Psychiatry Research 210 (2013) 274–280

Contents lists available at ScienceDirect

Psychiatry Research

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

Predictors of trait dissociation and peritraumatic dissociation induced via cold pressor

Lydia Gómez-Pérez [a,*], Alicia Eva López-Martínez [b], Gordon John Glenn Asmundson [a]

[a] Anxiety Illness and Behaviours Laboratory & Department of Psychology, University of Regina, 3737 Wascana Parkway, Regina, SK, Canada S4S 0A2
[b] Department of Personality, Assessment and Psychological Treatment, University of Málaga, Campus de Teatinos s/n, 29071 Málaga, Spain

ARTICLE INFO

Article history:
Received 5 July 2012
Received in revised form
1 June 2013
Accepted 3 June 2013

Keywords:
Peritraumatic dissociation
Trait dissociation
Dissociative Experience Scale
Anxiety sensitivity
Cold pressor
Stress
Catastrophizing

ABSTRACT

Understanding which factors predict individual dissociative response during stressful situations is important to clarify the nature of dissociation and the mechanisms associated to its use as a coping strategy. The present study examined (1) whether experiential avoidance (EA), anxiety sensitivity (AS), depressive symptoms, and state anxiety concurrently predicted trait dissociation (TD)—absorption, amnesia, depersonalization, and total TD scores—and laboratory induced dissociation (LID); and (2) whether TD and catastrophizing predicted LID. We also examined whether catastrophizing mediated the relationships between both AS and depressive symptoms and LID. A total of 101 female undergraduate students participated in a cold pressor task, which significantly induced dissociation. Results of hierarchical regression analyses showed that AS at Time 1 (9 months before the experimental session), as well as depressive symptoms and catastrophizing at the time of the experiment (Time 2), predicted LID at Time 2. Depressive symptoms at Time 2 predicted total TD, absorption, and amnesia scores. AS at Time 1 and depressive symptoms at Time 2 predicted depersonalization. AS, depressive symptoms, and catastrophizing seem to facilitate the use of dissociative strategies by healthy individuals, even in response to non-traumatic but discomforting stress.

1. Introduction

Dissociation is defined as a disruption in the integrated functions of consciousness, memory, identity, or perception of the environment (American Psychiatric Association, 2000). It has been traditionally viewed as a protective mechanism against emotional distress and physical pain during traumatic (Spiegel, 1991) and non-traumatic forms of stress (Sterlini and Bryant, 2002). Some authors posit dissociation as a risk factor for psychopathology (Briere et al., 2005), and positive relationships between dissociation and other psychopathology predictors have been reported (e.g. Kellner et al., 2002). Nevertheless, the role of dissociation remains unclear, and its potential adaptive value may depend on its level of presentation and its interactions with other constructs (Gómez-Pérez et al., 2011). Elucidating which factors promote dissociation during stressful situations is important to clarify its nature and the mechanisms associated to its use as a coping strategy.

Although trait dissociation (TD) has been hypothesized to strongly predict peritraumatic dissociation (PTD), evidence in favour of and against this assumption has been reported. For example, Leonard et al. (1999) found that individuals scoring high on TD, compared to those scoring low, reported more laboratory induced dissociation (LID) during different experimental procedures (i.e., dot-staring, pulsed photic administration, audio stimulation). Nevertheless, in a study in which dissociation was induced in undergraduate students via a cold pressor task, only the absorption subscale but not the total scores of the Dissociation Experience Scale (DES; Bernstein-Carlson and Putnam, 1993) was positively correlated with LID (Giesbrecht et al., 2008). Finally, Sterlini and Bryant (2002) failed to find an association between TD and dissociation elicited during skydiving.

One factor that may predict dissociation in response to stress is anxiety sensitivity (AS; i.e., the fear of anxiety-related bodily sensations derived from beliefs that these symptoms have harmful physical, psychological, or social consequences; Reiss and McNally, 1985). In the context of trauma, both AS (particularly the AS taxon; Bernstein et al., 2005) and dissociation (Briere et al., 2005) have been shown to predict posttraumatic stress symptoms. It has been argued that AS may contribute to dissociation because individuals may make attributions about their somatic experiences that lead to altered perceptions of awareness (Nixon and Bryant, 2006). In fact, AS (particularly the AS taxon) has been shown to be associated with body vigilance (Zvolensky et al., 2007) and with reports of freezing in the context of a biological challenge (i.e., 20-s

* Corresponding author. Tel.: +1 306 337 2473; fax: +1 306 337 3275.
E-mail addresses: lydia.gomez.perez@uregina.ca, lydiagp2@yahoo.es (L. Gómez-Pérez).

0165-1781/$ - see front matter © 2013 Elsevier Ireland Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.psychres.2013.06.001

inhalation of 20% CO_2/balance O_2; Schmidt et al., 2008), the latter of which may represent an extreme behavioural expression of dissociation (Abrams et al., 2009). Kellner et al. (2002) found higher AS scores in men with high TD than in those with low TD scores. Likewise, a positive relationship between AS and LID was reported in a study in which dissociation was elicited by having participants read a text about the 9/11 terrorist attacks (Kosloff et al., 2006). AS is considered a predisposing factor for several forms of catastrophizing (e.g., pain catastrophizing; Asmundson et al., 1999), and catastrophizing has been hypothesized to contribute to dissociation (Ehlers and Steil, 1995). Catastrophizing may be, therefore, one pathway linking AS and dissociation experienced in response to stress.

