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An Evolutionary Perspective on Family Studies: Differential Susceptibility to Environmental Influences

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An evolutionary perspective of human development provides the basis for the differential-susceptibility hypothesis which stipulates that individuals should differ in their susceptibility to environmental influences, with some being more affected than others by both positive and negative developmental experiences and environmental exposures. This paper reviews evidence consistent with this claim while revealing that temperamental and genetic characteristics play a role in distinguishing more and less susceptible individuals. The differential-susceptibility framework under consideration is contrasted to the traditional diathesis-stress view that "vulnerability" traits predispose some to being disproportionately affected by (only) adverse experiences. We raise several issues stimulated by the literature that need to be clarified in further research. Lastly, we suggest that therapy may differ in its effects depending on an individual's susceptibility.

Keywords: Differential susceptibility; Gene–environment interaction; Diathesis stress

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INTRODUCTION

Individual differences in sensitivity to the environment—or susceptibility to environmental influences—have long been recognized, especially in research considering contextual risk and resilience. Decades of theory and research support the claim that some individuals are more adversely affected than others by negative developmental experiences and environmental exposures—like marital discord and child maltreatment—whereas others prove relatively immune to or resilient in the face of such adversity. In the case of children, then, some prove more developmentally "plastic" or malleable, tending to be more affected by their experiences and exposures than other, less plastic or malleable age mates. Recent evolutionary inspired thinking calls attention, however, to the fact that those very characteristics that appear to make children—and adults—more vulnerable to adversity also make them more likely to benefit from support and enrichment. In other words, some are more affected "for better and for worse" than are others. Clinicians no doubt encounter variation in patients' response to therapy, with some clients benefiting substantially and others not at all. In this article, we consider theory and research on person-X-environment and gene-X-environment interaction studies consistent with the claims just advanced.

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DIFFERENTIAL SUSCEPTIBILITY

Applying an evolutionary perspective on human development, Belsky proposed that individuals should vary in their susceptibility to environmental influences and especially those of the rearing environment (Belsky, 1997, 2005; Belsky & Pluess, 2009, 2013a). This proposition was based on appreciation that the future is—and always has been—inherently uncertain. Thus, to maximize the likelihood of genes being passed from one generation to the next (i.e., reproductive fitness)—the ultimate goal of all living things—natural selection should have crafted offspring to vary in their susceptibility. If, for instance, the environment one grew up in did not match the environment one encountered later on, it would prove more costly for the child whose development was heavily influenced by his or her early environment. On the other hand, this environmental mismatch may have a negligible effect on a child who was less, if at all, shaped by his or her early experiences. To combat this ever-present risk of a potentially changing environment, Belsky theorized that nature "hedged its bets" by making humans variable in their susceptibility to parental influences and presumably other influences as well.

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! This example illustrates the point that even the best-intended parental goals and efforts can prove misguided—because the future is uncertain. Thus, effects that families have on children, whether consciously or unconsciously pursued, can engender outcomes never intended. By bearing and rearing some offspring who are and are not, or who are more and less responsive to parental (and other environmental) influence(s), a family basically secures an insurance policy against environmental effects paying off or proving costly.

The just highlighted example of environmental mismatch in the case of Cambodian children exemplifies one the potential costs of plasticity or malleability. Additional costs of increased plasticity derive from dependence on a more complex developmental system designed to receive and translate information from the environmental conditions to regulate development (Belsky & Pluess, 2013a). As delineated by DeWitt, Sih, and Wilson (1998), reliance on a more complex system incurs costs across several areas, some of which include energetic costs of the sensory and regulatory mechanisms of plasticity, the production cost of environmentally inducible structures and processes, and the cost of acquiring information about the environment. On the other hand, less plastic individuals may rely on more simple systems and thus can expend energy to enhance other areas of development. Therefore, plasticity is not an unmitigated good but rather a trade-off that confers both costs and benefits to the organism. It is for this reason, too, that we should expect variation across individuals in their plasticity or susceptibility to environmental influences.

