

Vantage Sensitivity: Individual Differences in Response to Positive Experiences

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The notion that some people are more vulnerable to adversity as a function of inherent risk characteristics is widely embraced in most fields of psychology. This is reflected in the popularity of the diathesis-stress framework, which has received a vast amount of empirical support over the years. Much less effort has been directed toward the investigation of endogenous factors associated with variability in response to positive influences. One reason for the failure to investigate individual differences in response to positive experiences as a function of endogenous factors may be the absence of adequate theoretical frameworks. According to the differential-susceptibility hypothesis, individuals generally vary in their developmental plasticity regardless of whether they are exposed to negative or positive influences—a notion derived from evolutionary reasoning. On the basis of this now well-supported proposition, we advance herein the new concept of *vantage sensitivity*, reflecting variation in response to exclusively positive experiences as a function of individual endogenous characteristics. After distinguishing vantage sensitivity from theoretically related concepts of differential-susceptibility and resilience, we review some recent empirical evidence for vantage sensitivity featuring behavioral, physiological, and genetic factors as moderators of a wide range of positive experiences ranging from family environment and psychotherapy to educational intervention. Thereafter, we discuss genetic and environmental factors contributing to individual differences in vantage sensitivity, potential mechanisms underlying vantage sensitivity, and practical implications.

Keywords: vantage sensitivity, positive psychology, differential susceptibility, diathesis-stress, gene-environment interaction

The notion that individuals often vary in their response to the same experience is widely appreciated in most subfields of psychology. In clinical and developmental psychology, there is an especially rich history of research aimed at investigating individual characteristics that predict differences in response to environmental influences (e.g., Garmezy, 1991; Luthar, 2006; Masten & Obradović, 2006; Rutter, Moffitt, & Caspi, 2006; Werner, 1997). Most such work is based, to varying degrees, on psychopathology and problematic development, thereby resulting in a focus on *vulnerability to adversity*. In fact, many of today's established concepts and models in psychology derive from or are influenced by a long history of empirical research on psychopathology and the treatment thereof. Appreciation of this disproportionate psychopathological bias in so much psychological theory and research

stimulated the emergence of the subfield of positive psychology at the beginning of this millennium (Seligman & Csikszentmihalyi, 2000). In consequence, the last decade has witnessed an outpouring of positive psychology research focused specifically on the investigation of optimal human functioning (Linley, Joseph, Harrington, & Wood, 2006; Lopez & Snyder, 2011; Seligman, 2011; Seligman, Steen, Park, & Peterson, 2005).

Though appreciated by many, positive psychology is not without its critics. Indeed, it has been chastised for pursuing a rather simplistic agenda while failing to acknowledge individual differences in its application (Gable & Haidt, 2005; Held, 2004; Lazarus, 2003). Whereas most subfields of psychology have a long history of investigating factors associated with variability in response to a variety of influences, research with a focus on positive psychology often appears based on the implicit assumption that positive influences will benefit most, if not all, individuals to the same degree. Nevertheless, variability in response to positive influences is ubiquitous. It can be easily observed, for example, that some children excel academically in a supportive educational environment, whereas other children do not, or at least not to the same degree. Similarly, not all children appear to achieve the same socioemotional maturity when growing up in a warm and supportive family environment.

Besides these simple examples of individual differences in benefit derived from positive environmental experiences and exposures, one can also consider variation in the effects of formal psychological interventions (e.g., Dinkel et al., 2012; Forbes et al.,

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We would like to express special appreciation to Stephen Manuck for providing us with the term *vantage sensitivity*.

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2012). After all, even the most generally efficacious treatments benefit some individuals more than others (e.g., DeRubeis et al., 2005; Ginsburg et al., 2011; Kennard et al., 2006). Although variability in response to treatment is generally appreciated in the literature and nonresponse is routinely assessed in evaluations of psychological interventions, the notion that individuals differ fundamentally in their general responsiveness *due to endogenous factors* rather than others—perhaps most especially fidelity of treatment—is rarely considered and therefore rarely investigated, except perhaps on a post hoc basis.

Conceivably, one important reason psychological research generally fails to entertain, or at least systematically examine, variability in response to positive experiences as a function of endogenous factors—beyond perhaps severity of disturbance—is due to the absence of theory stipulating that such should be the rule rather than the exception. It is the principal purpose of this article to address this lacuna. After introducing a new concept pertaining to variability in response to positive influences—derived from differential-susceptibility reasoning (Belsky & Pluess, 2009a)—we propose specific new terminology before highlighting conceptual differences between the new concept and both differential susceptibility and resilience. We then review recent empirical evidence consistent with the proposed framework. After subsequently addressing issues related to determinants and mechanisms of variability in response to positive experiences, we conclude by suggesting potential practical applications of the new concept.

From Diathesis-Stress to Differential Susceptibility

The dominant role that *diathesis-stress* thinking has played in much psychological research (Monroe & Simons, 1991; Zuckerman, 1999) no doubt accounts in part for the dearth of theoretical models addressing variation in response to putatively positive environmental experiences and exposures, a point Belsky and Pluess (2009a) recently made in this journal. The diathesis-stress framework presumes that some individuals are more vulnerable to the *adverse* effects of *negative* experiences and exposures than others due to some endogenous “vulnerability” characteristic (e.g., negative emotionality, “risk gene”). Even if numerous empirical findings in many areas of inquiry prove consistent with diathesis-stress thinking (e.g., Belsky, Hsieh, & Crnic, 1998; Caspi et al., 2003; Cummings, El-Sheikh, Kouros, & Keller, 2007), it must be acknowledged that this widely embraced framework has nothing to say about variation in response to putatively positive experiences. In fact, diathesis-stress reasoning suggests—at least implicitly—that there should be no differences between vulnerable and resilient individuals in the absence of adversity.

Recently, an alternative model of environmental action has been advanced—*differential susceptibility*—which is not restricted to negative effects of contextual adversity (Belsky, 1997, 2005; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009a). It shares with an independently developed and more mechanistically focused complementary model, *biological sensitivity to context* (Boyce et al., 1995; Boyce & Ellis, 2005), the view that some individuals are disproportionately susceptible to both positive *and* negative developmental experiences and environmental exposures (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011). Whereas these frameworks

have been recently referred to as *neurobiological susceptibility* (Ellis et al., 2011), for the sake of consistency with our previous work (Belsky et al., 2007; Belsky & Pluess, 2009a, 2009b), and given its now widespread adoption, we employ the terminology of differential susceptibility throughout this article.

Whatever terminology is employed, the theoretical framework in question regards more susceptible individuals as not just especially “vulnerable” to adversity, but more generally “developmentally plastic” or “malleable” (Belsky & Pluess, 2009b; Boyce & Ellis, 2005; Ellis et al., 2011). Thus, many of those whom the diathesis-stress framework considers disproportionately likely to be adversely affected by negative experiences and exposures may *also* be disproportionately likely to *benefit* from *supportive and enriching* ones. In other words, differential-susceptibility thinking encompasses both the “dark side” of environmental susceptibility, which refers to response to negative experiences, and what Bakermans-Kranenburg and van IJzendoorn (2011) have labeled the “bright side” or response to positive experiences and exposures (see also Homberg & Lesch, 2011).

Importantly, the differential-susceptibility framework—like that of biological sensitivity to context—is based on evolutionary reasoning rather than on clinical or other insights regarding origins of psychopathology. One of the most basic assumptions of many behavioral scientists is that humans are developmentally plastic, shaped in a myriad of ways by their developmental experiences. This is so whether one is an extreme environmentalist or a behavior geneticist appreciating the importance of nonshared environmental influences (Plomin & Daniels, 1987). What evolutionary thinking brings to this perspective is the view that such developmental responsiveness evolved in the service of reproductive fitness, the successful dispersion of genes in future generations (Belsky, Steinberg, & Draper, 1991). Implicit if not explicit in both evolutionary and nonevolutionary approaches to developmental plasticity is the assumption that response to early environmental influence serves to fit the organism to the environment in which it would seem most likely to find itself as it develops. Whereas evolutionists emphasize fit in terms of survival and reproduction, others conceptualize such developmental programming in mental- or physical-health terms.

What evolutionary-minded thinkers, like financial investors, are highly appreciative of, however, is that the future is inherently uncertain. As such, future environmental conditions for which developmental plasticity might have prepared an individual could end up being rather different than anticipated. Hence, and as stipulated by differential-susceptibility reasoning, natural selection should have shaped individuals to differ in their degree of susceptibility to environmental conditions. That way, the negative consequences of a discrepancy or mismatch between anticipated and eventual environment would affect predominately those individuals who are more susceptible to environmental influences (i.e., those with a higher degree of developmental plasticity), but not those generally less susceptible. This could be particularly beneficial if such interindividual variation in developmental plasticity occurred within families, as siblings varying in susceptibility would essentially provide “insurance” for each other—and their parents vis-à-vis their inclusive fitness prospects (Belsky, 2005).

However logical such an analysis appears, we should make clear, as Ellis and associates (Ellis et al., 2011) recently have, that there remains uncertainty about the evolutionary dynamics of

differential susceptibility. Alternative models have been advanced to explain how variation in plasticity might evolve, some highlighting the bet hedging just described, some conditional versus alternative strategies, and some frequency dependent selection. The issues involved, however, are beyond the scope of this review.

Evolutionary reasoning should also be considered regarding the understanding of what “positive” means. The distinction between negative and positive outcomes and environments, though shared by many within a society and in some cases also across different societies, may not be particularly relevant from an evolutionary perspective: “The idea that any form of phenotypic variation in and of itself is necessarily positive or negative is an anathema to biology” (Cameron et al., 2005, p. 846). What many conceptualize as manifestations of “nonoptimal” development (e.g., insecure attachment, aggression, risk-taking, early sexual debut), then, evolutionary-minded thinkers regard as potential alternative tactics for dispersing genes across generations and thereby enhancing reproductive fitness under the ecological conditions that give rise to them. In view of the fact that environments can and do change over time, however, what was once a fitness-enhancing characteristic at one point in time may undermine fitness at another point in time.

In light of the claim that, from an evolutionary perspective, we should expect individual differences in developmental plasticity, we make the case here that substantial variation in response to positive experiences should also be the norm. We derive this theoretical conclusion from the differential-susceptibility claim that individuals vary generally in the extent to which they are affected by both (commonly regarded) negative *and* positive environmental experiences and exposures. Before reviewing empirical evidence substantiating the proposition that some individuals are disproportionately susceptible to the benefits of positive environmental influences, we propose new terminology for such variability in response to positive experiences, making clear that while related to differential susceptibility, it is not the same as it.

From Differential Susceptibility to Vantage Sensitivity

Since our 2009 review, ever more evidence consistent with the differential-susceptibility hypothesis has emerged (e.g., Clasen, Wells, Knopik, McGeary, & Beevers, 2011; Dick et al., 2011; Poehlmann et al., 2011; Wetter & El-Sheikh, 2012), so much so, in fact, that a special section of the journal *Development and Psychopathology* was devoted recently to this topic, edited by Ellis and Boyce (2011), authors of the *Biological Sensitivity to Context* framework (Boyce & Ellis, 2005), which is closely related to differential susceptibility (Belsky, 1997, 2005; Belsky & Pluess, 2009a), as mentioned earlier. Intriguingly, some of the most recent work informed by differential-susceptibility thinking involves experiments designed to evaluate the beneficial effects of interventions intended not just to address the “dark side” by ameliorating problematic functioning (e.g., Eley et al., 2012) but also the “bright side” by promoting explicitly positive functioning (e.g., Cassidy, Woodhouse, Sherman, Stupica, & Lejuez, 2011). Because such work does not address the diathesis-stress concepts of *risk* and *resilience*, and because it is difficult to identify the linguistic converse of “vulnerability” (Belsky & Pluess, 2009a)—that is, a word that captures the notion that some are more likely than others to benefit from environmental support and enrichment—it is clear

that new terminology is called for. Especially important to note in this regard is that the terms *protection* and *buffering* commonly used in the vulnerability-resilience literature to denote the positive consequence of *not* succumbing to an adverse experience do not fit the “bright side” of the differential-susceptibility framework. As it turned out, the only term that Belsky and Pluess (2009b) could identify—in tongue-and-cheek fashion—to characterize those disproportionately likely to benefit from positive experiences and exposures was “lucky.” And this was after asking speakers of diverse languages, including French, German, Italian, Chinese, Czech, Spanish, Korean, and Polish, whether there was a word or term in their native tongue that captured the “bright side” of differential susceptibility. The fact that there did not seem to be such a term in any of these languages raised the intriguing possibility that one reason variability in response to positive—as opposed to negative—experiences went unnoticed, or at least unheralded, for so long was that we simply lack terminology to direct attention to it.

