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Extending and testing Tom Bouchard's Experience Producing Drive Theory

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ABSTRACT

In several papers in the 1990's, Tom Bouchard outlined and developed his Experience Producing Drive Theory, the idea that complex organisms have evolved through natural selection to be agents actively seeking circumstances in which they can optimally survive. Thus genes exert their influences on the development of patterns of human and other animal behaviors known as traits through their control of motivations, preferences, and emotional responses. Over time, these motivations, preferences, and emotional responses drive the acquisition of experiences that result in the development, practice, and pursuance of skills, habits, patterns of response, and environmental circumstances. In turn, these reinforce the underlying drivers through the creation of gene–environment interactions and correlations. This paper describes how recently emerging understandings of gene–environment interplay and behavioral genetic methodology can be used to extend and test this important theory.

> "EPD-R theory has the virtue of antagonizing participants on all sides of the debate regarding the roles of genes, environment, and evolution in the shaping of human personality." (Bouchard, 1997, p. 63).

Most scientists probably would not consider antagonizing their colleagues with their pet theory to be advantageous, but it is characteristic of Tom Bouchard that he did. At least arguably, the social sciences would be better off if more of their practitioners thought as he does. Tom has written that he considers the purpose of science to be to construct and test theories of how the world and its inhabitants 'work'. When our scientific theories are good, they can tell us that if we do *X* under conditions *Y*, the result will probably be *Z*. They cannot tell us, however, whether *Z* is the optimal result or even if *Z* is a result for which we should strive at all. That is the role of ideology: to work toward implementation of policies and choices that will bring about desirable results. But ideology is ultimately dependent on science. No matter how desirable our goals may be, they cannot be attained if they are based on a faulty scientific understanding of the underlying causal chain. As Tom and others (Kendler, 2006; McIntyre, 2006) have noted, ideology often gets confused with social science because so many social scientists want so badly to see a particular kind of result. And theories that 'sound good' (as if they can lead to desired results) to many are all too likely to receive acceptance without rigorous testing. If a theory that does not sound good to anyone can avoid being overlooked and can survive the testing that everyone will want to put it through, there must be something to it.

1. So what is Experience Producing Drive Theory?

Experience Producing Drive (EPD) Theory is the idea that complex organisms have evolved through natural selection to be agents actively seeking circumstances in which they can optimally survive. Thus genes exert their influences on the development of patterns of human and other animal behaviors known as traits through their control of motivations, preferences, and emotional responses. Over time, these motivations, preferences, and emotional responses drive the acquisition of experiences that result in the development, practice, and pursuance of skills, habits, patterns of response, and environmental circumstances, which in turn reinforce the underlying drivers through the creation of gene–environment interactions and correlations. In this basic form, the theory was originally proposed by Hayes (1962) to integrate then-recent relatively independent research findings in the areas of motivation, behavior genetics, and intelligence. At the time, Hayes was well known, with his wife Vicki, for having raised a chimpanzee in their home during the 1940's (Hayes, 1951).

Hayes (1962) focused on intelligence in writing about the theory, and one of the primary ways in which Tom Bouchard extended the theory was to consider it explicitly applicable to all individual differences of interest to psychologists: personality, mental abilities, interests, values, attitudes, and idiosyncratic traits unique to

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each individual (Bouchard, 1997). In fact, however, exposition of the theory entails the hypothesis that all of these areas of individual differences are related to individual differences in intelligence (Gottfredson, 1997); study of any one is simply a matter of selection of focal point. Here, I will maintain Hayes' original focus on intelligence, with the understanding that Bouchard's extension to all areas of individual psychological differences was appropriate. Within that focus, some of Hayes' (1962) comments are as fresh today as they were when he wrote them, and thus bear repeating.

