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Childhood adversity predicts reduced physiological flexibility during the processing of negative affect among adolescents with major depression histories

Shimrit Daches[a,*], Maria Kovacs[a], Charles J. George[b], Ilya Yaroslavsky[c], Eniko Kiss[d], Ágnes Vetró[d], Roberta Dochnal[d], István Benák[d], Ildikó Baji[d], Kitti Halas[d], Attila Makai[d], Krisztina Kapornai[d], Jonathan Rottenberg[e]

[a] Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA
[b] Department of Psychiatry, University of Pittsburgh Medical Center, Pittsburgh, PA, USA
[c] Department of Psychology, Cleveland State University, Cleveland, OH, USA
[d] Department of Child and Adolescent Psychiatry, University of Szeged, Szeged, Hungary
[e] Department of Psychology, University of South Florida, Tampa, FL, USA

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ABSTRACT

Background: Adversity during early development has been shown to have enduring negative physiological consequences. In turn, atypical physiological functioning has been associated with maladaptive processing of negative affect, including its regulation. The present study therefore explored whether exposure to adverse life events in childhood predicted maladaptive (less flexible) parasympathetic nervous system functioning during the processing of negative affect among adolescents with depression histories.

Methods: An initially clinic-referred, pediatric sample (N = 189) was assessed at two time points. At Time 1, when subjects were 10.17 years old (SD = 1.42), on average, and were depressed, parents reported on adverse life events the offspring experienced up to that point. At Time 2, when subjects were 17.18 years old (SD = 1.28), and were remitted from depression, parents again reported on adverse life events in their offspring's lives for the interim period. At time 2, subjects' parasympathetic nervous system functioning (quantified as respiratory sinus arrhythmia) also was assessed at rest, during sad mood induction, and during instructed mood repair.

Results: Extent of adverse life events experienced by T1 (but not events occurring between T1 and T2) predicted less flexible RSA functioning 7 years later during the processing of negative affect. Adolescents with more extensive early life adversities exhibited less vagal withdrawal following negative mood induction and tended to show less physiological recovery following mood repair.

Conclusions: Early adversities appear to be associated with less flexible physiological regulatory control during negative affect experience, when measured later in development. Stress-related autonomic dysfunction in vulnerable youths may contribute to the unfavorable clinical prognosis associated with juvenile-onset depression.

1. Introduction

An extensive body of research has documented that stressful events, particularly developmentally early adversities, can have lasting negative effects on neurobiological systems involved in the regulation of affect and response to stress (for reviews, see Kaffman and Meaney, 2007; Lupien et al., 2009; Heim and Binder, 2012). This work has included both non-human and human subjects and has mostly targeted neuroendocrine functioning via the hypothalamic-pituitary-adrenal (HPA) axis and its neural substrates (Chen et al., 2010; Pesonen et al., 2010; Rao et al., 2010; Bush et al., 2011; Hanson et al., 2015).

Autonomic regulation, often indexed as respiratory sinus arrhythmia (RSA; Porges, 2007), is another neurobiological system potentially vulnerable to the negative effects of early adversities. Associations between early-life adversity and RSA impairment have been documented despite considerable heterogeneity in the types of early-life adversities examined and in how impaired RSA was defined (for a review, see Propper and Holochwost, 2013). Namely, negative events as

* Corresponding author.
E-mail address: dachess@pitt.edu (S. Daches).

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diverse as parental depression (Field et al., 1995; Pickens and Field, 1995), domestic violence (Rigterink et al., 2010), marital conflict (Porter et al., 2003; Moore, 2010; El-Sheikh and Hinnant, 2011), neglect and disordered attachment (Oosterman et al., 2010), and institutional care (McLaughlin et al., 2015) have been associated with altered RSA, either in the form of lower baseline RSA (Field et al., 1995; Pickens and Field, 1995; Porter et al., 2003; Moore, 2010; Rigterink et al., 2010; El-Sheikh and Hinnant, 2011), more blunted RSA in response to an experimental stressor (Oosterman et al., 2010; McLaughlin et al., 2015), or both (Conradt et al., 2014; but see Gottman and Katz, 1989; Katz, 2007; Skowron et al., 2014 for contradictory findings).

In turn, in separate literatures, exposure to adverse life events and altered RSA each has been linked to depression risk (Hammen, 2005; Williamson et al., 2005; Stroud et al., 2008; Kemp et al., 2010; Heim and Binder, 2012; Hamilton and Alloy, 2016; Infurna et al., 2016). The association between stressful, adverse life events and depressive disorders and symptoms has been shown across the life span (e.g., Chapman et al., 2004; Hankin, 2015), both in prospective and retrospective studies (Tram and Cole, 2000; Ge et al., 2001; Pine et al., 2002; Southall and Roberts, 2002; Franko et al., 2004). Notably, the reported associations are robust regardless of how stressful events are defined (e.g., specific traumatic events, total negative event counts, weighted stressful event counts). For example, isolated adverse childhood experiences such as separation from or loss of a parent, as well as cumulative stressful adverse events, predict elevated depression symptoms and increased risk of a depressive disorder (e.g., Agid et al., 1999; Pine et al., 2002). Using total event counts, we found that clinically referred children with depressive disorders experienced about twice as many lifetime stressful events than did a school-based control group, and had accumulated those events by a younger age than did the controls (Mayer et al., 2009).