Experiential avoidance (EA)—the "unwillingness to remain in contact with aversive private experiences as well as the actions taken to alter the form or frequency of those events and the contexts that occasion them" (Hayes et al., 1996; p. 4)—may be another predictor of dissociation. In fact, dissociation has been proposed as a form of EA (e.g., Hayes et al., 1996). Nonetheless, few authors have examined the association between these two constructs. Marx and Sloan (2005) found a positive relationship between EA and PTD in trauma-exposed individuals (TEI). In addition, EA assessed before a university campus shooting longitudinally predicted PTD among the victims (Kumpula et al., 2011). The relationships between EA and TD and LID have, to the best of our knowledge, never been examined.

Positive correlations between depressive symptoms and TD have been reported (Sterlini and Bryant, 2002; Lipsanen et al., 2004; Sayar and Kose, 2003; Pastucha et al., 2009; Prasko et al., 2010); however, no studies regarding the relationship between depressive symptoms and dissociation experienced during stressful situations have been conducted. Catastrophizing—central to depression models (Clark et al., 1999)—may be linking depressive symptoms and this type of dissociation. Anxiety symptoms have also been associated with TD (Sayar and Kose, 2003; Pastucha et al., 2009; Prasko et al., 2010) but, as in the case of depressive symptoms, its relationship with dissociation experienced in response to stress has never been examined.

The present study had two objectives. First, to examine (1) whether AS Taxon, EA, depressive symptoms, and state anxiety concurrently predicted TD and LID (induced via a cold pressor task, Giesbrecht et al., 2008), and (2) whether TD and pain catastrophizing predicted LID. We specifically assessed pain catastrophizing, because the cold pressor task induces pain. Second, we determined whether pain catastrophizing mediated the relationships (a) between AS Taxon and LID and (b) between depressive symptoms and LID. We predicted positive significant relationships between the abovementioned variables, and significant indirect effects of AS Taxon and depressive symptoms on LID through pain catastrophizing.

2. Method

2.1. Participants

Data were from a sample of female undergraduate students from the University of Málaga (Spain) in which TEI were overrepresented. These data were collected as part of a larger study (all the participants participating in the present research were considered for Gómez-Pérez et al. (2012); and 80 of them participated in Gómez-Pérez and López-Martínez (2013)). Data from individuals with clinical levels of depressive (scores ≥26 in the Center for Epidemiologic Studies Depression Scale [CESD]; Radloff, 1977; Vázquez et al., 2007) or posttraumatic stress (scores ≥40 in the Davidson Trauma Scale [DTS]; Davidson et al., 2002) symptoms were not considered for the present investigation, as previous literature suggests that the role of dissociation and its relationship with the variables examined is different in clinical than in non-clinical samples (Gómez-Pérez et al., 2011). The final sample comprised 101 participants (*Mean* age [S.D.]=20.53 [3.15]). Based on the Stressful Life Event Screening Questionnaire Revised scores (Green et al., 2006), 63 of them reported trauma exposure (TEI) and 38 did not (non-TEI).

2.2. Apparatus and instruments

2.2.1. Questionnaires

Recent findings indicate that the latent structure of AS is taxonic (Zvolensky et al., 2007); therefore, AS was assessed with the *Anxiety Sensitivity Index Taxon Scale* (ASI Taxon Scale, Bernstein et al., 2005), which presents significant incremental validity above and beyond the traditional ASI scale from which it is derived (Bernstein et al., 2005). Its Cronbach's alpha was 0.75. EA was assessed with the *Acceptance and Action Questionnaire* (AAQ; Hayes et al., 2004). The Spanish version of this questionnaire shows an internal consistency of 0.74 (Cronbach's alpha), test–retest reliability coefficient of 0.71, and concurrent validity (Barraca, 2004). Unfortunately, the reliability of this questionnaire was very low for the sample of this study (Cronbach's alpha=0.48), and consequently we decided not to include this measure in any of the statistical analyses conducted. The frequency of depressive symptoms during the past week was assessed with the CES-D (Radloff, 1977), for which Cronbach's alpha values were 0.90 and 0.79 for Time 1 and Time 2, respectively. State anxiety during the past week was assessed with the state subscale of the *State Trait Anxiety Inventory* (STAI; Spielberger et al., 1970), for which Cronbach's alpha was 0.90. Catastrophizing experienced during the cold pressor task was assessed with the *Pain Catastrophizing Scale* (PCS; Sullivan et al., 1995) adapted to the specific experimental situation (Cronbach's alpha=0.86), which comprises 8 items on a 5-point rating scale ranging from 0 (nothing) to 4 (a lot). TD was assessed with the *Dissociative Experiences Scale* (DES; Bernstein-Carlson and Putnam, 1993), which provides a total TD score, three TD subscales scores (i.e., absorption, amnesia, depersonalization), and a TD-Taxon score, which is an index of pathological TD (Waller et al., 1996). Cronbach's alpha values were 0.90, 0.82, 0.74, 0.75, and 0.73 for the total, absorption, amnesia, depersonalization, and DES-Taxon scales, respectively. The *Peritraumatic Dissociative Experiences Questionnaire Modified* (PDEQ; Marshall et al., 2002) was used to quantify LID. Cronbach's alpha for this questionnaire was 0.75 at Time 1 and 0.87 at Time 2.

