Childhood Poverty and Parental Stress: Important Determinants of Health

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abstract

The following is a literature review examining the interaction between early–life stress and child development. First, evidence showing that early–life experiences can affect the cognitive abilities, behaviour, and health of an individual are reviewed. Next, possible mechanisms by which these changes occur are explained. These mechanisms include toxic stress–mediated dysregulation of the hypothalamic–pituitary–adrenal axis and epigenetics. Finally, up–and–coming research related to early–childhood interventions that target family stress relief and the child–parent relationship are touched upon. These interventions may impact the negative effects of toxic stress on cognition and health.

early–life poverty

Extensive research on the consequences of child poverty over the past two decades has demonstrated that chronic poverty is a significant mediator of the future quality of life of children from both a cognitive and physical health perspective. Persistent low socioeconomic status (SES) during childhood is consistently associated with lower academic achievement and more maladaptive socioemotional functioning.[1-3] Moreover, the cumulative number of years spent in poverty during childhood is more predictive of academic outcomes than acute poverty at a certain age.[4] Evidence from twin populations suggests that this association between chronic poverty and decreased cognitive ability persists even when genetic contributions are controlled.[5] It has been shown that poverty in early–life not only affects the immediate childhood period but also has a significant role in diseases previously thought to be caused by adult behaviours. For example, in a study of over 1100 physicians followed for a median of forty years, those with a low childhood SES had a 2.4 times increased risk of developing coronary heart disease (CHD) by age 50 independent of other CHD risk factors, compared to those who did not grow up in poverty.[6] Thus, reducing rates of child poverty may help to improve both the cognitive abilities and physical health of children throughout their lifespan.

the child–parent relationship and parental stress

In addition to chronic poverty, many large studies have demonstrated the importance of the child–parent relationship and parental stress in life outcomes. Child–parent interactions are essential to learning experiences in the home and have been found to account for up to half of the beneficial effects of both income–assistance and educational interventions on the cognitive development of children with low SES.[7,8] Moreover, the Minnesota Longitudinal Study of Risk and Adaptation—a landmark study for child development that followed individuals from birth until adulthood over 40 years—showed that measures of family stress and social support were two of the strongest predictors for life outcomes.[9] For instance, among kindergarteners who were anxiously attached to their primary caregiver as infants, the most important factor in predicting improved function over the school year was increased social support for their primary caregiver. More recent studies have corroborated these findings; they show that measures of parental stress were strong predictors of maladaptive externalizing and internalizing behaviours in children as well as decreased school engagement.[10,11] This research provides important recommendations for practice and policy. Firstly, interventions should be started at a young age if possible given the enduring influence of early relationships.[9-12] Secondly, instead of targeting the child in isolation, a complex interventional approach is required that focuses on both the child–parent relationship and the recruitment of social and community supports for parents to alleviate parental stress.

possible mechanisms of embedding experiences

The National Scientific Council on the Developing Child and the American Academy of Pediatrics use a model called "toxic stress" to explain the long–term effects of childhood adversity on the body.[13,14] Toxic stress is defined as stress that is chronic and uncontrollable and/or stress that is experienced in the absence of support from caring adults; this can cause changes to the developing brain as well as dysregulation of the stress response system. Risk factors for toxic stress include chronic extreme poverty, child abuse/neglect, maternal depression,

parental substance misuse, and family violence. The health impact and mechanisms of toxic stress associated with dysregulation of the stress response system and up-regulation of inflammation have been well documented.[15-17] In addition, numerous studies have shown that adrenocortical and pro-inflammatory mediators were up-regulated in individuals suffering from chronic stress related to low SES, maltreatment, and social isolation.[18] This was seen in a cross–sectional study of over 200 Quebec school children where morning cortisol levels were found to be negatively correlated with SES.[19] Moreover, independent of current SES, lifestyle practices, and perceived stress level, adults with low early–life SES were found to have increased baseline daily cortisol levels, increased pro-inflammatory mediators, and significantly up-regulated genes associated with the stress response and inflammatory systems compared to adults raised in higher SES households.[18] In addition to the model of toxic stress–mediated dysregulation of the stress response system, another plausible explanation for the biological embedding of early–life experiences is seen in the field of epigenetics.

