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Toppling Typologies

Developmental Plasticity and the Environmental Origins of Human Biological Variation

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The early scientific study of human variation was founded upon an assumption that human populations could be classified according to stable types, or races, that were viewed not only as immutable but also as representative of different stages of evolutionary advancement (Stocking 1968; Wolpoff and Caspari 1997). The quantitative study of body form, including stature, body proportions, and, especially, craniometric measures, was the primary means of classifying human variation at the time, and despite inconsistencies in many early findings, these data were used to reinforce the presumed natural status of Caucasoid, Mongoloid, and Negroid human subtypes (Gould 1996). An early anthropological challenge to the idea of stable racial types is credited to Franz Boas, who was a founding figure of American anthropology (Williams 1996). Employing measures of bodily dimensions in a large sample of immigrants, Boas (1912) found that the length of time that mothers spent in the United States influenced their children's size and cranial dimensions, implying that the environment played some role in shaping these traits. These findings were a clear demonstration of what we now call "developmental plasticity," or the capacity for developmental biology to be modified by environmental influences.

Although the work of Boas and others helped undercut typological notions of human variation, the renaissance in early genetics research was in full swing at the time and would soon be followed by developments such

as the melding of Darwin's and Mendel's work and by the discovery of DNA (Watson and Crick 1953). These radically restructured the field of biology, including the study of race. In the wake of the molecular revolution, it was no longer sufficient to focus solely on physical traits such as cranial form, skin color, or hair texture. Instead, human races were redefined initially in molecular terms, exemplified by early work on blood groups, and, with the subsequent advent of sequencing technologies, strictly genetic terms (Marks 1995, 1996). With the locus of human phenotypic stability shifting from physical to molecular units, the reality of race as a natural biological category ultimately hinged upon the question of whether human genetic diversity clustered within traditional racial categories.

The degree to which genes partition by continental race, and whether they support the race concept, remains hotly debated to this day (Jorde and Wooding 2004; Mountain and Risch 2004). Oddly, the same data are routinely used both to support the notion of genetic race and to undermine it (Barbujani 2005). For instance, those who believe that race is a valid biological category point to evidence for significant partitioning of human genetic variation by continent or self-identified ethnicity as evidence that race is not merely a social construct (Risch et al. 2002). However, others have noted that the majority of variation in such studies is found within continents or socially defined race groups rather than between them (Brown and Armelagos 2001). As an example, one high-profile study examined hundreds of single nucleotide polymorphisms (SNPs)—small markers of genetic variation within individuals—and found that only 3 to 5 percent of the variation represented in this global sample was explained by major population groups (Rosenberg et al. 2002).

Although debates about the genetic reality of race remain unsettled, the percentage of population variance that must be explained for race to be "biologically meaningful" is inherently subjective. And there are, arguably, deeper problems with the question as currently framed, for the debate hinges upon an unstated assumption that genes are an appropriate proxy for human phenotypic variation. The notion that genes code "for" traits is a pervasive one in popular and media coverage of genetics research but is also implicit in the thinking of many medical scholars. This misconception lingers on, despite a decade of brisk population genetics research that has largely failed to identify strong genetic predictors of most phenotypes (traits or behaviors). Extensive investment of research dollars in hope of discovering genes that contribute to common diseases, such as hypertension, obesity, or diabetes, has resulted in a modest list of consistent genetic predictors of these conditions, and, collectively, they explain what most

agree is a small fraction of the variance in such traits (Cruickshank et al. 2001; Rankinen et al. 2006; Sankar et al. 2004). If knowing someone's genotype tells us little about his or her phenotypic characteristics, the question of whether these genes partition according to race seems to lose some of its relevance.

