An Evolutionary Perspective on Developmental Plasticity

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Abstract

In this essay, we advance the argument that variations in developmental plasticity should be treated as an individual-difference construct in research on environmental influences on human development. As guided by the diathesis-stress framework, past research has focused mainly on the development of dysfunction and negative outcomes in "vulnerable" individuals and the absence of such effects in "resilient" ones in response to adverse developmental experiences and environmental exposures. An evolutionary perspective challenges this traditional and prevailing framework for understanding person-X-environment interaction, leading to the view that there are individual differences in developmental plasticity, with those individuals most susceptible to the negative effects of adverse experiences also most likely to benefit from positive ones. Evidence consistent with this view is summarized and directions for future research are outlined.

INTRODUCTION

It has long been appreciated that *Homo sapiens* are a developmentally plastic species whose growth and functioning are affected by environmental experiences and exposures encountered while growing up. Such sensitivity to the environment is presumed to enable the individual to prepare for the future and manage its resources effectively, thereby promoting reproductive fitness, that is, the passing on of genes to future generations.

Insufficiently appreciated by many developmental scholars, however, is that plasticity is not an unmitigated good, carrying just benefits (Sih, 2011). One potential cost derives from a "mismatch" between the early environment that a person develops in and adjusts to and the one the person encounters later in life. As a dramatic example, consider the cost incurred by Cambodian children (and their genetic relatives) who followed parental entreaties to study hard and do well in school, only to find when they grew up that they were the first to be murdered by the Khmer Rouge who distrusted the educated classes! As another example, consider a child who becomes (wisely?)

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hostile, aggressive, and excessively alert to threats to his or her well-being as a result of being abused at home, but then ends up in a school that is safe and fair-minded, expecting nevertheless that others will take advantage of him or her? Developmental plasticity may also carry costs associated with the greater complexity required of an organism capable of developing in a variety of ways depending on its early life experiences, given that a more complex system has more ways to go awry than a less complex one.

No matter what the benefits of plasticity, then, these observations suggest that natural selection should have "hedged its bets," with some individuals being more and others less—or hardly at all—developmentally plastic. This analysis implies that developmental plasticity should be regarded as a phenotype or individual-difference construct in its own right (Belsky & Pluess, 2013a).

As it turns out, appreciation of individual differences in sensitivity to the environment have been well recognized—at least in research on and thinking about contextual risk and developmental resilience. After all, some individuals have long been regarded as more likely to succumb to adversity (i.e., the "vulnerable") than others (the "resilient") as a function of their temperament, physiology, and/or genetic makeup (Luthar, Cicchetti, & Becker, 2000). Such "diathesis-stress" thinking says nothing about variation in response to environmental support or enrichment, however. Moreover, it remains unclear why natural selection would craft the development of some organisms to go awry in the face of contextual risk, as this predominant perspective on person-X-environment interaction seems to presume would be the case. Recently, an alternative to the prevailing diathesis-stress framework has been advanced.

DIFFERENTIAL SUSCEPTIBILITY

A perspective emphasizing differential susceptibility to environmental influence is built on evolutionary theory and acknowledges both the costs and benefits of plasticity (for extensive treatment, see Belsky & Pluess, 2009a; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2011). It differs from the traditional diathesis-stress framework by treating plasticity as a phenotype and highlighting the fact that evidence for what appears to be a disproportionate vulnerability of some to adversity may, in fact, reflect a more general susceptibility to both positive and negative environmental influences. This may have gone unnoticed owing to the absence of evolutionary thinking in so much developmental and psychiatric research and the fact that so much of this work has focused principally if not exclusively on contextual adversity and developmental dysfunction

instead of the full range of environmental exposures—from positive to negative—and their effects, again from positive to negative.

As a result, "vulnerable" individuals have been viewed as solely susceptible to the negative effects of adversity instead of being recognized as disproportionately susceptible to both positive and negative influences, that is, "for better and for worse" (Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2007). By the same token, individuals regarded as resilient in the face of adversity may also be unlikely to benefit from environmental support and enrichment. The latter clearly implies that resilience stemming from characteristics of the individual and so heralded in the developmental and psychiatric literature may not be the unmitigated good it is typically regarded as. Evidence consistent with the differential-susceptibility view that is extensively reviewed by Belsky and Pluess (2009a, 2013a) highlights temperamental, physiological, and genetic plasticity factors distinguishing those more and less susceptible to both positive and negative environmental effects.

