Parenting Effects in the Context of Child Genetic Differences

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It is widely acknowledged that sensitive parenting contributes to the healthy psychosocial development of children. This understanding is not simply based on popular wisdom. Meta-analyses of controlled experimental studies informed by attachment theory demonstrate that sensitive parenting causally influences the development of a secure attachment in the infant and young child (Bakermans-Kranenburg, van IJzendoorn, & Juffer, 2003; De Wolff & van IJzendoorn, 1997).

But does this mean that each and every child is equally likely to benefit from sensitive parenting? To address this critical question, we consider a selection of findings emerging from studies of gene-environment interaction (e.g., Bakermans-Kranenburg & van IJzendoorn, 2006; Knafo, Israel, & Ebstein, 2011). In a concluding section, we discuss different frameworks of gene-environment interaction before suggesting potential implications of research chronicling differential susceptibility to the influence of parenting.

Diathesis-stress versus differential susceptibility

Interactions between organism characteristics of children (e.g., temperament, genotype) and environmental influences, including parenting quality, are usually interpreted in terms of a diathesis-stress model of psychopathology (Monroe & Simons, 1991; Zuckerman, 1999) or dual risk model of development (Sameroff, 1983). Both frameworks are based on the view that negative consequences of adverse experiences disproportionately, if not exclusively, characterize children who are "vulnerable", with children not succumbing to the same adversity regarded as "resilient" (Zubin & Spring, 1977). Diathesis-stress/dual-risk models imply also that under conditions of environmental support, including sensitive parenting, "vulnerable" and "resilient" individuals will not differ in their functioning. In other words, it is only under conditions of adversity that their heightened sensitivity to environmental influence is operative.

But, are children that are more negatively affected by low parenting quality correctly characterized as being exclusively "vulnerable" to adversity with no differences to be expected in response to positive experiences as the diathesis-stress/dual-risk models imply? The pathology-focused view of the diathesis-stress/dual-risk models has been challenged recently by the differential susceptibility hypothesis (Belsky, 1997a, 1997b, 2005; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009) which stipulates that individuals may not so much vary in the degree to which they are vulnerable to the negative effects of adverse experience, but do so more generally in their *developmental plasticity*: More "plastic" or malleable individuals will be more susceptible to both the adverse developmental sequelae associated with negative environments *and* the positive developmental consequences of supportive environments; less susceptible individuals—so-called "fixed" (or "relatively fixed") ones—will be far less or not at all affected by the same environmental conditions (see Figure 1). Boyce and Ellis (2005) advanced a perspective similar to differential susceptibility with a more specific focus on the role of the stress-response system in moderating environmental effects (i.e., "biological sensitivity to context"; see also Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011).

An increasing number of gene-environment interaction studies provide empirical evidence for differential susceptibility as a function of genetic variation. In what follows, we summarize results from a variety of investigations documenting moderating effects of a polymorphism in the dopamine receptor D4 gene (DRD4) in respect to parenting. More extensive reviews of differential susceptibility findings involving other genes than the DRD4 and other environmental influences than parenting can be found elsewhere (Belsky & Pluess, 2009; Ellis, et al., 2011; Pluess & Belsky, 2010).

Differential susceptibility to parenting as a function of the Dopamine Receptor D4 Polymorphism

Variations of a single gene—so called polymorphisms—are frequent across the human population and are generally characterized by the existence of two or more different versions or variants of the same gene (i.e., alleles). The DRD4 gene codes for a protein that is transmitting dopamine signals from one neuron to another and plays an important role in the dopaminergic system which is engaged in attentional, motivational, and reward mechanisms. Variants of the DRD4 differ by the number of 48-base pair tandem repeats in exon III, ranging from 2-11. The 7-repeat variant 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), amongst 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

