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Emotional memory and perception of emotional faces in patients suffering from depersonalization disorder

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Previous work has shown that patients with depersonalization disorder (DPD) have reduced physiological responses to emotional stimuli, which may be related to subjective emotional numbing. This study investigated two aspects of affective processing in 13 patients with DPD according to the DSM-IV criteria and healthy controls: the perception of emotional facial expressions (anger, disgust, fear, happiness, sadness, and surprise) and memory for emotional stimuli. Results revealed a specific lack of sensitivity to facial expression of anger in patients, but normal enhancement of memory for peripheral aspects of arousing emotional material. The results are consistent with altered processing of threat-related stimuli but intact consolidation processes, at least when the stimuli involved are potently arousing.

Depersonalization is defined in the DSM-IV (APA, 1994) as 'a feeling of detachment or estrangement from one's self'. Other prominent symptoms include a lack of emotional feeling and reactivity. When persistent and accompanied by loss of function, it may be defined as depersonalization disorder (DPD). A recent study comparing historical with current cases of DPD indicates that the clinical manifestations of the condition have remained stable for the last century, suggesting a neurobiological basis (Sierra & Berrios, 2001). Depersonalization occurs in association with diverse neuropsychiatric conditions (Lambert, Sierra, Phillips, & David, 2002; Sierra, Lopera, Lambert, Phillips, & David, 2002a) or as a primary disorder in which case it tends to run a chronic and disabling course (Baker *et al.*, 2003; Simeon *et al.*, 1997). Prevalence studies have

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indicated that from 40 to 80% of psychiatric in-patients suffer from comorbid secondary depersonalization (Brauer, Harrow, & Tucker, 1970; Hunter, Sierra, & David, 2004). Although the prevalence of DPD in the general population is unknown, available data suggest it to range from 1.7 to 2.4% (for a systematic review see Hunter *et al.*, 2004).

Depersonalization has been shown to correlate with anxiety measures, and most patients with a diagnosis of DPD have been shown to have significant levels of anxiety or comorbid anxiety disorders (Baker *et al.*, 2003; Lambert, Senior, Fewtrell, Phillips, & David, 2001). This, together with the high prevalence of depersonalization in times of life-threatening situations, has been interpreted as suggesting that depersonalization represents an anxiety-triggered 'hard-wired' inhibitory mechanism on emotional processing intended to ensure the preservation of adaptative behaviour during situations that are normally associated with overwhelming and potentially disorganizing anxiety (Noyes & Kletti, 1977; Sierra & Berrios, 1998). In contrast to the fight or flight response, it has been proposed that depersonalization would normally be triggered by life-threatening situations in which the individual does not seem to have control over the situation or when the source of danger cannot be localized (e.g. earthquake, car crash, etc.). In such circumstances, depersonalization may result in the *inhibition* of non-functional emotional and autonomic responses. It has been suggested that in patients with DPD, this response becomes abnormally persistent and dysfunctional (Baker *et al.*, 2003).

Recent fMRI (Phillips *et al.*, 2001) and psychophysiological studies (Sierra *et al.*, 2002b) support this model and have indicated that patients with DPD show lack of activation in limbic areas, and marked autonomic attenuation in response to pictures depicting disgusting or distressing situations. In line with this is a study investigating the level of norepinephrine in DPD. Norepinephrine is a neurotransmitter which plays an important role in anxiety. Elevated levels of this neurotransmitter have been reported in trauma-related disorders, with high anxiety levels (Southwick *et al.*, 1999). Although Simeon, Guralnik, Knutelska, Yehuda, and Schmeidler (2003) found high levels of norepinephrine in DPD, a striking negative correlation of 0.8 between intensity of depersonalization and urine norepinephrine levels was reported, supporting an anxiety-suppressing mechanism in DPD. These findings may also be related to abnormalities in emotional subjective experiencing. For example, Phillips *et al.* found that patients with depersonalization rated unpleasant pictures as less emotional when compared with a control group of patients with obsessive compulsive disorder. Likewise, Sierra *et al.* (2002b) found that, compared with anxiety disorder patients and healthy controls, patients with DPD rated unpleasant pictures from a standard set as less arousing.

