

UC Davis

UC Davis Previously Published Works

Title

Applying the Quadruple Process model to evaluate change in implicit attitudinal responses during therapy for panic disorder

Permalink

<https://escholarship.org/uc/item/2h65q04q>

Journal

Behaviour Research and Therapy, 52(1)

ISSN

0005-7967

Authors

Clerkin, Elise M
Fisher, Christopher R
Sherman, Jeffrey W
et al.

Publication Date

2014

DOI

10.1016/j.brat.2013.10.009

Peer reviewed



Applying the Quadruple Process model to evaluate change in implicit attitudinal responses during therapy for panic disorder



Elise M. Clerkin^{a,*}, Christopher R. Fisher^a, Jeffrey W. Sherman^b, Bethany A. Teachman^c

^a Department of Psychology, Miami University, Oxford, OH, USA

^b Department of Psychology, University of California, Davis, CA, USA

^c Department of Psychology, University of Virginia, Charlottesville, VA, USA

ARTICLE INFO

Article history:

Received 14 June 2013

Received in revised form

23 September 2013

Accepted 29 October 2013

Keywords:

Quadruple Process model

Panic disorder

Implicit

Associations

Cognitive-behavioral group therapy

ABSTRACT

Objective: This study explored the automatic and controlled processes that may influence performance on an implicit measure across cognitive-behavioral group therapy for panic disorder.

Method: The Quadruple Process model was applied to error scores from an Implicit Association Test evaluating associations between the concepts Me (vs. Not Me) + Calm (vs. Panicked) to evaluate four distinct processes: Association Activation, Detection, Guessing, and Overcoming Bias. Parameter estimates were calculated in the panic group ($n = 28$) across each treatment session where the IAT was administered, and at matched times when the IAT was completed in the healthy control group ($n = 31$).

Results: Association Activation for Me + Calm became stronger over treatment for participants in the panic group, demonstrating that it is possible to change automatically activated associations in memory (vs. simply overriding those associations) in a clinical sample via therapy. As well, the Guessing bias toward the calm category increased over treatment for participants in the panic group.

Conclusions: This research evaluates key tenets about the role of automatic processing in cognitive models of anxiety, and emphasizes the viability of changing the actual activation of automatic associations in the context of treatment, versus only changing a person's ability to use reflective processing to overcome biased automatic processing.

© 2013 Elsevier Ltd. All rights reserved.

One of the biggest challenges when treating panic and other anxiety disorders is the seeming disconnect between what people report “knowing” at a more controlled, strategic level, versus what they report “experiencing” at a more automatic, uncontrollable level. This fundamental discrepancy has led researchers to posit that relatively automatic processing (e.g., processing that is outside one's conscious control or awareness) is critical in understanding the fear and anxiety response (e.g., Mathews & MacLeod, 1994; McNally, 1995; Williams, Watts, MacLeod, & Mathews, 1997). For instance, Beck and Clark (1997) theorize that anxiety problems result in part from the activation of a relatively automatic, reflexive “primal threat mode,” which is followed by a more strategic and elaborative form of cognitive processing. Indeed, Beck and Clark (1997) suggest that deactivating biased automatic processing, while activating more adaptive forms of thinking, is the critical goal when treating anxiety problems.

Several reviews have demonstrated that automatic processing biases are common and predict important outcomes among people with anxiety problems (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007; Teachman, Joormann, Steinman, & Gotlib, 2012). However, one major limitation with our current understanding of the role of automatic processing in anxiety pathology is that the measurement of automatic processes is not process pure (Conrey, Sherman, Gawronski, Hugenberg, & Groom, 2005; Sherman, 2009; Sherman et al., 2008). Instead, as implicit social cognition researchers have convincingly articulated, these measures capture the combined contributions of several, qualitatively different processes, including ones that are relatively controlled in nature (Conrey et al., 2005; Sherman et al., 2008; Sherman, Klauer, & Allen, 2010). In other words, simply using an indirect measure, like the Implicit Association Test (IAT, Greenwald, McGhee, & Schwartz, 1998), does not permit strong conclusions about the relative influences of automatic versus relatively more controlled components of processing.