Recently, Manuck and associates (Manuck, 2011; Sweitzer et al., 2012) introduced the term *Vantage Sensitivity* to characterize the “bright side” of differential susceptibility and more generally variability in response to positive experiences. *Vantage* is short for *advantage*, but in addition to implying benefit, gain or profit, it is also defined as “a position, condition, or opportunity that is likely to provide superiority or an advantage” (Houghton Mifflin, 2000). In Manuck’s own words (S. B. Manuck, personal communication, January 18, 2011), *vantage* “bespeaks a position conferring advantage, benefit or gain, without bearing the singularity of a particular advantage.” We embrace and promote the term *vantage sensitivity* to describe the notion that some individuals are more sensitive and positively responsive to the environmental *advantages* to which they are exposed. These advantages may take the form of security of attachment derived from sensitive parenting, academic achievement resulting from high-quality child care, prosocial behavior due to supportive friendship networks, and life satisfaction stemming from positive life events, as well as sense of efficacy following psychotherapy, to name just a few possibilities.

We propose the following concepts to characterize variability in response to positive experiences: (a) *vantage sensitivity* reflects the general proclivity of an individual to benefit from positive and presumptively well-being- and competence-promoting features of the environment, just as *vulnerability* depicts the tendency to succumb to negative effects of adversity in the diathesis-stress framework; (b) the degree of *vantage sensitivity* is a function of the presence of *vantage-sensitivity factors* (i.e., promotive factors), just as *vulnerability/risk factors* increase vulnerability to negative effects of adversity in the diathesis-stress framework; (c) *vantage resistance* describes the failure to benefit from positive influences, just as *resilience* characterizes resistance to negative effects of adversity in the diathesis-stress framework; and (d) the degree of *vantage resistance* is a function of the presence of *vantage-resistance factors* or absence of *vantage-sensitivity* ones, just as *protective factors* increase resilience to negative effects of adversity in the diathesis-stress framework. In summary, *vantage-sensitivity factors* increase *vantage sensitivity* to the *beneficial effects of positive experiences and exposures*, whereas *vantage-resistance factors* diminish or even completely eliminate *positive response* to the same supportive conditions. (see Figure 1 for graphical illustration.)

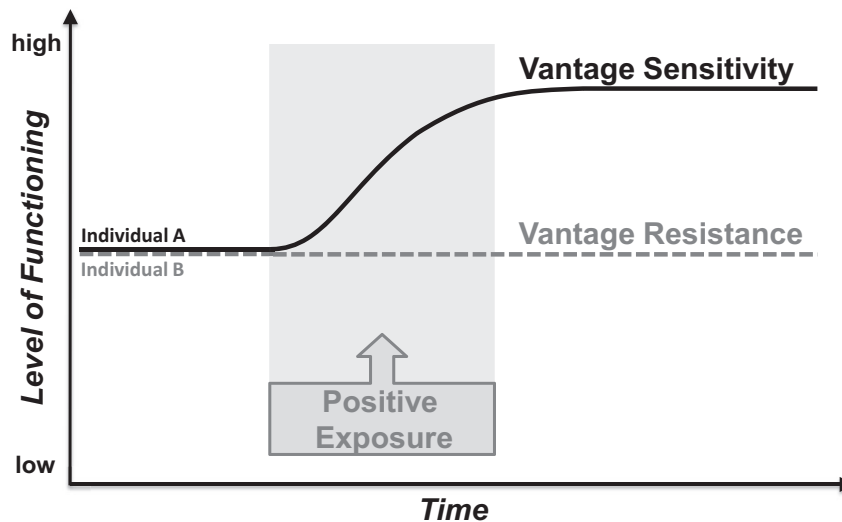


Figure 1. Graphical illustration of vantage sensitivity; in response to a positive exposure, the level of functioning increases in Individual A, reflecting vantage sensitivity, whereas it remains unchanged in Individual B, reflecting vantage resistance.

Though derived from and therefore closely related to differential susceptibility, vantage sensitivity represents more than just the “bright side” of that concept. Because the reasoning behind this claim may not be entirely clear, we next clarify conceptual differences between vantage sensitivity, differential susceptibility and also the resilience component of the diathesis-stress framework before reviewing empirical evidence consistent with vantage sensitivity.

Vantage Sensitivity Versus Differential Susceptibility

There are several reasons not to automatically equate vantage sensitivity with differential susceptibility. First, it is conceivable that, whereas some individuals might be more sensitive to the benefits of a supportive or enriching environment as a function of vantage-sensitivity factors, the same individual attributes may not make them more susceptible to the negative effects of contextual adversity. Thus, some individual differences in developmental response to environmental experiences and exposures may emerge exclusively under supportive conditions. To the extent that this is so, one would speak of vantage sensitivity, not differential susceptibility—which implies disproportionate sensitivity to both positive and negative experiences and exposures.

A further distinction between the two concepts being discussed emerges when we consider that individuals may be both *highly responsive to* environmental support (i.e., showing increased vantage sensitivity) and *unresponsive to and protected from* adversity (i.e., showing increased resilience), and this could be due to the very same endogenous characteristic. Consider, for example, a highly intelligent child who might profit disproportionately from high-quality education. There is no theoretical or empirical reason to presume that such a student would also be more adversely affected by low-quality schooling. On the contrary, children with high IQs tend to be more

resilient in the face of adversity (e.g., Masten et al., 1999). Thus, the same individual characteristic—in this case high IQ—may serve both a protective function in adverse environments (Rutter, 1987) and a promotive/vantage-sensitivity function in supportive environments (Sameroff, 2000).

Another distinction to be made between vantage sensitivity and differential susceptibility pertains to the empirical conditions required to evaluate each. Whereas demonstration of differential susceptibility is based ideally on investigation of contextual conditions that range from the negative to the positive (Belsky et al., 2007; Belsky & Pluess, 2009a), this is not so for vantage sensitivity. In fact, many truly positive exposures do not range from the positive to the negative but only from the positive to the absence of the positive (e.g., psychological intervention versus no intervention). Consequently, it cannot be inferred from a vantage-sensitivity finding chronicling an individual’s disproportionate response to, say, therapy, that the same person would be equally responsive—and more responsive than others—to adversity. The latter would be required to draw a conclusion of differential susceptibility. Finally, the same “range of measurement” issue applies to the outcome of interest. Whereas it should cover both the adaptive and the maladaptive spectrum in order to reflect true differential susceptibility, this is irrelevant to the evaluation of vantage sensitivity. This is because vantage sensitivity is exclusively about the *positive benefit* derived from an enriching or supportive experience, whether this is reflected in the reduction of problems or dysfunction (e.g., depression, antisocial behavior) or the enhancement of competence/well-being (e.g., prosocial behavior, academic achievement). Ultimately, the point to be made is that, whereas some individuals may be disproportionately likely to be affected positively and negatively by, respectively, positive and negative contextual conditions, others may only be susceptible to the former—and thus manifest vantage sensitivity rather than differential susceptibility.

Vantage Sensitivity Versus Resilience

Resilience, reflecting the absence of problematic functioning despite exposure to contextual adversity, is a concept central to many subfields of psychology ranging from clinical to developmental to positive psychology, to name just a few (e.g., Cicchetti & Garnezy, 1993; Masten & Obradović, 2006; Werner, 1997). Though not completely unrelated to vantage sensitivity, it is important to highlight fundamental differences between the two concepts. This is especially important given inconsistent use of the terms “resilience” and “protection” in different psychological literatures. Specifically, some have mistakenly referred to vantage-sensitivity-like findings as evidence of “protection” (for review, see Luthar, Cicchetti, & Becker, 2000), most probably due to the lack of a conceptual framework for thinking about variability in response to positive contextual conditions. While resilience is what “protective” factors and processes engender—by *preventing* an individual from succumbing to or being harmed by some *contextual adversity* (Rutter, 1987), vantage sensitivity refers to “promotive” influences (Sameroff, 2000) and is about an individual *benefiting*—more than others—from a *positive environmental experience or exposure*. Ultimately, then, vantage sensitivity is about variation in the promotion of well-being or competent functioning when exposed to an experience presumed to have a beneficial effect, whereas protection is about not having one’s well-being or competence undermined when subject to a negative experience.

To summarize, whereas the concept of resilience reflects protective processes within a diathesis-stress framework and refers to individual differences in response to adversity, it makes no claims about the potentially promotive function of particular protective factors in response to positive experiences. And whereas the concept of vantage sensitivity pertains to individual differences in response to positive contextual conditions as a function of promotive factors, it has nothing to say regarding the potentially protective function of the same factors in the face of adversity. Differential susceptibility, on the other hand, is based on the view that the same factors that increase vulnerability to adversity will also increase vantage sensitivity in positive environments and that factors that make some resilient to adversity will also make them less responsive to positive experiences.

Recent Evidence of Vantage Sensitivity

In our extensive analysis of evidence chronicling differential susceptibility to both negative and positive environmental influences, we identified three different categories of endogenous susceptibility factors (Belsky & Pluess, 2009a; see also Obradović & Boyce, 2009): (a) behavioral factors (e.g., negative emotionality; Pluess & Belsky, 2010), (b) physiological factors (e.g., cortisol stress reactivity; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010), and (c) genetic factors, perhaps most notably polymorphisms in the serotonin transporter (e.g., Taylor et al., 2006) and the dopamine receptor D4 genes (e.g., Bakermans-Kranenburg & van IJzendoorn, 2006).

In what follows, we review a selection of mostly very recent studies that provide substantive empirical evidence for individual differences in *vantage sensitivity* as a function of behavioral, physiological, and genetic factors. Rather than attempting to exhaustively delineate and summarize vantage-sensitivity findings,

we present here carefully selected evidence, for illustrative purposes, which derives from high-quality research that has appeared, with few exceptions, since the original Belsky and Pluess (2009a) review.

It is important to mention that the majority of the work selected for consideration is not positioned to test whether individuals that are more responsive to positive exposures are also more responsive to negative ones. This would be required, however, in order to distinguish vantage sensitivity from differential susceptibility, with the former predicting disproportionate responsiveness to only positive conditions and the latter predicting disproportionate responsiveness to both positive and negative conditions. Investigation of individual differences in response to both adverse and supportive conditions would be necessary in order to determine whether some people are more responsive to both positive and negative conditions (i.e., differentially susceptible), some to just supportive conditions (i.e., vantage sensitivity) and some to just adversity (i.e., diathesis stress).

Behavioral Vantage-Sensitivity Factors

Infant temperament and the personality trait of high sensory-processing sensitivity, both of which emerged as behavioral susceptibility factors in our previous review (Belsky & Pluess, 2009a), also appear to function as vantage-sensitivity markers.

Infant temperament. Developmental psychology has a long history of investigating the interaction between child temperament and early experiences (Rothbart & Bates, 2006). Most such research has been informed, implicitly if not explicitly, by the diathesis-stress perspective (Belsky & Pluess, 2009a), thus addressing how infant negative emotionality or difficult temperament predisposes infants to be negatively affected by adverse rearing conditions (e.g., poverty, harsh parenting). A growing body of evidence, informed by differential-susceptibility thinking, however, provides evidence that these putatively “vulnerable” children also show a higher degree of vantage sensitivity, benefitting disproportionately from positive environments.

