Differential Susceptibility to Parenting, Peers and Early-Life Threat and Deprivation: Domain Specificity or Domain Generality

By

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To my son,

Parker Reid Armstrong

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Abstract

It is widely appreciated that individuals vary in the extent to which developmental experiences and exposures shape their development. Individual differences in developmental plasticity have been highlighted in at least two different conceptual models—the diathesis-stress/dual-risk model which suggests that some individuals are more vulnerable to the negative effects of contextual adversity (Monroe & Simmons, 1991; Zuckerman, 1999)—and the differential susceptibility model which stipulates that individuals are developmentally plastic, and therefore vary more generally in their susceptibility to environmental effects both positive and negative (Belsky, Bakermans-Kranenburg & van Ijzendoorn, 2007; Ellis et al., 2011). Although much of the research on developmental plasticity is often cast in trait-like terms (Boyce & Ellis, 2005; Aron & Aron, 1997), there is also evidence that suggests otherwise (Belsky & Beaver, 2011; Belsky et al., 2021; Zhang, Widamen, & Belsky, 2021). Moreover, what remains to be further investigated is whether differential susceptibility is domain-general (i.e., trait-like) or domain-specific (Belsky et al., 2021).

The current dissertation extends research on differential susceptibility in terms of domain-general versus domain-specific developmental plasticity by exploring susceptibility to (a) parenting and peers, and (b) early-life adversity in the form of threat and deprivation. Paper 1 examines the interrelation of individual differences in susceptibility to parenting and peer effects. Paper 2 does the same with respect to effects of deprivation and threat in early life. Overall, this dissertation provides further evidence for individual differences in terms of domain-general versus domain-specific developmental plasticity.

Differential Susceptibility to Parenting, Peers and Early-Life Threat and Deprivation: Domain-Specificity or Domain-Generality?

How individuals vary in the extent to which developmental experiences and environmental exposures shape their development is a subject that has been long appreciated within the field of developmental psychology. Notably, individual differences in developmental plasticity have been highlighted in person-X-environment interaction research, typically guided by one of two different conceptual models: the diathesis-stress/dual risk model—which suggests that some individuals are more vulnerable than others to negative effects of contextual adversity (Monroe & Simmons, 1991; Zuckerman, 1999), and the differential susceptibility model—which stipulates that individuals vary more generally in their susceptibility to environmental effects both negative and positive (Belsky, Bakermans-Kranenburg & van Ijzendoorn, 2007; Ellis et al., 2011).

Empirical evidence in support of the differential susceptibility model comes from observational studies of gene X environment (G x E) interaction (for review, see Belsky & Pluess, 2009, 2013) and from experimental research using measured genes as moderators of intervention efficacy (for review, see Belsky & van Ijzendoorn, 2017). Despite being repeatedly noted in the above-cited work, what remains to be investigated is whether such differential susceptibility to environmental influence is domain-general (i.e., trait-like) or domain-specific (Belsky et al., 2021). In other words, are the same children affected more than others by different experiences and exposures?

Developmental plasticity is often characterized in domain-general terms, implying that individuals are more or less susceptible to a multitude of different experiences and exposures (e.g., parenting, peers, community) that influence many aspects of their development (e.g.,

social-emotional, cognitive). For instance, Aron and Aron's (1997) notion of the "sensitive" person calls attention to general variation in sensitivity to environmental influences and Boyce and Ellis' (2005) theory of biological sensitivity to context describes some children as highly susceptible "orchids" and others who are not as "dandelions". This trait-like characterization of developmental plasticity seems to imply that two types of people exist—those who are highly susceptible—and those who are less susceptible.

Yet, there is empirical evidence suggesting otherwise. Belsky and Beaver (2011) first chronicled a developmental plasticity gradient such that the more putative plasticity alleles adolescents carried, the more susceptible they proved to be to the effects of parenting on self-regulation. Numerous additional investigations of G x E interactions also revealed evidence of a similar continuum of developmental plasticity using different sets of genes (e.g., Gibbons et al., 2012; Simons et al., 2011). Moreover, similar evidence has emerged in studies focused on other plasticity factors (e.g., infant temperament, physiological reactivity; Rubinow et al., 2020; Tabachnich et al., 2021; Tstotsi et al., 2018).

Most of the differential susceptibility research just discussed has involved exploratory tests of Person x Environment interactions. These investigations generally utilize a genetic plasticity factor (e.g., a candidate gene or polygenic score) to determine whether it moderates the effect of an environmental factor on some psychological or behavioral outcome. If an interaction proves significant, it is decomposed to determine if its form is consistent with a hypothesized model of Person x Environment interaction (e.g., diathesis-stress). This is required because it cannot be presumed that any interaction detected in exploratory testing reflects differential susceptibility.

Recent inquiry, however, benefitted from utilizing a different approach based on influence statistics—to measure individual differences in susceptibility to specific environmental effects on predetermined developmental outcomes. Unlike previous work with Person X Environment interactions, influence statistics illuminate individuals within a sample that are more or less responsible for the estimate of any detected association of interest when using the entire sample (e.g., Belsey, Kuh & Welsch, 1980; Cook & Weisberg, 1982). Typically, this is accomplished by a "leave-one-out approach", in which the association in question is re-run repeatedly each time dropping a single case to see whether such (minor) sample modifications result in the association increasing (i.e., negative influencer) or decreasing (i.e., a positive influencer) modestly. In other words, the more the predictor-outcome association becomes less positive when a case is dropped, the more susceptible the child is to the effect in question. Conversely, the more the association becomes more positive when a case is dropped, the less susceptible the child is to the effect.

Work utilizing this novel influence statistics approach revealed that children whose cognitive-linguistic development proved most or least affected by the quality of childcare they experienced were generally not the same as those whose behavior problems proved most or least affected by the quantity (or dosage) of care they experienced (Belsky, Zhang & Sayler, 2021). Further inquiry by Zhang and colleagues (2021) using the same NICHD Study of Early Child Care and Youth Development sample also revealed that results across the environment-predictor/ child-outcome associations were normally, not bimodally (i.e., orchids/dandelions) distributed. Furthermore, children who as infants had difficult temperaments or those who scored higher on a polygenic plasticity score (5-HTTLPR, DRD4, BDNF) proved somewhat more susceptible to

some of the environmental effects investigated (e.g., familial and childcare) (Zhang, Widamen & Belsky, 2021).

The following two papers presented herein extend research on differential susceptibility in terms of domain-general versus domain-specific developmental plasticity by exploring susceptibility to parenting and to peers, and to early-life threat and deprivation. The first paper of my dissertation explores susceptibility to parenting and peer effects by addressing the extent to which individuals whose adolescent adjustment is most and least associated with the parenting they experienced is similarly or differentially associated with their experience with agemates. To achieve these goals, the influence statistic used in the previously cited work, DFBETAS, is used again to analyze data from the longitudinal NICHD study of Early Child Care and Youth Development (NICHD, $N = 1{,}364$).

The domain-general view of developmental plasticity would seem to conflict with current neurobiological thinking—which connects different contextual experiences with different brain processes and thereby, different phenotypes (e.g., McLaughlin, Sheridan, & Lamber, 2014). In this dissertation I will address this subject by investigating whether children vary in their susceptibility to experiences of deprivation and threat—which have been each shown to have distinct influences on children's emotional and cognitive development (McLaughlin & Sheridan, 2016). Therefore, in the second paper, the primary goal is to assess the extent to which individuals whose development—specifically their cognitive and social-emotional development—is most and least affected by different adverse developmental experiences (e.g., threat and deprivation). To achieve this, an influence statistic DFBETAS is used to analyze data from the ongoing Avon Longitudinal Study of Parents and Children (ALSPAC, $N = 14{,}541$).

Notably, this second study was pre-registered. In conclusion, a short summary describing the continuity of research between the two papers will follow.

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Paper 1

Parenting and Peer Effects on Psychosocial Adjustment:

Are the Same—or—Different—Children Affected Most or Least by Each?

Abstract

The prospective research presented herein extends work on parent and peer effects on adolescent psychosocial adjustment by looking beyond average effects. Instead, it considers variation in susceptibility to each source of influence in order to assess the extent to which those individuals most and least susceptible to parent effects are similarly—or differentially—susceptible to peer effects. Data from the NICHD Study of Early Child Care and Youth Development (n = 1364, 48.3% female, age range: 10–15 yrs., race/ethnicity: 80.4% white, 12.9% black and 6.7% other) was analyzed to assess the degree to which each child was susceptible to parent and peer effects. Toward this end, an influence statistic, DFBETAS, was used. A significant and moderate positive association between the two susceptibility effects indicated that children most or least susceptible to the effects of quality of parenting proved to be similarly affected by their peers, although this was not the case for a substantial minority of youth. The fact that at least some children vary dramatically in the effects to which they are most susceptible is discussed with respect to potential targets of intervention, namely, parents or peers.
Keywords: peer relations, parenting, differential susceptibility, psychosocial adjustment

Parenting, Peers, and Psychosocial Adjustment: Are the Same—or—Different—Children Affected by Each?

A primary focus of developmental science concerns the influence of early-life experiences and exposures on human development. It is well appreciated that individual differences in development are multiply determined, including genetic, physiological, and environmental sources of influence. Two well-studied influential contexts include the family and the world of peers. The prospective research presented herein extends research on differential susceptibility by looking beyond average effects to consider individual differences in susceptibility to each source of influence (e.g., parenting and peers). Therefore, the primary goal is to assess the extent to which individuals whose adolescent adjustment is most and least associated with the parenting they experienced is similarly—or differentially—associated with their experience with agemates. To achieve these goals an influence statistic DFBETAS, is used to analyze data from longitudinal NICHD Study of Early Child Care and Youth Development (*n* = 1,364). Throughout this report, terminology regarding parent and peer "effects" refer to statistical effects. These effects are not necessarily documented causal ones because this is an observational study and the analyses presented herein do not account for genetic and other potential confounders which would need to be discounted to chronicle true environmental causation. Therefore, within this paper terms like "putative influence" and "apparent influence" will be used.

Parent and Peer Effects

How differences in parenting quality (Darling & Steinberg, 1993), as well as experiences and relations with peers (Giletta et al., 2021) might influence child and

adolescent development has been the subject of inquiry for more than half a century (Bronfenbrenner, 1970). Regarding parenting, cross cultural evidence indicates that many parenting effects are similar around the world (Lansford et al., 2018)—and not just among Western societies (i.e., Asia, Africa)—including the adverse effects of low warmth and high control on externalizing problems (Rothenberg et al., 2020). Considering the context of the current inquiry, the latter is also true in the USA with both maternal and paternal unsupportive parenting predicting problematic development (Davies et al., 2021), and with conflicted-coercive mother-child interaction in childhood predicting adolescent antisocial behavior (Compton et al., 2003). There is also evidence that hostile or distant (Griffith et al., 2019) or overcontrolling (Rogers et al., 2020) parenting is associated with adolescent internalizing problems. Moreover, excessively harsh, permissive, or neglectful parenting is also linked to delinquency (Allen et al., 2019) and substance use (Neppl et al., 2016).