Here, we argue that principles of evolutionary biology, which Boas unwittingly demonstrated in his study of immigrants, help us understand why many genes *by necessity* are only loosely coupled with specific phenotypes in a species such as humans. Humans are long-lived organisms and have evolved mechanisms that allow more rapid adaptation to environmental change than can be accommodated by the gradual process of gene frequency change (Kuzawa and Thayer 2011). Natural selection has thus favored the retention of many human gene variants that do not code "for" traits but rather for flexible systems capable of a range of response states. We argue that processes of environment-driven developmental plasticity are important contributors to human variation that we see today. This is especially true for phenotypes that map onto the social categories of race and the gradients of environmental stress and opportunity that societies organize around these categories.

We begin by reviewing the evolutionary and adaptive importance of developmental plasticity, which enables organisms to respond to and cope with changes too rapid to be handled by genetic adaptation (West-Eberhard 2003). We next survey important mechanisms of plasticity that allow environmental experiences to shape developmental biology and thus the assembly of mature phenotypes. We show that plasticity is a pervasive feature of human biology that has important impacts on traits such as growth rate, maturational timing, age at first reproduction, brain organization, and immune function and on the metabolic and physiologic traits that influence how the body manages energy and reacts to stress and that ultimately determine risk for many chronic diseases. In concluding, we suggest that Boas's observations of a century ago remain all the more relevant today: just as evidence for plasticity helped topple essentialist notions of a racial body type, today an understanding of plasticity moves us beyond the simplified notions of genetic determinism upon which the presumed biological importance of genetic race now rests.

THE IMPORTANCE OF DEVELOPMENTAL PLASTICITY AS A MODE OF ORGANISMAL ADAPTATION

The concept of adaptation is among the organizing principles of evolutionary biology and refers broadly to changes in organismal structure,

function, or behavior that improve survival or reproductive success (Lasker 1969; Williams 1966). Genetic adaptation more specifically refers to the process by which gene variants that code for such beneficial traits emerge and stabilize within a population. When a gene variant present in the gene pool of a breeding population increases the survival and/or reproductive success of its carriers, the gene will increase in relative frequency in the gene pool of the next generation. Genes that increase survival to reproductive age or the number of offspring sired will, by a matter of simple arithmetic, become more common than other alleles at the same locus. Over many generations, this will tend to yield organisms with life cycles, reproductive strategies, morphology, metabolism, and behavior that are well suited for the range of conditions encountered by members of that population.

Although adaptation by this process of natural selection is a powerful mode of adjustment at the population level, many environmental changes occur on a more rapid timescale than can be efficiently dealt with by changes in gene frequency, changes that require many generations and hundreds if not thousands of years in order to accrue in the gene pool. To cope with this more rapid change, human biology includes additional, more rapidly acting adaptive processes (Ellison 2005; Kuzawa 2005; Lasker 1969). The most rapid ecological fluctuations (e.g., fasting between meals or the increase in nutrients that our bodies need when we run) are handled primarily via homeostatic systems, which respond to changes or perturbations in a way that offsets, minimizes, or corrects deviations from an initial state (negative feedback). Operating not unlike a thermostat, which maintains a constant temperature by turning the furnace on and off, homeostatic systems modify physiology, behavior, and metabolism to maintain relatively constant internal conditions despite fluctuations in features such as ambient temperature, dietary intake, and physical threat. The distinctive features of homeostatic systems include their rapid responsiveness and self-correcting tendencies. Also, the changes they induce are reversible, not permanent.

Some environmental trends are chronic enough that they are neither efficiently buffered by homeostasis nor sustained enough for substantial genetic change to consolidate around them. Such intermediate timescale trends would thus fall through the cracks if homeostasis and natural selection were the only means available of adjusting biological strategy. It is easy to see how a sustained change might overload the flexible capacities of a homeostatic system if this were the only way to help the organism cope with it (Bateson 1963). Take, for example, an individual who has recently moved to a high-altitude environment where oxygen pressure is too low for his or her lungs to efficiently handle. One immediate response will be an elevated

heart rate that increases the volume of blood and thus the number of oxygen-binding red blood cells that pass through the lungs. By engaging a homeostatic system—heart rate—the body has found a temporary fix to help compensate for the low oxygen pressure. However, this comes at a cost, for it "uses up" the ability to increase heart rate and deal with other challenges that might require bursts of higher blood flow, such as running from a predator. Thus, chronically elevating heart rate may work as a short-term solution but is a poor means of coping with chronic high-altitude hypoxia.