"As Hebb (1958, p. 246) put it, Binet 'learned how to measure something without any very clear idea as to what it was he was measuring'. Today, some 50 [now more than 100] years later, we may measure it a bit more satisfactorily, and we can certainly measure it with a greater variety of techniques; but we are still vague about what 'it' is" (Hayes, 1962, p. 299). Hayes goes on to note that being more specific about what we mean by intelligence is not a matter simply of defining it more clearly. It is instead a matter of understanding *how* genetic and environmental influences are involved in what is patently a characteristic that *develops* throughout the lifespan. There are characteristic patterns associated with many stages of the lifespan, but also appear to be extensive individual differences in developmental patterns within each of those stages. Offering an explanation of intelligence that recognizes these properties was the explicit purpose of EPD theory.

Hayes (1962, p. 337) took a strong position with respect to intelligence, at least as measured by IQ tests. To him it was "nothing more than the accumulation of learned facts and skills". That is, "innate intellectual potential consists of tendencies to engage in activities conducive to learning, rather than inherited intellectual capacities as such". To make sense of this, Hayes had to manipulate the commonly used meaning of the term 'drive'. To most motivation and emotion psychologists, 'drive' has referred to some innate mechanism that perceives some deficit state and acts to initiate behavior that will eliminate the deficit. For example, after some period without food consumption or sleep, an organism will seek food or a safe place to sleep, and if the drive becomes strong enough, the organism will eat even things that are not food or fall asleep standing up in the midst of a fight. But, as Hayes explained it, rats, perhaps unlike humans, show little enhanced interest in food after a period of deprivation, and they respond to food-rewarded learning tasks only after they have been on a deprivation schedule for awhile (Ghent, 1957). This suggests that they should starve, as they do not express interest in food until they have had the experience of eating after having been deprived of food. They are apparently saved by a tendency continuously to consume small amounts of food as available, whether they actively need it or not. In discussing EPD's, Hayes used the term 'drive' in this latter sense as the neural mechanism responsible for an organism's tendency to pursue certain activities regardless of any deficit state. He maintained that these drives were under genetic influence, and that intelligence developed through the skills and knowledge acquired in the process of engaging in the activities inspired by these drives.

No doubt related to his interest in and experience with raising a chimpanzee in his home, Hayes had a comparative psychological outlook that contributed to his ideas about EPD's. He was very aware that human individuals had a massive advantage over other animals in using EPD's to build intelligent performance because they could make use of language, and, in particular, written materials to access the experiences of those who had gone before them. Written (and now of course also electronic) materials can also be used to store information so that it does not need to be maintained actively in memory. Hayes also touched on how EPD's may be involved in the development of both intelligence *per se*, whatever it is, and the collection of skills and knowledge we use to test it, and I offer some updated thoughts on this below.

2. And what is EPD-R?

Bouchard (1997; Bouchard, Lykken, Tellegen, & McGue, 1996) found much in Hayes' EPD theory to recommend it as an explanatory mechanism to integrate the evidence for the pervasiveness of genetic influences on all psychological characteristics with the presence of strong environmental influences but the absence of evidence of effects of any *particular* measured environmental influences. At the same time, he could not reconcile Hayes' proposition that learning capacity was completely general, with no individual differences in learning or information processing capacity, with the empirical data. He therefore suggested that mechanisms involving specialized structural features of the brain that do show individual differences in capacity drive behavior and the acquisition of experience. He pointed out that this is consistent with the Darwinian idea that organisms have evolved to do something; they are active in their environments rather than passive. He posited that the brain mechanisms involve sensitivity to specific features of the environment in which the organism evolved, and that the drives they support are self-reinforcing. In complex organisms, he noted that these mechanisms will tend to be modifications and elaborations of mechanisms previously evolved to carry out other functions (Darwin's descent with modification due to tinkering, as described by Jacob (1977)) and so are unlikely to be unitary or simple.

