Chapter 6

DIFFERENTIAL SUSCEPTIBILITY TO MATERNAL SENSITIVITY

Michael Pluess and Jay Belsky

ABSTRACT

A vast body of empirical evidence highlights the contribution of maternal sensitivity to multiple features of children's development. Long appreciated, too, is that parenting effects frequently vary across children, often moderated by characteristics of the child such as early temperament. Most such work has been guided by conceptions of vulnerability stemming from negative or difficult temperament that interacts with insensitive, harsh or otherwise unsupportive parenting to undermine child well-being. Belsky's (1997b; 2005) differential-susceptibility framework challenges such diathesis-stress thinking, highlighting the fact that the very individuals who seem most susceptible to environmental adversity—including negatively emotional infants, toddlers, and preschoolers—may also benefit the most from developmentally supportive rearing. In other words, some children may be more affected than others by both highly sensitive and insensitive parenting. Evidence consistent with this view is reviewed, including research on temperament-X-parenting and gene-X-environment interaction. Finally, potential implications of the differential susceptibility perspective regarding the understanding of parenting effects are discussed.

INTRODUCTION

It is widely acknowledged, both in scientific and consumer publications, that maternal sensitivity is a—if not *the*—crucial ingredient for the healthy psychosocial development of children. This understanding is not simply based on popular wisdom but has been convincingly confirmed by empirical evidence. For example, controlled experimental studies informed by attachment theory demonstrate repeatedly that sensitive mothering causally influences the development of attachment security in the infant and young child (De Wolff & Van IJzendoorn, 1997; Bakermans-Kranenburg, Van IJzendoorn, & Juffer, 2003). In other words, infants with sensitive mothers are more likely to have a secure attachment.

But is that true for every child? Are all children affected by maternal sensitivity to the same extent? Evidence that maternal sensitivity may have different effects in different children emerges consistently in research investigating the interaction of temperament and parenting on developmental outcomes. For example, in children with difficult temperament at 12 months of age, low maternal sensitivity has been found to predict externalizing behavior problems at three years (Belsky, Hsieh, & Crnic, 1998). And infants with difficult temperaments at six months had more anxiety/depressive symptoms at two years when experiencing lower levels of maternal sensitivity whereas children with easy temperaments were not affected by low maternal sensitivity regarding such symptoms (Warren & Simmens, 2005). These findings suggest that some children—specifically those characterized by difficult temperament in infancy—are more affected by low levels of maternal sensitivity.

This form of temperament-X-parenting interaction is 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 temperamentally "vulnerable" (diathesis = difficult temperament), with children who fail to succumb to the same adversity regarded as "resilient" (Zubin & Spring, 1977). Diathesis-stress/dual-risk models imply also that under conditions of environmental support, including supportive parenting, "vulnerable" and "resilient" individuals will not differ in their functioning. In other words, it is only under conditions of adversity that their differential susceptibility to environmental influence is operative; Figure 1, an adaptation of Bakermans-Kranenburg and Van IJzendoorn (2007), graphically depicts this characterization.

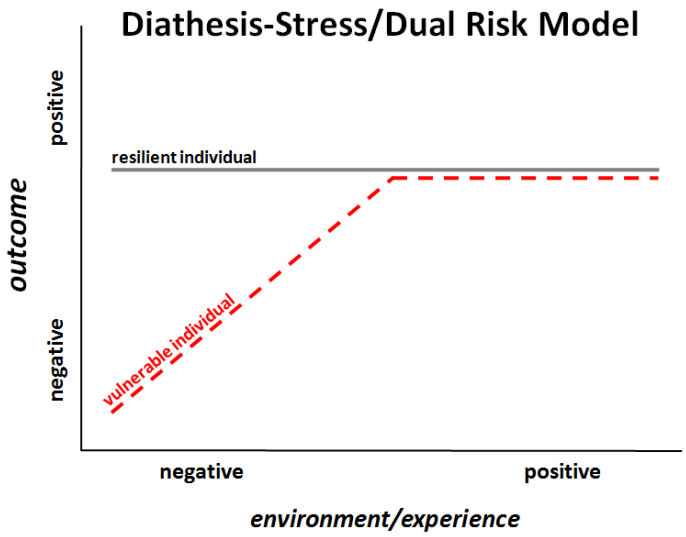

Figure 1. Graphical display of the diathesis-stress/dual risk model (an adaptation of Bakermans-Kranenburg and Van IJzendoorn's (2007) Figure 1). The X-axis indicates quality of the environment/experiences from negative to positive. The Y-axis indicates the developmental outcome from negative to positive. The lines depict two categorical groups that differ in their responsiveness to the environment: the "vulnerable" group shows a negative outcome when exposed to a negative environment, while the "resilient" group is not affected by it. No differences between the two groups emerge in a positive environment.

