee on Social Trends (26). Likewise, a representative British textbook of public health in 1939 matter-of-factly presented a data-based narrative of how social conditions are literally written into, and onto, the bodies of the poor and those in unfavorable occupations (27). Work on “social physiology” (28) was central to the establishment of social medicine as a discipline in the 1940s (1, 20, 29), including the founding, in 1948, of the landmark Social Medicine Research Unit under the directorship of Professor Jerry Morris (b. 1910) (30). Rising economic prosperity after World War II, plus the Cold War, reduced interest, but in the aftermath of the social movements of the 1960s, the field again experienced growth (1, 16, 29)—especially after the publication in 1980 of the Black Report (31), to which Morris was a major contributor. Since then, social epidemiology has come into its own (1, 12), reintroducing social concerns into a research milieu permeated by assumptions of biomedical individualism (1, 32–34), including a tendency to study decontextualized biology and bodies and to reduce

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**TABLE 1. Villermé's data on socioeconomic gradients in Paris, France, for average annual mortality among the total population (1817–1821) and for height among military recruits (1816–1823), by a neighborhood measure of wealth (percentage of untaxed rents, with taxed rents being paid only by the wealthy)**

<table>
<thead>
<tr>
<th>Arrondissement (neighborhood)</th>
<th>Population in 1817</th>
<th>% untaxed rents</th>
<th>Average annual mortality in the total population</th>
<th>Average height (m) of military conscripts (young men aged 18–21 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 (wealthiest)</td>
<td>65,623</td>
<td>7</td>
<td>1 in 62</td>
<td>1,612.9</td>
</tr>
<tr>
<td>3</td>
<td>44,932</td>
<td>11</td>
<td>1 in 60</td>
<td>1,666.7</td>
</tr>
<tr>
<td>1</td>
<td>52,421</td>
<td>11</td>
<td>1 in 60</td>
<td>1,666.7</td>
</tr>
<tr>
<td>4</td>
<td>46,624</td>
<td>15</td>
<td>1 in 58</td>
<td>1,724.1</td>
</tr>
<tr>
<td>11</td>
<td>51,766</td>
<td>19</td>
<td>1 in 51</td>
<td>1,960.8</td>
</tr>
<tr>
<td>6</td>
<td>72,682</td>
<td>21</td>
<td>1 in 54</td>
<td>1,851.8</td>
</tr>
<tr>
<td>5</td>
<td>56,871</td>
<td>22</td>
<td>1 in 53</td>
<td>1,785.7</td>
</tr>
<tr>
<td>7</td>
<td>56,245</td>
<td>22</td>
<td>1 in 52</td>
<td>1,923.1</td>
</tr>
<tr>
<td>10</td>
<td>81,133</td>
<td>23</td>
<td>1 in 50</td>
<td>2,000.0</td>
</tr>
<tr>
<td>9</td>
<td>42,932</td>
<td>31</td>
<td>1 in 44</td>
<td>2,272.7</td>
</tr>
<tr>
<td>8</td>
<td>62,758</td>
<td>32</td>
<td>1 in 43</td>
<td>2,325.6</td>
</tr>
<tr>
<td>12 (poorest)</td>
<td>80,079</td>
<td>38</td>
<td>1 in 43</td>
<td>2,325.6</td>
</tr>
</tbody>
</table>

* Sources: Villermé (17, p. 370; 18, pp. 227 and 246).
† In the original table, data were presented only as proportions.
TABLE 2. Selected defining aspects of embodied humans, as jointly biologic organisms and social beings