With time, additional biological adjustments ease the burden on the heart, such as increasing the number of oxygen-binding red blood cells in circulation. However, individuals *raised* at high altitude have a better strategy yet for coping with low oxygen availability, for they simply grow larger lungs, thus obviating the need for these fixes (Frisancho 1977). This change in developmental biology is an example of *developmental plasticity*, which allows organisms to adjust biological structure on timescales too rapid to be dealt with through genetic natural selection and too chronic to be buffered by homeostasis (Kuzawa 2005). These mechanisms can be viewed as enabling the organism to fine-tune structure and function to match the needs imposed by its idiosyncratic behavioral patterns, nutrition, stress, and environmental experiences, all of which cannot be anticipated by the genome (West-Eberhard 2003). Unlike homeostatic changes, which are transient, growth and development occur only once, so plasticity-induced modifications tend to be nonreversible once established. In this sense, developmental plasticity is intermediate between homeostasis and natural selection in both the phenotypic durability of the response and the timescale of ecological change that it accommodates. As discussed in detail next, the more durable, structural nature of changes induced by developmental plasticity makes it an especially powerful generator of human phenotypic variation within and between populations.

MECHANISMS OF DEVELOPMENTAL PLASTICITY AND PHENOTYPIC EMBODIMENT

Which biological processes enable phenotypic structure and function to be modified in response to environmental experience? Plasticity involves changes in the growth, structure, or function of a trait, an organ, or a physiological system. This can involve a change in the number of cells present in a tissue or an organ, in the properties or patterns of gene expression within individual cells (epigenetic changes), or in cells' integration at higher levels of biological organization. These processes do not negate the importance of genes but exemplify that the phenotypic effects of genes are contingent

upon interaction with environmental inputs. A handful of developmental strategies or "assembly rules" have evolved so that organisms can harness these properties to match form and function to individual needs (Gilbert and Epel 2009). These mechanisms of plasticity include phenotypic accommodation, reaction norms, and developmental programming. Each facilitates adaptation to a different type of environmental change. However, all help explain why phenotypes tend to map onto the environments or social conditions that humans experience and why many phenotypes are not easy to predict on the basis of knowing someone's genotype.[1]

Phenotypic Accommodation: Organizing Structure around Patterns of Use and Disuse

Phenotypic accommodation is a process in which developing structures organize around patterns of use or functional loading. Neuronal selection in the central nervous system (CNS) is the archetypic example: during brain growth, masses of redundant neurons and synaptic connections are generated. Cells and connections that are used are stabilized and retained and those not used are pruned away, resulting in a structure built through learning and experience (Changeux 1986). The immune system develops according to similar principles: millions of randomly spliced antibodies capable of binding millions of antigens are generated in infancy, but only those that come in contact with their associated antigen are retained in the pool of memory cells. Many of those that never find their associated antigen are pruned away, thus gradually developing of a repertoire of defenses well-suited to protecting the body against locally encountered pathogens (Edelman 1973). The skeletal system develops similarly. Viewing a cross-sectional slice of the femur reveals patterns of fine spongy bone (trabeculae) aligned along gradients of stress and strain. Individuals with different patterns of mechanical loading develop appropriate variations in bone structure (Pontzer et al. 2006). Because activity and mechanical loading cannot be known in advance, the nature of the fine structure of bone within the femur is not coded in the genome. Instead, it is assembled through a process of developmental plasticity in which redundant bone cells proliferate and are retained if they align with gradients of loading within the tissue (Pearson and Lieberman 2004; Ruff, Holt, and Trinkaus 2006).

