

Plasticity in Development

Jay Belsky and Michael Pluess

The Nature (and Nurture?) of Plasticity in Early Human Development

Jay Belsky and Michael Pluess

*Institute for the Study of Children, Families and Social Issues, Birkbeck University of  
London*

Address correspondence to Jay Belsky, Institute for the Study of Children, Families  
and Social Issues, Birkbeck University of London, 7 Bedford Square, London WC1B  
3RA, United Kingdom; e-mail: [j.belsky@bbk.ac.uk](mailto:j.belsky@bbk.ac.uk).

**ABSTRACT**—The effect of early experience is a long-standing concern in developmental psychology. Gaining further insight into the nature of human plasticity is central to efforts to prevent problems in development from arising and promote positive functioning. Evolutionary reasoning suggests that children should vary in their susceptibility to environmental influences, including parenting. Evidence indicates that rather than some children, such as those with negatively emotional temperaments or certain genotypes, being simply more vulnerable to the adverse effects of negative experiences, as commonly assumed, they may actually be more susceptible to both positive and negative experiences. In addition to raising questions about the nature of plasticity in human development, this article highlights unknowns regarding the role of nature and nurture in shaping individual differences in plasticity, including whether recent research linking maternal stress during pregnancy with child behavior problems illuminates a process whereby fetal programming shapes the child's susceptibility to postnatal environmental influences. Throughout this article, we raise concern about the potentially distorting influence that psychology's disproportionate focus on the adverse effect of negative experiences on developmental problems has on our understanding of human plasticity, and we propose that researchers should pay more attention to the positive side of the plasticity equation.

A central tenet of developmental psychology is that humans are affected by their experiences while growing up in ways that importantly shape their life course (though perhaps more so earlier in childhood than later). This interest manifests itself in field studies of parenting (Belsky, Fearon, & Bell, 2007) and child care (National Institute of Child Health and Human Development Early Child Care Research Network, 2006); in naturalistic investigations of especially depriving experiences, such as growing up in Romanian orphanages (Gunnar & van Dulmen, 2007); and in early intervention work evaluating the impact of systematic efforts to promote young children's well being and success in life (Olds et al., 2004). In other words, the notion that human development manifests a capacity for plasticity is widely embraced by students of child development. Thus, a core concern of many, be they basic neuroscientists, applied researchers, or educators, is elucidation of early developmental experiences and processes that undermine and enhance later functioning. Such interest has surely motivated many scholars and practitioners to work in this field.

A presumption implicit in much, although not all, work on environmental influences in early human development which is especially evident in most early intervention research is that children are similarly affected by developmental experiences, be it family poverty, parental warmth and hostility, or quality of child care to cite but a few widely investigated topics of inquiry. Variation in plasticity has been a primary focus of investigation, even if not in such terms, in the field of resilience (Cicchetti & Rogosch, 1997). Here, concerted effort has been made to identify children who are less or not at all affected by some contextual adversity presumed to undermine well-being. Even though much resilience research seeks to illuminate ecological conditions that protect children from being adversely affected by

a particular negative experience, much attention also has been paid to child characteristics (e.g., sense of humor, IQ) that operate protectively (Werner, 1997). Children who are resilient because of their own attributes might then be considered less plastic or malleable than others.

This observation that variation seems to exist in the manner and/or the degree to which negative early developmental experiences adversely affect human functioning highlights the central question we pose in this article for the field of developmental psychology: How much of plasticity in early human development is affected by nature and how much is affected by nurture? Given the premise that basic science knowledge can and should be used to guide the practice and policy of promoting human well-being while preventing problems from developing in the first place, it should be clear that advancing understanding of the nature of plasticity in early human development remains a core concern of developmentally minded psychologists.