<table>
<thead>
<tr>
<th>Body aspect</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reproduce: capacity to give rise to the next generation, whether asexually (typically the case for bacteria, the most common form of life on Earth) or sexually (involving contribution of genetic information from both biologic parents), even if not every organism itself reproduces.</td>
<td></td>
</tr>
<tr>
<td>Develop: life history change within an organism, involving generation of cellular diversity, differentiation, and morphogenesis.</td>
<td></td>
</tr>
<tr>
<td>Grow: increase in physical size; in multicellular organisms, by processes involving regulation of cell division, addition of new cells (by mitosis), and deletion of extant cells (by apoptosis).</td>
<td></td>
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<tr>
<td>Interact: with other members of the species (in deme), with other organisms in the same ecosystem, and with the physical environment(s) in which the ecosystem is located, so as to meet basic needs for food, safety, pleasure, procreation, and rest, including avoiding noxious stimuli, seeking life-sustaining environs, and reproducing.</td>
<td></td>
</tr>
<tr>
<td>Exist in time and space: from birth to death, bodies exist (and, if mobile, move around) in spatially and temporally delimited ecosystems with geographically contingent patterns of temperature, climate (if on land), altitude, and diurnal change (periods of light and dark).</td>
<td></td>
</tr>
<tr>
<td>Evolve: given key criteria of reproduction, inheritance, and genetic variation, evolution involves emergence of new traits and new species, reflecting possibilities enabled and constrained through historically contingent biologic processes that are “selected” or “filtered” in a context of altered environments.</td>
<td></td>
</tr>
<tr>
<td>Societal context: live life in the society (or societies) of which one is a member, vis-à-vis one’s historical period, economy, political and legal system, technology, and social, cultural, civil, economic, and political rights, resources, relationships, and institutions, plus one’s location in the global economy and global institutions of governance.</td>
<td></td>
</tr>
<tr>
<td>Social position: be born into and/or raised in, and later form, a specific type of family and/or household, vis-à-vis social relations of class, gender, sexuality, race/ethnicity, and other salient social divisions premised on power, property, and social inequality.</td>
<td></td>
</tr>
<tr>
<td>Social production: engage in socially delimited processes, relationships, and institutions, contingent on one’s social position, involving production, exchange, distribution, and consumption of goods and services, as well as ideas and information, with differential distribution and intergenerational transfer of assets, typically enforced by law.</td>
<td></td>
</tr>
<tr>
<td>Social consumption: engage in socially delimited processes, relationships, and institutions, contingent on one’s social position, involving acquisition and consumption of goods, services, and ideas and information required to meet basic needs (for physical survival) and social needs (for a socially meaningful life).</td>
<td></td>
</tr>
<tr>
<td>Social reproduction: engage in socially delimited processes that sustain, modify, or replace societal structures, relationships, and institutions.</td>
<td></td>
</tr>
</tbody>
</table>

problems of population health to a matter of individual lifestyles plus faulty genes. It was in this context that, in 1994, “embodiment” was proposed as a fundamental construct critical to the theoretical and substantive development of social epidemiology (3).

Bringing the body back, in context

To work with the construct of “embodiment,” we need first to consider several key defining and inseparable aspects of our lives as simultaneously social beings and biologic organisms. These aspects, on which we elaborate in table 2, draw on well-developed notions of the individual body, the social body, and the body politic (1, 35–44) and include the following: 1) for biologic organisms: reproduce, develop, grow, interact, exist in time and space, and evolve; 2) for social beings: societal context, social position, social production, social consumption, and social reproduction. All of these biologic and social aspects necessarily are integral to human existence and thus are inherent characteristics of each and every person.

Fresh thinking about bodies and embodiment in the biologic and social sciences enriches this picture. New developments in research connecting evolutionary and developmental biology have brought attention to the profound importance of hierarchically embedded, multi-level, and historically contingent biologic processes in both individual development and speciation (37–39). From this vantage point, biologic beings and species are constituted through their engaged interaction with biotic and abiotic environments they in part construct and embody, in the context of dynamic ecologic systems. This work stands in contrast to persistent ultra-reductionist accounts of the proverbial “selfish gene” (45) allegedly dictating, via a “master code,” a set of processes geared solely toward propagating gene replication. Shifting concern to determinants of gene expression, not simply gene frequency, this new perspective likewise calls into question the popular notion of “gene-environment interaction, since “genes” do not interact with environments—only organisms do, with consequences for gene regulation and expression (37–41, 46).

Similarly, within the social sciences, recent scholarship has drawn attention to social, cultural, economic, and political aspects of embodiment (35, 36, 42, 43). Focused on the particularly human societal issues of power, property, and politics, it explores the body as both a “cultural artifact” and a political entity. At issue are the myriad ways in which social beliefs and values, as expressed through legal codes, religious and civic institutions, marketing, and so forth, affect how people inhabit their bodies in their everyday lives. Topics especially relevant to health include consumption of food, sexual practices and identities, use of psychoactive...
substances, use and experience of violence, and the impacts of not only economic deprivation and social oppression but also affluence and privilege. Challenging essentialist and individualistic accounts of human behavior, including biologically deterministic analyses proffered by sociobiologists and evolutionary psychologists (47–49), this new scholarship emphasizes how actualization and suppression of people’s agency, that is, their ability to act within their bodies, intimately depends on socially structured opportunities for, and threats to, their well-being.

