Child Abuse & Neglect 72 (2017) 215–226

Contents lists available at ScienceDirect

Child Abuse & Neglect

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

Research article

Reflective function mediates the relationship between emotional maltreatment and borderline pathology in adolescents: A preliminary investigation

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ARTICLE INFO

Keywords:
Childhood maltreatment
Reflective function
Mentalization
Borderline personality disorder
Adolescence
Emotional abuse

ABSTRACT

Deficits in mentalizing, particularly within the context of attachment relationships i.e., reflective function (RF), are posited to result from childhood maltreatment and to influence the development of borderline personality disorder (BPD). Whilst a mentalization-based model of BPD provides a theoretical explanation, direct empirical support for this model, in linking childhood maltreatment to borderline pathology remains limited. This study examined the interrrelationships between childhood maltreatment, RF, and borderline pathology in a mixed adolescent sample, consisting of adolescents with BPD (n = 26) and a group of non-clinical adolescents (n = 25). With the aim of directly testing the mentalization-based model of BPD, we additionally investigated the influence of each form of childhood maltreatment within this developmental pathway. Self-report data supported the hypothesized indirect effect of childhood maltreatment on elevated borderline pathology through lowered RF in adolescents. Both emotional abuse and emotional neglect were found to indirectly influence borderline pathology through adolescent RF, however, only emotional abuse indirectly influenced borderline pathology through RF, after all other maltreatment types were controlled for. Findings support the promotion of mentalization, within attachment-related contexts, as an intervention target for adolescents with borderline pathology and as a potential target of prevention for at-risk children and adolescents with histories of childhood maltreatment, especially emotional abuse. Future research should delineate other underlying mechanisms, independent of RF, which may also link the influence of childhood maltreatment, and in particular, emotional abuse, to BPD.

1. Introduction

Borderline personality disorder (BPD) is a psychiatric disorder characterized by severe interpersonal dysfunction, emotional dysregulation, identity disturbances, impulsivity and self-injurious and suicidal behaviors (American Psychiatric Association [APA], 2013). Retrospective reports of adult BPD patients suggests that the disorder typically presents during adolescence (Clarkin, Levy,

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http://dx.doi.org/10.1016/j.chiabu.2017.08.008
Received 3 March 2017; Received in revised form 10 June 2017; Accepted 6 August 2017

Lenzenweger, & Kernberg, 2004; Zanarini et al., 2006). Diagnosing BPD prior to adulthood, however, has been controversial (Griffiths, 2011; Laurenssen et al., 2013); in large, concerns have been raised about the stability of personality prior to adulthood, and whether adolescent BPD features may instead be transient variations of normal developmental processes, as opposed to expressions of psychopathology (Miller, Muehlenkamp, & Jacobson, 2008). Other concerns include the potential stigmatization and iatrogenic harm associated with labelling adolescent patients with a personality disorder, particularly if simply viewed as a disorder of 'character' (Chanen & McCutcheon, 2008). Contrary to and in spite of some of these concerns, substantial evidence has emerged suggesting that the validity and clinical significance of BPD in adolescents is similar and comparable to BPD in adults (Miller et al., 2008). From transgenerational models that elucidate early etiological risk factors (Newman et al., 2016), BPD is also becoming increasingly seen as a developmental condition, irrespective of age of presentation (Tackett, Balsis, Oltmanns, & Krueger, 2009). As a result, adolescent BPD has gained recent recognition as a valid diagnostic entity, warranting further empirical attention during this developmental stage (Kaess, Brunner, & Chanen, 2014).

BPD in adolescence is estimated to affect 3% of the general population (Bernstein et al., 1993), 11% of outpatients (Chanen et al., 2004), and up to 50% of inpatients (Becker, Grilo, Edell, & McGlashan, 2002). Adolescents affected by BPD have been found to experience greater levels of functional impairment than adolescents with other personality or psychiatric disorders (Chanen, Jovev, & Jackson, 2007). Adolescents with BPD also experience higher rates of psychiatric comorbidity (Ha, Balderas, Zanarini, Oldham, & Sharp, 2014) and poor longer-term interpersonal and occupational outcomes, even when psychiatric comorbidity is accounted for (Winograd, Cohen, & Chen, 2008). While substantial research has been conducted concerning the validity and clinical importance of adolescent BPD, less is still known about its etiology. Research studies examining etiological factors that cause, maintain or exacerbate adolescent BPD are crucial for identifying treatment targets and fostering developmentally sensitive interventions. Although developmental theorists of BPD acknowledge that pathways to the disorder are likely complex, and involve interactions between genetic and environmental factors (Crowell, Beauchaine, & Linehan, 2009; Fonagy & Luyten, 2009; Gunderson & Lyons-Ruth, 2008), there is general consensus that early childhood experiences, particularly attachment relationships and disruptions of care, are often central to the development of BPD.