Toward that end, we begin by offering a brief evolutionary argument as to why plasticity should be variable across individuals, especially in a family. We make a case here that the disproportionate attention paid to adversity and its problematic consequences may distort as much as it illuminates the process of human development, leaving much still to be learned about how nature and/or nurture affects plasticity in early human development. We subsequently consider evidence suggesting that, for temperamental, physiological and/or genetic reasons, some children actually are more susceptible than others to both positive and negative rearing influences. Such data raise the prospect that plasticity is primarily a function of nature rather than nurture. Before drawing some conclusions while delineating future research directions, we challenge this view, highlighting evidence that suggests

not only that plasticity may be a function of one's environment but that, for genetic reasons, this may be more true for some individuals than others. Ultimately, this article should make clear that multiple questions remain about how nature and/or nurture affect plasticity in early human development while contending that just as much attention must be accorded to enriching experiences and positive outcomes as to contextual adversity and problematic functioning for progress to be made in answering them and that an evolutionary mindset can raise issues that, for the most part, have been neglected.

### **AN EVOLUTIONARY PERSPECTIVE ON PLASTICITY**

Elsewhere, we have made the case that because the future is and always has been inherently uncertain, ancestral parents, just like parents today, could not have known (consciously or unconsciously) what child-rearing practices would prove most successful in promoting the reproductive fitness of offspring and thus their own inclusive fitness. As a result, and as a fitness optimizing strategy involving bet hedging (Philipi & Seger, 1989), natural selection would have shaped parents to bear children varying in plasticity (Belsky, 2005). This way, if an effect of parenting proved counterproductive in fitness terms, those children not affected by parenting would not have incurred the cost of developing in ways that ultimately proved to be misguided. It is important to note that, in light of inclusive fitness considerations, the less-malleable children's "resistance" to parental influence would not only have benefited themselves directly but also their more malleable siblings, although it would do so indirectly, given that siblings, like parents and children, have 50% of their genes in common. By the same token, had parenting influenced children in ways that enhanced fitness, then not only would more plastic offspring have benefited directly from parental influence, but so, too, would their parents and even their less malleable

siblings who did not benefit from the parenting they received (again for inclusive fitness reasons).

This line of evolutionary argument leads to the prediction that children should vary in their susceptibility to parental rearing and perhaps to environmental influences more generally. As it turns out, a long line of developmental inquiry informed by a "transactional" perspective (Sameroff, 1983) is more or less based on this unstated assumption. Central to this perspective is the dual-risk model of problematic functioning on which studies of resilience are founded, a perspective that shares much with classical diathesis-stress models of psychopathology (Monroe & Simons, 1991; Zuckerman, 1999): Children who are vulnerable for reasons pertaining to their biology, temperament, genetics, or some other organismic reason (e.g., prematurity) will most likely manifest compromised development when exposed to some contextual adversity (e.g., hostile parenting, poverty).

A central premise of this essay is that the widely embraced dual-risk transactional model of development may seriously distort the nature of human plasticity. This is because it is based on developmental psychology's disproportionate focus on the adverse effects of negative experiences on problems in development and, thereby, the identification of children who, for organismic reasons, are vulnerable to contextual risks or resilient to them. What the aforementioned evolutionary analysis presupposes, in contrast, is that the very children who are putatively vulnerable to adversity vis à vis problems in development may be equally and disproportionately susceptible to the developmentally beneficial effects of supportive rearing environments. Thus, more so than other children, they are especially plastic or malleable (Belsky, 1997, 2005; Boyce & Ellis, 2005) and are affected by

developmental experiences (for better and for worse) (Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2007).

One noteworthy observation in this line of argument concerning the nature of human plasticity is that the English language—and thus the vocabulary of American psychologists—does not seem to have any terms to describe the positive side of plasticity and certainly no terms as pithy as *vulnerable* and *resilient*, which are used to characterize the negative side. For example, what would one call a child who for some organismic reason disproportionately benefits from nurturing or stimulating developmental experience, be it supportive parenting or high quality child care? To capture the notion of variation in children's plasticity, Belsky (1997) coined the term *differential susceptibility to environmental influences*, which is not unrelated to Boyce and Ellis's (2005) more recent notion of *biological sensitivity to context* (see the next section). In both cases, reference is made to the fact that children who are especially vulnerable to adversity may benefit disproportionately from positive experiences.

### THE NATURE OF PLASTICITY

It is one thing to assert that some children may be more affected by developmental experiences—for better and for worse—than others, and it is another to chronicle empirically such a fact. Three sets of emerging evidence can be cited in support of the claim: one points to early temperament as a marker of variation in plasticity, another pertains to physiological reactivity, and a third pertains to measured genes.

