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Priming Associations between Bodily Sensations and Catastrophic Misinterpretations: Specific for Panic Disorder ?

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Abstract

Cognitive models assume that panic disorder is characterised by a tendency to misinterpret benign bodily symptoms (e.g. breathlessness) in a catastrophic fashion (e.g. suffocation). This is a central part of the cognitive model which presents a core focus for treatment. Several studies have supported this hypothesis. These studies have, however, almost always relied on self-report. In addition to susceptibility to biases (e.g. distortions of memory), a limitation of research based on verbal report is its inability to capture the spontaneous/automatic nature that is attributed to these catastrophic interpretations. The present paper reports on two experiments in which a priming procedure was used to test the hypothesis that panic disorder is characterised by spontaneous catastrophic interpretations and whether this effect is 'specific' to panic disorder. In line with predictions from the cognitive model, it was observed in the first experiment that the panic group demonstrated facilitated responses to trials consisting of a 'symptom' prime and a 'catastrophic outcome' target (e.g. breathlessness – suffocate). Similar effects were not observed for an anxious control group and a non-clinical control group, supporting the specificity of this effect. Interestingly, however, significant priming effects were observed for a group of mental health professionals (part of the healthy control group) who had no history of panic disorder. Subsequently, this unexpected observation was explicitly addressed in a second experiment, which confirmed the findings of Experiment 1. Together, these results suggest that associations between mental representations of benign bodily symptoms and catastrophic outcomes might develop as part of professional knowledge and experience, and should not necessarily be viewed as pathogenic. Theoretical and clinical implications are discussed.

Key words: panic disorder, associative priming, misinterpretation, cognitive models

Priming Associations between Bodily Sensations and Catastrophic Misinterpretations: Specific for Panic Disorder ?

One of the central tenets of cognitive models of panic disorder (e.g. Clark, 1986, 1988) is that persons suffering from panic disorder (PD) are characterised by a relatively enduring tendency to misinterpret benign bodily sensations as indications of an immediately impending physical or mental catastrophe. Typically, autonomic responses like palpitations, breathlessness or headache are spontaneously interpreted as signals of an impending heart attack, choking or the presence of a brain tumour. This is assumed to be the basis for a vicious circle in which misinterpretations of bodily sensations increase anxiety, which subsequently amplifies the level of these (anxiety related) sensations, which in turn feeds the catastrophic misinterpretations and the level of anxiety that follows from them. This loop can then terminate in a panic attack. Because maladaptive interpretations are viewed as the central element in the cognitive model of panic disorder, they are one of the main targets for treatment according to the cognitive account of PD (e.g. Craske & Barlow, 2008).

Research has supported the view that patients suffering from panic disorder are characterised by an enhanced tendency to misinterpret benign symptoms. For example, in two studies Clark et al. (1997) administered the Body Sensations Interpretation Questionnaire (BSIQ) to patients suffering from panic disorder and controls. The BSIQ measures negative interpretations in four domains, including bodily sensations for which the cognitive theory predicts that these will be more likely misinterpreted by panic disorder patients (e.g. '*You notice that your heart is beating quickly and pounding*'). Each item was followed by the question '*Why ?*', after which participants wrote down the first explanation that came to mind (open-ended question). Next, three alternatives were presented, that had to be rank-ordered for the extent to which '*they would be most likely come to your mind if you found yourself in a similar situation*' (e.g. for the item presented above: *'because you have been physically active*', '*because there is something wrong with your heart*', and '*because you are feeling excited*'). Finally, participants rated the extent to which they actually believed in these

interpretations. Results showed that panic patients were more likely to interpret ambiguous autonomic sensations as signs of immediately impending physical or mental disaster. Moreover, as compared to other anxiety disorder patients and nonpatients, persons suffering from panic disorder were more likely to believe these interpretations.

Similar to the study of Clark et al. (1997), most studies that investigate catastrophic misinterpretations in panic disorder have been based on self-report. It is well-known that there are several limitations to using self-report measures like the BSIQ, such as distortions by memory biases. David Clark and colleagues pointed to another major limitation, which is that these measures "*… explicitly ask patients to make interpretations of bodily sensations, whereas the interpretations that are assumed to happen in panic attacks are more automatic and reflexive in nature*" (Clark et al., 1997, p. 211). To assess the more 'automatic' character of these catastrophic misinterpretations, Clark et al. advocated the use of alternative measures like priming procedures, which bypass self-report and more directly tap into the memory structures that are at the basis of these misinterpretations.

In the same year, Schniering and Rapee (1997) presented a study in which precisely this type of methodology was employed. They used a variant of the associative priming task in which associatively related (e.g. butter-bread) and associatively unrelated (e.g. dust-cats) prime-target pairs were presented. These trials were intermixed with an equal number of trials on which the target was a nonword (e.g. jird). Participants were asked to categorise targets as quickly as possible as a 'word' or a 'nonword' (lexical decision task). To reduce the impact of strategic processes, the stimulus onset asynchrony (SOA; i.e. the interval between the onset of the prime and the onset of the target) was limited to 240 ms. In line with traditional findings, the authors observed that 'word' responses were significantly faster in case the target was preceded by an associatively related prime, as compared to when it was preceded by an unrelated prime. As a crucial test of the catastrophic misinterpretation hypothesis, the authors also included trials on which the prime referred to a bodily sensation and the target to a catastrophic outcome (e.g. breathlessness-suffocate; dizzy-faint). It was hypothesised that if memory representations of bodily sensations automatically activate the

representations of associated fearful outcomes, panic patients should respond significantly faster on these trials as compared to trials on which the target was preceded by an unrelated and nonthreatening prime (e.g. sandman-suffocate; cardboard-faint). A similar effect was not predicted for the non-clinical control group. Even though a significant facilitation effect was observed for the threatening word pairs (as compared to the control trials), Schniering and Rapee (1997) found that this effect was equally strong in the group of panic patients and the nonclinical control group. Hence, the results did not provide support for the cognitive model of panic disorder.