Along these lines, Teachman, Marker, and Smith-Janik (2008) recently investigated implicit panic associations, or interconnected associations in memory that are difficult to consciously

* Corresponding author. Miami University, Psychology Department, 100 Psychology Building, Oxford, OH 45056, USA.

E-mail address: clerkie@miamioh.edu (E.M. Clerkin).

control, measured with the IAT. To evaluate whether change in panic-relevant IAT scores [i.e., associations between Me (vs. Not Me) + Calm (vs. Panicked)] led to subsequent change in panic symptoms across a 12-week course of cognitive-behavioral group therapy for panic disorder, researchers used dynamic bivariate latent difference score modeling. This test allows one to model both the change processes across the two variables, as well as the relationship between those change processes (see McArdle & Nesselrode, 2002). Given the sample size, the researchers constrained the change process to be the same over time to test whether one change process was a leading indicator of another change process, but this test did not address the question of exactly when in treatment the change was most predictive. Results indicated that changes in panic-relevant IAT scores predicted the degree of subsequent symptom change. This work was exciting because it suggests that change in cognition, including cognition that is activated at a relatively automatic level, occurs in advance of and predicts the extent of symptom reduction among patients with panic disorder. However, given that the IAT captures both relatively automatic and controlled components of anxious processing, it is not clear what components of the IAT change were driving the results in this study. For example, it could be that automatic, panic-relevant associations were being altered, or it could be that patients were becoming better at regulating these automatically activated associations. Given the mounting research demonstrating that implicit associations assessed with the IAT can be modified in a clinical context (e.g., Clerkin & Teachman, 2010; Teachman et al., 2008; Teachman & Woody, 2003; see review in Roefs et al., 2011), it is critical to understand the underlying processes driving these changes.

The current study seeks to better understand what aspects of automatic and controlled processing change over the course of treatment by applying the Quadruple Process or Quad model to a subset and extension of Teachman et al.'s (2008) data. Specifically, this study seeks to test the components of implicit attitudinal responses that change in response to a full dose of treatment, in addition to comparing differences in Quad model parameter estimates between a group diagnosed with panic disorder and a healthy control group. Ultimately, applying the Quad model is important because it enables a refined test of the underlying processes driving overall IAT effects.

The Quad model

The Quad model is a multinomial processing tree model that has been validated across a variety of tasks, including the IAT (Conrey et al., 2005; Sherman et al., 2008; see Methodological detail on the IAT, below). Similar to other mathematical modeling approaches (e.g., Control Default model: Jacoby, 1991; Diffusion model: Klauer, Voss, Schmitz, & Teige-Mocigemba, 2007; Process Dissociation model: Payne, 2008), applications of the Quad model are part of a growing recognition that crude classifications of implicit versus explicit dimensions likely miss critical distinctions within measures of cognitive processing. Specifically, the Quad model decomposes implicit task performance into four interdependent, but distinct processes: 1) Activation of Associations (AC) refers to the degree to which biased associations are activated when responding to a stimulus. All else being equal, the stronger the associations, the more likely they are to be activated and to influence behavior. In the current context, AC is measured with two parameters. Specifically, $AC_{me + calm}$ measures the degree to which an association between Me (i.e., the self) and Calm is activated, whereas $AC_{not\ me + panicked}$ measures the degree to which an association between Not Me (i.e., others) and Panicked is activated. 2) Detection (D) corresponds to a more controlled process that

enables detection of correct and incorrect responses (note that "Detection" is conceptually the same as the earlier "Discriminability" parameter outlined by Conrey et al., 2005). 3) Guessing (G) reflects a general response bias when no associations are activated and the correct response cannot be determined. 4) Finally, at times there is a conflict between automatically activated associations and the response detected as correct. In this case, the Quad model proposes that a self-regulatory process may override the influence of automatically activated associations. This self-regulatory process is referred to as the Overcoming Bias (OB) parameter.