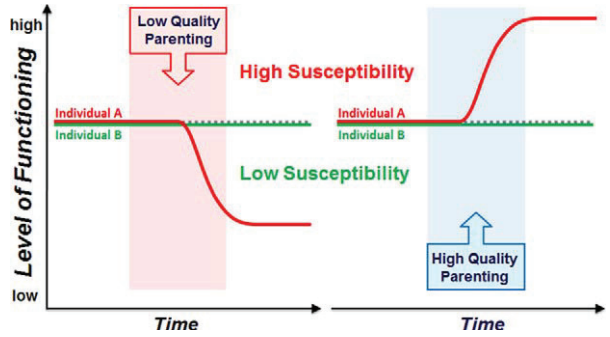

Figure 1. The left half of the figure illustrates the negative side of differential susceptibility (i.e. diathesis-stress): in response to low quality parenting, the level of functioning decreases in individual A, reflecting vulnerability, whereas it remains unchanged in individual B, reflecting resilience. The right half of the figure illustrates the positive side of differential susceptibility (i.e. vantage sensitivity): in response to high-quality parenting, the level of functioning increases in individual A, reflecting vantage sensitivity, whereas it remains unchanged in individual B, reflecting vantage resistance. Consequently, individual A reflects higher susceptibility to both negative and positive parenting experiences whereas individual B appears less responsive regardless of parenting quality.

quality parenting than other children, but also benefit more than others from high-quality rearing—consistent with a differential susceptibility framework. For example, in a longitudinal study of infants, maternal insensitivity observed when children were 10 months of age predicted greater externalizing problems reported by mothers more than two years later, but only for children carrying the 7-repeat DRD4 allele (Bakermans-Kranenburg & van IJzendoorn, 2006). Importantly, however, children with the 7-repeat DRD4 allele also manifested the least externalizing behavior when mothers were highly sensitive.

A cross-sectional investigation of sensation seeking in 18-21-month-old children generated results in line with those just cited, with toddlers carrying the 7-repeat allele rated by parents as showing less sensation-seeking behavior when parenting quality was high, yet more when parenting quality was low, compared to children without the 7-repeat allele (Sheese, Voelker, Rothbart, & Posner, 2007). Whereas parenting proved significantly associated with sensation seeking in the 7-repeat individuals, then, it did not in other children. Of importance is that genotype did not predict parenting or sensation seeking, thereby discounting the possibility that children carrying certain genes evoked the parenting they received. Were that to have been the case, the situation would be better characterized as gene-environment correlation rather than gene-environment interaction.

Experimental intervention research designed to enhance parenting also documents a moderating effect of the 7-repeat allele on parenting. When Bakermans-Kranenburg, van IJzendorn, 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 1-3 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 that experimental effects extended to improvements in child behavior, but only for those children carrying the DRD4 7-repeat allele. These results are particularly important, not just because they are consistent with the previous findings considered, but because they clearly chronicle indisputably causal effects of parenting—in the case of genetically susceptible children.

In a recent cross-sectional analysis of a longitudinal prospective study, Knafo, et al. (2011) investigated whether DRD4 moderated the effects of mother-reported positivity in parenting on prosocial behavior in early childhood, using a sample of 167 3.5-year-old boys and girls. Among children who did not carry the DRD4 7-repeat allele, there was no significant relation between positivity in parenting and prosocial behavior. Among children carrying the DRD4 7-repeat allele, however, evidence of increased susceptibility emerged, as more positive parenting by the mothers proved related to more prosocial behavior by their children.

Finally, Berry, Deater-Deckard, McCartney, Wang, and Petrill (in press) investigated in a subsample of the large-scale longitudinal prospective NICHD Study of Early Child Care (n = 711) whether DRD4 moderated the effects of early sensitive parenting on attention-problem trajectories across middle childhood. The DRD4 7-repeat allele was associated with higher levels of inattention in the context of low-sensitive parenting but, consistent with a differential susceptibility pattern of interaction, the same allele was also associated with lower levels of inattention in the context of high sensitive care.

Discussion

There is now growing evidence that some individuals are more susceptible to both negative and positive parenting experiences than others as a function of genetic differences—consistent with the differential susceptibility hypothesis. Consequently, parenting behavior seems to exert more influence on some children than on others. In extremis, the same parenting behavior may affect some children substantially yet not affect others at all, depending on children's genetic make-up. In contrast to the "vulnerability" perspective central to diathesis-stress/dual-risk models of environmental action, being more or less susceptible to rearing experiences is not exclusively associated with the effects of adversity; this greater susceptibility can also operate with respect to supportive rearing environments (Pluess & Belsky, submitted). What needs to be made clear, then, is that whether or not being highly susceptible is considered advantageous or not depends entirely on the environment to which the child is exposed. Whereas more susceptible individuals would seem to benefit from responding more positively to supportive parenting, less susceptible children would seem to benefit more from being resilient in the face of contextual adversity, including problematic parenting.