Essentially, what these arguments lead to is the theoretical proposition that some people, using evolutionary-biological terminology, are "fixed strategists" when it comes to making their way in the world, whereas others are "plastic strategists". Or, thinking dimensionally rather than categorically, some individuals are more plastic or malleable and thus shaped by their experiences more than others who are less affected by their developmental experiences and environmental exposures. Please appreciate that use of the term "strategist" here does not imply that a person consciously follows a particular strategy, only that he or she functions "as if" he or she did so. Of note, too, is that having a fixed strategy does not imply that all fixed strategists develop in the same way, only that these individuals are not particularly susceptible to environmental influence. Some of these individuals will function very well, average, or very poorly, depending on their personal characteristics.

One final comment is in order before considering empirical evidence consistent with the claims just made. There is no disputing that relative to many other species, humans are a highly plastic one. We change as we develop and in response to our experiences and exposures. Variation in nutrition affects our growth and variation in education affects our knowledge and skills. But such general characteristics do not obviate the possibility—asserted herein—that some prove much more developmentally plastic or malleable than others.

EVIDENCE FOR DIFFERENTIAL SUSCEPTIBILITY

The findings we now proceed to review provide evidence that those individuals who are more susceptible to the environment (i.e., plastic strategists) function in a for-better-or-for-worse manner. This means that more plastic individuals benefit disproportionately from supportive environments while also being more adversely affected by negative conditions (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009, 2013a). The last point contrasts with the prevailing diathesis-stress framework which has guided decades of research in the behavioral and health sciences.

Diathesis-stress thinking stipulates that some individuals are "vulnerable" or "at risk" due to individual differences in behavior or biology (e.g., difficult temperament, risk allele) and thus are disproportionately likely to be adversely affected by negative experiences (Zuckerman, 1999). For example, infants who have a negative temperament may be especially likely to develop insecure attachments when exposed to insensitive parenting or individuals carrying a putative "risk" allele may be most likely to become depressed upon encountering stressful life circumstances. However, those who do not have the "vulnerability" characteristic will be less or not at all affected by adversity; thus, they will prove "resilient".

Where the differential-susceptibility perspective contrasts to a diathesis-stress one is in asserting that those who are most vulnerable to adversity are, simultaneously, most likely to benefit from supportive environmental conditions—because they are more generally developmentally plastic or malleable. Basically because so many social and behavioral scientists, including family scholars and students of child development, focus virtually exclusively upon clinical conditions and environmental risk factors, they stand to misconstrue a heightened and general susceptibility to environmental influences of all kinds to a particular sensitivity—that is, vulnerability—to adversity alone. One of the interesting issues this argument raises, of course, is that those who benefit the most from therapy may be so-called plastic strategists who are most affected, for better and for worse, by environmental conditions. Indeed, this may be the very reason that some of them are in therapy to begin with (i.e., adverse developmental experiences and exposures).

Behavioral Markers of Differential Susceptibility

Two behavioral patterns have emerged as being indicators of heightened susceptibility (i.e., increased developmental plasticity): temperament/negative emotionality and a highly sensitive personality. Temperament, mostly studied in infants and young children, has been a long-standing focus of developmental inquiry (Rothbart & Bates, 2006). On the other hand, highly sensitive personality has been studied mostly in adults and is a relatively new area of interest. Both temperament and a highly sensitive personality are thought to have biological underpinnings, including elevated physiological reactivity (Rothbart, Ahadi, & Evans, 2000; Aron & Aron, 1997, respectively). Although having a highly sensitive personality is likely to be closely related to difficult temperament in childhood, it encompasses broader characteristics thought to relate to individual differences in

the underlying nervous system rather than negative emotionality alone. The following sections highlight evidence that both of these behavioral patterns may indicate a heightened susceptibility to the environment across childhood and adulthood.

Temperament and negative emotionality

Some of the earliest evidence chronicling differential susceptibility to environmental influences emerged from research on temperament-X-parenting interaction (Belsky, 1997). In Belsky's (2005) review, he made the empirical observation that the effect of rearing experience on a variety of behavioral outcomes was consistently greater for a subgroup of children characterized by a difficult temperament and thereby having high levels of negative emotionality. Thus, children who were irritable, fearful, and/or inhibited were more affected in their functioning in response to quality of the environment—and in for-better-and-for-worse manner. Therefore, what was previously viewed as solely a marker of vulnerability under contextual risks (e.g., poverty, maternal depression), was actually a marker of a more general susceptibility to the environment. While children with negative temperaments did indeed suffer the most when faced with adversity, they also benefited the most from supportive or benign circumstances.

