

Is there a common pathway to developing ASD and PTSD symptoms?

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COMMON PATHWAYS TO ASD AND PTSD

Abstract

Numerous studies have identified risk factors for acute and long-term posttraumatic stress symptoms following traumatic exposure. However, little is known about whether there are common pathways to the development of acute stress disorder (ASD) and posttraumatic stress disorder (PTSD). Research suggests that a common path to ASD and PTSD may lie in peritraumatic responses and cognitions. The results of structural equation modeling in a national sample of Danish bank robbery victims ($N = 450$) show that peritraumatic panic, anxiety sensitivity, and negative cognitions about self were significant common risk factors for both ASD severity and PTSD severity when controlled for the effect of the other risk factors. The strongest common risk factor was negative cognitions about self. Future research should focus on replicating these results as they point to possible areas of preventive and treatment actions against the development of traumatic stress symptoms.

Keywords: Acute Stress Disorder, Posttraumatic Stress Disorder, common pathways, risk factors, bank robbery.

COMMON PATHWAYS TO ASD AND PTSD

Is there a common pathway to developing short-term and long-term traumatic stress symptoms?

1. Introduction

Aiding understanding of the mechanisms behind the development of posttraumatic stress symptoms is essential in order to identify victims at risk of developing posttraumatic stress symptoms. Increasing our understanding of these mechanisms will facilitate the development of effective treatment strategies. Numerous studies have investigated the prediction of acute and, in particular, long-term posttraumatic symptoms following different forms of traumatic exposure (Brewin, Andrews, & Valentine, 2000; Hansen & Elklit, 2011; Ozer, Best, Lipsey, & Weiss, 2003). As a result, several factors have been shown to be predictive of acute stress disorder (ASD) and posttraumatic stress disorder (PTSD), respectively. ASD and PTSD are both diagnoses in the Diagnostic and Statistical Manual of Mental Disorders 4th edition and also the recently released 5th edition (DSM-IV; APA, 1994; 2013) which follow traumatic exposure. According to the DSM-IV ASD describes acute posttraumatic symptoms (two days to one month) following traumatic exposure, whereas PTSD describes long-term posttraumatic symptoms (more than a month). The diagnostic criteria for ASD and PTSD are similar as they require symptoms of intrusion/re-experiencing, avoidance, and arousal. However, the ASD diagnostic criteria also state a requirement of dissociative symptoms. At the same time, only the PTSD diagnosis requires that intrusion involves or causes distress and the presence of at least three symptoms of avoidance and two symptoms of arousal, whereas the ASD diagnostic criteria only state a diffuse requirement of marked arousal and avoidance (Bryant & Harvey, 2002). Due to the strong similarities between the ASD and the PTSD diagnostic criteria, it stands to reason that there will be a strong association between the two disorders. Indeed, research shows a strong relationship between ASD severity and PTSD severity following different forms of traumatic exposure

COMMON PATHWAYS TO ASD AND PTSD

(cf. Classen, Koopman, Hales & Spiegel, 1998; Hansen & Elklit, 2013). Additionally, when disregarding the dissociation symptom cluster, a similar factor structure has also been found in the two diagnoses (Hansen, Armour, & Elklit, 2012). Research, however, indicates that this may only be in relation to severity and not in relation to diagnostic status given that an ASD diagnosis appears to have limited capacity in identifying those with later PTSD diagnostic status (Bryant, 2011). Due to the close relationship between ASD and PTSD, risk factors for ASD are likely to also be risk factors for PTSD and vice versa (Bryant & Harvey, 2002). Together this suggests that there perhaps exists a common path (i.e. a common combination of risk factors) to the development of ASD and PTSD. Numerous risk factors of both ASD and PTSD have been identified. However, research has shown that peritraumatic (i.e. peritraumatic dissociation, peritraumatic panic, and tonic immobility) and cognitive responses (i.e. negative cognitions about self, the world and panic) may be of particular importance for the development of posttraumatic symptoms (Ehlers, & Clark, 2000; Kunst, Winkel, & Bogaerts, 2011; Nixon & Bryant, 2003; 2005).

