

For Better *and* For Worse

Differential Susceptibility to Environmental Influences

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ABSTRACT—*Evidence that adverse rearing environments exert negative effects particularly on children presumed “vulnerable” for temperamental or genetic reasons may actually reflect something else: heightened susceptibility to the negative effects of risky environments and to the beneficial effects of supportive environments. Building on Belsky’s (1997, 2005) evolutionary-inspired proposition that some children are more affected—both for better and for worse—by their rearing experiences than are others, we consider recent work on child vulnerability, including that involving measured genes, along with evidence showing that putatively vulnerable children are especially susceptible to both positive and negative rearing effects. We also consider methodological issues and unanswered questions in the differential-susceptibility equation.*

KEYWORDS—*differential susceptibility; gene–environment interaction; parenting; temperament*

Most students of child development probably do not presume that all children are equally susceptible to rearing effects; a long history of research on interactions between parenting and temperament, or *parenting-by-temperament interactions*, clearly suggests otherwise. Nevertheless, it remains the case that most work still focuses on parenting effects that apply equally to all children—so-called main effects of parenting—thus failing to consider interaction effects, which reflect the fact that whether, how, and how much parenting influences the child may depend on the child’s temperament or some other characteristic of individuality.

Like classic work in educational and clinical psychology on interactions between learning aptitude and treatment, research on parenting-by-temperament interactions is based on the premise that what proves effective for some individuals in fos-

tering the development of some valued outcome—or preventing some problematic one—may simply not do so for others. Commonly tested are hypotheses derived from multiple-risk/transactional frameworks in which individual characteristics that make children “vulnerable” to adverse experiences—placing them “at risk” of developing poorly—are mainly influential when there is at the same time some contributing risk from the environmental context.

After highlighting some research of just this kind, we raise questions—on the basis of other findings—about how the first set of data has been interpreted. We advance the evolutionary-inspired proposition that some children, for temperamental or genetic reasons, are actually more susceptible to *both* (a) the adverse effects of unsupportive parenting *and* (b) the beneficial effects of supportive rearing. The validity of this claim cannot be determined, however, so long as research focuses disproportionately on *vulnerable* (as opposed to merely *susceptible*) child characteristics and evaluates effects of *adverse* environments on *problematic* outcomes. What, then, would be required to distinguish vulnerability from susceptibility? We consider the answer after first reviewing research that meets the criteria for differential susceptibility. Finally, we draw conclusions and highlight some “unknowns in the differential-susceptibility equation.”

DUAL-RISK CONDITIONS AND CONSEQUENCES

The view that infants and toddlers manifesting high levels of negative emotion are at special risk of problematic development when they experience poor-quality rearing is widespread. Evidence of this comes from Morrell and Murray (2003), who showed that it was only highly distressed and irritable 4-month-old boys who experienced coercive and rejecting mothering at this age who continued to show evidence, 5 months later, of emotional and behavioural dysregulation. Relatedly, Belsky, Hsieh, and Crnic (1998) observed that infants who scored high in negative emotionality at 12 months of age and who experienced the least supportive mothering and fathering across their second and third years of life scored highest on externalizing problems

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at 36 months of age. And Deater-Deckard and Dodge (1997) reported that children rated highest on externalizing-behavior problems by teachers across the primary-school years were those who experienced the most harsh discipline prior to kindergarten entry and who were characterized by mothers at age 5 as being negatively reactive infants.

The adverse consequences of the co-occurrence of a child risk factor (e.g., negative emotionality) and problematic parenting also is evident in Caspi and Moffitt's (2006) ground-breaking research on gene-by-environment interaction. Young men followed from early childhood were most likely to manifest high levels of antisocial behavior when they had both a history of child maltreatment and a particular variant of the *MAO-A* gene, a gene previously linked to aggressive behaviour. Such results led Rutter (2006), like others, to speak of "vulnerable individuals," a concept that also applies to children putatively at risk for compromised development due to their behavioral attributes. But is "vulnerability" the best way to conceptualize the kind of parenting-by-child interactions under consideration?

VULNERABILITY OR DIFFERENTIAL SUSCEPTIBILITY?

Working from an evolutionary perspective, Belsky (1997, 2005) theorized that children, especially within a family, should vary in their susceptibility to both adverse and beneficial effects of rearing influences: Because the future is uncertain, in ancestral times, just like today, parents could not know for certain (consciously or unconsciously) what rearing strategies would maximize reproductive fitness. To protect against all children being steered, inadvertently, in a parental direction that proved disastrous at some later point in time, developmental processes were selected to vary children's susceptibility to rearing.

