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Facets of Psychopathy in Relation to Trauma-Exposure and Posttraumatic Stress Symptomology in a Sample of Incarcerated Male Offenders

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Abstract

Purpose: The aim was to investigate the moderating role of psychopathy facets on the relationship between traumatic exposure and posttraumatic stress disorder (PTSD) symptomology. Design/methodology/approach: Participants were male prisoners incarcerated in the U.K. Findings: The analysis revealed differential associations between the two facets of psychopathy, with potentially traumatic events and symptoms of PTSD. Specifically, neither primary psychopathy nor trauma exposure were significantly related to PTSD, while secondary psychopathy was positively and significantly related with PTSD symptoms. Furthermore, the effect of trauma exposure on PTSD was found to depend on the level of secondary psychopathy. More specifically, trauma exposure was strongly and positively associated with PTSD symptomology for individuals with high levels of secondary psychopathy. Originality/value: The findings clarify linkages among psychopathy facets, trauma, and PTSD, and extend our understanding of the presentation of PTSD in male prisoners.

Keywords: Psychopathy; Post Traumatic Stress Disorder; Trauma; Moderation; Prisoners.

Introduction

Posttraumatic stress disorder (PTSD) is a heterogeneous syndrome characterised by relatively disparate symptom clusters. In the recently published 5th edition of the Diagnostic and Statistical Manual of Mental Disorder (DSM-5; American Psychiatric Association, 2013), PTSD is comprised of four symptom clusters – intrusion symptoms, avoidance, negative alterations in cognitions and mood, and alterations in arousal and reactivity. The diagnosis of PTSD requires that four clusters of symptoms are present for at least one month, causing significant distress or impairment (APA, 2013). However, a significant proportion of trauma-exposed individuals do not develop full but sub-threshold PTSD (McLaughlin, Koenen, & Friedman et al., 2015). Such sub-threshold posttraumatic stress is clinically relevant, as it associated with significant and long-standing impairment (Marshall, Olfson, Hellman, et al., 2001). Although few studies have examined the prevalence of PTSD among offender samples, a recent systematic review found it to be more prevalent among prisoners than in the general population with a rate of between 4 and 21% (Goff, Rose, Rose, & Purves, 2007).

Various factors have been postulated to moderate and mediate the relationship between potentially traumatic events (PTEs) and the emergence of PTSD symptomology. Among these are personality characteristics such as attributional style (Yule, 2000), locus of control (Frye & Stockton, 1982; Solomon, Mikulincer & Benbenishty, 1989) and sensation seeking (Neria, Solomon, Ginzburg & Dekel, 2000; Solomon, Ginzburg, Neria & Ohry, 1995). A trait that may be of particular relevance in incarcerated offenders is psychopathy. Psychopathy is characterised by a callous, shallow and manipulative affective-interpersonal style (primary psychopathy) combined with antisocial and reckless behaviour (secondary psychopathy) (Hare, 1991).

Both PTSD and psychopathy are related with greater offense rates, violence, and recidivism (Goff et al., 2007; Hare & Neumann, 2008). The association between these constructs, however, is complex and not yet fully understood (Sellborn, 2015). Some researchers have argued that the two should not co-occur due to the fact that psychopathy is associated with reduced threat-sensitivity and poor fear conditioning (neutral stimuli present during the traumatic event acquire fear-eliciting properties through association with the traumatic event); whereas, PTSD is associated with heightened threat-sensitivity (Cleckley, 1941, 1976; Blair & Mitchell, 2009; Willemsen, De Ganck, & Verhaeghe, 2012). Indeed, Blair and Mitchell (2009) concluded that psychopathy is in some respects the functional inverse of PTSD. Others researchers, however, contend that these disorders tend to both be associated with substantial histories of trauma exposure and should consequently co-occur (Frick, Lilienfeld, & Ellis et al., 1999; Lilienfeld & Penna, 2001). Research has suggested that pre-existing traits, such as behavioural inhibition, leads to numerous stressful events (e.g. incarceration), which results in high levels of negative affect (e.g., Colder & O'Connor, 2004; Gudiño, Nadeem, Kataoka, & Lau, 2012; Muris, Meesters, de Kanter, & Timmerman, 2005). As such, individuals who score high on psychopathy measures are also likely to obtain high scores on measures of anxiety (Lilienfeld, 1994) and suicide risk (Dhingra, Boduszek, Palmer, & Shevlin, 2014; Verona, Edens, Howard, & Smith, 2001). According to this theory, heightened anxiety is not the result of temperament or personality, but instead is a reaction or result of their risk-taking and antisocial behaviour (i.e. secondary psychopathy). Consistent with their proposed greater exposure to negative life events, Tatar, Cauffman, Kimonis, and Skeem (2012) found that incarcerated boys classified in the secondary psychopathy group manifested greater past symptoms of PTSD than their primary psychopathy counterparts. Somewhat differently, Sellbom (2015) found that fearless-dominance traits have a protective

effect against the presence of PTSD symptoms in students scoring highly on the impulsiveantisocial psychopathy facet.