Different forms of peritraumatic responses are found to be associated with both the development of ASD and PTSD. In particular, peritraumatic dissociation has been found associated with the development of both ASD and PTSD following traumatic exposure (cf. Breh & Seidler, 2007; Bryant, 2009; Bryant & Panasetis, 2001; Ozer et al., 2003). The DSM-IV defines peritraumatic dissociation as a subjective feeling of emotional numbness, detachment from others, reduced responsiveness to one's surroundings, depersonalization, and derealization during the traumatic exposure (APA, 1994). Thus, dissociation can be regarded as an inner distancing mode allowing the individual to momentarily phase out reality (Breh & Seidler, 2007). Peritraumatic dissociation is assumed to be associated with the development of PTSD as this inner distancing prevents adequate processing and thus adequate integration of the traumatic memories. Ozer et al. (2003), in a meta-analysis,

COMMON PATHWAYS TO ASD AND PTSD

showed that peritraumatic dissociation is the strongest predictor of PTSD across different forms of traumatic exposure among seven investigated predictors. These results were largely replicated in the Breh and Seidler (2007) meta-analysis of prospective studies of peritraumatic dissociation and PTSD.

Peritraumatic panic and tonic immobility are also emerging as important risk factors associated with the development of posttraumatic stress symptoms (cf. Bryant et al., 2011; Marx, Forsyth, Gallup, Fusé, & Lexington, 2008; Rocha-Rego et al., 2009). Symptoms of panic are for instance palpitations, shaking, sweating, shortness of breath, nausea, dizziness, and fear of dying or losing control (APA, 1994). It has been suggested that peritraumatic panic may be associated with the development of posttraumatic stress symptoms as it may condition trauma related cues to subsequent anxiety (Bryant & Panasetis, 2001). Indeed, peritraumatic panic has been found to be associated with the development of both ASD and PTSD (Bryant & Panasetis, 2001; Bryant et al., 2011; Nixon & Bryant, 2003; Rocha-Rego et al., 2009). Tonic immobility is conceptualized as an evolutionary survival strategy and is characterized by involuntary immobility, analgesia, and unresponsiveness to external stimulus (Marx et al., 2008). Contrary to peritraumatic dissociation, tonic immobility is primarily a physical state and awareness of the surrounding environment is preserved (Heidt et al., 2005). The precise mechanism behind the relationship between tonic immobility and the development of PTSD is unclear, but several possible explanations have been put forward (Marx et al., 2008). For instance tonic immobility is said to be related to the development of PTSD as it hinders the use of more adaptive defensive responses during or after traumatic exposure. Indeed, tonic immobility has been found associated with the development of PTSD (Heidt, Marx, & Forsyth, 2005; Kunst et al., 2011; Rocha-Rego et al., 2009). However, to the best of our knowledge, tonic immobility has not been investigated in relation to ASD.

COMMON PATHWAYS TO ASD AND PTSD

Recently there has been an increased focus on role of cognitions in explaining the development and maintenance of posttraumatic stress symptoms (Cahill & Foa, 2007). According to cognitive theories on PTSD, negative cognitions appear to play a central role in the development of both acute and long-term posttraumatic symptomatology. Specifically, Ehlers and Clark's (2000) cognitive model of PTSD has emphasized the importance of negative cognitions about the traumatic event and one's capacity to respond to that experience for adapting to traumatic exposure. Negative cognitions may hinder the victims from processing corrective information and thus prevent the victims from realizing that the threat has passed. Thus, these negative cognitions may lead to a sense of current threat which can contribute to the development as well as the maintenance of posttraumatic stress symptoms. Indeed, negative cognitive responses to traumatic exposure have been associated with the development of both ASD and PTSD symptoms (Hansen & Elklit, 2011; Karl, Rabe, Zöllner, Maercker, & Stopa, 2009; Nixon & Bryant, 2005). Furthermore, according to Karl et al. (2009) research show that more theoretical grounded factors (i.e. cognitive factors) may be better predictors of PTSD than less theoretical and more empirically grounded factors such as peritraumatic dissociation. There are two broad classes of negative cognitions (Ehlers, & Clark, 2000): those that focus on the self and those that focus on the world. However, research indicates that these cognitive responses also include maladaptive thinking associated with panic (i.e. anxiety sensitivity; Nixon & Bryant, 2005). Indeed, anxiety sensitivity has been found associated with the development of both ASD and PTSD symptoms (cf. Bryant & Panasetis, 2001; Nixon & Bryant, 2005).

