ORIGINAL ARTICLE

Measuring Symptom-Specific Panic-Relevant Associations Using Single-Target Implicit Association Tests

Simon E. Blackwell[1] · Frank H. Wilhelm[2] · Andrea Reinecke[3,4] · Jürgen Margraf[1] · Marcella L. Woud[1]

Abstract

Background According to major cognitive accounts of panic disorder, bodily sensations can lead to automatic activation of an associative fear network, potentially triggering a cascade of cognitive, emotional, and physiological responses culminating in a panic attack. However, the evidence for the automatic associations assumed by these models is mixed. This may reflect the heterogeneous nature of panic disorder, in that the relative importance of different bodily sensations and symptoms varies between individuals. The current study aimed to test this possibility via measuring the associations between three different sets of panic symptoms (cognitive, respiratory, cardiac) and scores on three symptom-specific single target implicit association tests (STIATs).

Methods A total of 226 unselected female participants aged 18–35 completed the STIATs as well as questionnaires assessing panic symptoms and related measures in a web-based study.

Results Only limited evidence was found to support the idea of specific associations between STIAT stimuli sets and their related panic symptoms. Exploratory analyses indicated that there were only associations between STIAT scores and panic-relevant questionnaires amongst those participants who had experienced a panic attack in the previous 6 months.

Conclusions The results have implications for measuring panic-relevant associations and understanding their role in panic disorder.

Keywords Panic · Automatic associations · Implicit association test · Cognitive bias

Occasional anxiety is a normal part of our daily life and an adaptive response to danger. However, in the context of panic disorder (PD), intense anxiety can be triggered without a clear danger or threat. Individuals suffering from PD report frequent and unexpected panic attacks, which are defined as sudden periods of intense fear and discomfort and are accompanied by a sense of losing control. As a consequence, people with PD often worry about future panic attacks and actively avoid situations or behaviors they associate with such attacks (American Psychiatric Association, 2013). In trying to elucidate factors contributing to PD, a large amount of research has focused on a potential central role for cognitive processes (Clark, 1986; Kyriakoulis & Kyrios, 2023; Margraf & Ehlers, 1989). In brief, cognitive accounts propose that in PD, individuals are hypervigilant to their bodily responses. Once attention is paid to these responses, threat-related associations are immediately triggered, followed by catastrophizing misinterpretations. To illustrate, if an individual suffering from PD attends to their pounding heart, a fear association is rapidly activated, followed by a catastrophic misinterpretation such as "This is a sign of a heart attack". This leads to increased fear, which in turn triggers additional anxiety-related bodily responses, and ultimately escalates into a vicious cycle of panic culminating in a full-blown panic attack. Support for the role of panic-related cognitions in PD comes from both cross-sectional studies (Ohst & Tuschen-Caffier, 2018) and also longitudinal data finding panic-related misinterpretations to be predictive of new onsets of PD (Woud et al., 2014).

✉ Simon E. Blackwell
simon.blackwell@rub.de

1 Faculty of Psychology, Mental Health Research and Treatment Center, Ruhr-Universität Bochum, Massenbergstrasse 9-13, 44787 Bochum, Germany

2 Division of Clinical Psychology and Psychopathology, Department of Psychology, Paris-Lodron University Salzburg, Salzburg, Austria

3 Department of Psychiatry, University of Oxford, Oxford, UK

4 Oxford Health NHS Foundation Trust, Oxford, UK

While there is large body of research investigating the role of panic-related cognitive processes (e.g., Ohst & Tuschen-Caffier, 2018), the literature in relation to panic-related associations is rather mixed; this is particularly notable given the central role attributed to such associations in the etiology of panic attacks. Various computerized tasks have been used to assess panic-related association, with priming paradigms (e.g., Fazio et al., 1986) and the Implicit Association Test (IAT; Greenwald et al., 1998) most widely applied. Both tasks share similar operationalizations in that participants are required to assign stimuli to categories as quickly as possible. Participants' reaction times (RTs) then serve as the index of interest. These RTs are considered a measure of associative strength, with faster RTs indicating a stronger association between categories and stimuli. While this operationalization seems straightforward, results in the context of panic-related associations are mixed. To illustrate, a study by Hermans et al. (2010) employed a priming paradigm and found that panic patients, compared to anxious control patients, showed stronger panic-related associations in that they reacted faster to panic trials compared to control trials (e.g., breathlessness—suffocate vs. breathlessness—shopping). A study by Schneider and Schulte (2007) also employed a priming paradigm and initially failed to find the expected difference in priming effects between panic and control group patients. However, when priming effects were calculated for idiographically-selected stimuli, the expected group difference was found. Another study, by Teachman et al. (2007), used two different versions of an IAT to examine panic-related associations. Results showed that individuals with PD, compared to healthy controls, had stronger associations (i.e., faster RTs) when categorizing stimuli belonging to the concepts *self* and *panic*. In contrast, when stimuli related to the concepts *alarming* and *bodily changes* were used, no significant group difference were found (for related null findings using similar tasks see e.g., Cloitre et al., 1992; McNally et al., 1997; Schniering & Rapee, 1997; Woud et al., 2016).

One potential explanation for these mixed findings concerns the stimuli included in the tasks used. According to the DSM-5 (American Psychiatric Association, 2013), panic attacks are characterized by 13 different possible symptoms. While the computerized tasks assessing panic-related associations described above did represent these symptoms via their stimuli, it is possible that not all symptoms were relevant for each individual patient (see Schneider & Schulte, 2007). As a consequence, the (idiosyncratic) panic-relevant associative memory network may not have been sufficiently triggered, and this, in turn, could explain why the expected RT patterns were not found. The results found by Schneider and Schulte (2007) support this hypothesis, as does the suggestion that PD is characterized by different subtypes. Specifically, patients with PD often report symptoms around

one central bodily response such as cognitive, cardiac, or respiratory responses (see e.g., Caldirola & Perna, 2019; Sansone & Sansone, 2009). The respiratory subtype (RS), for example, is a well-studied subtype, and requires the presence of at least four of five respiratory-related symptoms, i.e., breathlessness, chest pain, choking, fear of dying, and paresthesia (Briggs et al., 1993).