More recently, Teachman, Smith-Janik and Saporto (2007) employed another measure to assess automatic associations between bodily sensations and their catastrophic interpretations. They used a variant of the Implicit Association Test (IAT; Greenwald et al., 1998) in which stimuli were presented that related to bodily changes (heart racing, rapid pulse, sweating, dizzy) or to body parts (arm, leg, shoulder, ear). These stimuli had to be categorised as 'bodily changes' or 'body parts' as fast as possible by pressing one of two keys. In addition, words related to threat (alarming, scary, terrifying, dangerous) or neutral words (e.g. meaningless, trivial) had to be categorised as 'alarming' or 'meaningless'. In line with results typically observed in the IAT, Teachman et al. (2007) predicted that panic patients would perform best if the same key was to be used to categorise stimuli as 'bodily changes' and 'alarming' relative to trial blocks in which the categories 'bodily changes' and 'meaningless' were mapped onto the same response. This was indeed the case. However, contrary to their expectations, no group differences were observed between persons suffering from PD and healthy controls. Similarly, in a previous study in which 'bodily changes' were compared to a category of 'weather changes', also no group differences were observed, while the 'panicked'/'me' IAT did produce the expected results (Teachman, 2005). In this latter study, groups of participants scoring high and low on Anxiety Sensitivity were compared, a concept which is proposed to be of central importance in panic disorder (Schmidt, Lerew, & Jackson, 1997).

Finally, also Schneider and Schulte (2007) failed to find differences between a group of panic patients and a group of nonclinical controls when they were compared for a fixed set of prime sentences and target words. In this study participants named catastrophic (e.g. infarction) and neutral target words (e.g. holiday) which were preceded by prime sentences that described an anxiety symptom (e.g. 'You notice an increasing palpitation') or a neutral situation (e.g. 'You notice an increasing drum of rain'). Prime sentences either preceded the target immediately (inter stimulus interval, ISI: 0 ms) or after 1500 ms. The expected advantage – for the panic group – in naming latencies for catastrophic targets preceded by 'symptom primes' was not observed.

The whole of these data fails to provide support for the view that panic disorder is characterised by automatic or reflexive catastrophic misinterpretations of bodily symptoms. Schniering and Rapee (1997) actually discussed the possibility that when experiencing palpitations, *everyone* might consider an equivalent probability that this symptom signals a coronary, but that panic patients attach a greater cost to coronaries than other individuals (p. 568). This would mean that persons suffering from panic disorder are not characterised by an enhanced tendency for spontaneous catastrophic misinterpretations, but do differ from others with respect to the valence associated with these negative outcomes (or the extent to which they believe they can control them). If so, this would be of consequence for the cognitive model of panic disorder.

Another possibility that has been offered is that associations between bodily sensations and certain misinterpretations are so specific and idiosyncratic, and are limited in number, that averaging over fixed sets of word pairs fails to reveal differences between groups of panic patients and nonclinical controls. In the study by Schneider and Schulte (2007) this possibility was investigated. Although priming effects that were calculated on the basis of all trials were similar in panic patients and controls (see above), the authors did find the expected group differences when priming effects were calculated for ideographically selected stimuli. To this end, participants were asked, at completion of the experiment, to explicitly judge as quickly as possible the associative relatedness of all prime-target pairs that

were employed in the priming procedure. Assuming that associative strength in memory should be reflected in faster decision times for prime-target pairs that are idiosyncratic associates, trials consisting of stimulus pairs that were responded to fastest were selected for further analyses Consistent with cognitive models of PD, these analyses now revealed the expected difference (at ISI 0 ms) between the panic group (who showed a 20 ms facilitation effect) and the nonclinical control group (who showed a -22 ms inhibition effect). According to the authors, these findings are in line with the idea that panic patients are effectively characterised by automatic catastrophic interpretations, but that the nature of these interpretations is so idiosyncratic that they cannot be picked up when effects are calculated across a large set of generic stimuli.

From a clinical perspective it makes sense to select idiographic stimulus pairs. Whereas some panic patients strongly focus on one type of catastrophe (e.g. heart attack), other patients will fear calamities of a different kind (e.g. brain tumour). This does not take away, however, that such a highly idiosyncratic dependency is not often observed for other types of information processing biases within the study of panic disorder (e.g. attentional bias, Maidenberg, Chen, Craske, Bohn, & Bystrwski, 1996) or in the study of other disorders (e.g. OCD). In fact, it is common practice to average over sets of stimuli which are assumed to be 'overall' relevant for the participant group under investigation. Also, it remains somewhat unclear why control participants did not exhibit a similar associative priming effect for the panic-related stimulus pairs for which the selection task had indicated that these were probably highly associatively related (and actually demonstrated an opposite trend; -22 ms). Hence, further replication and extension of these interesting findings is justified.

In this context, data from an experiment reported by Lefaivre, Watt, Stewart, and Wright (2006) can also be taken as support for the catastrophic misinterpretation hypothesis. In this study, participants who scored high or low on anxiety sensitivity completed an Extrinsic Affective Simon Task (EAST; De Houwer, 2003). In addition to bodily symptoms (e.g. dizzy, breathless, palpitation), stimulus words included threatening (e.g. faint, suffocate, heart attack) and positive health outcomes (e.g. benign, healthy, innocuous). Similar to the study by

Teachman et al. (2007) body parts (e.g. ankle, elbow, nose) were used as a contrast category for the category of bodily symptoms. Results indicated that high anxiety sensitive individuals were significantly faster on trials where target words related to anxiety symptoms were mapped on to the same response key as threatening consequences. No significant difference in performance was found for low anxiety sensitive individuals or when target words were body parts (Lefaivre et al., 2006, p. 295). These findings are consistent with the idea that persons who score high on anxiety sensitivity automatically associate anxiety-related symptoms with harmful consequences. Given the proposed central position of the anxiety sensitivity concept within panic disorder (McNally, 2002), these findings might also speak to this latter condition. A limitation of this study, however, was the small number of participants (high anxiety sensitivity: n= 10; low anxiety sensitivity: n=12).

In conclusion, we can state that spontaneous catastrophic misinterpretations of bodily sensations are assumed to be a hallmark of panic disorder. Several studies provide support for this cognitive view. However, most of these studies are based on self-report. According to Clark et al. (1997), one of the limitations of these procedures is that patients are explicitly asked to make interpretations, whereas the interpretations that are assumed to happen in panic attacks are more automatic and reflexive in nature. Indirect measures, such as priming paradigms would provide a good alternative in this context. Unfortunately, as we have discussed, results from studies that have employed such behavioural procedures have been far from clear-cut.

