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ARTICLE



## Assessing the existence of dissociative PTSD in sub-acute patients of whiplash

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### ABSTRACT

Numerous studies investigating dissociative posttraumatic stress disorder (D-PTSD) have emerged. However, there is a lack of studies investigating D-PTSD following a wider range of traumatic exposure. Thus, the present study investigates D-PTSD using latent class analysis (LCA) in sub-acute patients of whiplash and associated risk factors. The results of LCA showed a three-class solution primarily distributed according to posttraumatic stress disorder (PTSD) symptom severity and thus no indication of D-PTSD. Dissociative symptoms, psychological distress (i.e. anxiety/depression), and pain severity significantly predicted PTSD severity. Combined, the results support the component model of dissociation and PTSD, while still stressing the importance of dissociative symptoms when planning treatment for PTSD.

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### KEYWORDS

Whiplash; pain; dissociative PTSD; latent structure

Since the introduction of the dissociative posttraumatic stress disorder (D-PTSD) subtype in the fifth edition of the diagnostic and statistical manual of mental disorders (DSM-5; APA, 2013), numerous studies testing the existence of D-PTSD have emerged (Hansen, Ross, & Armour, 2017). Furthermore, several studies have indicated that coexisting dissociative symptoms may negatively affect a number of posttraumatic stress disorder (PTSD) treatments (Cloitre, Petkova, Wang, & Lu, 2012; Resick, Suvak, Johnides, Mitchell, & Iverson, 2012). For example, clients with high levels of dissociative symptoms may not respond as well to exposure-based psychotherapy compared to clients with low levels of dissociation (Bae, Kim, & Park, 2016; Cloitre et al., 2012; Resick et al., 2012). For this reason, D-PTSD in the context of whiplash injury may pose a specific challenge since the majority of interventions for pain and comorbid PTSD are exposure-based therapies (Asmundson, 2014). Whiplash refers to the mechanism of the injury, the rapid and sudden extension and flexion of the neck following a rear-end collision. At the same time, comorbid presentations of pain and PTSD are common (approximately 20%) after traffic injuries (Beck & Clapp, 2011). The identification

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of D-PTSD and associated risk factors therefore has potential clinical impact both in terms of preventive measures and treatment planning.

According to the DSM-5 diagnostic criteria for D-PTSD, individuals must in addition to endorsing the criteria for a PTSD diagnosis (i.e. a specific number of symptoms of intrusion (B1–B5), avoidance (C1–C2), negative alternations in cognitions and mood (D1–D7), and alterations in arousal and reactivity (E1–E6)) also report persistent or recurrent symptoms of either depersonalization (e.g. feelings of disconnectedness or detachment from self or body) or derealization (e.g. feelings of unreality of surroundings). Dissociative symptoms can more broadly be defined as ‘an experienced loss of information or control over mental processes that, under normal circumstances, are available to conscious awareness, self-attribution, or control, in relation to the individual’s age and cognitive development’ (p. 251) (Cardeña & Carlson, 2011). Although, the importance of dissociative symptoms in response to traumatic experiences has just recently been added to the diagnostic nomenclature, the occurrence of dissociative responses following traumatic experiences has been scientifically acknowledged for over a century (Janet, 1907). At the same time, numerous studies have shown associations between dissociation and posttraumatic stress symptoms following a wide range of traumatic exposure (c.f. Dalenberg et al., 2012). According to two of the most prominent models (e.g. the Component Model and the Subtype Model), dissociation is hypothesized to be a part of traumatic responding. In the Component Model, dissociative symptoms can co-occur with PTSD symptoms, whereas, in the Subtype Model, dissociative symptoms can change the phenomenology of PTSD symptoms (Dalenberg & Carlson, 2012). Thus, if the Subtype Model is accurate and the Component Model is not, victims with and without dissociative symptoms would display qualitatively different patterns of symptomatology. Hence the D-PTSD is also expected to be differently associated with psychosocial covariates compared to PTSD only (Dalenberg & Carlson, 2012).