Multiple excellent reviews explain epigenetics and its role in child development.[20-25] In short, epigenetic mechanisms, such as DNA methylation and histone modification, alter gene expression by modifying the accessory structures of the genome to facilitate or block gene transcription within the lifetime of the individual. The child–parent relationship and SES–related aspects of the prenatal and childhood environments have been found to be associated with persistent changes in epigenetic patterns. For example, 60 years after being prenatally exposed to famine, study subjects were found to have different DNA methylation patterns of a gene linked closely to growth and development compared to their same–sex siblings who were not exposed.[26] Similarly, researchers have repeatedly found that DNA methylation patterns were more associated with childhood SES than adulthood SES.[27,28] For instance, McGowan et al. found increased methylation of the promoter of the glucocorticoid receptor in the hippocampus of suicide victims who had been abused as a child but not in suicide victims who had not been abused or in the control group (no suicide).[29] Thus, childhood abuse had altered an aspect of the hypothalamic–pituitary–adrenal axis function and perhaps increased the susceptibility of these individuals to the effects of stress in adulthood. To support these retrospective associations, the first longitudinal prospective study on the connection between epigenetic changes and childhood adversity was published in 2013; this study involved 109 children from Wisconsin who were followed from birth until age 15.[30] Researchers examined 14,000 genes associated with environmental stress or behaviour and found that a composite score of parental stress measured in infancy and preschool was predictive of changes in DNA methylation at age 15. In conclusion, it is likely that epigenetics is one of the mechanisms by which prenatal/childhood experiences and parental stress can affect the future behaviour and health of children.

psychosocial interventions

The major focus of most large–scale early childhood intervention programs (ECIPs) in North America has been cognitive development.[31] However, as presented above, the current body of evidence shows that future cognitive and physical health can by significantly modified by toxic stress and the child–parent relationship. Therefore, experts in child development suggest that ECIPs are more beneficial when they target family stress relief, attachment, and education.[14,31] Traditional programs providing only low–income subsidies to combat family stress have led to a small reduction in cumulative poverty risk; however, they were not able to significantly affect the psychosocial aspects of at–risk families, including maternal depression, food insufficiency, parenting stress, and parenting behaviours.[32] ECIPs using relationship–centered therapies to alleviate family stress have been shown to be effective at improving attachment and behavioural problems, as well as biomarkers of chronic stress.

Examples of ECIPs that take a relationship–centered approach include those used by Cicchetti et al.: child–parent psychotherapy (CPP) and psycho-educational parenting interventions (PPI).[33] CPP is an attachment–based therapy that recognizes multi-generational trauma and a lack of parenting skills. Therefore, it tries to provide a "corrective emotional experience" by helping parents "form positive representations of themselves and the caregiver–child relationship".[33] PPI is a didactic intervention that teaches parents how to access social supports, including education and employment, and parenting skills to reduce parental stress. Both CPP and PPI were found to substantially improve attachment profiles of maltreated infants after 12 months of treatment in comparison to those maltreated children whose families received the standard services and to non-maltreated children.[34] Moreover, attachment/relationship–based therapies, including CPP and PPI, have been shown to normalize cortisol levels in maltreated and foster children.[17,33,35] Taken together, these studies suggest that disruptions in attachment and the HPA axis due to early–life adversity may be prevented or reversed by interventions designed to improve caregiving and the child–parent relationship. Therefore, adding interventions to educational programs that focus on strengthening the child-parent relationship, along with alleviation of poverty, may improve outcomes for at-risk children.

conclusions

In summary, chronic poverty, parental stress, and poor child–parent relationships can have a significant negative impact on future cognitive abilities, behaviour, and physical health through mechanisms, such as toxic stress and epigenetics. As a result, interventions that take a multi-generational approach to support the child and parent individually, as well as in their relationship, may provide some added benefit to current ECIPs. It is important to note that the term, "early," used in this article is not restricted to pre-adolescence. The teenaged years are a critical time in development with ample opportunity for harm as well as intervention, but adolescent–specific research was not discussed in this article. For a comprehensive review of the entire topic, please see the 2012 report by the Royal Society of Canada and the Canadian Academy of Health Sciences expert panel in Early Childhood Development led by Dr. Clyde Hertzman and Dr. Michel Boivin.[36]

In addition to improved, evidence–based ECIPs, it is imperative that child poverty and the many other social determinants of health of children and adolescents are made a priority. Notably, the Canadian Medical Association has identified that a main priority to improve health across Canada is to create federal and provincial/territorial level action plans to eliminate poverty.[37] Other important social determinants of child and adolescent health include access to education and housing, multigenerational trauma, income inequality, racial/ethnic disparities, and community violence. To start addressing these issues, numerous evidence–based initiatives have been suggested, such as a minimum wage that reflects the cost of living, national food security program, universal publicly–funded child care, affordable housing for low– and middle–income families as well as those with mental illness, and a focus on marginalized populations.[38] Obviously, there is no simple solution to these societal problems; however, better support for children/adolescents and their parents will be essential to any effective plan.

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