The current status of this line of research is such that there are still a number of questions that need to be addressed empirically. First of all, given the conflicting data that are available, additional research will need to address the main issue of whether panic disorder is truly characterised by spontaneous (automatic) catastrophic misinterpretations of bodily symptoms. Also, it remains to be examined whether possible effects are indeed highly idiosyncratic, as suggested by the data of Schneider and Schulte (2007), or whether more generic effects can be observed more generally for sets of stimuli (as suggested by the data of Lefaivre et al., 2006). Finally, a question that has not yet been investigated in this context

is whether potential effects of automatic misinterpretations are specific to panic disorder (i.e. not a characteristic of anxiety disorders in general). This issue has received extensive attention in studies employing self-report measures (e.g. Clark et al., 1997; Harvey, Richards, Dziadosz, & Swindell, 1993), where 'anxious control groups' were included (i.e. patients suffering from anxiety disorders other than panic disorder). Such comparisons have, however, not been made within studies employing paradigms that circumvent introspection. Hence, it is an open question whether findings like those reported by Schneider and Schulte (2007) are truly specific for panic disorder.

In an attempt to answer these questions, we investigated automatic catastrophic misinterpretations in a group of persons suffering from panic disorder (n=31), an anxious control group (n=25), and a nonanxious control group (n=30). An associative priming procedure was used (lexical decision task; SOA 250 ms). To maximize the chance of observing the predicted priming effects, all participants from the two clinical groups were included at a moment on which they were actively seeking treatment but did not yet enter the treatment programme.

Experiment 1

Method

Participants

Three groups participated in this study: a panic group, an anxious control group and a nonanxious control group. The *Panic Group* (PG) consisted of 34 outpatients who met DSM-IV criteria for panic disorder. Due to loss of data, three participants were excluded from the analyses. These participants had misunderstood the instructions, because of which a significant proportion of data were lost. The final sample consisted of 31 patients (16 women) with a mean age of 43.4 years (*SD* = 12.1; range = 20-64). In this group there were 19 patients who showed at least one form of comorbidity (eleven patients with one comorbid disorder, seven with two comorbid disorders and one patient with three comorbid disorders). In total, there were eleven patients with comorbid depression, five patients with comorbid

hypochondriasis, four patients suffering from benzodiazepine or alcohol dependence, four patients with social phobia, two patients with specific phobia, and one patient suffering from GAD. The *Anxious Control Group* (ACG) consisted of 25 outpatients (19 women) who were diagnosed with an anxiety disorder other than panic disorder (or agoraphobia). Of these, 35% had obsessive-compulsive disorder, 23% social phobia, 38% specific phobia, and 4% generalized anxiety disorder as their primary diagnosis. Their mean age was 36.5 years (SD = 13.6; range = 18-59). All participants from the panic group and the anxious control group were seeking treatment at the Academic Anxiety Center in Maastricht (at the time of testing treatment had not yet started). Diagnoses were based on a semi-structured interview (Mini International Neuropsychiatric Inventory, MINI; Lecrubier et al., 1997) conducted by a psychiatrist or psychologist. The *Nonanxious Control Group* (NCG) consisted of 30 healthy volunteers (12 women) who were recruited by advertisement. Their mean age was 42.7 years (SD = 11.3; range = 22-59). None of the participants in the NCG currently suffered from an anxiety disorder (or any other psychiatric disorder) and had no history of prior psychiatric problems.

Materials

In addition to the priming procedure, participants were assessed by means of the following instruments:

Panic and Agoraphobia Scale (PAS; Bandelow, 1995). The PAS is a 13-item clinical interview to assess the severity of panic disorder and agoraphobia. Previous work has demonstrated that the PAS has satisfactory values for internal consistency ($\alpha = .86$), test-retest reliability and correlations with other anxiety scales.

State Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Trait anxiety was measured using the Dutch version of the STAI. It has good validity and reliability ($\alpha > .90$) (Van der Ploeg, Defares, & Spielberger, 1980).

Montgomery–Åsberg Depression Rating Scale (MADRS; Montgomery & Åsberg, 1979). The MADRS is a ten-item clinical rating scale to assess severity of depressive

symptoms. Previous research showed that the MADRS is characterized by excellent internal consistency (Carmody et al., 2006). We used the Dutch version by Hartong and Goekoop (1985), which has an internal consistency of .91.

Self-rating Depression Scale (SDS; Zung, 1965). The SDS is a self-report measure of depression consisting of 20 items, with a four-point scale ranging from 'a little of the time' (1) to 'most of the time' (4). Research supports the validity of this measure (Schaefer et al., 1985).

Prime-target word pairs. Four sets of word pairs were used in the priming task. In addition to the crucial Panic-Panic (PP) word pairs there were three types of control pairs: Neutral-Neutral (NN), Panic-Neutral (PN) and Neutral-Panic (NP). The Panic-Panic prime-target word pairs consisted of a bodily symptom that is typically experienced during a panic attack (e.g. breathlessness) and the catastrophic misinterpretation that is related to this symptom (e.g. suffocate). Bodily symptoms were always used as prime, misinterpretations as target. Ten PP word pairs were created. Some of the word pairs were selected from work by Clark (1988) (e.g. dizziness – fainting); other word pairs were inspired by clinical phenomenology of panic disorder and previous research (Chambless, Beck, Gracely, & Grisham, 2000) (e.g. headache – brain tumor). All primes and targets were single Dutch words. An overview of all prime-target word pairs is provided in the Appendix. The Neutral-Neutral control pairs consisted of neutral, panic-unrelated words that were considered to be somewhat associatively related (e.g. flowers – picking; television – newsflash). Primes and targets from the NN list were matched for word length with those of the PP list. The two remaining sets of control pairs consisted of a neutral prime followed by a panic target (NP; e.g. flowers – stroke) or a panic prime followed by a neutral target (PN; e.g. headache – install). Like for the PP pairs, the three sets of control stimuli consisted of ten prime-target pairs (see Appendix).