According to a recent review (Hansen et al., 2017), the D-PTSD construct has been identified with estimated prevalence rates ranging from 6% to 44.6% ( $M = 20.35\%$ ) across 12 different trauma samples (primarily different kinds of interpersonal violence and/or military trauma) with estimated PTSD prevalence rates ranging from 2% to 100% (Hansen et al., 2017). Of the numerous investigated covariates across the studies, only childhood physical and sexual assault appeared to emerge as a consistent risk factor across the studies. This may suggest that common risk factors for D-PTSD following different forms of traumatic exposure do not exist as different risk factors emerged across the studies or that more prominent common risk factors for D-PTSD have simply not yet been identified.

Of note to the best of our knowledge, there has only been one study of D-PTSD in the context of patients suffering from whiplash (Hansen,

Müllerová, Elklit, & Armour, 2016; Hansen et al., 2017). Hansen, Müllerová et al (2016) found an estimated D-PTSD prevalence rate of 37.4% in victims of motor vehicle accidents meeting caseness for PTSD ( $M = 5.17$  years after traffic accident). Furthermore, only one study included in the review has failed to identify the dissociative subtype; this study assessed data of victims of bank robbery (Hansen, Hyland, & Armour, 2016). The negative result may be connected to the specific nature of the traumatic exposure. This underlines the need to investigate D-PTSD following a wider range of traumatic exposure, a wider range of risk factors for D-PTSD, and within a shorter time-frame than previously investigated. Thus, the present study investigates the D-PTSD within sub-acute whiplash patients. Neck-injury-related models have failed to explain the chronic whiplash symptoms (McLean, 2016). However, recently new promising models have emerged drawing attention to the potential negative impact of the psychological reactions to the traumatic event. Due to the nature of a motor vehicle crash being a sudden and uncontrollable event associated with helplessness, it has been hypothesized, that dissociation may be an important mechanism or risk factor in the development of both PTSD and chronic whiplash associated disorder (Carlson & Dalenberg, 2000; Scaer, 2001, 2014). Furthermore, when pain and PTSD are the result of the same traumatic event, several interrelated mechanisms such as anxiety, catastrophic thinking, hyperarousal, and avoidance behaviors may mutually maintain both conditions (Andersen, Karstoft, Brink, & Elklit, 2016; Sharp & Harvey, 2001). Moreover, Beck and Clapp (2011) have suggested that dissociation may also be an important factor which negatively contributes to the mutual maintenance of pain and PTSD.

The aim of the present study was twofold. The first aim was to determine if D-PTSD is present within sub-acute victims of whiplash trauma. The second aim was to investigate the relationship between a range of known risk factors for PTSD within sub-acute victims of whiplash trauma (i.e. the experience of subjective pain and pain catastrophizing) while controlling for the effect of previously identified risk factors in the D-PTSD literature (i.e. sex, age, anxiety/depression, and controlling for the days since the injury) and D-PTSD.

## **Method**

### ***Participants and procedures***

The present study is a prospective study with two measurement points and is part of a larger prospective cohort postal questionnaire survey investigating recovery from whiplash trauma (see Andersen et al., 2016). The study was approved by the Danish Data Protection Agency and the review board of the University of Southern Denmark. At the emergency room, all patients were assessed for neck pain, cervical range of movement, and sensibility

disturbances according to normal clinical procedure. Whiplash severity was classified from grade 0-IV according to the Quebec Task Force classification of whiplash-associated disorders (WAD; Spitzer, Skovron, Salmi et al., 1995). WAD grade IV was excluded from the study, since those with WAD grade IV are characterized by having a fracture or a dislocation and therefore not exclusively a soft-tissue injury. Only patients over 18 years of age suffering from whiplash grade I-III were included in the present study. All participants gave informed consent to participate in the study. Head injury, unconsciousness, and other serious treatment requiring sustained injuries lead to exclusion from the study. A total of 327 of 578 participants (57%) returned the questionnaire at T1 within the first month of their whiplash injury (T1,  $M = 19$  days,  $SD = 13.3$ ). A total of 234 answered (72%) the questionnaire at T2 three months after the traumatic exposure ( $M = 104.31$  days,  $SD = 12.68$ ). There were no significant group differences on any T1 scores in relation to the dropout between T1 and T2,  $p > .05$ . Demographic information is included in Table 1.

### Measures

All the following risk factors (i.e. pain severity and pain catastrophizing) and control factors (i.e. age, sex, and psychological distress (i.e. anxiety/depression)) were assessed at T1 and PTSD and days since the injury were assessed at T2. For the latent class analysis (LCA), dissociation was assessed at T2 as part of the PTSD symptom classes, but for the regression analyses dissociation was assessed at T1 as a risk factor.