Consideration of the above suggests that when investigating the role of panic-related associations in the context of PD, matching of the task and the stimuli used to those symptoms that are problematic for an individual may be a crucial factor. The present online study set out to test this idea. To do so we assessed panic-related associations in three specific symptom domains: respiratory, cardiovascular, and cognitive symptoms, within a single sample, enabling us to measure each participant's panic-relevant associations for each symptom domain separately. To assess the relevance of different panic symptom clusters for each participant we created a checklist based on the DSM-5 that included all panic-relevant symptoms. Using this checklist, we created an overall sum score and a specific symptom sum score for each symptom domain (e.g., adding the ratings of all items targeting cardiovascular symptoms), allowing us to capture the magnitude of both general and specific symptoms. In addition, we assessed levels of anxiety sensitivity (AS) via the Anxiety Sensitivity Index—3 (ASI; Taylor et al., 2007, German version: Kemper et al., 2011). AS has been shown to be a risk factor for PD development and maintenance (Olatunji & Wolitzky-Taylor, 2009), and previous studies have reported significant correlation between levels of AS and panic-related associations and catastrophic misinterpretations (e.g., Richards et al., 2001; Steinman & Teachman, 2010; Teachman, 2005; Zahler et al., 2020). As such, the AS provides an additional useful PD-analogue when examining panic-related associations in a general population sample. We also included other questionnaire measures of panic-related interpretations to allow investigation of their relationship with panic-related associations measured by the STIAT, as well as measures of general depression, anxiety and stress to help characterize the sample.

To assess panic-related associations we used a task that should provide a number of advantages compared to those previously used, namely the Single Target Implicit Association Test (STIAT; Wigboldus et al., 2004). The STIAT is a modified version of the IAT (Greenwald et al., 1998) and has been applied successfully in assessing various anxiety-relevant associations in e.g., fear of spiders (Woud et al., 2011) or generalized anxiety disorder (Reinecke et al., 2010). Using the STIAT compared to the IAT has the advantage that it does not require two opposing categories, and the associative strength between a stimulus and a category can be measured independently of the association with another category. Further, the psychometric properties of STIAT are

at least equally reliable as the IAT (Karpinski & Steinman, 2006).

We conceptualized panic-relevant associations as a cognitive risk factor for panic disorder that should be present to varying degrees within the general population, and we therefore did not use inclusion criteria based on anxiety sensitivity or panic disorder diagnoses in our participant selection (i.e., an experimental psychopathology approach; Davey, 2017). This also facilitated collection of a larger sample size and participants who scored across the whole range of the measures used. While previous studies have indicated that it is possible to measure the relative strength (or absence) of panic-relevant associations in non-clinical samples (e.g., Zahler et al., 2020), whether such cognitive factors are in fact risk factors for panic disorder (as opposed to, e.g., a correlate or consequence of panic-like experiences) is still an open question (e.g., see Teachman, 2005, for a discussion). There is evidence to support taking a dimensional approach to studying the relationships between cognitive risk factors and symptoms in the context of some other disorders, such as depression (e.g., Everaert et al., 2017; Liu et al., 2019; Persons et al., 1991). However, the picture has generally been less clear in the context of panic disorder. For example, there have been mixed findings with regard to the relationship between panic-relevant cognitions and interpretations and panic-relevant measures such as anxiety sensitivity, which may reflect variations in the kind of sample studied (Teachman, 2005). To increase the likely proportion of people with elevated levels of anxiety sensitivity and panic-like experiences, that is, people for whom we would be more confident to expect relationships between cognitive processes and panic-related measures, we decided to restrict the study to women aged between 18 and 35. This was based on data showing that PD has its onset in early adulthood (Solmi et al., 2022), and the prevalence of PD is higher in women than men (McLean et al., 2011).

The general hypothesis of the present study was that we would find symptom-congruent correlations between a corresponding STIAT version and symptom score. For example, we would expect scores on the subscale of the symptom checklist asking about cardiovascular symptoms to show a stronger correlation with the cardiovascular STIAT than with the respiratory and cognitive STIAT. We further hypothesized that we would find correlations between the DSM-5 checklist sum score, ASI-3 and all STIAT versions. Finally, we expected each specific STIAT version to have unique predictive validity, i.e., to explain unique variance when predicting a specific symptom score in addition to any explained by the other STIATs.

Methods

Design

The study used a cross-sectional design and was conducted online. It was pre-registered at aspredicted.org (#40464; https://aspredicted.org/at6r5.pdf).

Participants and Recruitment

Participants were recruited via online advertisements, including on social media and the study recruitment page for the Faculty of Psychology. Participant information explained that it investigated the relationship between thoughts, body sensations, and feelings. Inclusion criteria were being aged 18 or older, female gender, and fluent German. Participants were further informed that the study would not work on a tablet or smartphone. Participants read the information sheet online and provided informed consent online, at which point they also confirmed that they met the inclusion criteria.

Measures

The study procedures were implemented via a custom-built online platform implemented using JavaServer Pages and JavaScript on a secure server at the university (available for download at: https://osf.io/x48uh/). Participants accessed the study via their web browser.

Single Target Implicit Association Tests (STIATs)

Three Single Target Implicit Association Tests (STIATs) were used in this study, each mapping onto a different panic disorder domain (respiratory symptoms, cognitive symptoms, autonomic symptoms). The general structure was based on that used by Zahler et al. (2020). Participants were asked to categorise word stimuli[1] ('attribute words' and 'target words') presented in the middle of the browser window according to category words presented at the top of the window. These category words were two attribute categories (alarming and meaningless), presented in the two top corners of the browser window, and one target category (bodily change), presented underneath one of the two attribute categories in a top corner of the browser window. Participants

---

[1] German words used were: Attribute categories: bedeutunglos, alarmierend; target category: Körperveränderung; attribute words: bedeutungslos, unbedeutend, unwichtig, belanglos, beunruhigend, beängstigend, erschreckend, gefährlich; target words (respiratory): Atemnot, Kurzatmigkeit, ersticken, Atembeschwerden; target words (cognitive): Kontrollverlust, Verrücktheit, durchdrehen, Verwirrtheit; target words (cardiac): Herzklopfen, Herzrasen, Herzstolpern, Herzschwäche.

pressed the 'A' or the 'L' key respectively to indicate that the word belonged to the category on the left-hand side or right-hand side of the browser. Each STIAT used the same attribute words, for which there were four per attribute category (for meaningless: meaningless, insignificant, unimportant, trivial; for alarming: disturbing, scary, frightening, dangerous). Target words varied according to the relevant domain for the STIAT (respiratory: breathlessness, shortness of breath, choking, difficulty breathing; cognitive: loss of control, craziness, going crazy, confusion; cardiac: heart pounding, heart racing, palpitations, heart failure).