Longitudinal data collected as part of the National Institute of Child Health and Development (NICHD) Study of Early Child Care (NICHD Early Child Care Research Network, 2005) revealed that when children with more difficult and negatively emotional temperaments in their first 6 months of life experienced high-quality nonmaternal child care and more sensitive maternal care they scored higher on, respectively, social competence at 4.5 years (Pluess & Belsky, 2009) and academic competence and social skills at age 6 years (Stright, Cranley Gallagher, & Kelley, 2008). Similar results emerged when the predictor was maternal sensitivity across the first 4.5 years of life and the outcome was the growth of teacher-rated social skills from 4.5 years until 11 years (Pluess & Belsky, 2010). Indeed, when Roisman and associates (2012) subjected the latter findings to especially stringent evidentiary criteria, they found that children rated by their mothers as having difficult temperaments at 6 and 12 months had significantly more social and academic skills at 11 years of age when they experienced high-quality parenting in early life. Children with less difficult temperaments, on the other hand, did not benefit from positive parenting to the same degree.

The vantage sensitivity accruing to children with difficult or negatively emotional temperaments also emerged in a large British

cohort study, the Avon Longitudinal Study of Parents and Children (ALSPAC). Ramchandani, van IJzendoorn, and Bakermans-Kranenburg (2010) tested whether infant negative reactivity moderated effects of father involvement on prosocial behavior, finding that it did, but only for the almost 2,500 girls included in their analysis: Girls rated as highly reactive at 6 months manifested significantly more prosocial behavior at 6.5 years of age if fathers were highly involved in caring for the child during the early years. Nonreactive girls, in contrast, evinced no such benefit from father involvement.

Further evidence for the augmented vantage sensitivity of children high in negative emotionality comes from Kim and Kochanska's (2012) investigation of the influence of infant negative emotionality and mother-child relationship quality on the development of child self-regulation (i.e., effortful control) with a sample of 100 boys and girls. For children with low observed negative emotionality in infancy, there was no significant association between observer-rated mother-child relationship quality at 7 months and the child's ability at age 2 years to suppress a dominant response (e.g., unwrapping a gift) in favor of a subdominant response (e.g., waiting to unwrap a gift). Children who as infants were highly negatively emotional, however, manifested the most self-regulation in response to these challenging demands when mother-child relationship was of particular high quality, a finding consistent with vantage sensitivity.

Overcoming the causal-inference limits of correlational studies such as those just cited, Cassidy et al. (2011) sought to determine whether newborn irritability, observationally assessed within the first month of life, would moderate the effects of a brief parenting intervention on attachment security, measured using the Strange Situation at 12 months of age, in a randomized controlled study involving 169 families. Only in the case of highly irritable infants did the intervention succeed in promoting attachment security. In subsequent work focused only on the control group ($n = 84$), Stupica, Sherman, and Cassidy (2011) extended their investigation of vantage sensitivity, evaluating whether infant irritability moderated the effect of infant attachment security, a marker for the quality of the early environment, on sociability measured at 18 and 24 months. Children highly irritable as newborns who had established secure attachments to their primary caregiver proved to be the most sociable of all children in this inquiry.

High sensitive personality. Vantage sensitivity as a function of behavioral and psychological characteristics is not restricted to temperamental traits very early in life. One psychological attribute measured in adulthood that has emerged recently as a moderator of environmental influences is high sensory-processing sensitivity, a personality trait measured with the Highly Sensitive Person Scale (Aron & Aron, 1997). According to Aron, Aron, and Jagiellowicz (2012) about 20% of the population is characterized by a high-sensitive personality encompassing a highly sensitive nervous system, increased awareness of subtleties in surroundings, as well as the deep processing of them and a tendency to be more easily overwhelmed when in a highly stimulating environment.

Building on this work while testing the a priori hypothesis that children characterized by high sensitivity would be more responsive to psychological intervention, Pluess and Boniwell (2012) investigated variation in the anticipated positive effects of a school-based resilience-promoting program (Pluess, Boniwell, Hefferon, & Tunariu, 2012) administered to a sample of 166

11-year-old girls in one of the most deprived areas in London, United Kingdom. The intervention led to a significant decrease of depression symptoms observable up to the 12-month follow-up assessment, but, consistent with vantage sensitivity, exclusively among children who scored in the upper tercile of the highly sensitive-child questionnaire (Pluess et al., 2012). All other children failed to benefit from the intervention, at least regarding changes in depression symptoms.

The only published randomized experiment of high sensitive personality of which we are aware included 160 male and female undergraduates (Study 4: Aron, Aron, & Davies, 2005). All participants completed a very brief version of the Highly Sensitive Person scale (Aron & Aron, 1997), and participants were then asked to solve problems adapted from intelligence tests. For half of the sample, the problems were relatively easy, and for the other half, the problems were very difficult. After completion, participants rated their state negative affect. For those participants scoring low on high sensitivity, experimental condition had no effect on negative affect. Participants who were highly sensitive, on the other hand, reported not only more negative affect when the test was difficult but also the least negative affect when the test was easy. In other words, high-sensitive individuals were more sensitive to the emotional reward of successfully completing the test, thereby providing evidence for vantage sensitivity as a function of behavioral characteristics in adulthood. Admittedly, vantage-sensitivity would have been more compellingly documented had the outcome measured been positive affect and the findings the same.

Physiological Vantage-Sensitivity Factors

The notion that high physiological reactivity would render individuals highly susceptible to both positive and negative experiences is central to Boyce and Ellis's (2005) *Biological Sensitivity to Context* hypothesis. Physiological reactivity in the context of the stress response is controlled by both the autonomous nervous system (ANS), which is further divided into the sympathetic (SNS) and the parasympathetic nervous system (PNS), and the neuroendocrine system. The SNS controls activities that are mobilizing during stress and anxiety (e.g., accelerated heart rate, increased blood pressure, sweating, etc.), whereas the PNS controls physiologically opposing activities serving relaxation of the body and restoration of energy stores (e.g., decreases in heart rate, blood pressure, sleep, etc.). The neuroendocrine response to stress is primarily controlled by the hypothalamus-pituitary-adrenal axis (HPA). Corticotropin releasing hormone (CRH)—which is released from the hypothalamus in response to stress—activates the secretion of adrenocorticotropic hormone (ACTH) from the pituitary gland, which then causes the adrenal cortex to release cortisol into the bloodstream. Finally, cortisol stimulates numerous and diverse physiological and metabolic changes that prepare the organism for optimal functioning under stressful conditions (e.g., increase of blood pressure and blood sugar, breakdown of lipids and proteins, reduction of immune responses).

Empirical evidence examining the moderating role of physiological reactivity focused on indisputably positive experiences is relatively rare. However, there are a small number of studies indicating increased vantage sensitivity in children with high cor-

tisol or high Respiratory Sinus Arrhythmia (RSA; a cardiac measure for the activity of the PNS) activity and reactivity.

In a preliminary analysis of a longitudinal evaluation of an intervention provided to 22 10-year-old boys with disruptive behavior disorder, van de Wiel, van Goozen, Matthys, Snoeck, and van Engeland (2004) investigated whether cortisol stress reactivity would influence symptom severity 9 months later. Following treatment, boys who scored high on cortisol stress reactivity *before* treatment had significantly lower parent-rated aggression and oppositional behavior scores than boys whose cortisol stress reactivity was low, thereby providing the first experimental evidence that high cortisol reactivity is a marker of increased vantage sensitivity.

Similar results emerged in a recent observational study by Obradović et al. (2010) investigating effects of family adversity on prosocial behavior in 338 5- and 6-year-olds. For children with low cortisol reactivity, family adversity proved unrelated to prosocial behavior (based on child, parent, and teacher ratings). Children with high cortisol reactivity, however, were more prosocial when family adversity was especially low, suggesting greater vantage sensitivity, that is, enhanced capacity to benefit from a more supportive environment. In the same study, Obradović et al. (2010) also evaluated the moderating effect of RSA. Children with high RSA reactivity showed the highest school engagement (based on child, parent, and teacher ratings) of all children not exposed to high family adversity—and who thus experienced a more supportive rearing milieu—whereas this was not the case for children with low RSA reactivity.

The finding that RSA measures index vantage sensitivity also emerged in Eisenberg et al.'s (2012) recent work of 213 toddlers and their families. Baseline RSA moderated effects of the home environment (based on a composite of maternal and paternal education, family income, and marital quality) in early childhood on repeatedly assessed child aggression. For children with high RSA activity, high environmental quality was associated with less aggression at 54 months, whereas children characterized by low RSA activity—displaying vantage resistance—did not benefit from high quality environments at all.

Genetic Vantage-Sensitivity Factors

Two polymorphisms identified as potential “plasticity genes” in the Belsky and Pluess (2009a) analysis of differential susceptibility have consistently emerged in more recent work as markers of vantage sensitivity, DRD4, and 5-HTTLPR.

The dopamine receptor D4 gene. The dopaminergic system plays an important role in attentional, motivational, and reward processes and a polymorphism of the dopamine receptor D4 (*DRD4*) gene has been much studied in gene \times environment ($G\times E$) interaction research. 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 regarded as a vulnerability factor due to its links to attention-deficit/hyperactivity disorder (Faraone, Doyle, Mick, & Biederman, 2001), high novelty seeking behavior (Kluger, Siegfried, & Ebstein, 2002), and low dopamine reception efficiency (Robbins & Everitt, 1999), among other correlates. Findings of a recent meta-analysis of $G\times E$ studies on children up to age 10 years involving *DRD4* and other dopamine-related genes indicate that those carrying less efficient dopamine-related genes are more vulnerable to negative environments but,

supporting a differential susceptibility model, also show higher vantage sensitivity in response to positive environments (Bakermans-Kranenburg & van IJzendoorn, 2011). Indeed, in this work the vantage-sensitivity or “bright side” of the differential-susceptibility framework vis-à-vis dopamine-related genes proved stronger than the diathesis-stress or “dark side.” In other words, the apparent benefits of carrying putative “risk alleles” in the face of environmental support or enrichment was greater than the apparent costs under conditions of contextual adversity. Had what we are calling vantage sensitivity, after Manuck (2011; Sweitzer et al., 2012), not been considered, this finding surely would have been missed.

In their pioneering experimental study evaluating genetic moderation of a psychological intervention, Bakermans-Kranenburg, van IJzendoorn, Pijlman, Mesman, and Juffer (2008) investigated whether *DRD4* interacted with a video-feedback parenting intervention in reducing externalizing behavior in a sample of 157 families with 1-year-old to 3-year-old children randomly assigned to treatment condition. Providing evidence for vantage sensitivity, the intervention proved effective in decreasing externalizing behavior—but only for children carrying the *DRD4* 7-repeat allele. Children without the *DRD4* 7-repeat did not benefit from the intervention at all. Follow-up analyses revealed that the only children in the experimental group who actually benefited from the intervention were those carrying the *DRD4* 7-repeat whose mothers' parenting behavior improved as a result of it. The presence of the *DRD4* 7-repeat coupled with no change in mothers' positive discipline did not result in a decrease of externalizing behavior, thereby suggesting that it was the mother's increase in positive discipline as a result of the intervention that children with the *DRD4* 7-repeat were more sensitive to. This observation begs the question, what accounted for the vantage sensitivity of some mothers relative to others, an issue not addressed in this inquiry; that is, why did the positive discipline of only some mothers in the intervention group increase? Consideration of this question raises the prospect that parenting interventions to enhance child functioning may prove most effective when both members of the parent-child dyad—or all members of the family system—have genetic, physiological, temperamental, or other bases that make them highly susceptible to environmental support and enrichment.

Focusing on an indisputably positive outcome rather than reduction of a negative one, Kegel, Bus, and van IJzendoorn (2011) investigated genetic sensitivity as a function of the *DRD4* 7-repeat vis-à-vis a computer based literacy instruction program ($N = 182$ 4-year-old to 5-year-old boys and girls). Two intervention groups, one with positive feedback and one without, were compared to a control group on the development of emergent literacy skills. Only children carrying the *DRD4* 7-repeat increased their early literacy skills in response to the intervention. Notably, the positive effect of the intervention in children with the *DRD4* 7-repeat was restricted to the group that received positive feedback as part of the computer program. In the absence of positive feedback, there was no difference in literacy skills between children in intervention or control group, thereby suggesting that the presence of the *DRD4* 7-repeat allele predicted vantage sensitivity to the positive feedback component of the intervention.