To summarize, research has shown a strong relationship between ASD and PTSD, both with respect to symptom severity as well as in relation to common pathways /risk factors. Research suggests that common pathways may exist through the three above stated peritraumatic responses and the three cognitive factors. The aim of the present study was

COMMON PATHWAYS TO ASD AND PTSD

therefore to investigate whether these six risk factors are common risk factors for both ASD and PTSD severity while controlled for the effect of the other risk factors and control factors and not to investigate the strength of these risk factors as such. Given the extant literature, we hypothesized that the three peritraumatic risk factors and three forms of cognitions would be significantly associated with both ASD and PTSD severity. To the best of our knowledge no other study has previously investigated the above stated risk factors collectively in relation to both ASD and PTSD. Thus, it is unknown which factors will be significant common risk factor for ASD and PTSD severity when controlled for the effect of the other factors. However, due to previously mentioned research we expected that the effect of the peritraumatic factors would diminish whilst controlling for the effect of negative cognitions. The conceptual model investigating the common paths to ASD and PTSD is shown in figure 1.

[Please insert Figure 1 about here].

2. Method

The current study is part of a national Danish cohort questionnaire survey of the psychological impact of bank robberies conducted in collaboration with the Danish Bankers Association, the National Bank of Denmark, all Danish Banks, and the University Southern of Denmark. From April 2010 to April 2011, every bank employee exposed to bank robbery in Denmark ($N = 614$) received the first questionnaire. A total of 450 employees (73%) filled out the first questionnaire a week after the robbery ($T1$, $M = 9.89$ days, $SD = 6.30$), and a total of 371 of these (82 %) filled out the second questionnaire six months after the robbery ($T2$, $M = 191.7$ days, $SD = 13.15$). There were no significant group differences in ASDS scores or any of the control factors or risk factors in relation to the dropout between $T1$ ($N = 450$) and $T2$ ($N = 371$). All participants were informed of the purpose of the study orally and in writing. Furthermore, all necessary permissions for conducting this study, according to

COMMON PATHWAYS TO ASD AND PTSD

Danish Law, were obtained and participation was voluntary. The current study is based on the DSM-IV criteria as the DSM-5 criteria was not available when conducting the study. Please see Hansen and Elklit (2014) for further details on the study.

Measures

The questionnaire assessed demographic factors and the following factors related to the current study. All of the following described control factors, risk factors, and ASD severity were assessed at T1 and PTSD severity was assessed at T2. Prior traumatic exposure was assessed as the sum of endorsements from a checklist of 14 different kinds of traumas used in the U.S. National Comorbidity Survey (Kessler et al., 1995), and a separately dichotomously scoring (*yes* or *no*) indicating if the participant had experienced a previous bank robbery. Four of the following scales (e.g. the Peritraumatic Dissociation Questionnaire (PDEQ), Marmar, Weiss, & Metzler, 1997; Physical Reaction Scale (PRS), Falsetti & Resnick, 1992; Tonic Immobility Scale (TIS), Heidt et al., 2005, and Posttraumatic Cognition Inventory (PTCI), Foa, Ehlers, Clark, Tolin, & Orsillo, 1999) were translated into Danish for this study, whereas the remaining scales have already been translated and used in Danish populations. The original English versions of the PDEQ, the PRS, the TIS and the PTCI were translated into Danish by two independent translators, who were fluent in both Danish and English. The two translations were compared and combined into single translations by the two translators (i.e. the committee approach; Simonsen & Elklit, 2008), which was then back-translated into English by a third translator. The third translator (or back-translator) was also fluent in both Danish and English. The back-translation was then compared to the original English items with a very high correspondence in relation to conveying the meaning.