In the case of peer effects, adolescent risk taking, risk preference and risky decision making are systematically associated with peer experiences (Gardner & Steinberg, 2005). In fact, even when the probabilities of negative consequences are known the presence of peers increases adolescent risk taking (Smith et al., 2014). Adolescents highly sensitive to peer pressure are also more likely to engage in risk-taking and less likely to become socially competent (Allen et al., 2006). Even adolescent's perceptions of friend's risk-taking behavior can strongly influence their own (Young et al., 2014). Consider, for example, evidence that perceptions of peers' engagement in risky sexual behavior (e.g., not using a condom) predicts one's own sexual risk taking (Henry et al., 2007). According to many prospective studies, experiences with peers are associated with a wide range of problematic psychological outcomes in adolescence—including externalizing behavior

(e.g., aggression, delinquency; Rulison et al., 2013), internalizing symptoms (e.g., anxiety, depression; Troop-Gordon et al., 2019) and a diverse array of health risk behaviors (for review see Prinstein & Giletta, 2016). Therefore, it is not surprising that being part of a peer group that engages in delinquent acts predicts an individual's likelihood to do as well (Reynolds & Crea, 2015).

Importantly, the world of parent-child and peer relations are by no means independent, and this includes their documented effects. Consider recent work showing that parenting experienced in early childhood predicts neural responses to simulated peer rejection and adolescent-reported peer stress (Kujawa et al., 2020). There is also evidence that peer effects may be part of a developmental process by which parenting shapes development, from inquiry testing the proposition that peer acceptance in adolescence mediates effects of early harsh and manipulative parenting on psychological and behavioral development at age 32 (Loeb et al., 2021). Parent and peer effects can also moderate one another in other interactive ways including the two effects amplifying (e.g., Miklikowska et al., 2019) or mitigating each other (Hazel et al., 2014).

Differential Susceptibility to Environmental Influences

Much reporting of findings like those just summarized, perhaps particularly in the popular press, can often lead to the impression that any and all children exposed to the kinds of parent and peer experiences just highlighted have their development influenced in the manner implied. Nevertheless, most scholars appreciate that effects of almost all putative sources of influence—be they environmental, physiological, or genetic—reported in the scholarly literature represent average effects. In other words, there is typically variation in susceptibility even when effect sizes are statistically significant, even substantial. Such variation in susceptibility is a

subject that has a long history, often conceptualized in terms of person-X-environment interaction, with work focused on topics like whether and how child temperament interacts with parenting (Slagt, Dubas, Dekovic & van Aken, 2016) or genes interact with a variety of contextual conditions to account for variation in child, adolescent, and even adult development (Moffitt, Caspi & Rutter, 2005).

In recent years much of this work has been conceptualized in terms of "differential susceptibility to environmental influence" rather than "vulnerability to adversity"; this is because the focus has turned from a near-exclusive concern with who is vulnerable to the negative effects of adverse developmental conditions (as well as who proves resilient) to interest in variation in sensitivity to both supportive contextual conditions (e.g., sensitive parenting) and unsupportive ones (e.g., harsh parenting). In its original formulation, the differential susceptibility framework stipulated that some individuals are more generally developmentally plastic—and thus especially susceptible to both positive and negative environmental influences—whereas others are not or at least much less so (Belsky, Bakermans-Kranenburg & van Ijzendoorn, 2007). Children whose development was more and less shaped by their developmental experiences and environmental exposures were labeled, respectively, "orchids" and "dandelions" by one scholarly team (Boyce & Ellis, 2005) and "doves" and "hawks" by another (Suor, Sturge-Apple, Davies & Cicchetti, 2017).

Evolutionary analysis provided a theoretical foundation for theorizing about individual differences in developmental plasticity (Ellis, Boyce, Belsky, Bakerman-Kranenburg & van Ijzendoorn, 2011). This foundation was based, in part, on the developmental implications of the future being inherently uncertain (Belsky & Pluess, 2009). More specifically, because there could always be and presumably has been some, if not a great deal of, mismatch between the

environment in which children grow up—which is presumed to prepare them for the future—and the one they actually experience as adults, having later development shaped by earlier experiences could undermine, rather than facilitate, the ultimate goal of passing on genes to future generations. Thus, the theoretical presumption is that nature essentially "hedges its bets", with Darwinian natural selection resulting in human genetic variation making some children more susceptible to developmental influence and others less so.

Domain-General or Domain-Specific Susceptibility?

Theorists advancing these ideas wondered whether conceptualizing variation in developmental plasticity in trait-like and typological terms was most appropriate (Belsky & Pluess, 2013). Rather than such variation being domain-general, it has been proposed that a domain-specific alternative merits consideration (Zhang, Widaman & Belsky, 2021). After all, would it not be more likely, perhaps for neurobiological-sensitivity reasons, that individuals varied in terms of the particular experiences that most influenced their functioning rather than some being highly susceptible and others not or less so to a wide variety of developmental experiences and exposures? Might, for example, some children be very developmentally plastic when it came to effects of their families' musical environments, but not particular affected by their literacy environments, with the opposite being true of other children?

This distinction between a domain-general and a domain-specific view of developmental plasticity would seem to carry implications for efforts to provide services and interventions to children, be they directed at preventing or remediating problems or promoting well-being. If interventions were matched to the experiences and exposures to which children were especially sensitive, it seems likely that their efficacy would be enhanced. This, of course, is exactly the rationale for "personalized medicine."

Recent research has provided some empirical support for the domain-specific claim. In a proof-of-principle study that used an influence statistic to assess individual differences in children's susceptibility to two well-documented effects of child care—better quality of care predicting enhanced cognitive-linguistic development and greater quantity of care predicting more problem behavior (NICHD Early Child Care Research Network, 2002)—investigators evaluated whether the same children proved highly susceptible to both of these effects (Belsky, Zhang & Sayler, 2021). Notably, results revealed that this was generally not the case. Follow-up work using the same influence-statistic approach examined effects of multiple features of families (e.g., supportive parenting, economic resources, maternal depression) and of child care (quality, quantity, type) on a more extensive set of developmental outcomes (e.g., behavior problems, social competence, short-term memory) (Zhang et al., 2022). Not only did variation in overall susceptibility to eight environmental factors vis-a-vis five developmental outcomes prove to be distributed normally rather than bimodally (i.e., orchids-dandelions), but evidence also indicated that the more susceptible children were to the beneficial effects of family support and resources on social-behavioral development, the less susceptible they were to the detrimental effect of lots of time in care on cognitive competence.

The research reported in the present report seeks to extend such work by evaluating whether adolescents whose adjustment proves to be strongly or weakly associated with the parenting they experienced look similarly susceptible—or not—to effects of peers. At least one reason to entertain the possibility that different children's adolescent adjustment might be differentially susceptible to parent and peer effects is because the psychological dynamics of parent-child and peer relations are quite different. After all, popularity, attraction, and competition all involve dynamics that are not as central to parent-child relations as they are in the

world of peers. In sum, developmental scholars have long acknowledged that children are influenced by both parents and peers, but the ways in which parents influence their children (e.g., through disciplinary practices) are not the same as the ways in which peers influence each other (e.g., by establishing norms that are enforced through social acceptance or rejection).

In line with this analysis of differential parent-child and peer relational dynamics is a recent study that queried 11-year-old Israeli adolescent twins about their sensitivity to peer and parent effects (Markovitch & Knafo-Noam, 2020). Results indicated that teens highly sensitive to their parents are not necessarily highly sensitive to their peers. In fact, sensitivity to parents and to peers had different (genetic) etiologies, a finding consistent with the aforementioned evolutionary analysis of why it might be that children vary in their relative susceptibility to parent and peer effects. Importantly, to the extent that susceptibility varies within and across individuals in terms of which experiences they are most affected by, it could have implications, noted earlier, for intervention. It is certainly imaginable that whereas a focus on parents might be the most appropriate target for children highly susceptible to parenting effects, a focus on peers might be most appropriate for those highly sensitive to the influence of their agemates.

Puberty and Peer Influence

It is now well-established that adolescence is a period of heightened sensitivity to social contextual influences, particularly with peers (Blakemore & Mills, 2014). During puberty, the brain undergoes fundamental alterations including a phase of synaptic pruning, volumetric changes, extensive myelination, and changes to excitatory and inhibitory inputs (Monahan et al., 2015) which result in increased sensitivity to social stimuli (Blakemore & Mills, 2014). The extant literature on the relationship between puberty and brain plasticity indicates that in adolescence pubertal hormones play an essential role in regulating the mechanisms of

experience-dependent plasticity (Laube, van den Bos & Fandakova, 2020). Hence, it seems plausible that brain-based individual differences resulting from depending on age and pubertal timing could affect sensitivity to social contexts—such as those created through relationships with peers and with parents (Schriber & Guyer, 2016).

A substantial body of research has examined whether pubertal development is associated with developmental outcomes (for review see Vijayakumar et al., 2018). Adolescents whose pubertal development occurs earlier than their peers display greater reactivity to emotionally salient information (Ladouceur, 2012) and elevated levels of externalizing behavior (e.g., rule-breaking or delinquency) (Smith-Woolley, Rimfield, & Plomin, 2017). Consider also work by Willoughby and colleagues (2021) which revealed that early to mid-adolescents (including individuals in mid-to-late puberty) were more sensitive to a peer observation task (e.g., Balloon Analogue Risk Task) and displayed larger neural responses to loss-feedback in the peer condition than the alone condition. In contrast, children (and individuals in pre-early puberty) were unaffected by peer observation—displaying no difference in neural response to both peer and alone conditions. Therefore, pubertal development may be an important factor to consider when evaluating the extent to which peer influences affect adolescent adjustment and as such, it is incorporated into our current study.

Current Study

In sum, this study is designed to determine whether the degree to which children's susceptibility to independently measured parent and peer effects proves similar or different, a subject to our knowledge which has never before been addressed empirically. Using the aforementioned influence statistic to measure individual differences in susceptibility to both

parent and peer effects (DFBETAS), the work presented treats "persons as effects sizes" (Grice et al., 2020). Following this, if distinct susceptibility groups emerge, I will further examine whether pubertal maturation is associated with variation in susceptibility to peer influence.