In the present study, we explored the relative influence of each of the four Quad model parameters on implicit panic responses during therapy for panic disorder. Given our goal of decomposing the basic IAT effect into relatively automatic and controlled forms of processing, in this initial application of the Quad model to implicit panic data, it was important to consider all the parameters that reflect these different types of processes. For instance, it is possible that an overall IAT effect is driven by changes in the ability to detect the correct response, differences in the ability to overcome automatic or habitual associations, or differences in response bias toward a certain category.

With this in mind, we first, we evaluated whether $AC_{me + calm}$ and $AC_{not\ me + panicked}$ would increase over the course of treatment, reflecting greater activation of an automatic association between the self and calm. This test provides a critical opportunity to determine whether it is actually the automatically activated associations that are shifting over treatment, as suggested by cognitive models of anxiety treatment (e.g., Beck & Clark, 1997), distinct from the more controlled processing that the IAT also captures. Our modeling approach also allowed examination of changes in Detection and Guessing across treatment, and as a function of group status (panic vs. healthy control). There were no specific hypotheses for changes in Detection, other than possible improvement over time with practice. With respect to the Guessing parameter, it seemed plausible that the response bias would be relatively more oriented toward panicked (vs. calm) among the panic group compared to the healthy control group prior to treatment. Whether this Guessing parameter would shift following treatment was more exploratory.

Finally, recall that the OB parameter reflects an override of an automatic or habitual response. Given the structure of these data, the OB parameter in this study represents the ability to overcome the tendency to associate oneself with calm.¹ Thus, for OB to reduce one's implicit panic associations in this context, participants would have needed to lose their self-regulatory abilities across treatment, which does not seem highly plausible given the intention in treatment to gain self-regulatory skills (Barlow & Craske, 1994).

Therefore, this study was well designed to evaluate whether relatively pure measures of activation, detection, and guessing change over treatment, but was not as well-suited to test whether override responses change. Critically, the advantage of applying the Quad Model to basic IAT data is that it is possible to learn how different automatic and strategic facets of implicit associations change in response to treatment, rather than evaluating only a single general effect.

¹ As discussed in Teachman et al. (2007), individuals with panic disorder have relatively stronger panicked (versus calm) associations with the self (versus others) compared to a healthy control group, as measured with the IAT. However, both groups still have a stronger absolute association between the self and calm, in the sense that they are generally quicker to respond to words when "Me + Calm" are paired, as compared to "Me + Panicked." Given this, "Me + Calm" is the compatible pairing in this data (as opposed to "Me + Panicked," which is the incompatible pairing). Hence, the OB parameter reflects overcoming the tendency to associate oneself with calm.

Method

Participants

Participants from this study were part of a larger project evaluating adult outpatients who participated in a 12-week cognitive-behavioral group therapy for panic disorder, as well as a healthy control group (for additional discussion of the panic and healthy control group samples, and details on diagnostic inter-rater reliability, see Teachman et al., 2008; Teachman, Smith-Janik, & Sap- orito, 2007). Given our interest in evaluating trajectories of change over treatment, only participants who completed at least 8 sessions of treatment were included in the panic group, to insure that participants had received an adequate dose of therapy (consistent with Clerkin, Smith-Janik, & Teachman, 2008). Additionally, given that the Quad model parameters are based on error rates, two participants with extreme outliers in error rates (i.e., error rates greater than three times the standard deviation of the error rate for the IAT across sessions for the panic disorder sample) were excluded from analyses. This resulted in a final sample of 28 participants (Mean Age = 41.39, SD = 14.93, Range = 18–71; 67.9% female; 89.3% reported race as Caucasian, 7.1% as Black, 3.6% as “Other”) with a primary diagnosis of panic disorder, as assessed with the Structured Clinical Interview for DSM-IV diagnoses (First et al., 1995).²

Participants in the healthy control group were assessed with the MINI International Neuropsychiatric Interview (MINI Plus, version 5.0, 2003; original by Sheehan, Lecrubier, Sheehan, Amorim, & Janavs, 1998). This brief diagnostic interview was used to assess inclusion/exclusion criteria to confirm that participants in the healthy control group did not have a current or past anxiety disorder, a current eating or psychotic disorder, or a substance use disorder diagnosis during the previous year. Given our interest in evaluating change over time consistent with the time the panic group spent in treatment, only participants who completed both testing sessions (baseline and post-treatment) were included in analyses. As well, two participants were excluded who had error rates greater than three times the standard deviation of the error rate for the IAT across sessions for the healthy control sample. This resulted in a final sample of 31 control participants (Mean Age = 33.16, SD = 17.40, Range = 18–78; 54.8% female; 93.5% reported race as Caucasian, and 2 participants had missing data for race).