In their 2009 and 2013 reviews, Belsky and Pluess summarized a range of evidence for negative emotionality as a plasticity factor that spanned ages and contexts (Belsky & Pluess, 2009, 2013a). One illustrative example came from two reports using data collected as part of the large-scale and longitudinal NICHD Study of Early Child Care and Youth Development (NICHD Early Child Care Research Network [ECCRN], 2005). These studies provided evidence for differential susceptibility upon examining the interaction between maternally reported infant temperament at 1 and 6 months and parenting and child care.

In the first study, Bradley and Corwyn (2008) examined the effects of observed and reported maternal sensitivity, harshness, and productive activity on teacher-reported behavior problems. Results revealed that children who had more difficult temperaments during infancy had the most behavior problems in first grade if they experienced low-quality parenting across the infant, toddler, and preschool years, but the least problems if they experienced high-quality parenting compared to all other children (i.e., "for better and for worse"). On the other hand, the effect of parenting quality was weaker in the case for children with intermediate levels of difficult temperament and weaker still in the case of children scoring very low on difficult temperament (i.e., easy temperament). That is, the children with difficult temperaments were more responsive to positive or negative parenting.

In the second inquiry, Pluess and Belsky (2009, 2010) extended this work documenting for-better-and-for-worse environmental effects on children with difficult temperaments. They found that it was not just the case that infants with difficult temperaments were more affected by, respectively, sensitive and insensitive parenting, but that the same was true with regard to the quality of nonmaternal child care. That is, it was infants who had the most difficult temperaments whose early and later social functioning was affected by whether they experienced child care that was attentive, responsive, warm, and stimulating across their first 54 months of life—or not. Similarly, research by Essex, Armstrong, Burk, Goldsmith, and Boyce (2011) found that children who were more negatively emotional experienced the greatest change in mental-health symptomology in response to teacher–child conflict across the primary-school years (Essex et al., 2011).

Additional research also provides support for the claim that negative emotionality moderates environmental effects in a differential-susceptibility-like manner, especially with regard to the effects of parenting. This includes the following parenting predictor and child-outcome links: maternal empathy and externalizing problems (Pitzer, Jennen-Steinmetz, Esser, Schmidt, & Laucht, 2011); mutual responsiveness in the

mother–child dyad and effortful control (Kim & Kochanska, 2012); intrusive maternal behavior and executive functioning (Conway & Stifter, 2012); sensitive parenting and social, emotional, and cognitive-academic development (Roisman et al., 2012).

Intriguingly, there is even evidence that highly negatively emotional adults are disproportionately susceptible to environmental influences, at least with respect to forces that shape their parenting. Consider in this regard an investigation by Jessee et al. (2010) which found that maternal negative affect moderated the effect of marital quality on changes in parental sensitivity: Mothers high on negative affect who experienced little marital conflict showed the greatest increases in parenting sensitivity, whereas emotionally similar women who experienced high marital conflict decreased the most in parenting sensitivity.

Highly sensitive personality

Although mainly investigated in childhood, there is emerging evidence—from studies like that just cited by Jessee et al. (2010)—that differential susceptibility is also evident in adulthood, operating via an individual's sensory-processing sensitivity. The personality dimension of sensory-processing sensitivity is measureable by means of the Highly Sensitive Person scale (HSP; see Aron & Aron, 1997), and interacts with various environmental factors to predict adult shyness and negative affectivity (Aron, Aron, & Davies, 2005). According to Aron and Aron (1997), about 20% of the population is characterized by a highly sensitive personality which encompasses a sensitive nervous system, awareness of subtleties in surroundings, and a tendency to be more easily overwhelmed when in a highly stimulating environment. Work by Aron et al. (2005) reveals that a stressful (and retrospectively reported) childrearing history predicts high levels of (self-reported) shyness and negative affectivity and that the absence of such a stressful childhood forecasts low levels of these same outcomes within a sample of undergraduate students, but only for those scoring high on sensory-processing sensitivity.

Notably, a recent fMRI study found that individuals who scored high on sensory-processing sensitivity showed greater brain activation in regions associated with interpersonal awareness and empathy when exposed to their partner's faces and stranger happy and sad faces compared to those who scored lower on sensory-processing sensitivity (Acevedo et al., 2014). Interestingly, other research on adults indicates that certain personality characteristics (e.g., openness, agreeableness) may function as plasticity-related factors making individuals more and less susceptible to environmental influences and operating in the for-better-or-for-worse manner characteristic of differential susceptibility (e.g., Slagt, Dubas, Denissen, Dekovic, & Aken, 2014). Given that differential susceptibility appears to extend from infancy into adulthood, it may have implications for clinicians who treat various ages in therapy.