In a cross-sectional analysis of a longitudinal prospective study, Knafo, Israel, and Ebstein (2011) investigated whether *DRD4* moderated the effects of mother reported positivity in parenting on

prosocial behavior in early childhood in 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 vantage sensitivity emerged, as more positive parenting by the mothers proved related to more prosocial behavior by their children.

Similar findings emerged when Belsky and Pluess (in press) evaluated whether DRD4 moderated the effects of early nonmaternal child care quality in the first 4.5 years on repeatedly assessed social skills across middle childhood using a subsample of 508 Caucasian boys and girls from the aforementioned NICHD Study of Early Child Care. Although better quality child care—reflective of more attentive, positively affectionate, and stimulating care giving—proved unrelated to yearly teacher ratings of social skills from 4.5–11 years for children without the DRD4 7-repeat, in the case of children carrying this allele, the anticipated positive association emerged between predictor and outcomes at kindergarten and first grade.

The serotonin transporter gene. A large proportion of G×E studies is based on genetic variants in the serotonergic system, most prominently the serotonin-transporter-linked polymorphic region (5-HTTLPR), which is a degenerate repeat polymorphic region in SLC6A4, the gene that codes for the serotonin transporter. Most research focuses on two variants—those carrying at least one short allele (s/s, s/l) and those homozygous for the long allele (l/l)—though more variants than these have been identified (Nakamura, Ueno, Sano, & Tanabe, 2000). The short allele has generally been associated with reduced expression of the serotonin transporter molecule—which is involved in the reuptake of serotonin from the synaptic cleft—and thus considered to be related to depression, either directly (Munafò et al., 2009; Sen, Burmeister, & Ghosh, 2004) or in the face of adversity (Karg, Burmeister, Shedden, & Sen, 2011; Risch et al., 2009). However, in a substantial proportion of these studies, results are actually more indicative of differential susceptibility than diathesis-stress, with 5-HTTLPR short allele carriers having the worst outcomes under adverse conditions and the best outcomes under supportive—or at least benign—conditions (Belsky et al., 2009; Belsky & Pluess, 2009a). Here, we summarize recent studies that investigated the moderating effect of 5-HTTLPR specifically in response to positive experiential factors. Before doing so, however, we call attention to a recent meta-analysis of research on 2,276 Caucasian children under the age of 18 years, which shows that those with one or two short alleles benefited more from positive environmental exposures than children without them (van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012).

Kochanska, Kim, Barry, and Philibert (2011) investigated whether 5-HTTLPR moderated the effect of mothers' repeatedly observed responsiveness across the first 4 years on children's moral internalization at 67 months. Moral internalization for children carrying the 5-HTTLPR short allele was significantly higher when mothers were more responsive, whereas those homozygous for the long allele appeared resistant to the same beneficial effects of high maternal responsiveness.

Similar findings emerged in Hankin and associates' (Hankin et al., 2011) recent research on the interaction of 5-HTTLPR and positive parenting in predicting positive emotionality in middle childhood/adolescence using three independent samples totaling

1,874 9-year-old to 15-year-old boys and girls. Results from two of the three samples were supportive of vantage sensitivity, with children carrying the 5-HTTLPR short allele showing the highest positive affect scores when positive parenting was high, suggesting that these children were particularly sensitive to the benefits of high positive parenting.

Vantage sensitivity as a function of the 5-HTTLPR short allele is not restricted to positive experiences within the parenting domain, as revealed by Eley et al.'s (2012) evaluation of whether 5-HTTLPR moderated effects of cognitive behavioral therapy for anxiety disorders. Clinical diagnoses of anxiety disorders and symptom severity were assessed before and after treatment, as well as 6 months after treatment ended. Although all children appeared to benefit from the treatment, the positive effect of the intervention was particularly pronounced in the case of those children carrying the short allele in this work with 6-year-old to 13-year-old boys and girls ($N = 359$). More specifically, those homozygous for the 5-HTTLPR short allele showed a significantly greater reduction in symptom severity from pretreatment to follow-up assessment, so much so, in fact, that they proved 20% more likely than others to be free of anxiety disorder at the 6-month follow-up assessment.

Through this point, all work considered involving 5-HTTLPR relied on observational data that do not afford strong causal inference, making findings from a recent randomized controlled study especially noteworthy. Using data from the Bucharest Early Intervention Project (BEIP), Drury et al. (2012) sought to determine whether 5-HTTLPR would moderate the effect of early rearing on indiscriminate social behavior when children were 54 months old. Indiscriminate social behavior is often regarded as a "signature consequence" of deprived, institutional care. In the BEIP, 136 abandoned children between 6 and 30 months of age were randomly assigned to standard institutional care or a newly developed high-quality foster care program (Zeanah et al., 2003). Children homozygous for the 5-HTTLPR short allele randomly allocated to the high-quality foster care condition had the lowest indiscriminate social behavior scores of the whole sample at 54 months, whereas for children with the 5-HTTLPR long allele there was no beneficial effect of high-quality foster care. These data are the first experimental evidence highlighting the vantage-sensitivity character of 5-HTTLPR.

Vantage sensitivity associated with the 5-HTTLPR short allele is not restricted to experiences in childhood. Pluess, Belsky, Way, and Taylor (2010) tested the hypothesis that 5-HTTLPR would moderate effects of recent life events on neuroticism in a cross-sectional study involving 118 healthy young men and women. Individuals homozygous for the 5-HTTLPR short allele scored lower on neuroticism than all other genotypes if they experienced positive life events, suggesting that the short allele of the 5-HTTLPR increases vantage sensitivity to recent positive experiences in adulthood, at least with regard to self-reported neuroticism. Similar results emerged recently in a study involving 367 young adults (Kuepper et al., 2012) with women homozygous for the short allele scoring significantly lower in neuroticism than women homozygous for the long allele when having a history of predominately positive life events. In the same study both men and women homozygous for the short allele also scored significantly higher on a measure of life satisfaction than those homozygous for the long allele if they experienced a preponderance of positive life events.

Evidence of increased vantage sensitivity in adulthood as a function of the 5-HTTLPR also emerged in recent work by Schoebi, Way, Karney, and Bradbury (2012), whose focus of inquiry was sensitivity to affective interpersonal cues among 76 married couples. Husbands and wives each reported positive and negative affect before and after a lab-based discussion of a marital disagreement. Although there was no significant effect of preinteraction *partner* positive affect (i.e., the environmental exposure) on postinteraction positive affect (i.e., the outcome) for individuals homozygous for the 5-HTTLPR long allele, findings were different in the case of individuals carrying the 5-HTTLPR short allele. For them, the greater the partner's preinteraction positive affect, the greater the increase in their own positive affect from before to after the interaction, supporting the view that short alleles can be regarded as markers of heightened vantage sensitivity.

Discussion

The claim inherent in the concept of vantage sensitivity—as proposed herein—is that individuals differ fundamentally in their responsivity to positive experiences and exposures, a theoretical proposition derived from differential susceptibility reasoning. In this follow up to our rather recent report making the case for differential susceptibility (Belsky & Pluess, 2009a), we focus on very recent and diverse, cross-sectional, longitudinal, and experimental studies chronicling vantage sensitivity. Some but not all of this work has been directly stimulated by our earlier writings.

Consistent with the predictions of vantage sensitivity, some individuals proved especially likely to benefit from positive experiences, whereas others failed to do so completely or at least did not benefit to the same extent. In most cases the evidence was based on research that measured effects of positive environmental experiences on positive functioning (e.g., prosocial behavior), though in some studies benefit was demonstrated as reduction of problematic functioning (e.g., aggression).

The individual differences in vantage sensitivity highlighted in the research reviewed are associated with similar or in many cases the same behavioral, physiological, and genetic characteristics that have often been conceptualized as “risk” or “vulnerability” factors in the psychological and psychiatric literatures (Rutter, 1987). The empirical observation that many of these putative “risk factors” are also associated with increased vantage sensitivity to positive effects of supportive environments lends further weight to the claim that in many cases these characteristics should be reconceptualized as “plasticity markers” (Belsky et al., 2009; Belsky & Pluess, 2009a). However, it is important to differentiate between those plasticity markers that confer both risk and vantage sensitivity—that is, differential susceptibility—from those that confer only one or the other. In this section, we discuss this issue briefly, then turn attention to determinants of vantage sensitivity and mechanisms that could account for the phenomena under consideration before drawing some final conclusions regarding implications of this analysis of vantage sensitivity.

Risk, Vantage-Sensitivity, and Differential-Susceptibility Factors

Although the same factors often seem to moderate effects of environmental influences whether they are exclusively negative

(i.e., diathesis-stress), exclusively positive (i.e., vantage sensitivity), or ranging from the negative to the positive (i.e., differential susceptibility), it is important to caution against inferring that every risk factor will also, by default, function as a vantage sensitivity or susceptibility factor. There may very well be specific factors that play a predominant role in diathesis-stress but not in vantage sensitivity and vice versa. Whether an individual characteristic represents a risk, vantage-sensitivity, or differential-susceptibility factor depends not only on the observed interaction pattern but also on the nature and range of the environmental and outcome constructs subject to investigation. Most important, whether variability in response to environmental experience and exposure as a function of a specific moderating factor proves more consistent with diathesis-stress, vantage sensitivity, or differential susceptibility can only be determined empirically using adequate statistical approaches (Roisman et al., 2012). The most suitable approaches for differentiating between these models of environmental interaction, at this point, include the “regions of significance” analytic approach (Preacher, Curran, & Bauer, 2006) and a recently developed confirmatory and hypothesis-driven method using reparameterization of regression models (Widaman et al., *in press*). Unfortunately, these new methods have been applied too rarely to be used in evaluating the evidence reviewed in the preceding section.

An important issue raised in our previous analysis of differential susceptibility is whether some individuals are generally more susceptible to all kinds of environmental influences or whether susceptibility is confined to specific susceptibility factors in specific domains, with people being more or less susceptible in different areas (Belsky & Pluess, 2009a). Similar questions have to be considered regarding vantage sensitivity. Thus, is it the case that an individual likely to benefit disproportionately from one positive environmental exposure with respect to a specific positive outcome is more or less likely to benefit from other positive environmental exposures with respect to other positive outcomes? Or is it the case that people vary in what they do or do not show vantage sensitivity to? Not unrelated, could some individuals be vantage-sensitivity generalists, evincing disproportionate positive response to a myriad of positive experiences and with regard to a diverse set of positive outcomes, whereas others are vantage-sensitivity specialists, proving especially responsive to only some positive contextual conditions and with regard to only some positive outcomes? However attractive the notion may be that individuals are more specialists than generalists vis-à-vis what they are likely to benefit from, we would be remiss if we did not call attention to findings from the two Dutch experiments reviewed, one dealing with the enhancement of maternal positive discipline and reductions in externalizing behavior (Bakermans-Kranenburg et al., 2008) and the other with computerized literacy instruction and the development of reading skill (Kegel et al., 2011). The fact that children carrying the DRD4-7R allele proved most responsive to both of these experiments highlights the very real possibility that some are simply more susceptible to a host of environmental “nutrients” than are others. Clearly, though, far more research is called for before such a broad-sweeping conclusion can be embraced with any confidence.

Determinants of Vantage Sensitivity

Given the obvious advantage of being more sensitive to the benefits of positive experiences, one important question is whether the capacity for vantage sensitivity represents an inherited disposition or whether it can be influenced and fostered through developmental experiences, including intervention. The G×E studies reviewed earlier would seem to suggest, at first glance, that individual differences in vantage sensitivity have a primarily genetic basis. This reading would seem to be further substantiated by evidence linking putative vantage-sensitivity genes with other putative vantage-sensitivity markers that are behavioral in character (e.g., infant difficult temperament and DRD4; Holmboe, Nemoda, Fearon, Sasvari-Szekely, & Johnson, 2011) or physiological in nature (e.g., cortisol reactivity and 5-HTTLPR; Gotlib, Joormann, Minor, & Hallmayer, 2008). It must be acknowledged, however, that such direct relations between genetic and behavioral or physiological attributes are only inconsistently chronicled in the literature (e.g., Alexander et al., 2009; Armbruster et al., 2009; Pluess et al., 2011).