The capacity for all of these systems to align with environmental conditions and need is based upon a simple algorithm: generate more structure than needed, stabilize and keep what is used, and then prune away any unused excess. This ability to fine-tune developing structures in response to patterns of use and disuse allows relatively few genes to specify a vast array

of possible phenotypic configurations according to individual experience and behavior. Accommodation not only is key to learning and antibody acquisition but also likely influences brain regions involved with regulating the production of hormones that influence metabolism, reproduction, and behavior (Badyaev 2009). It also biases immune development to modify risk of allergy, asthma, or systemic inflammation related to many chronic diseases (McDade et al. 2010). Accommodation has broad effects on the shape and strength of individual bones and their articulations within the skeletal system, and use-driven development of the musculature during development has lifelong consequences for strength, body composition, and even body dimensions (West-Eberhard 2003).

Through accommodation, many developing systems acquire "information" about the environment in order to meet the needs of that individual, place, and time. This information cannot be anticipated by nucleotide sequences, which are inherited from parents and only change in character slowly over many generations. The intrinsic sensitivity inherent to processes of developmental biology helps explain why phenotypes come to serve as biological mirrors of our environments and illustrates in concrete terms one reason we should not be surprised that genotypes tend to be poor predictors of many complex phenotypes.

Reaction Norms: Growth, Maturational Tempo, Adult Size, and Reproduction

Some environmental inputs trigger a coordinated developmental response involving changes in multiple traits that flex together. Evolutionary biologists describe such patterns of response as reaction norms, which are assumed to trace to complex interactions between suites of genes and environmental inputs (Schlichting and Pigliucci 1998). One clear example of a human reaction norm is the response of body growth and maturational tempo to changes in nutrition, which influences traits such as adult stature, body weight, and age at reproductive maturity. Individuals raised under favorable nutritional conditions grow rapidly, reach reproductive maturity earlier, and are also taller and heavier as adults (Eveleth and Tanner 1990). In northern European countries with good historical records, menarcheal age has declined from 17 years in the mid-nineteenth century to the present mean of 12–13 years, during which time adult stature has also increased. This enormous phenotypic change—reflecting a 33 percent change from the original phenotype in just over a century—was too rapid to involve changes in genes and is understood as a developmental response to improvements in nutrition or hygiene during infancy and childhood (Tanner 1962).

Similar developmental responses to changing nutrition are observed across the animal kingdom, and evolutionary principles have been used to explain the evolution of the diverse reaction norms across species (Stearns and Koella 1986). The trade-off between age at maturity and size at maturity is essential for understanding this variation. For many species, including humans, larger adults tend to have greater physical strength and lower risk of predation (if predators are a local fact of life, as they often were and remain in some settings), and their offspring are also larger and more likely to survive (Stearns 1992). These and other benefits of being a large adult must be balanced against the risks associated with delaying reproduction—and the possibility of dying before reproducing—in order to take more time to grow. This sets in motion a trade-off between age and size at maturity, and the mean age at maturity that strikes a good balance for that species will tend to be favored by natural selection (Stearns 1992).

Nutritional influences on growth rate and the threat of unavoidable mortality vary widely across species, populations, and individuals, and developmental plasticity enables individuals to modify their strategy of growth and maturation in response to these factors. Evolutionary models predict that improvements in nutrition lead to earlier maturity at a larger adult size, much like what is seen in humans undergoing the secular trend in menarcheal age described above (Hill and Hurtado 1996). Many species speed up maturational tempo in response to cues signaling heightened risk of mortality or predation, thus reducing the likelihood of dying before reproducing (Crespi and Denver 2005; Stearns and Koella 1986). This same logic is believed to help explain why children exposed to stressful social cues indicating a risky environment tend to speed up maturation and begin their reproductive careers earlier than children raised in more stable and lower-risk settings (Belsky, Steinberg, and Draper 1991; Chisholm 1993; Ellis et al. 2009).

