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doi:10.1017/S1537592713000893

Nature and Nurture

In the Introduction to this volume, Peter K. Hatemi and Rose McDermott assert that "nature–nurture" is an "outdated juxtaposition," an "atavistic dichotomy," and a "dualism [that] remains absurd on its face" (p. 3). This a surprising assertion in a book titled "Man is *by Nature* a Political Animal." In rejecting this dichotomy, they claim to "support the dominant interpretation across sciences which assumes the inextricable interaction between biology [by which the authors intend both genes and "physiology"] and environment in determining the growth and development of any given biological organism within a particular ecological context" (p. 3). It would be characteristic of this dichotomy to assume that genes could contribute to phenotypic variation in a manner that did not involve "inextricable interaction" between genes and the environment; likewise, it would be absurd to assume that a given physiological response could develop in a manner that did not involve environmental interaction. Such assumptions, however, are ubiquitous in this volume.

For example, Lindon J. Eaves, Hatemi, Andrew C. Heath, and Nicholas G. Martin (Chap. 4), after considering how to partition the contribution of genes versus environment to variation in political behavior, note a "potential limitation" of their models: "[T]hey are linear and additive [and] do not take into account . . . interactions between genes and environments" (p. 157). But if biology (which includes genes) and environment "remain codependent and interactive," what is the value of a model that treats them *as if they are not interactive*? It is one thing to acknowledge the shortcomings of a model; it is another to construct a model on the basis of a principle that one simultaneously claims is "absurd on its face." A similar "atavistic dichotomy" is found in Kevin B. Smith and John R. Hibbing's (Chap. 7) account of the development of "physiological" differences: "The evolutionary process has imbued all humans with an autonomic nervous system, but genetic variation has instilled a remarkable amount of diversity in the specific response levels of individuated nervous systems to standardized stimuli" (p. 225). Hence, diversity in autonomic responses is ascribed entirely to "genetic variation" (inasmuch as "genes build physiology" [p. 225]) in defiance of "the inextricable interaction between biology and environment."

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Consider the role of the environment in shaping the autonomic nervous system's stress response (the "fight or flight" response), inasmuch as it provides an illustration of the kind of biology–environment interaction that is conspicuously absent from this volume, as well as the evolutionary significance of such interaction. Differences in early maternal care translate into differences in the stress response of offspring, and cross-fostering studies have shown repeatedly that the key determinant is the rearing rather than the biological mother.[1] Offspring that receive low levels of maternal care exhibit, as adults, increased levels of stress as measured behaviorally and physiologically, with females also exhibiting earlier mating behavior and lower levels of maternal care, while high maternal-care offspring exhibit, as adults, the opposite phenotypes. Significantly, these behaviors are associated with postnatal alterations to the epigenome, the complex, environmentally reactive, biochemical system that regulates the accessibility of genes to transcription factors and, thereby, the extent to which any given gene can be transcribed. The observed epigenetic changes, and corresponding changes in gene transcription rates, are consistent with increased and decreased stress responses. In other words, the environment reprograms the epigenome and the epigenome reprograms gene transcription rates.[2]

This is an example of *phenotypic plasticity,* the ability of an organism to modify developmental trajectories and change phenotypic state or activity in response to variations in environmental conditions. Developmental phenotypic plasticity evolved because it is *adaptive,* promoting Darwinian fitness by enhancement of survival and reproductive success by using environmental cues to optimize the life-course strategy. A heightened stress response and diminished maternal care enhance the mother's chances of survival in a dangerous ("high stress") environment, but by shaping the stress response and mating and rearing behavior of her offspring, she enhances their survival as well. Offspring behavioral plasticity enables the mother to adjust offspring phenotype in response to the environment she inhabits. Adversity during perinatal development can forecast an increased level of demand in the environment the offspring will occupy. Under such conditions, the animal's best interest is to enhance its behavioral (e.g., vigilance, fearfulness) and endocrine responsiveness to stress. These responses promote detection of potential threat, avoidance learning, and metabolic/cardiovascular responses that are essential under the increased demands of the stressor.