Some evidence that environmental exposures can influence vantage sensitivity is found in research on the putatively adverse effects of maternal stress during pregnancy (for review, see Ruiz & Avant, 2005). After all, research on “fetal programming” indicates that prenatal adversity predicts both elevated infant negative emotionality and physiological reactivity (for review, see Pluess & Belsky, 2011), developmental phenotypes that our analysis of vantage-sensitivity evidence revealed to be potential markers of vantage sensitivity. Apparent programming of vantage sensitivity would also seem operative postnatally, given evidence, for example, of effects of early maternal sensitivity on infant difficult temperament (Kaplan, Evans, & Monk, 2008) and of a childhood family bereavement program on cortisol reactivity (Luecken et al., 2010). Considered together, these data make clear that the study of potential determinants of vantage sensitivity should not be restricted to genetic factors (Boyce & Ellis, 2005; Ellis et al., 2011). Moreover, the fact that recent evidence also indicates that some of the genetic polymorphisms already identified as plausible vantage-sensitivity factors (e.g., 5-HTTLPR) interact with the early environmental experience to predict behavioral vantage-sensitivity factors (e.g., infant negative emotionality; Pluess et al., 2011) raises the possibility that some individuals may be genetically predisposed to the environmental inducement of vantage sensitivity.

Mechanisms Accounting for Vantage Sensitivity

Although explicit efforts to identify mechanisms and processes of vantage sensitivity have not been undertaken yet, a range of studies focusing on behavioral and neurological correlates of some of the endogenous characteristics that emerged as vantage-sensitivity factors in our review indicate that there are likely multiple processes involved in vantage sensitivity. One may concern the degree of attention directed to qualitative aspects of experiences. For example, infants rated high on negative emotionality have been found to look longer at new stimuli, suggesting increased visual attention and cognitive engagement may play a role in accounting for vantage sensitivity (Vonderlin, Pahnke, & Pauen, 2008). Not inconsistent with this inference is evidence that healthy adults carrying the 5-HTTLPR short allele outperform others on the Wisconsin card sorting test, a task that requires,

among other things, good functioning in attention and visual processing (Borg et al., 2009). The notion that some individuals benefit more from positive experiences due to attentional processes is consistent with Suomi's (1995, 1997) claim that highly fearful, inhibited, “up-tight” rhesus macaques learn more than others about how to function effectively in their social environment because they spend more time than other young monkeys observing the world around them.

The attentional processes implicated in the above-cited work may themselves be a function of deeper central nervous functioning, as recently shown in an imaging study of 18 healthy adults (Jagiellowicz et al., 2011). Participants scoring high in self-reported *sensory-processing sensitivity* took longer to respond to minor changes in neutral photos and showed more activation in visual attentional brain areas, suggesting they attended more closely to the subtle details of the photos.

Other work suggests that some of the putative vantage-sensitivity factors highlighted in this report may be related to enhanced attention to emotionally relevant stimuli in particular. For example, healthy adults carrying 5-HTTLPR short alleles show attentional bias to both negative and positive emotional compared to neutral stimuli (Beevers, Wells, Ellis, & McGeary, 2009). Yet other research suggests that this attentional bias for emotionally relevant aspects related to the 5-HTTLPR short allele may actually be stronger for positive stimuli (Beevers et al., 2011); were this the case, it could help explain why individuals with this genotype benefit more than others from positive influences. The fact, however, that other studies report a stronger bias for negative stimuli only (for meta-analysis, see Pergamin-Hight, Bakermans-Kranenburg, van IJzendoorn, & Bar-Haim, 2012) or no emotional bias at all (Fox, Ridgewell, & Ashwin, 2009) certainly invites caution before any conclusions are drawn as to why short-allele carriers seem to evince greater vantage sensitivity than do others.

One potential explanation for the inconsistent findings just summarized may be that short-allele carriers are not so much inherently biased toward negative or positive stimuli but, rather, that their attentional bias is more easily influenced. Evidence consistent with this claim is found in a recent experimental study involving a standard *Attention Bias Modification* (ABM) procedure in which adults with 5-HTTLPR short alleles developed stronger biases for both negative and positive affective pictures than those with long alleles (Fox, Zougkou, Ridgewell, & Garner, 2011). Consequently, the authors concluded that individuals carrying the 5-HTTLPR short allele should gain most from therapeutic interventions such as ABM. This suggestion fits nicely with the findings of a recent study by Clarke, Chen, and Guastella (2012) in which the ability to adopt selective attentional processing was assessed with ABM before adult patients went through group CBT therapy for social anxiety disorder. Confirming and extending Fox et al.'s (2011) proposition, those most ready to adopt selective attentional processing (toward threatening stimuli) in the ABM experiment were also those who showed the most positive change in response to treatment.

Another potential mechanism involved in vantage sensitivity may be that individuals who benefit more from positive influences are especially sensitive to social forces. On the basis of empirical observations that individuals with the 5-HTTLPR short allele often prove more sensitive to social aspects of the environment, Way and Taylor (2010) recently made the case that activity within the

serotonin system might be critically involved in setting sensitivity to social experiences. This is certainly intriguing given the fact that most of the earlier-reviewed evidence of vantage sensitivity as a function of 5-HTTLPR include positive experiences and exposures predominately of a social nature (e.g., parenting, child care, psychotherapy).

Differences in reward sensitivity may be another mechanism underlying vantage sensitivity. In an experimental study by Roiser, Rogers, Cook, and Shahakian (2006) individuals with 5-HTTLPR short alleles attended to differences in the probability of winning gambles more than those with long alleles, suggesting that the former have greater reward sensitivity. Similar results emerged for adolescents with a history of childhood inhibited temperament in an imaging study (Bar-Haim et al., 2009). Teenagers who were more behaviorally inhibited in early childhood—and thus more negatively emotional—showed more activity in reward-related brain regions in experimental conditions in which they believed that their choice of an action determined reward acquisition.

As suggested by the *Biological Sensitivity to Context* framework (Boyce & Ellis, 2005) and more recently in the *Adaptive Calibration Model* (Del Giudice, Ellis, & Shirliff, 2011), vantage sensitivity may also be a function of a highly reactive stress response system (e.g., high cortisol reactivity). According to this view, the stress response system plays an important role in regulating sensitivity to environmental resources. Indeed, it can be regarded as an information-acquisition device that relays to the inside of the body what is going on outside the body.

According to the *differential-susceptibility* framework (Belsky & Pluess, 2009a) as well as the concept of *sensory-processing sensitivity* (Aron & Aron, 1997; Aron et al., 2012), the primary reason why some individuals are more responsive to positive influences than others may be because they have a more sensitive central nervous system on which experiences register more easily and deeply. In a simple attempt to integrate this “neurosensitivity” claim with the empirical observations already made regarding three different categories of vantage-sensitivity factors, we speculate that specific gene variants (e.g., 5-HTTLPR short allele, DRD4 7-repeat) contribute to the increased sensitivity and responsiveness of specific brain regions. The increased neurosensitivity in these brain regions then manifests itself in increased negative emotionality and physiological reactivity (Pluess, Stevens, & Belsky, in press), in part because highly sensitive individuals are easily aroused.

One brain region that seems very likely to be involved in vantage sensitivity (as well as differential susceptibility) is the amygdala, which is part of the limbic system and plays an important role in the processing of emotional stimuli (Sander, Grafman, & Zalla, 2003). Importantly—and contrary to the outdated view that the amygdala primarily functions to detect and process fearful stimuli (Adolphs et al., 1999; Davis & Whalen, 2001)—recent research shows that it responds even more strongly to positive stimuli (for meta-analysis, see Sergerie, Chochol, & Armony, 2008). It is no stretch of the imagination, then, to infer that amygdala reactivity might be one possible central nervous mechanism by which vantage sensitivity operates. Certainly consistent with this claim is evidence that amygdala reactivity is greater in individuals carrying the 5-HTTLPR short allele (Munafò, Brown, & Hariri, 2008) and in individuals with difficult infant temperament (Pérez-Edgar et al., 2007; Schwartz, Wright, Shin, Kagan, &

Rauch, 2003), both of which we have identified already as vantage-sensitivity factors.

Also important—and consistent with our contention that vantage sensitivity may be environmentally induced—is recent research chronicling effects of early environmental quality on amygdala size (Lupien et al., 2011; Tottenham et al., 2010). Regardless of whether amygdala reactivity should be conceptualized as a neurological mechanism underlying vantage sensitivity, it would be a mistake to conclude that it is the only or even necessarily most important neurological substrate of vantage sensitivity. Most likely, vantage sensitivity is the function of different central nervous mechanisms, including processes related to attention, reward sensitivity, social cognition, and the stress response system. Whether these different mechanisms represent independent and domain-specific vantage sensitivity or whether they are all a function of general vantage sensitivity, due to being associated with each other, remains to be determined.

Finally and on a related note, some vantage sensitivity factors may build over time in response to positive and supportive exposures. Consider in this regard the fact that adults with higher initial levels of vagal tone (measured as RSA), which as noted earlier is related to increased vantage sensitivity (Eisenberg et al., 2012; Obradović et al., 2010), increased in positive emotions and social connectedness more rapidly than others over a period of 9 weeks, and these increases themselves forecast further growth in vagal tone independent of baseline RSA measures (Kok & Fredrickson, 2010). In other words, the individual propensity for vantage sensitivity may increase over time as a result of exposure to positive influences—consistent with the notion of upward spiral dynamics (Fredrickson & Joiner, 2002). Given that such an upward and positive spiral could characterize some more than others, one might expect interindividual differences in vantage sensitivity to become larger over time in a positive environment. For example, individuals with high cognitive abilities (i.e., IQ) may be more likely to benefit from high quality education which then increases their cognitive abilities even further and with that the probability that they will also benefit more from future high quality education experiences.

Conclusion

There is now emerging evidence that individuals differ in their *positive* response to *beneficial* experiences and exposures. Such variation is not simply a result, at least in intervention studies, of the quality of service delivery but also a function of endogenous—behavioral, physiological, and genetic—characteristics of individuals. Not until the move from diathesis-stress to differential-susceptibility thinking, though, has this become especially apparent. As we have sought to make clear, while the vantage sensitivity concept we are formally and explicitly introducing here is indisputably related to differential susceptibility, it is not the same, just as differential susceptibility is also not the same as diathesis-stress. Whereas differential susceptibility calls attention to individual differences in developmental plasticity—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 nega-

tive experiences and exposures, consistent with diathesis stress, others may be disproportionately susceptible to positive environmental conditions, consistent with vantage sensitivity. Still others may be disproportionately susceptible to both—or to neither. Besides calling special attention to individual differences in response to positive experiences and providing terminology (borrowed from Manuck, 2011; Sweitzer et al., 2012) to facilitate discussion, this analysis of vantage sensitivity should stimulate researchers, perhaps especially those within the subfields of clinical and positive psychology, to pay more attention to endogenous determinants of variation in response to presumptively beneficial experiences and exposures. Failure to do so will most likely lead to misestimation of treatment effects: For those who are especially vantage sensitive, treatment effects will be *underestimated*, whereas for those who are vantage resistant they will be *overestimated*.

Clearly, more research is required to address at least two important questions related to vantage sensitivity. First and in line with an observation we made prior to reviewing empirical evidence for vantage sensitivity, the majority of existing studies do not afford determination of whether a vantage-sensitivity finding reflects simply the positive end of differential susceptibility, due to the fact that only positive components of the environment have been studied, or exclusively vantage sensitivity as defined herein, increased sensitivity to supportive but not to adverse environmental conditions. Consequently, whether a moderating factor represents a diathesis-stress-related risk factor, a vantage-sensitivity factor, or both (i.e., a general susceptibility factor) can only be determined if both the diathesis-stress and vantage sensitivity components of differential susceptibility are tested within the same study using adequate statistical procedures (Belsky, Pluess, & Widaman, 2012; Roisman et al., 2012; Widaman et al., in press). While it is possible to compare associations between naturally occurring variation in environmental quality and outcomes across *different* individuals—with some being exposed to a more negative and others to a more positive environment—it must be appreciated that it is rather challenging to carry out experimental research in which the *same* individuals are exposed to *both* negative and positive conditions as it would be very unethical to purposefully expose study participants to negative experiences. What will be called for—and remains almost completely absent even in studies of differential susceptibility—are repeated-measures investigations that evaluate the responsiveness of the same individuals—not just individuals sharing the same plasticity factors—to naturally occurring changes over time in both positive and negative environmental conditions (e.g., Verschoor & Markus, 2011). Second, future research should address whether there are psychological, behavioral, or neurobiological mechanisms that are specific to vantage sensitivity.