In accordance with Hansen, Hyland, Armour, Shevlin, and Elklit (2015) and Hansen, Müllerová et al. (2016), we assessed DSM-5 PTSD symptoms and dissociative symptoms with a combination of items from the Harvard Trauma Questionnaire Part IV (HTQ; Mollica et al., 1992) and the Trauma Symptom Checklist (TSC; Briere & Runtz, 1989; see Table 2 for item specifications). Although designed to reflect the DSM-IV and more general posttraumatic stress symptoms, the HTQ items from the full 31 item scale largely reflect the newly introduced DSM-5 PTSD symptoms. Answers are rated on a four-point Likert-type scale (1 = *not at all*, to 4 = *all the time*). The TSC measures symptoms associated with traumatic stress rated on a four-point Likert scale (1 = *never*, 4 = *always*). Cronbach alpha values ( $\alpha$ ) were satisfactory for the used HTQ items ( $\alpha = .92$ ) and the two TSC items used to measure dissociation at T1 ( $\alpha = .75$ ) and T2 ( $\alpha = .78$ ). The diagnostic DSM-5 criteria for PTSD are met if the participants endorse at least one symptom of intrusion, one symptom of avoidance, two symptoms of negative cognitions and mood, and two arousal symptoms, all indicated by item scores  $\geq 3$  on the HTQ or the TSC for the E2 criteria. The diagnostic criteria for D-PTSD was met if the participants in addition to meeting the criteria for PTSD also endorsed a symptom of either depersonalization or



**Table 2.** Item mapping for DSM-5 PTSD symptoms including dissociation (Hansen et al., 2015; Hansen, Müllerová et al. 2016).

DSM-5 symptoms of PTSD	HTQ and TSC items
B1. Intrusive thoughts	HTQ1 Recurrent thoughts or memories of the most hurtful or terrifying events
B2. Distressing dreams	HTQ3 Recurrent nightmares
B3. Dissociate reactions	HTQ2 Feeling as though the event is happening again
B4/5. Emotional reactivity and physiological reactivity	HTQ16 Sudden emotional or physical reaction when reminded of the most hurtful or traumatic events
C1. Efforts to avoid thoughts	HTQ15 Avoiding thought or feelings associated with the traumatic or hurtful events
C2. Efforts to avoid reminders	HTQ11 Avoiding activities that remind you of the traumatic or hurtful event
D1. Trauma-related amnesia	HTQ12 Inability to remember parts of the most hurtful or traumatic events
D2. Negative beliefs about oneself	HTQ14 Feeling as if you don't have a future
D3. Self-blame	HTQ19 Blaming yourself for the things that have happened
D4. Negative emotional state	HTQ23 Feeling ashamed of the hurtful or traumatic events that have happened to you/HTQ21. Feeling guilty for having survived/HTQ31. Feeling guilty for not doing anything or not doing enough
D5. Diminished interest in activities	HTQ13 Less interest in daily activities
D6. Detachment	HTQ4 Feeling detached or withdrawn from people
D7. Inability to feel positive emotions	HTQ5 Unable to show emotions
E1. Irritability/anger	HTQ10 Feeling irritable or having outburst of anger
E2. Reckless or self-destructive behavior.	TSC21 Do you want to harm yourself physically?
E3. Hypervigilance	HTQ9 Feeling on guard
E4. Exaggerated startle response	HTQ 6 Feeling jumpy and easily startled
E5. Difficulty concentrating	HTQ7 Difficulty concentrating
E6. Sleep disturbance	HTQ8 Trouble sleeping
Depersonalization	TSC32 Do you sometimes feel as you are outside your body?
Derealization	TSC30 Do you have a sense of unreality?

Note. HTQ: Harvard Trauma Questionnaire; TSC: Trauma Symptom Checklist. PTSD symptoms are assessed with the HTQ in accordance with Hansen et al. (2015), and dissociation were assessed with TSC in accordance with Hansen, Müllerová et al. (2016).

derealization indicated by indicated by item scores  $\geq 3$ . Thus, for the LCA symptom endorsement was scored 1 (TSC and HTQ item scores of 3–4), and lack of symptom endorsement was scored 0 (TSC and HTQ item scores of 1–2). PTSD severity was assessed by the HTQ total score.

