Original Paper

Human Development 2017;60:95–106
DOI: 10.1159/000477994

Human Development

The Epigenesis of Obesity

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Keywords
Biological embedding · Developmental systems theory · Embodiment · Epigenetics · Epigenesis · Obesity

Abstract

What would it mean to take seriously a radically dynamic, life course approach to the epigenesis of obesity? This essay brings together concepts and perspectives from developmental systems theory, evolutionary developmental psychology, critical epidemiology, and public and population health into a complex systems framing of the problem of obesity. It begins with a survey of a variety of partial (reductionistic) approaches, and then synthesizes them more adequately and productively via the notion of *biological embedding*. As a hypothesis, biological embedding forces our attention toward the biology of embodiment, the pathways and mechanisms by which multilevel factors at multiple time scales constitute us even within our own skin. In this view, embryology, anthropology, urban planning, and geriatrics are as important to understanding obesity as nutritional science and health promotion. The essay concludes with reflections on this synthetic epigenetic approach in the quest for understanding human development, in sickness as in health.

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Two Pressing Problems

First, a central problem in the study of human development is to understand how such a complex and creative (and destructive) organism emerges from a historical lineage of protobionts and a more proximal union of such unassuming materials as sperm and oocyte. In *Embryology, Epigenesis, and Evolution: Taking Development Seriously* [Robert, 2004], I argued that a dynamic epigenetic perspective was critical in adequately theorizing and exploring our evolution as *Homo sapiens sapiens* and our development as particular Jasons and Davids and Courtneys. Many others have argued similarly [e.g., Gottlieb, 1992; Johnston & Edwards, 2002; Overton, 2013; Oyama, 1985; Oyama, Griffiths, & Gray, 2001]. I wasn't the first and I won't be the last to champion the adoption of a richer ontogenetic ontology and epistemology. But adopting a rich dynamic epigenetic perspective in practice is much easier said than done, which helps to explain the persistence of more simple and simplistic approaches to the study of our kind.

Second, a central problem in contemporary health care, population health, and public policy is to understand how some of us particular humans (me, for instance) wind up overweight or obese, with attendant health and other sequelae, and to understand the population-level patterns (incidence, prevalence, distribution, etc.) of overweight and obesity societally, both locally and more broadly. Obesity is disabling, demoralizing, expensive, and widespread. The World Health Organization refers to it as worldwide epidemic – "globesity" – resulting in social, psychological, physical, and economic challenges on a global scale. These challenges and, indeed, obesity itself are mediated by a complex of social and environmental factors that are difficult to identify – let alone control [World Health Organization, 2017]. Obesity is also, apparently, scientifically and politically intractable: an "elusive epidemic" [Callahan, 2013], as one commentator put it. In the face of such complexity, scientists seek a foothold in something tangible – usually by employing some simplifying heuristics [Robert, 2003a, 2004]. And so they frame obesity in a variety of different ways: maybe obesity is a behavioral disorder; or maybe it is a genetic or gene-regulatory disorder; or maybe it is a social disorder; or maybe it is a microbiomic disorder. Massive research and intervention efforts have explored each of these reductionistic possibilities, with virtually no success. Of course, obesity may be none of these things, and we just need to keep seeking the correct reductionistic frame. But, in fact, obesity is all of the above, and it is all of them at once. Our simplifying strategies have, quite simply, failed us.

In this essay I attempt a civil union of epigenesis and obesity by introducing a broad approach to the epigenesis of obesity, one that captures a variety of the partial explanations of obesity and synthesizes them more productively. For reasons that will become clear, this epigenetic approach is more aligned with developmental systems theory than with "molecular epigenetics." I draw on a variety of interdisciplinary and cross-disciplinary resources to advance our understanding of obesity – and of epigenesis. The end result is a more plausible, more scientific, and more actionable conceptualization of obesity and globesity than many of its predecessors.

The first part of the essay reviews and problematizes a variety of approaches to obesity. The second part introduces some conceptual innovations from population health, critical epidemiology, and allied fields. The third part applies this conceptual work to the problem of obesity. The final part articulates the "cash value" of the new approach, for scientists, policymakers, and the rest of us.