Each STIAT was organized into five blocks: attribute practice (20 trials), first combined practice (20 trials), first combined block (40 trials), second combined practice (20 trials), second combined block (40 trials). In the attribute practice block only the two attribute category words were presented in the top corners of the window, and participants practiced categorizing the attribute words according to these two categories. For the combined blocks, the target category word was also presented on the screen, either under the attribute category 'alarming' (compatible block) or under the category 'meaningless' (incompatible block). Participants were instructed to respond as quickly as possible but to try not to make errors. If a participant made an error, a red cross appeared on the screen and the participant was required to press the correct key to continue.

The three STIATs were presented one after the other with a single instruction screen in between. Order of the three STIATS was counterbalanced across participants (6 possible orders). Order of the combined blocks was also counterbalanced across participants (compatible blocks followed by incompatible blocks or vice versa), with this order the same across all three STIATS for any one participant. The initial position of the target category word (left or right) on the screen was also counterbalanced across participants, and the same for each STIAT for any one participant. This resulted in a total of 24 counterbalanced conditions.

Scoring for the STIATs followed the procedure detailed by Bluemke and Friese (2008). That is, first of all trials with incorrect responses or from the attribute practice block were removed. Next, responses with reaction times < 300 ms or > 3000 ms were replaced with these values. Reaction times for each trial were then Z-transformed by subtracting the mean reaction time for all trials (across all four blocks) then dividing by the standard deviation for all trials (across all four blocks). The first trial from each block was then removed. Final scores for the STIAT were then calculated by subtracting the mean z-scores for the incongruent trials from the mean z-scores for the congruent trials. This meant that a positive STIAT score indicated longer reaction times for congruent vs. incongruent blocks and was interpreted as a weaker association between the relevant panic symptom (target words) and the attribute label 'alarming'.

Split-half reliabilities [95% confidence intervals] were calculated for each STIAT separately using 2000 randomly-selected split halves, and were as follows: respiratory STIAT: 0.91 [0.88, 0.93], cardiac STIAT: 0.92 [0.90, 0.94], cognitive STIAT: 0.74 [0.66, 0.80].

Questionnaire Measures

Anxiety Sensitivity Index—3 (ASI; Taylor et al., 2007; German version: Kemper et al., 2011) The ASI used here is an updated version of the original ASI (Reiss et al., 1986), which was developed as a measure of the belief that anxiety has negative implications or consequences. It comprises 18 items asking about three domains of concern: physical (e.g., "When I feel pain in my chest, I worry that I'm going to have a heart attack"), social (e.g., "I worry that other people will notice my anxiety"), and cognitive (e.g., "When my thoughts seem to speed up, I worry that I might be going crazy."). Participants rate the extent to which each item applies to them on a five-point scale from 0 (*very little*) to 4 (*very much*). Cronbach's alpha in our sample was $\alpha = 0.87$ [0.83, 0.89].

Body Sensations Questionnaire (BSQ; Chambless et al., 1984; German version: Ehlers et al., 1993b) The BSQ comprises a list of 17 body sensations that people might experience when feeling anxious (e.g., heart palpitations, nausea). Participants are asked to rate how frightened they are by each sensation on a scale from 1 (*not at all*) to 5 (*extremely*). Cronbach's alpha in our sample was $\alpha = 0.87$ [0.84, 0.90].

Agoraphobic Cognitions Questionnaire (ACQ; Chambless et al., 1984; German version: Ehlers et al., 1993a) The ACQ comprises a list of 14 cognitions that people might experience when feeling nervous or frightened (e.g., "I am going to throw up", "I will have a heart attack"). Participants are asked to rate how often each cognition occurs to them when they are nervous on a scale from 1 (*thought never occurs*) to 5 (*thought always occurs*). Cronbach's alpha in our sample was $\alpha = 0.79$ [0.73, 0.83].

DSM-5 Panic Symptom Checklist In this self-report questionnaire, adapted from that used in previous studies (Wilhelm et al., 2001; Zahler et al., 2020), participants were presented with the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) symptoms of panic disorder. Participants were asked to think of a time when they had suddenly experienced a high level of anxiety and then rate the extent to which they experienced each symptom at that time on a scale from 0 (*not at all*) to 10 (*extremely*). The 13 panic symptoms listed in the DSM-5 were included, split into individual phenomena such that each was a separate item. For example, the

symptom "Palpitations, pounding heart, or accelerated heart rate" was split into three items (Palpitations, pounding heart, accelerated heart rate), which were each rated separately. This resulted in a 26-item questionnaire, with Cronbach's $\alpha = 0.94$ [0.93, 0.95]. We further created subscales for cardiac symptoms (3 items; $\alpha = 0.83$ [0.79, 0.86]), cognitive symptoms (2 items; $\alpha = 0.76$ [0.69, 0.82]), and respiratory symptoms (3 items; $\alpha = 0.91$ [0.89, 0.93]).

Depression Anxiety Stress Scales—Short form (DASS-21; Lovibond & Lovibond, 1995; German version: Nilges & Essau, 2015) The DASS-21 comprises three 7-item subscales asking about the experience of symptoms of depression, anxiety, and stress over the past week. Participants rate each symptom on a scale from 0 (*did not apply to me at all*) to 3 (*applied to me very much or most of the time*) Cronbach's alpha in our sample was $\alpha = 0.90$ [0.87, 0.92] for the depression subscale, $\alpha = 0.85$ [0.80, 0.89] for the anxiety subscale, and $\alpha = 0.86$ [0.83, 0.89] for the stress subscale.

Experience of Panic Attacks A series of questions were asked to assess whether participants had experienced panic attacks in the past 6 months according to DSM criteria. A first question asked whether they had experienced an 'anxiety attack' in the past 6 months, and explained that this meant suddenly experiencing intensive anxiety without a recognizable cause. A second question asked whether this anxiety had reached a peak within a few minutes, and a third whether this anxiety was associated with strong bodily reactions and changes. Answering yes to all three of these questions led to the participant being classed as having a recent history panic attacks for analysis purposes. A final question asked how many such attacks the participant had experienced in the past month.

Procedure

After reading the study information and providing informed consent, participants provided demographic information, then completed the ASI and the BSQ followed by the three STIATs. Afterwards participants reported their experience of panic attacks and completed the panic symptom checklist, followed by the ACQ and finally the DASS-21. Participants then read some debriefing information about the study.

Statistical Analyses

Statistical analyses were conducted in RStudio 2022.12.0 running R version 4.2.2 (R Core Team, 2022; RStudio, Inc., 2016). Data and analysis scripts are available at https://osf.io/x48uh/.