Chi-square tests indicated that the panic and healthy control groups were not significantly different across race ($\chi^2(2) = 3.28$, $p > .10$) or sex ($\chi^2(1) = 1.05$, $p > .10$). As well, an independent samples t -test indicated that groups did not significantly differ by age ($t_{57} = 1.94$, $p = .06$, Cohen’s $d = .51$).

Treatment

All participants in the panic group completed a 12-week group treatment based on the manualized Panic Control Treatment manual (Barlow & Craske, 1994). Treatment, which involved structured, 90-min weekly sessions, consisted of groups of 4–6 participants diagnosed with panic disorder. A range of topics were covered, including psychoeducation about panic symptoms and the “fear of fear” model, cognitive restructuring that focused on re-

evaluating thoughts about the catastrophic nature of changes in bodily sensations and beliefs about personal vulnerability to panic, exposures (both interoceptive to address fear of bodily sensations, and in the environment to return to situations that had been avoided), and relaxation training to reduce resting levels of anxiety. Each group was co-led by a licensed clinical psychologist (the fourth author), or a doctoral student who received extensive training and supervision. For additional detail on the treatment, therapist training and supervision, participant recruitment and attrition, see Teachman et al. (2008).

Measures³

Implicit panic responses were evaluated with the *Implicit Association Test* (IAT; Greenwald et al., 1998). The IAT has adequate psychometric properties (Greenwald & Nosek, 2001), including good test–retest reliability (Bosson, Swann, & Pennebaker, 2000). Previous papers have also demonstrated that this version of the IAT has high split-half reliability (Teachman et al., 2008), as well as good construct validity (Teachman et al., 2007). Specifically, at baseline, participants diagnosed with panic disorder had relatively lower self + calm (and correspondingly greater self + panicked) implicit associations as compared to a healthy control group.

Similar to many measures used by social cognition researchers, the IAT is a reaction time task that is meant to evaluate the relative strength of association between two concepts in a person’s memory (Fazio & Hilden, 2001). The task compares the time taken to classify stimuli into superordinate categories that have been paired together, based on the idea that classification will be more rapid and involve fewer errors when the paired categories are automatically associated in memory vs. when the categories are not associated. Used in this context, “pairing” refers to the mapping of categories to response keys (e.g., the categories “Panicked,” and “Me” can be paired together on the left side of the screen so that items from either category are classified by the same keyboard letter, indicating “left” side).

Categories in the present study were words reflecting the concepts of “Calm” versus “Panicked,” and “Me” versus “Not Me.” The primary IAT outcome (i.e., the IAT D score) reflects the time it takes to classify stimuli when paired categories are congruent with another (e.g., when “me” is paired with “calm”) versus when paired categories are incongruent (e.g., “me” is paired with “panicked”). Category labels and stimuli in the present study were as follows: Panicked (Panicked, Scared, Anxious); Calm (Calm, Relaxed, Serene); Me (Me, Self, I); and Not Me (Not Me, Other, Them). For additional detail on the IAT measure used in this study, please see Teachman et al. (2007, 2008).

Panic symptoms were evaluated with the *Panic Disorder Severity Scale* (PDSS; Shear et al., 1997). The PDSS is a 7-item measure that evaluates the frequency, distress, and impairment associated with panic disorder. The self-report version has good psychometric properties and is sensitive to treatment changes (Houck, Spiegel, Shear, & Rucci, 2002; see also Otto, Pollack, Penava, & Zucker, 1999; Teachman, 2005). In the current study, it was implemented as a self-report scale, with total possible scores on the PDSS ranging from 0 to 28. Note that the PDSS was slightly modified in the present study to include a definition of “panic attacks,” so participants could more accurately complete the scale as a self-report measure.

