Commentary: Childhood Abuse: New Insights into its Association with Posttraumatic Stress, Suicidal Ideation, and Aggression

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The publication of the Battered Child Syndrome (Kempe, Silverman, Steele, Droegemueller, & Silver, 1962) opened our eyes to the large number of abused children. With this awareness came the dawning realization that exposure to early adversity is a major risk factor for psychopathology and poor health. The most sobering statistics arise from the Adverse Childhood Experiences Study. Based on retrospective reports from 17,337 adult HMO members they concluded that exposure to early abuse and adversity accounted for 50–78% of the population attributable risk for drug abuse, depression, alcoholism, and suicide attempts [see (Anda et al., 2006) for a review]. Further, early adversity leads to the adoption of unhealthy habits (e.g., smoking, risky sex) associated with premature death. To paraphrase Fellitti, ''*what we recognize as common disorders in adult medicine and psychiatry are likely the result of what we fail to recognize or address in childhood (Felitti, 2002).*''

Developmental traumatology is still in its infancy. We do not understand why exposure to abuse appears to profoundly affect some individuals but not others, or why it may be associated with posttraumatic stress disorder (PTSD) in some; depression, substance abuse, or antisocial behaviors in others still. The articles in this issue bring new findings to bear on these questions.

Cicchetti and colleagues (2010) addresses two key factors likely to influence outcome: genetic polymorphisms and the number of different types of adversity endured. They provided evidence for a gene × environment (G × E) interaction involving abuse, the serotonin transporter promoter polymorphisms (5HTTLPR), and suicidal ideation. They found that the short form of the 5HTTLPR gene conveyed vulnerability while two copies of the high efficiency long form provided protection. However, this protective effect was only apparent in individuals with moderate levels of exposure, but not in individuals exposed to three or four different types of abuse. Dose-dependent protection also emerged in a recent paper by Weder et al., (2009). Hence, it is likely that some polymorphisms act to right or left shift the dose-response relationship between trauma and outcome, predominantly affecting individuals with moderate levels of exposure.

Gordis and colleagues (2010) explored the hypothesis that autonomic nervous system (ANS) activity buffers the effects of maltreatment on aggression. They presented interesting findings that presume that the ANS was unaffected by abuse. This premise is reasonable as they failed to observe ANS differences associated with maltreatment. However, there is an important alternative. Exposure to early stress may program some individuals to have an enhanced sympathetic fight–flight reaction (Heim et al., 2000), and others to have a blunted response (Macmillan et al., 2009). We suspect that heightened sympathetic responses emerge in situations where fight or flight is adaptive. In contrast, a submissive parasympathetic response may be lifesaving in other situations. Hence, the association between ANS and aggression may depend on the adaptive programming of the ANS to foster aggressive, avoidant, or submissive responses.

Carrión and colleagues (2010) and De Bellis (2001) focused on the relationship between abuse, PTSD, and hippocampal function. This is an intriguing area as at least five studies have shown an association between childhood abuse and reduced hippocampal volume in adults while at least three studies have failed to find a similar association in children. It is likely, as we proposed (Teicher, Andersen, Polcari, Anderson, & Navalta, 2002),

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that exposure to early stress affects trophic factors and results in developmental differences in synaptic overproduction (Andersen & Teicher, 2004) leading to changes in hippocampal morphometry by late adolescence or early adulthood (Andersen & Teicher, 2004; Andersen et al., 2008). However, it is probable that alterations in hippocampal function predate differences in morphometry.

Carrión et al. (2010) found that youths with symptoms of PTSD had a diminished degree of right hippocampal activity during memory retrieval. Further, left hippocampal deficits during memory retrieval correlated with avoidance and numbing symptoms of PTSD. These findings dovetailed with the hierarchical regression analysis of De Bellis who reported that deficits in visual memory significantly enhanced the regressive relationship between exposure to abuse and symptoms of PTSD. These findings support a developmental traumatology model that posits that maltreatment leads to PTSD symptoms through effects on stress response systems and their effects on brain and cognitive development. It is important to note, however, that visual memory deficits need not be hippocampal. For instance, we recently reported that 18- to 22-year-olds exposed to repeated episodes of childhood sexual abuse (CSA) had substantial reductions in gray matter volume (GMV) in right and left visual cortices, which correlated with visual memory performance (Tomoda, Navalta, Polcari, Sadato, & Teicher, 2009).

These studies cover a great deal of territory but there is much that remains to be pursued. First, the developmental traumatology model (De Bellis, 2001) may be insufficiently developmental. As we proposed in our similar cascade model (Teicher et al., 2002), brain regions will likely differ in their sensitivity to the effects of abuse, based, in part, upon development stage. In support of this hypothesis, we provided evidence for sensitive periods when specific brain regions were maximally susceptible to the effects of CSA (Andersen et al., 2008). This finding needs to be replicated and extended to other brain regions. The temporal intersection of abuse with sensitive periods may help explain why different disorders emerge in different individuals.