The Danish version of the PDEQ (Marmar et al., 1997) was used to assess peritraumatic dissociation. The summed score for the PDEQ comprises responses to 10 items reflecting dissociative responses rated on a five-point Likert type scale (0= *not at all* to 5=

COMMON PATHWAYS TO ASD AND PTSD

extremely true) during the robbery. The PDEQ has shown satisfactory internal consistency and test-retest reliability (.72; cf. Kunst et al., 2011). The reliability coefficient in the current study was satisfactory (Cronbach's $\alpha = .88$).

Peritraumatic panic was assessed using a modified Danish version of the PRS (PRS; Falsetti & Resnick, 1992). The PRS is a 16-item self-report questionnaire that measures how severely the participants experienced DSM-IV panic symptoms during the robbery rated on a six-point Likert type scale (0= *not at all* to 5= *extremely*). Bryant et al. (2011) have reported difficulties with the factor structure on the 16 items, but not on 10 of the items and so only the 10 items were used. The reliability coefficient for the scale in the current study was high (Cronbach's $\alpha = .96$).

The Danish version of the TIS (Heidt et al., 2005) was used to assess tonic immobility. The TIS originally comprised 10 items rated on a seven-point Likert scale (a low score indicates no symptom presence and a high score indicates high symptom severity). In order to avoid item overlap with the PDEQ and the PRS only the four items assessing the motor aspects of tonic immobility were used. Other studies have used the same procedure (Kunst et al., 2011; Rocha-Rego et al., 2009). Previous studies report good internal consistency Cronbach's $\alpha = .88$ (cf. Kunst et al., 2011). The reliability coefficient in the current study was satisfactory (Cronbach's $\alpha = .86$).

Anxiety sensitivity (i.e. proneness to panic reactions or negative cognitions about bodily sensations) was assessed using the Danish version of the Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986). The ASI is a 16-item questionnaire that measures beliefs about the harmfulness of anxiety symptoms rated on a five-point Likert type scale (1= *very little*, 5= *very much*). Good test-retest reliability (.75) and construct validity have been reported (Reiss et al., 1986). The reliability coefficient in the current study was high (Cronbach's $\alpha = .91$).

COMMON PATHWAYS TO ASD AND PTSD

The Danish version of the PTCI (Foa et al., 1999) was used to assess posttraumatic cognitions. The PTCI is a 33 item self-report scale with three subscales assessing negative cognitions about self (NCS), negative cognitions about the world (NCW), and self-blame (SB) rated on a seven-point Likert Scale (1 = *totally disagree*, 7 = *totally agree*). Scale scores are formed for the three subscales. The PTCI has demonstrated high convergent validity with other trauma-related cognition scales and good sensitivity and specificity in relation to correctly identifying PTSD diagnostic status (Beck et al., 2004; Foa et al., 1999). Difficulties with the self-blame subscale have been reported previously (cf. Beck et al., 2004) and so only the NCS ($\alpha = .85$) and NCW ($\alpha = .85$) scales were used.

ASD severity was assessed using the Danish version of the ASDS (Bryant, Moulds & Guthrie, 2000). The ASDS is a 19 item self-report scale with four subscales assessing the four DSM-IV symptom clusters dissociation, re-experiencing, avoidance, and arousal rated on a five-point Likert type scale (1 = *not at all* to 5 = *very much*). Previous studies using the Danish ASDS have used this procedure and have reported good reliability (Cronbach's $\alpha = .76$ -.96; cf. Hansen, Armour, & Elklit, 2012) for the total scale. The reliability of the total scale in the current study was high (Cronbach's $\alpha = .93$).

The Danish version of the Harvard Trauma Questionnaire (HTQ; Mollica et al., 1992) was used to assess PTSD severity. The HTQ is a 17 item self-report scale with three subscales assessing the three separate symptom clusters intrusion, arousal, and avoidance as specified by the DSM-IV rated on a four-point Likert type scale (1 = *not at all*, to 4 = *all the time*). The Danish version of the HTQ has been used in a wide range of trauma populations with reports of good reliability and validity (cf. Bach, 2003). The reliability of the total scale in the current study was high (Cronbach's $\alpha = .92$).

