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The Effects of Early Trauma and Deprivation on Human Development - From Measuring Cumulative Risk to Characterising Specific Mechanisms

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Editorial: The effects of early trauma and deprivation on human development – from measuring cumulative risk to characterising specific mechanisms

Science is not a linear process of accumulating knowledge. To the contrary, progress in understanding is most likely to occur, especially in less 'mature' disciplines, when healthy debate between opposing points of view create a dialectic in which thesis and antithesis force a new synthesis. In developmental psychopathology such tension between opposing schools of thought continue to play a vital role in driving discovery across a wide range of topics. One core question for the field has been: how do environmental exposures to different adverse experiences and events early in life produce negative outcomes many years later? The cumulative risk model – that long-term adverse outcomes are better predicted by the total number, rather than the specific nature of environmental risk exposures – has quickly established itself as the dominant thesis. In fact, it is widely accepted as a central principle in the developmental psychopathology canon. Although perhaps most famously demonstrated in the Kaiser Permanente Adverse Childhood Experiences Study ([ACES] Felitti et al., 1998), the general principle has been repeatedly confirmed in many other studies, both preceding and following that study.

Despite this considerable body of evidence, the idea that risk exposure-outcome links lack specificity may well reflect our limited knowledge about pathways and mechanisms. Developmental investigators and theorists have plenty of reasons to believe that the nature, timing and dose of adverse experiences matters. Because the developing brain anticipates certain kinds of environmental

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input at certain times, substantial deviations from what is needed and anticipated can compromise brain and behavioral development (Fox, Levitt & Nelson, 2010; Rutter & O'Conner, 2004). Nevertheless, until the pathophysiology of adversity is better delineated, the power of cumulative risk remains easier to demonstrate.

To advance our understanding of links between risks and outcomes, developmental psychopathologists are increasingly focusing on the specific neurocognitive and neurobiological mechanisms that mediate the risk-outcome pathways as they try to understand if and why particular exposures predispose to particular outcomes – especially the way brain and biological systems are disrupted by deprivation and trauma. For instance, several investigative groups have suggested that adverse experiences can be conceptualised by distinguishing between inadequate input (neglect/deprivation) and harmful input (threat/abuse/violence exposure). Consistent with this view, Humphreys & Zeanah (2015), suggested that deprivation and trauma represented distinct deviations from the expectable environment and reviewed evidence linking each to several types of psychopathology. Likewise, McLaughlin, Sheridan & Lambert (2014) proposed that deprivation and threat are distinct pathways to psychopathology through known brain circuits. Teicher & Samson (2016) recently reviewed evidence for an even greater level of specificity, suggesting that different forms of abuse can have specific effects on targeted brain regions.

The papers in this special issue, including several from landmark studies in developmental psychopathology, offer ground-breaking insights into both the specific and general risk processes that cause persistent negative effects of serious

early trauma and deprivation, approaching the task through varied methods and examining multiple levels of maladaptation in diverse samples of children and adolescents. Cumulative risk in this issue is studied prospectively in a long-term longitudinal study. Investigations involving inadequate input are represented by studies of the effects of social neglect and the deprivation involved in institutional rearing. The traumas studied in these investigations involve all types of harmful input – witnessing violence, war trauma, physical abuse, and sexual abuse. Assessments in the papers that follow include structural and functional imaging, event related potentials, neurocognitive assessments, laboratory observational paradigms, structured psychiatric interviews, as well as parent and self-report ratings.

In an important and innovative study of cumulative risk, Reuben et al. (this issue) examined adverse childhood experiences in the well-known Dunedin Study sample. They compared associations of adverse childhood experiences (ACEs) to physical, cognitive, mental and social health outcomes in adults. This unique longitudinal sample allowed them to compare adverse childhood experiences assessed both prospectively and retrospectively, overcoming the most important limitation of the well-known ACE study, which was retrospective (Felitti et al., 1998). Although the reports of numbers of adverse childhood experiences using these two methods converged moderately, they associated with outcomes differently. Specifically, prospectively assessed ACEs better predicted objectively assessed outcomes whereas retrospectively assessed ACEs better predicted subjective (self-report) outcomes. In addition to providing interesting data about

bias, this study represents an important prospective replication of the original Felitti et al. (1998) retrospective analysis, and an illuminating extension by demonstrating that even 'forgotten' adverse experiences relate to adult outcomes. The breadth, depth and length of the Dunedin study make the prospective results reported here the most important validation yet conducted of the link between childhood adversity and adult outcomes.