Some research has found that primary psychopathy is associated with lower anxiety (Blonigen, Sullivan, Hicks, & Patrick, 2012; Harpur, Hare, & Hakstian, 1989; Hicks & Patrick, 2006) and greater psychological well-being (Verona et al, 2001). Consequently, primary psychopathic traits might protect individuals from the impact of traumatic exposure as they may mean that individuals are less likely to develop conditioned fear, to avoid unpleasant stimuli, and to focus on threat stimuli, the impact of traumatic exposure may be less significant for them. In line with this proposition, research has shown that primary psychopathy traits are negatively associated with PTSD (Pham, 2012; Willemsen et al., 2012), and that such traits are protective against development of this disorder when an individual is exposed to trauma (Willemsen et al., 2012).

The current study

The aim of the present study is to elucidate further the associations between trauma exposure, psychopathy, and PTSD. An important issue that we address in examining the anticipated differential associations is the need to control for the association between the two dimensions of psychopathy (see Sellbom, 2015). Controlling for the effects of each dimension we believe will provide a clearer picture of the role of each psychopathy dimension in the relationship between trauma exposure and PTSD symptomology (see also Frick et al., 1999; Patrick, 1994). To our knowledge, a model incorporating the two facets of psychopathy in this way has never been tested before in a sample of prisoners. Based on prior research (Frick et al., 1999; Lilienfeld & Penna, 2001), it was expected that psychopathy would moderate the trauma–PTSD relationship. Moderation analysis tests whether the prediction of a dependent variable, Y (PTSD symptomology in this case), from an independent variable, X (trauma exposure in this case), differs across levels of a third variable, Z (psychopathy levels in this

case). Moderating variables affect the strength and/or direction of the relation between a predictor and an outcome: reducing, enhancing, or changing the influence of the predictor. Specifically, we expect that trauma exposure will be positively associated with PTSD symptoms for high levels of secondary psychopathy. This relation will be due to the fact that individuals scoring higher on this facet of psychopathy may have encountered a greater number of potentially traumatic events (e.g., physical assault, transportation accidents etc.) that lead to heightened PTSD symptomology. It is further expected that trauma exposure will be negatively associated with PTSD symptomology for individuals scoring highly on primary psychopathy. This is because such traits may confer a protective effect against the development of this disorder when an individual is exposed to trauma (Willemsen et al., 2012).

Method

Participant and sampling procedure

An opportunistic sampling procedure was used. Ethical Approval for the study was granted by the National Offender Management Service (NOMS). Two hundred and eighty (N = 280) male prisoners incarcerated in a medium security UK prison were invited to participate in the study (via survey distribution). The only exclusion criteria applied was an inability to read English. All inmates were informed verbally that they should not participate in the study if they had poor literacy skills, but that they did not have to inform data collectors of the specific reason for not participating in the research. A brief description of the study was provided to each participant along with the questionnaire. Individuals were assured about the confidentiality of their participation, and informed that they could withdraw from the study at any time without having to provide a reason for doing so. Participants completed the questionnaires in their living units. Participation was voluntary without any form of reward. After completing the questionnaire, prisoners were asked to return it to the prison officer in a sealed envelope, which was provided alongside the questionnaire.

One hundred and nine (N = 109) surveys were returned to data collectors but only 101 were included in final analysis (eight prisoners were excluded due to significant missing data). The participant age range was between 18 and 61 years (M = 33.01, SD = 8.50). Most participants were heterosexual (93.9%), White (68.7%), and single (56.6%). Most participants were also housed in a single cell (96.0%), had spent time in three or more prisons (38.0%), and were employed by the prison (54.5%). The frequency of prior imprisonment ranged from zero to 25 (M = 4.56, SD = 5.88). The sample consisted prisoners convicted of property offenses (n = 19), drug offenses (n = 15), robbery (n = 14), physical assault (n = 6), offenses with weapons or firearms (n = 5), fraud (n = 4), murder (n = 2), domestic violence (n = 1), and mixed offenses (n = 34).