With respect to predictions, it would seem reasonable to hypothesize that children who are more or less susceptible to putative parental influence would be somewhat similarly susceptible, on average, to that of their peers—given that these social worlds tend to be positively related and that each is also predictive of children's psychosocial adjustment (Collins et al., 2000). But because the focus here is on whether the same children are most or least susceptible to effects of parenting and of peers, there may be reason to temper this expectation. After all, in the vast literatures addressing the apparent influence of parents (e.g., Steinberg, 2001) and peers (e.g., Bechwald & Prinstein, 2011) on adolescent development, effects are rarely very large, just as in the case of so many developmental experiences and exposures. Lastly, regarding pubertal development, I predict that children with advanced pubertal development would be most affected by peer influence.

In this report, the measurement of potential peer influence is based on children's reports of the risky behavior of friends and their own resistance to peer influence, whereas measures of parenting are based on both observational data and self-reports of parenting and parent-child relations. This was considered an appropriate approach given evidence that "deviant" peer behavior can influence child and adolescent development (Haynie, 2002), just as parenting behavior and parent-child relations can (Collins et al., 2000). Even though the variables used to estimate parenting and peer effects are not the same, they are both of a kind that predict child and adolescent functioning and have been used in prior work, including direct comparison of parenting and peer effects (e.g., Trudeau, Mason, Randall, Spoth & Ralston, 2012) and indirect

pathways of apparent influence from parenting via peer relations (e.g., Defoe, Dubas, & van Aken, 2018).

Method

The methods for the paper "Parenting and Peer Effects: Are the Same—or Different—Children Affected by Each?", are delineated below, starting with the hypothesis, study population, measures, and data analyses.

Hypothesis

(1) There will be individual differences in the extent to which children are affected by their parents and peers, such that (a) while children most and least susceptible parent effects will generally be similarly susceptible to peer effects, (b) —at least some children most susceptible to one source of influence will be least susceptible to the other.

Population

Data for the proposed study will come from the NICHD Study of Early Child Care and Youth Development (NICHD Early Childcare Research Network, 2005). Families were recruited through hospital visits to mothers shortly after the birth of a child in 1991 in 10 locations in the U.S. During selected 24-hour intervals, 8, 986 women giving birth were screened for eligibility. From that group, 1,364 families (boys = 705; white = 1,097, black = 176; other = 91) completed a home interview when the infant was 1 month old and became the study participants. Details of the sampling plan can be found in NICHD ECCRN (2005). In terms of demographic characteristics at study enrollment, 26% of the mothers had no more than a high school education and 21% had incomes no greater than 200% of the poverty level.

Study children were followed from birth to 15 years of age. Data on demographic control variables (sex, race/ethnicity, maternal years of education, family income/needs) was obtained in the first 54 months of life and when children were in 5th grade (maternal depression, father presence). Parenting and peer predictors were collected when children were in 5th and 6th grade. Data on child socioemotional well-being was obtained in 5th grade and at age 15.

Measures

Predictors. Two sets of variables will serve as predictors, those pertaining to parenting and to peers. All variables will be scaled so that higher scores reflect more of the construct's (*italicized*) label.

Parenting. Five indices of parenting will be used. In 5th grade, mothers participated in a structured interaction with their child that included a discussion task and a shared problem-solving activity. Working from videotapes, highly trained coders rated (on 7-point scales) the 3 dimensions of parent behavior: supportive presence, respect for autonomy and hostility. A *maternal sensitivity* composite will be based on the mean of the three scales: supportive presence, respect for autonomy and hostility (reverse scored).

In 6th grade, mothers reported, using a 5-point rating adapted from the Student Teacher Relationship Scale (Pianta, 2001), on *closeness* with their child (e.g., warm relationship with child, child shares feelings and experiences; 7 items) and *conflict* (e.g., child is sneaky or manipulative, child's feelings toward mother are unpredictable; 6 items). Study children also reported on their relationship with mothers during a 6th grade lab visit using a 4-point rating scale. Questions assessed *maternal warmth* (e.g., cares about you, helps you with things that are important to you; 9 items) and *maternal hostility* (e.g., gets angry with you, criticizes you, insults or swears at you; 8 items).

Peers. Three indices of peer relations will be used. In 6th grade each child reported, using a 3-point rating, on their friends' engagement in "*major risk taking*" (e.g., carried a weapon, taken something worth a lot, purposely damaged property) and on their "*minor risk taking*" (e.g., ridden without a seatbelt, done something dangerous on a dare) using a questionnaire developed for the NICHD Study based on work by Conger and Elder (1994). The major-risk-taking scale will consist of 14 items and minor-risk taking 3 items. Children also reported on their own *resistance to peer pressure* using a modified version of the Steinberg and Monahan's (2007) Resistance to Peer Influence instrument. The questionnaire contains 9 items rated on a 4-point scale (e.g., It's pretty hard for my friends to get me to change my mind, I act the same way alone as I do with friend).

Pubertal Development. Two indices of pubertal development taken at 4th, 5th, 6th, 7th, and 8th grade assessments will be used. These indices were based on nurse reports of age of menarche and annual physical exams of pubertal status using Tanner Criteria (Marshall & Tanner, 1969, 1970).

15-Year Outcomes. Six indices of adolescent social-behavioral functioning at age 15 will be used. Mothers reported on their teen's *peer competence* using the Social Skills Rating System (SSRS). This subscale consists of 11 items (e.g., controls temper when arguing with other children, follows rules when playing games with others). Mothers also reported on their teen's *externalizing behavior* using the Child Behavior Checklist (CBCL; Achenbach, 1991). A 33-item standardized externalizing t-score will be created based on the delinquent and aggressive behavior syndrome subscales (e.g., cruelty, bullying or meanness to others, argues a lot, doesn't feel guilty after misbehavior).

Adolescents reported on their own *externalizing behavior* using the Youth Self-Report (YSR) scale. A 30-item standardized externalizing t-score will be created from delinquent and aggressive behavior syndrome scales (e.g., I argue a lot, I am mean to others, I don't feel guilty after I do something I shouldn't). They also reported on their own *lack of impulse control*, using 7 of the 8 items of this subscale of the Weinberger Adjustment Inventory (WAI; Weinberger & Swartz, 1990; e.g., I'm the kind of person who will try anything even if it is not safe, I do things without giving it much thought, I do something fun but tend to go too far). Teens also self-reported *sexual risk taking* and *non-sexual risk taking* using a 55-item questionnaire created for the NICHD Study. The adolescent was asked how many times in the past year they have engaged in 55 different risky behaviors. Two items will comprise the sexual risk-taking subscale (e.g., oral sex, sexual intercourse) and the remaining 53 the non-sexual risking taking subscale (e.g., ridden in car without a seatbelt, done something dangerous on a dare).

Covariates. Two sets of covariates will be measured, one set pertaining to child functioning in 5th grade and the other to demographic factors.

Child Control Variables. To afford prediction of change in child functioning at age 15, I will treat as covariates 7 measures of child behavior in 5th grade. Mothers reported their child's competent child functioning using the SSRS. A *social skills* total score will consist of 38 items (e.g., introduces him or herself to new people, is self-confident in social situations, politely refuses unreasonable requests). A *peer competence* score will be based on 10 items (e.g., joins group activities without being told, makes friends easily, controls temper when arguing with another child). A *disruptive behavior* score, adapted from the Diagnostic and Statistical Manual of Mental Disorders, fourth edition, will consist of 26 items (e.g., has difficulty playing quietly, has difficulty awaiting turn, is spiteful or mean).

Mothers also reported on their child's *minor risk-taking* behaviors using the same instrument already described and on child *externalizing behavior* using 33-item scale of the Child Behavior Checklist (CBCL; Achenbach, 1991; e.g., temper tantrums or hot temper, disobedient at home, argues a lot).

In 5th grade, teachers reported on the study child's *social-emotional well-being* using the Teacher Checklist on Peer Relations (Coie & Dodge, 1988) which assesses children's social skillfulness with peers. This subscale includes 7 items rated on a 5-point scale (e.g., skills understanding others feelings, generates good solutions to interpersonal problems, socially aware of what's happening). Teachers also completed a 7-item scale assessing *conflict with child* (e.g., child becomes easily angry with me, the child and I always seem to be struggling with each other, child remains angry/resistant after discipline).

Demographic Control Variables. To control for potentially confounding demographic factors, I will control for child sex, child race/ethnicity, maternal education, income to needs ratio and father presence in the home. Also, I plan to control for maternal depressive symptoms. *Mother's level of educ*ation (in years), the study children's *race/ethnicity* and *sex were* all collected at the 1-month interview. A mean *family income-to-needs* (ITN) ratio will be calculated from data gathered at 6, 15, 24, 36, and 54 months based on dividing total family income by the poverty threshold for family size (see NICHD ECCRN, 2002). Information about household composition was collected in 5th grade and *father presence in the home* indicates whether the child's father (adoptive or biological) resided full-time within the household. *Maternal depression* was also measured in 5th grade using the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977; e.g., felt sad, felt fearful, trouble keeping my mind on what I was doing).

Data Analyses

Preliminary analyses will set up the primary analyses addressing the issue of individual differences in susceptibility to parenting and peer effects.

Preliminary analyses

The first analytic step involved imputing missing data. Next, I conducted a data-reduction oriented, preliminary analysis using principal components analysis to reduce the number of constructs subject to analysis and thereby limit statistical testing in the primary analysis. More specifically, I separately factored parenting, peer influence and $5^{th}/6^{th}$ grade social and behavioral functioning variables. Based on these results, composite scores were created. The third step included a regression analysis to evaluate, separately (in two regression equations), the power of parenting or of peers to predict age-15 socioemotional well-being, net of all covariates.

Primary analyses

Primary analyses were carried out using influence statistics to assess the degree to which each child was susceptible to the parent and peer effects documented in the prior regression analyses so that the two resulting susceptibility scores can be associated with each other; this determined the extent to which children affected more or less by one experience were or were not affected by the other in a similar way.

Toward this end, I relied on an influence statistic, DFBETAS, a continuous and standardized index assigned to each and every observation; it reflects the *degree and direction* of change of the regression coefficient after removing an observation. The next step in the analysis involved determining whether children most and least affected by one of the effects under investigation were the same as the ones most and affected by the other. This included simply correlating the two susceptibility variables based on DFBETAS and cross-tabulating tercile splits

of the two DFBETAS' distributions to determine what percentage of children fall in the same tercile for both effects (i.e., high-high, moderate-moderate, low-low) and what percentage of children score high on susceptibility to one effect but low on the other (i.e., high-low). Finally, a one-way ANOVA was employed to test for significant mean differences in pubertal development among tercile peer influence susceptibility groups (e.g., low, moderate, and high).