Pain intensity was measured as one combined total score of the four 11-point Likert scales (Turk & Melzack, 2001). Each scale measured pain intensity on a numerical rating scale ranging from 0 (no pain) to 10 (the worst possible pain). Patients marked their answers on each scale corresponding to their pain now, highest level of pain, lowest level of pain, and finally average pain over the past week. Cronbach's alpha was satisfactory ( $\alpha = .93$ ).

The Pain-Catastrophizing Scale (Sullivan, Bishop, & Pivik, 1995) was used to measure catastrophic thinking related to pain. Patients are asked to reflect on past painful experiences, and to indicate the degree to which they experienced each of 13 thoughts or feelings when experiencing pain, on a five-point

Likert scale (0 = *not at all*, 4 = *all the time*). A summed scale score was calculated from all items, with higher scores indicating high levels of pain catastrophizing. Cronbach's alpha was satisfactory ( $\alpha = .94$ ).

To assess the level of psychological distress (i.e. anxiety and depressive symptoms), we used the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). The HADS was originally constructed to detect psychological distress (i.e. anxiety and depression) in non-psychiatric medical patients. The scale consists of 14 items assessing psychological distress, 7 items assessing depression, and 7 items assessing anxiety. Answers are rated on a four-point Likert-type scale (0 = *no symptoms* to 3 = *maximum impairment*). Cronbach's alpha was satisfactory ( $\alpha = .93$ ).

### **Analytical plan**

The analyses for the present study proceeded in a number of steps. First an LCA was conducted to determine if D-PTSD was identifiable within the present sample. LCA was chosen as it is the appropriate statistical method for determining the correct number of homogeneous groups from multivariate, categorical indicators (i.e. the HTQ and the TSC). Given the diagnostic nature of the constructs under investigation in the present study, LCA can be further argued as a more appropriate analytical technique than latent profile analysis (LPA) for the identification of a possible D-PTSD subtype. These analyses were conducted in Mplus 7.1 (Muthén & Muthén, 2013) using the robust maximum likelihood estimator (Yuan & Bentler, 2000). Inspection of the standardized regression residuals indicated only 3 scores within the data were beyond the critical Mahalanobis distance value for determination of a multivariate outlier. According to Tabachnick and Fidell's (2013) recommendations, the presence of a small number of multivariate outliers is normal and not a matter of concern and thus they were included in the analyses. Missing data were minimal (1.7%) and were estimated using the full information maximum likelihood method. The covariance coverage ranged from .970 to 1.00. Five latent class models were estimated (two- to six-class solutions), and 500 random start values were utilized followed by 50 final stage optimizations to avoid class solutions based on local maxima. Model selection was based on several model comparison indices including the Akaike information criteria (AIC), the Bayesian information criteria (BIC), the sample size adjusted Bayesian Information Criteria (ssaBIC), the Lo-Mendell-Rubin's adjusted likelihood ratio test (LMRA-LRT), and the bootstrapped likelihood ratio test (BSLRT). Lower values on the AIC, BIC, and ssaBIC are indicative of better fit (Nylund, Asparoutiov, & Muthén, 2007a; Nylund, Bellmore, Nishina, & Graham, 2007b; Yang, 2006). Nylund et al. (2007a) and Nylund et al. (2007b) reported that the most reliable indicator of fit is the BIC; thus, we focused our comparisons on this particular indicator. The LMRA-LRT and the BSLRT assess whether a latent model with one additional class is superior to a latent model with one less class. In the case of both tests, a



non-significant value ( $p > .05$ ) indicates that the latent model with one less class is the preferred option (Lo, Mendell, & Rubin, 2001). We also consulted the Entropy (Ramaswamy, Desarbo, Reibstein, & Robinson, 1993) value as an indicator of classification quality within each individual model. Entropy is a standardized measure of how accurately participants are classified to a latent class. Superior classification is indicated by values which approach 1 and value greater than .80 suggest good classification (Celeux & Soromenho, 1996).

