Psychiatry Research: Neuroimaging 254 (2016) 92–102

Contents lists available at ScienceDirect

Psychiatry Research: Neuroimaging

journal homepage: www.elsevier.com/locate/psychresns

Cumulative trauma, adversity and grief symptoms associated with fronto-temporal regions in life-course persistent delinquent boys

Amy E. Lansing [a,c,*], Agam Virk [a], Randy Notestine [a], Wendy Y. Plante [a,c], Christine Fennema-Notestine [a,b]

[a] Department of Psychiatry, University of California, San Diego, La Jolla, CA, USA
[b] Department of Radiology, University of California, San Diego, La Jolla, CA, USA
[c] Department of Sociology, San Diego State University, San Diego, CA, USA

ARTICLE INFO

Article history:
Received 12 November 2015
Received in revised form
6 May 2016
Accepted 14 June 2016
Available online 18 June 2016

Keywords:
Structural magnetic resonance imaging
Cumulative adversity
Complex trauma
Grief
Loss
Life-course persistent delinquent youth
Fronto-temporal regions

ABSTRACT

Delinquent youth have substantial trauma exposure, with life-course persistent delinquents [LCPD] demonstrating notably elevated cross-diagnostic psychopathology and cognitive deficits. Because adolescents remain in the midst of brain and neurocognitive development, tailored interventions are key to improving functional outcomes. This structural magnetic resonance imaging study compared neuroanatomical profiles of 23 LCPD and 20 matched control adolescent boys. LCPD youth had smaller overall gray matter, and left hippocampal, volumes alongside less cortical surface area and folding within the left pars opercularis and supramarginal cortex. LCPD youth had more adversity-related exposures, and their higher Cumulative Trauma, Adversity and Grief [C-TAG] symptoms were associated with less surface area and folding in the pars opercularis and lingual gyrus. Neuroanatomical differences between LCPD and control youth overlap with data from both maltreatment and antisocial literatures. The affected left frontal regions also share connections to language- and executive-related functions, aligning well with LCPD youths' cognitive and behavioral difficulties. These data also dovetail with research suggesting the possibility of neurodevelopmental delays or disruptions related to cumulative adversity burden. Thus, concurrent treatment of LCPD youths' C-TAG symptoms and, cognitive deficits with overlapping neuroanatomical bases, may be most effective in improving outcomes and optimizing neurodevelopmental trajectories.

1. Introduction

Over 1.3 million youth are arrested annually in the United States (Puzzanchera, 2014), and each delinquent youth costs $2.5–$3.9 million (cost adjusted for 2015, Consumer Price Index, 2016) in their lifetime (Cohen, 1998). Substantial costs derive from their functional impairments in educational, occupational and social competence, which intensify with increased autonomy and responsibilities during young adulthood. Unfortunately, maltreatment increases the risk for delinquency, adult criminality, violence, cognitive deficits and poor functional outcomes (Cisler et al., 2012; Van der Kolk et al., 2001; Widom, 2014). In turn, delinquent youth demonstrate significant cognitive deficits (Lansing, et al., 2014), psychiatric impairment (Teplin et al., 2002), and cumulative childhood adversities (total accumulated adversity burden, with poorer mental health among those experiencing the most severe adversities, Lloyd and Turner, 2003; Schilling et al., 2008). "Adversities" experienced by delinquent youth include exposure to multiple traumatic events (e.g., poly-victimization; Abram et al., 2013; Baglivio et al., 2014; Duke et al., 2010), losses (Perkins-Dock, 2001), deprivation (e.g., neglect), and complex trauma; which is defined as typically severe (direct harm), interpersonal (involving betrayal often by caregivers), repetitive and/or pervasive traumas occurring during development, such as ongoing childhood maltreatment (Ford and Courtois, 2013). This is especially true for Life-Course Persistent Delinquents [LCPD], who exhibit early onset disruptive behavior and engage persistently in antisocial behavior (Moffitt, 1993; Moffitt, 2006). Notably, these youth remain in the midst of continued brain and neurocognitive development (Giedd et al., 2010; Lenroot et al., 2006; Lebel et al., 2011; Giedd et al., 2015); therefore, adversity during early life may cause or exacerbate brain abnormalities, including delayed or disrupted development of myelination and cortical maturation.