NEGATIVE EMOTIONALITY

The first evidence for differential susceptibility indicated that it was infants, toddlers, and children with difficult or negatively emotional temperaments who developed especially poorly in the face of contextual adversity (e.g., poverty, and maternal depression), but proved most likely to benefit from supportive experiences (for review, see Belsky, 2005). Further evidence to this effect comes from work linking maternal empathy (Pitzer *et al*., 2011) and anger (Poehlmann *et al*., 2012) with externalizing problems; mutual responsiveness observed in mother-child dyads with effortful control (Kim & Kochanska, 2012); intrusive maternal behavior (Conway & Stifter, 2012) and poverty (Raver, Blair, & Willoughby, 2012) with executive functioning; and sensitive parenting with social, emotional, and cognitive-academic development (Roisman *et al*., 2012). One issue that merits additional empirical attention concerns the plasticity marker, negative emotionality, given that it has been operationalized in diverse ways across studies, including fear, inhibition, difficult temperament, and negative affect.

PHYSIOLOGICAL REACTIVITY

A claim central to Boyce and Ellis' (2005) biological-sensitivity-to-context (BSC) model of differential susceptibility is that physiological reactivity is a plasticity factor regulated by environmental experience, with heightened physiological reactivity in children being the mechanism responsible for greater environmentally sensitivity—in a for-better-and-for-worse manner. This has been shown in research on effects of actual marital conflict

(Obradovic, Bush, & Boyce, 2011) and simulated interparental aggression (Davies, Sturge-Apple, & Cicchetti, 2011) on externalizing problems; family adversity on school achievement (Obradovic, Bush, Stamperdahl, Adler, & Boyce, 2010); and teacher-child conflict on change in symptom severity (Essex, Armstrong, Burk, Goldsmith, & Boyce, 2011). What remains unclear is the stability of physiological reactivity across childhood and into adulthood and thus whether those who are highly reactive early in life but are exposed to nurturing experiences that promote well-being remain highly physiologically reactive—and thus highly susceptible (for better and for worse)—as they develop. After all, it could be that supportive rearing down regulates reactivity, thereby reducing environmental susceptibility.

GENETIC POLYMORPHISMS

Like other polymorphisms, the serotonin transporter gene, 5-HTTLPR, and the dopamine receptor gene, DRD4, have been regarded by psychiatric geneticists as "vulnerability genes" predisposing carriers of particular alleles to depression, and ADHD, respectively, in the face of adversity. Ever more evidence indicates, however, that they be regarded as "plasticity genes" (Belsky & Pluess, 2009b), making carriers of the putative risk alleles especially susceptible to environmental influences—for better and for worse.

Regarding 5-HTTLPR, individuals carrying one or more short alleles show greater "for-better-or-for-worse" plasticity when the rearing predictor and child outcome are, respectively, maternal responsiveness and moral internalization (Kochanska, Kim, Barry, & Philibert, 2011), child maltreatment and antisocial behavior (Cicchetti, Rogosch, & Thibodeau, 2012), and supportive parenting and positive affect (Hankin *et al*., 2011). Differential-susceptibility-related findings also emerge (among male African-American adolescents) when perceived racial discrimination is used to predict conduct problems (Brody *et al*., 2011); when life events are used to predict neuroticism (Pluess, Belsky, Way, & Taylor, 2010) and life satisfaction of young adults (Kuepper *et al*., 2012); and when retrospectively reported childhood adversity is used to explain aspects of impulsivity among college students (e.g., pervasive influence of feelings, and feelings trigger action) (Carver, Johnson, Joormann, Kim, & Nam, 2011). Nevertheless, other work makes clear that 5-HTTLPR operates in a manner consistent with diathesis-stress thinking (e.g., Bakermans-Kranenburg, Dobrova-Krol, & van IJzendoorn, 2012; Brody *et al*., 2012). This underscores the need to determine when one process or the other proves operative. A recent meta-analysis reveals that in the case of Caucasian children under 18 years of age, short-allele carriers are more susceptible than long-allele carriers

to both positive and negative developmental experiences (van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012).

Regarding DRD4, heightened if not exclusive susceptibility emerged for individuals who carry the 7-repeat allele in contexts where the environmental predictor and developmental outcome were, respectively, maternal positivity and pro-social behavior (Knafo, Israel, & Ebstein, 2011); early nonfamilial childcare and social competence (Belsky & Pluess, 2013b); contextual stress and support and adolescent negative arousal (Beach *et al*., 2012); childhood adversity and young-adult persistent alcohol dependence (Park, Sher, Todorov, & Heath, 2011); and newborn risk status (i.e., gestational age, birth weight for gestational age, length of stay in the hospital) and observed maternal sensitivity (Fortuna *et al*., 2011). Notable also is that a meta-analysis of G×E research involving dopamine-related genes revealed that children of 8 years and younger respond to positive and negative experiences in a manner consistent with differential susceptibility (Bakermans-Kranenburg & van IJzendoorn, 2011).