However, are infants with difficult temperaments 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 temperament-parenting interactions from a diathesis-stress/dual-risk perspective has been challenged recently by Belsky's (1997b; 1997a; 2005) differential susceptibility hypothesis, which stipulates that individuals do not so much vary in the degree to which they are vulnerable to the negative effects of adverse experience, but more generally so 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" ones—will be far less or not at all affected by the same environmental conditions (see Figure 2, an adaptation of Bakermans-Kranenburg & Van IJzendoorn, 2007). Boyce and Ellis (2005) recently advanced a perspective somewhat similar to differential susceptibility which focused exclusively on the role of the stress-response system in moderating environmental effects (i.e., "biological sensitivity to context").

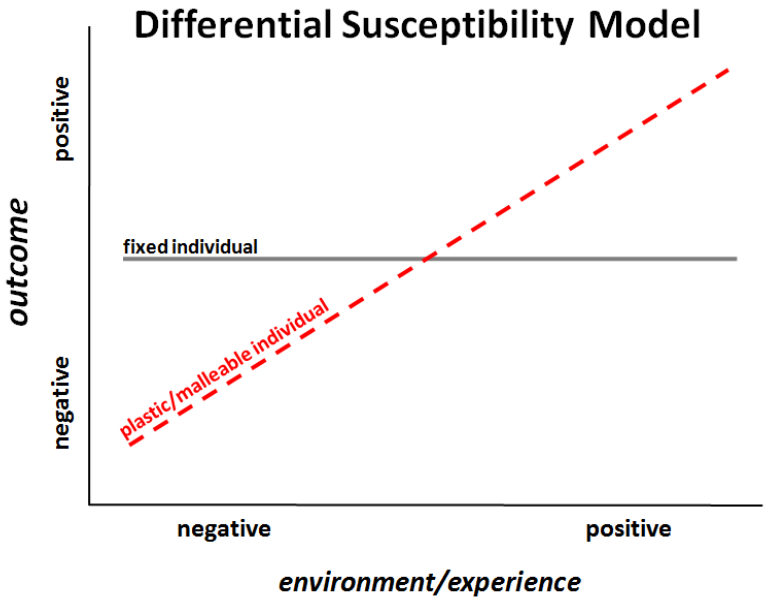

Figure 2. Graphical display of the differential susceptibility model (an adaptation of Bakermans-Kranenburg and Van IJzendoorn's (2007) Figure 1). The X-axis indicates quality of the environment/experiences from negative to positive. The Y-axis indicates the developmental outcome from negative to positive. The lines depict two categorical groups that differ in their responsiveness to the environment: the "plastic" group is disproportionately more affected by both negative and positive environments. Plastic individuals have a more negative outcome in response to negative environments (i.e. negative slope) but also a more positive outcome in response to positive environments (i.e. positive slope) compared to the "fixed" individuals.

In what follows, we first delineate the theoretical foundation of Belsky's (1997b; 2005) differential susceptibility hypothesis and then provide a review of recent evidence regarding developmental effects of maternal sensitivity consistent with it. In a concluding section, we

discuss potential implications of the differential susceptibility perspective regarding the understanding of parenting effects and developmental processes in general.

THEORETICAL FOUNDATION OF DIFFERENTIAL SUSCEPTIBILITY

The view that children should vary in their susceptibility to rearing experiences is founded on evolutionary logic which regards the dispersion of genes in future generations as the ultimate biological imperative and thus goal of all living beings. Indeed, from the perspective of modern evolutionary biology, natural selection does not just shape living things to survive, but to reproduce. Importantly, such reproduction can be direct, as when one produces immediate descendants (i.e., children, grandchildren), but also indirect, as when one's kin—such as brother, sister, niece or nephew—reproduce and, in so doing, pass on genes that they share, in varying proportions, with the individual in question. "Reproductive fitness" refers to the dispersion of one's genes in future generations and "inclusive fitness" calls attention to the fact that one's genetic material is distributed both directly and indirectly. With this foundation established, we turn to the theoretical argument.