It is not difficult to imagine the practical benefits that might accrue from the theoretically anticipated discovery of such variation in response to treatments intended to benefit children, parents, and others, be those treatments intended to remediate problems, prevent them from developing in the first place or becoming worse, or promote positive functioning. After all, if one could identify in advance—due to explicit consideration of vantage sensitivity—those most likely to benefit from a treatment or intervention and the endogenous resistance factors most likely to undermine service effectiveness, then that service could perhaps be

provided on a more efficient basis. Consider in this regard not just the financial cost of endeavoring to enhance the functioning of someone who evidence indicates is unlikely to benefit—or at least not to the degree of others—but also the disappointment experienced by such recipients and their service providers.

On the basis of the notion—as of yet untested—that vantage sensitivity might be domain specific, the preceding discussion should not be regarded as inherently pessimistic vis-à-vis those who seem to be vantage resistant and thus unlikely to benefit from a particular treatment. If vantage-sensitivity resistance factors undermining treatment efficacy could be identified, then it might prove possible to match individuals to particular treatments. But if such an effort is going to succeed, one must first begin with the notion of vantage sensitivity and, as a result, entertain promotive and resistance factors when planning and providing a service. Although it is not news to argue that different people may require different treatments to achieve the same outcome—whether that be the amelioration of some problem or the promotion of some valued outcome—we have until recently lacked theory and evidence regarding what endogenous factors might matter in this regard. Just to be clear here, the range of treatments we are considering extends well beyond those provided in clinical settings to “patients” and “clients,” and thus includes widely utilized routine child care, educational, and other services.

A final and related point concerns whether vantage sensitivity itself can be directly influenced through intervention. Evidence cited earlier suggesting that some vantage-sensitivity factors are shaped by early environmental influences certainly suggests that this might be possible. If so, efficacy of existing psychological interventions and services might be increased drastically by interventions that target the promotion of vantage sensitivity.

In conclusion, vantage sensitivity provides a new concept for the ubiquitous observation that individuals differ generally in their response to positive experiences. Application of vantage sensitivity reasoning to clinical, developmental, and educational psychology may significantly enhance the person-environment fit for a variety of interventions and services, eventually maximizing efficacy on an individual basis.

References

- Adolphs, R., Tranel, D., Hamann, S., Young, A. W., Calder, A. J., Phelps, E. A., . . . Damasio, A. R. (1999). Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia*, *37*, 1111–1117. doi:10.1016/S0028-3932(99)00039-1
- Alexander, N., Kuepper, Y., Schmitz, A., Osinsky, R., Kozyra, E., & Hennig, J. (2009). Gene-environment interactions predict cortisol responses after acute stress: Implications for the etiology of depression. *Psychoneuroendocrinology*, *34*, 1294–1303. doi:10.1016/j.psyneuen.2009.03.017
- Armbruster, D., Mueller, A., Moser, D. A., Lesch, K. P., Brocke, B., & Kirschbaum, C. (2009). Interaction effect of D4 dopamine receptor gene and serotonin transporter promoter polymorphism on the cortisol stress response. *Behavioral Neuroscience*, *123*, 1288–1295. doi:10.1037/a0017615
- Aron, E. N., & Aron, A. (1997). Sensory-processing sensitivity and its relation to introversion and emotionality. *Journal of Personality and Social Psychology*, *73*, 345–368. doi:10.1037/0022-3514.73.2.345
- Aron, E. N., Aron, A., & Davies, K. M. (2005). Adult shyness: The interaction of temperamental sensitivity and an adverse childhood envi-

- ronment. *Personality and Social Psychology Bulletin*, 31, 181–197. doi:10.1177/0146167204271419
- Aron, E. N., Aron, A., & Jagiellowicz, J. (2012). Sensory processing sensitivity: A review in the light of the evolution of biological responsibility. *Personality and Social Psychology Review*, 16, 262–282. doi:10.1177/1088868311434213
- Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2006). Gene-environment interaction of the dopamine D4 receptor (DRD4) and observed maternal insensitivity predicting externalizing behavior in preschoolers. *Developmental Psychobiology*, 48, 406–409. doi:10.1002/dev.20152
- Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2011). Differential susceptibility to rearing environment depending on dopamine-related genes: New evidence and a meta-analysis. *Development and Psychopathology*, 23, 39–52. doi:10.1017/S0954579410000635
- Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., Pijlman, F. T., Mesman, J., & Juffer, F. (2008). Experimental evidence for differential susceptibility: Dopamine D4 receptor polymorphism (DRD4 VNTR) moderates intervention effects on toddlers' externalizing behavior in a randomized controlled trial. *Developmental Psychology*, 44, 293–300. doi:10.1037/0012-1649.44.1.293
- Bar-Haim, Y., Fox, N. A., Benson, B., Guyer, A. E., Williams, A., Nelson, E. E., . . . Ernst, M. (2009). Neural correlates of reward processing in adolescents with a history of inhibited temperament. *Psychological Science*, 20, 1009–1018. doi:10.1111/j.1467-9280.2009.02401.x
- Beevers, C. G., Marti, C. N., Lee, H. J., Stote, D. L., Ferrell, R. E., Hariri, A. R., & Telch, M. J. (2011). Associations between serotonin transporter gene promoter region (5-HTTLPR) polymorphism and gaze bias for emotional information. *Journal of Abnormal Psychology*, 120, 187–197. doi:10.1037/a0022125
- Beevers, C. G., Wells, T. T., Ellis, A. J., & McGeary, J. E. (2009). Association of the serotonin transporter gene promoter region (5-HTTLPR) polymorphism with biased attention for emotional stimuli. *Journal of Abnormal Psychology*, 118, 670–681. doi:10.1037/a0016198
- Belsky, J. (1997). Theory testing, effect-size evaluation, and differential susceptibility to rearing influence: The case of mothering and attachment. *Child Development*, 68, 598–600. doi:10.2307/1132110
- Belsky, J. (2005). Differential susceptibility to rearing influences: An evolutionary hypothesis and some evidence. In B. Ellis & D. Bjorklund (Eds.), *Origins of the social mind: Evolutionary psychology and child development* (pp. 139–163). New York, NY: Guilford Press.
- Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). For better and for worse: Differential susceptibility to environmental influences. *Current Directions in Psychological Science*, 16, 300–304. doi:10.1111/j.1467-8721.2007.00525.x
- Belsky, J., Hsieh, K. H., & Crnic, K. (1998). Mothering, fathering, and infant negativity as antecedents of boys' externalizing problems and inhibition at age 3 years: Differential susceptibility to rearing experience? *Development and Psychopathology*, 10, 301–319. doi:10.1017/S095457949800162X
- Belsky, J., Jonassaint, C., Pluess, M., Stanton, M., Brummett, B., & Williams, R. (2009). Vulnerability genes or plasticity genes? *Molecular Psychiatry*, 14, 746–754. doi:10.1038/mp.2009.44
- Belsky, J., & Pluess, M. (2009a). Beyond diathesis-stress: Differential susceptibility to environmental influences. *Psychological Bulletin*, 135, 885–908. doi:10.1037/a0017376
- Belsky, J., & Pluess, M. (2009b). The nature (and nurture?) of plasticity in early human development. *Perspectives on Psychological Science*, 4, 345–351. doi:10.1111/j.1745-6924.2009.01136.x
- Belsky, J., & Pluess, M. (in press). Genetic moderation of early child care effects on behavior problems and social competence across childhood: A developmental analysis. *Child Development*.
- Belsky, J., Pluess, M., & Widaman, K. F. (2012). *Confirmatory and competitive evaluation of alternative gene-environment interaction hypotheses*. Manuscript submitted for publication.
- Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, 62, 647–670. doi:10.2307/1131166
- Borg, J., Henningson, S., Saijo, T., Inoue, M., Bah, J., Westberg, L., . . . Farde, L. (2009). Serotonin transporter genotype is associated with cognitive performance but not regional 5-HT1A receptor binding in humans. *International Journal of Neuropsychopharmacology*, 12, 783–792. doi:10.1017/S1461145708009759
- Boyce, W. T., Chesney, M., Alkon, A., Tschann, J. M., Adams, S., Chesterman, B., . . . Wara, D. (1995). Psychobiologic reactivity to stress and childhood respiratory illnesses: Results of two prospective studies. *Psychosomatic Medicine*, 57, 411–422.
- Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*, 17, 271–301. doi:10.1017/S0954579405050145
- Cameron, N. M., Champagne, F. A., Parent, C., Fish, E. W., Ozaki-Kuroda, K., & Meaney, M. J. (2005). The programming of individual differences in defensive responses and reproductive strategies in the rat through variations in maternal care. *Neuroscience and Biobehavioral Reviews*, 29, 843–865. doi:10.1016/j.neubiorev.2005.03.022
- Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., . . . Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, 301, 386–389. doi:10.1126/science.1083968
- Cassidy, J., Woodhouse, S. S., Sherman, L. J., Stupica, B., & Lejuez, C. W. (2011). Enhancing infant attachment security: An examination of treatment efficacy and differential susceptibility. *Development and Psychopathology*, 23, 131–148. doi:10.1017/S0954579410000696
- Cicchetti, D., & Garnezy, N. (1993). Prospects and promises in the study of resilience. *Development and Psychopathology*, 5, 497–502. doi:10.1017/S0954579400006118
- Clarke, P. J., Chen, N. T., & Guastella, A. J. (2012). Prepared for the best: Readiness to modify attentional processing and reduction in anxiety vulnerability in response to therapy. *Emotion*. doi:10.1037/a0025592
- Clasen, P. C., Wells, T. T., Knopik, V. S., McGeary, J. E., & Beevers, C. G. (2011). 5-HTTLPR and BDNF Val66Met polymorphisms moderate effects of stress on rumination. *Genes, Brain & Behavior*, 10, 740–746. doi:10.1111/j.1601-183X.2011.00715.x
- Cummings, E. M., El-Sheikh, M., Kouros, C. D., & Keller, P. S. (2007). Children's skin conductance reactivity as a mechanism of risk in the context of parental depressive symptoms. *Journal of Child Psychology and Psychiatry*, 48, 436–445. doi:10.1111/j.1469-7610.2006.01713.x
- Davis, M., & Whalen, P. J. (2001). The amygdala: Vigilance and emotion. *Molecular Psychiatry*, 6, 13–34. doi:10.1038/sj.mp.4000812
- Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress reactivity. *Neuroscience and Biobehavioral Reviews*, 35, 1562–1592. doi:10.1016/j.neubiorev.2010.11.007
- DeRubeis, R. J., Hollon, S. D., Amsterdam, J. D., Shelton, R. C., Young, P. R., Salomon, R. M., . . . Gallop, R. (2005). Cognitive therapy vs medications in the treatment of moderate to severe depression. *Archives of General Psychiatry*, 62, 409–416. doi:10.1001/archpsyc.62.4.409
- Dick, D. M., Meyers, J. L., Latendresse, S. J., Creemers, H. E., Lansford, J. E., Pettit, G. S., . . . Huizink, A. C. (2011). CHRM2, parental monitoring, and adolescent externalizing behavior: Evidence for gene-environment interaction. *Psychological Science*, 22, 481–489. doi:10.1177/0956797611403318
- Dinkel, A., Herschbach, P., Berg, P., Waadt, S., Duran, G., Engst-Hastreiter, U., . . . Book, K. (2012). Determinants of long-term response to group therapy for dysfunctional fear of progression in chronic dis-