The analysis plan followed that outlined in the pre-registration form. Initially we conducted Pearson's correlations to examine the relationships between scores on the STIATs, the questionnaire measures, and DSM symptom scores. Bootstrapped versions of each correlation (5000 bootstrap samples) were conducted as an additional robustness check. We then conducted regressions to further examine the relationship between scores on the STIATs and (i) total score on the symptom checklist, and (ii) scores on the individual symptom checklist subscales. These regressions included scores on the three STIATs as independent variables in order to test the specificity of their relationships with their corresponding symptom checklist subscales. Assumptions of the regression were checked following the steps outlined by Field et al. (2012), including computation of bootstrapped regression coefficients (5000 bootstrap samples). In additional exploratory analyses we examined the potential moderating role of previous history of panic attacks via regressions including an interaction between history of panic attacks as self-reported by participants (yes vs. no) and STIAT scores (see Results section for full details).

Results

Participant Characteristics

A total of 226 participants completed the study, albeit with missing data for at least one STIAT (due to the data not saving correctly) for four participants. The majority were native German speakers ($n = 209$, 92.5%), and students ($n = 193$, 85.4%). Other occupations were unemployed/job-seeking ($n = 3$, 1.3%), self-employed ($n = 1$, 0.4%), and employed ($n = 29$, 12.8%). Highest educational levels achieved were doctorate ($n = 1$, 0.4%), Masters ($n = 6$, 2.7%), Bachelors ($n = 23$, 10.2%), Vocational qualification ($n = 40$, $n = 17.7\%$), and school-leaving certificate/exams ($n = 156$, 69.0%). Scores on the DASS-21 were comparable to other unselected student samples in Germany (e.g., Bibi et al., 2020), indicating a relatively healthy sample overall.

In total, 31 (13.7%) of participants reported having experienced a panic attack in the last 6 months that potentially fulfilled DSM criteria, and of these, 7 (22.6%) had experienced none in the past month, 10 (32.3%) reported experiencing one in the past month, 10 (32.3%) reported experiencing two in the past month, and 4 (12.9%) reported experiencing more than two in the past month. Independent sample t-tests indicated that there was no difference in scores on any of the three STIATs between people who had or had not experienced a panic attack in the past 6 months (ps > 0.50). One-sample t-tests indicated that the mean scores on all STIATS were less than 0, indicating on average stronger associations between panic symptoms and the attribute 'alarming' relative to 'meaningless' (ps < 0.001).

Table 1 Participant Scores on Questionnaire Measures and Correlations with STIAT Scores

	Mean (SD)	Range	STIAT		
			Cognitive	Respiratory	Cardiac
STIAT					
Cognitive	− 0.41 (0.34)	− 1.31–0.82	–	0.33***	0.40***
Respiratory	− 0.35 (0.32)	− 1.48–0.83	0.33***	–	0.26***
Cardiac	− 0.31 (0.30)	− 0.91–1.18	0.40***	0.26***	–
Age	22.44 (3.54)	18–35	0.00	− 0.05	0.06
ASI	24.69 (11.03)	1–59	− 0.07	− 0.08	− 0.06
BSQ	23.41 (11.44)	1–58	− 0.12	− 0.01	− 0.06
ACQ	24.00 (6.90)	14–51	− 0.13*	− 0.07	− 0.07
Panic symptoms					
Total score	88.04 (51.27)	1–230	− 0.14*	− 0.04	− 0.12
Cognitive	3.24 (2.98)	0–10	− 0.16*	− 0.08	− 0.15*
Respiratory	3.14 (2.97)	0–10	− 0.10	0.01	− 0.03
Cardiac	4.62 (2.67)	0–10	− 0.11	− 0.05	− 0.13
DASS depression	5.98 (4.86)	1–22	− 0.02	0.01	− 0.04
DASS anxiety	4.58 (4.52)	0–21	− 0.08	− 0.04	− 0.08
DASS stress	7.25 (4.84)	0–21	− 0.07	− 0.05	− 0.08

STIAT = Single Target Implicit Association Test (scores < 0 indicate on average stronger associations between panic symptoms and the attribute 'alarming' relative to 'meaningless')

ASI anxiety sensitivity index, *BSQ* body sensations questionnaire, *ACQ* agoraphobic cognitions questionnaire, *DASS* depression anxiety stress scale

*$p < .05$; ***$p < .001$

Participants' scores on the questionnaires and the STIATs are shown in Table 1.

Correlational Analyses

Correlations between STIAT scores and other variables are shown in Table 1.

General Panic Measures

None of the STIATs correlated with scores on the ASI or BSQ, but scores on the dysfunctional cognitions STIAT correlated with scores on the ACQ and total panic symptom checklist scores.

Measures of Specific Panic Symptoms

Scores on the dysfunctional cognitions STIAT correlated with cognitive symptoms, as measured by the cognitive subscale of the panic symptoms checklist, and not with the other symptom subscales (see Table 1). However, scores on the cardiac STIAT also showed the same pattern, correlating with the cognitive symptoms subscale but none of the others. Scores on the respiratory STIAT did not correlate with any symptom subscales. Almost all correlations were in the expected direction but were small in magnitude, and there

was little difference in magnitude between those correlations that reached statistical significance and those that did not.

Regression Analyses

In regression analyses with all three STIAT scores as independent variables, no STIAT score was a predictor of either total score on the panic symptoms checklist or any individual subscale (see Supplementary Material for details).

Exploratory Analyses

An alternative hypothesis for the lack of consistent associations between scores on measures of panic-relevant associations and panic-related outcome measures across studies could be that associations need to be made salient by recent experience of panic symptoms; generally the studies finding such associations have included patients with panic disorder. We therefore included variations in sample characteristics, in particular participants' history of panic attacks, in the analysis. As this hypothesis did not relate to matching of specific STIAT stimuli to specific panic symptoms, we first created an aggregate STIAT score consisting of the mean of the scores on the three STIATs. This aggregate STIAT score did not show statistically significant correlations with scores on the ASI ($r = − .09$; $p = .15$) or BSQ ($r = − .10$; $p = .14$), but did correlate with scores on the ACQ ($r = − .13$,

$p = .045$) and total score on the panic symptoms checklist ($r = -.14$, $p = .03$).

We then carried out regressions including as dependent variable a panic-related outcome measure (ASI, BSQ, ACQ, total score on the panic symptoms checklist), and as independent variables score on the aggregate STIAT (mean-centered), a binary variable indicating history of a panic attack (in the past 6 months), and their interaction. There was a statistically significant interaction between STIAT score and history of panic attacks when the dependent variable was the ASI ($p = .001$), BSQ ($p = .008$), and ACQ ($p = .03$), but not when it was score on the panic symptoms checklist ($p = .22$). When the statistically significant interactions were broken down, amongst individuals who had recently experienced panic attacks ($n = 31$) there were substantial correlations between scores on the aggregate STIAT and scores on the ASI ($r = -.54$, $p = .002$), BSQ ($r = -.48$, $p = .006$), and ACQ ($r = -.33$, $p = .07$), albeit not statistically significant for the ACQ. Amongst individuals who had not previously experienced panic attacks ($n = 195$), none of the correlations were statistically significant (ASI: $r = -.02$, $p = .75$; BSQ: $r = -.04$, $p = .61$; ACQ: $r = -.09$, $p = .19$). See Supplementary Material for full details.