In order to further characterize emotional processing in DPD, this study focused on two different aspects of emotional processing, that is, explicit recognition of emotional faces and memory for emotional events. In view of the above findings suggesting that depersonalization produces a dampening effect on neurobiological and subjective responses to aversive stimuli, it was predicted that patients with DPD would show a selective deficit in the detection of threat-signalling emotional facial expressions, such as fear, anger, and disgust. A control task was administered in order to examine the capability of matching neutral unfamiliar faces (i.e. facial perception regardless of emotional content). With respect to emotional memory, it has been well established that long-term memory for emotional arousing events is generally enhanced compared with memory for neutral events (Burke, Heuer, & Reisberg, 1992; Hamann, 2001). Although it has been proposed that this effect only holds for the emotionally arousing details of the remembered event (Payne *et al.*, 2006), at the exclusion of peripheral information, not all studies confirm this pattern (Heuer & Reisberg, 1990). Neurobiological findings

suggest that this enhancement is mediated by the amygdala (Adolphs, Cahill, Schul, & Babinsky, 1997; Brierley, Medford, Shaw, & David, 2004), a brain structure in the limbic system which is involved in emotion processing in general (e.g. Adolphs, Tranel, & Damasio, 1998; Young *et al.*, 1995) and may be specifically related to the processing of threat-related information (e.g. Adolphs, Tranel, Damasio, & Damasio, 1994, 1995). In view of the model of depersonalization as selectively impairing the processing of aversive, arousing information it was predicted that patients with depersonalization would not show the expected emotional enhancement of memory for arousing stimuli.

Methods

Participants

Thirteen patients meeting DSM-IV criteria for DPD were recruited from the Depersonalization Disorder Clinic of the Maudsley Hospital (see Table 1). The patients were diagnosed by an experienced psychiatrist (MS) and underwent a 2-hour semi-structured psychiatric interview, which incorporated the Present State Examination (PSE; Wing, Cooper, & Sartorius, 1974). Case definition required a total score of ≥ 2 on either or both of the depersonalization/derealization items (Baker *et al.*, 2003). Previous to the interview, patients also filled in a self-report questionnaire which contained depersonalization-related questions of the Structured Clinical Interview for Dissociative Disorders (Steinberg, Cicchetti, Buchanan, Rakfeldt, & Rounsaville, 1994) adapted to a self-rating format. In addition, information was obtained regarding current anxiety symptoms, history of medical and neurological illness, drug and alcohol abuse and history of, or current comorbid disorders. Exclusion criteria included lifetime incidence of psychotic disorder, current substance abuse disorder, current major depression, other dissociative disorder, and history of neurological disorder. Patients meeting criteria for anxiety disorders were excluded where depersonalization was found to be circumscribed to episodes of intense anxiety. Patients scored in the moderate dissociation range on the Dissociative Experiences Scale (DES) and above the cut-off for DPD of 70 on the Cambridge Depersonalization Scale (CDS; Sierra & Berrios, 2000; see Table 1).

Table 1. Demographic data, face recognition performance, and self-report depression scores for all participants plus self-report dissociation and depersonalization scale scores for the depersonalization patients only

	Patients ($N = 13$)		Control group ($N = 33$)	
	Mean	*SD*	*Mean*	*SD*
Age	33.2	8.2	36.0	11.7
Sex (male:female)	9:4		19:14	
Test of facial recognition	23.8	1.8	23.1	2.5
Beck depression inventory	21.2	10.3	2.4	3.5
Beck anxiety inventory	18.7	13.3	–	–
Dissociative experiences scale (%)	29.7	12.7	–	–
Cambridge depersonalization scale	129.8	56.3	–	–

Materials

Neuropsychological tests

All participants completed the National Adult Reading test (Nelson & Willison, 1991), which provides an estimate of premorbid IQ, and the Beck Depression Inventory (Beck, Steer, & Garbin, 1988; BDI range 0–63). Patients also completed the 28-item DES (Bernstein & Putnam, 1986; Carlson & Putnam, 1993), which includes a broad range of symptoms, including depersonalization/derealization, and the CDS (Sierra & Berrios, 2000) range 0–290.

In order to control general memory deficits or non-specific anomalies in face perception, participants were administered the Benton and Van Allen Test of Facial Recognition (Benton, Sivan, deS Hamsher, Varney, & Spreen, 1994) comprises a series of sheets containing a single photographed target face to be matched to a set of six face photographs. In the first six trials, an identical face has to be selected among five decoys. In the remaining seven trials, three different views (changed in orientation or lighting conditions compared with the target photograph) have to be discriminated from three incorrect alternatives. Scoring ranges from 0 to 27. Patients with DPD also completed the Wechsler Memory Scale-III (Wechsler, 1997) and all performed within the normal range (data not shown).