Bouchard et al.'s (1996) modification of Hayes' (1962) theory proposed that inherited predispositions to develop abilities are evolutionarily selected sensitivities to ubiquitous environmental features. This was Bouchard et al.'s (1996) conception, and it works well when we consider actively extended phenotypes such as beaver dams that are constructed by all members of the species. It also works well to explain universally human features such as the acquisition of language. I believe it requires some modification, however, to account for individual differences in intelligence among humans. That is, the environmental features involved cannot be necessarily ubiquitous, or every individual would have the same EPD's and individual differences in intelligence would solely be differences in capacity, as they appear to be for language. That is, with language, emergence of use of language is almost universal, but there are large individual differences in vocabulary, verbal fluency, etc. Instead, the environmental features involved in intelligence appear to be commonly but not constantly occurring, so that individual differences in the kinds of activities inspired by EPD's are preserved through balancing selection. This is what makes it possible for EPD theory to account for the range of specific abilities that show individual differences independent of general intelligence.

So long as the organism is exposed to these environmental stimuli during the appropriate development periods, Bouchard's (1997) revision of EPD theory posits that the organism is genetically predisposed to pay attention to them, thus acquiring particular kinds of information. This is the case whether we are considering abilities that are expressed universally across a species or specialized skills and abilities not necessarily shared by all within the species. The neural mechanisms are thus motivational, and must mediate the relevant skills and abilities. Lack of timely availability of exposure to the relevant environmental stimuli may retard their development, but a key point here is that the organism is actively involved in seeking the relevant environmental stimuli, canalized to make use of the experience of the stimuli, and, having made use of it, that much more prepared to seek and make use of the next one.

Importantly, Bouchard (1997) emphasized that EPD's may not necessarily contribute to better environmental adaptation. Environments are generally complex and contain many different kinds of stimuli, and individual humans may have many different

combinations of EPD's, not to mention conflicting goals and objectives. Matches among EPD's, goals, and environmental stimuli may differ in strength or quality. Behavioral decisions that are advantageous in the short run may be disadvantageous in the long run. Environmental responses to behavioral choices are not always constructive. The net result is that people may increase their risks of both physical and psychiatric illness by responding to EPD's that lead them to stressful environments that exacerbate their EPD's, which in turn increase their environmental stressors. An example could be a drive to express anger aggressively, leading to punishment that is at least perceived to be unfair, which in turn inspires further anger and aggressive behavior and punishment.

Though they have not articulated them as fully, others have voiced themes similar to those of EPD theory. For example, Roberts and Caspi (2003) have discussed several important principles of personality development, including the corresponsivity principle. This is the idea that life experiences deepen the characteristics that led people to those experiences in the first place. Scarr (1996; Scarr & McCartney, 1983) have noted that people are not randomly assorted into their environments: they make active choices that have environmental consequences. And Plomin (1994, chaps. 2–3)) called for a new approach to understanding the environment, or "the nature of nurture." Crucially, as noted by McGue, Bouchard, Lykken, and Finkel (1991), this requires the development of measurement instruments and observational and statistical methods that can identify and quantify the individual's role in creating experience. These tools remain largely outstanding.

3. How can we test EPD Theory?

Any good theory needs to be testable. EPD theory is a difficult one to test, however, because it steps directly into what is probably the messiest problem facing the social sciences: genetically influenced multicollinearity (Rutter, 2006). Poor environmental conditions ranging from toxic pollutants and high crime rates to dilapidated housing and lack of access to health care are associated with personal characteristics ranging from lack of education and low IQ and income to poor mental and physical health and antisocial behavior. At the other end of the spectrum, things are also tied together: Good environmental conditions ranging from clean air and water and safe and stately neighborhoods to high-tech health care and schools with both state-of-the art enrichment and special education programs are associated with personal characteristics ranging from professional occupational status to moderate alcohol consumption, regular exercise, and feelings of self-efficacy. And of course the environmental conditions are correlated with each other and so are the personal characteristics. Moreover, it has become evident that the personal characteristics and even the environmental conditions generally show genetic influences (Plomin, 1994; Turkheimer, 2000). This makes identifying the causal effects of any one variable on any one outcome, whether environmental or personal characteristic, extremely difficult, and makes clear that the failure by many social scientists to recognize the involvement of genetic influences in unfortunate life outcomes was simply wrong (Bouchard, 1993; Rowe, 1994; Rutter, 2007; Scarr, 1997). We know that multicollinearity exists and that it is genetically influenced. What we do not know is how it comes about and how manipulable it might be. EPD theory provides an explanation (arguably the most direct explanation extant) for how it comes about and thus could be helpful in figuring how manipulable it might be and how to go about it. In the process, however, the very possibility that EPD theory is correct disrupts our ability to make use of the kinds of behavior genetic analyses that would otherwise have been most likely to be effective in testing it and thus our ability to make inferences about the very mechanisms whose existence it proposes.