Procedure

Measures

The Posttraumatic Stress Disorder-Checklist version 5 (PCL-5; Weathers, Litz, Herman, Huska, & Keane, 1993) is a commonly used self-report measure of PTSD symptomology (Elhai, Gray, Kashdan, & Franklin, 2005). Weathers, Litz, and Herman et al. (2013) adapted the PCL so that items map directly onto DSM-5 symptom criteria for PTSD. Respondents indicated how distressed they were by each symptom over the past month by rating items on a five-point Likert-type scale (1 = "not at all" to 5 = "extremely"). Respondents were instructed to anchor their ratings to their worst nominated traumatic event.

Traumatic exposure. The life events checklist (LEC-5) (Blake, Weathers, & Nagy et al., 1995) is a self-report report measure designed to screen for exposure to Criterion A events. It consists of 16 potentially traumatic events, each rated on a 6-point nominal scale (1="happened to me"; 2="witnessed it"; 3="learned about it"; 4 = "part of my job", 5="not sure"; and 6="does not apply") and one catch-all category labelled "any other very stressful event or experience." A frequency score, based on personal experience, for traumatic exposure was calculated by totalling all potentially traumatic events reported. In our sample, 93.1% of the participants had experienced at least one potentially traumatic event in accordance with the A1 criterion, and the mean frequency score was 5.29 (*SD* = 3.32, range = 0-16). The most commonly endorsed directly experienced traumas included physical assault (n = 77; 76.2%), assault with a weapon (n = 67; 66.3%), transportation accident (n = 56; 55.4%), and serious injury, harm, or death caused to someone else (n = 42; 41.6%).

SRP-SF. Self-Report Psychopathy Scale-Short Form (Paulhus, Neumann, & Hare, in press) was used to assess self-reported psychopathic traits. Based on the "gold standard" of clinical psychopathy assessment, the Psychopathy Checklist–Revised (PCL-R; Hare, 1991), the SRP-SF is a 29-item measure that yields a total score as well as four subscale scores: Interpersonal Manipulation, 7 items, (e.g. "I have pretended to be someone else in order to get something"); Callous Affect, 7 items, (e.g. "I like to see fist-fights); Erratic Lifestyle, 7 items, (e.g. "I'm a rebellious person"); and Antisocial Behaviour, 8 items, (e.g. "I have never been involved in delinquent gang activity"). Items are scored on a 5-point Likert scale (1 = *strongly disagree* to 5 = *strongly agree*).

Analysis

Descriptive statistics, Pearson product-moment correlation coefficients, and regression analysis were calculated using SPSS 22. A hierarchical moderated multiple regression analysis, as the recommended method for testing interaction effects (Cohen & Cohen, 1983), was applied in order to investigate the moderating role of psychopathy factors in the relationship between trauma and PTSD (only standardised solution was reported). Simple slopes for the relationship between trauma and PTSD were investigated for low (1 SD below the mean), medium (mean), and high (1 SD above the mean) levels of primary and secondary psychopathy factors (see Bate, Boduszek, Dhingra, & Bale, 2014; Boduszek, Adamson, Shevlin, & Hyland, 2012; Cohen & Cohen, 1983) using ModGraph 3.0 (Jose, 2013).

Results

Descriptive Statistics and correlations

Descriptive statistics including means (*M*) and standard deviations (*SD*) for trauma, PTSD, and two psychopathy factors are presented in Table 1 together with bivariate correlations. Results indicate significant positive associations between the number of traumatic events experienced, PTSD scores, and scores on both facets of the psychopathy scale.

Table 1: Descriptive statistics and correlations for trauma exposure, PTSD symptomology,and primary and secondary psychopathy

Variable	Trauma	PTSD	P1	P2
Trauma exposure	1			
PTSD	.22*	1		
Primary psychopathy (P1)	.32**	.23*	1	
Secondary psychopathy (P2)	.40***	.32**	.83***	1
М	5.29	32.49	32.18	41.36
SD	3.32	21.52	11.14	12.49
Min	0	0	14	19
Max	16	.76	64	66
Cronbach's alpha	6	. 90	.88	.86

Note: * *p* < .05; ** *p* < .01; *** *p* < .001

Moderated Regression Analysis

Hierarchical moderated regression analysis was performed to investigate the moderating effect (enhancing, reducing, or changing the influence) of the two psychopathy factors on the relationship between trauma exposure and PTSD symptoms. Preliminary analyses revealed no violation of the assumptions of normality, linearity, multicollinearity, and homoscedasticity.