Results

Preliminary Analyses

To reduce the number of dependent variables and thus multiple testing, the measures of parenting, peer relations, child socioemotional well-being in 5th grade and teen functioning were subjected to separate exploratory principal components analyses (see Table 1). Unit-weighted composite scores were created based only on the first factor because in all cases it accounted for the large majority of explained variance along with the largest eigenvalues, with other factors having eigenvalues equal to or greater than 1.0. In all cases, negative loading items were reverse scored before composite measures were created so that high scores on each composite represented more positive parent and peer relations and child functioning. The internal consistency of each composite was as follows: parenting (α = .70), peers (α = .62), child (5th grade) adjustment (α = .72) and adolescent psychosocial adjustment (α = .74).

Table 2 displays the inter-correlations of these composite measures. Recall that these correlations provide validation information on the factor-derived constructs specific to this investigation. Specifically, evidence of validity is based on whether the correlations reflect the following pattern of associations, as it turns out they do: both predictor constructs of parenting and of peer experiences are significantly and positively associated with one another and with

adolescent adjustment; and the latter composite is significantly and positively associated with the antecedent 5th-grade psychosocial-adjustment composite. Based on confirmation of the three validational hypotheses, primary analyses were undertaken. Results in Table 2 also suggest that pubertal development and susceptibility are related—with greater pubertal development resulting in more parental influence rather than peer influence, contrary to predictions.

The final step of the preliminary analyses involved evaluating, in two separate exploratory regression analyses which included the entire sample—and on which DFBETAS susceptibility scores would be based—whether parenting and, separately, peer experiences predicted adolescent adjustment after controlling for covariates, including the composite measure of psychosocial adjustment in 5th grade (see Table 3). As expected, results indicated that more supportive parenting predicted greater growth in socioemotional well-being (*beta* = .36, $p <$ .001), with the same being true of more positive peer relations (*beta* = .18, $p < .001$).

Primary Analyses

The first step in the primary analyses involved calculating DFBETAS scores for each of the two significant associations—representing the degree of susceptibility of each child to the effects of parenting and peers. Recall this was accomplished via the leave-one-out procedure and re-running the two whole-sample regression analyses 1,364 times, once using the parenting predictor and once the peer predictor. With individual susceptibility scores estimated for each case, the DFBETAS for parenting and peers were correlated. Recall that the prediction was that the association would be significant and positive but not necessarily very large. The correlation between the two influence-statistic-derived susceptibility scores proved consistent with this expectation: $r = .40$ ($p < .0001$). This result indicates that children who appeared to benefit the most from supportive parenting in terms of their socioemotional development were also most

likely to be positively affected by peers. Conversely, children who appeared to benefit the least from supportive parenting were also least likely to benefit from their peer relations.

Because the detected effect size was moderate, even if in the expected direction, exploratory efforts were undertaken to provide further descriptive insight into the association between susceptibility to the putative influence of parents and peers by, arbitrarily, dividing each DFBETAS' distribution in thirds and cross tabulating them. The DFBETAS mean (and range) of the top tercile (i.e., most susceptible) was, respectively, .02 (0.00 - .21) for parenting and 0.02 (0.0 – 0.17) for peers; for the middle tercile -0.01 (-0.01 – 0.01) for parenting and 0.00 (- 0.01 – 0.01) for peers; and for the lowest tercile (i.e., least susceptible) - 0.01 (- 0.13 – - 0.003) for parenting and -0.02 (-0.20 – - 0.003) for peers.

Inspection of the diagonal running from the top-left corner to the bottom-right corner of Table 4 indicates that of a total of 1,364 children, one third of the children (n = 450) scored in the same tercile of both susceptibility distributions (i.e., low, moderate, high). Having said that, only 7% of the full sample proved highly susceptible to the (putative) effects of both parents and peers (n = 94) and 10% highly unsusceptible to both exposures (n = 132). Thus, less than a fifth of the sample proved extremely susceptible or unsusceptible to the two effects under consideration.

Perhaps just as notable are the cells that reflect children highly susceptible to parent or peer effect (top tercile) and highly unsusceptible to the other (bottom tercile). These reveal that 15% of the total sample (n = 198) proved highly susceptible to peers but highly unsusceptible to parenting, with the comparable figures for the reverse configuration of susceptibility being 19% (n = 254). Thus, more than a third of the sample proved highly susceptible to one exposure and highly unsusceptible to the other.

Sensitivity Analysis

The final set of analyses re-evaluated parent-peer consistency in individual differences in susceptibility to parenting and peer effects by including both predictors in a single regression analysis before calculating individual differences in susceptibility (i.e., DFBETAS). Recall that it was expected that would result in smaller associations between parent and peer effects because the susceptibility index of each effect was based only on the unique variance that each predictor explained. Results of the regression analysis are displayed in Table 1 (supplementary material) and again indicate that more supportive parenting predicted greater growth in socioemotional well-being (*beta* = .25, $p < .001$), with the same being true of more positive peer relations (*beta* = .14, $p < .001$), though somewhat less so than in the primary analyses. On the basis of these results, individual differences in susceptibility were calculated for each individual for both parenting and peer effects and the resulting DFBETAS were correlated. The correlation between the two influence-statistic-derived susceptibility scores proved consistent with this expectation: $r = .29$ ($p < .0001$), though again it proved smaller than in the primary analyses, accounting for 8.4% of variance rather than the prior 16%. Once again, then, the result indicates that children who appeared to benefit the most from supportive parenting in terms of their socioemotional development were also most likely to be positively affected by peers. Conversely, children who appeared to benefit the least from supportive parenting were also least likely to benefit from their peer relations.

To gain descriptive insight into the degree of consistency in susceptibility to the two effects, exploratory efforts were undertaken once more by dividing each DFBETAS' distribution in thirds and cross tabulating them. Inspection of the diagonal running from the top-left corner to the bottom-right corner of Table 2 (supplementary material) indicates that of the total of 1,364 children, more than half of the children ($n = 730$) scored in the same tercile of both susceptibility

distributions (i.e., low, moderate, high). Having said that, 16.94% of the full sample proved highly susceptible to the (putative) effects of both parents and peers (n = 231) and 17.96% highly unsusceptible to both exposures (n = 245). Thus, more than a third of the sample proved extremely susceptible or unsusceptible to the two effects under consideration. Just as notable is that that 8.58% of the total sample (n = 117) proved highly susceptible to peers but highly unsusceptible to parenting, with the comparable figure for the reverse configuration of susceptibility being 8.43% (n = 115). Thus, somewhat less than a fifth of the sample proved highly susceptible to one exposure and highly unsusceptible to the other.

The role of pubertal development

To test the hypothesis that peer influences would be most pronounced in adolescents with more advanced pubertal development, I compared, by means of a one-way Analysis of Variance (ANOVA), three groups of adolescents (i.e., those scoring low, moderate and in high susceptibility to peer influence). Adolescents in the susceptibility groups did not significantly differ by their pubertal development F (2, 1361) = 1.80, p = .17. Specifically, adolescents who were highly susceptible to peer influence (M = 2.89) did not differ from adolescents in the moderate (M = 2.88) and low (M = 2.91) susceptibility groups to peer influence on their pubertal development.

Discussion

Despite extensive theorizing regarding the influence of parenting on child and adolescent development, as well as both observational and experimental evidence consistent with that claim, some decades ago a controversy arose when the argument was advanced that developmentalists had mistakenly privileged parents as the primary source of influence on child and adolescent

development because peers were more influential (Harris, 1995). The counterargument was that it was a mistake to pit parent and peer effects against one another (Collins et al., 2000), if only because the peer processes considered to be of developmental importance were themselves systematically related to what transpired in the family (Mounts & Steinberg, 1995). The research reported herein sought to extend work on parenting and peer effects by evaluating the extent to which children apparently most and least susceptible to effects of parenting on change in psycho-social adjustment were similarly—or differently—susceptible to the effects of peers. The primary results clearly indicated that apparent susceptibility to these much-studied sources of influence were by no means unrelated, just as anticipated; recall as well that the sensitivity analysis revealed less consistency in individual differences in susceptibility than did the primary analysis, basically half as much (18% vs. 8%). At the same time, descriptive information (i.e., cross-tabulation of tercile splits) made clear that there was a not insubstantial number of children who proved highly susceptible to one putative source of influence but not to the other, though the sensitivity analysis revealed this, once again, to be somewhat less the case than the primary analysis did.

More specifically, the very children who appeared to developmentally benefit more or less from supportive parenting in terms of their psychosocial adjustment were likely to be similarly affected by their peer relations. This anticipated result is consistent with a large literature indicating that adolescents who have been raised by more supportive parents (i.e., authoritative, reflecting warmth plus firmness) are more apparently influenced by positive peer relations peers, whereas those who are raised by less supportive parents (i.e., authoritarian, or permissive) are seemingly more influenced by negative peer relations (Collins & Steinberg, 2006). Notably, evidence in line with such prior work emerged from this investigation as well.

Recall the significant positive association detected between composite constructs of parent and peer exposures that helped to validate these measurements. Regarding pubertal development, there is a relationship between puberty and susceptibility. Contrary to initial predictions, the association was not in the expected direction in that more advanced pubertal development was associated with less peer and more parent influence rather than the reverse. Further investigation of this relationship did not reveal any significant mean difference between susceptibility groups with respect to peer influence. This could be because the process of terciling susceptibility groups reduces power.

All this is not to say that Harris (1995, 1998) was entirely wrong—or right—in arguing that peers are more influential in shaping development than parents. In line with her claim, recall that the illustrative (terciled) cross-tabulation of children scoring high, moderate and low on the susceptibility to parenting and to peer effects in the primary analysis revealed that while a full third of the sample appeared highly susceptible to one and highly unsusceptible to the other (with figures clearly smaller when quartile and quintile distributions were used), some children displayed the pattern that Harris might have predicted (15% highly susceptible to peers with low susceptibility to parents). Just as notable, perhaps, was that there were more such children (i.e., low parenting/high peer: 15%) than those who proved most susceptible to both effects (i.e., high-high: 7%) or least susceptible to both (i.e., low-low, 10%).

These figures should not be reified because in both primary and sensitivity analyses reliance on terciles to characterize the influence-statistic-derived susceptibility distributions represented an arbitrary decision. Nor should it be presumed that the results just summarized, which apply to a particular period of development, albeit one now considered a period of heightened developmental plasticity resulting from ongoing brain development (Aoki, Romeo &

Smith, 2017), would be the same were the parent-peer susceptibility issue investigated addressed at younger or older ages (Schulenberg & Maslowsky, 2015). The same caveat should be noted when it comes to the measures used to create the parenting, peer, child and adolescent composites in the current report. Different measures could yield different results. Ultimately, the goal of this inquiry was not to be positioned to draw definitive and widely generalizable conclusions about individual differences in the relative susceptibility to parent and peer effects, but to raise this fundamental developmental—and unstudied issue—while illustrating a "person-as-effect-size" approach to addressing it.