Emotion recognition task

Stimuli for this test were based on colour pictures from actors mimicking emotional expressions and a neutral face. There were four actors (two males and two females) who each posed six emotions (anger, disgust, fear, happiness, sadness, and surprise). A computer-generated programme, developed from algorithms designed by Benson and Perrett (1991), was used to develop the stimuli. This programme was devised to create intermediate morphed images between a neutral face (0% emotion) and a full-blown expression (100% emotion). The original colour pictures of the faces were manually delineated by 179 feature points that define the shape of the important facial features (Rowland & Perrett, 1995). For each expression, two photographs were available, the neutral and the full-blown expressions. The intermediate expressions were simulated. This was done by generating 19 morphed images by calculating the difference in spatial position between two corresponding feature points in the neutral and full-blown expressions.

In the morphed images, both the dimension of shape and texture underwent gradual transitions. These images were used to construct video clips that incrementally increase the degree of expression by 10% steps. Consequently, each complete video clip entailed 21 images and 20 image steps and was used to make the individual video clips for the different trials. The first image is the neutral face, which has 0% emotion on the face, the second image has an increment of 5% emotion on the face, and each next image has an additional 5% more emotion on the face, ending with the last image, which is a 100% emotional face. Thus, the video clip from 0 to 20% contains the first five images, and for each subsequent video clip increments of 10% were used, which consists of two images (e.g. the sequence from 0 to 30% are the first seven images, from 0 to 40% are the first nine images, and so on). For each actor, nine video clips (0–20, 0–30, 0–40, 0–50, 0–60, 0–70, 0–80, 0–90, and 0–100%) were constructed for each of the six emotions by increasing the number of morphed images presented in succession (see Figure 1). In total, there were 216 trials, i.e. the number of emotions used (6) × the number of actors (4) × the number of sequences (9). The presentation procedure was as follows. First, the participants saw,

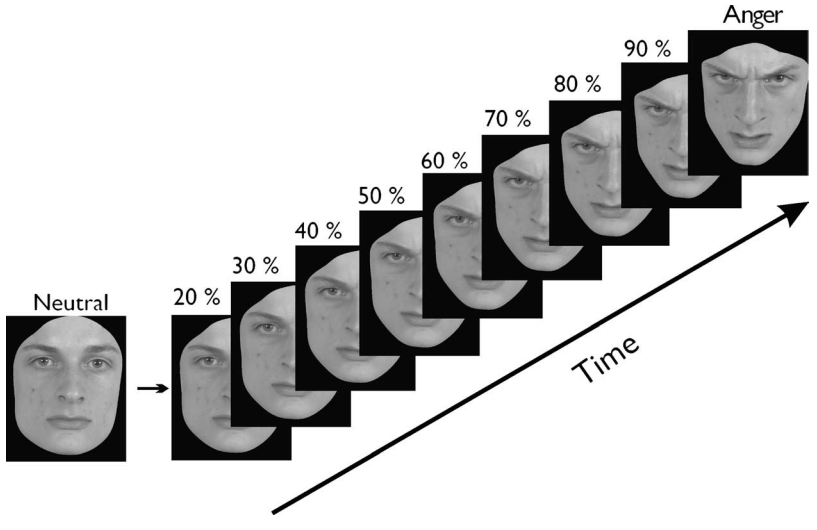

Figure 1. Example of a morphed facial expression of the emotion anger in 10% incremental steps of emotional intensity.

in a random order, the first 24 trials (six emotions × four actors), which were video clips from 0 to 20% (there was no set of 24 trials with video clips from 0 to 10%). The second set of 24 trials was video clips from 0 to 30% and then for each set of 24 trials, the video clip goes up with increments of 10%, until they reached the final sequence of clips in which the neutral face changed into a full-blown expression. In each trial, the participant was required to make a forced choice between one of six emotional expression labels that were displayed on the screen after the presentation of the video clip. There was no time restriction (Montagne, Kessels, Wester, & de Haan, 2006).