Framing Obesity[1]

How we conceive of obesity as a problem determines how we go about understanding it and solving it. In their comprehensive survey of the various framings to which obesity has recently been subjected, Saguy and Riley [2005] identify several overarching frames: (a) obesity as risky behavior; (b) obesity as disease; (c) obesity as epidemic disease.

Within frame 1, the risky behavior frame, obesity is a function of poor lifestyle choices; if we can alter those choices, then we can have people make better decisions and overcome obesity. On this frame, fat people have failed in either one or both of two ways: either they are ignorant (not knowing what to eat) or weak-willed (not being able to control oneself even when one knows better). These are both moral failures; the former can be addressed by education, the latter by shame. Indeed, weakness of the will is an ancient moral failing (*akrasia*), and that this is what causes people to become or remain obese is at the heart of persistent proposals that the appropriate remedy is stigmatization. Consider the recent remarks of ethicist Daniel Callahan [2013]:

> It will be … necessary to find ways to bring strong social pressure to bear on individuals, going beyond anodyne education and low-key exhortation. It will be imperative, first, to persuade them that they ought to want a good diet and exercise for themselves and for their neighbor and, second, that excessive weight and outright obesity are not socially acceptable any longer. … It will be necessary to make just about everyone strongly want to avoid being overweight and obese. Education has not shown itself to be up to that task. Fear of illness has not, either. No technologies – surgery or pills – have made a major difference. … [We must aim to] make people acutely aware of pervasive stigmatization, but then to invoke it as a danger to be avoided: don't let this happen to you! If you don't do something about yourself, that's what you are in for [name-calling, discrimination, loathing]. (pp. 37–39)

Callahan was widely chastised for his remarks, although perhaps unfairly inasmuch as the overall approach he takes is nuanced and sensitive to the potential harms of stigmatization. Others found it refreshing that someone finally was willing to call a spade a spade: sloth and gluttony are deadly sins, not points of pride or otherwise off-limits to criticism.[2]

Frame 2, obesity as disease, takes almost the opposite approach: obesity is not a result of moral failure, not something deserving of criticism or contempt, but rather a medical issue requiring care and cure. Within this frame, the medicalization of obesity brings it within the scope of health care, and so medical solutions (e.g., the surgery and pills that Callahan dismisses as ineffective) become not only possible, but prescribable and reimbursable. Subframes include the equation of obesity with a genetic disorder, say, of lipid metabolism (suggesting genome editing or gene "therapy" as possible solutions), the claim that obesity is caused by gut flora deficiencies (suggest-

[1] The phrase "framing obesity" appears in an excellent survey of obesity researcher and activist conceptions of and attitudes toward obesity [Saguy & Riley, 2005].

[2] On sloth and gluttony: prior to the publication of Callahan's piece, see the remarks of Michael Fumento [cited in Saguy & Riley, 2005, p. 885], for instance. In *The Fat of the Land*, Fumento writes that "the fat acceptance people… have turned what had been two of the Seven Deadly Sins – sloth and gluttony – into both a right and a badge of honor … That's a sin in and of itself" [Fumento, 1997, p. 130]; he elaborates in an interview that "when somebody shows prejudice to an obese person, they are showing prejudice toward overeating and what used to be called laziness. It's a helpful and healthful prejudice for society to have" [quoted in Lasalandra, 1998, p. 20].

ing fecal matter transplant as a possible treatment), and the claim that obesity is a neurological condition (suggesting deep-brain stimulation as the next big thing in weight loss). But note that mere medicalization does not do away with stigmatization: "biological inferiority" has a long and troubled history, and "at-risk" individuals may be penalized for actions related to their diagnosis. Moreover, medicalization wrongly equates obesity with ill health, and forces a "sick role" on fat people who may be perfectly healthy, with all kinds of individual and social consequences.