The results presented would seem of importance for two reasons, each highlighted in the Introduction. First and from a basic-science point of view, it would now seem problematic to think about differential susceptibility to environmental influences in domain-general rather than domain-specific terms. While it is likely, as the evidence reported herein indicates, that some individuals will be consistently high or low in susceptibility to parent and peer effects, it is clear that susceptibility to environmental influences may vary depending on the source of influence under consideration. This may not be surprising, but it challenges a view that has emerged in recent years (i.e., orchids and dandelions; Boyce & Ellis, 2005). From an applied-science perspective, the fact that some children may be more susceptible to parent than peer influence—and others the reverse—suggests that targets of effective intervention may vary across children. Interventions targeting parents would seem to be most appropriate for children highly susceptible to parenting but not peer effects, whereas one targeting peers would seem most appropriate for those highly susceptible to peers but not parents.

This inference would seem to point to a possible agenda for developmental scholars going forward: What child, parent, peer—or other—factors might predispose certain children to

be highly responsive to one source of influence more so than another? Although this investigation was not positioned to explore this issue, some possibilities come to mind. For children for whom being popular with peers is especially important, targeting peer processes might be most appropriate. For children who identify strongly with their parents—wanting to be like them, perhaps—targeting parents might be most appropriate when it comes to intervention. Given the well documented heterogeneity in intervention efficacy, perhaps a more personalized approach is called for, just as has become evident in medicine.

In point of fact, the evidence presented herein warns against turning central tendencies of any kind, including the highly significant average associations between the two susceptibility effects chronicled herein, into all-too-general conclusions. All too often developmental scholars, journalists and the lay public erroneously conclude that a significant relation between variables observed in a sample—or an overall treatment effect in intervention work—applies at the individual level and thus equally so to all individuals. The authors of this report are not themselves entirely immune to this tendency. Yet what it captures is what is going on in the aggregate. Failure to make the aggregate-individual distinction is known as the ecological fallacy (Robinson, 1950). Clearly, the variation in patterns of apparent susceptibility to parent and peer effects revealed in Tables 4 and 5 strongly underscores the problem associated with over-interpreting the significant correlation between parent and peer susceptibility effects at the aggregate level. As the descriptive (cross-tab) analyses indicate, there is clear variability among adolescents in their relative degrees of susceptibility to the apparent influence of parents and peers.

Whatever the strength of this effort, whether addressing a previously unaddressed and fundamental developmental question using a large sample followed longitudinally, it is not

without limits. Perhaps the major ones that should temper any and all conclusions are that the parenting and peer measurements used herein by no means cover all features of these domains of potential influence and that their imperfect equivalence could surely have affected the results reported. Additionally, not relying on latent variables for parenting and peer constructs may limit the ability to capture all shared variance among all measures. Also meriting consideration is reliance on a residualized measure of change in psychosocial adjustment, necessitated by having relatively comparable measurements of the outcome construct only in 6th grade and at age 15. It seems quite likely that had identical child and adolescent measurements been available, that change—assessed by a residualized dependent variable—would have been more accurately captured. In fact, it would have been even more preferable to have at least three measurement occasions of child and adolescent psychosocial development so that growth curve estimates of slope could have served as the outcome to be explained, as this would have enabled the measurement of susceptibility with regard to within-person change over time rather than just between-person change. Either of the design improvements just highlighted could affect the degree of consistency in susceptibility discerned herein. One also has to be wary that at least some of the influence-statistic estimation of susceptibility reflects error rather than precise assessment of such variation.

That the relation between susceptibility to parenting and peer effects showed a moderate effect size was no doubt at least partly the result of the aforementioned significant and positive association linking the composite parenting and peer predictor constructs. The more associated these measurements, the more consistency in apparent susceptibility to parent and peer effects there would be. This raises at least two interesting research directions going forward. First, what would results like those generated here look like if exposures to parents and peers were unrelated

or at least far less related? Second, could experimental manipulations of parenting and peer behavior be induced to determine if some children prove more differentially responsive to these social agents. And, if so, what attributes of children or parents or peers might account for any detected differential susceptibility to parent and peer exposures.

Limitations

Whatever the strength of this effort, whether addressing a previously unaddressed and fundamental developmental issue using a large sample followed longitudinally, it is not without limits. Perhaps the major ones that should temper any and all conclusions are that the parenting and peer measurements used herein by no means cover all features of these domains of potential influence and that their imperfect equivalence could surely have affected the results reported. Additionally, not relying on latent variables for parenting and peer constructs may limit the ability to capture all shared variance among all measures.

Also meriting consideration is reliance on a residualized measure of change in psychosocial adjustment, made possible by having relatively comparable but not identical measurements of adjustment in 6th grade and at age 15. It seems quite likely that had identical child and adolescent measurements been available, that change—assessed by a residualized dependent variable—would have been more accurately captured. In fact, it would have been preferable to have at least three measurement occasions of child and adolescent adjustment so that growth curve estimates of slope could have served as the outcome to be explained. This would have enabled the measurement of susceptibility with regard to within-person change over time, rather than just between-person change. Either of the design improvements just highlighted could affect the degree of consistency in susceptibility discerned herein. One also must be wary

that at least some of the influence-statistic estimation of susceptibility reflects error rather than precise assessment of such variation.

The last issue to be raised concerns the degree of association between the parent and peer predictor constructs and how that can affect results. Had they been less related, would the susceptibility to these two sources of influence proven more independent? That is certainly what the results of the sensitivity analysis suggest. Recall that by focusing on only the unique variance in change in adjustment accounted by parents and by peers—by including both predictors in the regression analysis—the degree of consistency in susceptibility to parents and peers was cut almost in half.

Conclusion

Parents and peers are two well-documented sources of influence on adolescent psychosocial adjustment. The findings presented herein extend prior research by considering, for the first time, the extent which children whose psychosocial development appears most and least susceptible to parenting effects are similarly—or differentially—influenced by their experience with peers. Results revealed, as anticipated, that susceptibility to the influence of parents and peers was positively related, even if only modestly so. At the same time, the descriptive (cross-tabulated, terciled) data made clear that there are a substantial number of children who appear highly susceptible to one putative source of influence but not to the other. Our results contribute to work focused on better understanding of how parents or peers appear to influence adolescent psychosocial adjustment beyond average effects, by highlighting individual differences in susceptibility to these developmental experiences.

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Table 1. *Principal Component Weightings of Data-Reduction Analyses*

Parenting Composite		Peer Composite	
Variable	Loading Weight	Variable	Loading Weight
Maternal Sensitivity	.46	Major Risks by Friends	-.79
Maternal Warmth	.68	Minor Risks by Friends	-.75
Maternal Hostility	-.70	Resistance to Peers	.71
Child Conflict	-.67		
Child Closeness	.67		
5th Grade Psychosocial-Adjustment Composite		15 yr. Psychosocial-Adjustment Composite	
Variable	Loading Weight	Variable	Loading Weight
Social-emotional well-being	.60	Peer Competence	.39
Social Skills	.80	Non-Sex Risk Taking	-.80
Peer Competence	.78	Sexual Risk Taking	-.63
Teacher Conflict	-.62	Lack of Impulse Control	-.73
Minor Risk Taking	-.36	Externalizing Behavior (mother-report)	-.58
Disruptive Behavior	-.79	Externalizing Behavior (self-report)	-.79
Externalizing Behavior	-.73		

Table 2. *Inter-correlation of Composite Measures*

Composite	Parenting	Peers	5th grade Adjust.	15 yr. Adjust.
Parenting	-			
Peers	.39**	-		
5th grade Adjust.	.56**	.20**	-	
15 yr. Adjust.	.43**	.35**	.48**	-
Pubertal Dev.	.03**	-.05**	.04**	.05**

Notes: Adjust: psychosocial adjustment, Pubertal Dev: pubertal development
** = $p < .01$.

Table 3. *Regression Results of (A) Parenting and (B) Peer Effects on Age-15 Psychosocial Adjustment*

A. Testing Parenting Effect					
Predictor Variables	*B*	*SE B*	*t*	*df*	*p*
Parenting	.36	.05	7.09	236	.001
Child Sex	-.36	.24	-1.50	226	.13
Child Race/Ethnicity	-.25	.24	-1.02	178	.31
Maternal Education	.07	.05	1.34	463	.18
Maternal Depression	-.04	.02	-2.26	138	.03
Income to Needs	.08	.05	1.67	508	.10
Father in Home	.81	.27	3.03	254	.001
5th Grade Psychosocial Adjustment	.16	.02	7.15	292	.001

B. C. Testing Peer Effect					
Predictor Variables	*B*	*SE B*	*t*	*df*	*p*
Peer Influence	.18	.04	4.78	133	.001
Child Sex	-.43	.24	-1.81	241	.08
Child Race/Ethnicity	-.21	.25	-.81	145	.42
Maternal Education	.09	.05	1.56	514	.12
Maternal Depression	-.05	.02	-2.91	131	.01
Income to Needs	.06	.05	1.29	531	.20
Father in Home	.96	.27	3.52	241	.001

5th Grade Psychosocial Adjustment	.21	.02	10.17	275	.001

Table 4. *Tercile Splits of Susceptibility to Parenting and Peer Effects (based on DFBETAS)*

	Susceptibility to Parenting			
Susceptibility to Peers	Low	Moderate	High	Total
Low	132	68	254	454
Moderate	124	224	107	455
High	198	163	94	455
Total	454	455	455	1364

Paper 2

Early Life Threat and Deprivation: Are children similarly affected by exposure to each?

Abstract

Extensive evidence documents negative consequences of adversity for children's development. Here we extend such work by looking beyond such average effects to consider variation in susceptibility to both threat and deprivation in terms of cognitive and social-emotional development, using an influence-statistic methodology. Data comes from the Avon Longitudinal Study of Parents and Children (N = 14,541, 49.1% female, age range: 6mo. to 12yrs., race/ethnicity: 97.8% white, .4% black and .6% other). With respect to documented associations of threat with problem behavior and of deprivation with cognition, results of this pre-registered research revealed that a roughly equal proportion of children proved to be susceptible in a domain-general manner (similarly influenced) and a domain-specific one (dissimilarly influenced). Implications for intervention are considered.

Keywords: ALSPAC, early-life adversity, differential susceptibility, cognitive development, social-emotional development

Early Life Threat and Deprivation:

Are children similarly affected by exposure to each?