In the first step of the analysis, trauma exposure and the two psychopathy factors were entered. This model (model 1) was statistically significant ($F_{(3, 92)} = 4.03$, p = .01) and explained 12% ($R^2 = .12$) of variance in PTSD symptoms. Only secondary psychopathy was statistically correlated with the PTSD (see Table 2). The second step of the analysis consisted of entering interaction terms, coding the interaction between trauma exposure and the two psychopathy factors. After entering the interaction terms, an additional 6% of variance in PTSD symptoms was explained (R^2 Change = .06, p = .048), and the final model (model 2) as a whole explained 18% of variance in PTSD symptoms ($R^2 = .18$; $F_{(5,90)} = 3.79$, p = .004). Just as in model 1, in model 2, secondary psychopathy formed a statistically significant direct relationship with PTSD symptoms. There was no significant direct relationship between primary psychopathy and PTSD symptoms or between trauma exposure and PTSD symptoms. However, the relationship between the interaction term (trauma by secondary psychopathy) and PTSD symptoms was statistically significant, indicating that the effect of trauma exposure on PTSD symptoms depends on the level of secondary psychopathy. In other words, levels of secondary psychopathy affect the strength and/or direction of the relationship between trauma exposure and PTSD symptomology.

Table 2

Moderated effect of psychopathy traits on relationship between trauma exposure and PTSD symptoms

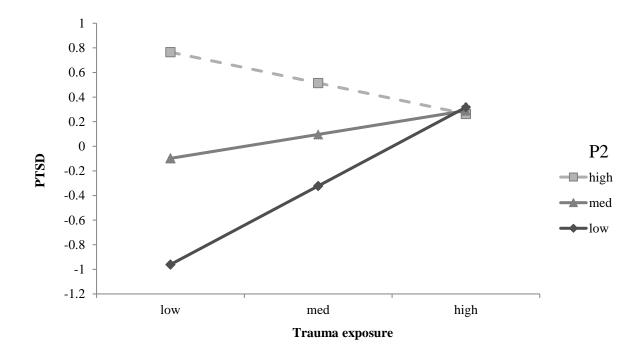
Model	Variable	β	SE	95% CI
1	Primary psychopathy (P1)	11	.17	47/.25
	Secondary psychopathy (P2)	.37*	.18	.01/.74
	Trauma exposure	.10	.10	11/.32
2	Primary psychopathy (P1)	18	.18	54/.19
	Secondary psychopathy (P2)	.42*	.18	.05/.79
	Trauma exposure	.19	.11	03/.41
	Trauma by P1	.29	.20	15/.65
	Trauma by P2	50*	.21	88/01

Note: * *p* < .05.

To aid the interpretation of the moderation, and show how the slope of Y (PTSD symptomology) on X (trauma exposure) is dependent on the moderator (psychopathy levels), interaction effects were plotted (Figure 1). Regression slopes that correspond to the prediction of Y from X at a single value of Z, which are termed simple slopes, were investigated for low (1 SD below the mean), medium (mean), and high (1 SD above the mean) levels of secondary psychopathy (see Figure 1). Trauma exposure was strongly and positively associated with PTSD symptoms for low levels (-1 SD) of secondary psychopathy ($\beta = .64$, SE = .20, p = .01). A significant negative association between trauma exposure and PTSD symptoms was found for high levels (+1 SD) of secondary psychopathy ($\beta = .25$, SE =

.22, p = .04). The association between variables for medium (mean) levels of secondary psychopathy was non-significant ($\beta = .19$, SE = .15, p = .12).

Figure 1: The moderating role of secondary psychopathy (P2) on the relationship between trauma exposure and PTSD symptomology



Discussion

The aim of the present study was to elucidate further the associations between exposure to potentially traumatic events, psychopathy factors, and PTSD symptomology in a sample of male prisoners. The findings support our hypothesis that the two psychopathy facets (primary and secondary) would have differential associations with PTSD symptomology, although the direct relationship between primary psychopathy and PTSD symptomology was not statistically significant in either step of the analysis. The non-significant association between primary psychopathy and PTSD symptoms further supports the growing literature that suggests that primary psychopathic traits might protect individuals from the impact of traumatic exposure (Sellbom, 2015; Willemsen et al., 2012). Our results also indicated somewhat unexpectedly that trauma exposure was strongly and positively associated with PTSD symptoms for low levels of secondary psychopathy and negatively associated with PTSD symptomology for individuals with high levels of secondary psychopathy. This suggests that individuals lower on these traits are more vulnerable to the development of PTSD in response to trauma exposure whereas, for those higher on these traits, the impact of traumatic exposure may be less significant. Thus, for male prisoners with higher levels of secondary psychopathy, more exposure that is traumatic might mean diminished levels of PTSD over time, rather than accumulating symptomology.