Genetic Markers of Differential Susceptibility

Most of the research cited thus far, even if not all, which examined the moderating role of temperament, focused on children; however, evidence of differential susceptibility in the form of gene-X-environment (GXE) interaction derives mainly from work on adults. Most of the GXE research has been guided by the diathesis-stress framework, with a focus on psychopathology and with the goal of illuminating when and how particular "risk alleles" result in disturbed or dysregulated functioning. This is not surprising, given that most of this research was undertaken by psychiatric geneticists. In the following sections, we call attention to GXE findings involving two of the most studied candidate genes—the serotonin-transporter-linked polymorphic region (*5-HTTLPR*) and the dopamine receptor

D4 gene (*DRD4*)—that reveal findings more consistent with differential susceptibility than diathesis stress.

Serotonin-transporter-linked polymorphic region

The serotonin transporter (5-HTT) has a central role in regulating the effects of serotonin within the brain because it modulates the neural transmission of serotonin through the reuptake of serotonin from the synaptic cleft. In fact, 5-HTT is the key protein pharmacologically targeted through antidepressants including serotonin-reuptake inhibitors. The *5-HTTLPR* is a genetic polymorphism that exists within the promoter (i.e., regulatory) region of the 5-HTT gene, SLC6A4. Different lengths of *5-HTTLPR*, so-called alleles, are thought to affect promoter activity and, thus, how efficiently 5-HTT is transcribed (Canli & Lesch, 2007). These *5-HTTLPR* alleles are often classified as either being short or long, although more variants than these have been identified (Nakamura, Ueno, Sano, & Tanabe, 2000). The short allele has been associated with reduced expression of 5-HTT and consequently lower serotonin-reuptake activity as compared to the long allele (Canli & Lesch, 2007).

Therefore, most research on *5-HTTLPR* distinguishes two groups of people—those carrying at least one short allele (s/s, s/l) and those homozygous for the long allele (l/l). These different combinations result because each person inherits two alleles of the 5-HTT gene, one from his or her mother and the other from his or her father. Using this short versus long approach, *5-HTTLPR* has been extensively investigated both directly and GXE interactions with regard to clinical conditions such as depression (Caspi et al., 2003) and anxiety (Conway, Slavich, & Hammen, 2014).

It has been more than a decade since Caspi et al. (2003) conducted their ground breaking GXE study showing that *5-HTTLPR* moderates the relation of stressful life events during early adulthood on later depression and suicidal ideation. Their findings indicated that individuals with two short alleles (s/s) proved most adversely affected by stressful life events and that parallel effects on those with two long alleles (l/l) were weaker or entirely absent. Although guided by a diathesis-stress framework, this study also showed, even if it went unmentioned, that s/s homozygotes scored best on the outcomes just mentioned when stressful life events were absent.

Perhaps of particular interest are GXE findings pertaining to family processes. In one such study, Sturge-Apple, Cicchetti, Davies, and Suor (2012) observed that *5-HTTLPR* moderated the effect of marital quality on parenting in a differential-susceptibility-related manner. More specifically, mothers carrying at least one copy of the s allele evinced greater sensitivity and less harsh/punitive parenting when there was little marital conflict, but just the opposite when marital conflict was high. Notably, there was no association between marital quality and parenting in the case of mothers homozygous for the long allele.

In another inquiry, Schoebi, Way, Karney, and Bradbury (2012) used a marital interaction task within a laboratory setting to investigate whether *5-HTTLPR* moderated a spouse's sensitivity to their partner's self-reported positive and negative affect on their own emotional experience. Both spouses self-reported on their affect before and after a marital interaction task. Results revealed that spouses carrying at least one copy of the short allele were more responsive, via changes in their own reported affect, to their partner's preinteraction positive and negative affect compared to spouses with two long alleles.