I briefly described frame 3, obesity as epidemic (that is, "globesity"), in the introduction. This framing expands the reach of physicians and also affords a greater role for government agencies, in the name of public health. Moreover, the epidemic framing of obesity suggests that obesity is contagious – or at least "socially contagious" [Fumento, 1997, p. 245]: what we eat and how much can be influenced by those with whom we eat (friends, family), and bad behaviors spread easily. But at the same time as frame 3 expands the purview of health care professionals and public health professionals, it also reopens the door to stigmatization: Callahan and others specifically think of stigmatization *in the name of public health* as defensible (it worked with smokers, why not fatties?).

Each framing of obesity is plausible. Each is also incomplete, offering only partial access to reality. So how ought we to study and solve the problem of obesity? Let's take a conceptual detour through population health, so as to begin to make sense of the complex pathways tying together development, social and physical environments, and health outcomes.

How the Environment Gets under the Skin[3]

Over the past four decades or more, research in population health and on the social determinants of health has demonstrated that social, economic, individual behavioral, cultural, historical, biological, and physical contexts have differential effects on health outcomes for both individuals and populations. Factors within, across, and between these contexts interact in complex ways, on complex timescales, in determining health outcomes and variation in health outcomes. But even though we "know" this to be the case, researchers are inclined or encouraged to pretend otherwise, often seeking simpler or apparently more proximal explanations for disease. This is understandable as a research strategy; tractability is key. But it raises the threat that what our research ends up telling us about is some domesticated idealization rather than the wild reality of nature – and the former may have nothing to do with the latter [Robert, 2008].

It might seem that the complaint here is that we too quickly jump to the biological level to explain some phenomenon of interest at another level. But biology is not the enemy here. The problem lay in the kind of biological research that we undertake – and, accordingly, what we do not end up studying. As Heymann and Hertzman [2006] note:

[3] The compelling phrase "How the environment gets under the skin" was introduced by Hertzman and Frank [2006].

> Although extensive research has demonstrated that social determinants make a difference [to our health outcomes], at some level, most of us do not truly believe it – not instinctively, not down to our bones. We often do not understand how social conditions can affect biology, and we've been thoroughly ingrained with the notion of the *biology* of illness that all explanations starting and finishing with biology seem more plausible than any others. (p. 4; italics in the original)

This key observation should lead us not to reject biological explanations but rather to learn to understand the pathways by which "social conditions can *affect* biology" (p. 4; italics added), and also the ways in which the biological can help us understand the social. That is, we are both and simultaneously biological and social creatures, and our research strategies must honestly capture this dual nature if they are ever to be adequate to and for us.[4]

The epidemiologist Nancy Krieger [2005] makes exactly this point in discussing the concept of "embodiment" in epidemiology: "Clues to current and changing population patterns of health, including social disparities in health, are to be found chiefly in the dynamic social, material, and ecological contexts into which we are born, develop, interact, and endeavour to live meaningful lives" (p. 350). She contrasts this contextual "ecosocial" perspective with "pervasive aetiological hypotheses concerned mainly with decontextualised and disembodied 'behaviours' and 'exposures' interacting with equally decontextualised and disembodied 'genes'" (p. 350). She observes that "from the conditions of our bodies – and those of the animals and plants whose environs we now shape – you can gain deep insight into the workings of the body politic" – that is, the social (p. 350). Acknowledging our embodied existence in studies of human development forces us to integrate (not explain away or otherwise ignore) the material contexts of our lives, and how those contexts – "the environment" – get under our skin.

Another epidemiologist, the late Clyde Hertzman, introduced the related concept of "biological embedding." Biological embedding can help us study what Krieger calls "embodiment" by forcing our attention on the biology of embodiment, the pathways and mechanisms by which factors at multiple time scales and levels of organization manage to deeply contextualize us, and constitute us even within our own skin. As summarized by Hertzman and Frank [2006], "biological embedding is an overarching hypothesis that seeks to explain how systematic differences in human experience can systematically affect the healthfulness of life across the life course" [p. 41, following Hertzman & Wiens, 1996]. Hertzman hypothesizes that experience (broadly construed) achieves its generative effects via a "spectrum of specific biological mechanisms … through embedding of the environment in human functioning,