Nonword targets. In addition to the ten neutral and the ten panic targets, twenty pronounceable nonwords were used as targets in the lexical decision task (e.g. preester; farnasie). In addition to 8 nonwords taken from Hermans, Smeesters, De Houwer, and Eelen

(2002), twelve additional nonword targets were created. Length of the 'nonword' targets matched word length of the 'word' targets (neutral; panic).

During the priming task, the presentation of all stimuli, as well as the registration of all responses was controlled by an Affect 3.0 program (Spruyt, Clarysse, Vansteenwegen, Baeyens, & Hermans, in press). This program was run on an AMD Athlon personal computer. Responses to targets (word/nonword) were recorded using two keys of the keyboard. For the rating of the word pairs, a tablet PC with touch screen was used (1027×748 pixel resolution).

Procedure

Before the start of the actual study, all participants were screened for presence (panic group) or absence (anxious and nonanxious control groups) of panic disorder (and/or agoraphobia). Participants in the two clinical groups filled out the MADRS and SDS as a part of a larger clinical assessment. The PAS was assessed in this context as well.

At the start of the experiment[1], participants were informed about the study and signed the informed consent form. Instructions for the priming task were presented on the computer screen. The participant was informed that words would be presented and that the task consisted of categorising each word as an 'existing' or a 'non-existing' word by pressing one of two keys on the keyboard. Half of the participants pressed the left button for 'words' and the right button for 'non-words'. For the other participants this assignment was reversed. The experiment started with a practice phase (20 trials), during which participants were trained in the classification task. Only target words were presented during this phase. Each trial started with the presentation of a fixation cross during 500 ms. Five hundred milliseconds after termination of the fixation cross, the target word was presented. Targets were selected randomly from the larger set of targets (see above). For half of the practice trials the target was a word; for the remaining trials this was a non-word. Targets disappeared from the screen upon response of the participant.

After the practice phase, 80 experimental trials were presented. Each of the ten prime-target pairs within each of the four trial types (PP, PN, NP, NN) was presented once

(see Appendix). In addition, all of the primes were presented once with a nonword target. As a result, each of the 10 panic primes was followed by a panic target on 10 trials, by a neutral target on 10 trials, and by a nonword target on twenty trials. The same was the case for the neutral primes. Assignment of nonword targets to primes was random.

Each of the 80 experimental trials started with the presentation of a fixation cross during 500 ms. Five hundred milliseconds after termination of the fixation cross, the prime word was presented for 200 ms. This was followed by an inter-stimulus interval of 50 ms, after which the target was presented (resulting in a stimulus onset asynchrony of 250 ms). Targets remained on the screen until a response was recorded. In case of no response, the target disappeared after 2000 ms. As was the case for the practice trials, participants categorised each target as a 'word' or a 'nonword' by pressing one of two buttons on the keyboard. The inter-trial interval (ITI) ranged between 1000 and 2000 ms, with a mean of 1500 ms. The order of all trials was randomized for each participant.

In addition to the priming task, a subgroup of 20 panic patients and 20 anxious controls also rated the 20 word pairs (panic-panic; neutral-neutral) for the extent to which they elicited fear. This was included as an examination of the validity of the word pairs (panic-related and control) that were included in this study. It was hypothesised that the panic-related word-pairs (e.g. *breathlessness - suffocate*) would elicit significantly more fear in the panic group as compared to the anxious control group; whereas no differences were predicted for the control pairs. During this rating phase, each of the 20 word pairs (see Appendix) was presented once on the computer screen. Presentation duration was 8 seconds, with an ITI that ranged between 12 and 20 s. After each presentation, the participant was asked to indicate the level of fear experienced during the presentation of the word pair, on a scale ranging from 0 ('*no fear at all*') to 100 ('*the worst imaginable fear*'). The level of fear was marked by tipping with a stylus the correct position on a 20 cm x 1 cm horizontal visual analogue scale (for more details of this electronic VAS, see Van Duinen, Rickelt, & Griez, 2008). Half of the participants completed the ratings before the priming task; for the other participants the order was reversed.

Results

Data reduction

The data from priming trials on which no response (2.64 %) or a wrong response (2.03 %) was given, were excluded from all analyses. To reduce the impact of outlying values, we also excluded all response latencies (0.64 %) that deviated more than 2.5 standard deviations from a participant's mean latency in a particular priming condition (see Ratcliff, 1993). Subsequently, for each participant and for each experimental condition, mean response latencies were calculated. The means for the three types of control trials (PN, NP, NN) were averaged. Mean response latencies were analysed using a 3 x 2 analysis of variance, with group (panic / anxious controls / nonanxious controls) as a between-subjects variable, and trial type (panic / control) as a within subjects variable.

For the fear ratings, t-tests were conducted for each of the 20 word pairs, comparing the scores of the panic group with the scores of the anxious control group.

Clinical instruments

As expected, the panic group had significantly higher scores on the Panic and Agoraphobia Scale (M = 24.7; SD = 10.1), as compared to the anxious control group (M = 9.12; SD = 10.3), $t(54) = 5.68$, $p < .001$. The two groups did not differ on trait anxiety (STAI-trait), M_{panic} = 54.5, SD = 9.2, $M_{anxious\ controls}$ = 48.7, SD = 14.2, $t(54) = 1.85$, *n.s.*. Similarly, both groups did not differ in their level of depression, as indicated by the scores on the SDS, M_{panic} = 46.6, SD = 9.3, $M_{anxious\ controls}$ = 44.0, SD = 11.9, $t(54) = 0.89$, *n.s.*, and the MADRS, M_{panic} = 13.3, SD = 7.2, $M_{anxious\ controls}$ = 12.6, SD = 11.1, $t(54) = 0.25$, *n.s.*. These results indicate that in spite of equal levels of depression and general level of anxiety, the panic group had substantially higher levels of symptomatology related to panic and agoraphobia.