2.2.2. Cold pressor task

We employed a cold pressor task in order to induce dissociation (Giesbrecht et al., 2008). Participants were instructed to keep their arm in cold water (1.5–3 °C) for as long as possible. They were told they could remove it at any time if they could no longer tolerate the pain. They were asked to remove their arm from the water at 3 minutes, but were not informed of this upper time-limit.

2.3. Procedure

As part of a screening for several research projects, participants completed a battery of questionnaires—including the ASI-Taxon, AAQ, and the CES-D—prior to or after scheduled undergraduate Psychology classes (Time 1). Nine months later, they were contacted by phone and invited to participate in an experimental session (Time 2), during which they completed the CES-D, the STAI, the DES, and the PCS, and completed the cold-pressor task. The PDEQ was administered before and after the cold pressor. LID was determined by subtracting baseline state dissociation scores from state dissociation scores assessed after the cold pressor task. Informed consent was obtained, and the study was approved by the University of Malaga's Research Ethics Board.

2.4. Data analyses

We conducted a Wilcoxon Signed Rank Test to examine if the cold pressor significantly increased dissociation using the total sample. To test if the increase of dissociation was significant in both TEI and non-TEI, this analysis was repeated separately for each group. We performed a Mann–Whitney *U* test to examine whether TEI and non-TEI differed in any of the variables, because groups differed in size and some variables were non-normally distributed. Then, we conducted zero-order correlations and a set of hierarchical regression analyses considering total TD scores—as well as absorption, amnesia, depersonalization, and TD-Taxon scores—and LID as dependent variables. Those variables significantly correlated with the dependent variables were considered as predictors. As previously mentioned, EA was not included in any of these analyses due to the low reliability of the AAQ in our sample. Finally, we performed bootstrap analyses (Zhao et al., 2010), using 5000 bootstrap samples to calculate 95% bias-corrected confidence intervals, to examine if catastrophizing mediated the relationship between (a) AS and LID and (b) depressive symptoms and LID.

3. Results

3.1. Preliminary analyses

Descriptive statistics of the raw data of the variables examined are presented in Table 1. The Wilcoxon Signed Rank Test conducted

Table 1
Descriptive statistics (medians, means, standard deviations).

	Non-TEI ($n=38$)			TEI ($n=63$)			Total sample ($N=101$)		
	Md	*M*	S.D.	*Md*	*M*	S.D.	*Md*	*M*	S.D.
LID	1.5	4.42	6	1	1.84	3.73	1	2.81	4.85
AS (T1)	13	14.10	4.90	13	13.49	4.41		13.72	4.59
TD (T2)	41	47.16	27.40	42	49.67	29.97	41	48.72	28.92
Depressive symptoms T1	17	13.76	6.78	16	18.65	9.91	17	12.97	6.49
Depressive symptoms T2	12.5	12.49	6.32	13	17.70	10.62	13	18.06	10.32
State anxiety (T2)	21	22.74	9.95	21	21.54	9.01	21	21.99	9.35
Catastrophizing (T2)	12.5	38.61	9.99	10	37.71	10.63	11	11.71	6.30

Note: TEI=Trauma-exposed individuals; AS=Anxiety sensitivity; LID=Laboratory induced dissociation; TD=Trait dissociation.

Table 2
Zero-order correlation analyses.

Variables	1	2	3	4	5	6	7	8	9	10
1. LID	–									
2. DES_{T2}	0.207*	–								
3. DES-AB	0.232*	0.935***	–							
4. DES-AM	0.113	0.820***	0.685***	–						
5. DES-D	0.231*	0.659***	0.586***	0.446***	–					
6. DES-T	0.173	0.792***	0.701***	0.704***	0.784***	–				
7. ASI-T_{T1}	0.24*	0.211*	0.229*	0.104	0.309**	0.195	–			
8.$CESD_{T1}$	0.188	0.190	0.130.	0.125	0.335**	0.340**	0.411***	–		
9. CES-D_{T2}	0.242*	0.444***	0.405***	0.341***	0.387***	0.441***	0.140	0.473***	–	
10. STAI-S_{T2}	0.146	0.344***	0.301**	0.250*	0.241*	0.356***	0.068	0.351***	0.688***	–
11. PCS	0.436***	0.144	0.187	0.155	0.145	0.072	0.044.	−0.034	−0.045	−0.103

Note: two- tails. LID=Laboratory induced dissociation; DES=Dissociation experience Questionnaire; DES-AB=Absorption; DES-AM=Amnesia; DES-D=Depersonalization; DES-T=DES Taxon scale; ASI-T=Anxiety Sensitivity Index Taxon; STAI-S=State subscale of the State and trait Anxiety questionnaire; PCS=Pain Catastrophizing Scale.

*** $P<0.001$.

** $P<0.01$.

* $P<0.05$.

with the entire sample revealed a statistically significant increase in LID following cold-pressor exposure, $Z=-5.82$, $p<0.001$. The effect size was moderately large ($r=0.41$). Median LID scores increased from pre- ($Md=8$) to post-cold pressor ($Md=10$). Similar results were found when these analyses were conducted separately in TEI ($Z=-3.91$, $p<0.001$, $r=0.35$) and non-TEI ($Z=-4.34$, $p<0.001$, $r=0.50$). TEI and non-TEI did not differ significantly on any of the variables (although TEI tended to present lower LID than non-TEI, $p=0.06$); therefore, subsequent analyses where conducted on the entire sample ($N=101$). Transformations were conducted to reduce the influence of univariate outliers and improve distributions; however, for clarity in the description of the results, the original names of the variables are retained. Results of the correlational analyses are presented in Table 2.