Thirty-three healthy controls were volunteers selected from staff and students of the Institute of Psychiatry and King's College London or from students of Utrecht University, the Netherlands (the controls for the emotional memory task were different from those for the emotional recognition task). By self-report, none of the controls had a history of psychiatric illness or neurological disorder. The study was approved by the local research ethics committee, and all participants provided informed consent. Men and women were equally distributed in both groups ($\chi^2(1) = 2.2, p = .14$). As expected, the DPD patients scored higher than controls on the BDI (most in the mild or moderate range ($t(37) = 8.55, p < .001$). NART IQ did not differ significantly between the groups.

Emotional memory task

This is a version of the Heuer and Reisberg (1990) test of emotional memory modified by Cahill and colleagues (Cahill & McGaugh, 1995). It consists of 11 picture slides and accompanying text, depicting a story of a boy who suffers a terrible car accident after which he is rushed to the hospital. This emotional, central section of the story follows and precedes neutral sections that set the scene and describe the repercussions, respectively. Memory for story details is assessed, without prior warning, 1 week after presentation with a 76-item multiple choice questionnaire, focusing on central and peripheral aspects of emotional and neutral information presented. Twenty healthy age-matched control participants were included (see Table 2) with no history of psychiatric or neurological disorder. Men and women were equally distributed over both groups

($\chi^2(1) = 1.49$, $p = .22$). NART IQ scores were significantly higher in the controls ($t(31) = 3.16$, $p < .01$).

Table 2. Characteristics of the depersonalization patients and controls participating in the emotional memory task

	Patients ($N = 12$)		Control group ($N = 20$)	
	Mean	*SD*	*Mean*	*SD*
Age	33.2	8.2	38.1	12.3
NART-IQ	115.0	7.1	103.0	12.3
Sex (male:female)	8:4		11:9	

Note. NART, national adult reading task.

Procedure

The tests were presented to each participant in a fixed order. Participants were comfortably seated in front of a computer screen (15 inch) and asked to perform the emotional expression task. Participants performed four practice trials followed by the actual task. Participants had to indicate which emotion they perceived from a set of six possible emotion labels (i.e. forced choice). There was no time restriction. Subsequently, the Test of Facial Recognition was administered and participants were asked to fill out the BDI and other self-report scales. Finally, they were given the Emotional Memory Task followed a week later by the memory test.

Results

Emotion recognition task

Sensitivity was conceptualized as the minimum amount of expression information required for systematic correct identification, that is, the percentage of emotion from which point onwards the expression was consistently recognized in subsequent trials (i.e. a sensitivity of 40% for a given emotional expression indicates that the participant correctly identified the emotion at 40% and gave a correct answer for all subsequent percentages between 40 and 100%). A 6 × 2 MANOVA was carried out with expression (six levels: anger, fear, disgust, happiness, sadness, and surprise) as within-subject factor and group (two levels: patient, control) as between-subject variable. This MANOVA revealed a main effect of expression ($F(5, 29) = 27.56$, $p < .001$) and an interaction effect of expression × group ($F(5, 29) = 2.80$, $p < .04$). There was no main effect of group ($F(1, 33) = 0.12$, $p = .74$). Bonferroni-corrected *post hoc* tests (α set at .008) showed a significant difference in sensitivity between the two groups only for the emotion anger ($F(1, 43) = 8.16$, $p = .007$), with the depersonalization patients being less sensitive compared with the control subjects (see Figure 2). Since the patients had elevated scores on the BDI, data were also analysed with the BDI scores as covariate. Still, there was a significant difference for the emotion anger ($F(2, 35) = 5.90$, $p = .006$; α set at .008), indicating that the depressive symptoms could not account for the differences in emotion perception.

Test of facial recognition

Table 1 shows the results on the Test of Facial Recognition. Performance was analysed using an independent sample *t* test, which revealed no significant difference between