Upon selection of the appropriate class solution, descriptive statistics including measures of central tendency, variance, and estimated diagnostic rates were computed. Finally, we conducted a hierarchical regression analysis post hoc to shed more light on the relationship between pain, dissociation (T1), and PTSD severity. Thus, a hierarchical multiple regression analysis using SPSS version 22 was conducted to determine the unique effect of the pain variables (pain levels, and pain catastrophizing) and dissociation to predict PTSD scores, and to determine whether these effects remain after controlling for a range of established risk factors for PTSD (age, sex, time since injury, and psychological distress (i.e. anxiety/depression)).

## Results

### *Descriptive statistics and regression analyses*

The estimated DSM-5 PTSD prevalence rate for the full sample was 9.1%. A total of 5.6% ( $n = 13$ ) of the sample met the dissociation criteria, 3.4% ( $n = 8$ ) endorsed the DIS1 item, and 3.9% ( $n = 9$ ) endorsed the DIS2 item, and 3.0% ( $n = 7$ ) met the D-PTSD diagnostic criteria. [Table 1](#) reports the descriptive statistics and correlations between all measured variables in the study.

### *LCA results*

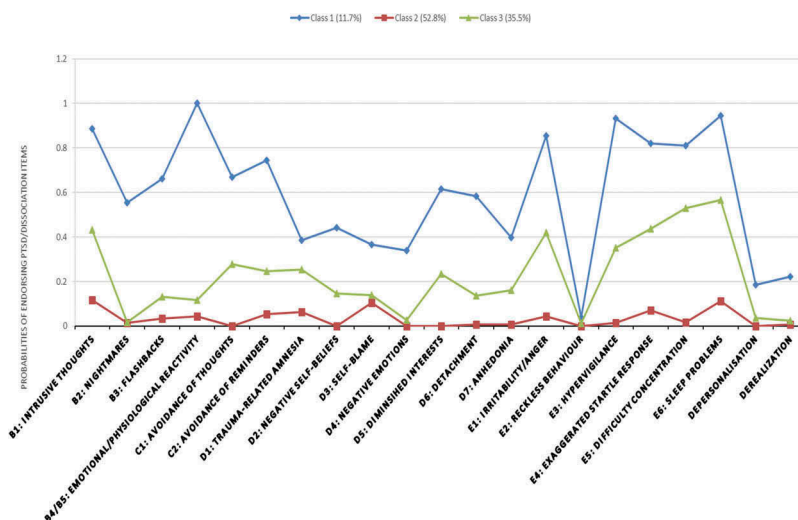
The results of the LCA analyses are presented in [Table 3](#). A three-class solution indicated the best fit. The BIC value, which has been shown to be the best indicator of model fit was lowest for the three-class solution, while the LMRA-LRT test became non-significant for the four-class solution (thus favoring a three-class solution). The entropy value of .89 reflected good classifications of participants.

As displayed in [Figure 1](#), the latent class solution suggested a mainly quantitative distribution of classes across the PTSD and dissociation symptom indicators. No evidence of a D-PTSD construct was found, and all three classes displayed very low probabilities of endorsing the two dissociative symptoms. Class 1 was characterized as the most symptomatic class by generally moderate probabilities of endorsing most of the PTSD symptoms and low probability of endorsing dissociation and was termed the ‘Symptomatic Class’. This class

**Table 3.** Fit statistics for LCA of PTSD and dissociation symptom indicators.

Classes	Loglikelihood	AIC	BIC	ssaBIC	Entropy	LMRA-LRT (p)	BS-LRT (p)
2	-1636	3358	3506	3370	.92	728 (.000)	734 (.000)
<b>3</b>	<b>-1563</b>	<b>3256</b>	<b>3480</b>	<b>3274</b>	<b>.89</b>	<b>144 (.018)</b>	<b>146 (.000)</b>
4	-1512	3198	3498	3223	.90	100 (.240)	101 (.000)
5	-1475	3169	3545	3199	.90	72 (.201)	73 (.000)
6	-	-	-	-	-	-	-

Note. The model with six classes did not estimate. AIC: Akaike information criterion; BIC: Bayesian information criterion; ssaBIC: sample-size adjusted BIC; LMRA-LRT: Lo-Mendell-Rubin adjusted likelihood ratio test; BSLRT: bootstrapped LMRA. Selected class solution in bold.

