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Research paper

The effects of childhood maltreatment and anxiety proneness on neuropsychological test performance in non-clinical older adolescents

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ABSTRACT

Background: The effect of childhood maltreatment (CM) on neuropsychological performance is well established, but the effect of anxiety proneness (AP) on such performance has been underexplored. We assessed the predictive ability of CM and AP, and their interaction, in non-clinical adolescents, for a range of previously documented neuropsychological deficits.

Methods: Multiple linear regression models were used to assess the unique and combined influences of CM and AP on neuropsychological performance in 104 non-clinical adolescents, who underwent both neuropsychiatric and neuropsychological assessment.

Results: The interaction of CM and AP was associated with poorer performance in executive functioning skills, processing speed, and estimated IQ. CM and AP were uniquely associated with verbal working memory performance, while verbal and visual memory performance and learning, and visuo-spatial ability, were not associated with either CM, AP or the interaction of CM and AP.

Limitations: The use of self-report measures to determine participants' levels of CM, AP, and depression. The CTQ-SF, a retrospective self-report measure, may have introduced recall bias. The neuropsychological evaluation was not conducted in the Xhosa language, the first language of most African participants. Most instruments utilized have not been validated in a South African adolescent sample. The impact of important moderator variables (e.g., age of onset of maltreatment) was not assessed.

Conclusions: Increased levels of CM and AP may be risk markers for poor performance in several key neuropsychological domains. Our findings underscore the importance of assessing the impact of both CM and anxiety-related temperamental traits on neuropsychological performance.

1. Introduction

The periods of childhood, adolescence and early adulthood are associated with significant developmental changes (Gunnar et al., 2009; Tamnes et al., 2010; Yurgelun-Todd, 2007) coupled with an increased risk for the development of anxiety disorders. Nationally representative household surveys and prospective community-based studies have documented the onset of any anxiety disorder, or specific anxiety subtypes, across these critical developmental periods (Asselmann and Beesdo-Baum, 2015; Kessler et al., 2005; Merikangas et al., 2009; Pine et al., 1998). The prevalence of DSM-IV anxiety disorders is high in adolescent and young adult samples, with lifetime rates approaching 30% (Asselmann and Beesdo-Baum, 2015). These disorders are frequently comorbid (Merikangas et al., 2009), persistent, chronic (Kessler et al., 2012), associated with significant impairment, adverse functioning and disability in early adulthood (Asselmann and Beesdo-Baum, 2015; Essau et al., 2014). This includes increased interpersonal problems, poor health (Copeland et al., 2014) and psychosocial (Essau et al., 2014) outcomes, and an increased risk of alcohol and substance use disorders (Essau et al., 2014).

A number of well-documented factors have been implicated in the aetiology of child and adolescent anxiety disorders (Murray et al., 2009). These include biological vulnerability factors, such as genetic variant influences (Domschke and Reif, 2012; Norrholm and Ressler, 2009), cognitive or information processing styles, such as attention, interpretation and memory biases (Bar-Haim et al., 2007; Marques et al., 2013; Watts and Weems, 2006), environmental influences, reflective of negative and stressful life events and childhood maltreatment (CM)/trauma (Benjet et al., 2010; De Bellis and Thomas, 2003; Lewis et al., 2012; McCullough et al., 2010; Young and Dietrich, 2015), and

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learning factors, such as social modelling (e.g., of parental anxious/avoidant behaviour) and information transfer (e.g., anxious parental communication with child) (Fisak and Grills-Taquechel, 2007). These vulnerability factors commonly influence or interconnect with one another (Franic et al., 2010) to produce potentially maladaptive outcomes.

High anxiety proneness (AP) is characterized by high levels of self-reported anxiety-related temperamental traits in non-clinical individuals (i.e., those who have not sought treatment for their anxiety symptoms) (Stein et al., 2007) and include traits such as anxiety sensitivity (AS) (Reiss et al., 1986) and trait anxiety (TA) (Eysenck, 1992). These are both developmentally stable risk factors for anxiety disorder (Garcia et al., 2013; Zavos et al., 2012a, 2012b), with genetic influences exerting a greater effect on stability during adolescence and early adulthood than environmental influences, which are generally more age- or time-specific (Garcia et al., 2013; Zavos et al., 2012a, 2012b). AS is defined as fear of anxiety-related or arousal-related sensations and symptoms (Reiss and McNally, 1985) stemming from a belief that such sensations or symptoms may have negative consequences, such as feelings of embarrassment, illness or added anxiety (Reiss et al., 1986). TA refers to the tendency to respond fearfully to stressors in general (McNally, 1989). AS is positively and significantly associated with both TA and neuroticism (Esteve and Camacho, 2008; Muris et al., 2001) and both AS and TA are associated with anxiety disorders and associated symptoms in youth (McLaughlin et al., 2007; Muris et al., 2001; Schmidt et al., 2006; Weems et al., 2007). High AP in youth and young adults has been found to be associated with a number of negative outcomes, including functional impairment in social and occupational domains (Korte et al., 2013) and a range of negative health behaviours, including increased alcohol and drug use and dependence (Otto et al., 2016).