² Note that the demographic characteristics for the panic group in this sample (i.e., those panic participants who had completed most treatment sessions) are comparable to those reported in Teachman et al. (2008): $N = 43$, Mean Age = 40.14, SD = 15.17, Range = 18–71; 70% female; 90.7% reported race as Caucasian, 4.7% as Black, and 2.3% as “Other”.

³ Only measures directly relevant to the study’s current hypotheses are included here. For details on the larger study evaluating cognitive processing in the context of panic disorder, see Teachman et al. (2007, 2008), or contact the fourth author.

Procedure

Participants in the panic group completed the PDSS at the start of every therapy session to provide a weekly measure of panic symptoms. They also completed the IAT at a testing session held immediately prior to session 1, and then following sessions 3, 6, 9, and 12. Participants in the healthy control group completed the IAT at baseline, and at 12-week follow-up (corresponding to the 12 weeks of the treatment protocol). Note that we were not testing the efficacy of this treatment given its established strong outcomes, on average, across numerous trials (see meta-analysis by [Gould, Otto, & Pollack, 1995](#)); hence, we did not include a full waitlist control group. Note that all procedures were approved by the university's Institutional Review Board, and all participants consented to participate.

Data analytic plan

The Quad model uses the frequency of correct and incorrect responses to measure four interdependent, but distinct underlying processes: the Activation of Associations ($AC_{me + calm}$ and $AC_{not me + panicked}$), Detection (D), response bias toward the calm category (G), and Overcoming Bias (OB). As shown in [Fig. 1](#), the Quad model can be conceptualized as a processing tree in which a series of processes are linked to responses via a many-to-one mapping. Each branch in the processing tree represents a likelihood, and each parameter represents the probability that a given process will be engaged, conditional upon the preceding processes. For example, Overcoming Bias (OB) is conditional upon the processes of both Activation of Associations (AC) and Detection (D).

For illustration, [Fig. 1](#) provides an example of an incompatible trial where the Me + Calm association is automatically activated, but the category label Me is paired with the label Panicked on the screen. Consider the top branch in the figure (highlighted in light gray) leading to a correct response on this incompatible trial; incompatible in that there is a conflict between activated associations (Me + Calm) and the detected correct response (Me + Panicked). When a stimulus that belongs to the Me category is presented, an association between Me and Calm will be activated with probability $AC_{me + calm}$. There also is a probability D that the correct response will be detected. Detection is a controlled process that discriminates between the correct and incorrect response (correct here refers to the required left/right classification for a correct response on the task). If the correct response is detected,

there is a probability OB that the response driven by the association will be overridden and the correct response will be chosen instead. The probability that this top branch will be followed is the product of the probabilities within the branch: $(AC_{me + calm})(D)(OB)$. (Note, the probability of G is not discussed in this example because it reflects a response bias toward the calm category when the other processes are not engaged. Given the Me + Calm association was activated, detection of the correct response occurred, and this bias was overcome, G is not represented in this branch.) The overall probability of a correct response can be found by summing the probability of each branch in [Fig. 1](#) leading to a correct response: $P(\text{Correct}) = (AC_{calm})(D)(OB) + (1 - AC_{calm})(D) + (1 - AC_{calm})(1 - D)(1 - G)$.