Experiencing early life adversity is a well-studied and influential source of influence on children's development (Nelson et al., 2020). Two core underlying dimensions of adversity—threat and deprivation—cover a wide range of adverse experiences common in childhood. Experiences involving threat (i.e., experiences that encompass harm or threat of harm to the child), and deprivation (i.e., a reduction in expected environmental inputs during development) each have distinct influences on children's emotional and cognitive development (McLaughlin & Sheridan, 2016; Sheridan & McLaughlin, 2014). Apart from research informed by diathesis-stress or dual-risk thinking, which posits that some children are more vulnerable to adversity than others, virtually all research on adversity documents what are essentially average effects of adverse childhood experiences on the sample under study (Ellis, Sheridan, Belsky & McLaughlin, 2022). As such, it disregards the fact that there is variation in terms of which children are affected and the extent to which this is the case. Differential susceptibility theory posits that some individuals are more developmentally plastic and therefore, vary more generally in their susceptibility to both negative and positive environmental effects (Belsky, Bakermans-Kranenburg & van IJzendoorn, 2007; Belsky & Pluess, 2009, 2013; Ellis et al., 2011). Yet, what has been repeatedly noted in the work just cited but only recently been subject to empirical inquiry is whether developmental plasticity should be regarded as a general trait (Belsky, Zhang & Sayler, 2021). That is, are the same children affected, for better or for worse, more than others by different adverse experiences and exposures, such as threat and deprivation? This is the focus of the present report. To address this issue, the research reported herein draws on data from the Avon Longitudinal Study of Parents and Children (ALSPAC); assesses the extent to which

particular children's cognitive and social-emotional development are most and least affected by threat and deprivation; and evaluates whether children are similarly affected to each adversity. The work presented was pre-registered (https://osf.io/wpcxh) using Open Science Framework (OSF).

Threat and Deprivation Effects

Exposure to early life adversity increases the risk of compromised well-being, broadly conceived, including psychosocial and cognitive functioning (Milojevich et al., 2019). Much prior research documenting such effects has relied on cumulative-risk approaches that sum adverse experiences regardless of type, chronicity, or severity in an effort to account for variation in children's development (Anda et al., 2005). This body of work chronicles strong links between experiencing adversity and many negative mental health outcomes (McLaughlin et al., 2010, 2012). For example, a cumulative-risk index compromised of seven indicators—physical abuse, sexual abuse, single-parent household, number of caregiver transitions, number of school transitions, exposure to community violence, and intellectual functioning—predicted mental health symptoms among a sample of maltreated youths (aged 9-11), reliably differentiating between those children who did and did not score in the clinical range for anxiety and externalizing/internalizing problems (Raviv et al., 2010). Thus, the more adversity children experienced, the more likely they were to develop mood, anxiety, and disruptive behavior disorders (for review see Evans, Li & Whipple, 2013; Juwariah et al., 2022).

Importantly, childhood adversity exposure is not only associated with the risk of mental disorder onset in childhood and adolescence, but also with a greater risk of developing a mental disorder for the first time in adulthood (Green et al., 2010). Cumulative childhood adversity is linked specifically to mental health problems in adulthood, including personality disorders

(Björkenstam et al., 2017), eating disorders (Steine et al., 2017), and depressive, anxiety, and stress-related disorders (Björkenstam et al., 2021; Houtepen et al., 2020). Despite their power to predict variation in development, cumulative-risk models provide little insight into the mechanisms accounting for detected links between adverse exposures and developmental outcomes (McLaughlin al., 2021).

The Dimensional Model of Adversity and Psychopathology (DMAP) addressed this lacuna by distinguishing two core underlying dimensions of adversity: threat and deprivation (McLaughlin et al., 2014, 2021; McLaughlin & Sheridan, 2016) The distinct dimensions of adversity cover a wide range of negative experiences and exposures that are all too common in the childhoods of many children. Threat encompasses experiences involving actual harm or threat (e.g., physical, or sexual abuse), and deprivation involves reductions in cognitive or social inputs (e.g., neglect). It is well appreciated that these kinds of adversity often co-occur, even as these dimensions can be measured separately and have unique effects on development (Ellis et al., 2020; McLaughlin et al., 2012). Because neural plasticity is heightened in early childhood and adolescence (Fandakova, 2020), adverse experiences and exposures during these developmental periods are especially likely to produce lasting changes in the brain—and thus psychological and behavioral development.

According to the DMAP, brain plasticity is thought to be the primary mechanism through which environmental exposures and experiences shape learning and development. This is because neural plasticity mechanisms are sensitive to specific types of environmental inputs (Takesian & Hensch, 2013; Kolb & Gibb, 2014; Ho & King, 2021), making it unlikely that the neurodevelopmental processes influenced by early life adversity that in turn influence psychological and behavioral development are similar across all forms of adverse environments.

Therefore, the central tenant of the DMAP is that different dimensions of adversity will have influences on neurodevelopment and its phenotypic consequences that are at least partially distinct. Notably, evidence is consistent with this theoretical claim (Kolb & Gibb, 2014; McLaughlin & Gabard-Durnam, 2021; Nelson & Gabard-Durnam, 2020).

Disruptions in threat processing are thought to be a key neurodevelopmental mechanism underlying associations between exposure to childhood trauma and the onset of internalizing and externalizing symptoms (McLaughlin & Lambert, 2017). Specifically, DMAP predicts that experiences of threat during childhood alter developing neural networks in ways that facilitate the swift identification of danger in the environment and initiate defensive responses for safety (Sheridan & McLaughlin, 2014; McLaughlin & Lambert, 2017). It further stipulates that experiences of threat are related to problematic behavior via fear learning, emotional reactivity, and difficulties with emotion regulation (McLaughlin & Lambert, 2017). There is strong evidence to support this claim; a review of relevant evidence revealed that experiences of threat early in life are associated with changes to both the structure and function of brain regions involved in emotion learning, including reduced amygdala and hippocampal volume and elevated amygdala responses to threat cues (McLaughlin, Weissman & Bitran, 2019).

In contrast, children exposed to deprivation in early life do not generally exhibit similar alterations in emotional processing associated with threat. Instead, experiences of deprivation are linked to cognitive difficulties, including language ability and executive functioning. Like threat, though, deprivation is also associated with increased risk for psychopathology and difficulties in school (Miller et al., 2021; Lonigan et al., 2017). Reductions in expected environmental inputs influence future cognition by altering the foundation on which more complex forms of thinking

are based. More specifically, the brain selectively eliminates synaptic connections that are utilized infrequently (Faust, Gunner & Schafer, 2021).

The DMAP predicts that exposure to environments characterized by limited social and cognitive stimulation can contribute to accelerated and extreme synaptic "pruning," thereby, leading to reductions in the thickness and volume of cortical regions essential for social and cognitive functioning (McLaughlin et al., 2017; Sheridan & McLaughlin, 2016). Evidence supporting these claims comes from research on children who experience extreme deprivation, such as growing up in understaffed Romanian orphanages, as well as those exposed to less severe forms of deprivation, including poverty and neglect. Children experiencing such deprived conditions exhibit reductions in grey matter volume and, presumably in consequence, difficulties in executive functioning and linguistic development.

Differential Susceptibility to Environmental Influences

Regarding effects of two different types of adversity just reviewed, there is ever more theory and evidence that children vary in the extent that these forces shape development. According to differential susceptibility thinking, it is not just, as with diathesis stress or dual risk models, that some children are especially vulnerable to adversity but, rather that children vary in their susceptibility to both positive and negative developmental experiences and environmental exposures (Belsky, Bakermans-Krannenburg & van Ijzendoorn, 2007; Belsky & Pluess, 2009, 2013; Ellis et al., 2011). Differential susceptibility to environmental influences is often conceptualized in trait-like terms, implying that children whether proving high or low in susceptibility develop this way across diverse contextual inputs (e.g., harsh parenting, cognitive stimulation) and developmental sequelae (e.g., aggression, executive function). Reliance on terminology of "orchids and dandelions" (Boyce & Ellis, 2005) and of "highly sensitive persons"

(Aron & Aron, 1997) makes this explicit, as have graphic depictions of differential susceptibility (Belsky et al., 2007).

This domain-general view of differential susceptibility would seem mis-aligned with current neurobiological thinking which presumes that that different adverse experiences influence different brain processes and thereby different developmental phenotypes (e.g., McLaughlin, Sheridan, & Lambert, 2014). Nevertheless, there are grounds for taking a domain general approach seriously. Perhaps the best reason why such a domain-general perspective remains not entirely unreasonable is due to empirical evidence. Truth be told, many studies of differential susceptibility, whether focused on temperament, physiology, or genes as moderators of environmental effects in a for-better-and-for-worse manner, document that the same putative plasticity factors condition—in very similar ways—the effects of a wide variety of environmental features, like prenatal stress, maternal empathy, marital conflict, teacher–child conflict and economic hardship, on a wide variety of developmental phenotypes, such as externalizing problems, executive function, attentional bias, sleep and pubertal development) (Belsky & Pluess, 2009; 2013; Ellis et al., 2011). In other words, however simplistic a general trait-like view of susceptibility to environmental influences might first appear, this possibility is not without empirical support.

Current Study

The research reported herein extends prior work on differential susceptibility by looking beyond average effects to consider individual differences in susceptibility to the consequences of threat and deprivation. Therefore, the primary goal is to assess the extent to which children prove similarly susceptible to effects of threat on problem behavior and deprivation on cognitive functioning. To achieve this goal, a novel influence-statistic approach is utilized that assesses

individual differences in susceptibility to environmental influence. This methodology has been used in prior studies looking at the same issue of domain-generality vs. domain-specificity of environmental effects when considering two distinct effects of child care (Belsky, Zhang & Sayler, 2022), effects of environmental harshness and unpredictability (Zhang, Schlomer, Ellis & Belsky, 2021), the timing of adversity exposure (i.e., early childhood and adolescence; Belsky & Andersen, 2022) and parent and peer effects (Sayler, Zhang, Steinberg & Belsky, 2022). Data for the proposed study comes from the ongoing Avon Longitudinal Study of Parents and Children .

The OSF pre-registration (https://osf.io/wpcxh) delineated the following hypotheses: (1) that greater deprivation exposure will be associated with poorer cognitive abilities and intelligence; (2) that greater threat exposure will be associated with poorer socioemotional development (i.e., more externalizing and internalizing problems); and (3) that children who prove most and least susceptible to threat will be somewhat similarly affected by deprivation, but there will be children who prove highly susceptible to one exposure but not to the other (with regard to their respective anticipated outcomes).