Given this pattern of results, we then investigated whether our initial hypothesis of specificity in the relationships between scores on the different STIATs and the scores on the associated subscales of the panic symptoms checklist would be supported within the subsample with a history of recent panic attacks. We examined the correlations between scores on the individual STIATs and scores on the symptom subscales. However, there was little evidence for specificity even within this subsample, in that all STIATs showed the strongest correlation with the cognitive symptom subscale, followed by the respiratory symptom subscale and then the cardiac symptom subscale (see Supplementary Material for details). Given these results and the small sample size we did not conduct follow-up regressions in this subsample.

Discussion

The primary aim of this study was to further our understanding of the relationship between panic-related associations and symptoms of panic disorder, and in particular relevance of symptom specificity. To do so, we examined the correlation between three specific STIAT versions (cognitive, cardiac, and respiratory) and specific panic symptoms as well as general panic-relevant measures (the ASI-3, BSQ, and ACQ) in an online study. We also examined the specificity of relationships between scores on the different STIATs and their associated symptom clusters, and via exploratory analyses, tested whether the relationship between STIAT scores and panic-relevant outcomes varied depending on whether participants had recently experienced anxiety attacks or not.

Results showed that all three STIAT versions had acceptable to excellent split-half reliability and moderate correlations with each other. However, we only found limited evidence to support our hypothesis of symptom-congruent correlations between a specific STIAT and symptom score. For the cognitive STIAT, the only statistically significant correlation with a panic symptom subscale score was with cognitive symptoms (and not respiratory or cardiac symptoms), indicating that stronger cognitive panic-related associations were correlated with higher panic-related cognitive symptoms. However, while correlations between the cognitive STIAT and other panic symptom subscales were not statistically significant, they were of similar (small) magnitude to the correlation with cognitive symptoms. Additionally, scores on the cognitive STIAT did not explain unique variance in cognitive symptom scores when controlling for scores on the other STIATs in a regression, and the cardiac STIAT showed a similar correlation with cognitive symptoms. Otherwise, the other cardiac and respiratory STIATs did not correlate with any questionnaire measures, and interestingly none correlated with scores on the ASI-3. In exploratory analyses, however, we found an interaction between an aggregated (mean) score derived from the STIATs and self-reported history of panic attacks in predicting scores on several panic measures (ASI-3, BSQ, ACQ), indicating that there were only correlations between panic-related associations and these panic measures amongst those participants who had experienced a panic attack in the past 6 months.

The pattern of a general lack of relationship, or only small correlations, between scores on the STIATs used in this study and overall panic-related measures (e.g., the ASI) is consistent with the literature indicating a mixed picture in terms of the relationship between measures of panic-related associations and panic symptoms or panic disorder diagnoses (e.g., Schneider & Schulte, 2007; Teachman et al., 2007; Zahler et al., 2020). However, the limited evidence for specific relationships between matched STIAT and panic symptom subscales is not consistent with our prediction that matching of STIAT stimuli to panic symptoms would reveal stronger relationships. This prediction stemmed initially from the finding of Schneider and Schulte (2007) that a difference in responses on a panic-related priming task between patients with panic disorder and healthy controls was only found when calculating the priming score using idiographically-selected stimuli. However, in the study by Schneider and Schulte (2007), selection of stimuli for calculation of the individualized priming effect was based on reaction times on a separate semantic relatedness task, that is another behavioral task closely related to the priming task itself. In contrast, in our study the matching of STIAT to relevant panic symptoms was not so highly individualized at

an item level, and was between two methodologically much more weakly-related measures, namely a reaction time task and a self-report symptom measure. Further, we did not include a patient sample, and only a small proportion of our sample had a recent history of panic attacks, and this may have reduced our sensitivity to observe relationships with symptom measures. Recruitment of a predominantly healthy sample could lead to insufficient variance in the data to find reliable relationships, as well as to a sample for whom there is not a strong panic-relevant associative network structure (see Lang, 1979). Such a memory structure is hypothesized to store feared bodily sensations along with panic-related cognitions (such as panic-related associations), and it is assumed that this associative network structure can be activated once one of its components is triggered. We expected that our STIAT would serve as such a trigger, but this of course would be dependent on the existence of the associative network in the first place. It may well be that within a larger sample of patients with panic disorder we would find differential relationships between our different STIATs and different symptom clusters or patient subtypes responses (e.g., cardiac or respiratory subtypes; Briggs et al., 1993; Caldirola & Perna, 2019; Sansone & Sansone, 2009). Finally, our measure of panic-related associations was a STIAT rather than the priming task used by Schneider and Schulte (2007); although from a theoretical perspective we think of these as measuring the same associative network, it may be that differences in the precise operationalization of the measurement between these two tasks means that they have different sensitivities for detecting panic-relevant associations.

Our exploratory analyses may shed more light on the current mixed literature. That is, if there are only relationships between panic-relevant associations and panic-related measures such as anxiety sensitivity amongst people who have a recent history of panic attacks, it may be that whether a study finds such correlations is highly dependent on the sample characteristics. The present results have to be interpreted with the caveat that they were based on exploratory rather than pre-planned analyses. Further, only a small number of participants in our study (13.7%; $n = 31$) indicated a history of panic attacks in the past 6 months. This proportion is higher than the estimated lifetime prevalence of panic disorder amongst women (e.g., 7.1%; McLean et al., 2011), potentially indicating that our restriction of the study to younger women was successful in increasing the prevalence of panic-like experiences in the sample. However, the categorization in our study was based on participants' self-report rather than a diagnostic assessment. It is notable that the difference between this subset of participants and those with no panic attacks in the past 6 months was not in total scores on the STIATs, but rather the relationship between STIAT scores

and other panic-related measures. From the perspective of cognitive models, in which panic-relevant associations play a central role (e.g., Clark, 1986; Kyriakoulis & Kyrios, 2023; Margraf & Ehlers, 1989), there are a number of ways in which these results could be interpreted in the context of the existing literature.