**Figure 1.** Latent class plot of three PTSD classes.

contained the fewest participants (11.7%). Class 2 was characterized by extremely low probabilities of endorsing all items and was the largest class (52.8%). This class was termed the ‘Non-Symptomatic Class’. Class 3 (35.5%) was characterized by low probabilities of endorsing the PTSD symptoms and dissociation items and were thus termed the ‘Low-Symptomatic Class’. The LCA results offered no evidence of D-PTSD and were instead strongly indicative of a mainly quantitative distribution of PTSD symptom endorsement probabilities (i.e. Symptomatic, Low-Symptomatic, and Non-Symptomatic class).

As the three identified PTSD classes appeared to be mainly quantitatively distributed, it made more sense to look at risk factors for PTSD severity within the present sample. Furthermore, to shed more light on the relationship between dissociation and PTSD, dissociation was added as a risk factor rather than part of the dependent variable. Thus, post hoc hierarchical multiple regression analysis was performed to determine the unique predictive effects of pain severity, and pain catastrophizing (block 1) and dissociation (T1, block 2) to predict PTSD severity at T2, and how these effects changed when controlling for age, sex, time since injury, and psychological

**Table 4.** Hierarchical multiple regression model predicting posttraumatic stress disorder severity (T2).

	<i>R</i>	<i>R</i> <sup>2</sup>	<i>R</i> <sup>2</sup> Change	<i>β</i>	<i>t</i>	<i>B</i>	SE	CI 95% (B)
Block 1	.602	.362***						
Pain severity (T1)				.32***	4.21	0.34	0.08	0.18/0.50
Pain catastrophizing (T1)				.35***	4.70	0.42	0.09	0.24/0.60
Block 2	.701	.491***	.129***					
Pain severity (T1)				.24**	3.50	0.26	0.07	0.11/0.40
Pain catastrophizing (T1)				.19**	2.66	0.23	0.09	0.06/0.39
Dissociation (T1)				.42***	6.90	4.23	0.61	3.02/5.44
Block 3	.765	.585***	.094***					
Pain severity (T1)				.17**	2.73	0.19	0.07	0.05/0.32
Pain catastrophizing (T1)				-.01	-0.13	-0.01	0.09	-0.18/0.16
Dissociation (T1)				.28***	4.52	2.79	0.62	1.57/4.01
Age (T1)				.02	0.44	0.02	0.04	-0.06/0.09
Sex (Males = 0, Female = 1)				-.01	-0.22	-0.23	1.06	-2.32/1.86
Days since injury (T2)				.06	1.31	0.05	0.04	-0.03/0.13
Psychological distress (T1, i.e. Anxiety/Depression)				.45***	6.21	0.55	0.09	0.37/0.72

Note. Statistical significance: \*\*\**p* < .001, \*\**p* < .01, \**p* < .05.

distress (T1, i.e. anxiety/depression) (block 3). Preliminary analysis indicated no serious violations of the assumptions of normality, linearity, and homoscedasticity. Possible violations of multicollinearity were investigated through using the tolerance and Variance Inflation Factor (VIF) statistics and no evidence of multicollinearity was identified. However, additional post hoc analyses showed a high correlation between PTSD severity T1 and PTSD severity T2. The high correlation was expected as PTSD severity T1 and T2 are the same variable and only separated in time. Thus, it was not possible to control for the effect of early PTSD in the regression analyses. The results are shown in Table 4.

## Discussion

The aim of the present study was to examine the possible existence of D-PTSD and to investigate the relationship between pain-related factors, previously identified risk factors for D-PTSD, and D-PTSD if identified within acute whiplash patients. The LCA results indicated a three-class solution for PTSD symptoms (i.e. Symptomatic Class, Low-Symptomatic Class, and Non-Symptomatic Class). Contrary to our expectations we were not able to identify D-PTSD or any PTSD subtypes per se as the three identified PTSD classes were mainly quantitatively distributed. Thus, it made more sense to look at risk factors for PTSD severity to shed more light on the relationship between PTSD, pain, and dissociation within the present sample. Pain severity, psychological distress (i.e. anxiety and depression), and dissociation were identified as significant risk factors for PTSD severity in the final block of the regression analyses.