Such environmental shaping of physiological response is not to be confused with either "G × E" ("gene × environment" interaction or "gene-environment interplay" [Chap. 5]) or what Eaves and his colleagues refer to as the "ontogenetic process" (p. 154). G × E is the idea that a particular polymorphism will result in a given phenotype in one environment and a different phenotype (or no

phenotype) in a different environment. The "ontogenetic process" refers to purported (and inexplicable, both in terms of physiology and adaptive value) changes in the heritability of a particular phenotype at a particular point in life as indicated by the twin-study methodology. Neither G × E nor the ontogenetic process involves phenotypic plasticity, that is, modification of developmental trajectories to meet the demands of a particular environment and, hence, neither has any apparent adaptive value. Neither epigenetics nor phenotypic plasticity—mechanisms that embody "the inextricable interaction between biology and environment"—is mentioned anywhere in this volume, although the former represents the most important development in molecular genetics and the latter the most important development in evolutionary biology over the past 50 years.

Both absurdly high heritability estimates—60% for political participation (p. 215), 60% for strength of political partisanship (p. 216), 50%–70% for attitudes toward homosexuality and gay rights—and the assumption that single polymorphisms can predict complex political behaviors (Chaps. 5, 6, 8) reinforce, rather than overcome, the traditional nature–nurture dichotomy. If we were to take such heritability estimates seriously, then we would have to conclude that humans were less biologically influenced by, and reactive to, their environments than fruit flies (heritability of aggression in fruit flies is ~10%).[3] Aggressive strains of fruit flies exhibit differences in transcription rates of more than 1,539 genes as compared to controls.[4] In a situation such as this, no single gene could possibly predict aggressive behavior, yet we are told that a single polymorphism can predict, for example, voting (pp. 218–19, 268).

The assumption that a single polymorphism could predict a behavior such as voting results from conceptualizing complex behaviors in the manner of "simple" genetic disorders (p. 27):

> [D]owns syndrome results from just a small genotypic and biological variation, a relatively small variation that can alter an entire set of social and physical behaviors. Considering that we have endless small genetic differences across the entire population, the logical assumption would be that these differences also affect attitudes and behaviors, including political behaviors.

This is not a logical assumption but, rather, a profound error. Huntington's disease is a neurological disorder caused by a mutation on a gene (HTT) that is coded for the protein huntingtin. Symptoms include changes in personality and cognitive ability and, over time, the eventual loss of the ability to walk, talk, think, and reason. Can we infer from this that polymorphisms of single genes are likely to influence a wide array of complex normal behaviors associated with the healthy human brain (such as, e.g., voting)? Certainly not. An HTT gene mutation has far-reaching phenotypic consequences because the end result of an abnormal huntingtin protein is neuronal degeneration. But voting, for example, is not a monogenic disorder, or an oligogenic disorder, or a complex polygenic disorder. Normal complex human behavior is not a cluster of disorders, nor is it a cluster of distinct "behaviors," each behavior predicted by a gene or set of genes. Rather, normal behavior is the integrated output of a highly integrated biological system interacting with a particular environment.

Physiology

In their discussion of physiology and political ideology, Kevin Smith and John Hibbing reference a study by Douglas Oxley and his coauthors[5] in which the latter "used SCL [skin conductance levels] to detect physiological differences in response to threatening stimuli between conservatives and liberals" (p. 233). The Oxley coauthors assumed that what they were measuring using SCL was a "threat response" because they showed participants what they classified as three "threatening images." But whether or not an image, for example, of a woman laughing at a large spider crawling on her face, is "threatening" is certainly in the eyes of the beholder. This point is important because measures of SCL are *nonspecific* measures of arousal *of any kind;* for example, surprise, joy, disgust, empathy, or interest can all be manifested by *identical* increases in SCL. Furthermore, Smith and Hibbing claim that this study showed differences in "threat responses" between "conservatives" and "liberals," when, in fact, associations were purported (and highlighted) for only a few items of the Wilson-Patterson "conservatism" index. Nonetheless, the general takeaway of this study seems to have been that "conservatives have greater threat responses than liberals." But, of course, what distinguishes liberals and conservatives is not that one or the other has greater levels of "fear" in some general sense, but that (to continue with the language of fear) they fear different things: Liberals fear global warming and gun violence; conservatives fear the economic impact of environmental restrictions and the loss of Second Amendment rights.