A first possible interpretation could be that strong panic-relevant associations are not in themselves problematic, but could potentially become so when activated, for example by a panic attack (which could be triggered by a variety of physiological or psychological stressors). Once activated, strong panic-relevant associations could then be a risk factor for experiencing further panic attacks. Such an explanation would fit with other research finding no relationship between anxiety sensitivity and various measures of panic-relevant cognitive biases in samples with no history of panic attacks (e.g., McNally et al., 1999; although see Teachman, 2005), but it is also possible that these biases are simply correlates of panic disorder rather than playing any causal role (see Teachman, 2005, for a discussion).

A second possibility is that panic-relevant associations may only become problematic in the context of other factors that differentiate people who experience repeated panic attacks from people who do not. For example, it might be that panic-relevant associations are only risk factors for panic attacks amongst individuals who also have strong attentional biases towards bodily sensations, or who then follow up these associations with a catastrophic misinterpretation. Amongst such individuals (or those with a history of panic attacks), scores on the STIAT could reflect strength of activation of an associative fear network, whereas for individuals without such additional vulnerabilities (and no history of panic-like experiences, meaning no such associative fear network) they could simply reflect semantic associations. This proposition might help explain both our results and those of Hermans et al. (2010). While Hermans et al. (2010) found that panic patients showed stronger panic-relevant associations than anxious control patients in a priming task, the panic patients did not differ from a group of mental health professionals without a history of panic disorder. The authors suggest that this finding can be explained by the mental health professionals' knowledge, i.e., they are confronted with the association of 'bodily response—panic' on a daily basis, and this repeated experience, in turn, may have led them to also develop stronger panic-related associations. Due to the nature of the recruitment and incentivization for our study, it is likely that a large proportion were undergraduate psychology students, who may also have already had some education about panic disorder and thus be aware of such associations. However, as we did not collect information about the subject studied by student participants this is not possible to test within our sample and remains speculative.

A third possibility is that STIAT scores amongst the participants with no history of panic attacks could in fact measure a meaningful risk factor for future panic attacks, but investigating this would require longitudinal studies in high-risk samples (e.g., scoring high on the ASI, but with no history of panic attacks). While longitudinal data has found that panic-relevant interpretations predicted future new onsets of panic disorder (Woud et al., 2014), this has yet to be investigated for panic-relevant associations. It is possible that tending to interpret bodily sensations such as symptoms of anxiety as threatening could make someone more vulnerable to experience a first panic attack under circumstances of physiological arousal or psychological stress, and after this a fear network is formed that might be detectable via tasks like the STIAT and which would present a risk for future, more easily-triggered, panic attacks. Such an explanation would fit well with a recently-proposed computational model of panic disorder (Robinaugh et al., 2019). Within this framework, a tendency to interpret certain bodily sensations in a threatening manner could lead to the individual 'escaping' a situation in which they find their anxiety rapidly escalating (i.e. an initial panic attack or panic-like experience), leading to formation of an 'arousal schema', which could conceivably include the panic-relevant associations as measured by the STIAT. This in turn could lead to rapid escalation between arousal and perception of threat (i.e. activation of the associations), increasing the risk of future panic attacks (and further strengthening of the arousal schema).

Interestingly, our exploratory results and the possible interpretations outlined above fit with previous findings from the study by Woud et al. (2016) in which patients with panic disorder were compared to a healthy control group. As noted in the introduction to this paper, there was no difference between the two groups on a measure of panic-relevant associations (an Extrinsic Affective Simon Task). However, within the panic patient group, scores on the EAST reflecting less positive associations with agoraphobia-related situations correlated with trait anxiety; this correlation was not found within the healthy control group or across the whole sample. However, before drawing strong interpretations, it would be useful to replicate our exploratory results in a larger sample, ideally comparing patients with a panic disorder diagnosis to healthy controls. Further, hypotheses of interplays between cognitive processes as suggested here could be best tested via experimental set-ups that combine measures of all relevant processes into one task, as has been done under the 'combined cognitive biases' hypothesis in other areas (e.g., Everaert & Koster, 2020). It would also be useful to go beyond static cross-sectional measures of what are essentially dynamic processes in order to examine the pathway from panic-related association to panic attacks more closely, as well as model the expected dynamic relationships more formally (Robinaugh et al., 2019). Finally,

in order to test the hypothesized causal role of panic-related associations in panic disorder it would be useful to conduct studies with experimental manipulations, as has been done for panic-related interpretations (e.g., Steinman & Teachman, 2010; Würtz et al., 2023).

The present study has several limitations. First, the sample only includes young women, limiting the generalizability of the present findings. For example, it could be that in a sample with a larger age range a greater proportion would have some past experience of panic attacks, which may in turn alter their responding on the STIAT. Second, although the symptom subscale scores extracted from the DSM-5 checklist revealed good internal consistency, these scores were derived from a very small number of items. While this may have allowed us to measure the core manifestation of a specific kind of symptom, it may have also reduced the sensitivity of these measures via excluding broader aspects of their experience. Third, the present study did not include a clinical or at risk-sample, and not many participants reported having experienced a panic attack. In line with the explanations presented earlier related to the role of associative network structures, this may have reduced our chances to find the expected effects. It would also have been useful to ask about lifetime history of panic attacks (not just the past 6 months), as this might reveal a larger proportion of the sample for whom the panic-related associations were relevant. Further, given the online format of the present study, we could not include a task that would theoretically activate a fear network, such as a symptom provocation task, which may have been needed to allow such a network to be adequately measured by the STIAT. It is also possible that the sensitivity of the STIAT could be improved by using computational modelling of the semantic relationships between words to refine the stimuli set (Gladwin, 2023). Finally, as the study was completed online, we do not know in which circumstances participants completed the study procedures, and variation in these circumstances would likely introduce further variability in the data quality.

To summarise, in this study only limited evidence was found to support the proposal of specificity in the relationship between panic-related associations and panic symptoms. However, results indicate that what might differentiate people with or without a history of panic attacks is not necessarily the presence or absence of measurable panic-relevant associations, but the extent to which these show relationships with key aspects of panic disorder such as anxiety sensitivity. Following up this exploratory result and re-testing our initial hypothesis in clinical and subclinical samples has potential to shed important light on the role of automatic associations in panic disorder.

Declarations

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Publishing.