In contrast to the majority of previously conducted LCA and LPA studies of D-PTSD (Hansen et al., 2017) including the only other study of whiplash

patients and D-PTSD (Hansen, Müllerová et al., 2016), we were not able to identify the D-PTSD. Of note the samples included in the previous whiplash study, and the present study are very different. The Hansen, Müllerová et al. (2016) whiplash study sample included a more chronic sample with a more complex traumatic exposure than the present study. Indeed, very different periods of time had passed since the traumatic exposure in the present sample (3 months) and the previous whiplash study (several years). At the same time, in the sample used in the Hansen, Müllerová et al. (2016) study, 15% of the victims had more than one whiplash trauma and half of the sample had also sustained other physical injuries. In comparison with the present whiplash sample, no one had sustained other physical injuries or previous whiplash traumas. This may indicate that the failure to identify D-PTSD in the present study may be attributed to the specific nature of the traumatic exposure. Only one other study has failed to identify D-PTSD (i.e. a recent study of victims of bank robbery, Hansen, Hyland et al., 2016). As argued by Hansen et al. (2017) in relation to victims of bank robbery, it is possible that the negative results of the present study are related to the more clearly defined nature of the traumatic event and that posttraumatic stress symptoms following acute Motor Vehicle Accident (MVA) present more simple compared to the more complex nature of the traumatic exposure across the other studies. At the same time, the lack of positive results is unlikely to be attributed to the specific measurement of D-PTSD in the present study. Indeed, the HTQ and the TSC have been used in other studies which have identified the dissociative subtype (Armour, Elklit, Lauterbach, & Elhai, 2014; Hansen, Müllerová et al., 2016). Furthermore, the D-PTSD has been identified across several studies with only two-three items assessing dissociation as in the present study (cf. Hansen et al., 2017) and not just the studies assessing dissociation more broadly (cf. Müllerová, Hansen, Contractor, Elhai, & Armour, 2016; Wolf et al., 2015). Thus, Hansen et al. (2017) review concludes that assessing dissociative symptoms more broadly than depersonalization and de-realization do not add to the understanding of D-PTSD. Combined this suggests that the negative results of the present study are not attributed to the measurements, but rather attributed to the nature of the traumatic exposure.

Of note, both the present study and the bank robbery study have relatively low PTSD levels. Thus, it would appear reasonable to assume that the D-PTSD rate would also be low and perhaps even explain the negative results of both studies. However, as underlined by Hansen et al. (2017), the results are not that straightforward and D-PTSD cannot be said to be merely a function of the PTSD prevalence rate. This is further indicated by the fact that Wolf et al. (2015) found that 8.3% of the participants could be assigned to the dissociative PTSD profile in a sample with an estimated PTSD rate of 2%. As pointed out by Hansen et al. (2017), there are several reasons to why dissociative PTSD is not

just a function of PTSD. For instance, PTSD is a dimensional construct and therefore individuals can display rather different symptom patterns. Individuals without a full PTSD diagnosis may display severe symptoms of PTSD and dissociation that may be captured in a LCA as indicative of D-PTSD. Thus, diagnostic rates of dissociative PTSD do not have to correspond to the results of LCA. In a similar vein, dissociative symptoms can also exist out of the context of PTSD. Finally, as also indicated by the results of the present study the Hansen et al. (2017) review suggest that the varying rates of D-PTSD across the studies, may indicate that D-PTSD is not consistent across traumatic exposure. Indeed, D-PTSD may be more pronounced following more complex traumatic exposures than MVA and bank robbery.

The results of the LCA also indicate that only low levels of dissociative symptom endorsement are found in the symptomatic group suggesting that dissociative symptoms may be less central within this population and while the dissociative subtype model cannot be supported. The low estimated rates of dissociation symptom endorsement also indicate that dissociation is not very pronounced in the present sample. However, the results of the bivariate correlations between PTSD severity and dissociative symptoms (T2,  $r = .62$ ,  $p < .001$ ) and the hierarchical regression analyses indicate that a strong relationship between dissociative symptoms and PTSD and a high predictive value of dissociative symptoms (T1) within this specific sample. It therefore appears that dissociation can exist alongside PTSD and that the dissociation is likely to be increased if posttraumatic symptoms are present as indicated by the Component Model, but that dissociation cannot change the phenomenology of PTSD within this particular type of traumatic exposure (Dalenberg & Carlson, 2012). In agreement with Beck and Clapp (2011), the level of dissociative symptoms correlated highly with all the outcomes indicating that dissociation may be an important factor negatively contributing the mutual maintenance of pain and PTSD as hypothesized by Scaer (2001, 2014)) and Sharp and Harvey (2001).