Smith and Hibbing (p. 233) also reference a study by K. Smith and colleagues[6] purporting to show that "conservatives" exhibit greater "disgust responses" than "liberals" (based upon the same dubious methodology), and the authors of this study speculate that greater disgust responses toward, for example, homosexuality, might explain conservatives' opposition to gay marriage. But how does one get from a feeling of disgust to a political point of view? In and of itself, a reaction of disgust has no political implications whatsoever. The fact that I find the smell and taste of Limburger cheese disgusting does not entail that I think Limburger should be banned and those who make or consume it imprisoned (or that its consumption by adults should be tolerated, but behind closed doors). There is simply no way to derive political

principles from a sensation of disgust without presupposing a host of principles to which the sensation of disgust itself is supposed to give rise (e.g., how far-reaching the authority of governmental power should be, or whether disgust is a legitimate reason to ban an activity and punish those who engage in it). Furthermore, what does my disgust with Limburger cheese have to do, for example, with my disgust at the current state of American politics (besides the use of the word *disgust* in both instances)? The latter is a consequence of my political and moral views regarding American politics; the former is a consequence of my sense of taste and smell.

Politics and Religion

To be a member of a particular religion (as distinguished from "religiosity" in some abstract sense [p. 35, Chap. 4]) is the only complex behavior that, as far as I can tell, practitioners of behavior genetics appear reluctant to claim is heritable or predictable by a polymorphism. But this is not, despite what Hatemi and McDermott (and many others) claim (p. 35), because this behavior has not been found to be heritable. For example, being a Born Again Christian is, we are told, 65% heritable,[7] Sabbath Observance is 35% heritable,[8] a belief in Biblical Truth is 25% heritable,[9] and religious affiliation in general exhibits a moderate, "statistically significant" heritability.[10]

Note how such findings are handled, however. For example, despite the fact that *Sabbath* observance and belief in the *Bible* are said to be heritable, they are treated not as *Christian* religious doctrines but as examples of "religious *conservatism*" (minus the specific religious affiliation).[11] In reporting that being a Born Again Christian is 65% heritable, the authors note that "the wording of this particular question forces us to interpret this finding with caution (i.e., it combines a generic commitment to Jesus Christ that may be shared among all Christians with the conservative Protestant-specific concept of being 'born-again')," [12] and they classify this not as the heritability of being a Born Again Christian but the heritability of "transformations and commitment." When A. B. Heath and colleagues[13] found that polymorphisms of the ADH2 gene predicted *Anglicanism,* they did not publish a study titled "Gene Predicts Anglicanism," but rather speculated (quite sensibly) that these findings were driven by population stratification.[14] And when Brian D'Onofrio and his colleagues discovered that the heritability of religious affiliation was "statistically significant," they speculated that genetic factors play a role in "determining which children decide not to follow in the religious traditions of their parents."[15] In other words, following the religious tradition of one's parents was distinguished from the religious tradition itself!

Why are religion and politics treated in such different ways? It is likely because the absurdity of claiming, for example, that a gene predicts Anglicanism, or Confucianism, or Zoroastrianism, or that they are "heritable," is manifest even to practitioners of behavior genetics; that is, the historical and cultural particularity of religious commitments is as manifest as their mode of transmission. That twin studies generate results that even partisans of the methodology acknowledge as absurd is further evidence that they are to many what they have always seemed to be: an obviously confounded, unreliable methodology. Yet political ideologies, which exhibit the same rate of parent-to-child transmission as religious "ideologies," are just as culturally and historically particularistic and manifold, a fact obvious to anyone even casually acquainted with the history of political beliefs and practices.

This particularity is masked by the almost universal tendency to speak of "liberalism" and "conservatism" as if these were the two necessary and universal modes of political behavior (while typically using measures of twenty-first-century American liberalism and conservatism; *passim* in the current volume). Assuming that all political ideologies that have ever existed are manifestations of either liberalism or conservatism is like assuming that all religions that have ever existed are manifestations of either Christianity or Judaism. A claim that being a (twenty-first-century American) liberal or conservative is 50% heritable (p. 35) is as absurd as the claim that being a Born Again Christian is 65% heritable (construed as a claim about *Christianity*); the claim that a gene predicts (twenty-first-century American) liberalism (p. 219) is as absurd as the claim that a gene predicts (twenty-first-century) Anglicanism.