Bibi, A., Lin, M., Zhang, X. C., & Margraf, J. (2020). Psychometric properties and measurement invariance of Depression, Anxiety and Stress Scales (DASS-21) across cultures. *International Journal of Psychology, 55*(6), 916–925. https://doi.org/10.1002/ijop.12671

Bluemke, M., & Friese, M. (2008). Reliability and validity of the Single-Target IAT (ST-IAT): Assessing automatic affect towards multiple attitude objects. *European Journal of Social Psychology, 38*(6), 977–997. https://doi.org/10.1002/ejsp.487

Briggs, A. C., Stretch, D. D., & Brandon, S. (1993). Subtyping of panic disorder by symptom profile. *The British Journal of Psychiatry: The Journal of Mental Science, 163*, 201–209. https://doi.org/10.1192/bjp.163.2.201

Caldirola, D., & Perna, G. (2019). Toward a personalized therapy for panic disorder: Preliminary considerations from a work in progress. *Neuropsychiatric Disease and Treatment, 15*, 1957–1970. https://doi.org/10.2147/NDT.S174433

Chambless, D. L., Caputo, G. C., Bright, P., & Gallagher, R. (1984). Assessment of fear in agoraphobics—The body sensations questionnaire and the agoraphobic cognitions questionnaire. *Journal of Consulting and Clinical Psychology, 52*(6), 1090–1097.

Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy, 24*, 461–470.

Cloitre, M., Heimberg, R. G., Holt, C. S., & Liebowitz, M. R. (1992). Reaction time to threat stimuli in panic disorder and social phobia. *Behaviour Research and Therapy, 30*(6), 609–617. https://doi.org/10.1016/0005-7967(92)90006-3

Davey, G. C. L. (2017). A research pathway for experimental psychopathology: The role of external validity criteria. *Psychopathology Review, 4*(2), 129–140. https://doi.org/10.5127/pr.045015

Ehlers, A., Margraf, J., & Chambless, D. (1993a). *ACQ Fragebogen zu angstbezogenen Kognitionen*. Beltz.

Ehlers, A., Margraf, J., & Chambless, D. (1993b). *BSQ Fragebogen zur Angst vor körperlichen Symptomen*. Beltz.

Everaert, J., & Koster, E. H. W. (2020). Chapter 9—The interplay among attention, interpretation, and memory biases in depression: revisiting the combined cognitive bias hypothesis. In T. Aue & H. Okon-Singer (Eds.), *Cognitive biases in health and psychiatric disorders* (pp. 193–213). Academic Press. https://doi.org/10.1016/B978-0-12-816660-4.00009-X

Everaert, J., Podina, I. R., & Koster, E. H. W. (2017). A comprehensive meta-analysis of interpretation biases in depression. *Clinical Psychology Review, 58*, 33–48. https://doi.org/10.1016/j.cpr.2017.09.005

Fazio, R. H., Sanbonmatsu, D., Powell, M., & Kardes, F. (1986). On the automatic activation of attitudes. *Journal of Personality and Social Psychology, 50*, 229–238.

Field, A., Miles, J., & Field, Z. (2012). *Discovering statistics using R*. SAGE Publications.

Gladwin, T. E. (2023). Toward the nature of automatic associations: Item-level computational semantic similarity and IAT-based alcohol-valence associations. *Addiction Research & Theory, 31*(2), 100–105. https://doi.org/10.1080/16066359.2022.2123474

Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. (1998). Measuring individual differences in implicit cognition: The implicit association test. *Journal of Personality and Social Psychology, 74*(6), 1464–1480.

Hermans, D., De Cort, K., Noortman, D., Vansteenwegen, D., Beckers, T., Spruyt, A., & Schruers, K. (2010). Priming associations between bodily sensations and catastrophic misinterpretations:

Specific for panic disorder? *Behaviour Research and Therapy, 48*(9), 900–908. https://doi.org/10.1016/j.brat.2010.05.015

Karpinski, A., & Steinman, R. B. (2006). The single category implicit association test as a measure of implicit social cognition. *Journal of Personality and Social Psychology, 91*(1), 16–32. https://doi.org/10.1037/0022-3514.91.1.16

Kemper, C. J., Ziegler, M., & Taylor, S. (2011). ASI-3. Angstsensitivitätsindex-3 [Verfahrensdokumentation aus PSYNDEX Tests-Nr. 9006208 und Fragebogen]. In *Leibniz-Zentrum für Psychologische Information und Dokumentation (ZPID) (Hrsg.), Elektronisches Testarchiv*. ZPID. https://doi.org/10.23668/psycharchives.403

Kyriakoulis, P., & Kyrios, M. (2023). Biological and cognitive theories explaining panic disorder: A narrative review. *Frontiers in Psychiatry*. https://doi.org/10.3389/fpsyt.2023.957515

Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology, 16*(6), 495–512. https://doi.org/10.1111/j.1469-8986.1979.tb01511.x

Liu, R. T., McArthur, B. A., Burke, T. A., Hamilton, J. L., Mac Giollabhui, N., Stange, J. P., Hamlat, E. J., Abramson, L. Y., & Alloy, L. B. (2019). A latent structure analysis of cognitive vulnerability to depression in adolescence. *Behavior Therapy, 50*(4), 755–764. https://doi.org/10.1016/j.beth.2018.11.004

Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the depression anxiety stress scales* (2nd ed.). Psychology Foundation.

Margraf, J., & Ehlers, A. (1989). Etiological models of panic: psychophysiological and cognitive aspects. In *Panic disorder: Theory, research and therapy* (pp. 205–231). New York.

McLean, C. P., Asnaani, A., Litz, B. T., & Hofmann, S. G. (2011). Gender differences in anxiety disorders: Prevalence, course of illness, comorbidity and burden of illness. *Journal of Psychiatric Research, 45*(8), 1027–1035. https://doi.org/10.1016/j.jpsychires.2011.03.006

McNally, R. J., Hornig, C. D., Hoffman, E. C., & Han, E. M. (1999). Anxiety sensitivity and cognitive biases for threat. *Behavior Therapy, 30*(1), 51–61. https://doi.org/10.1016/S0005-7894(99)80045-8

McNally, R. J., Hornig, C. D., Otto, M. W., & Pollack, M. H. (1997). Selective encoding of threat in panic disorder: Application of a dual priming paradigm. *Behaviour Research and Therapy, 35*(6), 543–549. https://doi.org/10.1016/s0005-7967(96)00125-8

Nilges, P., & Essau, C. (2015). Die Depressions-Angst-Stress-Skalen: Der DASS—ein Screeningverfahren nicht nur für Schmerzpatienten. *Der Schmerz, 29*(6), 649–657. https://doi.org/10.1007/s00482-015-0019-z

Ohst, B., & Tuschen-Caffier, B. (2018). Catastrophic misinterpretation of bodily sensations and external events in panic disorder, other anxiety disorders, and healthy subjects: A systematic review and meta-analysis. *PLoS ONE, 13*(3), e0194493. https://doi.org/10.1371/journal.pone.0194493

Olatunji, B. O., & Wolitzky-Taylor, K. B. (2009). Anxiety sensitivity and the anxiety disorders: A meta-analytic review and synthesis. *Psychological Bulletin, 135*(6), 974–999. https://doi.org/10.1037/a0017428

Persons, J. B., Miranda, J., & Perloff, J. M. (1991). Relationships between depressive symptoms and cognitive vulnerabilities of achievement and dependency. *Cognitive Therapy and Research, 15*(3), 221–235. https://doi.org/10.1007/BF01173015

R Core Team. (2022). *R: A language and environment for statistical computing*. R Foundation for Statistical Computing.