Adversity exposure and trauma disorders among delinquents are up to eight times higher than in community youth, and high

* Correspondence to: University of California, San Diego, 8950 Villa La Jolla Drive, Suite A109, La Jolla, CA 92037, USA.
E-mail address: alansing@ucsd.edu (A.E. Lansing).

http://dx.doi.org/10.1016/j.pscychresns.2016.06.007
0925-4927/

density housing with other delinquents and separation from family can result in re-traumatization and exacerbation of symptoms (Abram et al., 2004 and 2013; Arroyo, 2001; Cauffman et al., 1998; Hennessey et al., 2004; Holman and Ziedenbeg, 2006; Wolpaw and Ford, 2004). LCPD youth are at particular risk for poor lifespan outcomes (Piquero et al., 2007) and intensive mental health and education service needs, yet are unlikely to receive traditional health services (Liebenberg and Ungar, 2014), emphasizing the need for a more sophisticated nosology and treatment strategies. The Adverse Childhood Experiences [ACE] (Anda et al., 2006; Baglivio et al., 2014) studies assess ten factors, including childhood abuse (physical, sexual, emotional); physical and emotional neglect; and parental distress (incarceration, mental illness, substance use; maternal-directed domestic violence); however, there is a clear need to address a broader range of adversities common among delinquents (Child Protective Services [CPS] removal; living in violent communities; Perkins-Dock, 2001). To provide effective programs, reduce academic disengagement, improve functional outcomes and prevent delinquency escalation, we must better understand early adversity, along with the associated neurodevelopmental (neurological, psychiatric, cognitive) sequelae, among high-risk populations.

Neuroimaging research in criminally-engaged populations mainly focuses on violent and/or psychopathic adults; psychopathic youth (a theoretically controversial pathology for children; Blair, 2010); or youth seen in mental health settings (Conduct Disorder; Kruesi et al., 2004) rather than justice-involved youth. These data indicate smaller prefrontal gray matter (Raine et al., 2000) and reduced frontal functional activation (Blair, 2010), as well as smaller temporal lobe, particularly amygdala, volumes, and abnormal amygdalar activation (Kruesi et al., 2004; Blair, 2010). Studies of adult and adolescent incarcerated psychopaths suggest less gray matter and abnormal functional connectivity in limbic and paralimbic areas (hippocampus, amygdala, cingulate, prefrontal cortex; Ermer et al., 2012), with cortical thinning among adults (Ly et al., 2012), and paralimbic involvement among adolescents (Ermer et al., 2012). Meta-analysis implicates less gray matter or lower functional activation in orbitofrontal, dorsolateral frontal and anterior cingulate cortex in a range of antisocial, violent and psychopathic adults (Yang and Raine, 2009). Compared to controls, Conduct Disordered youth demonstrate cortical thinning in the superior temporal gyrus and, both childhood- and adolescence-onset conduct disordered youth demonstrate reduced surface area in the orbitofrontal cortex (Fairchild et al., 2015). While these findings provide insight supporting critical associations with temporal and frontal areas, little is known about LCPD youth, not all of whom are psychopathic (Dembo et al., 2007), or the role of dimensions of early adversity exposure in brain development among delinquents.