Differential-susceptibility-related findings also emerged when life events were used to predict neuroticism (Pluess, Belsky, Way, & Taylor, 2010) and the life satisfaction of young adults (Kuepper et al., 2012). There is also evidence showing that (retrospectively reported) childhood adversity was used to explain aspects of impulsivity among college

students (e.g., pervasive influence of feelings, feelings trigger action; Carver, Johnson, Joormann, Kim, & Nam, 2011).

Beyond this adult-related research, there is a wealth of evidence that chronicles increased susceptibility of children carrying one or more *5-HTTLPR* short alleles—and in a for-better-and-for-worse, differential-susceptibility-related manner. For example, one study found that *5-HTTLPR* moderated the relation between maternal responsiveness and moral internalization (Kochanska, Kim, Barry, & Philibert, 2011), another that between child maltreatment and antisocial behavior (Cicchetti, Rogosch, & Thibodeau, 2012). Other work revealed the same genetic moderation of the effect of maternal prenatal depression on child emotional dysregulation (Babineau et al., 2015), as well as of supportive parenting on child positive affect (Hankin et al., 2011). Moreover, a recent meta-analysis of GXE findings pertaining to children under 18 years of age found that short allele carriers are more susceptible to the effects of positive and negative developmental experiences and environmental exposures, at least in Caucasians (van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012).

Although the evidence cited regarding the moderating effect of *5-HTTLPR* proves most interesting and consistent with differential-susceptibility thinking, one particular limitation to such observational research is the possibility that results are confounded by a gene–environment correlation (rGE). That is, the observational work just summarized cannot discount the possibility that individuals with certain characteristics select or evoke environmental experiences and exposures; and these rGE effects could "masquerade" as GXE interaction. Experimental intervention research can overcome this limitation because environmental exposures are randomly assigned to individuals—and thus not selected or evoked as a result of individual characteristics. Fortunately, such work is available to consider.

One relevant gene-X-intervention (GXI) by Brett et al. (2015) examined differential-susceptibility in Romanian orphans randomly assigned to either high-quality foster care or care as normal in an institution. Results showed that *5-HTTLPR* significantly moderated intervention effects on later externalizing behavior. Children homozygous for the short allele (s/s) had the lowest levels of externalizing behavior in the foster care group, while children with the s/s genotype had the highest rates of externalizing behavior when they remained in an institution. And there were no intervention group differences in predicting externalizing behavior for children who carried the long allele (s/l or l/l). Thus, children with s/s genotypes had increased susceptibility to intervention effects.

Likewise, an investigation by Brody, Beach, Philibert, Chen, and Murry (2009) examined the effects of preventive parenting intervention on youth's risky behavior. They found that youths with at least one short allele (s/s or s/l) benefited more from the preventive parenting program than did youths with the l/l genotype. Not only does this intervention work provide compelling evidence for differential susceptibility but it is also especially relevant to clinicians considering that therapy may be similar to an intervention.

Dopamine receptor D4

There is also evidence that genes related to the dopaminergic system, which is engaged in attentional, motivational, and reward mechanisms, also moderate environmental effects in a differential-susceptibility-related manner. Most notable is the variable number tandem repeat (VNTR) of the dopamine receptor D4 (*DRD4*) gene. The *DRD4* gene codes for the dopamine receptor subtype D4 which is activated by the neurotransmitter dopamine. The variants, or alleles, of the *DRD4* differ by the number of 48-base pair tandem repeats in coding region exon III, ranging from 2 to 11. Once again, because each person inherits two alleles, one from each parent, there are different combinations of variants. Specially, presence of the seven-repeat variant (i.e., having at least one allele with seven

repeats) has been identified as a vulnerability factor due to its links to ADHD (Faraone, Doyle, Mick, & Biederman, 2001), high novelty seeking behavior (Kluger, Siegfried, & Ebstein, 2002), and low dopamine reception efficiency (Robbins & Everitt, 1999), among other correlates.

As it turns out, a number of studies indicate that children carrying this putative risk allele are not only more adversely affected by poorer quality parenting than other children, but also benefit more than others from good-quality rearing. Of special importance is that some of this work reflects efforts to determine whether intervention efficacy varies by genetic make-up. But, before considering such research, the next investigation to be considered may be regarded as particularly important for another reason—because a "good" environment is not just operationalized as the absence of adversity, as in much of the GXE research already cited, but in terms of high-quality parenting. In a longitudinal study of infants, maternal insensitivity observed when children were 10 months of age predicted externalizing problems reported by mother more than 2 years later, but only for children carrying the seven-repeat *DRD4* allele (Bakermans-Kranenburg & van Ijzendoorn, 2006). Moreover, although children with the seven-repeat *DRD4* allele displayed, consistent with a diathesis-stress model, the most externalizing behavior of all children when mothers were judged insensitive, they also manifested the least externalizing behavior when mothers were highly sensitive.