The notion of “embodiment” accordingly embraces biologic processes while avoiding the trap of equating “biologic” with “innate,” and simultaneously embraces social processes without assuming the soma is governed solely by the psyche. In the case of social inequalities in health, it likewise presumes that observed differences reflect biologic expressions of social inequality. Sapolsky’s cautionary tale bears repeating: Study adrenal glands only among the cadavers of the poor, long since hypertrophied due to excess stimulation of cortisol secretion, and—as occurred in the early 20th century—the wealthy will be diagnosed with adrenal deficiency disorders; simplistic divisions between the social and the biologic will not suffice (50, 51).

EMBODYING INEQUALITY: EXAMPLES

To demonstrate the importance of embodiment for social epidemiology, we now explore five examples, chosen in relation to the five key biologic aspects of organisms described above. Each example connects these core characteristics of biologic organisms to macroscopic aspects of bodies and health. In each case, we present brief descriptions of likely pathways of embodiment and subsequent health consequences of these embodied characteristics.

Reproduce: the example of low birth weight

Bodies reproduce. Because embodiment arguably begins with conception (intergenerational effects notwithstanding), it should not be surprising that birth weight is one macroscopic characteristic that has long been associated with socioeconomic conditions (15, 26, 31, 52–55). Low birth weight (<2,500 g) among births of ≥37 weeks’ gestation usually reflects intrauterine growth retardation, which results in infants who are small for gestational age and for whom mortality risk is high during the postneonatal period (from day 28 to the first birthday) (53, 54). By contrast, low birth weight among premature births (<37 weeks’ gestation) greatly elevates risk of neonatal mortality (<28 days of age), whether or not the premature infant is the appropriate size for his or her gestational age (53, 54).

Low birth weight as an embodied expression of social inequality reflects socially patterned exposures (during and prior to the pregnancy) to such factors as maternal malnutrition, toxic substances (e.g., lead), smoking, infections, domestic violence, racial discrimination, economic adversity in neighborhoods, and inadequate medical and dental care (e.g., lack of dental care can result in persistent periodontal infection; inadequate prenatal care can preclude screening and treatment for bacterial vaginosis) (52–60). Consequences of low birth weight for persons surviving past infancy include greater overall mortality risk in middle age, especially via association with an elevated risk of cardiovascular disease (52, 61). Mortality from some causes of death, particularly from some non-smoking-related cancers, shows a direct association with birth weight, reflecting the etiologic specificity of relations between embodied social experience and disease outcomes (61).

Finally, since birth weight is clearly dependent on the social circumstances, nutritional status, and health of mothers, there are potentially important intergenerational influences on health in later adulthood, such that the cardiovascular disease risk of older adults may reflect the social conditions prevailing during the early life of their mothers many decades before. Illustrating this, research shows that nutritional status and growth during mothers’ infancy and childhood influences the birth weight (and hence future adulthood health) of their offspring (62, 63).

Develop: the example of age at menarche

Bodies develop. Age at menarche, a macroscopic marker of biologic development, is both historically contingent and socially patterned, suggesting that it too reflects processes of embodiment. Data from Europe and the United States indicate that since the mid-19th century, the average age at menarche has declined considerably, from about age 16 years to about age 12 years (64, 65). Moreover, until recently, within any given country, later age at menarche was associated with socioeconomic deprivation, while earlier age at menarche was typically associated with affluence (64–67). However, newer evidence suggests that obesity in childhood, which is often associated with poverty in high-income countries, is associated with earlier age at menarche (68); participation in intense athletic activity, by contrast, is associated with delayed onset (64).