3.2. Hierarchical regression analyses predicting TD

To examine which variables predicted total TD scores, we conducted a regression analysis in which AS at Time 1 was first introduced (step 1), followed by depressive symptoms (step 2) and state anxiety (step 3) at Time 2. Step 1 significantly accounted for 3.5% of total TD. The inclusion of depressive symptoms in the model (step 2) significantly increased the proportion of variance explained by the model, which now accounted for 20.4% of total TD. Only depressive symptoms significantly contributed to this model, accounting for 42.3% of variance. When state anxiety was included (step 3) the proportion of variance explained by the model did not significantly increase. See Table 3.

To examine which factors predicted absorption, we conducted a regression analysis in which AS at Time 1was first introduced (step 1) followed by depressive symptoms (step 2) and state anxiety (step 3) at Time 2. The first model was statistically significant and accounted for 4.3% of the variance. The second model—including AS and depressive symptoms—was also statistically significant and explained 17.8% of variance. Only depressive symptoms at Time 2 significantly contributed to this model, accounting for 38% of its variance. AS approached but did not reach statistical significance within the model ($\beta=0.18$; $p=0.57$). When state anxiety was introduced (step 3) the proportion of variance explained by the model did not significantly increase.

To examine which variables predicted amnesia, we conducted a regression analysis including depressive symptoms (step 1) and state anxiety (step 2) at Time 2 as predictors. The first model accounted for 10.8% of variance, with depressive symptoms significantly explaining 34.1% of the variance of the model. The inclusion of state anxiety (step 2) did not significantly increase the proportion of variance explained.

To predict depersonalization, we first entered AS at Time 1 (step 1), depressive symptoms at Time 1 (step 2), and depressive symptoms (step 3) and state anxiety (step 4) at Time 2 in another regression model. The first step accounted for 8.6% of variance, with AS at Time 1 significantly contributing to the model. The second model (in which depressive symptoms at Time 1 were added) was also significant, explaining 13% of variance. Both AS and depressive symptoms at Time 2 were statistically significant. The third model (in which depressive symptoms at Time 2 were added) was also statistically significant, explaining 19.8% of variance. Both AS at Time 1 and depressive symptoms at Time 2 significantly contributed to the model, accounting for 22.7% and 31.1% of its variance, respectively. Although the fourth model was also significant, the inclusion of state anxiety did not significantly increase the proportion of variance explained by the model. See Table 3.

Table 3
Hierarchical regression analysis predicting total trait dissociation (TD), absorption, amnesia, depersonalization, and TD-Taxon scores.

		β	*Square semipartial correlation*	*Adjusted* R^2	ΔR^2	*F*	*gl*
VD: Total TD scores							
Model 1	AS (T1)	0.21*	0.045	0.035	0.044*	4.60*	1, 99
Model 2	AS (T1)	0.151	0.023	0.204	0.175***	13.78***	1, 98
	DS (T2)	0.423***	0.175				
Model 3	AS (T1)	0.154	0.023	0.199	0.004	9.28***	3, 97
	DS (T2)	0.366***	0.069				
	State anxiety (T2)	0.082*	0.003				
VD: Absorption							
Model 1	AS (T1)	0.23*	0.052	0.043	0.053*	5.50*	1, 99
Model 2	AS (T1)	0.18	0.030	0.18	0.142***	11.82***	2, 98
	DS (T2)	0.38***	0.141				
Model 3	AS (T1)	0.18	0.031	0.164	0.001	7.87***	3, 97
	DS (T2)	0.35**	0.062				
	State anxiety (T2)	0.05	0.001				
VD: Amnesia							
Model 1	DS (T2)	0.34***	0.116	0.108	0.116***	13.05***	2, 99
Model 2	DS (T2)	0.32*	0.054	0.099	0.000	6.49**	1, 98
	State anxiety (T2)	0.29	0.000				
VD: Depersonalization							
Model 1	AS (T1)	0.31**	0.095	0.086	0.096**	10.47**	1, 99
Model 2	AS (T1)	0.21*	0.035	0.130	0.052*	8.49***	2, 98
	DS (T1)	0.25*	0.052				
Model 3	AS (T1)	0.23*	0.042	0.198	0.075**	9.24***	3, 97
	DS (T1)	0.10	0.006				
	DS (T2)	0.31**	0.075				
Model 4	AS (T1)	0.23*	0.042	0.191	0.001	6.89 ***	4, 96
	DS (T1)	0.10	0.006				
	DS (T2)	0.34*	0.053				
	State anxiety (T2)	-0.04	0.001				
VD: TD-Taxon							
Model 1	DS (T1)	0.34**	0.116	0.107	0.116***	12.93**	1 99
Model 2	DS (T1)	0.17	0.022	0.201	0.101***	13.58***	2, 98
	DS (T2)	0.36**	0.101				
Model 3	DS (T1)	0.17	0.021	0.197	0.004	9.19***	3, 97
	DS (T2)	0.30*	0.042				
	State anxiety (T2)	0.91	0.004				

Note: DP=Depressive symptoms.
*** $p < 0.001$.
** $p < 0.01$.
* $p < 0.05$.

