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The influence of proximal risk on the early development of the autonomic nervous system

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ABSTRACT

In this paper we review the evidence linking the early development of the autonomic nervous system in early childhood to four proximal risk factors: maternal stress during pregnancy, maternal substance use during pregnancy, poor-quality parent–child interactions, and specific disruptions in parenting behavior. A clear pattern of altered autonomic function emerges in children exposed to proximal risk, marked by reduced parasympathetic tone under conditions of both homeostasis and challenge, accompanied by increased sympathetic tone in some cases. This pattern of autonomic activity would support increased vigilance and active defense responses, which, in an environment of high proximal risk, may be adaptive in the short-term. However, in the long-term maintaining such a state may impose a high allostatic load. The current paper reviews and interprets the current literature and discusses future directions.

Introduction

The link between environmental risk and poor developmental outcomes is one of the most robust findings in the developmental literature (e.g., Brooks-Gunn & Duncan, 1997; Buckner, Mezzacappa, & Beardslee, 2009; Pungello, Kupersmidt, Burchinal, & Patterson, 1996). This association has led to an effort to understand how environmental risk may "get under the skin" by affecting physiological or biological change (Taylor, Repetti, & Seeman, 1997, p. 411). In this review we summarize the evidence supporting one link in the causal chain from environmental risk to developmental outcome: the influence of proximal risk on the development of the autonomic nervous system in early childhood, which

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together with the hypothalamic–pituitary–adrenal (HPA) axis, underlies self-regulation in infants and toddlers (Porges, 2001) and lays the foundation for the emergence of more sophisticated forms of behavioral, emotional, and cognitive self-regulation later in childhood and into adulthood (Calkins & Marcovitch, 2010).

According to ecological (Bronfenbrenner & Morris, 1998), transactional (Sameroff & Chandler, 1975), and family-stress (Conger & Elder, 1994) theories, environmental or distal risk factors achieve their effects, in part, through their impact on proximal systems, such as the family (Brooks-Gunn & Duncan, 1997; Burchinal, Roberts, Hooper, & Zeisel, 2000; Duncan & Brooks-Gunn, 2000; Gerard & Buehler, 2004; Pungello et al., 2010; Sameroff, Seifer, Baldwin, & Baldwin, 1993). Accordingly, a number of studies have found that levels of proximal or familial risk mediate the relationships between distal/environmental risk and a variety of outcomes (Brody, Kim, & Murry, 2003; Jones, Forehand, Brody, & Armistead, 2002; Krishnakumar & Black, 2002; Luster & McAdoo, 1994; National Institute of Child Health, 2005; Trentacosta et al., 2008). By reviewing how exposure to proximal risk across multiple stages of development (in *utero*, neonatal, infancy, and very early childhood) may influence the development of the autonomic nervous system, we hope to inform empirical work designed to validate, explore, and ultimately change the course of the pathways from environmental to proximal risk, from proximal risk to physiological functioning, and finally from physiological functioning to developmental outcome.

The autonomic nervous system

The human nervous system is divided into the central and peripheral nervous systems, and the peripheral nervous system can itself be divided by volition, into somatic and autonomic components. The autonomic nervous system (ANS) can in turn be further divided into two branches: the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS). Early research employed "global constructs" (Porges, 1995), such as heart rate (HR), in an attempt to measure autonomic activity. However, as Mezzacappa et al. (1997) point out, increased HR may be a function of increased sympathetic input or decreased parasympathetic input; alternatively, reductions in HR could be a function of increased parasympathetic input or decreased sympathetic activity. Thus, a recent direction within this area of research has been the use of more precise measures and analytical tools to elucidate the independent activity of the PNS and SNS.

The activity of the PNS is most often indexed by vagal tone, or the neural control of the heart via the vagus nerve. Vagal tone is itself measured by respiratory sinus arrhythmia (RSA), or high frequency variability in the inter-beat interval (IBI) of the heart (Porges & Byrne, 1992). RSA is frequently used as a direct measure of vagal tone (see Grossman & Taylor, 2007) even though it is in fact only one of many influences on vagal tone. To reconcile the inconsistencies among studies in the terminology used to describe similar processes (i.e., vagal tone, heart rate variability, RSA) across studies, the current paper will use the term 'vagal tone' or 'vagal functioning' in order to draw comparisons across studies and to facilitate ease of reading and interpretation (see Appendix for additional information on how vagal tone was operationalized and quantified in each study).

Baseline vagal tone is considered to be a stable neurophysiological mechanism underlying autonomic and behavioral reactivity, and thus provides a measure of resting state in the absence of environmental challenge. Typically, the greater the variability of the heart's IBI (i.e., higher vagal tone, RSA, or heart-rate variability) at baseline, the greater the response potential or possible range of behavior, leading to more adaptive regulation. For example, findings reveal that higher resting vagal tone during infancy is associated with less temperamental difficulty (Stifter & Fox, 1990), secure attachment (Izard et al., 1991), more sociable and explorative behavior (Fox, 1989; Stifter, Fox, & Porges, 1989), and greater behavioral reactivity (Porges, Doussard-Roosevelt, Portales, & Suess, 1994). However, during times of environmental stress, these internal processes are disrupted and vagal tone (or the vagal 'brake' on cardiac output) is withdrawn to support an increase in heart rate. When environmental demands have ceased, vagal tone is increased (i.e., the vagal 'brake' is reengaged) to promote decreases in metabolic output and a return to a calm state. Thus, effective vagal functioning has been related to the ability to maintain homeostasis in the face of situational challenge by allowing attention to shift