These examples illustrate how reaction norms often involve a suite of related traits that flex in unison to achieve a common goal as ecological conditions change. Developmental plasticity allows organisms to reach adulthood at an average age that effectively balances trade-offs between nutrition, which influences how fast the organism is capable of growing, and risks to survival, which determine whether to delay reproduction in order to grow larger. As nutrition improves, growth speeds up and the organism reaches maturity earlier and at a larger adult size. On the other hand, as cues of unavoidable mortality increase—signaling that waiting to reproduce may be risky—maturity and reproduction are initiated earlier. Although traits such as stature and menarcheal age have relatively high heritabilities when phenotypic variation is viewed within a single population

sharing the same environment (Demerath et al. 2007), evolved reaction norms help explain why much of the worldwide population variation in traits such as growth rate, adult body size, and age at first reproduction map onto underlying gradients of nutritional adequacy and social privilege (Eveleth and Tanner 1990; Fogel and Costa 1997; Komlos 1994).

Biological Programming: Plasticity in Hormone Regulation, Metabolism, Physiology, and Long-Term Chronic Disease Risk

There is now extensive evidence from a wide range of animals, including humans, that early life experiences of nutritional or psychosocial stress can have profound and lasting effects on hormone production, metabolism, and physiology (Festa-Bianchet, Jorgenson, and Reale 2000; Gluckman et al. 2008; Lummaa and Clutton-Brock 2002). These recently described capacities for developmental plasticity show that maternal health, stress, or nutrition during or even prior to pregnancy influence how the offspring's body responds to stress or handles nutrients, fat deposition, and other functions across the life course. Much of this evidence comes from findings in humans who were born as lower-birth-weight babies, suggesting that they experienced prenatal nutritional stress (Barker et al. 1989). Similar biological and disease outcomes have been shown to result from experimental nutritional stress in animal model research (Gardner et al. 2006; Langley-Evans 2001; McMillen and Robinson 2005; Sayer et al. 2001).

Among the better-documented changes observed in adults who were born small is a tendency to be resistant to the effects of insulin in skeletal muscle. Reducing glucose use in muscle effectively conserves this prized resource for more essential functions such as the brain or immune system (Hales and Barker 1992; Kuzawa 2010). Individuals who experienced prenatal nutritional stress also tend to put on less fat in the lower body or appendages and to preferentially deposit it in the abdominal region (leading to a so-called "apple-shaped" or "android" pattern of unhealthy fat deposition). Fat in the abdominal depot is distinct because it is perfused with nerve fibers from the brain that release hormones such as adrenaline (sympathetic response), which allows the brain to rapidly mobilize stored free fatty acids for use as energy when the body is confronted with a stressor or challenge. Not only do individuals who were born light deposit more fat in this depot, but also, during stress, their fat cells are more sensitive to the effects of sympathetic activation, allowing them to mobilize these stored fats for energy use more rapidly (Girard and Lafontan 2008). As free fatty acids are mobilized to fuel the body, this also triggers insulin resistance in both the liver and muscle, thus further reducing glucose

uptake throughout the body and sparing it for other, more essential functions (Girard and Lafontan 2008).

The potential benefits of adopting such a glucose-sparing strategy in utero are easily seen in light of the nutritional challenges that infants face soon after birth. At this age, more than half of the body's energy use is accounted for by the brain, which almost exclusively uses glucose as fuel (Chugani, Phelps, and Mazziotta 1987; Holliday 1986). Because the brain has inflexible energy requirements and is quickly damaged in the event of energetic shortfall, there is an imperative to protect its glucose supply at this age. Infancy *also* happens to be an age when infectious diseases, such as diarrheal illnesses, occur concurrent with weaning and the introduction of supplemental foods and thereby heighten nutritional stress. It has been hypothesized that this confluence of an energetically demanding and fragile brain and common infectious and nutritional stress helps explain the unprecedented degree to which body fat stores are used as energy backup by human babies, who are the fattest mammalian newborns on record (Kuzawa 1998). The finding that the fetus modifies its pattern of glucose use in response to cues indicating nutritional stress suggests that the body's energetic priorities can be adjusted—increasing the priority of the brain as needed. These responses may be immediately beneficial as a buffer for fetal brain development in the event of a difficult pregnancy (Hales and Barker 1992). In addition, because babies born to high-stress mothers are likely to enter a more stressful world, it has also been hypothesized that this developmental plasticity may have evolved to enable the fetus to make adjustments in anticipation of nutritional stress likely to be experienced after birth (Gluckman and Hanson 2005; Kuzawa 2005, 2010).

