Childhood adversity as the harbinger for future affective disorders and health problems

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DESCRIPTION

Among the several factors contributing to depressiveness among children, maternal alcohol use/abuse, prenatal smoking habit and prenatal maternal obesity and other family-based poor health encumbrances remain a consideration although modulated by social and peer associations, childhood adversity and socioeconomic status (1-7). Adverse exposure to psychosocial traumatic events during the early part of the child's life, which incorporates negative experiences involving child maltreatment, caregiver stress and/or depression, and domestic or community violence, with wide-ranging effects on neural, endocrine, immune, and metabolic physiology, bear strong relationships within epidemiological studies with an elevated lifetime risk of adverse health outcomes, such as diabetes, heart disease, cancers, and psychiatric illnesses (8). The dysregulation and disharmony of brain neural networks, neuroendocrine stress regulation disruptions, and chronic inflammation, as well as a number of other pathophysiological factors predispose the developing individuals to innumerable health hazardous disorder and risk factors across the life course. In the context of obesity, prenatal stress, adversity among mothers, was linked to reduced levels of childhood adiposity although their cord blood interleukin-6 methylation was related to an increased level of childhood adiposity among Mexican children (9). Incremental adverse childhood experiences each reduce the infant's birth weight by 16.33 g and decline its gestational age by 0.063 with smoking emerging as the strongest mediator of the effect on gestational age thereby causing detrimental influences upon maternal reproductive health and well-being, as manifested by mothers' delivery of the newborn infants who were of reduced birth weight and shorter gestational age (10). Childhood adversity has been shown to be linked to some 10% elevated risk for developing smoking behavior during adulthood with both childhood adversity and smoking behavior during adulthood related to greater levels of anxiety and depressive symptoms in adulthood (11). Trauma during childhood, nicotine dependence combined with a greater prevalence for previous hospitalizations was observed among inpatients presenting for both cardiovascular diseases and the positive screening for depressive illness (12).

Several aspects of childhood adversity are associated with later-life health problems and neurodevelopmental detriment. For example, demographic features and depression, such as the long-lasting fear of a family member, long-lasting financial difficulties within the family, serious conflicts in the family, parental divorce and separation, serious or chronic illnesses in the family and alcohol and/or problems within the family were shown to be harbingers for fibromyalgia, expressed through widespread pain and a variety of somatic symptoms (13). Childhood maltreatment and trauma presents also an increased risk for a pre-diabetic status which is substantiated by the affected individuals' glucose intolerance, impaired insulin sensitivity, increased levels of C-reactive protein and tumor necrosis factor-α levels, and beta cell function dysregulation (14). In this regard, it has been shown that omega-3 composition and general trauma during childhood estimates had no relationship to each other although both factors were correlated, in the predicted directions, with negative emotionality with low omega-3 composition and a history of childhood trauma related to the persistence of depressive symptoms during follow-up analysis (15). It has been found that telomeric length is reduced in individuals exposed to psychosocial stress and in those with depression (16,17). In male and female participants screened for stressors, depressive status, social interaction, coping strategies and telomere length, it was observed that depressive status and age had direct negative effects on telomere length and both stressors in childhood and recent adulthood had indirect negative effects on telomere length among the female participants whereas for mae participants the effects of stressors and depressive status on telomere length were mediated by social interaction and the coping strategy worry for failure (18). Childhood adversity, e.g. those traumatic events experienced or perceived violence, low socioeconomic status, maternal depression, family disruption, and institutionalization, all exert an impact on telomere length implying exposed individuals show signs of accelerated erosion of telomeric ends from an early age. Telomere shortening relates to negative health outcomes later on in the lives of these individuals as well as presenting a biomarker to predict health and longevity outcomes (19). Finally, among patients presenting major depressive disorder it was observed that the exposure to both early and recent stressful environments exerted a widespread effect on white matter microstructure, i.e. reduced fractional anisotropy and axial diffusivity, with the latter correlating negatively with adverse childhood events and the latter with recent stressful events (20).