Belsky (1997, 2005) further observed that children high in negative emotion, particularly in the early years, appeared to benefit disproportionately from supportive rearing environments (Boyce & Ellis, 2005). Crockenberg (1981) showed that social support predicted infant attachment security but only in the case of highly irritable infants. Denham et al. (2000) reported that the beneficial effects of proactive parenting (i.e., supportive presence, clear limit setting) at age 7 and/or age 9 were most pronounced in the case of children who scored high on externalizing problems (i.e., disobedient, aggressive, angry) at an earlier time of measurement (i.e., mean age 55 months), even after controlling for problem behavior at the initial measurement occasion.

Experimental studies designed to test Belsky's (1997) theory are even more suggestive of differential susceptibility than the longitudinal-correlational evidence. Blair (2002) discovered that it was highly negative infants who benefited most—in terms of both reduced levels of externalizing behavior problems and enhanced cognitive functioning—from a multifaceted infant-toddler intervention program whose data he reanalyzed. More recently, Klein Velderman, Bakermans-Kranenburg, Juffer, and

Van IJzendoorn (2006) found that experimentally induced changes in maternal sensitivity exerted greater impact on the attachment security of highly negatively reactive infants than it did on other infants. In both experiments, environmental influences on "vulnerable" children were for better instead of for worse.

Better Evidence of Differential Susceptibility

Even though studies highlight the heightened susceptibility of temperamentally negative or genetically vulnerable offspring to either positive or negative rearing influences, more compelling would be data on a single sample substantiating the for-better-and-for-worse predictions of the differential-susceptibility hypothesis. Feldman, Greenbaum, and Yirmiya (1999) found that 9-month-olds scoring high on negativity who experienced low levels of synchrony in mother–infant interaction manifested more noncompliance during clean-up at age two than other children did. When such infants experienced mutually synchronous mother–infant interaction, however, they displayed greater self-control than did children manifesting much less negativity as infants. More recently, Kochanska, Aksan, and Joy (2007) observed that highly fearful 15-month-olds experiencing high levels of power-assertive paternal discipline were most likely to cheat in a game at 38 months, yet when cared for in a supportive manner such negatively emotional, fearful toddlers manifested the most rule-compatible conduct.

Recent studies involving measured genes and measured environments also document both-for-better-and-for-worse rearing effects in the case of susceptible infants, specifically those with a particular allele (variant) of a gene called *DRD4*, which codes for a type of dopamine receptor. Because the dopaminergic system is engaged in attentional, motivational, and reward mechanisms and the variant in question, the 7-repeat allele, has been linked to lower dopamine reception efficiency, Van IJzendoorn and Bakermans-Kranenburg (2006) predicted this allele would moderate the association between maternal unresolved loss or trauma and infant attachment disorganization. Having the 7-repeat *DRD4* allele substantially increased risk for disorganization in children exposed to maternal unresolved loss/trauma, as expected; but when children with that allele were raised by mothers who had no unresolved loss, they displayed significantly less disorganization than age-mates without the allele, regardless of mothers' unresolved-loss status (Bakermans-Kranenburg & Van IJzendoorn, in press).

Similar results emerged when the interplay between *DRD4* and observed parental insensitivity in predicting externalizing problems was studied in a group of 47 twins (Bakermans-Kranenburg & Van IJzendoorn, 2007). Children with the 7-repeat *DRD4* allele and insensitive mothers displayed more externalizing behaviors than children without that allele (irrespective of maternal sensitivity); and children with the 7-repeat *DRD4* allele and sensitive mothers showed the lowest

levels of externalizing problem behavior (Bakermans-Kranenburg & Van IJzendoorn, 2007). Such results suggest that conceptualizing the 7-repeat *DRD4* allele exclusively in risk-factor terms is misguided, as this variant of the gene seems to heighten susceptibility to a wide variety of environments, with supportive and risky contexts promoting, respectively, positive and negative outcomes.

DETECTING DIFFERENTIAL SUSCEPTIBILITY

An environmental effect, be it involving parenting or something else, moderated by an organismic characteristic, be it temperamental negativity or genetic makeup, is a necessary condition for differential susceptibility but not a sufficient one. It would thus be a mistake to presume that all gene-by-environment (or temperament-by-parenting) interactions are examples of differential susceptibility. Differential susceptibility needs to be distinguished from other interaction effects, including that of “dual risk,” which arises when the most “vulnerable” individuals (i.e., risk #1) are disproportionately affected in an adverse manner by a negative environment (i.e., risk #2) but do not also benefit disproportionately from positive environmental conditions). It is also important that there be no association between the moderator (i.e., the susceptibility factor) and the environment (i.e., the predictor). Belsky et al. (1998) tested the independence of negative emotionality and parenting as a step in their investigation of differential susceptibility. Had these factors been correlated, then the evidence would not have shown that the predictive power of parenting was greater for highly negative infants; it would instead have indicated either that high-negativity infants elicit negative parenting or that negative parenting fosters infant negativity. Similarly, Caspi and Moffitt (2006) determined that boys’ *MAO-A* genotype did not elicit maltreatment.