To examine which variables predicted TD-Taxon, we conducted a regression analysis in which depressive symptoms at Time 1 were included (step 1). This model significantly accounted for 10.7% of variance. Depressive symptoms at Time 2 were then added to the model (step 2), which now accounted for 20.1% of variance. Depressive symptoms at Time 2 were the only variable significantly contributing to the model, explaining 36.2% of its variance. State anxiety was finally included (step 4), but its inclusion did not significantly improve the model. See Table 3.

3.3. Hierarchical regression analyses predicting LID

We introduced AS at Time 1 (step 1), TD at Time 2 (step 2), depressive symptoms at Time 2 (step 3), and pain catastrophizing (step 4) in a regression model predicting LID. Step 1 was statistically significant, with AS at Time 1 explaining 5% of variance. Although Steps 2 and 3 were also statistically significant, neither the inclusion of TD nor the inclusion of depressive symptoms at Time 2 significantly increased the variance explained by the model. However, when pain catastrophizing was introduced (step 4), the variance explained significantly increased to 26.6% with AS significantly accounting for 19.2%, depressive symptoms for 23.6%, and pain catastrophizing for 43.9% of variance. A similar regression analysis was conducted in which TD was replaced by absorption and depersonalization as predictors. None of these significantly predicted LID. See Table 4.

3.4. Bootstrap analyses

Neither the indirect effect (IE) of AS (*IE*=0.003, *SE*=0.006, *BCa 95% CI* [−0.009, 0.017]) nor the IE of depressive symptoms (*IE*=−0.002, *SE*=0.005, *BCa 95% CI* [−0.012 to 0.007]) on PTD through pain catastrophizing was significant. Therefore, pain catastrophizing did not mediate the relationship observed between AS and PTD nor the relationship found between depressive symptoms and LID. In light of the relationship found between AS at Time 1 and depressive symptoms at Time 2 with TD and because AS was shown to predispose to psychopathology (Cox et al., 2001; Schmidt and Zvolensky, 2007), we decided to examine if depressive symptoms at Time 2 were mediating the relationship found between AS and TD. The results showed that the IE of AS on TD through depressive symptoms (*IE*=0.37, *SE*=0.25, *BCa 95% CI* [−0.074, 0.092]) was not statistically significant, indicating absence of mediation.

Table 4
Hierarchical regression analyses predicting laboratory induced dissociation (LID).

		β	*Square semipartial correlation*	*Adjusted* R^2	ΔR^2	*F*	*gl*
VD: LID							
Model 1	AS (T1)	0.24*	0.060	0.050	0.60*	6.28*	1, 99
Model 2	AS (T1)	0.21*	0.042	0.066	0.25	6.54*	2, 98
	TD (T2)	0.16	0.025				
Model 3	AS (T1)	0.20*	0.039	0.082	0.25	3.98*	3, 97
	TD (T2)	0.09	0.006				
	Depressive symptoms (T2)	0.18	0.025				
Model 4	AS (T1)	0.19*	0.035	0.266	0.185***	10.04***	4, 96
	TD (T2)	−0.00	0.000				
	Depressive symptoms (T2)	0.24*	0.044				
	Catastrophizing (T2)	0.45***	0.186				

Note:
*** $p < 0.001$.
* $p < 0.05$.

4. Discussion

The main aim of the present study was to concurrently examine the role of several psychological variables in the prediction of TD and LID. We hypothesized AS, depressive symptoms, and state anxiety would predict both TD and LID. In addition, we expected to find that TD and pain catastrophizing predict LID. Our hypotheses were partially confirmed.

AS uniquely predicted 3.5% of the total variance of LID 9 months later. AS also significantly predicted 4.5% of the total variance of TD 9 months later; however, when depressive symptoms at Time 2 were considered, its contribution to the model was no longer statistically significant, and the unique total variance explained by AS was reduced to 2.3%. This loss of significance might reflect a mediational effect of depressive symptoms in the relationship between AS and TD; however, our results did not support this mediational effect. Nonetheless, AS at Time 1 uniquely predicted 4.2% of the total variance of depersonalization, after removing the variance explained by depression at Time 2, which uniquely accounted for 7.5% of the total variance of depersonalization. Both variables were statistically significant in a model explaining 19.8% of depersonalization at Time 2. In addition, AS significantly predicted absorption. Nonetheless, when taking into account depressive symptoms at T2, AS approached but did not reach statistical significance within the model ($\beta = 0.18$; $p = 0.57$). It uniquely predicted 3% of the variance in absorption. The relationships found between AS and both TD and LID are in line with the results of studies reporting positive correlations between AS and dissociation (Kellner et al., 2002; Kosloff et al., 2006). Individuals with higher AS may perceive cold pressor induced dissociation symptoms (i.e. dizziness) in a more threatening way, increasing the distress and LID provoked by this task. Alternatively, the heightened attentional focus on bodily sensations of individuals with higher AS may facilitate the capacity to dissociate during stressful situations. In fact, AS longitudinally predicted the tendency to depersonalizate and, together with the absorption subscale, this trait dissociation dimension was the only one that correlated with LID.