2.1 Data-analysis

COMMON PATHWAYS TO ASD AND PTSD

Descriptive and bivariate statistical analyses were performed using SPSS version .21. A general statistical model, based on the conceptual model in Figure 1, was specified and tested using Mplus 6.1 (Muthén & Muthén, 2010). All models were estimated using robust maximum likelihood (MLR) as it is the most appropriate estimator under conditions of non-normality (Satorra & Bentler, 1994) and allows the use of all available information rather than using listwise deletion (Schafer & Graham, 2002). The directly observed variables (in rectangles in Figure 1) were entered directly, and the scale scores representing latent variables (ellipses in Figure 1) were corrected for measurement error by specifying a latent variable with one observed variable and fixing the error variance (1-scale reliability * scale variance) using the estimate of Cronbach's alpha as an estimate of reliability (Anderson & Gerbing, 1988; Sörbom & Jöreskog, 1982).

The overall aim of the analyses was to find the most parsimonious model that adequately explained the data. Specifically we aimed to find the degree to which ASD and PTSD shared common risk factors, or common pathways; a common pathway was operationalized by the regression coefficients from the same risk factor for both ASD and PTSD being the same (not significantly different). To this end we specified the general model with no restrictions; this was the baseline model. Then the model was estimated with equality constraints placed on each of the risk factors separately to determine the impact of this restriction on model fit. If these constrained models did not result in a poorer fit compared to the baseline model then that particular risk factor was considered to have a common pathway to ASD and PTSD. We then tested how many equality restrictions could be included in the model simultaneously without the model fit becoming worse than the baseline model. Successive restrictions were added, and the order that they were included was based on the fit statistics from the preceding analyses. The adding of the constraints was guided primarily by the Bayesian Information Criterion with lower value representing better model fit. The BIC

COMMON PATHWAYS TO ASD AND PTSD

was used due to its ability to identify the best parsimonious model by penalizing model complexity. The BIC allows for the assessment of the relative fit of different models. Other fit statistics were used to assess overall fit. We also inspected the chi-square (χ^2), the Comparative Fit Index (CFI), Tucker-Lewis Fit Index (TLI), the Akaike's Information Criterion (AIC), the Root Mean Square Error of Approximation (RMSEA), and the standardized root mean squared residual (SRMR). Adequate model fit is demonstrated by a non-significant χ^2 , CFI's and TLI's greater than .90, RMSEA's less than .08, and SRMRs less than .09.

3. Results

Descriptives and correlations of all control factors, risk factors, ASD severity, and PTSD severity are shown in Table 1. The percentages of missing values ranged from 0% to 18.7%. A total of 53 participants (11.8%) suffered from ASD measured by the ASDS (Bryant et al., 2000) and a total of 27 participants (6.0%) suffered from PTSD measured by the HTQ (Mollica et al., 1992). Table 2 shows the fit statistics for the models of common paths to ASD severity and PTSD severity from a model with no constraints (the baseline model), models with single constraints on a particular control factor or risk factor, models with multiple constraints on the control factors and/or risk factors to the fully constrained model.

[Please insert Table 1 and Table 2 about here].

Table 2 shows that the χ^2 values of all models were statistically significant. However, this should not lead to rejection of models given that chi-square values tend to become non-significant when assessing model fit with large samples (Tanaka, 1987). All of the models reached the recommend values of the CFI and the SRMR for adequate fit, and the majority of the models reached the recommend values of the RMSEA for adequate fit. However, none of the models reached the recommended values of the TLI for adequate fit. The relative fit of

COMMON PATHWAYS TO ASD AND PTSD

the models improved as the number of constraints increased; Model 17 had the lowest AIC and Model 18 had the lowest BIC value. Furthermore, Model 17 and Model 18 had identical values on the CFI (.92), the TLI (.87), RMSEA (.073), and SRMR (.039). The difference between the models in relation to the BIC values was too small to be significant. However, we argue that model 18 is slightly superior to model 17 as model 18 is more parsimonious. Thus, the regression coefficients for model 18 (constraints on all control factors and risk factors except peritraumatic dissociation) are shown in table 3.

[Please insert Table 3 about here].

