ple” (p. 50). Rushton presented a vast array of scientific evidence in his conceptualization, for example, a correlation of .62 between cranial capacity and distance from the equator with 20,000 crania representing 122 ethnically distinguishable populations (Beals, Smith, & Dodd, 1984). Templner and Arikawa (2003) reported a correlation of −.71 between mean IQ and mean high winter temperature and a correlation of −.61 between mean IQ and mean low winter temperature with 129 countries. There are alternative explanations to those of Rushton for such findings. To relegate Rushton’s theory to the realm of absurdity, however, would neither constitute an optimal scientific reasoning nor represent an ideal spirit of scholarly disagreement.

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DOI: 10.1037/0003-066X.61.2.177

Examining Unproven Assumptions of Galton’s Nature–Nurture Paradigm

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Sir Francis Galton’s (1869/1892) notion of nature versus nurture is a cornerstone of psychology: It was recently featured in two issues of the Monitor (March and April 2004) and was infused throughout the January 2005 issue of the American Psychologist. Sternberg, Grigorenko, and Kidd (January 2005) offered keen insights into the pitfalls in the study of intelligence and race, discerning between folklore and science. Similar scrutiny is needed of the premise underlying these articles: that the nature–nurture paradigm is a scientific fact. Ultimately, the validity of statistical formulae derived from Galton’s thesis depends on unproven assumptions. Further, a dimensional ontology allows expansion of the theoretical perspective.

The idea that nature and nurture make us who we are is easily distilled into a statistical formula: In terms of variance, “heritability and environmentality add to unity” (Sternberg et al., 2005, p. 53). The first assumption, termed exclusivity, stems directly from that idea: Only nature and nurture make us who we are. This leads to a paradigmatic requirement: No influences exist other than genetics and environment.

The second assumption implicit in references to nature–nurture research, termed universality, is that the paradigm is valid for every human trait studied. An example will illuminate both assumptions.

A century ago, phenylketonuria (PKU) was purely heritable in terms of variability: The inability to metabolize phenylalanine resulted from a genetic deficiency. Fifty years ago, a scientist discovered its mechanism and prescribed a palliative diet deficient in phenylalanine. Today, PKU is cited as exemplary of nature–nurture interaction (e.g., Sternberg et al., 2005). However, a crack has appeared: Science has given each person the freedom to be tested and to follow the diet. Even for a physiological disorder such as PKU, nature and nurture do not account for all influences; the ability to choose likely accounts for most of the variability today, thus violating the exclusivity assumption.

If one extends this example, on that theoretical day when human DNA is completely understood, Galton’s (1869/1892) paradigm will be refuted definitively: Scientists will have maximized human choice while minimizing adverse aspects of genetic inheritance.

If a physiological disorder might result from more than nature and nurture, then what about psychological traits, in which the effects of human abilities such as choice, free will, responsibility, meaning, purpose, and spirituality may be magnified? If PKU provides even a single exception to the universality assumption, then Galton’s paradigm must be validated for each trait studied.

A third assumption, complementarity, must also be addressed: Nature and nurture constitute a linear dichotomy, even in interaction. Note the wording of Sternbergs et al.’s (2005) translation of Galton’s paradigm quoted earlier: Nature and nurture “add to” one. As Sternberg et al. (2005) noted, “Heritability has a complementary concept, that of environmentality” (p. 53). This dichotomous structure requires that variation from any other source automatically be included under heritability, environmentality, and/or their interaction, thus precluding its consideration outside of the paradigm (Biddell & Fischer, 1997). In the example of PKU, if heritability is minimized, is environmentality correspondingly increased? However, as mentioned earlier, the ability to choose likely has become the greatest source of variability.

A dimensional ontology allows a more parsimonious inclusion of these factors. Viktor Frankl (1967) noted that the person lives in three interpenetrating dimensions: soma (the physical body), psyche (the emotions and intellect), and the noetic (the soul). The noetic dimension includes free will, responsibility, choice, spirituality, and the unique meaning capacity of Homo sapiens. Although animals share soma and psyche with humans, the noetic is defined as that which differentiates us from animals (Frankl, 1967). In this ontology, genetic expression is somatic in origin—genes synthesize amino acids, pure and simple; nurture occurs in both soma and psyche. From this perspective, nature and nurture are dimensionally different rather than complementary.

The existence of dimensional causes other than heredity and environmentality violates all three assumptions. Theorists have proposed factors that operate in conjunction with genetics and environment, such as symbol systems (language, science, math, musical notation; Gardner, Hatch, & Torff, 1997) and human agency (choice, free will; Biddell & Fischer, 1997; Frankl, 1967). Assuming that animals do not have language, science, and math, nor the choice and free will needed for agency, these distinctively human variables are noetic (Frankl, 1967). How do they affect nature–nurture research?