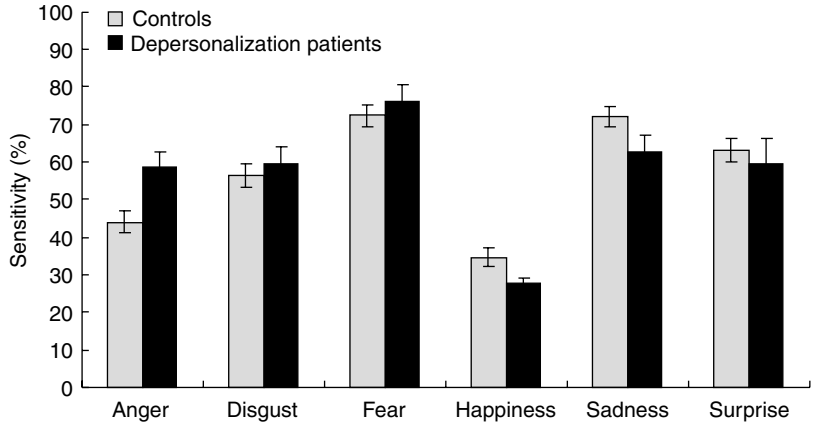

Figure 2. The sensitivity (± SEM) for all six emotional expressions for the depersonalization patients ($N = 13$) and the controls ($N = 33$). Significant group difference for anger only.

the patient and the control group ($t(43) = 0.89$). Both performances are within the normal range according to the normative data for the test.

Emotional memory task

Data for this task are from 12 patients, since one patient could not be contacted 1 week later. Figure 3 shows the data for the Emotional Memory Task for the central and peripheral aspects of the presented emotional and neutral information. A 2 × 2 × 2 ANOVA with arousal (neutral and emotional) and information type (peripheral and

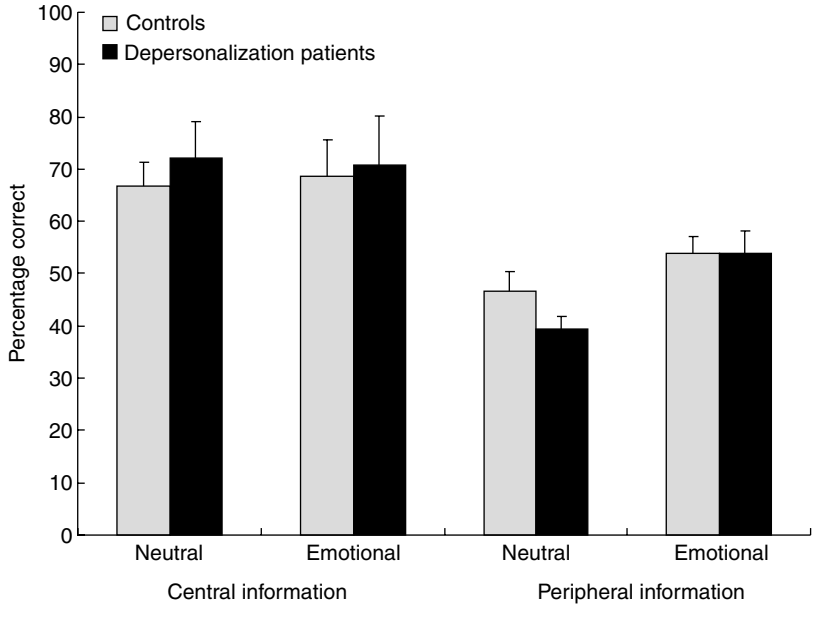

Figure 3. Performance (± SEM) on central and peripheral aspects of emotional or neutral information in the Cahill emotional memory task for the depersonalization patients ($N = 12$) and the controls ($N = 20$).

central) as within-subject variables, group (patient and control) as between-subject variable showed a main effect of arousal ($F(1, 28) = 5.24, p < .03$). In addition, a main effect of information type was found ($F(1, 28) = 43.82, p < .0005$), as well as an interaction between arousal and information type ($F(1, 28) = 4.24, p < .05$). Follow-up paired t tests showed that peripheral aspects were remembered worse than central aspects, both for neutral ($t(29) = 7.25, p < .0005$) and for emotional ($t(29) = 3.46, p < .002$) information. With respect to arousal, follow-up paired t tests showed that peripheral information was remembered more accurately when this information was emotional compared with neutral ($t(29) = 4.24, p < .0005$), but no arousal effect was found for central information ($t(29) = .13$). There was neither a main effect of group ($F(1, 27) = 0.01, p = .98$) nor were there any interactions with group (all F values < 1.40). This non-significant group effect was not due to differences in NART IQ (ANCOVA with NART IQ as covariate showed a non-significant group effect as well: $F(1, 27) = 0.56$).