Although many of these glucose-sparing metabolic adjustments could improve survival under conditions of nutritional stress—especially, early in life, when such stress is common and brain energy needs are unusually high—the strategies of reducing the body's response to insulin and prioritizing abdominal fat deposition are also among the most important precursors for diseases such as diabetes and cardiovascular disease (Phillips and Prins 2008; Ritchie and Connell 2007). In this way, the fetal capacity to modify energetic priorities in response to the mother's experience of stress can also set up heightened risk for adult chronic disease (Hales and Barker 1992; Kuzawa 2010).

Evidence for Multigenerational Consequences of Maternal-Fetal Metabolic Programming

There is increasing evidence that fetal responses to gestational conditions can perpetuate a transgenerational cycle that modifies biology across

multiple generations, illustrating how environmental experiences in one generation can be felt multiple generations into the future (Gluckman et al. 2007b; Rakyan et al. 2003). This is best documented in the case of a pregnancy in which the mother has diabetes (a common outcome associated with being overweight or obese), which exposes her fetus to high levels of glucose and insulin. Such babies are born with more body fat, and they are also more prone to becoming obese and developing diabetes as children and adults. When a female fetus is exposed to a diabetic gestational environment, her heightened adult risk of diabetes increases the likelihood that the *grandoffspring* of the originally diabetic mother will also be exposed to a high glucose, high-insulin gestational environment, thus perpetuating the pattern (Aerts and Van Assche 2006; Castro and Avina 2002). That this pattern of inheritance is at least partially nongenetic is demonstrated by the finding that offspring born after formerly obese mothers have lost weight as a result of gastric bypass surgery are much less likely to become obese compared with siblings born prior to their mother's surgery, when the mothers were heavier and had elevated glucose and insulin during pregnancy (Smith et al. 2009).

In a similar fashion, when a woman experiences stress during pregnancy, this can change how the offspring responds biologically to stress (O'Connor et al. 2013; Tollenaar et al. 2011). In one recent study, women who had high levels of the stress hormone cortisol while pregnant gave birth to offspring who produced cortisol differently when faced with a stressor in early childhood, strongly suggesting that the mother's stress experience had intergenerational effects (O'Connor et al. 2013). Because this hormone is involved in a range of disease and degenerative processes, children born to mothers who experienced psychosocial stress during pregnancy may be especially prone to adverse health outcomes in later life (Kuzawa and Sweet 2009; Thayer and Kuzawa 2011). And in female offspring, prenatal exposure to the mother's stress is predicted to modify the gestational stress-hormone environment experienced by her future offspring, thus potentially perpetuating a multigenerational pattern of stress-related biological strain (Drake and Walker 2004; Kuzawa and Sweet 2009; Wells 2010).

These examples illustrate that the mother's body conveys biological cues reflecting her experiences—and the *grandmother's* experiences—to her developing offspring. It has been speculated that this ability to pass along lingering biological "memories" reflecting multiple generations of ancestral experience could allow offspring to adjust developmental biology in anticipation of conditions, such as nutrition or stress, that have dominated in recent generations, thus serving as a best guess of conditions