In addition to the two polymorphisms just highlighted, there is evidence that others may operate as plasticity factors, making some individuals more susceptible to environmental influences—for better and for worse. Especially important to appreciate is that most polymorphisms that have emerged as potential plasticity factors derived from psychiatric-genetic studies guided by diathesis-stress thinking. Researchers should expand their list of candidate genes beyond polymorphisms associated with disturbed functioning to incorporate a biologically driven inquiry of physiological processes affecting plasticity. A recent example of such an effort yielding evidence of differential susceptibility focused on the CHRNA4 genotype because of its role in acetylcholine production, a component strongly related to plasticity and learning (Grazioplene, DeYoung, Rogosch, & Cicchetti, 2013).

FUTURE RESEARCH DIRECTIONS

However much progress has been made in chronicling differential-susceptibility to environmental influences, many issues remain to be explored or illuminated.

Polygenetic Plasticity

Rather than regarding some individuals as plastic or malleable and others as not, it probably makes more sense to think of a gradient, with some being especially malleable, some reasonably malleable, some less so, and some not at all. Certainly that is suggested by work using multiple plasticity genes, as it reveals a dose–response relation between the number of plasticity genes

and the extent to which individuals are affected for better and for worse (e.g., Belsky & Beaver, 2011; Conner, Hellemann, Ritchie, & Noble, 2010). Future work of this kind should be guided by a "system-level genetic approach" involving the composting of putative plasticity genes on an *a priori* basis guided by knowledge of a particular biological process or pathway, such as the dopaminergic or serotonergic system, or neurological morphology. Even further insight may be gained through analysis of subsystems, including those involved with synthesis, degradation/transport, receptor and modulation (Chen *et al*., 2011).

Repeated Measures

A key assumption underlying the notion of differential susceptibility is that if those who prove vulnerable to adversity or who benefit the most from support found themselves growing up under opposite conditions that they would develop in a manner opposite to that which they do. For example, children, carrying the 7-repeat allele of the DRD4 gene who show high levels of externalizing problems when their parents are unskilled would show especially low levels of problems—and perhaps greater competence than others—if raised with more skilled parents. It is difficult to evaluate this proposal owing to ethical issues; one cannot create a bad rearing milieu for a child who has a good one.

Researchers may overcome this limitation by experimentally manipulating environments in a repeated-measures design. Consider as an example work by Roiser *et al*. (2006) on decision making during a gambling game in which individuals in one condition had good chances of winning and in the other poor chances. Results revealed that individuals with more 5-HTTLPR short alleles took the most risk (i.e., placed a bet) when chances of winning were good yet these same individuals engaged in the least gambling risk-taking when chances of winning were poor. Another repeated-measures study took advantage of a natural experiment, discovering that short allele carriers experienced the most stress, tension, and negative mood on exam days but the least on nonexam days (Verschoor & Markus, 2011).

Experimental Testing of Plasticity Factors

Most differential-susceptibility-related research has been observational in nature. This can challenge interpretation because environmental experiences may be selected rather than randomly assigned, creating the possibility that gene–environment correlation masquerades as gene–environment interaction. One solution to this problem involves conducting intervention experiments with random assignment of participants to experimental or

control conditions, work that is just beginning (Bakersman-Kranenburg, van IJzendoorn, Mesman, Alink, & Jeffer, 2008; Scott & O'Connor, 2012). Because of the previously mentioned ethical considerations, this approach has only been used to detect plasticity factors affecting response to interventions promoting positive functioning. Even though this design is limited to examining just the "for-better" side of plasticity, it still enables evaluation of whether those found to be most vulnerable to adversity in other research actually do benefit disproportionately from intervention efforts to promote positive functioning, whereas those regarded as resilient in the face of adversity turn out to be unaffected by the intervention.

Domain Specific or Domain General?

Reflection on this observation raises a related issue about whether plasticity is domain general or domain specific. That is, are more malleable individuals especially responsive to and influenced by a wide variety of environmental conditions and developmental exposures and other not particularly influenced by the same large set of experiences? Alternatively, are individuals mostly "mosaics" of plasticity, being highly sensitive to some contextual conditions but not others and/or with respect to some developmental outcomes but not others?