Method

Participants

Data for this report comes from the ongoing Avon Longitudinal Study of Parents and Children (ALSPAC, $N = 14{,}541$). ALSPAC was launched in the early 1990s to investigate modifiable influences on individuals' health and development (among many other topics). Pregnant women were enrolled with estimated delivery dates between 1 April 1991 and 31 December 1992 within the Avon area of England (Boyd et al., 2013; Fraser et al., 2013). 20,248 pregnancies have been identified as being eligible and the initial number of pregnancies enrolled

was 14,541. Further details about ALSPAC are found at http://www.bristol.ac.uk/alspac/. The study website contains details of all the data within a fully searchable data dictionary and variable search tool (http://www.bristol.ac.uk/alspac/researchers/our-data/). Ethical approval for this study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees. Informed consent for the use of data collected via questionnaires and clinics was obtained from participants following the recommendations of the ALSPAC Ethics and Law Committee at the time.

Sample Size

The analytic sample size is based on the total ALSPAC sample of 14,541 children, minus cases that met exclusionary criteria (see Data Exclusion), as multiple imputation is used to estimate all missing data for variables included in the current report.

Data Exclusion

Children with scores of mother-reported poor health within the past year from 18 months to 3.5 years and those whose teachers reported that the child had physical disabilities, medical conditions, or developmental delay in grades 3 and 6 were excluded from the analysis. Excluded cases based on the mother and teacher-reported health indices resulted in a final analytic sample size of 14,029 children.

Measures

Predictor and outcome variables, as well as covariates, are delineated below. All variables were scaled so that a higher score reflects greater threat, deprivation, problem behavior or cognitive deficits (i.e., reverse scaling as needed).

Environmental Predictors

Two sets of variables—one for threat and the other for deprivation exposures—served as predictors.

Deprivation. Three indices of deprivation were used: (i) To assess (lack of) *maternal cognitive stimulation*, we relied on mothers' reports of cognitively stimulating activities with their children at 6 months, 18 months, 2.5 yrs. and 3 yrs., such as taking child to local shops, supermarkets, parks and homes of friends/family; answers were given on a 5-point scale from "more than once a week" to "never". (ii) (Lack of) p*arental education* was self-reported by mothers and fathers separately at 32 weeks' gestation reflecting highest level of educational attainment. (iii) Lastly, deprivation-related aspects of *parenting* were reported by mothers at 2 yrs., 3 yrs., and 3.5 yrs. At 2 yrs. and 3 yrs. mothers were asked how often they allow their child to play with paints/messy objects, use objects to build towers and sing to them, read stories, and go to the park or playground. Answers were given on a 5-point scale ranging from "every day" to "never". At 3.5 yrs., mothers were asked slightly different questions focused on different activities, including singing and reading to child, playing with toys and other fun activities, and taking the child for walks. Answers were given on a 5-point scale from "Nearly every day" to "Never.

Threat. Four indices of threat exposure were used: (i) The *Neighborhood Stress Score* was based on mothers' report on frequency of burglary, attacks, vandalism, and problems with youth in their neighborhoods at 1.75 yrs. and 2.75 yrs. The score was computed as the mean on 11 items, each rated on a 3-point scale (0 = no problem or opinion, 1 = minor problem, 2 = serious problem). (ii) To assess exposure to *domestic violence*, mothers were asked when their child was 1.75 yrs. old if they or their partners had "hit or slapped their partner in the past 3 months" and if they "threw something in anger within the past 3 months". Answers included

"yes mum did", "yes partner did", "yes both did" or "no not at all" and "no partner". (iii) Mothers were also asked when their child was *physically hurt* by another person at 6, 12, and 18 months, and 3 and 5 yrs. Answers included "Yes" or "No". (iiii) Lastly, mothers reported on their *parenting* at 2 yrs. Mothers were asked, "When you're at home with your child how often do you do the following: shout at them and slap them". Answers were given on a 5-point scale ranging from "Every day" to "Never".

Developmental Outcomes

Two sets of variables relating to children's cognition and behavior served as outcomes.

Cognitive Deficits. Two indices of cognition were used: (i) To assess *intelligence*, children completed a clinic-based assessment at age 8 using a short form of the Wechsler Intelligence Scale for Children- III (WISC; Woogler, 2001). This included both verbal and performance IQ. (ii) To assess *learning, speech, and language difficulties* teachers provided information in grades 3 and 6 by endorsing or not the existence of such difficulties.

Problem Behavior. Two indices of children's behavior were used. To assess children's emotions and behavior, teachers in grades 3 and 6 completed the short form of the Strengths and Difficulties Questionnaire (Goodman, 1997) which yielded sub-scores for (i) *internalizing problems* (emotional symptoms, peer problems) and (ii) *externalizing problems* (conduct problems, hyperactivity/inattention).

Covariates

Mothers reported on child sex and race/ethnicity (e.g., white, or other race) upon enrollment, as well as child's exact age at the scheduled 24-month assessment.

Analysis Plan

Multiple steps in the analysis are organized in terms of preliminary and primary analyses. A sensitivity analysis is also described, though it was not cast as such in the pre-registration, even though the statistical analysis was specified.

Preliminary Analyses

The first step of the preliminary analyses involved multiple imputation of missing data using Multivariate Imputation by Chained Equations (MICE; van Buuren & Groothuis-Oudshoorn, 2011) in R; this involved all listed study variables. All subsequent preliminary, primary and sensitivity analyses were then conducted based on 20 imputed data sets. Two data-reduction-oriented principal components analyses, one including threat and the other deprivation indicators, were conducted to limit statistical testing in the primary analysis. Pre-registration neglected to stipulate that this step would be conducted on outcomes as well as predictors. Results of principal components analyses provided the basis for creating single composite scores for each predictor and outcome. The final preliminary analyses relied on two least squares regression analyses, one using threat to predict problem behavior and the other using deprivation to predict cognitive deficits, after controlling for covariates. The decision to focus on only these associations was based on prior research reviewed earlier showing these distinctive and discriminating links between particular dimensions of adversity and their particular sequelae.

Primary Analyses

Following these preliminary analyses, primary analyses were carried out using influence statistics to assess the degree to which each child appeared susceptible to consequences of threat and deprivation documented in the prior regression analyses so that the two resulting susceptibility scores could be associated with each other; this would determine the extent to which children affected more or less by one experience were or were not affected by the other in

a similar way. We relied on an influence statistic, DFBETAS, a continuous and standardized index assigned to each and every observation.

DFBETAS reflects the degree and direction of change of the regression coefficient after removing a single observation. Therefore, DFBETAS is calculated using a "leave-one-out" approach by re-estimating an association repeatedly, each time dropping a single case to measure how much such (minor) sample modification causes the full-sample association to increase (i.e., a negative influencer) or decrease (i.e., a positive influencer), usually ever so modestly. The resultant change of the slope parameters attributed to each observation for the association of interest (i.e., threat: problematic behavior; deprivation: cognitive deficits) indicates how—and the extent to which—particular individuals affect the full-sample estimate of the association (e.g., Belsley, Kuh, & Welsch, 1980; Cook & Weisberg, 1982). More concretely, the more the predictor-outcome association becomes less positive when a case is dropped, the more susceptible the child is to the effect in question. Conversely, the more positive the association becomes when a case is dropped, the less susceptible the child is to the effect.

The final step in the primary analysis evaluated whether children most and least affected by one of the predictor effects under investigation (i.e., threat, deprivation) were similarly affected by the other. This involved correlating the DFBETAS' threat and deprivation susceptibility scores and cross-tabulating tercile splits of the two DFBETAS' distributions. No significance test will be applied to the latter because it is not independent of the former.

Sensitivity Analysis

Sensitivity analysis involved first repeating the preliminary regression analysis, followed by a repeat of the primary susceptibility analysis. But this time the preliminary regression analysis included both predictors, threat and deprivation, when predicting each of the two

outcomes, so that only the *unique variance* explained by each predictor would be attributed to it. Thus, for example, when evaluating the effect of threat on the problem-behavior outcome, deprivation is treated as a covariate. Recall that while the modified regression analyses (and resulting susceptibility scores) were preregistered, they were not framed as sensitivity analyses. The expectation was that the two effects in question would become more independent relative to the primary analysis because including a second adversity dimension in the regression analysis would reduce multicollinearity.

Results

Preliminary Analyses

Table 1 presents results of the (separate) principal component analyses of predictor and outcome variables. In each case, we created unit-value-based summary scores for all variables loading on the first principal component. These are labeled threat, deprivation, cognitive deficits, and problem behavior.

Intercorrelations of these composite measures are displayed in Table 2, an analysis not specified in the pre-registration of the study. These correlations, even though modest, provide validation of the factor-derived constructs specific to this investigation in that associations are consistent with expectations. Specifically, greater threat is significantly associated with more behavior problems and greater deprivation is significantly associated with greater cognitive deficits. Notably, threat predicts problem behavior more strongly than cognitive deficits, as well as more strongly than deprivation. Relatedly, deprivation predicts cognitive deficits more strongly than problem behavior, as well as more strongly than threat. Also worth mentioning is

that threat and deprivation are positively associated. All these results are consistent with observations made in the Introduction.

The final preliminary analysis involved evaluating—in two separate exploratory regression models on which DFBETAS susceptibility scores would be based—whether, net of covariates, greater threat was associated with greater behavior problems and whether greater deprivation was associated with more cognitive deficits. Inspection of Table 3 reveals that these expectations were confirmed.

Primary Analyses

The first step in the primary analyses involved calculating, by means of DFBETAS, the degree of susceptibility of each child to the effects of threat on problem behavior and deprivation on cognitive deficits. Recall that this was accomplished via the leave-one-out procedure and thus re-running the two whole-sample regression analyses 14,029 times, once using the threat composite to predict problem behavior and once the deprivation composite to predict cognitive deficits. With individual susceptibility scores estimated for each case, the resulting DFBETAS for threat and deprivation were correlated. The correlation between the two influence-statistic-derived susceptibility scores was highly significant but small ($r = .07$, $p < .0001$), indicating that children most or least susceptible to the adverse effects of threat on problem behavior proved somewhat similarly susceptible to the adverse effects of deprivation on cognitive deficits. The fact that a small effect proved so statistically reliable no doubt is a result of the very large sample.

Pre-registered exploratory efforts were undertaken to provide further descriptive insight into the association between the two susceptibility scores, dividing each DFBETAS' distribution, arbitrarily, into thirds and cross tabulating them. Inspection of the diagonal in Table 4 running

from the bottom-right corner to the top-left corner indicates that a bit more than a third of the children (36.8%) scored in the same tercile of both susceptibility distributions (i.e., low, moderate, high), thus appeared to be influenced in a relatively domain-general manner. Having said that, only a quarter of the sample proved highly susceptible to effects of both threat and deprivation (12.7%) or highly unsusceptible to both of these effects (12%).

Further inspection of cells in Table 4 indicates that 10.3% of the total sample proved highly susceptible to threat but highly unsusceptible to deprivation, with the comparable figure for the reverse configuration also being 10.3%. Thus, a fifth of the sample proved highly susceptible to one experience and highly unsusceptible to the other, thus appearing to be susceptible in a more domain-specific manner. In other words, only a little more of the sample proved similarly susceptible to both effects (i.e., ~25%) than did those who proved very different in their susceptibility to the two effects (i.e., ~20%).