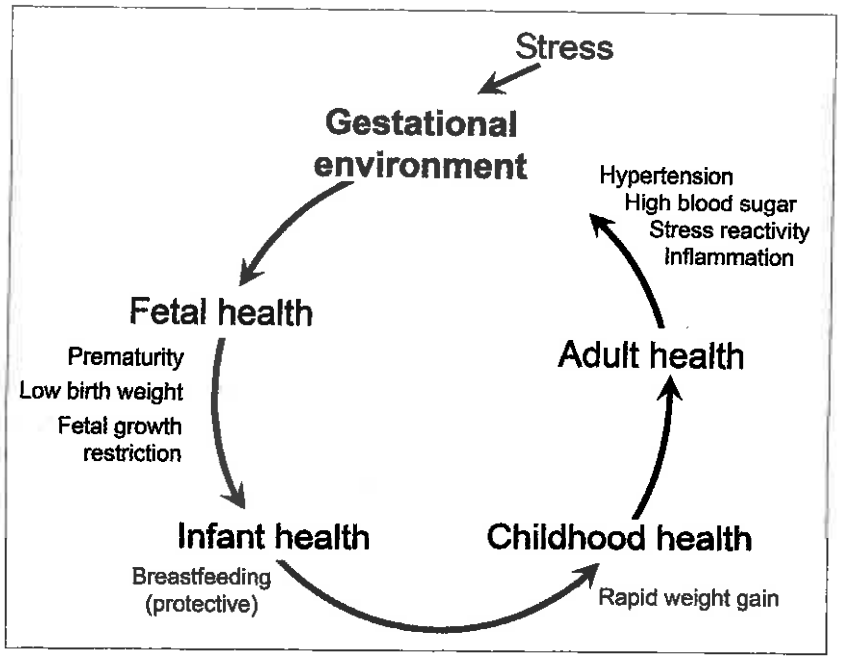

FIGURE 3.1

Recursive model for the intergenerational perpetuation of health disparities operating through effects of maternal stress on metabolic status in adult offspring, which elevates or amplifies risk experienced by grandoffspring (from Kuzawa 2008 with permission).

likely to be experienced in the near future (Kuzawa 2005; Kuzawa and Thayer 2011). Whether these intergenerational effects are adaptive or merely unavoidable consequences of the sensitivity of developmental biology to stress remains to be determined. From a practical perspective, these examples show that the experience of a stressor in one generation can impact long-term biology and health not only in offspring but also in grandoffspring (figure 3.1). Evidence for such multigenerational relationships is providing new insights into the ways that disparities in human experience can shape patterns of biological difference within and across societies (Thayer and Kuzawa 2011).

CONCLUSIONS

The examples of developmental plasticity reviewed above illustrate several means by which human phenotypes come to reflect their ecological and social environments. Boas (1912) provided an early demonstration of

this principal as applied to outward features of human growth and cranial form. Today, we know that plasticity is also integral to the development of many metabolic and physiologic traits. Many human biological systems have not only a capacity but also a *need* to incorporate information from the environment to complete their development. The various mechanisms of developmental plasticity allow the human body to assemble systems that are adjusted in response to social and ecological gradients of resource access, climate, physical activity, and stress. This is an essential means of adaptation that has helped human populations cope with the immense variety of environments inhabited by our species during its long history of migration and ecological diversification (Wells and Stock 2007).

Today, the environmental niches that humans occupy are largely shaped by human institutions (Singer 1989). "Skin-deep" traits such as skin color and facial features have long been used as a basis for defining race, justifying historical and contemporary patterns of exploitation, racism, and discrimination and determining access to resources and exposure to stress within societies. In light of this, it is not surprising that patterns of many diseases are organized around the social construction of race. As one prominent example, metabolic diseases, including hypertension, diabetes, stroke, and heart attacks, are major contributors to the black-white health and mortality gap in the United States (Williams and Collins 1995). Although the "wear and tear" of adult stress experience has long been known to be an important contributor to these disparities, there is mounting evidence that social disparities can also become embodied in a more durable sense as a result of the types of developmental plasticity we review (figure 3.2) (Kuzawa 2008; Kuzawa and Sweet 2009). Most notably, African Americans not only have higher rates of adult cardiovascular diseases but are also disproportionately affected by the early-life developmental antecedents to these conditions, such as lower birth weight, intrauterine growth retardation, and premature delivery. These early-life health disparities, in turn, have been linked to the mother's experiences of stress and discrimination rather than to genes (Collins, Wu, and David 2002; David and Collins 1997) and to predictions of diabetes and cardiovascular diseases in adult offspring (Cruickshank et al. 2005; Mzayek et al. 2004).