Discussion

The aim of the study was to look at emotional processing in patients with DPD, focusing on recognizing emotional faces and memory for an arousing story. The results on the task measuring recognition of facial expressions showed that patients with DPD were less sensitive in the detection of angry facial expressions compared with the control group. This result is unlikely to be due to a general face-processing deficit, as both groups performed within the normal range on a perception test of neutral faces. It could be argued that deficits in anger recognition are due to differences in depressive symptoms, since facial recognition studies in depression have found either impairments in the recognition of neutral faces (Leppanen, Milders, Bell, Terriere, & Hietanen, 2004) or an overall attenuation of recognition sensitivity (Mandal & Bhattacharya, 1985). However, none of our patients was clinically depressed. Moreover, by statistically adjusting for depressive symptoms between the controls and the patients showed that the deficit in emotion perception was not due to underlying differences in depressive symptoms.

Although we had predicted a general deficit for the recognition of threat-signalling facial expressions, a selective deficit for anger recognition is in keeping with our prediction and offers support to the idea that depersonalization functions as a dampening filter for the perception of unpleasant stimuli. Interestingly, of the three facial expressions with threat-signalling value (i.e. fear, disgust, and anger), only anger has direct communicative value: the perception of anger becomes a threat in its own right. Fear and disgust can only signal threat via a cognitive process involving 'theory of mind' deduction (e.g. if somebody is displaying fear it means that individual is perceiving a threat, which may be of relevance for my safety).

Given the intimate relationship between anxiety and depersonalization, it is interesting that a number of studies have found evidence suggesting that anxiety sensitizes individuals to the detection of threat. This effect has also been shown in some studies using facial emotional expressions as stimuli. Byrne and Eysenck (1995), for example, found that high trait-anxious individuals were quicker to identify an angry face in a neutral crowd compared with low trait-anxious individuals. Likewise, using a morphed face paradigm found that regardless of baseline measures of anxiety, the acute elicitation of anxiety by a stress-inducing situation selectively sensitized participants to recognize angry faces (Richards *et al.*, 2002). Interestingly, it has also been found that

medications with anxiety-suppressing effects impair the ability to recognize angry expressions. For example, 15 mg of diazepam (a benzodiazepine) selectively impaired the ability to recognize angry expressions in healthy participants, but did not affect recognition of any other emotional expression (Blair & Curran, 1999). The dopamine system does also play an important role in several brain structures known to be involved in the processing of several emotions, for one the dopamine system has been implicated in the processing of signals of aggression in social encounters.

Additionally, Lawrence, Calder, McGowan, and Grasby (2002) found that a single dose of the dopamine D2-class receptor antagonist sulpiride, led to a selective disruption in the recognition of facial expressions of anger. If, as has been proposed, depersonalization has a dampening effect on potentially disorganizing levels of anxiety (triggered in-turn by perceived threat and lack of control), it is not surprising that the alleged inhibitory action of depersonalization on emotional processing would be most noticeable for that emotion whose recognition is normally amplified by anxiety.

Contrary to our prediction, the results of the emotional memory task did not reveal a difference between the patient and the control groups. Both groups showed an enhanced memory effect for emotionally arousing peripheral aspects of a story, but neither groups displayed an arousal effect for the central aspects. This supports other work on non-clinical participants who scored highly on the DES, which failed to show the predicted reduced recall of an emotive story compared with those who had low DES scores (Candel, Merckelbach, & Kuijpers, 2003). Thus, there does not seem to be an attenuation of the potent arousal effect of Cahill emotional narrative in patients with DPD. Future studies should replicate these findings using other emotional memory tasks, such as the one designed by Brierley and Medford. Their task has been developed to examine memory performance for emotionally aversive words, as well as for neutral words encoded in an aversive context (Brierley *et al.*, 2004; Kensinger, Brierley, Medford, Growdon, & Corkin, 2002; Medford *et al.*, 2005). Finally, we would like to stress that the lack of anxiety data for controls is a limitation of our study, and future studies should include these data for all participants.

In conclusion, patients with DPD showed deficits in recognition of a fear-inducing facial expression. This finding is consistent with the view that depersonalization functions as a filter for aversive stimuli. However, we did not find evidence for a reduction of memory-enhancing effects of particularly potent emotional material. Although both perception and memory are mediated by arousal, there may be crucial differences in the underlying mechanisms. Further exploration of the effects of depersonalization and related states on different aspects of emotion processing is warranted.

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References

Adolphs, R., Cahill, L., Schul, R., & Babinsky, R. (1997). Impaired declarative memory for emotional material following bilateral amygdala damage in humans. *Learning and Memory*, *4*, 291–300.