[4] These considerations point to the need to overcome the persistence of the nature-nurture dichotomy: The psychologist and developmental systems theorist Susan Oyama observes "that the dynamic of the nature-nurture debate is a matter of trap-setting and -tripping: the debate is gerrymandered such that critics of genetic determinism open themselves to the charge of environmental determinism, while critics of environmental determinism risk exposing themselves as supporters of genetic determinism." Oyama further notes that merely conjoining "nature" and "nurture" will not work, given that both of these are flawed constructs in the first place; conjunction simply compounds the problem in a debate that was misguided from the very outset. "What we need is not ever more sophisticated ways to prize them apart [as reviewed, for instance, in Schaffner, 2001], but rather a view of life and history that is rich enough to integrate the genetic, morphological, psychological, and social levels (each 'biological,' each with a history) in such a way that we are not tempted to indulge in phenotype partitioning at all" [Robert, 2003b, p. 94, citing Oyama, 2000].

with varying lag times (or latencies), as well as pathway and cumulative effects, from childhood to old age" [Hertzman & Frank, 2006, p. 41]. Biological embedding has a declining effect on epigenesis over time, with the likelihood of more dramatic impacts earlier in life. This declining influence is explained in part by the especial fecundity of developmental windows or critical periods [e.g., Gluckman, Hanson, & Beedle, 2007; Schettler, Solomon, Valenti, & Huddle, 1999; Smith & Robert, 2008], and in part by a kind of canalization or what William Wimsatt [2001] calls "generative entrenchment," according to which what happens early in development can radically constrain future possibilities.

The pathways through which biological embedding occurs affect mechanisms of hormonal regulation, and hormones mediate a host of complex interactions within an organism and beyond. As a result, it makes sense to think of biological embedding as primarily affecting psychoneuroimmunological processes. Additionally, it is critical to assume a life course perspective (basically from oocyte to urn) in order to capture the dynamics of timescale effects such as cumulative or additive effects, generative interactivity, and latency [Hertzman & Frank, 2006; in a different but related context, see also Daaleman & Elder, 2007]. Hertzman and Frank [2006, p. 41] note that there are "chronic targets" of systematic differences emergent from "different socioeconomic, psychological, and developmental environments" over the life course:

- the "objective" (measurable) level of stressfulness of daily life circumstances;
- one's subjective experience of stressfulness;
- the development of coping skills (emotional, social, cognitive);
- the "physiological pattern of host response" to daily stressors, and
- a person's "biological responses to the circumstances and experiences of daily life."

Biological embedding suggests that "systematic differences across these five factors, day by day, week by week, and year by year over the life course will lead to systematic differences in the function of organ systems and create systematic differentials in morbidity and mortality that will cut across a wide variety of disease processes" [Hertzman & Frank 2006, p. 41].[5]

The details are less important for present purposes than the *gestalt*, but Hertzman and Frank provide the hypothalamic-pituitary-adrenal (HPA) system and the sympatho-adrenal-medullary (SAM) system as exemplars of the mechanisms by which biological embedding occurs. For instance, in early life, mediated by the HPA system, prenatal, perinatal, and immediately postnatal experiences are linked to developmental risk and eventual health outcomes; especially illustrative here is Michael Meaney's work on the epigenetics of maternal licking and grooming behavior in rats toward their pups [see, e.g., Meaney, 2001]. By midlife, as the environment has been getting under the skin for decades, chronic exposure to objectively challenging circumstances of daily life may engender a "gradual switchover" of mechanisms to respond to stress, which we might characterize as "learned helplessness": as individuals increasingly believe that they have inadequate resources to respond to challenges, their body may switch over from a mobilize-the-required-energy (SAM) response to

[5] For present purposes, I will set aside the roles of unsystematic or idiosyncratic differences in experience, but see Turkheimer [2000] for some important cautionary notes on "the gloomy prospect" that it is the very capriciousness of developmental experiences that makes experience as such resistant to standard research designs and methodologies.

a not-worth-trying/defeat (HPA) response [Meaney, 2001, p. 38]. And later in life, it is apparent that defense systems "age" at different, context-dependent rates, determined in part by early and midlife experiences: "All individuals in society are on a similar trajectory of increasing risk of a range of diseases with increasing age, but life circumstances (and the biological responses to them) determine how rapidly individuals move along that trajectory" [Meaney, 2001, p. 40]. A key concept at work here is "allostatic load" [McEwen, 1998], which is partly determined by SAM, HPA, and other psychoneuroimmune system interactions.