The formal test of differential susceptibility consists of five steps (see Box 1). The first step concerns the application of conventional statistical criteria for evaluating genuine moderation (Dearing & Hamilton, 2006), with some emphasis on

BOX 1

Stepwise Testing for Differential Susceptibility

Distinguishing true differential susceptibility from other types of interaction proceeds in five steps, as follows:

1. Statistical test for genuine (cross-over) interaction
2. Test of the independence of the susceptibility factor and the predictor
3. Test of the association between the susceptibility factor and the outcome; if the association is nonzero, there is no support for differential susceptibility
4. Comparison of the regression plot with the prototypical graphical displays shown in Figure 1; only the first model (a) represents differential susceptibility
5. Test of the specificity of the model by replacing susceptibility factors and outcomes

excluding interactions with regression lines that do not cross (sometimes referred to as *removable* interactions). The next steps distinguish differential susceptibility from gene–environment correlations that may reflect rearing experiences evoked by genotypes (step 2) and from dual-risk models (steps 3 and 4), as defined above. If the susceptibility factor and the outcome are related, dual risk (or gain, when positive factors are involved) is suggested (Fig. 1, model d). For example, early negativity would itself lead to externalizing behavior, but even more so when combined with negative parenting. The specificity of the effect is demonstrated (step 5) if the model is not replicated when other susceptibility factors (i.e., moderators) and outcomes are used (Caspi & Moffitt, 2006; Rutter, 2006). Differential susceptibility is demonstrated when the moderation reflects a cross-over interaction (Fig. 1, model a) that covers both the positive and the negative aspects of the environment (i.e., susceptibility instead of dual risk). The slope for the susceptible subgroup should be significantly different from zero and at the same time significantly steeper than the slope for the nonsusceptible subgroup (i.e., differential instead of general susceptibility). If both slopes are significantly different from zero but in opposite directions, contrastive effects are indicated (Fig. 1, model c), as in the case of positive and negative effects of harsh discipline on, respectively, African American and White children (Deater-Deckard & Dodge, 1997).

UNKNOWN IN THE DIFFERENTIAL-SUSCEPTIBILITY EQUATION

The notion of differential susceptibility, derived as it is from evolutionary theorizing, has only recently been stated in a clear and testable form (Belsky, 1997, 2005). Although research summarized here suggests that the concept has utility, there are many “unknowns,” four of which are highlighted.

Domain General or Domain Specific?

Is it the case that some children, perhaps those who begin life as highly negatively emotional, are more susceptible both to a wide variety of rearing influences and with respect to a wide variety of developmental outcomes—as is presumed in the use of concepts like “fixed” and “plastic” strategists (Belsky, 2005), with the latter being highly malleable and the former hardly at all? Boyce and Ellis (2005) contend that a general psychobiological reactivity makes some children especially vulnerable to stress and thus to general health problems. Or is it the case, as Belsky (2005) wonders and Kochanska et al. (2007) argue, that different children are susceptible to different environmental influences (e.g., nurturance, hostility) and with respect to different outcomes? Pertinent to this idea are findings of Caspi and Moffitt (2006) indicating that different genes differentially moderated the effect of child maltreatment on antisocial behavior (*MAO-A*) and depression (*5HTT*).