### **Limitations**

The present study has several limitations. Firstly, PTSD symptoms and dissociative symptoms were not assessed using a validated DSM-5 PTSD measurement. Instead, we used a DSM-IV PTSD self-report measurement (the HTQ) and a single item from the TSC to assess DSM-5 PTSD and two items from the TSC to assess symptoms of depersonalization and derealization. Furthermore, as pointed out by Hansen, Müllerová et al. (2016) and Hansen et al. (2015), the B4 and the B5 criterion were assessed with one item, and the E2, D2, D4, and D7 criterion were assessed rather specific (see Table 2 for item specifications). Despite the close resemblance between the TSC and the HTQ and the DSM-5 PTSD criteria and the fact

that the dissociative subtype previously has been identified using these, potential bias connected to using these measurements cannot be ruled out. At the same time, we were not able to assess the functional impairment criteria. Thus, the results of the present study need to be replicated with clinical interviews to see if similar symptom profiles can be uncovered. Secondly, the results were based on Danish acute patients suffering from whiplash and thus it is unknown, whether the results can generalize to acute patients in general. Although, non-responders did not differ in severity of whiplash grade, only 57% of all whiplash injured patients volunteered to participate in the study. It is plausible that those who did not participate were those with most severe symptoms. Thirdly, we were not able to control for childhood physical and sexual assault, which is one of the more consisted supported risk factors for D-PTSD. Although, we were unable to identify D-PTSD within the present study, information on prior childhood physical and sexual assault would have been valuable information to shed more light on the importance of the specific nature of traumatic exposure in connection to D-PTSD and the development of D-PTSD more broadly. As we were unable to identify D-PTSD, we would expect low rates of childhood physical and sexual assault in the present study as they are established risk factors of D-PTSD, however, unfortunately we are unable to test this. Finally, due to multicollinearity we were unable to control for the effect of PTSD (T1) in the regression analysis.

### ***Clinical implications***

Of note, the clinical implications of the results need to be interpreted in light of the limitations. The results of the present study suggest that D-PTSD may not be identified in acute MVA patients suffering from whiplash. At the same time, dissociative symptoms do not appear to be very pronounced within this population. However, this does not necessarily mean that dissociative symptoms should be ignored. In fact, both early and more long-term dissociative symptoms are highly associated with PTSD severity and pain. Furthermore, dissociative symptoms appear to predict PTSD severity. This means that there is still utility in early screening for dissociative symptoms to facilitative early treatment and preventive actions to minimize the risk of developing PTSD in acute victims of MVA suffering from whiplash. Furthermore, the finding that dissociative symptoms are an important predictor of PTSD and highly associated with PTSD indicates that high levels of dissociation may need to be addressed in treatment planning. The fact that dissociation may interfere with habituation during traumatic memory processing (Foa & Kozak, 1986) indicates that exposure therapy may not be sufficient for those with high levels of dissociative symptoms (Cloitre et al., 2012; Resick et al., 2012). However, a

recent study raised doubt about this and suggested that prolonged exposure therapy may work equally well for clients with and without the dissociative subtype (Wolf, Lunney, & Schnurr, 2016). Thus, future research is needed to shed more light on these matters.

## Conclusion

The present study is the first study of acute patients of MVA suffering from whiplash. We found support for three-class solution for PTSD but no dissociative subtype. Instead, the classes appeared to be mainly quantitatively distributed according to endorsement probabilities (i.e. Symptomatic, Low-Symptomatic, and Non-Symptomatic class). Although there are several possible explanations as to why we were not able to find evidence of D-PTSD in the present study, the most likely explanation is attributed to the specific less complex nature of the MVA in the present study (i.e. no other physical injuries or prior whiplash traumas) compared to existing studies, which have identified D-PTSD. Although, the estimated prevalence rates of dissociation and PTSD were not high, the results did indicate that dissociation and PTSD are co-occurring phenomenon and dissociation is likely to be increased if PTSD symptoms are present and vice versa as indicated by the Component model. Thus, the results suggest that the Component Model of dissociation and PTSD rather the subtype model better explains the relationship between dissociation and PTSD in the present sample. Future studies are needed to shed more light on both dissociation and PTSD and their mutual relationship in different trauma populations and how this affects treatment.

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