With regard to behavioral manifestations of temperament, many investigations now show that negatively emotional or "difficult" infants and toddlers are more affected by rearing experience than are children (see Belsky, 2005, for review). Two recent studies, each drawing on data collected for the large scale National Institute of

Child Health and Human Development Study of Early Child Care (National Institute of Child Health and Human Development Early Child Care Research Network, 2005), found that infants who are rated as highly negatively emotional at 6 months by their mothers not only manifest more behavior problems in early childhood when experiencing low quality parenting (Bradley & Corwyn, 2008) or low quality child care (Pluess & Belsky, 2009) than do other children, they also display fewer problems and more social skills than other children when exposed to high quality parenting or child care. Relatedly, Kochanska, Aksan, and Joy (2007) observed that highly fearful 15-month-olds experiencing high levels of power-assertive paternal discipline were most likely to cheat in a game at 38 months, yet when they were cared for in a supportive manner such negatively emotional, fearful toddlers manifested the most rule-compatible conduct.

With regard to physiology, highly reactive children also appear more susceptible to environmental influences than do less reactive ones. Boyce et al. (1995) found that, when growing up in stressful rearing contexts 365-year-olds showing high reactivity of mean arterial blood pressure during a stress test exhibited higher rates of respiratory illness than did other children, yet under low-stress conditions such high-reactive children had a significantly lower incidence of respiratory illnesses than did other children. These findings are reminiscent of data on young adults indicating that the adverse effect of daily hassles on physical symptoms varied as a function of change in blood volume pulse amplitude during a psychological stress test (Gannon, Banks, Shelton, & Luchetta, 1989). Not only did high-reactive individuals manifest the most symptoms when hassles were high, they also exhibited the least when hassles were low; a similar set of results emerged when depression was the outcome of hassles, and speed of heart rate recovery after the stress test was the physiological

moderator. Similarly, there are more recent child data from Boyce et al. (2006) highlighting the moderating effect of anticipatory activation of the hypothalamic-pituitary-adrenal axis as indexed by salivary cortisol at the beginning of a lengthy battery of psychological assessments. For children scoring low in reactivity at age 7, father involvement in infant care failed to predict mental health at age 9. But for those 7-year-olds who were highly reactive, low levels of early father involvement forecast more severe symptoms 2 years later.

Recent research involving measured genes and measured environments may most compellingly document both positive and negative rearing effects for susceptible children. One set of studies involves infants with a particular allele (variant) of a gene called *DRD4*, which codes for a type of dopamine receptor. Because the dopaminergic system is engaged in attentional, motivational, and reward mechanisms and the variant in question, the 7-repeat allele, has been linked to lower dopamine reception efficiency, Bakermans-Kranenburg and van Ijzendoorn (2006) predicted and found that this allele moderated the association between parental insensitivity and externalizing problems. Children with the 7-repeat *DRD4* allele who experienced insensitive mothering displayed more externalizing behaviors than did children without the *DRD4* 7-repeat (irrespective of maternal sensitivity), whereas children with the 7-repeat *DRD4* allele who were reared by sensitive mothers showed the lowest levels of externalizing problem behavior. Such results suggest that conceptualizing the 7-repeat *DRD4* allele exclusively in risk-factor terms is misguided, as this variant of the gene seems to heighten susceptibility to a wide variety of environments (Bakermans-Kranenburg & van Ijzendoorn, 2007), with supportive and risky contexts promoting positive and negative outcomes, respectively.

The same may be true of alleles that are linked with high and low monoamine oxidase A activity and found to moderate the effect of child maltreatment on antisocial behavior in adulthood. What is perhaps not fully appreciated in the widely cited and ground-breaking Gene  $\times$  Environment (G  $\times$  E) interaction research of Caspi et al. (2002, see Figure 1) is that not only were those with alleles associated with low monoamine oxidase A activity proven to be most antisocial if they experienced maltreatment, but they also manifested the lowest levels of antisocial behavior when not exposed to maltreatment while growing up.