This is because, if in fact there are genes that control drives for experience rather than for traits themselves, their very actions introduce gene–environment correlations. And, to the extent that there are individual differences in the strengths of the genetically influenced drives and the kinds of experiences they influence, the actions of these drives also introduce gene–environment interactions. The behavior genetic analytical methods in use today all rely to varying degrees on the assumption that genetic and environmental influences are independent. When this assumption is violated because genetic and environmental influences are correlated and/or interact, the estimates from behavior genetic models are distorted. The ways in which they are distorted are systematic and can be specified (Purcell, 2002), but the extent of the distortion cannot, without knowing the magnitudes of the gene–environment correlation and interaction effects. These are generally precisely what we would like to measure, leaving us with a chronically underspecified model. Even methods such as the comparison of co-twin controls or discordant monozygotic twin pairs can only be used to test for purely nonshared environmental effects at a quasi-causal level. It is often clear from such analyses that the quasi-causal effects identified are so much smaller and even in the opposite direction to the overall observable association that the processes most important to understanding that association involve a crucial 'lump' of gene–environment correlation that the analyses have left completely unaddressed (e.g., Harden, Mendle, Hill, Turkheimer, & Emery, 2008).

Moreover, EPD theory is unabashedly developmental. As extended by Bouchard (1997), it implies that development within some genetically influenced reaction range depends on the match between EPD's and available environmental stimuli. Without the appropriate stimuli, the theory implies that the EPD-relevant characteristics would not emerge. But the issue of what might be relevant stimuli is complex. At the broadest level, measures such as sensation seeking or openness to experience might be expected to capture them (e.g., Bates & Shieles, 2003), but these measures are intended to capture curiosity and interest in novelty in general, and many EPD's may be much more narrowly focused, to the point that people strongly motivated by them may actually score rather low on such broad exploratory measures. For example, an EPD might involve fascination with manipulation of spatial configurations. A developing child pursuing this EPD might become a highly skilled chess player with intense interest in studying chess strategies if the opportunity to learn to play chess was present. But this child might be so busy with chess that s/he had little enthusiasm for playing a game such as basketball or attending a musical concert or trying a new food. At the same time, even this more narrowly focused EPD is likely to be flexible. If chess was not readily available in the child's environment but the opportunity to learn to make cabinets was, this same child might become a skilled cabinetmaker and furniture designer who still had no interest in basketball, music, or new foods. Of course, the cultures surrounding chess and cabinetmaking and furniture design are rather different and the child would likely absorb much from whichever of those cultures was available. This means that, even with the same EPD for manipulation of spatial configurations and the same capacity for absorption of relevant stimuli, the child could still grow to be a very different adult in one environment than in the other. We need measures that can identify both narrow and broad exploratory intensity of activity and that can do so in very young infants as well as children and adults.