The relations of altered RSA and depression have been examined in samples that varied in age and type of depression-proneness (Rottenberg, 2007; Kemp et al., 2010; Hamilton and Alloy, 2016). For example, among adults, atypical RSA reactivity (compared to controls) is most consistently found when subjects are currently depressed (Hamilton and Alloy, 2016). Among children, atypical development of resting RSA has been reported for those at high familial risk for depression (Gentzler et al., 2012). Additionally, combinations of high resting RSA and robust RSA reactivity to negative mood induction were found to predict reduced depressive symptoms among depression-prone adults and their children (Yaroslavsky et al., 2013, 2014; Yaroslavsky et al., 2016), and indirectly contributed to lower risk of recurrent depression among adolescents (Kovacs et al., 2016).

While the contribution of early adversities to physiological dysfunction, and the role of stressful or adverse events in depression risk, have been documented in mostly separate literatures (for a review see Anacker et al., 2014), an emerging body of work has sought to link these areas. For example, specific trauma, namely sexual or physical abuse among depressed adults and youths has been associated with greater HPA activation in response to experimental stress (Kaufman et al., 1997; Heim et al., 2000). Further, among depressed women, those with severe trauma history evidenced blunted RSA reactivity to a stress-inducing task, relative to depressed women without severe trauma history (Cyranowski et al., 2011). Since these studies focused on extreme trauma, it is unclear if cumulative exposure to a variety of adverse life events has similar consequences on physiological response systems.

The present study seeks to extend the literature by integrating information on early adversities and physiological dysfunction measured years later, in the context of high depression risk. Specifically, we explored for the first time if reduced RSA flexibility among adolescents, who had histories of childhood-onset major depressive disorder, is related to developmentally early adversities. We focused on reduced physiological flexibility because context-appropriate RSA modulation is thought to reflect a functionally adaptive response to environmental demands (Porges, 1995; Porges et al., 1996). Early adversities were quantified as event counts, based on parental reports at initial (time 1 or T1) assessment. Several years later (time 2 or T2), we assessed RSA flexibility during the processing of sad affect and focused on individual differences in the context of a repeated-measures design. We hypothesized a dose-response relationship between greater exposure to childhood adversities and less flexible RSA functioning while adolescents were processing dysphoric affect.

Although the literature on the detrimental effects of adversities on later physiological functioning is most compelling for exposure during the *early phases of development*, we also examined the potential contribution of temporally closer T2 (more recent) adversities. In order to eliminate the confounding effects of current depression on physiological functioning (Salomon et al., 2013), we tested our hypotheses with adolescents who were remitted from their last episode of major depression.

2. Methods and materials

2.1. Participants

We report on adolescent probands with histories of childhood onset major depressive episodes (MDE) who were assessed at two time points, approximately 7.01 years (SD = 1.15) apart, and whose depressions were in remission at the time of the physiological assessment. The Time 1 (T1) assessments were part of a genetic investigation in a national sample of clinically referred children in Hungary, recruited through 23 child mental health clinical sites. At T1, probands were 7- to 14-years old and met DSM-IV (American Psychiatric Association [APA], 1994) criteria for a depressive disorder, as determined via a stringent series of research diagnostic interviews (for details, see Tamás et al., 2007; Kovacs et al., 2015).

The subsequent study of emotion regulation (Time 2 [T2] assessments) enrolled only a portion of the prior, original sample due to funding constraints. Because the T2 study included a laboratory-based physiological protocol, living within commuting distance of the research laboratories was the primary enrollment criterion. Subjects were aged 14–18 years at enrollment in the T2 study of emotion regulation.

The present sample includes 189 probands (96.3% Caucasian). Average age at T1 was 10.17 years (SD = 1.42); there were 122 (64.6%) boys. Age at onset of first MDD episode was 8.97 years (SD = 1.71); the average number of major depressive episodes was 1.5 (SD = 0.73). At T1, average socioeconomic status (SES), using the Hollingshead (2011) index was M = 32.93, SD = 13.18, which corresponds to middle class. At T2, subjects' average age was 17.18 years (SD = 1.28). By T2, 59.8% of the youths still had only one MDD episode, while 30.2% had 2 episodes, and 10.1% had 3 or more episodes. At T2, only 3 probands were taking psychotropic medication, consistent with the sample's remitted status. The temporal windows for the data collection in this article included January 2001 to February 2006 for T1, and April 2010 to May 2013 for T2.

2.2. Procedures

At T1, all participants received a psychosocial assessment that included the following: a) 2 separate standardized semi-structured clinical diagnostic interviews, at least one month apart, by different trained interviewers with parents about the subjects and with the subjects about themselves, which used DSM-IV diagnostic criteria (APA, 1994), b) review of the diagnoses by two trained senior child psychiatrists who then had to achieve “best estimate” diagnoses (Maziade et al., 1992), c) ascertainment of psychosocial information from parents, including offspring's history of stressful life events, medication use, and cigarette smoking history, d) completion of self-rated questionnaires, including the Paffenbarger Physical Activity Questionnaire (PPAQ; Paffenbarger et al., 1978), and e) determination of participants' weight and height.