Kennedy et al. (this issue) report adult follow-up data from the English and Romanian Adoptees Study (Kumsta et al., 2015) regarding attention deficit hyperactivity disorder (ADHD). Considerable research has documented the persistence of impairing ADHD symptoms in children exposed to institutional rearing, but this is the longest longitudinal study that has prospectively examined this symptomatology in the context of early life deprivation. They showed that between 15 and 22-25 years, risk was considerably elevated for children with more than 6 months of institutional rearing, whereas those with less than 6 months exposure had no increased risk – a pattern of continuity and persistence that was perhaps even more marked than in standard clinical cases. Importantly, ADHD following early life deprivation differs from ADHD that is unrelated to deprivation in several important ways. First, persistence from adolescence to adulthood was unusually high, both with regards to symptoms and impairment. Second, males and females were equally affected in contrast with the usual male preponderance of cases. Third, the inattentive symptoms predominated. Finally, the ADHD symptomatology was associated with other deprivation related abnormalities, including cognitive impairment, disinhibited social behavior, autistic-like traits, and

callous unemotional traits. Thus, despite phenomenological similarities to ADHD that do not involve deprivation, the clinical correlates of deprivation-associated ADHD seem to diverge.

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In a short-term longitudinal study of children who had experienced neglect from early institutional rearing, Lawler et al. (this issue) assessed disinhibited social behavior longitudinally following adoption into U.S. families. They found that observed disinhibited social behavior initially increased in the months following adoption (which occurred at 16 - 36 months) and then plateaued. In keeping with this group's previous work, they also distinguished between physical and non-physical engagement with strangers. This was important because immediately after adoption, this sample of post-institutionalized children were distinguished primarily by physical rather than by non-physical contact. However, over time, physical social engagement decreased whereas non-physical social engagement increased. Children diagnosed with Disinhibited Social Engagement Disorder at 5 years of age were better predicted by the increase in disinhibited social behavior rather than by the level of disinhibited social behavior evident at adoption. This finding has important implications both for prognosis and for designing preventive interventions.

Green et al. (this issue) report results from a short-term longitudinal study of post-institutionalized school aged children who had been adopted into U.S. families at a mean of 25 months of age. At mean of 9 years of age, they were assessed with fMRI as they were exposed to trustworthy versus untrustworthy faces. They found that compared to never institutionalized children, those with histories of

institutional rearing showed reduced amygdala signal differences to trustworthy and untrustworthy faces. Further, reduced differentiation of faces predicted separation anxiety symptomatology two years later. Interestingly, the post-institutionalized children had greater differential responses in cortical face processing areas, in contrast with the amygdala findings, compatible with increased higher order, top-down face processing to differentiate trustworthiness in this group.

In another examination of deprivation effects, Troller-Renfree et al. (this issue) also report on children exposed to early life deprivation in their contribution from the Bucharest Early Intervention Project (Nelson, Fox & Zeanah, 2014). They found impaired error monitoring in children with histories of institutional rearing, with smaller amplitudes of error related negativity (ERN) in event related potentials in children with more exposure. Children randomized to care as usual (who had more institutional exposure), were significantly different from never institutionalized children whereas children randomized to foster care (who had less institutional exposure) were not significantly different. At lower amplitudes of ERN, time spent in institutional care was related to externalizing and ADHD symptomatology but at higher amplitudes of ERN there was no association between time in institutional care and ERN. These findings add to the growing body of research implicating early life social neglect and executive functioning impairment and psychopathology.