Depressive symptoms assessed at Time 2 uniquely accounted for 4.4% of the total variance of LID, 17.5% of TD at Time 2, 14.1% of absorption, 11.6% of amnesia, 7.5% of depersonalization, and 11.1% of TD-Taxon. These results are in line with previously reported correlations between depressive symptoms and dissociation (Sterlini and Bryant, 2002; Lipsanen et al., 2004; Sayar and Kose, 2003; Prasko et al., 2010). Depressive symptoms at Time 1 did not significantly predict LID or TD at Time 2. This is not surprising, as our sample comprised healthy participants, whose levels of depression were not likely stable over time. Depressive symptoms may facilitate the use of dissociative strategies in healthy individuals in response even to non-traumatic situations in which the levels of stress are relatively low. Attentional biases may underlie this relationship. The reported detrimental effect of dissociation in individuals exposed to extreme stress (Briere et al., 2005) might be due to its relationship with AS and depressive symptoms and not necessarily to the use of dissociation itself as a coping strategy. These issues warrant future research.

Pain catastrophizing was the strongest predictor of LID, explaining 18.6% of its total variance, which suggests the way individuals appraise stressful situations may determine their dissociative response, probably via the influence of catastrophic thinking over distress. Nevertheless, contrary to our hypothesis, pain catastrophizing did not mediate the relationships found between AS or depressive symptoms and LID. Studies examining the relationship between catastrophizing and LID in which catastrophizing is experimentally manipulated and some measure of stress is included (i.e., stress perceived during the procedure) are needed. Examining the mediational role of perceived stress in the relationship between catastrophizing and LID will help to clarify the functioning of human coping stress mechanisms. Catastrophizing may serve as a general cognitive vulnerability which increases the probability that people perceive situations as stressful and develop stress-related disorders. Treatment targeting catastrophizing tendencies in populations exposed to stressful contexts (e.g., military population, health care providers) may diminish the level of stress perceived by these individuals and its detrimental consequences. Regarding state anxiety, it significantly correlated with all the TD scales (Sayar and Kose, 2003); however, it did not predict any of them when considered together with other predictors.

In line with Leonard et al. (1999), we found modest and positive correlations between LID and TD; however, none of the trait dissociation scales significantly predicted LID when they were concurrently considered with other predictors. The DES may not be assessing the tendency to dissociate in response to stressful situations (Sterlini and Bryant, 2002), at least not in response to non-traumatic stress. It has been argued that this questionnaire assesses psychopathology, at least in part (Merckelbach and Giesbrecht, 2006). Future studies using other dissociation scales (e.g. the ARAS; Carleton et al., 2010) are needed. TD seems to be a complex phenomenon. A differential pattern of correlations was found between the different DES subscales. The distinction between the different dimensions of dissociation must be considered in future investigations.

There is a well-documented relationship between EA and psychopathology (Hayes et al., 2004). Furthermore dissociation has been proposed as a form of EA (e.g., Hayes et al., 1996). In line

with this, one of the aims of the study was to investigate the relationship between EA and dissociation. Unfortunately, we were not able to test such relationship, as the AAQ presented a very low reliability in our sample which prevented us from including this variable in the analyses. Others authors have also criticised the low reliability of the AAQ as well as the difficulty to tease apart EA from the outcomes to which it is theoretically related (i.e., psychological distress; Chawla and Ostafin, 2007) when it is assessed using the AAQ. Future studies—in which other questionnaires to assess EA are used—should be conducted in order to test the relationship between EA and trait dissociation, which has not yet been empirically examined.

Furthermore, the present study has some other limitations. First, only women participated, and studies including men are necessary. Second, as the relationship between dissociation and other psychological variables and its adaptive value may not be the same in clinical than in non-clinical samples (Gómez-Pérez et al., 2011), we only include participants without clinical levels of depression of posttraumatic symptoms. Consequently, the present results can only be generalized to non-clinical samples, and should be replicated in clinical ones. The relationships we found could be more pronounced in individuals presenting higher and more pathological levels of dissociation, as higher levels of all the variables considered in this study would be expected in these individuals. It may also be that clinical and non-clinical individuals employed different subtypes of dissociation when faced with stress. Whereas healthy individuals may use more frequently strategies traditionally considered positive (e.g., absorption; Nijenhuis et al., 1996; Bowins, 2012), clinical populations may use pathological forms of dissociation (i.e., amnesia) more frequently. This hypothesis deserves attention in future investigations. Although we found that anxiety and depressive symptoms were related to positive (i.e., absorption) and negative (i.e., amnesia) types of trait dissociation, suggesting an association between dissociation and psychopathology, the debate about the protective/pervasive role of dissociation remains open. Studies examining the positive and negative consequences of each type of dissociation should be performed. Third, certain variables previously related to dissociation (e.g., alexithymia, somatisation; Lipsanen et al., 2004) were not considered and should be included in future studies. Fourth, due to the exploratory character of this study, we did not correct alpha levels for any of the statistical analyses (Bender and Lange, 2001); our findings should be replicated in confirmatory investigations. Finally, the PCS and the PDEQ were completed by the participants immediately after the cold pressor task, which could have artificially increased the correlation between both scales. Despite these limitations, this is the first longitudinal study in which the influence of AS and depressive symptoms on LID and DT is examined. These variables warrant further consideration as predictors of dissociation.