Table 3 shows that two of the control factors (female sex and prior robbery exposure) and three of the risk factors (anxiety sensitivity, peritraumatic panic, and negative cognitions about self) contributed significantly to the variance explained in both ASD severity (74 %) and PTSD severity (53 %). On the other hand, peritraumatic dissociation contributed significantly to the variance explained in ASD severity only. A further inspection of the standardized regression coefficients showed that negative cognitions about self was the strongest common contributing factor for both ASD severity ($\beta = .22, p < .001$) and PTSD severity ($\beta = .31, p < .001$).

4. Discussion

The current study investigated peritraumatic responses and negative cognitions as a possible common pathway, or risk factors for, ASD severity and PTSD severity in a cohort study of 450 Danish bank employees exposed to bank robbery. In accordance with previous research, we found that all the investigated risk factors were significantly and moderately to strongly associated with both ASD severity and PTSD severity. However, only anxiety sensitivity, peritraumatic panic, and negative cognitions about self were significant risk factors for both ASD severity and PTSD severity, whereas peritraumatic dissociation was only a significant risk factor for ASD severity whilst controlled for the effect of the other

COMMON PATHWAYS TO ASD AND PTSD

factors. Additionally, two of the control factors (female sex and prior robbery exposure) were also significant risk factors for both ASD and PTSD severity. However, the relation between prior robbery exposure and ASD/PTSD in the multivariate analyses is likely to be a statistical artifact as prior exposure was not significantly associated with ASD severity or PTSD severity in bivariate analyses. Thus, a common pathway to acute and long-term traumatic stress symptoms seems to exist through peritraumatic panic, anxiety sensitivity, and negative cognitions about self (as well as female sex). Several aspects of the results need to be commented on.

Contrary to our expectations, the significant bivariate association between peritraumatic dissociation and PTSD severity was no longer present in the multivariate analyses. However, peritraumatic dissociation was still a significant risk factor for ASD severity whilst controlling for the other factors. At the same time, the effect of peritraumatic panic on ASD severity and PTSD severity did not diminish after controlling for the effect of the other factors. Finally, as expected, the effect of tonic immobility did diminish after controlling for the effect of the other factors. Research suggest that the relationships between these three peritraumatic factors are rather complex (Bryant et al., 2011; Heidt et al., 2005; Kunst et al., 2011). Although we reduced the overlap between the three constructs by removing overlapping items from the TIS with the PDEQ and the PRS (i.e. only assessing the motor aspects of tonic immobility), the three peritraumatic factors were still moderately to strongly associated with each other. Similarly, theoretical discussions of the relationship between these factors also indicate that they may not be independent factors. For instance tonic immobility has been suggested to represent a behavioral manifestation of peritraumatic dissociation, and the effect of panic on posttraumatic stress symptoms has been found to be mediated by peritraumatic dissociation (Bryant et al., 2011; Heidt et al., 2005; Kunst et al., 2011). However, despite the complex relationship between the three peritraumatic factors,

COMMON PATHWAYS TO ASD AND PTSD

peritraumatic panic seems to have had an independent direct effect on both ASD and PTSD severity in the current study. Thus, peritraumatic panic appears to add something unique to the explanation of both acute and long-term posttraumatic stress symptoms which goes beyond what the other included factors can account for. In particular, this unique contribution seems to be beyond the cognitive element of panic as peritraumatic panic is still a significant risk factor when controlling for the cognitive part of panic (i.e. anxiety sensitivity). Thus, this unique part of peritraumatic panic seems to be related to peritraumatic arousal. This is in accordance with fear conditioning models of PTSD, which assume that peritraumatic panic may independently condition trauma related cues to subsequent anxiety (i.e. ASD & PTSD; Bryant & Panasetis, 2001). The arousal part of peritraumatic panic may be directly related to ASD and PTSD in relation to the development of more persistent symptoms of arousal and/or symptoms of intrusion/re-experiencing. Indeed, hyperventilation induced arousal (measured by the PRS) has been found associated with an increased activation of trauma memories and greater distress in ASD participants compared to non-ASD participants (c.f. Hopwood & Bryant, 2006). Similarly, other forms of interoceptive exposure (e.g. breath holding or spinning) have also been shown to trigger arousal related sensations and trauma memories in PTSD patients (Wald & Taylor, 2008). This is also in accordance with the Dual Representation Theory of PTSD, which assumes that sensory information can be processed both consciously (verbally accessible memory; VAM) and unconsciously (situationally accessible memory; SAM) (Brewin, Dalgliesh & Joseph, 1996). Peritraumatic panic and other physical reactions are more likely to be stored as SAM rather than VAM. Furthermore, SAMs are likely to be experienced as flashbacks or cued physiological arousal and unintentionally triggered by both external and internal (e.g. physiological arousal) trauma reminders (Brewin et al., 1996). Thus, peritraumatic panic may be uniquely connected to