Pathways of embodiment relevant to the social patterning of age at menarche include malnutrition (both undernutrition and obesity), childhood infection arising from household crowding and inadequate sanitation, and birth weight combined with rate of early childhood growth (64–68). The association of undernutrition and infection with delayed menarche is hypothesized to be due to slowed growth and development (64–67) and possibly also to fewer ovarian follicles (69). Evidence remains mixed as to whether early obesity is causally related to early menarche or whether their association arises because both result from an underlying endocrine disorder (68, 70). Other hypotheses posit that age at menarche is “programmed” in utero, albeit subject to modification by weight gain in infancy and childhood (71, 72). To date, little data exist on whether exposure to putative endocrine disrupters could also contribute to the social, including geographic, patterning of age at menarche (73).

Onset of menstruation has social as well as biologic salience for both adolescent and adult health. Socially, it has often served as a marker for “womanhood,” with considerable symbolic and practical value, whether revered as a source of strength or reviled as a form of “pollution” (74, 75). Setting the lower bound for possible age at first preg-
nancy, age at menarche can also be predictive of disease risk later in life (e.g., risk of breast cancer) (76, 77).

**Grow: the example of adult height**

Bodies grow. As was discussed above, body height has long been taken to be an index of social conditions (4, 14, 17, 64). The same applies to body proportions, with adversity being associated with shorter leg length and a lower leg length:trunk length ratio (78), as noted by Villermé nearly two centuries ago (17).

Final stature, a macroscopic characteristic, is influenced by both environmental and genetic factors, with the latter being reflected by the correlation between midparental height and the height of offspring (79). In economically affluent countries, the heights of fathers and mothers are equally strongly correlated with the heights of their offspring, suggesting a limited influence of intrauterine factors. However, in economically impoverished countries with a high prevalence of malnutrition among women across the life course, limited data suggest that the contribution of intrauterine growth is greater (79). For decades it was suggested that the lower height of the Japanese as compared with Europeans was genetic; however, second- and third-generation Japanese migrants to the United States, and now Japanese people living in Japan, have come to attain heights akin to those of their European counterparts (65, 80).

Both final achieved stature and bodily proportions express the embodiment of nutrition and disease (especially infectious disease) in infancy and childhood (78–81), with risk of exposure being influenced by such social factors as household crowding, family size, birth order, general socioeconomic circumstances, and sociocultural aspects of diet (e.g., veganism). In extreme circumstances, psychosocial deprivation can attenuate growth (82, 83), although whether this ultimately works through nutrition or through direct endocrinologic influences is not well understood (84).

Height has both biologic and social consequences. Evidence indicates that robust inverse associations exist between height and mortality from chronic obstructive pulmonary disease, hemorrhagic stroke, and stomach cancer (85). In the case of coronary heart disease and ischemic stroke, inverse associations appear to depend on an interaction between restricted early growth and later-life risk factors, such as dietary saturated fat or obesity, and thus inverse associations between height and mortality from these conditions are not universally observed (86). Studies also provide evidence of positive associations between height and other types of mortality, especially several non-smoking-related cancers (including breast, prostate, and colorectal cancer, possibly through an insulin-like growth factor pathway) (87). Accentuating the link between height and economic well-being, studies also indicate that being tall—a socially desirable attribute—increases the likelihood of upward social mobility (88, 89).

**Exist in time and space: the example of *Helicobacter pylori* infection**

Bodies exist, and move, in time and space. Migrant studies reveal that people bring a mixture of physical, cultural, and social risks (or protections) with them. Bodies contain within them or on them other life forms that can influence their health trajectories. This is most evident with persistent infections that, once acquired, may remain unless deliberately or accidentally eradicated.

*H. pylori* infection, typically acquired during infancy and childhood, is a case in point, reflecting embodiment of transmission dynamics shaped by inadequate sanitation, household crowding, and poverty during early life (90). The infection is probably fecally-orally or orally-orally transmitted, with vomit and diarrhea being more efficient modes of transmission than excreta or expectorations from healthy persons (91)—thereby enhancing the likelihood, in a crowded household, of a sick child with diarrhea and vomiting infecting other children. Once acquired, *H. pylori* infection tends to be asymptomatic but long-lasting, and in later adulthood it increases the risk of peptic ulcer and stomach cancer morbidity and mortality. Consequently, migrants from high-prevalence countries bring with them not only the bacterium but also an increased risk of stomach cancer (92). The exact age of acquisition of *H. pylori* may influence disease outcome, with very early infection being related to gastric ulcer and later infection to duodenal ulcer.