Reinecke, A., Becker, E. S., Hoyer, J., & Rinck, M. (2010). Generalized implicit fear associations in generalized anxiety disorder. *Depression and Anxiety, 27*(3), 252–259. https://doi.org/10.1002/da.20662

Reiss, S., Peterson, R. A., Gursky, D. M., & McNally, R. J. (1986). Anxiety sensitivity, anxiety frequency and the prediction of fearfulness. *Behaviour Research and Therapy, 24*(1), 1–8. https://doi.org/10.1016/0005-7967(86)90143-9

Richards, J. C., Austin, D. W., & Alvarenga, M. E. (2001). Interpretation of ambiguous interoceptive stimuli in panic disorder and nonclinical panic. *Cognitive Therapy and Research, 25*(3), 235–246. https://doi.org/10.1023/A:1010783427196

Robinaugh, D., Haslbeck, J., Waldorp, L., Kossakowski, J., Fried, E. I., Millner, A., McNally, R. J., Ryan, O., Ron, J. de, Maas, H. van der, Nes, E. H. van, Scheffer, M., Kendler, K. S., & Borsboom, D. (2019). *Advancing the network theory of mental disorders: A computational model of panic disorder*. PsyArXiv. https://doi.org/10.31234/osf.io/km37w

RStudio, Inc. (2016). *RStudio: Integrated Development for R*. http://www.rstudio.com/

Sansone, R. A., & Sansone, L. A. (2009). Panic Disorder Subtypes. *Psychiatry, 6*(8), 33–37.

Schneider, R., & Schulte, D. (2007). Panic patients reveal idiographic associations between anxiety symptoms and catastrophes in a semantic priming task. *Behaviour Research and Therapy, 45*(2), 211–223. https://doi.org/10.1016/j.brat.2006.02.007

Schniering, C. A., & Rapee, R. M. (1997). A test of the cognitive model of panic: Primed lexical decision in panic disorder. *Journal of Anxiety Disorders, 11*(6), 557–571. https://doi.org/10.1016/s0887-6185(97)00029-7

Solmi, M., Radua, J., Olivola, M., Croce, E., Soardo, L., Salazar de Pablo, G., Il Shin, J., Kirkbride, J. B., Jones, P., Kim, J. H., Kim, J. Y., Carvalho, A. F., Seeman, M. V., Correll, C. U., & Fusar-Poli, P. (2022). Age at onset of mental disorders worldwide: Large-scale meta-analysis of 192 epidemiological studies. *Molecular Psychiatry, 27*(1), 1. https://doi.org/10.1038/s41380-021-01161-7

Steinman, S. A., & Teachman, B. A. (2010). Modifying interpretations among individuals high in anxiety sensitivity. *Journal of Anxiety Disorders, 24*(1), 71–78. https://doi.org/10.1016/J.JANXDIS.2009.08.008

Taylor, S., Zvolensky, M. J., Cox, B. J., Deacon, B., Heimberg, R. G., Ledley, D. R., Abramowitz, J. S., Holaway, R. M., Sandin, B., Stewart, S. H., Coles, M., Eng, W., Daly, E. S., Arrindell, W. A., Bouvard, M., & Cardenas, S. J. (2007). Robust dimensions of anxiety sensitivity: Development and initial validation of the Anxiety Sensitivity Index-3. *Psychological Assessment, 19*(2), 176–188. https://doi.org/10.1037/1040-3590.19.2.176

Teachman, B. A. (2005). Information processing and anxiety sensitivity: Cognitive vulnerability to panic reflected in interpretation and memory biases. *Cognitive Therapy and Research, 29*(4), 479–499. https://doi.org/10.1007/s10608-005-0627-5

Teachman, B. A., Smith-Janik, S. B., & Saporito, J. (2007). Information processing biases and panic disorder: Relationships among cognitive and symptom measures. *Behaviour Research and Therapy, 45*(8), 1791–1811. https://doi.org/10.1016/j.brat.2007.01.009

Wigboldus, D. H. J., Holland, R. W., & van Knippenberg, A. (2004). Single target implicit associations. *Unpublished Manuscript*.

Wilhelm, F. H., Gerlach, A. L., & Roth, W. T. (2001). Slow recovery from voluntary hyperventilation in panic disorder. *Psychosomatic Medicine, 63*(4), 638–649. https://doi.org/10.1097/00006842-200107000-00017

Woud, M. L., Becker, E. S., Rinck, M., Harmer, C. J., & Reinecke, A. (2016). Assessment of automatic associations with bodily sensations and agoraphobic situations in panic disorder. *Journal of Behavior Therapy and Experimental Psychiatry, 52*, 105–109. https://doi.org/10.1016/j.jbtep.2016.04.001

Woud, M. L., Ellwart, T., Langner, O., Rinck, M., & Becker, E. S. (2011). Task-irrelevant spider associations affect categorization performance. *Journal of Behavior Therapy and Experimental Psychiatry, 42*(3), 309–316. https://doi.org/10.1016/j.jbtep.2010.12.008

Woud, M. L., Zhang, X. C., Becker, E. S., McNally, R. J., & Margraf, J. (2014). Don't panic: Interpretation bias is predictive of new onsets of panic disorder. *Journal of Anxiety Disorders, 28*(1), 83–87. https://doi.org/10.1016/j.janxdis.2013.11.008

Würtz, F., Steinman, S., Blackwell, S. E., Wilhelm, F. H., Reinecke, A., Adolph, D., Margraf, J., & Woud, M. L. (2023). Effects of training

body-related interpretations on panic-related cognitions and symptoms. *Cognitive Therapy and Research, 47*(6), 494–509. https://doi.org/10.1007/s10608-023-10358-9

Zahler, L., Sommer, K., Reinecke, A., Wilhelm, F. H., Margraf, J., & Woud, M. L. (2020). Cognitive vulnerability in the context of panic: Assessment of panic-related associations and interpretations in individuals with varying levels of anxiety sensitivity. *Cognitive Therapy and Research, 44*(4), 858–873. https://doi.org/10.1007/s10608-020-10103-6