Adolphs, R., Tranel, D., & Damasio, A. R. (1998). The human amygdala in social judgment. *Nature*, *393*, 470–474.

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. R. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature*, *372*, 669–672.

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. R. (1995). Fear and the human amygdala. *Journal of Neuroscience*, *15*, 5879–5891.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington DC: Author.

Baker, D., Hunter, E., Lawrence, E., Medford, N., Patel, M., Senior, C., *et al.* (2003). Depersonalisation disorder: Clinical features of 204 cases. *British Journal of Psychiatry*, *182*, 428–433.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck depression inventory: Twenty-five years of evaluation. *Clinical Psychology Review*, *8*, 77–100.

Benson, P. J., & Perrett, D. I. (1999). Synthesising continuous-tone caricatures. *Image and Vision Computing*, *9*, 123–129.

Benton, A. L., Sivan, A. B., deS Hamsher, K., Varney, N. R., & Spreen, O. (1994). *Contributions to neuropsychological assessment. A clinical manual* (2nd ed.). New York: Oxford University Press.

Bernstein, E. M., & Putnam, F. W. (1986). Development, reliability, and validity of a dissociation scale. *Journal of Nervous and Mental Disease*, *174*, 727–735.

Blair, R. J., & Curran, H. V. (1999). Selective impairment in the recognition of anger induced by diazepam. *Psychopharmacology (Berl)*, *147*, 335–338.

Brauer, R., Harrow, M., & Tucker, G. J. (1970). Depersonalization phenomena in psychiatric patients. *British Journal of Psychiatry*, *117*, 509–515.

Brierley, B., Medford, N., Shaw, P., & David, A. S. (2004). Emotional memory and perception in temporal lobectomy patients with amygdala damage. *Journal of Neurology Neurosurgery and Psychiatry*, *75*, 593–599.

Burke, A., Heuer, F., & Reisberg, D. (1992). Remembering emotional events. *Memory and Cognition*, *20*, 277–290.

Byrne, A., & Eysenck, M. W. (1995). Trait anxiety, anxious mood, and threat detection. *Cognition and Emotion*, *9*, 549–562.

Cahill, L., & McGaugh, J. L. (1995). A novel demonstration of enhanced memory associated with emotional arousal. *Consciousness and Cognition*, *4*, 410–421.

Candel, I., Merckelbach, H., & Kuijpers, M. (2003). Dissociative experiences are related to commissions in emotional memory. *Behavior Research and Therapy*, *41*, 719–772.

Carlson, E. B., & Putnam, F. W. (1993). An update on the dissociative experiences scale. *Dissociation*, *6*, 16–27.

Hamann, S. (2001). Cognitive and neural mechanisms of emotional memory. *Trends in Cognitive Sciences*, *5*, 394–400.

Heuer, F., & Reisberg, D. (1990). Vivid memories of emotional events: The accuracy of remembered minutiae. *Memory and Cognition*, *18*, 496–506.

Hunter, E., Sierra, M., & David, A. S. (2004). The epidemiology of depersonalisation and derealisation. A systematic review. *Social Psychiatry and Psychiatric Epidemiology*, *39*, 9–18.

Kensinger, E. A., Brierley, B., Medford, N., Growdon, J. H., & Corkin, S. (2002). Effects of normal ageing and Alzheimer's disease on emotional memory. *Emotion*, *2*, 118–134.

Lambert, M. V., Senior, C., Fewtrell, W. D., Phillips, M. L., & David, A. S. (2001). Primary and secondary depersonalisation disorder: A psychometric study. *Journal of Affective Disorders*, *63*, 249–256.

Lambert, M. V., Sierra, M., Phillips, M. L., & David, A. S. (2002). The spectrum of organic depersonalization: A review plus four new cases. *Journal of Neuropsychiatry and Clinical Neurosciences*, *14*, 141–154.

Lawrence, A. D., Calder, A. J., McGowan, S. W., & Grasby, P. M. (2002). Selective disruption of the recognition of facial expressions of anger. *Neuroreport*, *13*, 881–884.

Leppanen, J. M., Milders, M., Bell, J. S., Terriere, E., & Hietanen, J. K. (2004). Depression biases the recognition of emotionally neutral faces. *Psychiatry Research*, *128*, 123–133.

Mandal, M. K., & Bhattacharya, B. B. (1985). Recognition of facial affect in depression. *Perceptual and Motor Skills*, *61*, 13–14.