Acknowledgements

Part of the research was conducted when the first author was supported by a predoctoral fellowship from the Spanish Ministry of Education and Innovation (FPU, AP2005-1629. 2007–2010 National Scientific Research, Technologic Development, and Innovation). This work has been also supported by the Spanish Ministry of Science and Innovation (PSI2008-01803/PSIC), the Andalusian County Council (Proyectos Excelencia Junta de Andalucía, P07-SEJ-3067) and the Canadian Institutes of Health Research (MOP-PSD-178753-49284). The authors wish to thank anonymous reviewers whose comments and suggestions aided in the preparation of this article.

References

Abrams, M.P., Carleton, R.N., Taylor, S., Asmundson, G.J.G., 2009. Human tonic immobility: Measurement and correlates. Depression and Anxiety 26, 550–556.
American Psychiatric Association, 2000. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. Author, Washington DC.
Asmundson, G.J.G., Norton, P.J., Norton, G.R., 1999. Beyond pain: the role of fear and avoidance in chronicity. Clinical Psychology Review 19, 97–119.
Barraca, J., 2004. Spanish adaptation of the Acceptance and Action Questinnaire (AAW). International Journal of Psychology and Psychological Therapy 4, 505–515.
Bender, R., Lange, S., 2001. Adjusting for multiple testing—when and how? Journal of Clinical Epidemiology 54, 343–349.
Bernstein-Carlson, E., Putnam, F.W., 1993. An update on the Dissociative Experiences Scale. Dissociation 6, 16–27.
Bernstein, A., Zvolensky, M.J., Feldner, M.T., Lewis, S.F., Fauber, A.L., Leen-Feldner, E.W., Vujanovic, A.A., 2005. Anxiety sensitivity Taxon and trauma: discriminant associations for posttraumatic stress and panic symptomathology among young adults. Depression and Anxiety 22, 138–149.
Bowins, B.E., 2012. Therapeutic dissociation: compartmentalization and absorption. Counselling Psychology Quarterly 25, 307–317.
Briere, J., Scott, C., Weathers, F., 2005. Peritraumatic and persistent dissociation in the resumed etiology of PTSD. American Journal of Psychiatry 162, 2295–2301.
Carleton, R.N., Abrams, M.P., Asmundson, G.J.G., 2010. The Attentional Resource Allocation Scale (ARAS): psychometric properties of a composite measure for dissociation and absorption. Depression and Anxiety 27, 775–786.
Chawla, N., Ostafin, B., 2007. Experiential avoidance as a functional dimensional approach to psychopathology: an empirical review. Journal of Clinical Psychology 63, 871–890.
Clark, D.M., Beck, A.T., Alford, B.A., 1999. Scientific Foundations of Cognitive Theory and Therapy of Depression. Wiley, New York.
Cox, B.J., Enns, M.W., Taylor, T., 2001. The effect of rumination as a mediator of elevated anxiety sensitivity in major depression. Cognitive Therapy and Research 25, 525–534.
Davidson, J.R.T., Tharwani, H.M., Connor, K.M., 2002. Davidson Trauma Scale (DTS): normative scores in the general population and effect sizes in placebo-controlled SSRI trials. Depression and Anxiety 15, 75–78.
Ehlers, A., Steil, R., 1995. Maintenance of intrusive memories in posttraumatic stress disorder: a cognitive approach. Behavioural and Cognitive Psychotherapy 23, 217–249.
Giesbrecht, T., Smeets, T., Merckelbach, H., 2008. Dissociative experiences on ice—peritraumatic and trait dissociation during the cold pressor test. Psychiatry Research 157, 115–121.
Gómez-Pérez, L., Abrams, M.P., López-Martínez, A.E., Asmundson, G.J.G., 2012. Trauma exposure and health: the role of depressive and hyperarousal symptoms. Journal of Traumatic Stress 25, 641–648.
Gómez-Pérez, L., López-Martínez, A.E., 2013. Trauma, posttraumatic stress disorder, and pain response in healthy young women. Clinical Journal of Pain 29, 425–434.
Gómez-Pérez, L., Thibodeau, M.A., López-Martínez, E.A., Asmundson, G.J.G., 2011. Dissociation and Pain Sensitivity in Individuals with and Without PTSD. ABCT's 45th Annual Convention of the International Association for Behavioral and Cognitive Therapies. Toronto, Canada.
Green, B.L., Chung, J.Y., Daroowalla, A., Kaltman, S., DeBedenedictis, C., 2006. Evaluating the cultural validity of the Stressful Life Events Screening Questionnaire. Violence Against Woman 12, 1191–1213.
Hayes, S.C., Strosahl, K., Wilson, KG, Bissett, R.T., Pistorello, J., Toarmino, D., Polusny, M.A., Dykstra, T.A., Batten, S.V., Bergan, J., Stewart, S.H., Zvolensky, M.J., Eifert, G.H., Bond, F.W., Forsyth, J.P, Karekla, M., Mccurry, S.M., 2004. Measuring experiential avoidance: a preliminary test of a working model. The Psychological Record 54, 553–578.
Hayes, S.C., Wilson, K.W., Gifford, E.V., Follette, V.M., Strosahl, K., 1996. Experiential avoidance and behavioral disorders: a functional dimensional approach to diagnosis and treatment. Journal of Consulting and Clinical Psychology 64, 1152–1168.
Kellner, M., Yassouridis, A., Hua, Y., Wendrich, M., Naber, D., Wiedemann, K., 2002. Trait dissociation affects the behavioral response to cholecystokinin tetrapeptide in healthy man. Psychiatry Research 111, 93–96.
Kosloff, S., Solomon, S., Greenberg, J., Cohen, F., Gershuny, B., Routledge, C., Pyszczynski, T., 2006. Fatal distraction: the impact of mortality salience on dissociative responses to 9/11 and subsequent anxiety sensitivity. Basic and Applied Social Psychology 28, 349–356.
Kumpula, M.J., Orcutt, H.K, Bardeen, J.R., Varkovitzky, R.L., 2011. Peritraumatic dissociation and experiential avoidance as prospective predictors of posttraumatic stress symptoms. Journal of Abnormal Psychology 120, 617–627.
Leonard, K.N., Telch, M.J., Harrington, P.J., 1999. Dissociation in the laboratory: a comparison of strategies. Behaviour Research and Therapy 37, 49–61.
Lipsanen, T., Saarijärvi, S., Lauerma, H., 2004. Exploring the relations between depression, somatization, dissociation and alexithymia—overlapping or independent constructs? Psychopathology 37, 200–206.
Marshall, G.N., Orlando, M., Jaycox, L.H., Foy, D.W., Belzberg, H., 2002. Development and validation of a modified version of the Peritraumatic Dissociative Experiences Questionnaire. Psychological Assessment 14, 123–134.
Marx, B.P., Sloan, D.M., 2005. Peritraumatic dissociation and experiential avoidance as predictors of posttraumatic stress symptomatology. Behaviour Research and Therapy 43, 569–583.