Sensitivity Analysis

The final set of analyses re-evaluated consistency in individual differences in susceptibility to threat and deprivation exposures by first including, in a revised version of the preliminary regression analysis, both predictors in a single prediction model (along with covariates) before calculating the intercorrelation of susceptibility scores (i.e., DFBETAS). Recall that it was expected that this would yield even more evidence of inconsistency in susceptibility to the two effects under consideration—because the variance that threat and deprivation shared would be attributed to neither, thus making their effects more independent. Results proved consistent with this domain-specific expectation in that the two derived susceptibility effects (e.g., for threat and deprivation) proved somewhat less correlated ($r = .01$, p

< .001) than in the primary analysis (r = .07, p<.001), with this difference proving statistically significant ($z = 5.03$, $p < .001$).

Discussion

Recall that the primary goal of the work reported was to look beyond average effects of two distinct dimensions of adversity, threat and deprivation, on, respectively, two different developmental sequelae, problem behavior and cognitive deficits. Toward this end, we relied on an influence-statistic methodology to estimate the nature and magnitude of each of the two consequences on individual children in order to evaluate whether children proved to be similarly or differently affected by the two effects in question. Results of the preliminary analysis revealed that our initial expectations outlined in the preregistration were confirmed in that greater threat predicted more behavior problems, whereas greater deprivation predicted more cognitive deficits. These findings align with prior work showing that experiences of threat and deprivation are differentially associated with different developmental phenotypes in children (Usacheva et al., 2022; Schäfer et al., 2023; Vogel et al., 2021; Machlin et al., 2019) and have distinct influences on neurobiology (Banihashemi et al., 2021).

The primary results further indicated that children most or least susceptible to the adverse effects of threat on problem behavior proved somewhat similarly susceptible to the adverse effects of deprivation on cognitive deficits. To provide further descriptive insight into this association, pre-registered exploratory efforts made clear that more than a third of the children scored in the same tercile of both susceptibility distributions (i.e., low, moderate, high). In fact, a full quarter of the sample proved highly susceptible to the effects of both threat and deprivation or highly unsusceptible to both of these sources of influence. Just as notable, however, is that further consideration of categorical tercile splits revealed that a fifth of the sample proved highly

susceptible to one exposure and highly unsusceptible to the other. In sum, only a little more of the sample proved similarly susceptible—whether high or low—to both dimensional effects (i.e., ~25) than did those who proved very different in their susceptibility to the two effects (i.e., ~20%). In other words, whereas some children proved to be influenced in a domain-general way to the two effects under investigation, others appeared to be affected in a domain-specific manner. Such variation in children's susceptibility to environmental influences has never before been considered, much less documented, in research on threat and deprivation. The findings of the primary analysis are consistent with other recent investigations of differential susceptibility to environmental influences using the same influence-statistic approach (Zhang, Widaman, & Belsky, 2021). Recall that this prior work also chronicled both domain-generality and domain-specificity—in the case of different children—when with respect to two different child care effects (Belsky et. al., 2022), effects of environmental harshness and unpredictability (Zhang et al., 2021), adversity exposure in early childhood and adolescence (Belsky & Andersen, 2022), and parent and peer effects (Sayler, et al., 2022).

Taking these findings at face value, while appreciating that the foci of the just-cited studies included only two distinct environmental effects, would seem consistent with the conclusion that there are children who are more generally susceptible to environmental effects, others who are generally unsusceptible, and still others, including most individuals, falling somewhere in between. Just as notable, however, is that a separate report considering multiple family and child care predictors of multiple developmental phenotypes yielded evidence more consistent with domain specificity than domain generality (Zhang, Widaman & Belsky, 2023). The same was true, of course, once only the unique effects of threat and deprivation were examined in the sensitivity analysis in the current study. Collectively, these results seem in line

with evolutionary analysis stipulating that it would have been beneficial—in terms of reproductive fitness—over the course of human evolutionary history for individuals to vary in their susceptibility to specific experiences and exposures (Belsky, 2005; Belsky & Pluess, 2009; Ellis et al., 2011).

What remains unclear in the present case, given the limited focus of the research reported herein, is what distinguishes highly susceptible, highly unsusceptible, and intermediately susceptible children who were and were not similarly influenced by threat and deprivation. Is it their temperaments as young children, physiological reactivity, or genetics, all of which have been highlighted as potential "plasticity factors" in the differential susceptibility literature (Belsky & Pluess, 2009, 2013; Ellis et al., 2011), or some other source of influence? This empirical question was not pursued because of the absence of strong theory when it comes to guidance. It seemed like the time had passed to simply conduct exploratory work checking out a variety of seemingly alternative hypotheses (Belsky et al., 2021; Zhang et al. 2022). More theory and research are called for when thinking about plasticity factors.

This study has several strengths. First, by utilizing a dimensional approach to studying adversity, it was possible to distinguish differential associations of distinct experiences, threat, and deprivation with, respectively, cognitive deficits and problem behavior. Second, reliance on the influence statistic DFBETAS once again enabled the evaluation of the degree to which individual children appeared susceptible to the adversity effects investigated in order to determine the extent to which children affected by one experience were or were not affected by the other in a similar way and to a similar extent. Lastly, this study benefitted from its large sample size and longitudinal design. Recall, as well that it was preregistered.

Some limitations should also be noted. One potential limitation is that the analytic approach doesn't completely account for measurement error in the susceptibility scores. Composite scores were used in this report to index the environmental predictors, outcomes, and the susceptibility measure. Therefore, at least to some degree it is likely that the influence-statistic estimates of susceptibility reflect measurement error rather than precise assessment of such variation. Another limitation is the majority white (i.e., ~ 98%) sample, as the findings from this study may not be generalizable to other races/ethnicities or even necessarily different geographic locales.

Results of this study also carry potential implications for intervention. Some children in our study proved more susceptible to certain environmental exposures than to others (i.e., threat and deprivation). This suggests that intervention efficacy would likely vary across individuals, as is it is known to do (Belsky & van Ijzendoorn, 2015). After all, heterogeneous effects are quite common to virtually all interventions, whether targeting children, parents, or other adults. It seems eminently, possible then that, for example, interventions that address behavioral problems may be more effective with children with a history of threat-related experiences (e.g., physical abuse) and susceptibility to them. In contrast, interventions that target executive functioning or language skills may be more appropriate for children who proved more susceptible to deprivation effects (e.g., neglect).

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Table 1. *Principal Component Weightings of Data-Reduction Analyses: (A) Threat Predictors, (B) Deprivation Predictors, (C) Behavioral Outcomes and (D) Cognitive Outcomes**

A. Threat Predictors		B. Deprivation Predictors		
Loadings		Loadings		
	Factor 1		Factor 1	Factor 2
Parental Domestic Violence	.59	Parental Cognitive Stimulation	.80	.13
Neighborhood Threat	.56	Parental Deprivation	.76	-.05
Child Physical Abuse	.51	Frequency of Visiting Places	.54	-.58
Parental Threat	.49	Parental Education	.29	.85
Eigenvalue	1.16	Eigenvalue	1.59	1.07
Variance	29.04%	Variance	39.66%	26.88%

C. Behavioral Outcomes				D. Cognitive Outcomes		
Loadings				Loadings		
	Factor 1	Factor 2	Factor 3		Factor 1	Factor 2
Hyperactivity Grade 6	.63	-.17	-.35	Verbal IQ	.63	-.48
Conduct Prob. Grade 6	.60	-.27	-.31	Performance IQ	.57	-.55
Hyperactivity Grade 3	.58	.36	-.18	Learning Diff. Grade 6	.55	.29
Conduct Prob. Grade 3	.54	.41	-.15	Learning Diff. Grade 3	.54	.31
Emotional Symp. Grade 6	.48	-.49	.26	Speech/Language Grade 3	.36	.39
Peer Problems Grade 6	.48	-.49	.26	Speech/Language Grade 6	.30	.48
Peer Problems Grade 3	.42	.40	.40			
Emotional Symp. Grade 3	.23	.31	.63			

Eigenvalue	1.95	1.17	1.13	Eigenvalue	1.54	1.11
Variance	24.34%	14.65%	14.07%	Variance	25.64%	18.43%

Notes: Conduct Prob. = conduct problems; Emotional Symp. = emotional symptoms; Learning diff. = learning difficulties; Speech/Language = speech and language difficulties.

*Only factors with eigenvalues equal to or greater than 1.0 are displayed.

Table 2. *Inter-correlation of Constructs*

Composite	Threat	Deprivation	Problem Behavior
Threat	-		
Deprivation	.11**	-	
Problem Behavior	.13**	.14**	-
Cognitive Deficits	.08**	.22**	.29**

Notes: ** = $p < .01$.

Table 3. *Regression Results of (A) Threat Effects on Problem Behavior and (B) Deprivation Effects on Cognitive Deficits.*

D. Threat Effect Model

Predictor Variables	*B*	*SE B*	*t*	*df*	*p*
Threat	.10	.02	4.20	140.91	<.001
Child Sex	-.60	.02	-27.84	478.59	<.001
Child Race/Ethnicity	.05	.04	1.56	59.97	.12
Age	.04	.01	2.59	40.94	<.05

E. Deprivation Effect Model

Predictor Variables	*B*	*SE B*	*t*	*df*	*p*
Deprivation	.04	.01	7.16	755.09	<.001
Child Sex	-.12	.01	-9.64	1681.05	<.001
Child Race/Ethnicity	.03	.02	1.60	78.83	.11
Age	.02	.01	2.83	63.87	<.01

Table 4. *Tercile Splits of Susceptibility to Threat and Deprivation Effects (based on DFBETAS)*

	Susceptibility to Threat			
Susceptibility to Deprivation	Low	Moderate	High	Total
Low	1695	1542	1439	4676
Moderate	1540	1681	1456	4677
High	1441	1454	1781	4676
Total	4676	4677	4676	14,029

Summary Statement of Continuity

The two empirical studies included in this dissertation both highlight individual differences in terms of domain-general versus domain-specific developmental plasticity by exploring susceptibility to salient environmental influences on development (e.g., parenting/peers, and threat/deprivation). Overall, this dissertation addresses whether differential susceptibility is domain-general (i.e., trait-like) or domain-specific by investigating in Paper (1) the extent to which individuals whose adolescent adjustment is most and least associated with the parenting they experienced is similarly or differentially associated with their experience with peers and whether susceptibility differed by pubertal development, and in Paper (2) whether individuals whose cognitive and social emotional development are most and least affected by different adverse developmental experiences (e.g., threat and deprivation).