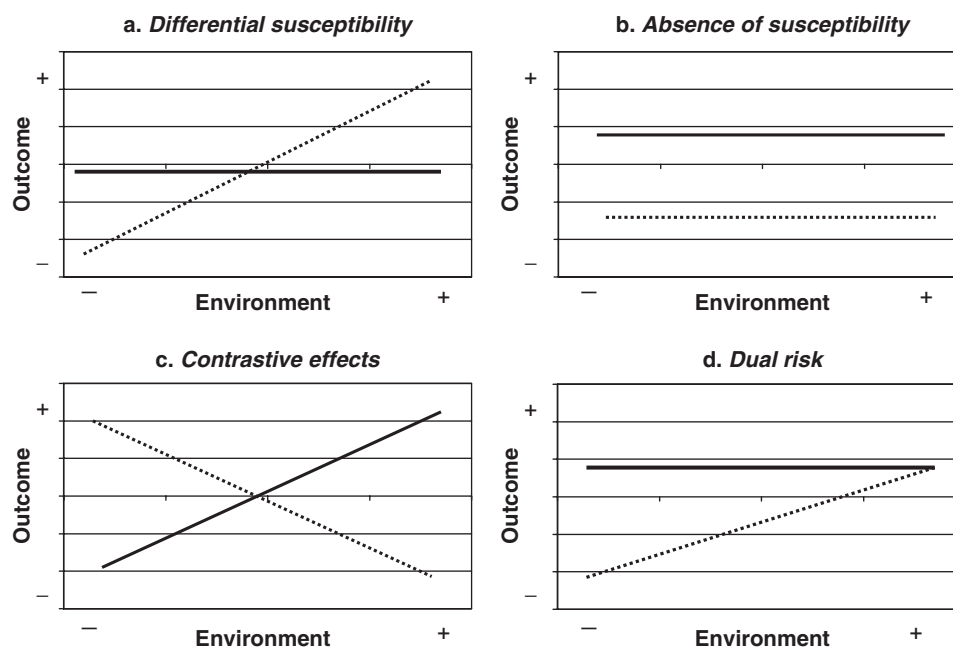


Fig. 1. Graphical display of different moderation effects. The x-axis indicates variation in the environmental factor from negative to positive; the y-axis indicates the outcome from negative to positive; and the lines depict the two groups differing on the susceptibility factor. Model a represents differential susceptibility. Model b depicts absence of susceptibility (fixed strategies)—that is, the two groups show different outcomes but variation in the environmental factor does not affect the outcome. In model c, the regression lines reflect contrastive effects. Model d represents a fan-shaped interaction, with the moderator affecting the outcome in just one direction.

Also worth considering is the prospect that heritable (or experientially induced) variation in positive emotionality (e.g., exuberance) moderates effects of rearing experiences on positive developmental outcomes (e.g., empathic concern). Perhaps negative emotionality emerges as a differential-susceptibility marker due to the disproportionate focus upon negative developmental outcomes in so much research.

Continuous Versus Discrete Plasticity?

The central argument that children vary in their susceptibility to rearing influences raises the question of how to conceptualize differential susceptibility: categorically (some children highly plastic and others not so at all) or continuously (some children simply more malleable than others)? It may even be that plasticity is discrete for some environment–outcome relations, with some individuals affected and others not at all (e.g., gender-specific effects), but that plasticity is more continuous for other susceptibility factors (e.g., in the case of the increasing vulnerability to stress of parents with decreasing dopaminergic efficiency; Van IJzendoorn, Bakermans-Kranenburg, & Mesman, 2007).

Mechanisms

Susceptibility factors are the moderators of the relation between the environment and developmental outcome, but they do not elucidate the mechanism of differential influence. Several (non-

mutually exclusive) explanations have been advanced for the heightened susceptibility of negatively emotional infants. Suomi (1997) posits that the timidity of “uptight” infants affords them extensive opportunity to learn by watching, a view perhaps consistent with Bakermans-Kranenburg and Van IJzendoorn’s (2007) aforementioned findings pertaining to *DRD4*, given the link between the dopamine system and attention. Kochanska et al. (2007) contend that the ease with which anxiety is induced in fearful children makes them highly responsive to parental demands. And Belsky (2005) speculates that negativity actually reflects a highly sensitive nervous system on which experience registers powerfully—negatively when not regulated by the caregiver but positively when coregulation occurs—a point of view somewhat related to Boyce and Ellis’ (2005) proposal that susceptibility may reflect prenatally programmed hyperreactivity to stress.

Within-Family Differences in Susceptibility

In light of evolutionary thinking about differential susceptibility (e.g., parental “bet hedging” or the trading off of costs and benefits), it is crucial to investigate within-family variation in susceptibility (Sulloway, 1996). Studies that include twins and other siblings from the same family might prove especially powerful, as they could distinguish genetically and environmentally induced variations in susceptibility. This will be especially the case if, in addition to measuring genes and

environments, studies also measured hypothesized moderators, thereby enabling investigators to move beyond globally attributing variance to “nonshared” family environment (i.e., those experiences that make children in the same family different from each other).

At best, work on differential susceptibility has only just begun. Issues raised here remain to be addressed empirically. Doing so may shed further light on why environmental effects seem so much smaller than they are often presumed to be.

Recommended Reading

- Belsky, J. (2005). (See References). A comprehensive presentation of theory and research on differential susceptibility that goes into more detail than the current article.
- Boyce, W.T., & Ellis, B. (2005). (See References). A thoughtful evolutionary analysis of differential susceptibility, advancing the original claim that heightened susceptibility may itself be environmentally induced, not just genotypically determined.
- Caspi, A., & Moffitt, T. (2006). (See References). A clearly written scholarly review of recent gene-by-environment interaction findings involving psychiatric disturbances in humans.
- Rutter, M. (2006). (See References). A book-length treatment of “gene–environment interplay” for non-geneticists, highlighting work on genotypic vulnerability to adverse rearing environments (and much more).
- Bakermans-Kranenburg, M.J., & Van IJzendoorn, M.H. (in press). (See References). The first experimental evidence of differential susceptibility in the case of attachment and externalizing behavior involving a genetic susceptibility factor.
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