Even more compelling in this regard is recent work linking recent life events and/or childrearing history (retrospectively reported) with depression. In results that were consistent with other research by Caspi et al. (2003), Taylor et al. (2006) found that university students homozygous for short alleles (s/s) of the serotonin transporter gene polymorphism manifested greater depressive symptomatology when exposed to early or recent adversity than did individuals with other allelic variants. Just as important, however, individuals with seemingly vulnerable s/s genotypes exhibited significantly less depressive symptomatology when they experienced a supportive early environment or recent positive experiences than did those with other genotypes. Such evidence agrees with results from Wilhelm et al. (2006) research, in which they linked the number of adverse life events in a 5-year period with probability of lifetime major depression: Even though s/s individuals manifested the highest probability of depression when adverse life events were high (i.e.,  $>3$ ), they had the lowest probability when not exposed to such events.

The molecular genetic findings just considered most certainly raise the prospect that plasticity is a function of nature more than nurture. Supporting this view is extensive evidence that measured plasticity is heritable in many species (Bashey,

2006; Pigliucci, 2007) and that it may function as a selectable character in and of itself (Sinn, Gosling, & Moltchanivskyj, 2007). There is also data showing that some of the very behavioral and physiological factors highlighted above as moderators of environmental influences and thus markers of plasticity— infant negative emotionality (Auerbach, Faroy, Ebstein, Kahana, & Levine, 2001) and hypothalamic–pituitary–adrenal axis reactivity (Gotlib, Joormann, Minor, & Hallmayer, 2008)— are themselves related to at least one of the genotypes associated with heightened susceptibility to rearing: the s/s allele of the serotonin transporter promoter polymorphism. This raises the following, unanswered question regarding research highlighting differential susceptibility to rearing influence: Are investigations that show some children to be more susceptible to environmental influences than others for temperamental, physiological, or genetic reasons identifying the same highly malleable individuals using different plasticity markers? To answer this question, it will be necessary to measure all three kinds of plasticity indicators in the same investigation, thereby affording determination of whether they are correlated and whether they redundantly— or uniquely and independently—moderate environmental influences on development.

### **THE “NURTURE” OF PLASTICITY**

Despite evidence suggesting that plasticity may be a function of genetics rather than experience, a growing body of theory and evidence raises the prospect that plasticity may be a function of nurture as well as nature (Boyce & Ellis, 2005). Indeed, there is now extensive work showing that very early experiences occurring in the womb can affect later development, most notably disease susceptibility (i.e., metabolic diseases) in middle age (Gluckman & Hanson, 2005). Of particular interest to psychologists, though, will be evidence documenting adverse effects of negative

fetal experiences on problem behavior in childhood. For example, maternal anxiety during late pregnancy predicts behavioral/emotional problems at age 7, even with postnatal anxiety and depression controlled (Huizink, de Medina, Mulder, Visser, & Buitelaar, 2002; see also O'Connor, Heron, Golding, Beveridge, & Glover, 2002; O'Connor, Heron, Golding, & Glover, 2003).

Even more central to our argument that much remains to be learned about plasticity in early human development is the evidence that such "fetal programming" may shape several of the very susceptibility factors mentioned above. Consider in this regard research showing (a) that maternal stress during pregnancy predicts difficult temperament at 3 months of age (Huizink et al., 2002) and emotional reactivity to novelty in 4-month-olds (Möhler, Parzer, Brunner, Wiebel, & Resch, 2006), (b) that prenatal maternal depression and elevated cortisol levels in late pregnancy predict negative reactivity at age 2 (Davis et al., 2007), and (c) that maternal prenatal anxiety predicts awakening cortisol in 10-year-olds (O'Connor et al., 2005).

On one hand, such data suggest that, in addition to genetics, very early experience in the womb may shape plasticity, as these outcomes are among the very characteristics found in work cited above to demarcate heightened susceptibility to environmental influences. Just as important, this reinterpretation of putatively negative effects of prenatal stress raises fundamental questions about the problem-focused perspective that pervades virtually all research and theory on prenatal programming: Is it the case that prenatal stressors compromise later development, as prevailing thinking presumes, or do these prenatal experiences promote plasticity and thus the organism's openness to future experiential inputs, be they positive or negative? That is, is there fetal programming of postnatal programming? Oberlander et al. (2008) recent epigenetic findings showing that maternal depressed mood in

pregnancy predicts increased methylation of the human glucocorticoid receptor gene (*NR3C1*, measured in neonatal cord blood), which itself forecasts elevated cortisol stress reactivity at 3 months of age, illuminates at least one biological mechanism that may be central to such fetal programming of postnatal plasticity. Also, recall that cortisol reactivity may well demarcate heightened susceptibility to rearing influences.