In sum, embodiment and biological embedding help to provide a dynamic, life course perspective on the multiple mechanisms and pathways by which environmental factors (experiences, exposures, absolute and relative standing, etc.) manifest in our biology. They are not always easy to understand, they are often not independent of each other, and they are not really amenable to randomized controlled studies. They are tractable, nonetheless, but not obvious.

Biological Embedding of Obesity, over the Life Course

With this conceptual apparatus in place, we can consider the epigenesis of obesity as the interactive result of socially mediated experiences and behaviors that culminate in biological qualities that are themselves subject to systematic and idiosyncratic change over developmental time. (Whether those qualities or changes in them are pathological is highly contingent, as will become clear in the next section.) But developmental time does not exist in a vacuum; it is rather evolutionarily constrained [Gluckman & Hanson, 2004; Robert, 2004; see Lickliter & Honeycut, 2003]. Accordingly, we should view obesity as a predictable though complex outcome of the biological embedding of our contemporary world within the scaffolding afforded by our species' evolutionary-developmental history.

On this broadly epigenetic approach to obesity, it is a mistake to partition the causes of obesity into personal and social and biological determinants; nature versus nurture makes as little sense here as it does anywhere else in developmental science. The claim that obesity is partly caused by nature and partly by nurture, partly caused by biology and partly by sociology and psychology, is at best trivially true and at worst seriously misleading. Instead, a more relational account of causation must be invoked, as in Overton's [2013] relational-development systems, wherein "the relational nature of the system emphasizes causality as reciprocal bi- or multi-directional (←→) or circular (positive and negative feedback loops)" [Overton, 2013, p. 102]. On this account, "all facets of the individual and the context exist in mutually influential relations" [Overton, 2013, p. 102], and failure to understand these complex relations is failure to comprehend the system of interest [Robert, 2004].

Adoption of this perspective does not lead to abandonment of the quest to identify particular causes, but rather enrichment of that quest and enhanced likelihood of success. We must look far, wide, and deep, and employ fewer simplifying assumptions, to make sense of complex systems. Consider the Developmental Origins of Health and Disease (DOHaD) paradigm, which holds that chronic diseases manifesting in adulthood can have their developmental basis in early (fetal, perinatal, infant) life [e.g., Hales & Barker, 2001]. The causal story in the background here does not invoke emergent magic, but neither does it reduce away the complexity of develop-

ment. Gluckman et al. [2007] have argued that "the DOHaD phenomenon can be considered as a subset of the broader processes of developmental plasticity by which organisms adapt to their environment during their life course" (p. 1). Developmental or phenotypic plasticity refers to the capacity of an organism to produce different phenotypes (morphologies, behaviors) in response to specific environments [for an overview, see Pigliucci, 2001]. Developmental plasticity is important evolutionarily as well as developmentally. It may involve trade-offs of various sorts, allowing for the persistence of apparently maladaptive morphologies or behaviors within the context of the entire organismal system, while nonetheless enhancing the survival and eventual reproductive capacity of the organism.