COMMON PATHWAYS TO ASD AND PTSD

ASD and PTSD through arousal and result in triggering both flashbacks and subsequent arousal.

The fact that peritraumatic dissociation was only a significant risk factor for ASD severity, whilst controlling for the other factors, is likely to be due to a unique relationship between ASD severity and peritraumatic dissociation. This was indicated by the stronger bivariate association between ASD severity and peritraumatic dissociation ($r = .70$) compared to the bivariate association between peritraumatic dissociation and PTSD severity ($r = .47$), and a larger part of the variance explained in ASD severity compared to PTSD severity. This difference is not surprising as the ASD diagnosis includes a dissociation symptom cluster whereas the PTSD diagnosis does not. Indeed, previous research has treated peritraumatic dissociation as somewhat equivalent to the ASD dissociation symptom cluster (Bryant, 2009). The structural equation modelling analyses in the current study also underlined that the peritraumatic dissociation was not a common risk factor for ASD severity and PTSD severity as models containing constraints on the PDEQ were the worst fitting models. At the same time, the results are in accordance with a more recent review of methodologically sound prospective studies of the relationship between peritraumatic dissociation and PTSD (van der Velden & Wittmann, 2008). This review showed that peritraumatic dissociation may not be an independent predictor of PTSD. Instead, the relationship between peritraumatic dissociation and PTSD tends to become non-significant when controlling for other factors. In the current study, these other factors are likely to be anxiety sensitivity, negative cognitions about self, and peritraumatic panic; as these were significant risk factors and at the same time were correlated moderately to strongly ($r_s = .42-.57$) with peritraumatic dissociation. Thus, our results confirm previous findings of the association between peritraumatic dissociation and posttraumatic stress symptoms losing its significance when controlling for variables reflecting early mental health responses.

COMMON PATHWAYS TO ASD AND PTSD

As expected, the effect of the motor aspects of tonic immobility diminished after controlling for the effect of the other factors, and tonic immobility was not a significant risk factor for either ASD severity or PTSD severity. This is in accordance with the Kunst et al. (2011) study, which found that tonic immobility was not a significant risk factor for PTSD severity when the effect of other peritraumatic responses was controlled for. However, it has been argued that the effect of tonic immobility on the development of PTSD may depend on a third factor such as self-blame or guilt for not fighting back, which we did not account for in the current study (cf. Heidt et al., 2005; Kunst et al., 2007). It is also possible as argued by Hansen and Elklit (2014) that tonic immobility following bank robbery is not of the same clinical relevance as it is following other forms of assault; for example, being told to stand still during a robbery does not equate to not being able to move. However, it remains possible that tonic immobility have had an indirect effect on the development of both ASD severity and PTSD severity as tonic immobility was moderately associated with peritraumatic panic, anxiety sensitivity, and negative cognitions about self ($r_s = .36 - .48$). In relation to ASD severity, the indirect effect is also likely to have been through peritraumatic dissociation. Indeed, tonic immobility was strongly associated with peritraumatic dissociation ($r = .53$).

