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Childhood trauma, antisocial personality typologies and recent violent acts among inpatient males with severe mental illness: Exploring an explanatory pathway

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ABSTRACT

Background: Prevalence of childhood trauma is elevated among individuals with severe mental illness (SMI) compared to the general population and associated with poor prognosis, substance misuse, lower treatment compliance and violence. Antisocial personality disorder (ASPD) typologies (childhood vs adult onset) also represent possible mediating mechanisms to explain risk of violence among men with SMI. The current study aimed to explore an explanatory pathway linking childhood traumatic exposure, antisocial personality typologies and risk of violent behaviour among adult male inpatients with SMI.
Methods: A total of 162 male inpatients with SMI were examined using a cross-sectional survey design. Information was extracted from medical files, interviews and official criminal records.
Results: Fifty-two participants (32.1%) reported experiencing a childhood trauma before 15. This group was 2.8 times more likely to engage in violent acts within the past 6 months than those without such a history. Furthermore, those with childhood onset ASPD (early starters) were more likely to report childhood trauma and engage in violence compared to adult onset ASPD (late starters) and those without antisocial histories. Multivariate analyses revealed that early starter ASPD was the only variable that independently predicted violence and mediated the relationship between childhood trauma and recent violent acts.
Conclusions: A significant subset of men reporting trauma and antisocial conduct from childhood (early starter ASPD) is at considerably elevated risk of engaging in violent behaviours. Assessment of antisocial typologies in men with SMI may assist effective and defensible case prioritisation, resource allocation and treatment planning.

1. Introduction

Individuals with severe mental illness (SMI) report elevated rates of traumatic experiences compared to the general population (Spidel et al., 2010) with estimates of lifetime interpersonal trauma exposure ranging between 16% and 98% (Morgan and Fisher, 2007; Husted et al., 2010; Bebbington et al., 2011; Subica et al., 2012). Trauma has been associated with more complex presentations and poorer prognoses among persons with SMI (Alvarez et al., 2011). Trauma can be defined as events that threaten one's physical and psychological integrity (Briere and Scott, 2006) and can refer to a number of adverse events, which include (but are not limited to) physical and sexual abuse. More specifically, a history of childhood physical and/or sexual trauma among individuals with SMI has been associated with earlier illness onset, severity of symptoms (Üçok and Bikmaz, 2007; Maniglio, 2009), poorer social functioning (Cusack et al., 2004), lower treatment compliance (Lecomte et al., 2008), substance abuse (Maniglio, 2009), higher rates of admissions (Alvarez et al., 2011), and elevated risk of suicide (Conus et al., 2010). These associated complications and adversities highlight the importance of consideration of childhood trauma in the treatment and management of adults with SMI.

A history of trauma among individuals with SMI has further been associated with increased rates of violent behaviour. The developmental pathway from victim to victimiser has been thoroughly documented within prior research (Widom, 1989; Nikulina et al., 2011). Swanson et al. (2002) found that individuals with SMI who reported experiencing sexual or physical trauma in childhood were three times more likely to engage in recent violent behaviour than controls. Brekke and colleagues (2001) also found similar findings within an outpatient sample. However, a large-scale epidemiological twin study by Forsman and Långström (2012) found a weak causal link between childhood maltreatment and adult violent offending, attributing familial and environmental factors as more predictive.

Accordingly, it is important to explore possible intermediary factors that might explain the pathway from childhood trauma to violent

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behaviour in adulthood among men with SMI. One such factor could be antisocial personality disorder (ASPD), which is a pervasive condition characterised by life course persistent antisocial behaviour with both genetic and environmental determinants. Antisocial personality disorder (which has onset before 15) complicates the course of SMI (Bruce et al., 2012; Gwaspari et al., 2011) and increases the risk of violence (Bruce et al., 2014; Ndegwa, 2003). In a sample of male psychiatric military outpatients, those reporting a physical and/or sexual childhood trauma were over three times more likely to meet criteria for ASPD than those without such history (Semiz et al., 2007). Hodgins et al. (2008) espoused a developmental framework for understanding violent and antisocial behaviours among men with SMI. She proposed three distinct typologies distinguished by the age of onset: (i) early starters (whose antisocial behavioural that emerges in childhood, well before illness onset, and remains stable across the lifespan, defined herein as early starter ASPD); (ii) late starters (who display no antisocial behaviour before illness onset and then repeatedly engage in antisocial behaviour, defined herein as late starter ASPD) and; (iii) serious offenders (these individuals display no antisocial behaviour for many years and then engage in very serious violence later in life).

Extant literature suggests that individuals displaying early starter ASPD can be distinguished by deficits in the orbital prefrontal cortex which has been linked to antisocial behaviour, impaired inhibition, attention and lower order executive functioning (Damasio, 2000). Furthermore, the orbital prefrontal cortex is particularly vulnerable in the context of early childhood trauma, which has been argued to negatively impact the maturation of this brain region that is responsible for self-regulatory functioning, social engagement and attachment systems (Schore, 1996, 2002) and linked to the development of personality disorder (Goodman et al., 2004). This evidence may suggest that childhood trauma may place individuals at risk of life course persistent antisocial behaviour. Moreover, such neurobiological markers do not appear present in late starter ASPD men with SMI implying distinct aetiological pathways (Hodgins, 2008). The relationship between childhood trauma and risk of violence as a function of antisocial typologies remains unexplored among men with SMI.