In regard to obesity, the DOHaD/developmental plasticity approach suggests a "thrifty phenotype" hypothesis to account for the prevalence and incidence of metabolic disorders in the contemporary world [Hales & Barker, 2001]. Focusing on the life course of the system, we can begin by identifying the developing fetus in utero as adapting to its novel (and perhaps adversarial) environment by optimizing limited nutritional resources simply in order to survive. If the nutritional resources are especially scarce (say, the pregnant woman is malnourished), then the need for such optimization is that much more acute, and the fetus "makes the best of a bad situation," developmentally, to serve evolutionary ends (e.g., survival as a condition of reproduction). But this optimization "behavior" – the thrifty phenotype – results in preferential treatment of some organs (such as the brain) over others, with more or less permanent developmental effects. Now, when the fetus grows up into an adult, her/his "thrifty phenotype" may be challenged by a completely different environment, one marked not by scarcity but rather by abundance. This manifests in what Gluckman and colleagues refer to as "the match-mismatch paradigm of metabolic disease" [Gluckman et al., 2007; see Gluckman & Hanson, 2005, 2006]. As a result, the developing adult may be incapable of dealing appropriately with a high-caloric intake because of the metabolic set points established gestationally. Despite the thrifty phenotype having been highly advantageous and fit in utero, it now predisposes toward obesity, hypertension, and insulin resistance.

These metabolic set points are not carved in stone or cast in concrete. They are, rather, embedded, embodied, in biology. And biology is not fixed but dynamic, changing, flexing, adapting. Embodiment is historical and foundational, but also conservative and generatively entrenched. Adjusting set points is neither impossible nor particularly easy. The biological embedding of the social thus involves complex causality. Social influences may be latent, with a long time passing between exposure and effect (as with the match-mismatch hypothesis), cumulative, whether in a standard dose-response form, or involving the additive effects of multiple exposures, or evincing more interactive effects, and/or pathway dependent, whereby a particular exposure at one point in time predisposes toward other kinds of exposures or the likelihood of certain kinds of context-dependent effects [Hertzman & Frank, 2006]. The relationships captured in these models of influence are all part of a complex, interactive trajectory – the life course. To understand the epigenesis of obesity, then, we must attend to biological embedding over the life course.

Saguy and Riley [2005] introduce a fourth framing of obesity that is radically different from the behavioral, medical, and public health frames. Frame 4 refers to "fatness as body diversity" and is the product of the ideas (and corresponding movement) that obesity is not a disease and that health is possible at every size. The overarching

aim of this framing is acceptance – acceptance that even fat people can be healthy, and also acceptance that there is a wide variety of biological normal "weights" despite the official definitions of overweight and obesity.[6] Within this frame, everyone individually has a biologically normal weight or weight range that is set developmentally and so weight is largely beyond immediate personal control. Within this frame, diets will likely fail (because of our particularly constituted biology), while exercise may lower the norm (by altering the underlying biology).[7] Emphasize health instead of size, and the problem of obesity looks very different, indeed. While obesity may be pathological, it need not be. Context matters and manifests in our individual and collective biology. A life course focus on the epigenesis of obesity thus affords new foci for conceiving, studying, and (where appropriate) intervening in obesity.

Reconceiving Obesity Developmentally: Science, Policy, and Praxis

Adequately grappling with obesity conceptually, and via interventions, requires us to take development seriously in all its complexity. If we want to *understand* patterns of obesity in groups and susceptibility to obesity in individuals, we need to pay attention to "the interplay – moment to moment, hour to hour, day to day – between the environments where people live, work, and grow up, on the one hand, and the development and responses of the SAM, HPA, and PNI [psychoneuroimmune] axes, on the other, which lead to systematically differing health expectancy over a lifetime" [Hertzman & Frank 2006, p. 44]. This is no easy task. Additionally, we must pay attention to the interplay between development and evolution so as to appreciate how context and contingency conspire to generate the bundle of trade-offs and compromises that is the human body [Robert, 2004]. And if we want to *alter* patterns of obesity in groups and susceptibility to obesity in individuals, we need to build public policy and public practices with full appreciation of these complex ecologies and their biological embedding over the life course. Again, no easy task.

In regard to biological embedding, Hertzman and Frank observe that considerably more research is needed on "exposures" to key social factors (including identification and measurement issues), on conceiving biological endowments properly biologically (epigenetically), and on the complexities of biological pathways and of objective and subjective psychological constructs [Hertzman & Frank, 2006, pp. 51–54]. More practically, more research is needed on precisely what it is going to take to make any of the science meaningful and actionable to policy makers. As Rychetnik, Hawe, Waters, Barratt, and Frommer [2004] note:

[6] For adults, the body mass index (BMI) of an overweight person is between 25 and 29.9 (women: 5'4" and >146 lb; men: 5'10" and >174 lb); the BMI of an obese person is higher than 30 (women: 5'4" and >175 lb; men: 5'10" and >209 lb); any adult with a BMI higher than 40 is considered morbidly obese (women: 5'4" and >233 lb; men: 5'10" and >279 lb). The BMI as a measure of obesity and as a risk factor for disease has been widely criticized, in part because it fails to capture variation in body composition (very muscular people will have a very high BMI as muscle is denser than fat).