Because the future is and always has been inherently uncertain, ancestral parents, just like parents today, could not have known (consciously or unconsciously) what childrearing practices would prove most successful in promoting the reproductive fitness of offspring—and thus their own inclusive fitness. As a result, and as a fitness optimizing strategy involving the hedging of bets, natural selection would have shaped parents to bear children varying in developmental plasticity, with some children being more affected by the parenting they experience than others (Belsky, 2005). This way, if an effect of parenting proved counterproductive in fitness terms, those children less—or not at all—affected by parenting would not have incurred the cost of developing in ways that ultimately proved "misguided" when it came to passing on genes to future generations. Importantly, in light of inclusive-fitness considerations, these less malleable children's "resistance" to parental influence would not only have benefited themselves directly, but their more malleable siblings as well—but indirectly, given that siblings, like parents and children, have 50% of their genes in common. By the same token, had parenting influenced children in ways that enhanced fitness, then not only would more plastic or malleable offspring have benefited directly by virtue of parental influence, but so, too, would their parents and even their less malleable siblings who did not benefit from the parenting they received, again for inclusive-fitness reasons (i.e., shared genes). This line of evolutionary argument leads directly to the expectation that children should vary in their plasticity and thus susceptibility to parental rearing and perhaps to environmental influences more generally. It might also lead to the prediction that variation in malleability should have differential fitness payoffs today, just like it is presumed to have had in ancestral times. To date, no such empirical tests of reproductive fitness have been conducted. Moreover, even if they were, it remains possible that fitness would no longer be related to individual differences in plasticity due to how much the modern world differs from the one in which humans evolved and have even lived until recently.

EVIDENCE OF DIFFERENTIAL SUSCEPTIBILITY

It is one thing to assert that some children may be more affected by parenting experiences than others—for better *and* for worse—and yet another to chronicle empirically such a fact. In this section we present selected evidence of differential susceptibility to the effects of parenting; for analysis of differential susceptibility to a wide variety of environmental influences, see Belsky & Pluess (2009a). First we consider research that addresses behavioral or phenotypic characteristics of children found to moderate effects of maternal sensitivity, followed by gene-by-environment interaction (GXE) evidence showing that genotype moderates such environmental effects.

Temperament as a Phenotypic Marker of Differential Susceptibility

Some of the earliest and most suggestive evidence of differential susceptibility to environmental influences emerged in research on temperament-X-parenting interaction (Belsky, 1997a), a long-standing focus of developmental inquiry (Rothbart & Bates, 2006). Belsky's (2005) review of relevant research revealed that predictive links between rearing experience and a variety of behavioral outcomes often were consistently greater for a subgroup of children characterized by a temperamental propensity for high negative affectivity, whether operationalized in terms of difficult temperament, irritability, fearfulness, or inhibition. But even though most of the work considered revealed that *greater variance* in a variety of developmental outcomes could be explained by rearing experiences in the case of more negatively emotional children, statistical analyses in the studies in question often did not afford determination of whether this result was itself a function of a for-better-*and*-for-worse parenting effect. Fortunately, the situation is different today, with many new studies chronicling such individual differences in developmental plasticity.

Consider, for example, Van Aken, Junger, Verhoeven, and Dekovic's (2007) evidence that 16-19-month-old boys with difficult temperament (i.e., susceptibility factor) manifest the smallest increase six months later in externalizing problems scores when reared by highly sensitive mothers who only infrequently used negative control, but the largest increase when highly insensitive mothers relied heavily on negative control. These striking parenting effects simply did not obtain in the case of other children.

Drawing on data of the large scale longitudinal NICHD Study of Early Child Care and Youth Development (NICHD Early Child Care Research Network [ECCRN], 2005) and focusing upon maternally-reported difficult temperament (i.e., susceptibility factor) at one and six months (composited), Bradley and Corwyn (2008) also discerned evidence of differential susceptibility when it came to evaluating effects of observed maternal sensitivity, harshness and productive activity on teacher-reported behavior problems: Children with more difficult temperaments had more behavior problems in first grade than all other children if they experienced low-quality parenting across the infant, toddler, and preschool years, but fewer problems than all other children if they experienced high-quality parenting; the anticipated effect of maternal sensitivity 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).

Dopkins-Stright, Cranley-Gallagher, and Kelley (2008) were able to extend findings of differential susceptibility to positive developmental outcomes, also drawing on data from the NICHD Study. Once again an interaction emerged, this time between difficult temperament (at 6 months) and parenting quality (composited across 6, 15, 24, 36 and 54 months) in the prediction of teacher-rated academic competence, social skills, teacher-child relationships, and peer-status at first grade. Predictive power proved greater for infants with more difficult temperaments than for infants with less difficult temperaments. Although all interactions were of a cross-over nature and in line with a for-better-*and*-for-worse parenting effect for only some children, not all criteria for differential susceptibility were met (Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2007); of special significance is that the temperament susceptibility factor itself predicted the parenting predictor (as well as at least one outcome measured).