Information-processing theories of anxiety suggest that anxiety is associated with selective processing of information that is perceived as threatening or dangerous to personal wellbeing or safety (Beck and Clark, 1997). Both clinical anxiety and high AP are associated with increased levels of fear- or threat-related attentional bias, either towards or away from threat (Carmona et al., 2015; Dalgleish et al., 2003; Puliafico and Kendall, 2006; Schoth et al., 2015), compared with levels in non-anxious or low-anxious individuals (Bar-Haim et al., 2007; Eysenck et al., 2007; Telzer et al., 2008). Similarly, interpretation and memory biases are also evident in clinically anxious individuals and in children, adolescents and young adults with high AP (Muris and Field, 2008; Richards et al., 2001; Teachman, 2005; Watts and Weems, 2006). These information processing biases may be considered automatic as they are voluntary, although not capacity-free as they require cognitive resources (McNally, 1995). High-anxious individuals, therefore, commonly employ more processing resources in task performance than low-anxious individuals and consequently have fewer available processing resources (Eysenck et al., 2007). Furthermore, a number of comparable outcomes on task performance have been reported in high-anxious and low-anxious individuals, however, anxiety is thought to reduce processing efficiency, with highly anxious individuals commonly reporting increased mental effort on task performance (Eysenck et al., 2007).

Few studies have examined key aspects of neuropsychological functioning in adolescents with high levels of anxiety-related temperamental traits, and therefore, the literature reporting on the impact of AP on neuropsychological performance in non-clinical adolescents is relatively limited. Results from studies in adults indicate that traits such as neuroticism and TA are associated with deficits in working memory, verbal fluency, and IQ (Moutafi et al., 2006; Qi et al., 2014; Sutin et al., 2011). There is some evidence to indicate that certain neuropsychological domains are impacted by high levels of anxiety-related traits in youth. For example, Barnard et al., (2011) found, in a sample of college students, that verbal working memory performance was significantly impacted by AS although mathematical and psychomotor performance were unaffected (Barnard et al., 2011). Visual working memory deficits have also been found to correlate with symptoms of anxiety and depression in healthy children, and TA has been found to correlate with verbal working memory deficits in children and young adults (Aronen et al., 2005; MacLeod and Donnellan, 1993; Owens et al., 2008).

CM, which is associated with both AS and TA (Martin et al., 2014), is a well-documented environmental risk factor for psychopathology across the life course (Collishaw et al., 2007; Kessler et al., 2010). The effects of anxiogenic events experienced during childhood can be mapped onto changes in vulnerable brain regions (Hanson et al., 2013; Teicher et al., 2003) and are associated with dysfunctional neuropsychological processing that may persist into adulthood (Wilson et al., 2011). Numerous studies have explored the effects of CM on neuropsychological test performance in samples of youths grouped according to exposure status (i.e., CM exposed vs. non-exposed youths) [e.g., (Irigaray et al., 2013; Kirke-Smith et al., 2014; Mothes et al., 2015)] and grouped according to exposure status and clinical disorder (i.e., CM exposed youth with clinical disorder(s), CM exposed youth without clinical disorder(s) and non-exposed, non-clinical youths) [e.g., (De Bellis et al., 2009, 2010, 2013; Kavanaugh and Holler, 2014b; Masson et al., 2015a)]. Others have compared neuropsychological functioning in youths with CM histories to youths with trauma histories other than CM (e.g., DePrince et al., 2009). Relatively few studies have assessed neuropsychological functioning in samples of non-clinical adolescents (Masson et al., 2015b) comprised of those with varying levels of CM. One such study demonstrated that non-clinical adolescents exposed to CM, quantified using Childhood Trauma Questionnaire scores, demonstrated deficits in aspects of executive functioning (Spann et al., 2012). Executive functioning can be defined as a set of control processes that regulate thoughts and actions (Miyake and Friedman, 2012) to achieve a certain goal in a flexible way (Funahashi, 2001). Executive functioning includes a number of cognitive processes, such as working memory, set shifting or task flexibility in information processing, and planning (Lezak et al., 2012). Besides CM being associated with poorer executive functioning, the effect of CM on other neuropsychological domains in youth and adults is well established. A history of CM has been shown to be associated with poorer performance in attention, language, verbal episodic memory, working memory, visuo-spatial skills, and executive functioning (De Bellis et al., 2013; Irigaray et al., 2013; Kavanaugh and Holler, 2014b; Kirke-Smith et al., 2014; Nadeau and Nolin, 2013; Nolin and Ethier, 2007). Furthermore, both intellectual impairment and academic underachievement are frequently evident in maltreated children, adolescents, and young adults (De Bellis et al., 2013; Jones et al., 2004; Kavanaugh and Holler, 2014b; Maguire et al., 2015; Mills et al., 2011; Navalta et al., 2006; Perez and Widom, 1994). Comparable findings have been reported in severely maltreated adolescents in which deficits in learning and memory, executive function, processing speed, working memory, visuo-perceptual function and language, have been demonstrated (Vasilevski and Tucker, 2016).

Given the existing evidence for neuropsychological deficits in youths with maltreatment histories and the few studies of neuropsychological performance in AP youth, the current study aimed to extend the evidence base by exploring the predictive ability of AP and CM (including the interaction of these) for a number of key neuropsychological domains (i.e., estimated IQ, visual and verbal memory and learning, executive functioning, processing speed, and visuo-spatial skills) previously reported in maltreated youth and which have been underexplored in AP, non-clinical adolescents.

2. Methods

2.1. Design

The present study was a two-tier study in a non-clinical sample of adolescents. In tier 1, a stratified two-stage cluster sampling was employed whereby schools and adolescents within schools, from four

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