As expected from previous research, negative cognitions about self and anxiety sensitivity were significant risk factors for both ASD severity and PTSD severity. Thus, in accordance with previous research both maladaptive thinking associated with one's self and bodily sensations (i.e. anxiety sensitivity) appear to be associated with the development of both ASD and PTSD. Additionally, negative cognitions about self were the strongest common risk factor. However, negative cognitions about the world were not a significant risk factor for ASD severity or PTSD severity. The pronounced role of the cognitions found in the current study is in accordance with previous research showing that theoretical grounded factors may be better predictors of posttraumatic stress symptoms than less theoretical

COMMON PATHWAYS TO ASD AND PTSD

grounded factors (cf. Karl et al., 2009). The results are also in accordance with research showing that negative cognitions about the self, appear to have a more pivotal role in the development of posttraumatic stress symptoms than other forms of cognitions (cf. Karl et al., 2009; O'Donnell, Elliott, Wolfgang, & Creamer, 2007). The importance of cognitions in relation to the development of PTSD is also in accordance with the newly released DSM-5, where cognitions have been recognized as a fourth symptom cluster in the PTSD diagnosis (APA, 2013). Although previous research suggest that the relationship between negative cognitions about the self and PTSD symptoms increases over time (O'Donnell et al., 2007), the current study suggest that the relationship is already pronounced in the acute phase following traumatic exposure. However, the potential significance of cognitions in relation to the ASD diagnosis is not reflected by the DSM-5 criteria (APA, 2013).

4.1 Limitations

The current study has several limitations. It was only possible to assess the participants twice. Thus, the results are based on a cross-sectional or quasi-prospective study at the best in relation to ASD and interpretations about causality should be made carefully as potential recall biases cannot be ruled out. However, recall biases are likely only to be small as only a week has passed since the traumatic exposure. Furthermore, we could have used modification indices to generate better fitting models. However, doing so would have made the models more specific to our data and perhaps less generalizable to alternative trauma populations. Instead, we chose a strategy of model modification, guided by the BIC, to identify the most parsimonious model that adequately fitted the data. A potential problem with this approach is that the comparison of constrained and non-constrained models may lack sensitivity to small differences in effects in the population. This would increase the likelihood of finding 'common' effects for ASD and PTSD. However, the addition of these model constraints only caused very small changes in the overall model fit statistics compared to the baseline

COMMON PATHWAYS TO ASD AND PTSD

indicating that our conceptual model was robust. Finally, both the ASD and the PTSD diagnosis and severity were based on self-report measures rather than clinical interviews. Despite both self-report measures being standardized scales, the estimated ASD and PTSD prevalence rates and severity may be biased. Additionally, the majority of the participants had relatively low levels of both ASD and PTSD. Although, this study has a high response rate and the prevalence rates are comparable to rates found in other bank robbery studies (Hansen & Elklit, 2013), it remains possible that participants with the most severe symptomatology chose not to enroll in the study. Additionally, it is possible that risk factors for posttraumatic stress symptomatology may depend on the severity of posttraumatic stress symptoms. Thus, the results of the current study may not be generalizable to trauma populations known for higher levels of symptomatology, for example rape victims.

4.2 Conclusion

The current study is the first to investigate peritraumatic dissociation, peritraumatic panic, tonic immobility, anxiety sensitivity, negative cognitions about the world, and negative cognitions about self as possible common pathways to the development of both ASD and PTSD in a highly representative study of bank robbery victims. Results show that a common pathway to ASD severity and PTSD severity appears to exist through peritraumatic panic, negative cognitions about bodily sensations, and negative cognitions about the self. At the same time, peritraumatic dissociation was only a significant risk factor for ASD severity indicating that different pathways to ASD and PTSD may also exist. Together with one control factors (female sex), these factors explained 74% of the variance in ASD severity and 53% of the variance in PTSD severity suggesting that if there does exist a common pathway to ASD severity and PTSD severity then a large part still remains to be accounted for. The strongest common risk factor was negative cognitions about self. The pronounced role of negative cognitions in the development of both ASD severity and PTSD severity found in the

COMMON PATHWAYS TO ASD AND PTSD

current study supports the inclusion of negative cognitions in the DSM-5 PTSD diagnosis. At the same time, the results suggest that it may be beneficial to also recognize cognitions in the ASD diagnosis. Future research should focus on replicating these results across different trauma populations with higher prevalence rates of ASD and PTSD as they point to possible areas of preventive and treatment actions to be taken against the development of both acute and long-term posttraumatic symptoms.

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