Medford, N., Phillips, M. L., Brierley, B., Brammer, M. J., Bullmore, E. T., & David, A. S. (2005). Emotional memory: Separating content and context. *Psychiatry Research: Neuroimaging*, *138*, 247–258.

Montagne, B., Kessels, R. P. C., Wester, A. J., & de Haan, E. H. F. (2006). Processing of emotional facial expressions in Korsakoff's syndrome. *Cortex*, *42*, 705–710.

Nelson, H., & Willison, J. (1991). *The National adult reading test (NART): Test manual* (2nd ed.). Windsor, UK: NFER Nelson.

Noyes, R. Jr., & Kletti, R. (1977). Depersonalisation in response to life-threatening danger. *Comprehensive Psychiatry*, *18*, 375–384.

Payne, J. D., Jackson, E. D., Ryan, L., Hoscheidt, S., Jacobs, J. W., & Nadel, L. (2006). The impact of stress on neutral and emotional aspects of episodic memory. *Memory*, *14*, 1–16.

Phillips, M. L., Medford, N., Senior, C., Bullmore, E. T., Suckling, J., Brammer, M. J., *et al.* (2001). Depersonalization disorder: Thinking without feeling. *Psychiatry Research*, *108*, 145–160.

Richards, A., French, C. C., Calder, A. J., Webb, B., Fox, R., & Young, A. W. (2002). Anxiety-related bias in the classification of emotionally ambiguous facial expressions. *Emotion*, *2*, 273–287.

Rowland, D. A., & Perrett, D. I. (1995). Manipulating facial appearance through shape and colour. *IEEE Computer Graphics and Applications*, *15*, 70–76.

Sierra, M., & Berrios, G. E. (1998). Depersonalization: Neurobiological perspectives. *Biological Psychiatry*, *44*, 898–908.

Sierra, M., & Berrios, G. E. (2000). The Cambridge depersonalisation scale: A new instrument for the measurement of depersonalisation. *Psychiatry Research*, *93*, 153–164.

Sierra, M., & Berrios, G. E. (2001). The phenomenological stability of depersonalization: Comparing the old with the new. *Journal of Nervous and Mental Disease*, *189*, 629–636.

Sierra, M., Lopera, F., Lambert, M. V., Phillips, M. L., & David, A. S. (2002a). Separating depersonalisation and derealisation: The relevance of the lesion method. *Journal of Neurology Neurosurgery and Psychiatry*, *72*, 530–532.

Sierra, M., Senior, D., Dalton, J., McDonough, M., Bond, A., Phillips, M. L., *et al.* (2002b). Autonomic response in depersonalisation disorder. *Archives of General of Psychiatry*, *59*, 633–638.

Simeon, D., Gross, S., Guralnik, O., Stein, D. J., Schmeidler, J., & Hollander, E. (1997). Feeling unreal: 30 cases of DSM-III-R depersonalization disorder. *American Journal of Psychiatry*, *154*, 1107–1113.

Simeon, D., Guralnik, O., Knutelska, M., Yehuda, R., & Schmeidler, J. (2003). Basal norepinephrine in depersonalization disorder. *Psychiatry Research*, *121*, 93–97.

Southwick, S. M., Bremner, J. D., Rasmusson, A., Morgan, C. A., Arnsten, A., & Charney, D. S. (1999). Role of norepinephrine in the pathophysiology and treatment of posttraumatic stress disorder. *Biological Psychiatry*, *46*, 1192–1204.

Steinberg, M., Cicchetti, D., Buchanan, J., Rakfeldt, J., & Rounsaville, B. (1994). Distinguishing between multiple personality disorder (dissociative identity disorder) and schizophrenia using the Structured clinical interview for DSM-IV Dissociative disorders. *Journal of Nervous and Mental Disease*, *182*, 495–502.

Wechsler, D. (1997). *Wechsler memory scale: Third edition manual*. San Antonio, TX: Psychological Corporation.

Wing, J. K., Cooper, J. E., & Sartorius, N. T. (1974). *Measurement and classification of psychiatric symptoms: An instruction manual for PSE and category system*. Cambridge: Cambridge University Press.

Young, A. W., Aggleton, J. P., Hellawell, D. J., Johnson, M., Broks, P., & Hanley, J. R. (1995). Face processing in impairments after amygdalectomy. *Brain*, *118*, 15–24.

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