Pluess and Belsky (2010) overcame this problem in their longer-term analysis of differential susceptibility using the same NICHD Study data. Parenting quality prior to school entry predicted reading, math, picture vocabulary, social competence, and academic work habits in the fifth grade more strongly for children with difficult temperament than for those with easy temperaments. After repeating the analysis using a parenting quality measure that has been statistically adjusted for the effect of six-month, mother-reported difficult temperament the findings remained virtually unchanged.

Although the work of Pluess and Belsky (2010) indicates that differential-susceptibility effects pertaining to early parenting and involving temperament-based susceptibility factors extend beyond the early childhood years, the question arises as to whether rearing and related experiences *in later childhood* operate in a similar manner. Some suggestive evidence that they do comes from Lengua's (2008) temperament-X-parenting interaction study which sought to explain change in externalizing problems using a community sample of 8-12 year olds. Children's reports of their mothers' parenting style (i.e., rejection/acceptance, inconsistent discipline) predicted change over a one-year period in mother-reported internalizing and externalizing problems, but differentially as a function of temperament. Children highly prone to frustration increased in externalizing problems over time when mothers were rejecting, but decreased when mothers manifest little rejection, with no such apparent effects of rejection evident in the case of children scoring low on frustration.

The repeatedly discerned moderational effect of negative emotionality/difficult temperament in the case of parenting (and other environmental experiences, see Belsky & Pluess, 2009a) raises the question of why this should be the case. This would seem especially important to address in view of the fact that even though Belsky (1997a; 2005) theorized that children should vary in their susceptibility to environmental influences (i.e., plasticity), his differential-susceptibility hypothesis did not stipulate that more negatively emotional children or those with difficult temperament (or with high sensory-processing sensitivity) would prove especially malleable; this was an empirical observation (Belsky, 2005). As it turns out, several non-mutually exclusive explanations have been advanced with regard to the issue at hand. Kochanska (1993) drew explicitly on Dienstbier's (1985) thinking on anxiety to account for her differential-susceptibility results, arguing that more negatively emotional/fearful/inhibited infants have lower thresholds for anxiety, thereby making them more easily aroused by discipline and thus responsive to it. Not unrelatedly, Belsky (2005) contended that a negatively emotional/difficult temperament reflects a highly sensitive

nervous system on which experience—of both the positive and negative variety—registers especially strongly (see also Aron & Aron, 1997).

Whatever the mechanisms involved in making more negatively emotional children seemingly more malleable—in an often for-better-*and*-for-worse manner—it would be mistaken to conclude that this is the most important phenotypic marker of plasticity. Even though this could turn out to be the case, it could also be an artifact of the disproportionate attention that investigators guided by the diathesis-stress perspective pay to so-called "risk factors" that interact with contextual adversity in producing problematic functioning (Belsky & Pluess, 2009b). If this is so, then it certainly behooves us to consider other potential behavioral markers of plasticity/malleability rather than rely on a single one. At this point, however, the only empirically supported behavioral susceptibility markers besides negative emotionality and difficult temperament are sensory-processing sensitivity (Aron & Aron, 1997; Aron, Aron, & Davies, 2005), impulsivity (Lengua, Wolchik, Sandler, & West, 2000), and anger proneness (Smeekens, Riksen-Walraven, & van Bakel, 2007). Certainly there might be other susceptibility factors and, indeed, identification of them remains a wide-open theoretical, as well as empirical, issue.

Genetic Markers of Differential Susceptibility

Findings of differential susceptibility are not restricted to behavioral markers of plasticity. Recent advancement in technology enabled the inclusion of genetic data in developmental studies in order to investigate how specific genes interact with the environment in the prediction of developmental outcomes. 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 of the same gene (alleles). Because every individual has two complete sets of chromosomes, the existence of a two-allelic polymorphism of a specific gene means that an individual can have one out of three different combinations of gene variants: (1) both chromosomes carry the gene variant A; or (2) both chromosomes carry the gene variant B; or (3) one of the chromosome sets carries gene variant A and the other one variant B. Those carrying two copies of the same gene variant are called homozygotes, whereas those with one of each are referred to as heterozygotes.