Ratings

When all ten panic-panic word pairs were included, as expected, markedly higher fear scores were expressed by the panic group (M = 44.7; SD = 24.7) as compared to the anxious control group (M = 20.8; SD = 16.7), $t(38) = 3.58$, $p < .001$. For the ten neutral word pairs, low fear

scores were obtained for both the panic group (*M* = 10.7; SD = 11.8) and the anxious control group (*M* = 5.1; *SD* = 5.8). Even though both groups did not statistically differ in their ratings of the neutral word pairs, the difference did approach significance, $t(38) = 1.89$, $p = .067$. Closer inspection revealed that for one particular neutral word pair (*groceries – shopping*), the panic group experienced significantly more fear, $t(38) = 2.36$, $p < .05$, $M_{panic} = 28.0$, $M_{anxious\ controls} = 9.9$. In retrospect, this word pair was probably strongly related to the agoraphobia component in the panic group. Hence, it was decided to omit the data related to this word pair from the priming analyses. For the other nine pairs, there were no reliable differences in fear ratings between the two groups. When the rating data were re-analysed after removing the 'groceries – shopping' item, the two groups no longer differed in their fear ratings for the neutral items, $t(38) = 1.52$, $p = .14$, $M_{panic} = 8.7$, $SD = 11.0$, $M_{anxious\ controls} = 4.6$, $SD = 5.5$.

Priming data

The response latencies were analysed using a 3 (group: panic / anxious controls / nonanxious controls) x 2 (trial type: panic / control) analysis of variance with repeated measures for the last variable. This revealed a significant main effect of trial type, $F(1, 83) = 17.6$, $p < .0001$, $MSE = 27651$. Overall, responses were significantly faster for Panic-Panic trials (*M* = 672; *SD* = 115) as compared to the control trials (*M* = 699; *SD* = 128). As predicted, this main effect was qualified in a reliable Group x Trial type interaction, $F(2, 83) = 3.2$, $p < .05$, $MSE = 1806$. Follow-up analyses showed a significant main effect of trial type in the panic group, $F(1, 83) = 21.0$, $p < .0001$, $MSE = 1806$. Response latencies for panic-panic trials (*M* = 687; *SD* = 106) were significantly shorter as compared to the control trials (*M* = 736; *SD* = 112). A similar priming effect was not observed in the anxious control group, $F(1, 83) < 1$, $MSE = 1806$, $M_{panic} = 668$ ($SD = 99$), $M_{control} = 679$ ($SD = 115$). Based on these results, the priming task thus clearly supports the assumption that prime stimuli that are related to bodily sensations automatically activate threatening interpretations in panic patients, while a similar phenomenon is absent in anxious controls who have no history of

panic disorder and who showed comparable levels of trait anxiety and symptoms of depression.

Quite surprisingly, however, contrast analyses revealed that for the nonanxious control group a significant priming effect was observed as well, $F(1, 83) = 4.11$, $p < .05$, $MSE = 1806$. Analogous to the panic group, this group responded faster to panic-panic trials (M = 660; SD = 137) than to control trials (M = 682; SD = 148). Because this priming effect was unexpected, further post-hoc analyses were conducted.

Post-hoc analyses for the nonanxious control group (priming data)

Even though the priming effects for the panic group ($F = 21.0$) and the anxious control group ($F<1$) were completely in line with predictions, it was striking to observe a significant priming effect for the nonanxious controls. Further inspection of this finding revealed a possible explanation in terms of participant characteristics. As a matter of fact, based on recruitment, this nonanxious control group largely consisted of two subgroups. Sixteen of these 30 participants were volunteers with no affiliation to the mental health profession. In contrast, the other 14 participants were professionals working within the health services (i.e. the academic anxiety clinic). Among others (e.g. a secretary), this group included persons who were working as a psychiatric nurse, psychologist or social worker, or who were students doing an internship as part of psychology-related education. Even though these persons were not currently suffering from panic disorder and had no history of panic or agoraphobia, it could well be argued that due to their education or occupation they had built up a knowledge base concerning panic symptoms and related catastrophic interpretations. It might be argued that this knowledge, which is rooted in long-term memory as part of professional expertise, could serve as a basis for priming effects similar to those observed in the panic patients.

To test this post-hoc hypothesis, the priming effect was analysed for both subgroups separately. For the volunteers who were not associated with the hospital, the priming effect was absent, $F(1, 82) < 1$, $MSE = 1806$, $M_{panic} = 680$ (SD = 133), $M_{control} = 692$ (SD = 156). For

the subgroup of ‘mental health professionals’, however, the priming effect was significant, $F(1, 82) = 4.47$, $p < .05$, $MSE = 1806$, $M_{panic} = 638$ ($SD = 143$), $M_{control} = 672$ ($SD = 143$).

Discussion

In this study an associative priming task was employed to assess automatic associations between bodily symptoms and catastrophic misinterpretations in panic disorder. As an improvement over previous studies, the panic group was compared to an anxious control group to test the specificity of the effect. In line with predictions from the cognitive model, facilitated responses were observed for the crucial panic trials in the panic group, but not in the group of anxious controls. To our knowledge, this is the first demonstration of (a) an automatic interpretation effect in a clinical panic group using unselected stimuli, and (b) of the fact that this finding is selective to patients suffering from panic disorder (i.e. not observed in a clinically anxious control group that does not suffer from panic disorder). Post-hoc analyses further revealed that akin to the anxious control group, no priming effect was observed in a group of persons who had no current or previous history of anxiety disorders, and who were not professionally connected to mental health services.

An intriguing finding, however, was that a panic-misinterpretation effect was also revealed in a group of health professionals without history of panic disorder. In this group, presentation of primes related to ‘bodily symptoms’ automatically activated representations of catastrophic outcomes. Our interpretation of this finding is that for this latter group the ‘symptom-catastrophe’ associations are part of professional knowledge. This observation is of theoretical and clinical importance, because it seems to indicate that automatic catastrophic interpretations do not necessarily point to ‘pathological mechanisms’. Most probably such priming effects are a mere index of the extent to which these concepts are related in memory (i.e. associative strength). This associative strength can be influenced by a number of factors, among which the amount of previous exposure to the contiguous presentation of these concepts. This can be part of one’s own personal illness history (e.g. in the case of panic patients) or as part of educational or professional experiences (e.g. in the

case of the health professionals). However, because of the post-hoc nature of these arguments, these results need replication before firm conclusions can be drawn. In Experiment 2, we therefore selected four groups of participants on an a priori grounds. In addition to a group of panic patients, and anxious control group, two nonclinical control groups were included: one group consisted of mental health professionals and one group consisted of participants who were not professionally (or otherwise) associated with mental health services. Based on the findings of Experiment 1, we predicted significant priming effects for the panic group and the health professionals, but not for the anxious control group and the nonclinical control group people outside the mental health profession.