Before concluding on the basis of fetal programming research that plasticity is a function of experience as much as a function of genetics, we should not lose sight of the fact that the  $G \times E$  interaction may characterize the fetal programming process (Gluckman & Hanson, 2005). This raises the following unanswered question: Are some fetuses more susceptible to fetal programming than others, for genetic reasons? If they are and as of yet we simply do not know it would suggest that plasticity is a function not just of nature or nurture, but that some individuals may be more likely than others to be affected by experience, most notably perhaps, fetal experience, in ways that subsequently affect whether or to what degree they will be influenced by the postnatal world they encounter. By incorporating molecular-genetic measurements into fetal programming studies, it should prove possible to illuminate the issue of the  $G \times E$  interaction in this fast developing arena of inquiry.

### CONCLUSION

The very possibility that individuals may either vary in their plasticity for genetic reasons or that they may vary in the degree to which their plasticity is subject to fetal programming for genetic reasons raises even more interesting issues, especially those pertaining to population genetics that also merit investigation: Do populations differ in the degree to which children are malleable? If we consider for a moment the fact that selection for plasticity only makes evolutionary sense if what happens at one point in time is systematically related to what happens at a later point

in time because there would be no fitness payoff adjusting future functioning based on early-life experience if the future was not systematically related to the past the possibility emerges that human populations may vary in the degree to which children are malleable. Future research can thus address whether in ecological niches in which the present and future are (or have been) more related, the payoff for plasticity could well be greater, with greater selection for plasticity in human populations.

Three observations seem noteworthy in this context. First, the 7R-DRD4 allele discussed above as a possible plasticity marker not only recently emerged in human populations (~40,000 years ago), but varies substantially across them, having an extremely low incidence in Asia yet a high frequency in the Americas (Ding et al., 2002); intriguingly, the reverse is true of another possible plasticity gene: the s/s allele of 5-HTTP (Kim et al., 2007). Second, one wild bird population shows evidence that selection favoring individuals who are highly plastic with regard to the timing of reproduction has intensified over the past three decades, perhaps in response to climate change causing a mismatch between the breeding times of the birds and their caterpillar prey (Nussey, Postma, Gienapp, & Visser, 2005). Finally, Suomi (2006) has observed that only two species of primates, humans and rhesus macaques, fill diverse ecological niches around the world and that the presence of the 5-HTTP short (plasticity?) allele in some individuals distinguishes these two weed species from all other primates.

Whether it makes sense to conceptualize the 5-HTTP short allele or the 7R-DRD4 allele as plasticity genes, or whether human populations vary in the degree to which children are malleable, either as a result of the presence of these alleles or some other characteristics, remain to be determined. The same is true with regard to whether unborn children vary for genetic reasons in their susceptibility to fetal

programming with respect to plasticity in response to developmental experiences after birth. The bottom line is that much remains unknown about the nature of plasticity in early development.

One thing should be clear, though, given what has already been said about the study of resilience on the one hand and of fetal programming on the other and thus about so much of developmental psychology: So long as disproportionate emphasis is placed on the adverse effects of negative experience on problems in development, we risk misunderstanding the process of human development. If the very children found to be especially vulnerable to adversity are also, as theory and evidence is beginning to suggest, disproportionately susceptible to the beneficial effects of positive experiences, then focusing principally on the former could obscure as much as it illuminates the nature of human plasticity. And if prenatal stress does not just foster difficult temperament or physiological reactivity or even behavior problems, but regulates susceptibility to postnatal experience (i.e., plasticity), perhaps via methylation-related epigenetic means, then our obsession with disturbances in development will lead us to misconstrue how development operates. Each of these errors based on an all-too-great emphasis in psychology on poor mental health and problems in development would affect not just how we think about and study human development, but how we seek to promote it. And that is because knowledge of plasticity should pave the way for the most effective interventions, be they seeking to prevent problems before they develop, remediate them once they have emerged, or promote well-being without concern for developmental risks.