The complications of testing EPD theory acknowledged, if EPD's exist, we should be able to track developmental trajectories of relations between interests and abilities. There should be genes for patterns of abilities such as verbal vs. spatial that function

independently of general intelligence (Johnson & Bouchard, 2007b; Johnson, Carothers, & Deary, 2009) and we should be able to link them to patterns of brain structure and function (Johnson & Bouchard, 2007a). We should be able to see evidence of individual differences in canalization; that is, learning should come more easily in areas of EPD's than in other areas. We should also be able to see individual differences in the salience of particular kinds of stimuli. Even in early infancy there should be consistent patterns of paying greater attention to some kinds of stimuli than to others, and these patterns should show individual differences (e.g., Connellan, Baron-Cohen, Wheelwright, Batki, & Ahluwalia, 2000). We should be able to see inter-individual differences in willingness to expend energy and tolerate frustration in the process of attaining some goal, and intra-individual differences of these same kinds in the process of attaining different *particular* goals. It should be possible to use some measure of exploratory or goal oriented behavior in infancy to predict later IQ. All of these measures may be difficult but should not be impossible to develop and validate.

4. Do we have any real evidence for EPD's now?

To date, the evidence for EPD's is indirect, but some does exist. To illustrate the kinds of evidence available, I will continue to focus on general intelligence. Though it is also consistent with other possible explanations, one of the clearest pieces of evidence is the pattern of the heritability of intelligence throughout the lifespan. Fig. 1 illustrates an informal synthesis of results from many studies carried out in many different samples over the past 50 years. As the figure shows, genetic influences increase from roughly 30% of variance in infancy to as much as 80% in adulthood, while shared environmental influences decrease from roughly 35% in infancy to effectively 0% in adulthood. Nonshared environmental influences decrease from roughly 35% of variance in infancy to about 20% of variance in adulthood, likely at least partly because we can measure intelligence better in adulthood than we can in infancy. Recognizing the intercorrelations among specific abilities that create what we know as general intelligence, if actualization of EPD's is possible primarily through shared environmental conditions such as parental assistance in making available educational materials and opportunities in areas of offspring interest and brighter parents do more of this, shared environmental and genetic influences on intelligence will be correlated. In most twin models that rely on the assumption that sources of influence are independent, variance involved in correlations between shared environmental and genetic influences falls into the estimate of shared environmental influences, causing it to be overstated (Purcell, 2002). As children grow more independent of their parents, however, actualization of EPD's should be expected to shift from correlation of genetic and shared environmental influences to correlation of genetic and nonshared environmental influences. But variance involved in correlations between nonshared environmental and genetic influences falls into the estimate of genetic influences, overstating it (Purcell, 2002). EPD's that create gene–shared environment correlations in childhood and gene–nonshared environmental correlations in adulthood could explain the pattern of heritability of intelligence throughout the lifespan.

Fig. 2 illustrates another kind of evidence. Cronbach and Snow (1977) developed and tested a model of the associations among general intelligence and educational practices such as mastery of particular curriculum goals, teaching approach (didactive vs. inductive), locus of responsibility for activity (teacher vs. student), and method by which lesson is conveyed (direct vs. indirect). They observed that it was common for students of high ability to learn more than they otherwise did when the instructional approach was open-ended and they were responsible for discovery, while students of low ability tended to learn more than they otherwise did when the instructional approach was structured and the teacher made clear exactly what they were to absorb. This observation has been replicated in situations ranging from college statistics (Shute, Gawlick-Grendel, Young, & Burnham, 1996) courses to elementary school reading lessons (Freebody & Tirre, 1985). Further exploration of this interaction and implementation of programs based on it have not exactly been embraced by educators. One