[7] Diets – or changes in nutritional intake – can also alter the underlying biology and/or how it works. But there is so much epistemic chaos in the nutritional sciences that these issues are very poorly understood to date. For instance, a typical human body will fuel itself initially and primarily on carbohydrates; but a human body in diet-induced cyclical ketosis will burn fat before sugars – a radical change that often leads to biologically explicable suppression of appetite and immediate weight loss, but with largely unexplored mid- and long-term consequences.

The advocacy and lobbying that are required to influence policies, change practice, and achieve public health action are an important component of public health. The process of achieving influence is often more difficult, and requires more complex social and political negotiations, than appraising evidence and formulating recommendations. In public health advocacy, research provides only one type of evidence, and evidence of any type is but one consideration that is taken into account. (p. 541; references omitted)

The key lesson here is that even if we rehabilitate developmental science, there will always be more work to be done to move forward. And as the literature on public planning has made abundantly clear for over 40 years [e.g., Rittel & Webber, 1973], any policy or praxis intervention in a complex system may effectively change that system in unpredictable and uncontrollable ways. Sometimes those changes are positive. Not always. We can never hope to definitively characterize a complex system. But the more we factor complex causality into our models, the more we appreciate the function of contingency, and the less we seek to explain away context as noise rather than integrate it as signal, the likelier we are to intervene productively.

It is clear that, at least in America, as a nation we eat badly, build unwalkable cities, and search for cures for obesity rather than preventing it in the first place. Lifestyle theory is predominant in American public health, and it is almost un-American to seriously suggest that social (rather than personal) determinants of health and disease are at play. The epigenesis of obesity as explored in this paper suggests that the better we understand the science of biological embedding – the actual biological effects of social determinants – the easier it will be to shed flabby concepts and practices that constrain us all.

To avoid sacrificing both healthspan and lifespan – that is, to avoid getting fatter and potentially sicker and maybe dying sooner and certainly sadder – we need to get beyond blame and shame, naïve and counterproductive medicalization, and health promotion that amounts to little more than information-sharing (with often incomplete information being shared). Shaming exacerbates disordered behavior and biology. Physician recommendations to seek diet and exercise are easier offered than received and implemented, and often misinformed.[8] Diets rarely work. Exercise stands a chance, but social factors and structures radically undermine the likelihood of regular exercise for many segments of the population.

A well-rounded, epigenetic conception of obesity is a critical starting point. Redressing obesity once fully manifest will prove more intractable than a more prospective, preventive approach that captures the dynamics of biological embedding, especially in early life. But again what is embedded is not permanently fixed. Dis-embedding or re-embedding or other reconfigurations may be possible, especially as we learn more about the complex relational causality at work in developmental systems. Let's start there.

[8] And following doctors' ill-conceived orders is problematic in itself, inasmuch as the wildly popular "low-fat" movement to prevent diabetes and combat obesity very likely resulted in making things worse. The alternative to satiating fat to which people turned is the remarkably fattening carbohydrate, and diabetes and obesity are literally everywhere. Meanwhile, diets typically fail, resulting in overall weight gain in between dieting attempts; the negative health effects of such weight cycling or so-called yo-yo dieting are well documented [Saguy & Riley, 2005].

Acknowledgment

This work is dedicated in memory of Clyde Hertzman (1953–2013). Clyde and I served for some time together on the Institute Advisory Board of the Canadian Institutes of Health Research's Institute of Population and Public Health. He might not have known who I am, but I am ever grateful for Clyde's careful research and compelling remarks that inspired me and continue to influence my work.

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