Experimental intervention research designed to enhance parenting also documents a moderating effect of the seven-repeat allele on parenting. When Bakermans-Kranenburg, IJzendoorn, Pijlman, Mesman, and Juffer (2008) looked at change over time in parenting —from before to well after a video-feedback parenting intervention was provided on a random basis to mothers of 1- to 3-year-olds who scored high on externalizing problems—they not only found that the intervention succeeded in promoting more sensitive parenting and positive discipline but also that experimental effects extended to improvements in child behavior, though only for those children carrying the *DRD4* 7-repeat allele.

The same team of Dutch investigators also reported that the *DRD4* 7-repeat allele moderated the effect of maternal unresolved loss or trauma, as measured by means of the Adult Attachment Interview, on early infant development. More specifically, unresolved loss predicted infant attachment disorganization, an early developmental marker of psychological disturbance later in life (Carlson, 1998), but only in the case of infants carrying the seven-repeat allele (van IJzendoorn & Bakermans-Kranenburg, 2006). Indeed, these infants manifest both the most and least disorganized attachment behavior when stressed, depending on whether their mothers had or had not experienced unresolved loss or trauma in their own lives.

In their review, Belsky and Pluess (2013a, 2013b) highlighted observational differential-susceptibility related evidence showing heightened or exclusive susceptibility of individuals carrying the seven-repeat allele. Seven-repeat carriers were more susceptible to maternal positivity as indicated by greater variation in later prosocial behavior (Knafo, Israel, & Ebstein, 2011). Similarly, children with the seven-repeat allele showed greater variation in social competence in response to early nonfamilial childcare (Belsky & Pluess, 2013b). Furthermore, seven-repeat carriers proved more susceptible to childhood adversity when predicting young-adult persistent alcohol dependence (Park, Sher, Todorov, & Heath, 2011).

It is important to note that at least one investigatory team finds that it is those without the seven-repeat allele who prove most responsive to childhood adversities in a diathesis-stress manner (Das, Cherbuin, Tan, Anstey, & Easteal, 2011). Nevertheless, two meta-analyses have supported dopamine-related genes as markers of differential susceptibility. The first revealed that children 8 years and younger responded to positive and negative developmental experiences and environmental exposures in a manner consistent with

differential susceptibility and more so than those not carrying this allele (Bakermans-Kranenburg & van IJzendoorn, 2011). The second meta-analysis, this one of intervention rather than of observational, field studies, found that dopamine-related genotypes were among the differential-susceptibility-related moderators of intervention effects (Bakermans-Kranenburg & Van IJzendoorn, 2015).

CONCLUSIONS AND IMPLICATIONS FOR FAMILY AND COUPLE THERAPY

What the research reviewed herein indicates is that it is not just that some individuals are more vulnerable to adversity than others, as traditionally appreciated by diathesis-stress-informed investigators studying psychopathology, but that in some, perhaps many, cases these same putatively vulnerable individuals are actually disproportionately susceptible to the benefits of positive environmental conditions, even when this just means the absence of adversity. This raises the obvious question, of course, whether the same applies to the therapeutic experience. More pointedly, are there clients who simply are unresponsive to therapy? And, if so, are these individuals' psychological difficulties less a function of experience, which is often presumed by etiological models guiding treatment, and more a product of inherited disposition? Conversely, are there some individuals who have not only been greatly—and adversely—affected by their experience, but who, as a result of their general susceptibility to environmental influences, benefit the most from therapy? Certainly if one generalizes broadly from the parenting intervention studies in the preceding paragraphs dealing with the prevention of child behavior problems, this would seem to be the case.

But what are the implications of this view for family therapy? We raise this question because in couple or family therapy there is more than a single individual—and these people may be similar or different in terms of their responsiveness to environmental influences. Does this matter for the efficacy of such therapy? Might this explain why, in some cases, not all but some family members benefit and others do not? These, of course, are questions we do not have answers for—but which future research could and should address.