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## REFERENCES

- Auerbach, J.G., Faroy, M., Ebstein, R., Kahana, M., & Levine, J. (2001). The association of the dopamine D4 receptor gene (DRD4) and the serotonin transporter promoter gene (5-HTTLPR) with temperament in 12-month-old infants. *Journal of Child Psychology & Psychiatry, 42*, 777-783.
- 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.
- Bakermans-Kranenburg, M.J., & van Ijzendoorn, M.H. (2007). Research review: Genetic vulnerability or differential susceptibility in child development. The case of attachment. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 48*, 1160-1173.
- Bashey, F. (2006). Cross-generational environmental effects and the evolution of offspring size in the Trinidadian guppy *Poecilia reticulata*. *Evolution: International Journal of Organic Evolution, 60*, 348-361.
- Belsky, J. (1997). Variation in susceptibility to rearing influences: An evolutionary argument. *Psychological Inquiry, 8*, 182-186.
- 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. 1396-163). New York: Guilford.

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.

Belsky, J., Fearon, R.M.P., & Bell, B. (2007). Parenting, attention and externalizing problems: Testing mediation longitudinally, repeatedly and reciprocally. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 48*, 1233-1242.

Boyce, W.T., Chesney, M., Alkon, A., Tschann, J.M., Adams, S., Chesterman, B., et al. (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.

Boyce, W.T., Essex, M.J., Alkon, A., Goldsmith, H.H., Kraemer, H.C., & Kupfer, D.J. (2006). Early father involvement moderates biobehavioral susceptibility to mental health problems in middle childhood. *Journal of the American Academy of Child and Adolescent Psychiatry, 45*, 1510-1520.

Bradley, R.H., & Corwyn, R.F. (2008). Infant temperament, parenting, and externalizing behavior in first grade: A test of the differential susceptibility hypothesis. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 49*, 1246-131.

- Caspi, A., McClay, J., Moffitt, T.E., Mill, J., Martin, J., Craig, I.W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*, 8516854.
- Caspi, A., Sugden, K., Moffitt, T.E., Taylor, A., Craig, I.W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, *301*, 3866389.
- Cicchetti, D., & Rogosch, F.A. (1997). The role of self-organization in the promotion of resilience in maltreated children. *Development and Psychopathology*, *9*, 7976815.
- Davis, E.P., Glynn, L.M., Schetter, C.D., Hobel, C., Chicz-Demet, A., & Sandman, C.A. (2007). Prenatal exposure to maternal depression and cortisol influences infant temperament. *Journal of the American Academy of Child and Adolescent Psychiatry*, *46*, 7376746.
- Ding, Y., Chi, H., Grady, D.L., Morishima, A., Kidd, J., Kidd, K.K., et al. (2002). Evidence of positive selection acting at the human dopamine DF gene locus. *Proceeding of the National Academy of Sciences, USA*, *99*, 3096314.
- Gannon, L., Banks, J., Shelton, D., & Luchetta, T. (1989). The mediating effects of psychophysiological reactivity and recovery on the relationship between environmental stress and illness. *Journal of Psychosomatic Research*, *33*, 1676175.
- Gluckman, P., & Hanson, M. (2005). *The fetal matrix: Evolution, development and disease*. Cambridge, United Kingdom: Cambridge University Press.
- 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*, 8476851.