**Interact: the example of early childhood physical and sexual abuse**

Bodies interact. These interactions can be beneficial or adverse, and among the latter is early childhood physical and sexual abuse. The World Health Organization estimates that approximately 20 percent of girls and 5–10 percent of boys, worldwide, have experienced childhood sexual abuse (93). Despite the long-standing and widespread nature of these problems (93–95), epidemiologic research on the incidence, determinants, and health consequences of early childhood abuse remains relatively new (96). Complicating research is serious underreporting due to legal implications, fear, and shame (93–96).

Implicating the embodiment of gender relations, girls are estimated to be 1.5–3 times more likely than boys to be targeted for childhood sexual abuse. Boys are more likely to be subjected to physical abuse, and men are most likely to be the perpetrators of sexual violence and of severe physical violence (93, pp. 64–67). Limited evidence suggests that physical violence against children may be more common in impoverished households and communities (93, pp. 67–67); cases occurring among the more affluent, conversely, may be kept out of public scrutiny (93–96). Regarding intergenerational effects, limited evidence supports the hypothesis that children subjected to abuse are more likely to commit abuse at a later age; however, the majority of adults abused as children do not commit child abuse, and many child abusers have no history of child abuse (93, 96).

Multiple outcomes have been linked to experiencing and embodying the harms of early childhood abuse. In addition to injuries caused directly by the violence (e.g., broken bones and teeth, bruises, torn rectums, scarred vaginas) (93, 96, 97), adverse effects can continue to play out across the life course. Subsequent harms include impaired mental health and risk of suicide, adverse use of psychoactive substances...
(alcohol, drugs, tobacco, etc.), eating disorders (anorexia, bulimia, obesity), increased likelihood of unsafe sex (and subsequent risk of human immunodeficiency virus infection and other sexually transmitted infections), and impaired somatic health, including reproductive health (e.g., early onset of perimenopause) (93–103). Limited evidence also suggests socioeconomic harm, as reflected in reduced earnings and greater risk of unemployment in adulthood (93).

EMBODYING INEQUALITY: IMPLICATIONS FOR POPULATION HEALTH

We have discussed various aspects of bodies that incorporate elements of social experience and that in turn influence health and social trajectory in a mutually constitutive fashion. One implication is that processes of embodiment should be reflected in a coherency in population distributions of health outcomes attributable to bodily habitus and reflecting embodiment of social conditions. A corollary is that secular changes in causes, rates, and distributions of diseases and death (15, 26, 104–106) are more likely to be driven by embodied consequences of changes in societal policies, material circumstances, and bodily constitution than by changes in psychosocial stressors per se (13)—unless an argument can be made that these have likewise changed over time.

To illustrate the utility of considering “embodiment” for understanding changes in population health, consider the macroscopic phenomenon of height in relation to a variety of health outcomes. One important observation is that overall mortality rates in Britain demonstrate cohort-specific declines from the mid-19th century on, with decreases being seen in subsequent cohorts born from midcentury onwards as they reached older ages (107–109). Notably, this pattern reflects trends in height (110), with calculations of attributable risk suggesting that increases in height can account for a reasonable proportion of the trend in overall adult mortality (111). Additional coherence is provided by patterns of association between height and cause-specific mortality (107–109). Conditions inversely associated with height—hemorrhagic stroke and stomach cancer—have declined as adult height has increased; comparable trends in chronic obstructive pulmonary disease are difficult to determine given the secular changes in smoking, a major etiologic factor in this disease. Other conditions contingently related to height—coronary heart disease and ischemic stroke—have shown increases and then falls, reflecting secular changes in other contributory factors. Conditions positively related to height—several of the non-smoking-related cancers—in turn have shown increases in incidence. Thus, there is some coherence between secular changes in height and population health.

However, for other aspects of bodies that we have examined, such as birth weight, a similar coherence is not seen. Considering evidence from the late 19th century through the late 20th century, there is no evidence of major changes in birth weight during the period when people who have experienced large changes in disease incidence were born (112). This anomaly, barely studied, invites important questions about why birth weight distribution is not changing when other aspects of embodied inequality are, despite identification of diverse social determinants of low birth weight in myriad investigations (53). Because working with the construct of “embodiment” encourages comparative and historical research on corporally connected outcomes, it can spark new questions relevant for etiologic research, as illustrated by concurrent consideration of height, mortality, and birth weight.