Given the range of adversities experienced among delinquents (e.g., ACEs, deaths of loved ones, neighborhood violence), findings from maltreatment and post-traumatic stress disorder [PTSD] studies are additionally informative. Maltreatment studies suggest smaller intracranial vaults [ICV] and cerebral brain volumes, as well as frontal and limbic abnormalities including both smaller structural estimates and abnormal functional activation patterns (De Bellis et al., 2002; Fennema-Notestine et al., 2002; Hart and Rubia, 2012; Kelly et al., 2013, 2015), supporting the potential influence of trauma on the brain in LCPD youth. Early childhood adversity is associated with cognitive, psychiatric and neurologic sequelae that persist into adulthood (Fennema-Notestine et al., 2002; Hart and Rubia, 2012; McCrory et al., 2010; Sheridan et al., 2012), and the impact of stress on the brain has been associated with hippocampus, amygdala, cingulate, and prefrontal cortex abnormalities in children and adults (Bremner, 2006; De Bellis, 2005; Hart and Rubia, 2012; McCrory et al., 2010; Robinson and Shergill, 2011; Kelly et al., 2013, 2015). Among adult males, data specifically indicate that higher cumulative adversity exposure is associated with reduced gray matter volume in prefrontal and limbic regions (Ansell, et al., 2012). Structural estimates are also typically smaller in maltreatment and stress-related cohorts (although one reported greater amygdala volume, Karl et al., 2006). The separate study of cortical thickness and surface area, both components of cortical volume, is critical, particularly within developing populations, as these aspects are driven by separate underlying genetic influences (Panizzon et al., 2009) and have varied developmental trajectories over time (Raznahan et al., 2011). Indeed, there is recent evidence for thinner cortex and smaller estimates of surface area and folding in distinct areas of the brain in maltreated youth, particularly frontal regions (e.g., anterior cingulate, lingual gyrus, and pars opercularis Kelly et al., 2013).

In addition to the neural abnormalities consistently noted in the maltreatment and delinquency literatures, cognitive deficits among these populations suggest a clinical relevance to these neural findings. First, child maltreatment, early adversity and PTSD, are associated with cognitive deficits in executive, intellectual (especially Verbal IQ), memory and academic performance (e.g., Beers and De Bellis, 2002; see review Wilson et al., 2011) and other psychiatric/neurologic sequelae that may result from delayed or disrupted neurodevelopment and persist into adulthood (e.g., Perry et al., 2008). Second, delinquents exhibit prominent verbal and intellectual deficits, alongside verbal memory, early-onset visual-spatial and mixed executive deficit findings (Lansing, et al., 2014; Moffit and Caspi, 2001; Morgan and Lilienfeld, 2000; Raine et al., 2005). This cognitive dysregulation, alongside behavioral and affective dysregulation observed among delinquents, may be linked to noted brain abnormalities, driven by the neurodevelopmental consequence of their increasingly well-documented early adversity exposure (Abram et al., 2013; Baglivio et al., 2014). In line with evidence related to the developmental impact of complex trauma (Pynoos et al., 1999; Cloitre et al., 2009) on emotion regulation and symptom complexity (Cloitre et al., 2009; Shipman et al., 2000, 2005), Moffitt (1993) proposed a developmental taxonomy for early-onset and persistent delinquency, suggesting that the interaction of cognitive deficits and adverse environments across development result in pathological behavior. Moffitt's later work (Koenen, et al., 2007), implicating children's externalizing behavior and family stress (maternal distress, loss of a parent) in the developmental roots of PTSD, further aligns with these concepts. Despite the strong evidence of the developmental impact of adversity on cognitive, neural, and psychiatric functioning and delinquency, no neuroimaging studies have specifically characterized adversity-related symptomatology among LCPD youth.

Therefore, we aimed to compare the neuroanatomical profiles of LCPD youth with matched controls, and to characterize the associations among neuroanatomical effects and adversity and resulting symptomatology in LCPD boys. Our investigation focused on brain regions implicated in both the antisocial and maltreatment/adversity literatures (e.g., frontal cortex, hippocampus, amygdala), reviewed above, and on sensitive metrics previously shown to elucidate neuroanatomical differences in childhood development (e.g., surface area, cortical folding; Kelly et al., 2013). We hypothesized that LCPD youth may have smaller hippocampal and amygdala volumes, alongside evidence of smaller frontal cortices. Considering reported structural and functional effects in frontal regions alongside the ongoing neurodevelopment in these areas within LCPD youth, we also proposed that, in addition to thinner cortex, we may see smaller surface areas and less folding indicative of stunted or delayed development in frontal cortex. Finally, we propose that the measures of regional frontal cortical surface area and folding may be strongly associated with