Conclusion

Although the editors begin with the promising claim that nature-nurture is an "atavistic dichotomy," this dichotomy is reinforced throughout the volume for at least three reasons: 1) "Genes" and "environment" are treated as independent causal forces whose contribution to variation in complex behaviors can be precisely quantified; 2) claims that polymorphisms can predict phenotypes ignore, for the most part, the entire developmental (i.e., environmental) history starting with fertilization and ending with, for example, entering a voting booth; and 3) "physiology" and "environment" are treated as if the former develops in a manner independent of the latter. I agree that political beliefs are not simply, or even mostly, a matter of reason and reflection, but the assumption that political behavior is reducible to, or derivable from, physiological responses is clearly incorrect. Finally, the difficulty of discussing the biology of politics is compounded by the fact that specific political beliefs and practices resemble what most practitioners of behavior genetics claim are not "biological" (despite findings to the contrary based on their own methodologies), namely, specific (doctrinal) religious beliefs and practices.

Notes

1 Champagne and Curley 2009.
2 For an overview and references, see Charney 2012.
3 Edwards et al. 2009.
4 Ibid.
5 Oxley et al. 2008.
6 Smith et al. 2009.
7 Bradshaw and Ellison 2008.
8 Ibid.
9 D'Onofrio et al. 1999.
10 Ibid.
11 Bradshaw and Ellison 2008; D'Onofrio et al. 1999.
12 Bradshaw and Ellison 2008, 542.
13 Heath et al. 2001.
14 Population stratification refers to differences in allele frequencies among populations due to genetic ancestry. For population stratification as a source of spurious correlations in gene association studies, see Charney and English 2012 and 2013.
15 D'Onofrio et al. 1999, 967.

References

Bradshaw, Matt, and Christopher G. Ellison. 2008. "Do Genetic Factors Influence Religious Life? Findings from a Behavior Genetic Analysis of Twin Siblings." *Journal for the Scientific Study of Religion* 47(4): 529–44.

Champagne, Frances A., and James P. Curley. 2009. "The Trans-Generational Influence of Maternal Care on Offspring Gene Expression and Behavior in Rodents." In *Maternal Effects in Mammals,* ed. Dario Maestripieri and Jill M. Mateo. Chicago: University of Chicago Press.

Charney, Evan. 2012. "Behavior Genetics and Postgenomics." *Behavioral and Brain Sciences* 35(5): 331–58, 381–410. http://tinyurl.com/Charney-Behavior-Genetics (accessed January 10, 2013).

Charney, Evan, and William English. 2012. "Candidate Genes and Political Behavior." *American Political Science Review* 106(1): 1–34.

———. 2013. "Genopolitics and the Science of Genetics." *American Political Science Review* 107(2): 382–95.

D'Onofrio, Brian M., Lindon J. Eaves, Lenn Murrelle, Hermine H. Maes, and Bernard Spilka. 1999. "Understanding Biological and Social Influences on Religious Affiliation, Attitudes, and Behaviors: A Behavior Genetic Perspective." *Journal of Personality* 67(6): 953–84.

Edwards, Alexis C., Liesbeth Zwarts, Akihiko Yamamoto, Patrick Callaerts, and Trudy F. C. Mackay. 2009. "Mutations in Many Genes Affect Aggressive Behavior in *Drosophila melanogaster.*" *BMC Biology* 7(1): 29. http://www.biomedcentral.com/content/pdf/1741-7007-7-29.pdf (accessed January 10, 2013).

Heath, A. C., J. B. Whitfield, P. A. F. Madden, K. K. Bucholz, S. H. Dinwiddie, W. S. Slutske, L. J. Bierut, D. B. Statham, and Nicholas G. Martin. 2001. "Towards a Molecular Epidemiology of Alcohol Dependence: Analysing the Interplay of Genetic and Environmental Risk Factors." *British Journal of Psychiatry* 178(suppl. 40): s33–s40.

Oxley, Douglas R., Kevin B. Smith, John R. Alford, Matthew V. Hibbing, Jennifer L. Miller, Mario Scalora, Peter K. Hatemi, and John R. Hibbing. 2008. "Political Attitudes Vary with Physiological Traits." *Science* 321(5896): 1667–70.

Smith, K., D. Oxley, M. Hibbing, J. Alford, and John Hibbing. 2009. "The Ick-Factor: Physiological Sensitivity to Disgust as a Predictor of Political Attitudes." Presented at the Annual meeting of the Midwest Political Science Association, Chicago.