EMBODYING INEQUALITY: IMPLICATIONS FOR ETIOLOGIC RESEARCH

The notion of “embodiment” can prove useful to etiologic investigations in still other ways. Observational epidemiologic studies, for example, are prone to confounding, and the construct of “embodiment” highlights why socially patterned confounding factors are both likely and of especial concern. For example, a person is not one day a woman, another day Latina, another day heterosexual, another day a single mother, another day living in a relatively poor neighborhood, another day working as a data processor, and still another day caring for both a small child and aging parents. The body does not neatly partition these experiences. That said, such a woman’s risk of ergonomic injury may be most influenced by her work and by the demands of physically caring for others (113), while her risk of cardiovascular disease might be driven by her smoking to take breaks from these tasks (113–115)—as well as by her birth weight, age at menarche, height, and body mass index (61, 62) (also germane to risk of breast cancer) (116, 117). By contrast, gender relations (e.g., men’s having more sexual partners and not revealing this information) and policies affecting access to reproductive health services may be most relevant for her risk of cervical cancer (118–123). Thus, teasing out whether and which of these factors are relevant to the chosen health outcome under study requires conceptualizing confounding in relation to the embodied consequences of social position. Likewise, possibilities for socially patterned confounding are suggested by diverse embodied factors hypothesized to contribute to Black/White difference in high blood pressure, such as low birth weight, childhood growth, body mass index, lead exposure, and racial discrimination (46, 55, 124–136).

Recent well-defined examples of confounding arising through socially patterned correlations have included studies of the relation between hormone replacement therapy or consumption of various antioxidant vitamins and cardiovascular disease, with observational studies suggesting that these factors protect against cardiovascular disease but randomized controlled trials demonstrating no such protection (137). Figure 1 presents results from an observational epidemiologic study (138) and a randomized controlled trial (139), with precisely the same difference in plasma vitamin C levels being related to coronary heart disease risk in the two study designs (140). However, vitamin C intake is very strongly socially patterned, and in a large study of United Kingdom women it was demonstrated that not only were health behaviors (such as smoking, alcohol intake, and leisure-time exercise) and socioeconomic measures (such as occupational social class and being reliant on a state pension) related to vitamin C levels but so were height (taller women had higher vitamin C levels) and, within this var-
able, components of stature—with a greater leg length:trunk length ratio being associated with higher vitamin C levels (140). Thus, inclusion of embodied factors such as birth weight, offspring birth weight, height, components of stature, and infections acquired in childhood such as H. pylori can potentially aid in detection of the presence of otherwise difficult or impossible-to-measure confounding by factors acting right across the life course.

An alternative strategy for addressing such confounding is to consider those biologic aspects of our bodies that are little, if at all, related to socioeconomic factors: germ-line genetic constitution (141). Among groups with shared geographic origins and ancestry, for example, there is little evidence of systematic differences between the distributions of functional genetic variants by socioeconomic position, and hence little likelihood of confounding of associations between these genetic variants and disease outcomes by socially patterned behavioral or physiologic factors (142). Both the existence and the distribution of these polymorphisms are premised on yet another bodily aspect: the evolved ability to reproduce sexually. Genetic epidemiology is often seen as the antipathy of public-health epidemiology, but paradoxically it now offers a potentially valuable empirical solution to the problems of separating causal elements from confounding elements in the deeply associational world in which we live.

Mendelian randomization—the random assortment of genes from parents to offspring that occurs during gamete formation and conception—provides a method of assessing whether certain environmental exposures are causally related to a disease (142). The association between risk of a disease and a genetic variant that mimics the biologic link between a proposed exposure and a disease is not generally susceptible to the reverse causation or confounding that may distort interpretations of conventional observational studies. Several cases in which the phenotypic effects of polymorphisms are well-documented provide encouraging evidence of the explanatory power of Mendelian randomization (142).