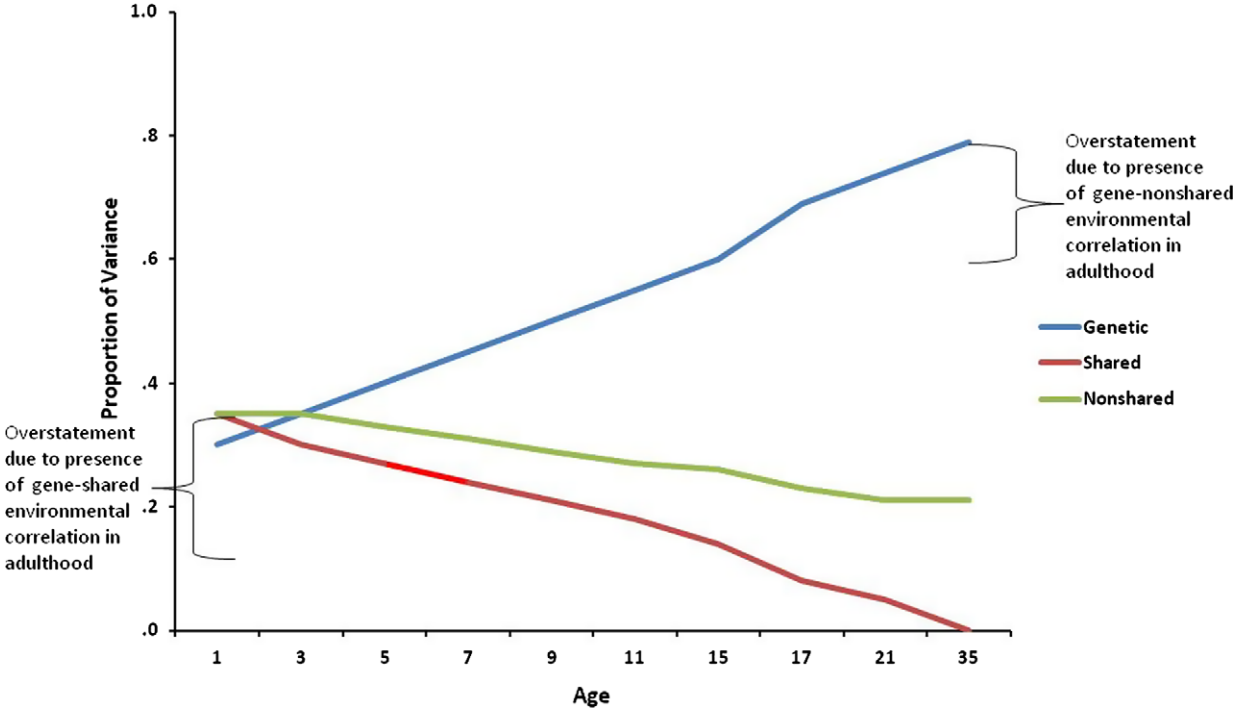

Fig. 1. Typical pattern of genetic influences on intelligence from early childhood to adulthood.

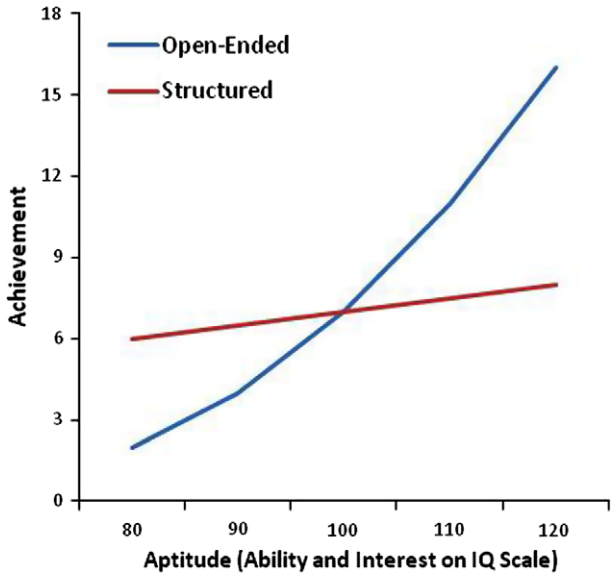

Fig. 2. Typical pattern of aptitude by educational program interaction based on Cronbach and Snow (1977).

reason for this may be that it stands directly between the two not always compatible goals of education in our society: to instill the basic skills needed for productive citizenship in all children and to help all children develop their intellectual potential to the fullest. Because there are so many political issues involved just in defining the first goal and so many resources are needed to reach it whatever the definition, the second goal often receives less attention.