Experiment 2

Method

Participants

Four groups participated in this study. In addition to the panic group and the anxious control group, two nonanxious control groups were included. The first consisted of volunteers who were mental health professionals (in training), and who worked with anxiety patients on a daily basis. The second group was recruited by advertisement and had no professional or educational background relating to anxiety disorders or therapeutic work in general.

The *Panic Group* consisted of 20 outpatients (11 women) who met DSM-IV criteria for panic disorder. Their mean age was 40.7 years (*SD* = 13.5; range = 20-65). In this group, there were eleven patients who showed at least one form of comorbidity (six patients with one comorbid disorder, five with two comorbid disorders). In total, there were nine patients with comorbid depression, two patients with comorbid hypochondriasis, one patient suffering from benzodiazepine dependence, one patient with social phobia, one patient suffering from PTSD, and two patients suffering from GAD. The *Anxious Control Group* consisted of 20 outpatients (9 women) who were diagnosed with an anxiety disorder other than panic disorder (or agoraphobia). Of these, 30% had OCD, 25% social phobia, 35% specific phobia, 10% generalized anxiety disorder as their primary diagnosis. Their mean age was 41.3 years (SD

= 15.1; range = 22-61). All participants from the panic group and the anxious control group were seeking treatment at the Academic Anxiety Center in Maastricht (at the time of testing treatment had not yet started). Diagnoses were based on a semi-structured interview (Mini International Neuropsychiatric Inventory, MINI; Lecrubier et al., 1997) conducted by a psychiatrist or psychologist.

The group of nonanxious controls consisted of 15 '*professionals*' (9 women; mean age = 37.4, SD = 11.7; range = 23-55) and 15 '*non-professionals*' (8 women; mean age = 39.1, SD = 13.7; range = 22-64) and were matched for age with the two anxious groups. None of the participants in the two nonanxious groups currently suffered from an anxiety disorder (or any other psychiatric disorder) and had no history of prior psychiatric problems.

Materials and procedure

Materials (including questionnaires) and procedure were identical to Experiment 1, with the exception of a small amendment in the stimulus materials for the priming task. As was already noted, one of the neutral control pairs in Experiment 1 (i.e. 'groceries – shopping') induced significantly more fear in the panic group as compared to the anxious control group, and could thus not be regarded as an appropriate control stimulus. Because of that reason, it was decided to exclude this word pair (and its combinations in PN and NP trials) from the present experiment. To balance the stimulus list, it was decided to delete one of the ten panic-panic pairs as well. For the word pair '*headache – brain tumour' the* ratings that were obtained in Experiment 1 were in the expected direction, M_{panic} = 35.0 (*SD* = 33.5), $M_{anxious\ controls}$ = 26.6 (*SD* = 26.8), but the difference failed reach the level of significance, $t(38) = 0.87$, $p = .39$. By deleting this word pair from the set, the present priming task was based on 9 panic-panic pairs and 9 neutral pairs (and their combinations in panic-neutral and neutral-panic). As a result, there were 36 trials on which the prime was followed by a word target (9 PP, 9 PN, 9 NP and 9 NN), and 36 trials on which the same primes were followed by a nonword. Because of the reduction in number of word pairs as compared to the previous

study, it was decided to double the number of trials. As a result, for the 'word' trials, there was a total of 72 trials, of which 18 PP, 18 PN, 18 NP and 18 NN.

Results

Data reduction

The data from priming trials on which no response (0.44 %) or a wrong response (1.90 %) was given, were excluded from all analyses. To reduce the impact of outlying values, we also excluded all response latencies (2.34 %) that deviated more than 2.5 standard deviations from a participant's mean latency in a particular priming condition. Subsequently, for each participant and for each experimental condition, mean response latencies were calculated. As in Experiment 1, the means for the three types of control trials (PN, NP, NN) were averaged.

Clinical instruments

The pattern of results for the questionnaires was almost identical to that of Experiment 1. The panic group scored significantly higher on the Panic and Agoraphobia Scale (M = 25.6; SD = 10.3), as compared to the anxious control group (M = 4.5; SD =7.9), $t(38) = 7.22$, $p < .001$. The two groups did, however, not differ on trait anxiety (STAI-trait), M_{panic} = 51.4 (SD = 10.7), $M_{anxious\ controls}$ = 46.1(SD = 15.1), $t(38) = 1.28$, *n.s.*. Similarly, both groups did not differ in their level of depression, as indicated by the scores on the SDS, M_{panic} = 47.3 (SD = 11.3), $M_{anxious\ controls}$ = 40.7(SD = 10.7), $t(38) = 1.89$, *n.s.*, and the MADRS, M_{panic} = 14.9 (SD = 7.6), $M_{anxious\ controls}$ = 10.6 (SD = 9.1), $t(38) = 1.62$, *n.s.*. These results indicate that with equal levels of depression and general level of anxiety, the panic group had substantially higher levels of symptomatology related to panic and agoraphobia.

Priming data

Because of our a priori hypotheses, contrasts between panic trials and control trials were calculated for each of the four groups of participants separately. It was predicted that for the

panic patients and the mental health professionals there would be a significant priming effect. No such effect was predicted for the anxious control group and the healthy control group. As hypothesised, panic patients responded significantly faster on panic-panic trials (M = 685; SD = 157) as compared to control trials (M = 721; SD = 143), $F(1, 66) = 8.83$, $p < .005$, MSE = 1498. A similar effect was observed for the mental health professionals, $F(1, 66) = 5.31$, $p < .05$, MSE = 1498, M_{panic} = 631 (SD = 122), $M_{control}$ = 664 (SD = 136). For the anxious control group, there was no significant difference between panic-panic trials (M = 716; SD = 134) and control trials (M = 732; SD = 131), $F(1, 66) = 1.81$, *n.s.*, MSE = 1498. Similarly, for the healthy control group, responses to panic trials (M = 642; SD = 119) did not differ from responses to control trials (M = 656; SD = 132), $F(1, 66) = 1.04$, *n.s.*, MSE = 1498.