Developmental sensitive periods also provide a potential mechanism for G × E interactions. We hypothesize that the magnitude of sensitive period affects depends on polymorphisms regulating monoaminergic stress responses. Polymorphisms effecting trophic factors may influence the timing of sensitive periods. This needs to be studied and alternative models pursued to delineate the mechanisms underlying G × E interactions. Another issue requiring explanation is the strong dose–response relationship between outcome and number of different types of adversity. One explanation is that different types of abuse act as stressors to exert similar effects on corticolimbic development, but also exert unique effects on sensory systems that receive and process the adverse events (Teicher, Tomoda, & Andersen, 2006). This hypothesis fits with our emerging observations that CSA, parental verbal abuse, and physical maltreatment are associated with different regional effects on GMV and fiber tract integrity (Choi, Jeong, Rohan, Polcari, & Teicher, 2009; Tomoda, Suzuki et al., 2009).

Overall, findings on the neurobiological effects of abuse parallel preclinical observations on the effects of early stress (Teicher et al., 2006) and highlight the benefits of translational research. Even more remarkable is how well these studies bridge the chasm between developmental psychology and biological psychiatry to provide a true biopsychosocial synthesis.

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References

Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., et al. (2006). The enduring effects of abuse and related adverse experiences in childhood: A convergence of evidence from neurobiology and epidemiology. *European Archives of Psychiatry and Clinical Neuroscience, 256*(3), 174–186.

Andersen, S. L., & Teicher, M. H. (2004). Delayed effects of early stress on hippocampal development. *Neuropsychopharmacology, 29*(11), 1988–1993.

Andersen, S. L., Tomoda, A., Vincow, E. S., Valente, E., Polcari, A., & Teicher, M. H. (2008). Preliminary evidence for sensitive periods in the effect of childhood sexual abuse on regional brain development. *The Journal of Neuropsychiatry and Clinical Neurosciences, 20*(3), 292–301.

Carrión, V. G., Haas, B. W., Garrett, A., Song, S., & Reiss, A. L. (2010). Reduced hippocampal activity

in youth with posttraumatic stress symptoms: an fMRI study. *Journal of Pediatric Psychology, 35*, 559–569.

Choi, J., Jeong, B., Rohan, M. L., Polcari, A. M., & Teicher, M. H. (2009). Preliminary evidence for white matter tract abnormalities in young adults exposed to parental verbal abuse. *Biological Psychiatry, 65*(3), 227–234.

Cicchetti, D., Rogosch, F. A., Sturge-Apple, M., & Toth, S. L. (2010). Interaction of child maltreatment and 5-HTT polymorphisms: suicidal ideation among children from low-SES background. *Journal of Pediatric Psychology, 35*, 536–546.

De Bellis, M. D. (2001). Developmental traumatology: The psychobiological development of maltreated children and its implications for research, treatment, and policy. *Development and Psychopathology, 13*(3), 539–564.

Felitti, V. J. (2002). The relationship of adverse childhood experiences to adult health: Turning gold into lead. *Zeitschrift für Psychosomatische Medizin und Psychotherapie, 48*(4), 359–369.

Gordis, E. B., Feres, N., Olezeski, C. L., Rabkin, A. N., & Trickett, P. K. (2010). Skin conductance reactivity and respiratory sinus arrhythmia among maltreated and comparison youth: relations with aggressive behavior. *Journal of Pediatric Psychology, 35*, 547–558.

Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., et al. (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *JAMA: the Journal of the American Medical Association, 284*(5), 592–597.

Kempe, C. H., Silverman, F. N., Steele, B. F., Droegemueller, W., & Silver, H. K. (1962). The battered-child syndrome. *JAMA: the Journal of the American Medical Association, 181*, 17–24.

Macmillan, H. L., Georgiades, K., Duku, E. K., Shea, A., Steiner, M., Niec, A., et al. (2009). Cortisol response to stress in female youths exposed to childhood maltreatment: Results of the Youth Mood Project. *Biological Psychiatry, 66*(1), 62–68.

Teicher, M. H., Andersen, S. L., Polcari, A., Anderson, C. M., & Navalta, C. P. (2002). Developmental neurobiology of childhood stress and trauma. *The Psychiatric Clinics of North America, 25*(2), 397–426.

Teicher, M. H., Tomoda, A., & Andersen, S. L. (2006). Neurobiological consequences of early stress and childhood maltreatment: Are results from human and animal studies comparable? *Annals of the New York Academy of Sciences, 1071*, 313–323.

Tomoda, A., Navalta, C. P., Polcari, A., Sadato, N., & Teicher, M. H. (2009). Childhood sexual abuse is associated with reduced gray matter volume in visual cortex of young women. *Biological Psychiatry, 66*(7), 642–648.

Tomoda, A., Suzuki, H., Rabi, K., Sheu, Y. S., Polcari, A., & Teicher, M. H. (2009). Reduced prefrontal cortical gray matter volume in young adults exposed to harsh corporal punishment. *Neuroimage, 47* (Suppl 2), T66–T71.

Weder, N., Yang, B. Z., Douglas-Palumberi, H., Massey, J., Krystal, J. H., Gelernter, J., et al. (2009). MAOA genotype, maltreatment, and aggressive behavior: The changing impact of genotype at varying levels of trauma. *Biological Psychiatry, 65*(5), 417–424.