Merckelbach, H., Giesbrecht, T., 2006. Subclinical dissociation, schizotypy, and traumatic distress. Personality and Individual Differences 40, 365–374.
Nijenhuis, E.R.S., Spinhoven, P., Van Dick, R., Van Der Hart, O., Vanderlinden, J., 1996. The development and psychometric characteristics of the Somatoform Dissociation Questionnaire (SDQ-20). The Journal of Nervous and Mental Disease 184, 688–694.
Nixon, R.D.V., Bryant, R.A., 2006. Dissociation in acute stress disorder after a hyperventilation provocation test. Behavioural and Cognitive Psychotherapy 34, 343–349.
Pastucha, P., Prasko, J., Grambal, A., Latalova, K., Sigmundova, Z., Tichackova, A., 2009. Dissociative disorder and dissociation—comparison with healthy controls. Neuroendocrinology Letters 30, 769–773.
Prasko, J., Raszka, M., Diveky, T., Grambal, A., Kamaradova, D., Koprivova, J., Latalova, K., Pastucha, P., Sigmundova, Z., 2010. Obsessive compulsive disorder and dissociation—comparison with healthy controls. Biomedical Papers of the Medical Faculty of Palacky 154, 179–183.
Radloff, L.S, 1977. The CES-D scale: a self-report depression scale for research in the general population. Applied Psychological Measurement 1, 385–401.
Reiss, S., McNally, R.J., 1985. The expectancy model of fear. In: Reiss, S., Bootzin, R.R. (Eds.), Theoretical Issues in Behavior Therapy. Academic Press, New York, pp. 107–121.
Sayar, K., Kose, S., 2003. The relationship between alexithymia and dissociation in an adolescent sample. Bulletin of Clinical Psychopharmacology 13, 167–173.
Schmidt, N.B., Richey, J.A., Zvolensky, M.J, Maner, J.K., 2008. Exploring human freeze responses to a threat stressor. Journal of Behavior Therapy and Experimental Psychiatry 39, 292–304.
Schmidt, N.B., Zvolensky, M.J., 2007. Anxiety sensitivity and CO_2 challenge reactivity as unique and interactive prospective predictors of anxiety pathology. Depression and Anxiety 24, 527–536.
Spiegel, D., 1991. Dissociation and trauma. In: Tasman, A., Goldfinger, S.M. (Eds.), American Psychiatric Press Review of Psychiatry. American Psychiatric Press, Washington DC, pp. 261–275.
Spielberger, C.D, Gorsuch, R.R, Lushene, R.E., 1970. State-Trait Anxiety Inventory Test Manual for Form X. Consulting Psychologists Press, Palo Alto, CA.
Sterlini, G.L., Bryant, R.A., 2002. Hyperarousal and dissociation: a study of novice skydivers. Behaviour Research & Therapy 40, 431–437.
Sullivan, M.J.L., Bishop, S.R., Pivik, J., 1995. The Pain Catastrophizing Scale. Development and validation. Psychological Assessment 7, 524.
Vázquez, F.L., Blanco, V., López, M., 2007. An adaptation of the Center for Epidemiologic Studies Depression Scale for use in non-psychiatric Spanish populations. Psychiatry Research 149, 247–252.
Waller, N.G., Putnam, F.W., Carlson, E.B., 1996. Types of dissociation and dissociative types: a taxometric analysis of dissociative experiences. Psychological Methods 1, 300–321.
Zhao, X., Lynch, J.G., Chen, Q., 2010. Reconsidering Baron and Kenny: myths and truths about mediation analysis. Journal of Consumer Research 37, 197–206.
Zvolensky, M.J., Forsyth, J.P., Bernstein, A., Leen-Feldner, E.W., 2007. A concurrent test of the anxiety sensitivity taxon: its relation to bodily vigilance and perceptions of control over anxiety-related events in a sample of young adults. Journal of Cognitive Psychotherapy 21, 72–90.