- eases. *Behavioral Medicine*, 38, 1–5. doi:10.1080/08964289.2011.640364
- Drury, S. S., Gleason, M. M., Theall, K. P., Smyke, A. T., Nelson, C. A., Fox, N. A., & Zeanah, C. H. (2012). Genetic sensitivity to the caregiving context: The influence of 5HTTLPR and BDNF val66met on indiscriminate social behavior. *Physiology & Behavior*, 106, 728–735. doi:10.1016/j.physbeh.2011.11.014
- Eisenberg, N., Sulik, M. J., Spinrad, T. L., Edwards, A., Eggum, N. D., Liew, J., . . . Hart, D. (2012). Differential susceptibility and the early development of aggression: Interactive effects of respiratory sinus arrhythmia and environmental quality. *Developmental Psychology*. doi:10.1037/a0026518
- Eley, T. C., Hudson, J. L., Creswell, C., Tropeano, M., Lester, K. J., Cooper, P., . . . Collier, D. A. (2012). Therapygenetics: The 5HTTLPR and response to psychological therapy. *Molecular Psychiatry*, 17, 236–237. doi:10.1038/mp.2011.132
- Ellis, B. J., & Boyce, W. T. (2011). Differential susceptibility to the environment: Toward an understanding of sensitivity to developmental experiences and context. *Development and Psychopathology*, 23, 1–5. doi:10.1017/S095457941000060X
- Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. *Development and Psychopathology*, 23, 7–28. doi:10.1017/S0954579410000611
- Faraone, S. V., Doyle, A. E., Mick, E., & Biederman, J. (2001). Meta-analysis of the association between the 7-repeat allele of the dopamine D(4) receptor gene and attention deficit hyperactivity disorder. *The American Journal of Psychiatry*, 158, 1052–1057. doi:10.1176/appi.ajp.158.7.1052
- Forbes, E. E., Stepp, S. D., Dahl, R. E., Ryan, N. D., Whalen, D., Axelson, D. A., . . . Silk, J. S. (2012). Real-world affect and social context as predictors of treatment response in child and adolescent depression and anxiety: An ecological momentary assessment study. *Journal of Child and Adolescent Psychopharmacology*, 22, 37–47. doi:10.1089/cap.2011.0085
- Fox, E., Ridgewell, A., & Ashwin, C. (2009). Looking on the bright side: Biased attention and the human serotonin transporter gene. *Proceedings of the Royal Society: B. Biological Sciences*, 276, 1747–1751. doi:10.1098/rspb.2008.1788
- Fox, E., Zoukou, K., Ridgewell, A., & Garner, K. (2011). The serotonin transporter gene alters sensitivity to attention bias modification: Evidence for a plasticity gene. *Biological Psychiatry*, 70, 1049–1054. doi:10.1016/j.biopsych.2011.07.004
- Fredrickson, B. L., & Joiner, T. (2002). Positive emotions trigger upward spirals toward emotional well-being. *Psychological Science*, 13, 172–175. doi:10.1111/1467-9280.00431
- Gable, S. L., & Haidt, J. (2005). What (and why) is positive psychology. *Review of General Psychology*, 9, 103–110. doi:10.1037/1089-2680.9.2.103
- Garnezy, N. (1991). Resilience in children's adaptation to negative life events and stressed environments. *Pediatric Annals*, 20, 459–466.
- Ginsburg, G. S., Kendall, P. C., Sakolsky, D., Compton, S. N., Piacentini, J., Albano, A. M., . . . March, J. (2011). Remission after acute treatment in children and adolescents with anxiety disorders: Findings from the CAMS. *Journal of Consulting and Clinical Psychology*, 79, 806–813. doi:10.1037/a0025933
- Gotlib, I. H., Joormann, J., Minor, K. L., & Hallmayer, J. (2008). HPA axis reactivity: A mechanism underlying the associations among 5-HTTLPR, stress, and depression. *Biological Psychiatry*, 63, 847–851. doi:10.1016/j.biopsych.2007.10.008
- Hankin, B. L., Nederhof, E., Oppenheimer, C. W., Jenness, J., Young, J. F., Abela, J. R. Z., . . . Oldehinkel, A. J. (2011). Differential susceptibility in youth: Evidence that 5-HTTLPR \times positive parenting is associated with positive affect 'for better and worse'. *Translational Psychiatry*, 1, e44. doi:10.1038/tp.2011.44
- Held, B. S. (2004). The negative side of positive psychology. *Journal of Humanistic Psychology*, 44, 9–46. doi:10.1177/0022167803259645
- Holmboe, K., Nemoda, Z., Fearon, R. M., Sasvari-Szekely, M., & Johnson, M. H. (2011). Dopamine D4 receptor and serotonin transporter gene effects on the longitudinal development of infant temperament. *Genes, Brain & Behavior*, 10, 513–522. doi:10.1111/j.1601-183X.2010.00669.x
- Homberg, J. R., & Lesch, K. P. (2011). Looking on the bright side of serotonin transporter gene variation. *Biological Psychiatry*, 69, 513–519. doi:10.1016/j.biopsych.2010.09.024
- Houghton Mifflin. (2000). *The American heritage dictionary of the English language* (4th ed.). Boston, MA: Houghton Mifflin.
- Jagiellowicz, J., Xu, X., Aron, A., Aron, E., Cao, G., Feng, T., & Weng, X. (2011). The trait of sensory processing sensitivity and neural responses to changes in visual scenes. *Social Cognitive and Affective Neuroscience*, 6, 38–47. doi:10.1093/scan/nsq001
- Kaplan, L. A., Evans, L., & Monk, C. (2008). Effects of mothers' prenatal psychiatric status and postnatal caregiving on infant biobehavioral regulation: Can prenatal programming be modified? *Early Human Development*, 84, 249–256. doi:10.1016/j.earlhumdev.2007.06.004
- Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry*, 68, 444–454. doi:10.1001/archgenpsychiatry.2010.189
- Kegel, C. A. T., Bus, A. G., & van IJzendoorn, M. H. (2011). Differential susceptibility in early literacy instruction through computer games: The role of the dopamine D4 receptor gene (DRD4). *Mind, Brain, and Education*, 5, 71–78. doi:10.1111/j.1751-228X.2011.01112.x
- Kennard, B., Silva, S., Vitiello, B., Curry, J., Kratochvil, C., Simons, A., . . . March, J. (2006). Remission and residual symptoms after short-term treatment in the Treatment of Adolescents With Depression Study (TADS). *Journal of the American Academy of Child & Adolescent Psychiatry*, 45, 1404–1411. doi:10.1097/01.chi.0000242228.75516.21
- Kim, S., & Kochanska, G. (2012). Child temperament moderates effects of parent–child mutuality on self-regulation: A relationship-based path for emotionally negative infants. *Child Development*, 83, 1275–1289. doi:10.1111/j.1467-8624.2012.01778.x
- Kluger, A. N., Siegfried, Z., & Ebstein, R. P. (2002). A meta-analysis of the association between DRD4 polymorphism and novelty seeking. *Molecular Psychiatry*, 7, 712–717. doi:10.1038/sj.mp.4001082
- Knafo, A., Israel, S., & Ebstein, R. P. (2011). Heritability of children's prosocial behavior and differential susceptibility to parenting by variation in the dopamine receptor D4 gene. *Development and Psychopathology*, 23, 53–67. doi:10.1017/S0954579410000647
- Kochanska, G., Kim, S., Barry, R. A., & Philibert, R. A. (2011). Children's genotypes interact with maternal responsive care in predicting children's competence: Diathesis-stress or differential susceptibility? *Development and Psychopathology*, 23, 605–616. doi:10.1017/S0954579411000071
- Kok, B. E., & Fredrickson, B. L. (2010). Upward spirals of the heart: Autonomic flexibility, as indexed by vagal tone, reciprocally and prospectively predicts positive emotions and social connectedness. *Biological Psychology*, 85, 432–436. doi:10.1016/j.biopsycho.2010.09.005
- Kuepper, Y., Wielpuetz, C., Alexander, N., Mueller, E., Grant, P., & Hennig, J. (2012). 5-HTTLPR S-Allele: A genetic plasticity factor regarding the effects of life events on personality? *Genes, Brain & Behavior*. doi:10.1111/j.1601-183X.2012.00783.x
- Lazarus, R. S. (2003). Does the positive psychology movement have legs? *Psychological Inquiry*, 14, 93–109. doi:10.1207/S15327965PLI1402_02
- Linley, P. A., Joseph, S., Harrington, S., & Wood, A. M. (2006). Positive psychology: Past, present, and (possible) future. *The Journal of Positive Psychology*, 1, 3–16. doi:10.1080/17439760500372796
- Lopez, S. J., & Snyder, C. R. (Eds.). (2011). *The oxford handbook of positive psychology*. New York, NY: Oxford University Press.