Trauma

Of particular concern to clinicians may be whether or not differential susceptibility may be applied to the experience of trauma. Are some individuals who experience trauma more prone to develop symptoms such as depression or posttraumatic stress disorder while others remain relatively immune? Or is it the case that exposure to a severe stressor such as trauma causes everyone to be equally affected, while revealing none to be resilient? Differential susceptibility has been studied mostly with regards to environmental stressors that may be considered as more moderate such as inter-parental conflict or insensitive parenting. There are, however, a few studies to suggest that differential susceptibility applies to more severe stressors. For example, in the case of child maltreatment, an investigation by Cicchetti and Rogosch (2012) revealed that children who had the most plasticity alleles (i.e., the greatest genetic susceptibility) had the poorest levels of functioning (e.g., lower social competence, higher depression) when maltreated but the highest levels of functioning when not maltreated. Conversely, children who were less genetically susceptible showed no effect of maltreatment on their functioning.

Further work by Disner et al. (2013) found that among U.S. army soldiers, *5-HTTLPR* moderated the effect of war-zone stress and biased attention toward negative stimuli, a correlate of several mental illnesses. Soldiers homozygous for the short allele of *5-HTTLPR* showed increased negative attention bias when exposed to more war-zone stress

while soldiers with other genotypes showed no such relation. Clearly, more research needs to be done to illuminate whether there are differential-susceptibility effects among different types of trauma at various ages.

Subgroups of Plasticity?

One of the most striking features of the findings reviewed herein is how diverse the evidence base is, which suggests that individuals differ in their plasticity. The environmental factors highlighted as predictors of differential-susceptibility-related effects include parenting, child care quality, and life events to name but some investigated in the research cited above. And the domains of functioning which prove sensitive to differential-susceptibility-related effects include disorganized infant attachment; externalizing problems in the toddler, preschool, and childhood years; depression and parenting throughout adulthood, again to call attention to just a few. And, finally, the moderators (i.e., plasticity factors) of these diverse environmental effects on these varied outcomes include temperamental attributes of children and genotypic ones.

Reflection on these observations leads one to wonder if the very same more versus less susceptible individuals are being identified by different means, with some investigators focused on temperament, some on genetics, and some on personality. Seemingly relevant to this issue is that negative emotionality early in life is related to the *5-HTTLPR* gene (Auerbach, Faroy, Ebstein, Kahana, & Levine, 2001) and *DRD4* (Holmboe, Nemoda, Fearon, Sasvari-Szekely, & Johnson, 2010; Ivorra et al., 2010). Although the one study that sought to determine whether a moderating effect of negative emotionality could be accounted for by that of *DRD4* did not find evidence of such (Belsky & Pluess, 2013b), this one inquiry cannot fully resolve the issue at hand. This, then, is another unknown in the differential-susceptibility equation.

Domain-specific or Domain-general?

Another issue that merits further investigation is whether individuals who prove more or less susceptible to one experience or exposure are similarly responsive to others. In other words, is plasticity a more domain-general trait or a domain-specific one? Might it be the case that some individuals are affected by only specific aspects of the environment (e.g., parents but not peers) and with regard to select domains of functioning (e.g., social but academic competence)? If this were the case, it would have implications for therapists considering whether certain individuals may benefit from one type of treatment over another or if they would be unaffected by all types of treatment. It may be the case that some individuals will be on the extremes of plasticity—highly responsive or completely unresponsive to almost all contextual conditions (and treatment approaches?)—whereas most others might fall somewhere on the continuum between these two extremes.

CONCLUSION

In conclusion, we have reviewed theoretical reasoning and evidence for the claim that individuals differ in their susceptibility to environmental influences, be those of parenting, child care, life events, or some other potential factor long thought to shape human development and psychological functioning. More specifically, the work cited indicates that in many cases those most vulnerable to adversity will benefit the most from supportive contextual conditions, as these individuals are generally more environmentally responsive and developmentally plastic. Given that such differences in susceptibility are rooted in biology and shaped by natural selection, there is reason to believe that a similar differential response should characterize the effects of therapy, too. Only future research

will determine the validity of this claim. In summary, the differential-susceptibility framework adds a novel and evolutionarily grounded perspective by which to further investigate and interpret these complex dynamics between genetics and the environment.

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