General Discussion

In two studies, we tested whether panic disorder is characterised by a spontaneous (automatic tendency) to interpret benign bodily symptoms in a catastrophic fashion. To circumvent the disadvantages of self-report measures, we employed an associative priming task in which participants had to provide lexical decisions (word vs. nonword) for the targets. On the crucial panic-panic trials, primes were words referring to a bodily symptom (e.g. breathlessness), while the target words referred to a threatening outcome (e.g. suffocate). Control trials consisted of a symptom prime followed by an unrelated neutral target (e.g. palpitations – picking), a neutral prime followed by a catastrophic outcome (e.g. flower – stroke), or a neutral prime and a somewhat related neutral target (e.g. flower – picking). It was predicted that in a group of patients suffering from panic disorder responding should be significantly faster for panic-panic trials as compared to control trials, while no such effect was predicted for a standard nonclinical control group. This is exactly what was observed in Experiments 1 and 2, supporting the cognitive model of panic disorder. To our knowledge, this is the first time that the catastrophic misinterpretation hypothesis was confirmed in a clinical group using a procedure that did not rely on self-report. In several previous studies similar effects could not be observed (e.g. Schniering & Rapee, 1997; Teachman et al., 2005,

2007) or only when the crucial panic stimuli were selected on an individual basis (Schneider & Schulte, 2007). We will return to these discrepancies in a moment.

In addition to testing whether panic disorder is characterised by spontaneous catastrophic misinterpretations, we also investigated whether this effect is indeed 'specific' to panic disorder. No previous behavioural studies had included an anxious control group, so conclusions about specificity were awaiting further testing. In both experiments no priming effects were observed for panic-panic pairs in the anxious control group. This group was equal in all respects to the experimental group, except for the fact that they had no current (or history of) panic disorder. Like the panic group they sought treatment at the same academic treatment facility, but for a variety of other disorders including OCD, social phobia and generalised anxiety disorder. The fact that this group did not reveal the spontaneous misinterpretations that were observed in the panic group is consistent with the idea that this bias is specific to panic disorder. A similar question has been raised in studies employing self-report questionnaires (e.g. Harvey, 1993; McNally & Foa, 1987), which led to the conclusion that panic patients rate negative interpretations of panic body sensations as more likely to come to mind, and as more believable, than patients with other anxiety disorders, and thus seems to represent a specific feature of panic disorder (Clark et al., 1997, p. 209). The present studies further support the idea of selective and spontaneous negative interpretations.

Even though panic-panic pairs and control pairs were matched for word length, these pairs were not matched for 'associative strength', as the necessary Dutch word association norms were not available for all included stimuli. Future research could take this into account. In any case, even if the pair-types were not equal on associative strength, this does not undermine the crucial interaction with diagnostic group. Future research could also include one additional type of control stimulus. In the present experiments, panic-panic trials were compared to panic-neutral, neutral-panic and neutral-neutral trials. It would, however, also be interesting to include panic-panic pairs that are combinations of non-matching symptoms and outcomes. For instance, '*breathlessness – brain tumor*' and '*headache – suffocate*' could be

used as controls for the experimental pairs '*breathlessness – suffocate*' and 'headache – *brain tumor*'. This would inform us whether the automatic priming of threatening outcomes is specific to the presented primes, or whether the presentation of a specific bodily symptom can activate a whole range of threatening outcomes. A final possible limitation of the studies presented here, is that a 'categorical' approach was followed, rather than a dimensional. The priming effect was studied as a function of diagnostic group, irrespective of severity of the panic complaints. It would be interesting to learn whether priming effects as those observed in Experiments 1 and 2, are indicative of the severity of the panic disorder. Some preliminary analyses based on the Panic and Agoraphobia Scale, indicate that the severity of the complaints in the panic group indeed reliably correlated with an overall index of panic priming (i.e. $RT_{control} - RT_{panic\text{-}panic}$) in Experiment 1, $r(31) = .41$, $p < .05$. A similar correlation was, however, not present in Experiment 2. Also, no similar correlations were observed for the anxious control group.

An important, but unexpected finding that emerged in Experiment 1 was that a significant priming effect was observed in a subgroup of the nonclinical control condition. This group of mental health workers displayed a reliable facilitation effect for panic-panic trials which was comparable to that of the panic group in direction and size. Because of the post-hoc nature of this finding, four groups were included on an a priori basis in Experiment 2. The results of Experiment 1 were replicated: significant priming effects were observed in the panic group and the nonclinical group of health professionals, but not in the anxious control group and the nonclinical control group that was unrelated to health care. We believe that these findings suggest that there might be several sources for the accessibility of threatening interpretations. One source can be found in the illness history of panic patients. When bodily symptoms are repeatedly and consciously interpreted in a threatening fashion, these negative interpretations will become more and more accessible for activation. Alternatively, the origin and accessibility of these interpretations might also stem from the training and day to day professional activities in mental health workers such as psychologists and psychiatrists.

We believe that this observation has at least two important implications. The first is a methodological one. If education, training and professional experience contribute to the availability of certain cognitive representations and to the accessibility of certain associations, the decision to include groups like psychology students or mental health professionals should be weighed with great care in future experiments. In the introduction we already referred to a series of studies in which no group differences were observed with respect to spontaneous interpretation biases (e.g.Schniering & Rapee, 1997; Teachman, 2005; Teachman et al., 2007). There can be two explanations for the absence of such group differences. The classic analysis is that the panic group is not characterized by selective interpretations. An alternative explanation, however, should be considered, which is that the control group showed a bias similar to the panic group. In the study of Teachman et al. (2007) spontaneous associations between 'bodily sensations' and 'alarming' did not differentiate between panic patients and healthy controls. The control group in this study, however, was partly recruited through the psychology participant pool. It might well be that the absence of differentiation between the two groups was due to relatively strong panic associations in the group of psychology students (as a result of their education). The results of a previous study from the same lab (Teachman, 2005) can be interpreted in a similar way. In this experiment participants were all psychology students.