Turning to harmful threatening input, Gold et al. (this issue) demonstrate structural abnormalities in the cortex of adolescents with a history of strictly

defined physical or sexual abuse. They found abuse associated with cortical thinning in a number of regions in the frontal and temporal lobes. There were no reductions in gray matter volume or cortical surface area, however. They also found that parahippocampal gyrus thickness was inversely related to externalizing symptomatology, replicating findings of other groups and pointing to an important plausible mediator the effects of physical and sexual abuse on externalizing behavior.

Similarly, Puetz et al. (this issue) demonstrated that maltreated children showed selective hypoactivation in brain circuitry previously implicated in abuse related PTSD symptomatology during exposure to rejection words but not during a classic color Stroop test. The children studied were mixed with regard to maltreatment – having experienced both abuse and neglect, but interestingly, the self-report data for the maltreated and nonmaltreated groups were significantly different for amount of neglect but not for abuse. The authors make the case that the hypoactivation is evidence for an avoidant coping style and in keeping with other data may represent a vulnerability to subsequent internalizing psychopathology. The cross sectional design of the study needs to be bolstered by longitudinal exploration, but these findings raise important questions to pursue.

Humphreys et al. (this issue) assessed children's attentional biases to fearful faces and related them to parent reports of their children's exposure to trauma before the age of 6 years. Even after accounting for later occurring traumas, children with early traumas showed an attention bias away from fearful faces but showed no differences in attention to happy or sad faces. The investigators also

showed that avoidance of fearful faces mediated the association between traumatic events in early childhood and later social withdrawal. These findings are interesting to consider in the context of research on attention bias to threat, which consistently has shown angry faces lead to biases towards the threat following trauma. Angry faces seem to represent direct threat whereas fearful faces may represent an indirect threat. In any case, it seems that processing of fearful faces may differ from angry faces, though both seem to relate to risk for psychopathology.

Halevy et al. (this issue) report results from a longitudinal study of severe trauma in young children with the longest follow-up of PTSD symptoms reported to date. It is also the only longitudinal study of repeated war trauma in children. They found remarkably high rates of psychopathology (PTSD, anxiety disorders, disruptive behavior disorders, and ADHD) and co-morbidity in children exposed to war trauma. Importantly, they documented that mothers' emotional distress and fear eliciting behavior during trauma evocation were related to early and to chronic symptomatology. Social engagement of the child during interactions with mother predicted psychopathology in late childhood. A next step will be developing interventions that address the factors associated with different symptom profiles.

Treatment of traumatized Zambian children – many of whom were social or actual orphans – with trauma focused cognitive behavioral therapy is reported by Kane et al. (this issue). Despite the challenging sample and setting, this randomized controlled trial was very effective in reducing trauma symptoms and somewhat effective in enhancing functioning in these children. Children with parents caring for them were more responsive to treatment and more likely to demonstrate enhanced

functioning. Importantly, after application of a number of statistical controls, the authors demonstrated that a history of sexual abuse moderated treatment response, limiting both trauma symptom reduction and enhancement in functioning. This calls for augmented treatments when traumas include sexual abuse.

This set of papers provide compelling cross-sectional and longitudinal evidence that early experiences of inadequate input (neglect/deprivation) and unwanted input (threat/trauma) – remembered or not – lead to long-term developmental and clinical abnormalities. They also add to a growing body of evidence about the neurodevelopmental pathways involved in psychopathology. Further, a number of papers demonstrate that the presence and behaviors of caregivers moderate the effects of early adversity and response to interventions. Our hope is that this special issue provides a preliminary platform for synthesis between models of general and the specific risk processes. A definitive model of the delimiting pathophysiological mechanisms underlying psychiatric disorders and impaired social maladaptation is not yet within reach. Nevertheless, studies like those in this special issue are beginning to make important inroads. By extending questions to brain and biological systems impacted by deviations from the expectable environment, this research has moved us from delineating morbidity to characterizing mechanisms.

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Charles H. Zeanah and Edmund Sonuga-Barke

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