Adverse childhood experiences exerted a direct effect on depression and relational resilience, i.e. the development of mutually empowering, growth-fostering connection in the face of adverse conditions, but not upon individual, i.e. behaviors, thoughts, and actions that promote personal wellbeing and mental health, or contextual, i.e. the influence of environment (context) environment with associated capacity to facilitate growth, resilience (21). It has been observed that ongoing and life-long expressions of "deliberate self-harm" occurred more frequently among children presenting depression, attention-deficit/hyperactivity disorder and/or oppositional defiant disorder, and also in the context of multiple disorders models accounting for demographic variables and co-occurring neuropsychiatric disorders (22). They have found that the incidence of maternal anxiety was associated strongly with ongoing and life-long expressions of "deliberate self-harm" in their children, and alternative, among small children particularly expressions of "deliberate self-harm" were related to the mothers' mood disorders. Similarly, parental adverse childhood experiences were important in the experiences of development of eventual offsprings' psychological problems (23). It has been found also that the inattentional component of attention-deficit/hyperactivity disorder and executive functioning problems were associated with childhood depression according to parental ratings (24). Longitudial analyses of childhood suicide attempters has proved evidence of three types of patient: (i) those individuals presenting all low depressive/anxious (n=32%), (ii) those presenting all high depressive/anxious (n=16%), and (iii) those presenting high depressive/anxious combined with low aggressive (n=52%) symptom-profiles (25). Individual presenting the highest likelihood of all the symptom-profiles evidenced a greater probability of reporting younger age-levels for suicide attempts accompanied by substance abuse disorders and violent criminal behaviors.

A marked positive relationship between a variety of adverse life events during childhood and prenatal depression scores among women presenting perinatal depression combined with excessive adrenocorticotropic hormone reactivity to the cold-pressor test has been indicated (26). Furthermore, those patients presenting a history of any type of adverse life event description displayed a blunted cortisol response to the cold-pressor test and at four

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weeks showed a heightened adrenocorticotropic hormone stress reactivity to the test. The cold-pressor test presents a cardiovascular performance estimate by immersing the individual's hand into an ice water container, often for one minute, and measuring changes in blood pressure and heart rate with eventual changes relating to vascular responding and pulse excitability. Additionally, the authors observed a positive and markedly strong correlation between pain tolerance estimates and the cortisol response in the cold-pressor test over the whole sample. It was shown also that among female patients presenting a history of childhood onset depression but no evidence of anxiety disorder (n=37) there was a reduction in electroencephalogram left lateral frontal activity in comparison with a group of psychiatrically-healthy control individuals (27) whereas those women patients presenting a history of childhood-onset depressive disorder with pathological levels of anxious apprehension estimated through concurrent generalized anxiety disorder, obsessive compulsive disorder, or separation anxiety disorder diagnoses showed profiles that were indistinguishable statistically from the healthy control group. The authors have presented three conclusions: (i) prefrontal electroencephalogram activity asymmetry provides a neurophysiological marker for depressive illness, (ii) the co-morbidity inherent to depression and anxiety expressions, and (iii) the failure to observe the prevailing relationship between prefrontal electroencephalogram activity asymmetry and levels of diagnosed depression. The children's mothers' history of childhood maltreatment and adversity was shown to bear a marked relationship to their offsprings' internalizing and externalizing problems while maternal antenatal depression, postnatal depression and offspring child maltreatment were indicated as mediating significantly this relationship independently (28). In a self-report study of rumination, sadness and anger, and depressiveness among children (aged 7–14 years), it was found that anger rumination presented a trans-diagnostic factor for the depressive symptoms and aggressiveness of the children whereas sadness moderated the association between anger rumination and aggression (29).

Perinatal stress-related/inflammatory symptoms are common in women during pregnancy and are risk factors for neurobehavioral disorders, particularly affective and associated co-morbidities and health hazards (30). Exposure to childhood trauma and adverse environments, mediated by dysregulation of hypothalamic-pituitary-adrenal axis function and a plethora of stress hormone and neuro-immune mobilization of biomarkers, appears to impair a multitude of psychophysiological and health-benefitting process to the eventual detriment of individuals over the life-span.

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