If there are EPD's, we should be able to observe associations between occupational interests and specific abilities independent of general intelligence. The difficulty in getting at these associations, however, is that occupations tend to have strong associations with general intelligence (Gottfredson, 1986) and we do not generally have good ways of measuring specific abilities independently of general intelligence. Using an adult sample that included 42 different mental ability tests, however, (Johnson & Bouchard, 2007b) were able to develop measures of two dimensions independent of general intelligence. Individuals' positions along these distributions had demonstrable associations with expressions of occupational interests (Johnson & Bouchard, 2009). It would be preferable to be able to measure associations of this kind in children. A nice example is Raine, Reynolds, Venables, and Mednick (2002).

Evidence closer to the anecdotal comes from examination of the lives of extremely creative and illustrious people. Their energy in pursuit of their goals and persistence often in the face of enormous obstacles is a consistent theme (Simonton, 2000).

5. Concluding thoughts about EPD's and Intelligence

EPD theory posits specific kinds of transactions between genetic and environmental influences, acknowledging clear importance to both. Much of the evidence for the importance of both has come from the natural experiments afforded by twin and adoption studies. Both kinds of natural experiments support the importance of both kinds of influences. Monozygotic twins are consistently more similar in intelligence than are dizygotic twins, yet they are far from consistently identical (Plomin, DeFries, McClearn, & McGuffin, 2007). And infants with biological parents of low socioeconomic status (SES) adopted by parents of high SES tend to show IQ scores on the order of one standard deviation higher than those of their siblings reared by their biological parents (Capron & Duyme, 1989; van IJzendoorn, Juffer, & Klein Poelhuis, 2005), though their scores tend to be more highly correlated with those of their biological parents than those of their adoptive parents (Plomin et al., 2007). The adoption findings in particular are indicative of a rather direct effect of something about SES. There is nothing inherently contradictory about the coexistence of even high heritability and direct environmental effects (Visscher, Hill, & Wray, 2008), but they indicate that some environmental condition likely alters genetic expression patterns, raising the prospect of gene–environment interaction. There is evidence for greater expression of genetic influences on IQ but distinct from those on SES in higher SES environments (Harden, Turkheimer, & Loehlin, 2007; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003).

One possible, speculative, explanation for this pattern is that people may vary in the degree to which they carry *any* EPD alleles at all. From this perspective, people who carry few EPD alleles would not have low IQ. Instead they would tend to adapt to the intellectual level surrounding them within some reasonably broad range such as one standard deviation, creating primarily gene–shared environmental correlations. People who carry EPD alleles for particular kinds of experiences would create their own intellectual environments and gene–nonshared environmental correlations, and would tend to have higher IQ's regardless of their particular EPD's due to the tendency for reading, reasoning, and information processing to be involved in all kinds of intellectual skill development in our modern world. Of course, when EPD's involve particular kinds of skill development such as mathematics or scientific investigation, they could be associated with very high IQ's. Because EPD's build general as well as specific abilities in our educational environment and ability of all kinds builds SES, children without genes for EPD's would tend to be more common in lower SES environments.

In conclusion, Hayes (1962) introduced a powerful theory to explain how genes may be involved in behavior. Bouchard, 1997; Bouchard et al., 1996) modified and extended this idea in important ways, updating it and tying it more closely to evolutionary principles. Because of its breadth and the fact that it directly addresses the multicollinearity among important demographic and life outcome variables in our society, it is difficult to test. This does nothing, however, to diminish the possibility that it is an accurate description of the world in which we live; it only makes our jobs as scientists more difficult. Simpler ideas such as either genetic or environmental determinism and even important contributions from independently operating genetic and environment influences have been rather clearly unable to account for the empirical data about developmental life outcomes. We need explanations such as EPD theory that propose specific kinds of transactions between genes and environments, whether they 'sound good' or not. Bring on some more to compete with it and let us test them all.

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