Although our findings with respect to secondary psychopathy was not as predicted, it is consistent with research suggesting that individuals higher in sensation-seeking are better able to adjust to stressful situations and report lower levels of PTSD, combat-related stress, and other psychiatric symptoms following exposure to stressful events (e.g., Solomon et al., 1995). Alternatively, our results might be explained as the result of habituation to potential traumatic events. Over time, the brain may attempt to compensate for elevated limbichypothalamic-pituitary-adrenal (LHPA) axis activity in response to traumatic stress by a negative feedback loop which reduces response to stimulation of the LHPA axis, resulting in a desensitization to stress (De Bellis, Chrouso, & Dorn et al., 1994; Glaser, 2000). The results of this desensitization may include callousness, unemotionality, non-responsiveness to punishment, and a heightened need for external stimulation (i.e., traits that mimic secondary psychopathy).

Our results are inconsistent with those of Sellbom (2015) who found that fearlessdominance traits had a protective effect against the presence of PTSD symptoms in students scoring highly on the impulsive-antisociality psychopathy facet. However, as noted by Sellbom, PPI Fearless-Dominance (used in his study) and SRP-SF (used in the present study) measurements of such traits are not isomorphic (see also Dhingra & Boduszek, 2013), with the former typically being more strongly negatively associated with negative affectivity/internalizing disorders (including PTSD) at the zero-order level. Thus, as suggested by Sellbom, "associations between psychopathy and PTSD might to some degree depend on from what perspective the former construct is operationalized" (2015, p. 90). It is also possible that the discrepancy between our findings and Sellbom's may be the result of different samples (prisoners vs. students, respectively).

The results should be interpreted in the light of potential limitations. The primary limitation centres on the cross-sectional examination of the associations between psychopathy facets, trauma exposure, and PTSD symptomology and the resultant inability to address the causal mechanism behind the relationships. Consequently, it may be the case that secondary psychopathic traits lead to trauma exposure due to an insensitivity to punishment when primed with reward, a tendency to seek out novel and dangerous activities (Frick, Cornell, & Bodin et al., 2003), and a lack of responsiveness to others' distress cues (Kimonis, Frick, Fazekas, & Loney, 2006). The reverse may also be true. Lykken (1995), for example, posited that environmental factors, such as poor or ineffective parenting, lead to the development of outwardly displayed characteristics of psychopathy, which he referred to as sociopathy. Porter (1996) also theorised secondary psychopaths "turn-off" their emotions in order to cope with trauma, leading to the emotional blunting associated with psychopathy. It is also possible that antisocial traits and trauma exposure mutually influence the other such that exposure to trauma elevates rates of psychopathy (including increased aggressiveness and sensation seeking), thus increasing risk for further trauma exposure (Kimonis, Centifanti, Allen, & Frick, 2014; Lauterbach & Vrana, 2001). Thus, it is clear that further investigation of the associations between these variables is warranted and that longitudinal research may help elucidate possible temporal orderings.

A second potential limitation is the exclusive use of self-report measures. Future research efforts should address this concern through the use of clinician-ratings of PTSD and psychopathy. Third, although we used a valid and reliable measure of lifetime trauma exposure, because the study was cross-sectional rather than prospective, recall and reporting bias may have impacted the accuracy of participants' reports. Finally, we did not assess the impact of potentially traumatic events upon prisoners. Thus, in future research, a measure such as the Impact of Event Scale (IES) could be used as well to evaluate the predictive validity of a weaker traumatic stress reaction among more psychopathic prisoners.

In conclusion, the findings suggest an association between exposure to potentially traumatic events and PTSD among incarcerated male offenders. Importantly, this relationship was found not to be direct but was instead moderated by secondary psychopathy. Specifically, trauma exposure was positively associated with increased PTSD symptomology among individuals with low levels of secondary psychopathy, and negatively associated with PTSD symptomology with those with high levels of secondary psychopathy. These findings contribute to our understanding of the nature of the relationships between PTSD symptomology, psychopathy facets and trauma exposure, namely, that the association between trauma exposure and PTSD is explained by secondary but not primary psychopathic traits. Future longitudinal studies may advance our understanding of the mechanisms of the relationship between trauma exposure, psychopathy facets, and post-traumatic symptomatology.

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