The powerful capacity of developmental biology to organize structure and function around individual experience is a prime illustration of why the traditional dichotomy between biology and culture is an artificial one: real biological differences can emerge from both genetic *and* social forces (Gravlee 2009; Kuzawa and Sweet 2009). By controlling the environmental cues that developmental biology is designed to respond to, societies effectively

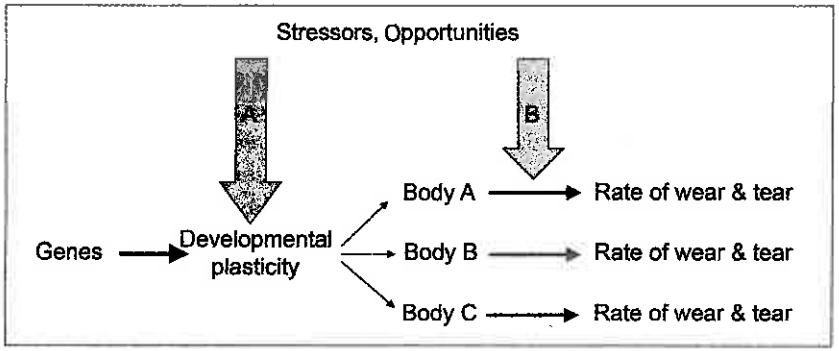

FIGURE 3.2

Environmental experiences can influence biology and health by modifying plastic developmental biology and epigenetic state (arrow A) *or through sustained effects of the environment on the "mature" phenotype across the life course* (arrow B).

project their ideological biases onto our biology, often with profound implications for health and well-being (Montagu 1962; Shapiro 1952). The research we review is providing new opportunities for anthropologists and other social scientists to extend the early critiques of essentialist race models and is helping explain why phenotypes that are poorly predicted by genotype tend to align with socially constructed categories. We are optimistic that future generations of researchers will continue to harness the principles of developmental plasticity to enrich our understanding of human variation and its many underlying causes.

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Note

1. In the following discussion, we often refer to plasticity as "adaptive." To evolutionary biologists, the concept of adaptation always implies trade-off and compromise. Two organisms with unequal resources and opportunities have their respective "optimal" solutions to maximizing survival and genetic fitness, even if the individual with greater resource access is surely better off. Adaptation and optimality are not absolutes and can be understood only in context—such as one's access to nutrition and health care or one's stress.

4

Toward a Cybernetics of Race

Determinism and Plasticity in Ideological and Biological Systems

Ron Eglash

What should anthropology do with the idea of race? Proponents of racial categories maintain that race is an effective way to categorize human variation and that ignoring it means less effective medicine, policy, and research. Critics of racial categories note how social priorities have resulted in misrepresentations of biological facts in much of the "science of race" development: if there is not really enough genetic differentiation between ethnic groups to qualify as a rigorous biological meaning of race, then continuing to use it is merely reinforcing a myth. Haslanger describes the first position as "naturalist" ("race is biologically real"), and the second as "eliminativist" ("race is an illusion, don't use it") (2008:57). She contrasts these two with the third (and most popular) option of "social construction," which maintains that although human race is biologically meaningless, it is nonetheless a socially powerful force that must be engaged rather than ignored (2008:58). This chapter regards all three positions as inadequate. Despite its popularity, the mantra "race is a social construction" has failed to directly engage the complex intertwining of biological and social processes involved (Hartigan 2008). As an alternative, this chapter reframes the question of race using conceptual tools from cybernetics, a discipline created for modeling the information flows within and between natural, social, and artificial systems. We will look for underlying dynamics that