- Gunnar, M.R., & van Dulmen, M.H. (2007). Behavior problems in postinstitutionalized internationally adopted children. *Development and Psychopathology, 19*, 1296-148.
- Huizink, A.C., de Medina, P.G., Mulder, E.J., Visser, G.H., & Buitelaar, J.K. (2002). Psychological measures of prenatal stress as predictors of infant temperament. *Journal of the American Academy of Child and Adolescent Psychiatry, 41*, 1078-1085.
- Kim, J.M., Stewart, R., Kim, S.W., Yang, S.J., Shin, I.S., Kim, Y.H., et al. (2007). Interactions between life stressors and susceptibility genes (5-HTTLPR and BDNF) on depression in Korean elders. *Biological Psychiatry, 62*, 423-428.
- Kochanska, G., Aksan, N., & Joy, M.E. (2007). Children's fearfulness as a moderator of parenting in early socialization: Two longitudinal studies. *Developmental Psychology, 43*, 222-237.
- Möhler, E., Parzer, P., Brunner, R., Wiebel, A., & Resch, F. (2006). Emotional stress in pregnancy predicts human infant reactivity. *Early Human Development, 82*, 731-737.
- 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.
- National Institute of Child Health and Human Development 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: Guilford.
- National Institute of Child Health and Human Development Early Child Care Research Network. (2006). Child-care effect sizes for the NICHD Study of

Early Child Care and Youth Development. *American Psychologist*, *61*, 996-1016.

Nussey, D.H., Postma, E., Gienapp, P., & Visser, M.E. (2005). Selection on heritable phenotypic plasticity in a wild bird population. *Science*, *310*, 3046-3066.

Oberlander, T.F., Weinberg, J., Papsdorf, M., Grunau, R., Misri, S., & Devlin, A.M. (2008). Prenatal exposure to maternal depression, neonatal methylation of human glucocorticoid receptor gene (NR3C1) and infant cortisol stress responses. *Epigenetics*, *3*, 976-1006.

O'Connor, T.G., Ben-Shlomo, Y., Heron, J., Golding, J., Adams, D., & Glover, V. (2005). Prenatal anxiety predicts individual differences in cortisol in pre-adolescent children. *Biological Psychiatry*, *58*, 2116-2127.

O'Connor, T.G., Heron, J., Golding, J., Beveridge, M., & Glover, V. (2002). Maternal antenatal anxiety and children's behavioural/emotional problems at 4 years. Report from the Avon Longitudinal Study of Parents and Children. *British Journal of Psychiatry*, *180*, 502-508.

O'Connor, T.G., Heron, J., Golding, J., & Glover, V. (2003). Maternal antenatal anxiety and behavioural/emotional problems in children: A test of a programming hypothesis. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *44*, 1025-1036.

Olds, D.L., Kitzman, H., Cole, R., Robinson, J., Sidora, K., Luckey, D.W., et al. (2004). Effects of nurse home-visiting on maternal life course and child development: Age 6 follow-up results of a randomized trial. *Pediatrics*, *114*, 1550-1559.

Philipi, T., & Seger, J.H. (1989). Hedging evolutionary bets, revisited. *Trends in Ecology & Evolution*, *4*, 416-444.

- Pigliucci, M. (2007). Do we need an extended evolutionary synthesis? *Evolution: International Journal of Organic Evolution*, *61*, 2743-2749.
- Pluess, M., & Belsky, J. (2009). Differential susceptibility to rearing experience: The case of childcare. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *50*, 396-404.
- Sameroff, A.J. (1983). Developmental systems: Contexts and evolution. In P. Mussen (Ed.), *Handbook of child psychology* (Vol. 1, pp. 237-294). New York: Wiley.
- Sinn, D.L., Gosling, S.D., & Moltischniowskyj, N.A. (2007). Development of shy/bold behaviour in squid: Context-specific phenotypes associated with developmental plasticity. *Animal Behaviour*, *75*, 433-442.
- Suomi, S.J. (2006). Risk, resilience, and Gene  $\times$  Environment interactions in rhesus monkeys. *Annals of the New York Academy of Sciences*, *1094*, 52-62.
- 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.
- Werner, E.E. (1997). Vulnerable but invincible: High-risk children from birth to adulthood. *Acta Paediatrica*(Suppl.), *422*, 103-105.
- Wilhelm, K., Mitchell, P.B., Niven, H., Finch, A., Wedgwood, L., Scimone, A., et al. (2006). Life events, first depression onset and the serotonin transporter gene. *British Journal of Psychiatry*, *188*, 210-215.
- Zuckerman, M. (1999). *Vulnerability to psychopathology: A biosocial model*. Washington, DC: American Psychological Association.