Findings consistent with differential susceptibility suggest that the widely embraced diathesis-stress/dual-risk model of development may seriously misrepresent some developmental processes, especially with respect to how developmental plasticity operates: Some children may not be simply more vulnerable to adverse environments but in fact more susceptible to both negative and positive

experiences. One reason this possibility has rarely been discussed in the literature is probably a result of psychology's disproportionate focus on the *adverse* effects of *negative* experiences on *problems* in development and, thereby, the identification of individuals, including children, who—for organismic reasons—are particularly "vulnerable" to contextual risks or "protected" from them. What the differential susceptibility hypothesis stipulates, in contrast, is that the very children who are putatively "vulnerable" to adversity vis-à-vis problems in development may be equally and disproportionately susceptible to the developmentally *beneficial* effects of *supportive* rearing environments.

This fundamentally different understanding may require some reconsidering—and recasting—of common concepts like "vulnerability" and "resilience". "Vulnerability" may represent just one side of plasticity—the negative or "dark" side—and therefore reflect only part of the developmental story. The observation that so-called "vulnerable" children will also benefit disproportionately from positive environments calls for a different, more neutral, term. Recently, Manuck and associates (2011; Sweitzer et al., submitted) introduced the term *Vantage Sensitivity* to characterize this "positive or bright side" of differential susceptibility which we embrace and promote not only to describe the positive end of differential susceptibility, but, more generally, variability in response to exclusively positive experiences (Pluess & Belsky, submitted). "Resilience", generally understood as the advantageous ability to withstand negative effects of adverse environments, may represent a general immunity to environmental influences of all kinds, including positive ones, not just to adversity. To the extent that this is the case, "resilience", typically regarded as an advantage (in adverse environments), would seem to be disadvantageous, too—in supportive environments. In these latter contexts, the highly susceptible will reap developmental benefits whereas the low susceptible, including perhaps the resilient, will not, or do so to a far lesser extent. We choose the term *Vantage Resistance* to describe this observation (Pluess & Belsky, submitted).

It is important to clearly distinguish between differential susceptibility, diathesis-stress, and vantage sensitivity. Whereas differential susceptibility calls attention to individual differences in developmental plasticity—both for better *and* for worse—and diathesis-stress calls attention to the for-worse, "dark side" only, vantage sensitivity is only about the for-better, "bright side". This distinction raises the intriguing possibility that whereas some individuals may be disproportionately susceptible to negative experiences and exposures, consistent with diathesis stress, others may be disproportionately susceptible to positive environmental conditions, consistent with vantage sensitivity. And still others may be disproportionately susceptible to both—or to neither (see Figure 1 for a graphic illustration of these concepts).

In conclusion, parenting does not seem to exert the same effect on all children. Although this observation itself is not new given the widely accepted notion of non-shared environmental effects (Plomin & Daniels, 1987), what differential-susceptibility thinking adds to this understanding is the possibility that some children will generally be more and some generally less affected by both positive and negative parenting. The differential susceptibility hypothesis, then, represents a new perspective regarding differences in response to parenting experiences, based on the theoretical premise that individuals differ fundamentally in their developmental plasticity. Differential susceptibility reasoning extends diathesis-stress claims that some individuals are more likely than others to be adversely affected by negative developmental experiences by calling attention to the fact that these same putatively "vulnerable" individuals are simultaneously more susceptible to positive environmental influences.

Finally, besides important implications of differential susceptibility for the understanding of parenting effects from a theoretical perspective, re-thinking and even re-conceptualizing so-called genetically "vulnerable" children as children highly susceptible to the benefits of supportive rearing environments as well as the costs of poor ones could prove highly useful to practitioners when dealing with parents and their children. Viewing children as having more or less developmental plasticity instead of simply being "vulnerable" to adversity or not may create hope for many parents who often feel overwhelmed when dealing with more challenging infants and toddlers. Knowing that the return on a heavy investment in sensitive parenting may be substantial on the upside could provide the motivation to work hard to do so when exhaustion and irritation rise to the surface.

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