Studies of twins with regard to language development and IQ invariably involve the measure of noetic symbol systems, using the manipulation of language and/or representations of math or logic. Similar studies of substance abuse ignore the noetic dimension: The most successful treatment involves 12-step programs that invoke a “Higher Power” to strengthen the ability to choose. Twin studies of career selection paradoxically assume that only nature and nurture determine choice, precluding the Jungian sense of “finding one’s
calling.” For each of these fields of study, the noetic dimension is a confounding variable, its variability subsumed into genetic and/or environmental components.

From a clinician’s standpoint, the small arena of choice is likely the most powerful fulcrum available for change. The healer’s role is arguably to transform the client’s sense of agency from “insignificant” into a force capable of harnessing and transcending nature and nurture. With choice come responsibility and the realization of freedom, which cannot occur without meaningful and purposeful options. Indeed, it is through exercising our ability to choose that we become fully human. Galton’s (1869/1892) paradigm ultimately assumes that the person has no true freedom. To rephrase Frankl (1967), nature and nurture represent our fate, whereas the noetic represents our capacity to take a stand toward that fate.

For decades, Galton’s (1869/1892) nature–nurture paradigm has anchored psychological research and theory. Though renowned, his idea and its derivations were never scientifically validated. Galton’s idea, and all studies based on it, bear reconsideration. Inclusion of the noetic dimension and its intercourse with genetics and environment allows the researcher to consider the person in studies of human development. It is time for a new paradigm: Nature, Nurture, and the Noetic.

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Carey (2006, this issue) cited in his response to our article (Sternberg, Grigorenko, & Kidd, January 2005) a study by Tang et al. (2005) showing that “of 3,636 subjects of varying race/ethnicity, only 5 (0.14%) showed genetic cluster membership different from their self-identified race/ethnicity” (p. 268). Carey (2006, p. 176) then stated that he would very much like to hear a response to this finding from Sternberg et al. (2005), who maintained that “race is a socially constructed concept, not a biological one” (p. 49), that refutes those physical correlates of ancient population dispersions “as deriving from some imagined natural grouping of people that does not in fact exist, except in our heads” (p. 51).

We are glad to provide a response.

Imagine the case of Dr. Smith. Dr. Smith has a hypothesis that the proper basis on which to classify people’s race is skin color, which has so often been used (mistakenly, he believes), but rather eye color. He discovers some interesting facts. Whereas 0.14% of a sample showed genetic cluster membership different from their self-identified race/ethnicity based on skin color and other such criteria, not even one person misidentified genetic membership on the basis of eye color. He is impressed. He then discovers that race defined by eye color is quite powerful. Blue-eyed races tend to live more in Northern climes, and brown-eyed races closer to the equator. Blue-eyed people, on average, are more susceptible to sunburn and concomitants of it. Blue-eyed races have health problems, on average, more typical of people in Northern climes, and brown-eyed races, those more typical of equatorial climes. Although the effects are not whopping, they are statistically significant.

There are even differences, on average, in the distribution of languages these races speak. Dr. Smith even notes that, in earlier times, eye color actually was used as a basis for classifying people on the basis of race, which gives historical weight to his scientific finding. (As an example, Sternberg’s mother escaped Nazi-occupied Austria only by virtue of her blue eye coloring, which suggested “Aryan” racial membership to police guards on a train.) The point of the apocryphal story above is that correspondence between self-identification and biological characteristics does not form a suitable basis for assigning people to “races.”

Moreover, it appears that Carey’s (2006) response to our article can be explained, at least partially, by his incomplete understanding of the data, analyses, and results presented in Tang et al. (2005). First, the Tang et al. study was done using samples primarily from the United States, where the primary ethnic groups are derived from very different parts of the globe: European (primarily Western European), African American (of mixed West African and European origin), East Asian (Chinese, Japanese, Vietnamese, and other groups not generally distinguished by U.S. policy-makers but genetically distinguishable), and Hispanic Mexican American (of mixed Native American and European origin).

Thus, there exist noticeable genetic differences that parallel the origins and culture differences among these groups, specifically, the immigration patterns from different parts of the globe into the United States. Correspondingly, given where these culturally defined groups came from geographically, it is not surprising that they have genetic differences. More relevant to the point that we make—that is, of the continuity of genetic diversity among various ethnic groups and, correspondingly, the artificial nature of the concept of race—is the observation that African Americans can have considerable differences in their ancestral contributions, depending on where, culturally and geographically, their ancestors came from. In the United States, culturally (socially) and historically, anyone with any African ancestry is viewed as or referred to as “African American.” Consider the children of Thomas Jefferson and Sally Hemings. She was already half European (a half sister of Thomas Jefferson’s wife), and their children were therefore three quarters European, yet they were still socially slaves and “African American.” Depending on how such individuals self-identify and how the genetic clusters are derived statistically, they will cluster with “pure” Europeans or a “partially African” cluster and will either fit or be outliers for these clusters.

Second, we also note that the analysis in Tang et al. used the “no admixture” option; in other words, the analyses did not allow for a careful examination of sources and structure of variability “added” by mixed ethnic marriages, which, clearly, would minimize estimates of within-group variation and maximize estimates of between-groups variation. Third, despite the