In the following section, we call attention to gene-by-environment (GXE) findings involving a polymorphism in the dopamine receptor D4 (DRD4) gene, which is coding for a protein that is transmitting dopamine signals from one neuron to another. The dopaminergic system 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 quality parenting than other children, but also benefit more than others from good-quality rearing. In a longitudinal study of infants, maternal insensitivity observed when children were 10 months predicted 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). Moreover, although children with the 7-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.

A cross-sectional study of sensation seeking in 18- to 21-month-old children generated results in line with those of Bakermans-Kranenburg and Van IJzendoorn (2006), with toddlers carrying the 7-repeat allele rated by parents as showing, compared to children without the 7-repeat allele, less sensation seeking behavior when parenting quality was high and more when parenting quality was low (Sheese, Voelker, Rothbart, & Posner, 2007). Whereas parenting proved significantly associated with sensation seeking in the 7-repeat individuals, 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 chararacterized as gene-environment correlation than gene-X-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- 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 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, in the case of some children, of parenting. In the other non-experimental studies carried out, which are correlational in nature even if longitudinal in design, one can never be certain that true effects of parenting are being detected. After all, some other unmeasured factor could be the source of apparent influence of parenting detected in such work.

CONCLUSION

A substantial number of studies, rather diverse in their focus and design, shows that some individuals are more susceptible to both negative and positive parenting experiences than others—just as the differential susceptibility hypothesis would predict. Consequently, parenting behavior—including maternal sensitivity—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 their temperament and/or genetic make-up. In contrast to the "vulnerability" concept 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 also operates with respect to supportive rearing environments. What needs to be made clear is that whether or not being highly malleable is considered advantageous or not depends entirely on the environment to which the child is exposed. Whereas more plastic individuals would seem to benefit from

responding more positively to supportive parenting, less malleable children would seem to benefit more from not being susceptible to the adverse consequences of problematic parenting.

The findings consistent with differential susceptibility summarized herein suggest that the widely embraced diathesis-stress/dual-risk model of development may seriously misrepresent some developmental processes, especially 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 postulates, 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 actually require the recasting of common concepts like "vulnerability" and "resilience." "Vulnerability" may represent just one side of plasticity—the negative one—and therefore reflect only half of the story. The observation that so-called "vulnerable" children will also benefit disproportionately from positive environments calls for a different, more neutral, term—susceptibility, plasticity, or malleability. "Resilience," generally understood as the advantageous ability to withstand negative effects of adverse environments, may in fact 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 malleable will reap developmental benefits whereas the less malleable, including perhaps the resilient, will not or do so to a far less extent.

The research considered herein suggests that parenting effects are moderated in a differential-susceptibility manner not just by a single factor but, rather, by different ones, some phenotypic (i.e., temperament) and some genotypic (i.e., DRD4). Interestingly, some data indicates that early infant temperament is related to the dopamine DRD4 polymorphism (Auerbach, Faroy, Ebstein, Kahana, & Levine, 2001) suggesting the possibility that putatively different differential-susceptibility factors may actually be manifestations of the same underlying disposition.

An important question is whether susceptibility is a function of nurture—consequently modifiable—or whether it represents an innate and therefore unchangeable trait, a function of nature. The review of GXE findings certainly suggests a direct genetic contribution to individual susceptibility. Moreover, the above mentioned significant association between genetic and phenotypical susceptibility factors (Auerbach et al., 2001) suggests that the genetic contribution—or at least some of it—is mediated by behavioral susceptibility factors.

However, evidence that susceptibility may also be a function of nurture stems from research showing that early experiences—both prenatal and postnatal—predict phenotypical susceptibility factors. Consider in this regard research showing that maternal stress during pregnancy predicts difficult temperament at three months of age (Huizink, de Medina, Mulder, Visser, & Buitelaar, 2002) and that maternal sensitivity predicts emotional

responsivity in four-month old infants (Kaplan, Evans, & Monk, 2007). Consequently, differential susceptibility may be best understood as the function of both nature and nurture (Boyce & Ellis, 2005; Belsky & Pluess, 2009b).

In conclusion, differential susceptibility represents a fundamentally new and different theoretical approach regarding how maternal sensitivity operates, based as it is on the theoretical premise that individuals differ 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. As already noted, this raises questions about the use of the terms "vulnerability" and "resilient" to describe individuals.

Reconceptualizing so-called "vulnerable" children as children highly susceptible to the benefits of supportive rearing environments as well as the costs of poor ones could prove useful to practitioners when dealing with parents and their "difficult" children. Viewing such children as having substantial developmental plasticity instead of simply being "difficult" and "vulnerable" to adversity may create hope for many parents who often feel overwhelmed when dealing with such 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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