A second implication of our findings is a more theoretical one and relates to the causal status of the spontaneous symptom-threat associations with respect to panic. If such selective threat associations can be observed in panic-free participants to the same extent as they are observed in panic patients, this at least raises doubts about these associations being a sufficient basis for increases in anxiety (or even the origin of panic attacks). One possibility is that not the accessibility of these threatening interpretations is of crucial importance, but their believability or the extent to which they are embedded in a representational structure of threat-related psychophysiological and motor responses (Lang, 1988). In other words, for both groups the word 'breathlessness' will automatically bring to mind the representation 'suffocate', but only for the panic group this will activate a related network of fear-responses

(e.g. avoidance tendencies, high arousal, increased blood pressure, …). In mental health professionals and psychology students the activation of the memory representation of 'suffocation' will not go hand in hand with an activation of these responses and response tendencies. Future research could aim at testing this view. We would predict that panic patients and mental health professionals would show similar priming effects, but that the same word pairs would lead to increased skin conductance responses in the panic group, as well as to avoidance tendencies as measures by tasks such as the Approach-Avoidance Test (Heuer, Rinck & Becker, 2007). Further research is also necessary to unravel the role of the accessibility aspect of threat representations. In this context, a recent study by Schneider and Schulte (2008) is highly relevant. Using a priming paradigm, these authors demonstrated that stronger associations between idiographically related anxiety symptoms and catastrophes predicted a smaller reduction in anxiety sensitivity in panic patients in response to a brief cognitive-behavioural treatment (Schneider & Schulte, 2008, p. 568).

A final element for discussion concerns the observation that group differences in panic priming only emerged after idiographical selection of prime-target pairs in the study of Schneider and Schulte (2007) whereas reliable differences emerged in our studies for an unselected set of prime-targets. To maximize the chance of observing the predicted (differences in) priming effects in the present two studies, all participants from the clinical groups were included on a moment at which they had been actively seeking treatment but did not yet enter the treatment programme. It was assumed that relevant associations between bodily sensations and negative outcomes would be more accessible at that time. However, in the study of Schneider and Schulte (2007) patients were included in a similar fashion. Possible differences in the results might relate to the types of stimulus materials use or the duration of prime presentations. In our studies primes consisted of prime words (e.g. breathlessness), whereas in the study of Schneider and Schulte (2007) prime sentences were employed (e.g. 'Your breath seems to you to be obstructed'). Another element, however, might be that some of the control sentences in the study of Schneider were actually emotional opposites of the threatening primes (e.g. 'Your breath seems to you to be *freed*'; 'In your

chest, you sense more and more clearly *relief*'; 'Your whole body is being gripped by a sensation of *calm*'). It is well possible that these sentences automatically activated the threatening opposites (for the examples above: obstructed, tightness and agitation). This is reminiscent of findings by Mathews and Klug (1993) who demonstrated that emotional Stroop effects in anxious persons are observed for words related to anxiety, irrespective of their emotional meaning. In their study, a word like 'relaxed' delayed colour naming as much as words like 'anxious'. In a similar fashion, it is possible that a selective panic priming effect in the Schneider study was not observed for the panic group because some of the control trials actually 'acted' as panic-panic trials and hence obscured the hypothesised difference between the two types of trials. The idiographical selection procedure might then have resulted in significant group differences, not so much because the panic-trials were more tailored to the individual, but because the 'inappropriate' control trials were filtered away (i.e. because the categorisation as 'unrelated' for these intended unrelated pairs took longer than for other pairs). Of course, future research will need to focus on this possibility in a more direct way. In any case, this study illustrates how subtle factors that are not the focus of a study, such as the nature of the control stimuli and the selection of control participants, can strongly influence results.

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Footnotes

1. In addition to the priming task, participants also completed two other tasks that were unrelated to the present research questions. The order of these tasks was counterbalanced.

Appendix: Stimuli for Experiment 1 (original Dutch words with English translations)

Panic-Panic		Neutral-Neutral	
ademnood – stikken	(breathlessness – suffocate)	bloemen – plukken	(flowers – picking)
beven – verlamming	(shake – paralyzed)	boekenkast – installeren	(book case – in stall)
borstpijn – hartaanval	(chest pain – heart attack)	boodschappen – winkelen	(groceries – shopping)
duizelig – flauwvallen	(dizziness – fainting)	krantenartikel – bestuderen	(paper article – study)
hartklopping – doodgaan	(palpitations – dying)	limonade – drinken	(lemonade – drink)
hoofdpijn – hersentumor	(headache – brain tumour)	maaltijd - klaarmaken	(meal – prepare)
misselijk – overgeven	(unwell – throw up)	onkruid – uittrekken	(weeds – pull)
onwerkelijk – krankzinnig	(unreal – insane)	schommelstoel – wiebelen	(rocking chair – wiggle)
tintelingen – beroerte	(tremor – stroke)	televisie – nieuwsbericht	(television – newsflash)
trillen – controleverlies	(tremble – loss of control)	tuintafel – zomertijd	(garden table – summer time)

Panic-Neutral		Neutral-Panic	
ademnood – winkelen	(breathlessness – shopping)	bloemen – beroerte	(flowers – stroke)
beven – nieuwsbericht	(shake – newsflash)	boekenkast – hersentumor	(book case – brain tumour)
borstpijn – zomertijd	(chest pain –summer time)	boodschappen – stikken	(groceries – suffocate)
duizelig – wiebelen	(dizziness – wiggle)	krantenartikel – doodgaan	(paper article – dying)
hartklopping – bestuderen	(palpitations – study)	limonade – krankzinnig	(lemonade – insane)
hoofdpijn – installeren	(headache – install)	maaltijd – overgeven	(meal – throw up)
misselijk – klaarmaken	(unwell – prepare)	onkruid – controleverlies	(weeds – loss of control)
onwerkelijk – drinken	(unreal – drink)	schommelstoel – flauwvallen	(rocking chair – faint)
tintelingen – plukken	(tremor – picking)	televisie – verlamming	(television – paralyzed)
trillen - uittrekken	(tremble – pull)	tuintafel - hartaanval	(garden table –hearth attack)