- Luecken, L. J., Hagan, M. J., Sandler, I. N., Tein, J. Y., Ayers, T. S., & Wolchik, S. A. (2010). Cortisol levels 6-years after participation in the Family Bereavement Program. *Psychoneuroendocrinology*, *35*, 785–789. doi:10.1016/j.psyneuen.2009.11.002
- Lupien, S. J., Parent, S., Evans, A. C., Tremblay, R. E., Zelazo, P. D., Corbo, V., . . . Seguin, J. R. (2011). Larger amygdala but no change in hippocampal volume in 10-year-old children exposed to maternal depressive symptomatology since birth. *Proceedings of the National Academy of Sciences of the United States of America*, *108*, 14324–14329. doi:10.1073/pnas.1105371108
- Luthar, S. S. (2006). Resilience in development: A synthesis of research across five decades. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Vol. 3. Risk, disorder, and adaptation* (2nd ed., pp. 739–795). Hoboken, NJ: Wiley.
- Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development*, *71*, 543–562. doi:10.1111/1467-8624.00164
- Manuck, S. B. (2011). *Delay discounting covaries with childhood socio-economic status as a function of genetic variation in the dopamine D4 receptor (DRD4)*. Paper presented at the Society for Research in Child Development, Montreal, Quebec, Canada.
- Masten, A. S., Hubbard, J. J., Gest, S. D., Tellegen, A., Garmezy, N., & Ramirez, M. (1999). Competence in the context of adversity: Pathways to resilience and maladaptation from childhood to late adolescence. *Development and Psychopathology*, *11*, 143–169. doi:10.1017/S0954579499001996
- Masten, A. S., & Obradović, J. (2006). Competence and resilience in development. *Annals of the New York Academy of Sciences*, *1094*, 13–27. doi:10.1196/annals.1376.003
- Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin*, *110*, 406–425. doi:10.1037/0033-2909.110.3.406
- Munafò, M. R., Brown, S. M., & Hariri, A. R. (2008). Serotonin transporter (5-HTTLPR) genotype and amygdala activation: A meta-analysis. *Biological Psychiatry*, *63*, 852–857. doi:10.1016/j.biopsych.2007.08.016
- Munafò, M. R., Freimer, N. B., Ng, W., Ophoff, R., Veijola, J., Miettunen, J., . . . Flint, J. (2009). 5-HTTLPR genotype and anxiety-related personality traits: A meta-analysis and new data. *American Journal of Medical Genetics: Part B. Neuropsychiatric Genetics*, *150*, 271–281. doi:10.1002/ajmg.b.30808
- Nakamura, M., Ueno, S., Sano, A., & Tanabe, H. (2000). The human serotonin transporter gene linked polymorphism (5-HTTLPR) shows 10 novel allelic variants. *Molecular Psychiatry*, *5*, 32–38. doi:10.1038/sj.mp.4000698
- NICHD Early Child Care Research Network. (2005). *Child care and child development: Results of the NICHD Study of Early Child Care and Youth Development*. New York, NY: Guilford Press.
- Obradović, J., & Boyce, W. T. (2009). Individual differences in behavioral, physiological, and genetic sensitivities to contexts: Implications for development and adaptation. *Developmental Neuroscience*, *31*, 300–308. doi:10.1159/000216541
- Obradović, J., Bush, N. R., Stamplerdahl, J., Adler, N. E., & Boyce, W. T. (2010). Biological sensitivity to context: The interactive effects of stress reactivity and family adversity on socio-emotional behavior and school readiness. *Child Development*, *81*, 270–289. doi:10.1111/j.1467-8624.2009.01394.x
- Pérez-Edgar, K., Roberson-Nay, R., Hardin, M. G., Poeth, K., Guyer, A. E., Nelson, E. E., . . . Ernst, M. (2007). Attention alters neural responses to evocative faces in behaviorally inhibited adolescents. *NeuroImage*, *35*, 1538–1546. doi:10.1016/j.neuroimage.2007.02.006
- Pergamin-Hight, L., Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., & Bar-Haim, Y. (2012). Variations in the promoter region of the serotonin transporter gene and biased attention for emotional information: A meta-analysis. *Biological Psychiatry*, *71*, 373–379. doi:10.1016/j.biopsych.2011.10.030
- Plomin, R., & Daniels, D. (1987). Why are children in the same family so different from one another? *Behavioral and Brain Sciences*, *10*, 1–16. doi:10.1017/S0140525X00055941
- Pluess, M., & Belsky, J. (2009). Differential susceptibility to rearing experience: The case of childcare. *Journal of Child Psychology and Psychiatry*, *50*, 396–404. doi:10.1111/j.1469-7610.2008.01992.x
- Pluess, M., & Belsky, J. (2010). Differential susceptibility to parenting and quality child care. *Developmental Psychology*, *46*, 379–390. doi:10.1037/a0015203
- Pluess, M., & Belsky, J. (2011). Prenatal programming of postnatal plasticity? *Development and Psychopathology*, *23*, 29–38. doi:10.1017/S0954579410000623
- Pluess, M., Belsky, J., Way, B. M., & Taylor, S. E. (2010). 5-HTTLPR moderates effects of life events on neuroticism: Differential susceptibility to environmental influences. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *34*, 1070–1074. doi:10.1016/j.pnpbp.2010.05.028
- Pluess, M., & Boniwell, I. (2012). *High sensitive personality moderates responsivity to positive effects of a school-based resilience-promoting intervention*. Manuscript in preparation.
- Pluess, M., Boniwell, I., Hefferon, K., & Tunariu, A. (2012). *Evaluation of a school-based resilience-promoting intervention in a high-risk population in England: A controlled mixed methods trial*. Manuscript in preparation.
- Pluess, M., Bosman, A., Bakker, J., Wald, S., Aron, A., & Aron, E. (2012). *The Highly Sensitive Child Questionnaire*. Manuscript in preparation.
- Pluess, M., Stevens, S., & Belsky, J. (in press). Differential susceptibility: developmental and evolutionary mechanisms of gene and environment interactions. In M. Legerstee, D. W. Haley, & M. H. Bornstein (Eds.), *Developing infant mind: Integrating biology and experience*. New York, NY: Guilford Press.
- Pluess, M., Velders, F. P., Belsky, J., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., Jaddoe, V. W., . . . Tiemeier, H. (2011). Serotonin transporter polymorphism moderates effects of prenatal maternal anxiety on infant negative emotionality. *Biological Psychiatry*, *69*, 520–525. doi:10.1016/j.biopsych.2010.10.006
- Poehlmann, J., Schwichtenberg, A. J., Schlafer, R. J., Hahn, E., Bianchi, J. P., & Warner, R. (2011). Emerging self-regulation in toddlers born preterm or low birth weight: Differential susceptibility to parenting? *Development and Psychopathology*, *23*, 177–193. doi:10.1017/S0954579410000726
- Preacher, K. J., Curran, P. J., & Bauer, D. J. (2006). Computational tools for probing interactions in multiple linear regressions, multilevel modeling, and latent curve analysis. *Journal of Educational and Behavioral Statistics*, *31*, 437–448. doi:10.3102/10769986031004437
- Ramchandani, P. G., van IJzendoorn, M. H., & Bakermans-Kranenburg, M. J. (2010). Differential susceptibility to fathers' care and involvement: The moderating effect of infant reactivity. *Family Science*, *1*, 93–101. doi:10.1080/19424621003599835
- Risch, N., Herrell, R., Lehner, T., Liang, K. Y., Eaves, L., Hoh, J., . . . Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. *JAMA: Journal of the American Medical Association*, *301*, 2462–2471. doi:10.1001/jama.2009.878
- Robbins, T. W., & Everitt, B. J. (1999). Motivation and reward. In M. J. Zigmond, S. C. Bloom, S. C. Landis, J. L. Roberts, & L. R. Squire (Eds.), *Fundamental neuroscience* (pp. 1245–1260). San Diego, CA: Academic Press.
- Roiser, J. P., Rogers, R. D., Cook, L. J., & Sahakian, B. J. (2006). The effect of polymorphism at the serotonin transporter gene on decision-making, memory and executive function in ecstasy users and controls. *Psychopharmacology*, *188*, 213–227. doi:10.1007/s00213-006-0495-z

- Roisman, G. I., Newman, D. A., Fraley, R. C., Haltigan, J. D., Groh, A. M., & Haydon, K. C. (2012). Distinguishing differential susceptibility from diathesis-stress: Recommendations for evaluating interaction effects. *Development and Psychopathology, 24*, 389–409. doi:10.1017/S0954579412000065
- Rothbart, M. K., & Bates, J. E. (2006). Temperament. In N. Eisenberg, W. Damon, & R. M. Lerner (Eds.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 99–166). Hoboken, NJ: Wiley.
- Ruiz, R. J., & Avant, K. C. (2005). Effects of maternal prenatal stress on infant outcomes: A synthesis of the literature. *Advances in Nursing Science, 28*, 345–355.
- Rutter, M. (1987). Psychosocial resilience and protective mechanisms. *American Journal of Orthopsychiatry, 57*, 316–331. doi:10.1111/j.1939-0025.1987.tb03541.x
- Rutter, M., Moffitt, T. E., & Caspi, A. (2006). Gene-environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry, 47*, 226–261. doi:10.1111/j.1469-7610.2005.01557.x
- Sameroff, A. J. (2000). Developmental systems and psychopathology. *Development and Psychopathology, 12*, 297–312. doi:10.1017/S0954579400003035
- Sander, D., Grafman, J., & Zalla, T. (2003). The human amygdala: An evolved system for relevance detection. *Reviews in the Neurosciences, 14*, 303–316. doi:10.1515/REVNEURO.2003.14.4.303
- Schoebi, D., Way, B. M., Karney, B. R., & Bradbury, T. N. (2012). Genetic moderation of sensitivity to positive and negative affect in marriage. *Emotion, 12*, 208–212. doi:10.1037/a0026067
- Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants “grown up”: Adult amygdalar response to novelty. *Science, 300*, 1952–1953. doi:10.1126/science.1083703
- Seligman, M. E. (2011). *Flourish: A visionary new understanding of happiness and well-being*. New York, NY: Free Press.
- Seligman, M. E., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist, 55*, 5–14. doi:10.1037/0003-066X.55.1.5
- Seligman, M. E., Steen, T. A., Park, N., & Peterson, C. (2005). Positive psychology progress: Empirical validation of interventions. *American Psychologist, 60*, 410–421. doi:10.1037/0003-066X.60.5.410
- Sen, S., Burmeister, M., & Ghosh, D. (2004). Meta-analysis of the association between a serotonin transporter promoter polymorphism (5-HTTLPR) and anxiety-related personality traits. *American Journal of Medical Genetics: Part B. Neuropsychiatric Genetics, 127*, 85–89. doi:10.1002/ajmg.b.20158
- Sergerie, K., Chochol, C., & Armony, J. L. (2008). The role of the amygdala in emotional processing: A quantitative meta-analysis of functional neuroimaging studies. *Neuroscience and Biobehavioral Reviews, 32*, 811–830. doi:10.1016/j.neubiorev.2007.12.002
- Stright, A. D., Cranley Gallagher, K., & Kelley, K. (2008). Infant temperament moderates relations between maternal parenting in early childhood and children’s adjustment in first grade. *Child Development, 79*, 186–200. doi:10.1111/j.1467-8624.2007.01119.x
- Stupica, B., Sherman, L. J., & Cassidy, J. (2011). Newborn irritability moderates the association between infant attachment security and toddler exploration and sociability. *Child Development, 82*, 1381–1389. doi:10.1111/j.1467-8624.2011.01638.x
- Suomi, S. J. (1995). Influence of attachment theory on ethological studies of biobehavioral development in nonhuman primates. In S. Goldberg, R. Muir, J. Kerr, S. Goldberg, R. Muir, & J. Kerr (Eds.), *Attachment theory: Social, developmental, and clinical perspectives* (pp. 185–201). Hillsdale, NJ: Analytic Press.
- Suomi, S. J. (1997). Early determinants of behaviour: Evidence from primate studies. *British Medical Bulletin, 53*, 170–184. doi:10.1093/oxfordjournals.bmb.a011598
- Sweitzer, M. M., Halder, I., Flory, J. D., Craig, A. E., Gianaros, P. J., Ferrell, R. E., & Manuck, S. B. (2012). Polymorphic variation in the dopamine D4 receptor predicts delay discounting as a function of childhood socioeconomic status: Evidence for differential susceptibility. *Social Cognitive & Affective Neuroscience*. doi:10.1093/scan/nss020
- Taylor, S. E., Way, B. M., Welch, W. T., Hilmert, C. J., Lehman, B. J., & Eisenberger, N. I. (2006). Early family environment, current adversity, the serotonin transporter promoter polymorphism, and depressive symptomatology. *Biological Psychiatry, 60*, 671–676. doi:10.1016/j.biopsych.2006.04.019
- Tottenham, N., Hare, T. A., Quinn, B. T., McCarry, T. W., Nurse, M., Gilhooly, T., . . . Casey, B. J. (2010). Prolonged institutional rearing is associated with atypically large amygdala volume and difficulties in emotion regulation. *Developmental Science, 13*, 46–61. doi:10.1111/j.1467-7687.2009.00852.x
- van de Wiel, N. M., van Goozen, S. H., Matthys, W., Snoek, H., & van Engeland, H. (2004). Cortisol and treatment effect in children with disruptive behavior disorders: A preliminary study. *Journal of the American Academy of Child & Adolescent Psychiatry, 43*, 1011–1018. doi:10.1097/01.chi.0000126976.56955.43
- van IJzendoorn, M. H., Belsky, J., & Bakermans-Kranenburg, M. J. (2012). Serotonin transporter genotype 5HTTLPR as a marker of differential susceptibility? A meta-analysis of child and adolescent gene-by-environment studies. *Translational Psychiatry, 2*, e147. doi:10.1038/tp.2012.73
- Verschoor, E., & Markus, C. R. (2011). Affective and neuroendocrine stress reactivity to an academic examination: Influence of the 5-HTTLPR genotype and trait neuroticism. *Biological Psychology, 87*, 439–449. doi:10.1016/j.biopsycho.2011.06.001
- Vonderlin, E., Pahnke, J., & Pauen, S. (2008). Infant temperament and information processing in a visual categorization task. *Infant Behavior & Development*.
- Way, B. M., & Taylor, S. E. (2010). Social influences on health: Is serotonin a critical mediator? *Psychosomatic Medicine, 72*, 107–112. doi:10.1097/PSY.0b013e3181ce6a7d
- Werner, E. E. (1997). Vulnerable but invincible: High-risk children from birth to adulthood. *Acta Paediatrica, 86*, 103–105. doi:10.1111/j.1651-2227.1997.tb18356.x
- Wetter, E. K., & El-Sheikh, M. (2012). Trajectories of children’s internalizing symptoms: The role of maternal internalizing symptoms, respiratory sinus arrhythmia and child sex. *Journal of Child Psychology and Psychiatry, 53*, 168–177. doi:10.1111/j.1469-7610.2011.02470.x
- Widaman, K. F., Helm, J. L., Castro-Schilo, L., Pluess, M., Stallings, M. C., & Belsky, J. (in press). Distinguishing ordinal and disordinal interactions. *Psychological Methods*.
- Zeanah, C. H., Nelson, C. A., Fox, N. A., Smyke, A. T., Marshall, P., Parker, S. W., & Koga, S. (2003). Designing research to study the effects of institutionalization on brain and behavioral development: The Bucharest Early Intervention Project. *Development and Psychopathology, 15*, 885–907. doi:10.1017/S0954579403000452
- Zuckerman, M. (1999). *Vulnerability to psychopathology: A biosocial model*. Washington, DC: American Psychological Association. doi:10.1037/10316-000

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