Childhood maltreatment, including physical, sexual, emotional abuse and neglect, has been well established in the literature as an important risk factor for BPD in adults. Retrospective and prospective investigations have consistently demonstrated significant associations between childhood maltreatment and adult BPD, even after accounting for other personality disorders (Johnson, Cohen, Brown, Smailes, & Bernstein, 1999; Zanarini et al., 1997). Research with adolescents have yielded comparable findings; a recent meta-analysis suggested that in adolescents, experiences of either childhood neglect, sexual abuse or verbal abuse were all associated with an almost five-fold increased risk of having a BPD diagnosis (Winsper et al., 2016). The same study also reported that physical abuse was associated with a nearly three-fold increased risk of suffering from BPD. Despite robust associations between childhood maltreatment and BPD in both adults and adolescents, the relative contributions of specific forms of childhood maltreatment to the development of BPD remains unclear; one significant reason has been due to the fact that different forms of childhood maltreatment often co-occur with each other (Dong, Anda, Dube, Giles, & Felitti, 2003). More importantly, there remains a limited understanding and a lack of empirical evidence about the specific mechanisms by which childhood maltreatment may affect the development of BPD.

Mentalization, referring to the ability to understand and interpret the meaning of one's own and others' behaviors by considering underlying mental states and intentions, has been proposed as a mechanism linking childhood maltreatment to BPD (Fonagy & Luyten, 2009); in spite of this, its role as a mediator between childhood maltreatment and BPD development has not been adequately tested. Closely related to and often interchangeably used with the term 'theory of mind' (ToM; Baron-Cohen, Leslie, & Frith, 1985), mentalization is also referred to, particularly within the context of attachment relationships, as 'reflective function' (RF; Fonagy, Gergely, Jurist, & Target, 2002; Sharp & Fonagy, 2008). It has been proposed that the capacity to mentalize is vulnerable and likely undermined in those with BPD, especially under states of attachment-related stress or arousal (Fonagy & Luyten, 2009). In support of this, Fonagy et al. (1996) provided evidence that mentalization, operationalized as RF, and which was rated according to mental state references based on transcripts from the Adult Attachment Interview (George, Kaplan, & Main, 1985), was significantly lower in adult BPD patients compared to psychiatric controls. Ha, Sharp, Ensink, Fonagy, and Cirino (2013), in using a self-report measure of RF, also recently found that lower levels of RF were significantly associated with BPD features in a sample of adolescent inpatients. Mentalizing abilities, as assessed by experimental measures of social cognition, have also been found to be significantly impaired in both adults and adolescents with BPD (see Sharp and Vanwoerden, 2015 for a review). According to Fonagy and Luyten (2009), deficits in mentalization, particularly in RF, underlie the significant interpersonal difficulties characteristically experienced by individuals with BPD. Interpersonal dysfunction has been suggested to be the most specific discriminator for BPD diagnosis (Gunderson, 2007), and for adults with BPD, are frequent precipitants for other core difficulties, such as affect instability, self-harm and suicidal behaviors (Brodsky, Groves, Oquendo, Mann, & Stanley, 2006; Sadikaj, Russell, Moskowitz, & Paris, 2010; Welch & Linehan, 2002).

The mentalization-based model of BPD theorizes that the capacity to mentalize develops within the context of early attachment relationships from infancy (Fonagy & Luyten, 2009). In forming early and secure attachment relationships with caregivers, it is believed that children are permitted the safety and freedom to explore and gradually understand what may be in their own minds, and minds of their caregivers, which in turn, fosters and promotes mentalizing capacity (Fonagy et al., 2002). Mentalizing development, as a result, is posited to be vulnerable to experiences that disrupt child-caregiver attachment (Fonagy & Luyten, 2009); as such, impaired mentalizing abilities may be affiliated with caregiving or environmental disruptions, exemplified by severe abuse, neglect or other forms of maltreatment. Studies have indeed provided evidence in support of this relationship, having found that children with a history of maltreatment exhibit significantly poorer ToM or mentalizing abilities compared to non-maltreated