Notably, literature regarding neurological and environmental pathways to violence among female inpatients with SMI remains scarce. Available literature of violent female offenders implicates similar risk factors to males with SMI such as early adverse childhood experiences, substance misuse and a diagnosis of a personality disorder (Weizmann-Henelius et al., 2004). However, de Vogel et al. (2012) argue that there are substantial gender differences in the way these risk factors develop. Females who commit violent acts have been found to report more disturbed backgrounds (Abram et al., 2003) and less antisocial behaviour in early childhood (Moffitt and Caspi, 2001) than males who engage in aggressive behaviours in adulthood.

The aim of the current study was to examine this conceptual pathway which links childhood traumatic exposure, development of antisocial behaviour (early vs late starter APSD) and subsequent risk of violent behaviour among adult inpatients with SMI around the time of hospitalisation. Due to the gender specific nature of this conceptual framework for understanding antisocial personality typologies in the pathway to violence and the low base rates of violence perpetrated by female inpatients with SMI the current study restricted its sample to male participants. We hypothesised that among men with SMI, early starter ASPD participants would be more likely to (i) report a history of childhood trauma and (ii) be at increased risk of perpetrating violence around the period of hospitalisation, compared to late starter ASPD participants and those without a history of antisocial behaviour. We further hypothesised that early starter, but not late starter, ASPD would explain the relationship between childhood trauma and acts of recent violence.

2. Methods

2.1. Participants

A total of 192 male inpatients were recruited from 10 secure psychiatric wards across four hospital sites within an inner-city mental health trust between September 2008 and July 2011. Inpatients were invited to take part in the study if they met the following inclusion criteria: male, aged between 18 and 65, a principal diagnosis of severe mental illness from the ICD-10 if codes F20–F29 (schizophreniform disorders) or F30–F39 (major mood disorders), had capacity to consent to participation and had a reasonable command of the English language. Individuals were excluded from taking part if they had no legal status in the UK, were detained for less than 48 h, presented with acute symptoms, and were assessed as having an IQ<70. Due to incomplete data for 18 participants and diagnostic exclusion of 12 further participants (see below) the final sample size was 162. Participants fell into the following major diagnostic categories; 58.6% (n = 95) were diagnosed with schizophrenia, 8.6% (n = 14) had a diagnosis of schizoaffective disorder, 19.8% (n = 32) were bipolar, and the remaining 13.0% (n = 21) received diagnoses of other severe mental disorders.

In order to assess the representativeness of the current sample a case register search was conducted to identify all individuals eligible for inclusion over the period of recruitment yielding a target pool size of n = 1, 104. Statistical analyses comparing basic demographic information confirmed no significant differences existed between recruited and non-recruited individuals. The current sample (representing 15% of the target population) was subsequently deemed representative.

2.2. Procedure and materials

The current investigation adopted a similar procedure successfully used by a previous study which examined the relationship between violence and severe mental illness in a psychiatric sample (Hodgins et al., 2007). Key clinical personnel of identified wards routinely advised researchers of potential inpatients who might meet eligibility criteria for the study. Subsequently, these inpatients were approached and their capacity to consent was assessed by the researcher and the nurse-in-charge in accordance with current legislation – Mental Capacity Act, (Department of Health, 2005). All participants provided full consent to engage in the research interview and access information from his medical files and criminal records from the Ministry of Justice. Inpatients were reimbursed for their time with a payment of £15.

2.2.1. Socio-demographic and clinical factors

An interview schedule was created to obtain information related to socio-demographic and clinical factors which have been demonstrated to be related to the both predictor and outcome variables (Bhui et al., 2003; Semiz et al., 2007; Joyal et al., 2011). See Table 1 in the Results section for information collected.

2.2.2. Structured clinical interview for DSM-IV (SCID-II; First et al., 1995)

This semi-structured interview was used to assess the DSM-IV Axis II personality disorders. The SCID-II was used to assess ASPD in the current sample of male psychiatric inpatients. The SCID-II has been recommended for use in both research and clinical settings (First et al., 1995). Previous research has demonstrated good inter-rater reliability of .98 (Maffei et al., 1997) for ASPD. For the purpose of the study, ASPD was coded as either (i) 'early starter ASPD' (i.e., men who met criteria for conduct disorder before age 12 and fulfilled antisocial criteria in adulthood), (ii) 'late starter ASPD' (i.e., men who did not meet criteria for conduct disorder before age 12 but fulfilled antisocial criteria after illness onset); and (iii) no ASPD (i.e., men who met neither criteria for conduct disorder or antisocial traits in adulthood). Individuals with adolescent (before 15) onset conduct disorder were excluded from the