However surprising it might seem, there is some evidence for the domain-general view. Consider the results of two interventions that used strikingly dissimilar methods to promote different aspects of development. In one case the intervention sought to promote sensitive parenting in order to reduce toddler's externalizing behavior (Bakersman-Kranenburg, van IJzendoorn, Pijlman, Mesman, & Juffer, 2008) and cortisol-related stress reactivity (Bakersman-Kranenburg, van IJzendoorn, Mesman, *et al*., 2008), whereas in the other a computerized instructional program was employed to foster preschooler's phonemic awareness and, thereby, early literacy (Kegel, Bus, & van IJzendoorn, 2011). Despite the dramatic differences in the interventions and in the features of development being studied, it was children carrying 7-repeat DRD4 allele who benefited disproportionately, if not exclusively, from both! Before it can be concluded, however, that plasticity is more domain general than domain specific, far more work is required. We suspect that some individuals will be on the extremes of plasticity—highly responsive or virtually unaffected by almost all contextual conditions—but that most might fall somewhere between these extremes.

Environmental Cue Reliability

There is likely a variation in the reliability of environmental cues that children receive in developmental contexts. For instance, harsh environments

can be characterized as consistently and thus predictably high in conflict or episodically but not as predictably high in conflict. Frankenhuis and Panchanathan (2011) propose that highly plastic individuals growing up in these differing environments could vary in the timing of their "commitment" to a developmental strategy. A person developing in a highly predictable harsh environment may "commit" early, having high confidence in the state of the environment, whereas someone in an erratic environment may delay commitment to allow more time for a better estimation of the current—and thus future environment. Future work should seek to illuminate this issue.

Timing of Susceptibility

It is widely assumed that environmental exposures and developmental experiences exert their greatest influence early in life when biological systems are forming. Even if this may generally be true, it is still possible that some individuals could be more plastic later rather than earlier in life. All it would have taken for this to be so is for later plasticity to have paid off more than earlier plasticity some time in our ancestral past and for natural selection to thus have preserved genes responsible for such deferred developmental plasticity. This raises the possibility that some individuals may be highly responsive to environmental influences—in a for-better-and-for-worse manner—across the life course, others never, some especially early and some especially later.

CONCLUSION

From an evolutionary perspective, we have outlined the need to move beyond considering only the benefits of plasticity—as is typically done when comparing our species to that of others—to an evolutionary-inspired theoretical framework that acknowledges both the costs and benefits of developmental plasticity. Moreover, rather than presuming that some individuals are simply more susceptible to the negative developmental effects of contextual adversity, as the prevailing person-X-environment, diathesis-stress framework does, differential-susceptibility thinking presumes that there are (i) individual differences in developmental plasticity; (ii) such that those most susceptible to adversity are also especially likely to benefit from environmental support and enrichment; and (iii) thus that developmental plasticity should be treated as an individual-difference construct. Evidence highlighting diverse plasticity factors proves consistent with this claim, though many questions remain to be addressed. Perhaps a major contribution of the differential-susceptibility perspective is to underscore the theoretical and empirical risks of focusing disproportionately on

contextual risk, dysfunctional development and vulnerability and benefits of viewing developmental from an evolutionary perspective.

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Jay Belsky is the Robert M. and Natalie Reid Dorn Professor of Human Development at the University of California, Davis. He is an internationally recognized expert in the field of child development and family studies. His areas of special expertise include the effects of day care, parent–child relations during the infancy and early childhood years, the transition to parenthood, the etiology of child maltreatment, and the evolutionary basis of parent and child functioning. He obtained his PhD in 1978 in Human Development and Family Studies from Cornell University. From 1999 to 2010, he served as founding Director of the Institute for the Study of Children, Families and Social Issues and Professor of Psychology at Birkbeck University of London. Before that, he served on the faculty at Penn State University for 21 years, rising to the rank of Distinguished Professor of Human Development. In 1983, he won the Boyd McCandless Award for Distinguished Early Contribution from the Developmental Psychology Division of the American Psychological Association. In 2002, the Institute of Scientific Information, Philadelphia, PA granted him the Highly-Cited-Researcher designation. In 2007, he was awarded the American Psychological Association Urie Bronfenbrenner Award for Lifetime Contribution to Developmental Psychology in the Service of Science and Society. In 2010, he was made a member of the Academy of Europe.

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