The example of the homocysteine-coronary heart disease association illustrates the approach (142, 143). The association between homocysteine and coronary heart disease, which has been repeatedly reported in observational epidemiologic studies, is heavily confounded by factors such as smoking and blood pressure, which tend to be related to elevated homocysteine levels. Polymorphisms of the methylenetetrahydrofolate reductase (MTHFR) gene result in lifelong differences in levels of serum homocysteine. The MTHFR TT genotype is associated with increased homocysteine levels, so examination of its relation with coronary heart disease risk provides an unconfounded test of the hypothesis that homocysteine increases coronary heart disease risk. Indeed, the association between MTHFR genotype and coronary heart disease is mathematically close to that predicted by a combination of the strength of association between the MTHFR genotype and homocysteine and the strength of association between homocysteine and coronary heart disease (142). Parts a and b of figure 2 contrast the

![FIGURE 1. Estimated effects of a 15.7-µmol/liter increase in plasma vitamin C concentration on 5-year coronary heart disease mortality as estimated from the observational European Prospective Investigation into Cancer and Nutrition (EPIC) (138) and the randomized, controlled Heart Protection Study (139). Black squares, relative risk; horizontal bars, 95% confidence interval. EPIC m, age-adjusted EPIC data for men; EPIC m*, EPIC data for men adjusted for systolic blood pressure, cholesterol, body mass index, smoking, diabetes, and vitamin supplement use; EPIC w, age-adjusted EPIC data for women; EPIC w*, EPIC data for women adjusted for systolic blood pressure, cholesterol, body mass index, smoking, diabetes, and vitamin supplement use. Source: Lawlor et al. (140).](image1)

![FIGURE 2. Flow chart outlining the study of associations via (a) conventional epidemiologic approaches and (b) the "Mendelian randomization" paradigm, using the example of associations between plasma homocysteine level and coronary heart disease. TT and CC refer to genotypes of the methylenetetrahydrofolate reductase (MTHFR) gene. Source: Davey Smith et al. (143).](image2)
logic of the associations studied in conventional epidemiology and those investigated through utilization of the “Mendelian randomization” paradigm (143). Table 3 demonstrates that the principle behind Mendelian randomization holds in the $MTHFR \rightarrow$ homocysteine $\rightarrow$ coronary heart disease case. There is no confounding of genotype by social class or smoking but considerable confounding of measured blood homocysteine levels by these factors.

**CONCLUSION**

In conclusion, taking embodiment seriously has the potential to sharpen social epidemiologic research and enhance its ability to provide insights relevant to improving population health and reducing social disparities in health. It can do so, however, only insofar as we understand our embodied selves to be simultaneously and historically contingent social beings and biologic organisms. Just as the “normal” age at menarche has declined among wealthier nations, so too has average body size increased, indicating the malleability of the human form. Yet, signaling the limits of our biology, falling above or below a specified value may nevertheless have adverse health consequences, even as the location of these values—in relation to the curve’s center or tails—may change over time.

The construct of embodiment also invites us to consider how our bodies, each and every day, accumulate and integrate experiences and exposures structured by diverse yet commingled aspects of social position and inequality. It can assist us with thinking through, systematically, what any given investigation measures, what are likely to be important unmeasured covariates, and what are relevant time frames for obtaining meaningful measurements in relation to etiologic period. With such knowledge, we better understand the meaning and limitations of the analytical findings any given study generates, plus potentially spark new hypotheses. It thus becomes an important empirical question, not a philosophical principle, as to which types of inequality—any, some, none, or all, independently or in synergistic permutations—are etiologically relevant (144). By starting with embodiment, reading from societal context to our bodies and then back again to population health, we are likely to enhance our ability to take on the challenge of explaining current and changing patterns of population health and social inequalities in health—a challenge which, after all, constitutes a core test of our etiologic hypotheses (1–4).

We recognize that incorporating the construct and reality of embodiment in epidemiologic research is not a simple matter. The difficulties of obtaining apt longitudinal data across the life course, with good measures of health outcomes, bodily characteristics, social position, social exposures, and social context, are well known (2, 4, 12, 26, 31, 61). Nor are data enough. Equally critical is how we frame our hypotheses—so we can make the best use, with the least confounding, of both the available data and the novel data we arduously obtain. Taking embodiment seriously can help us on all of these counts: conceptually, practically, and analytically. Lastly, by clarifying which aspects of embodied inequality can and cannot readily be altered and within what time span, and which biologic elements are not influenced or determined by social circumstances, we will be better able to predict the future consequences of current social inequalities and the benefits of preventing social disparities in health.

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