Given our interest in evaluating group differences and change that may influence implicit task performance, the Quad model was fit to the IAT data using multiTree, a specialized program for multinomial processing tree models ([Moshagen, 2010](#)). The parameters of the Quad model were estimated through maximum likelihood estimation. In particular, the branches in [Fig. 1](#) form a system of equations that predict correct and incorrect response frequencies across compatible (e.g., Me + Calm) and incompatible (e.g., Me + Panicked) trials. MultiTree uses a search algorithm to adjust the parameters simultaneously until the discrepancy between the predicted and observed frequencies is minimized, as measured by the G^2 statistic. For more complete information about the data analysis for the Quad model, please refer to [Conrey et al. \(2005\)](#) and [Sherman et al. \(2008\)](#). A hierarchical modeling approach was used for testing differences in parameters ([Moshagen, 2010](#)). First, a baseline model was fit to the data in which all parameters were free to vary. Next, hypothesis testing on model parameters was conducted by constraining parameters of interest to be equal. According to the logic of hierarchical model testing, differences in parameters may be inferred when fit of the constrained model decreases relative to the baseline model. In the present study, we used two indicators to assess model fit: 1) the likelihood ratio test (G^2), which compares the observed response frequencies to the expected response frequencies predicted by the model ($p > .05$ indicates an acceptable fit); and 2) the effect size w , an r -family measure that helps to account for large numbers of responses in aggregated data ($w \leq .05$ indicates an acceptable fit).

We provided converging support for our hypotheses by first comparing parameters aggregated across participants in the healthy control group versus the panic group (between-subjects effects), and then examining the trajectory of key aggregated

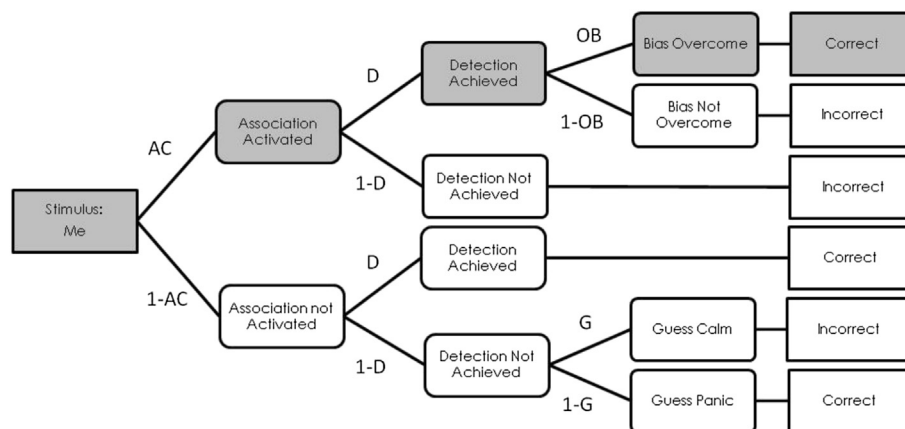


Fig. 1. The Quadruple Process (Quad) model of performance on an implicit task on an incompatible trial (where the Me + Calm association is automatically activated in memory, but Me is paired with Panicked on the screen). Note. Each branch represents a likelihood. All parameters are conditional upon the preceding parameters. In this diagram, AC = activation of habitual associations, or $AC_{me + calm}$; D = detection; OB = overcoming bias; G = guessing.

parameters over the course of treatment for the panic group (within-subjects effects). Last, parameters were calculated for each individual participant in the panic group, so we could evaluate whether the change in slope for parameters was associated with the change in slope for panic symptoms. As well, we conducted a time-lagged analysis to evaluate the temporal precedence of the Association Activation for Me + Calm predicting panic symptoms.

Results

Quad model application: between-subjects comparisons

The healthy control and panic groups were compared on Quad model parameters ($AC_{me + calm}$, $AC_{me + panicked}$, D , G , and OB) at the pre-treatment and post-treatment sessions, the only sessions for which data were available for the healthy control group. Before comparing the healthy control and panic groups, we first fit the baseline models for both groups. The Quad model fits the data well for the panic group, $G^2(df = 6) = 4.94, p = .55, w = .03$. Although the model did not fit the data as well for the healthy control group, the effect size indicated that the fit was acceptable, $G^2(df = 6) = 19.16, p < .01, w = .05$. See Table 1 for best fitting parameters.

Prior to treatment, $AC_{me + calm}$ was higher for the healthy control group compared to the panic group, $\Delta G^2(df = 1) = 10.65, p = .001, w = .03$, as expected. Critically, this difference was no longer statistically significant at post-treatment, $\Delta G^2(df = 1) = .09, p = .76, w < .01$. See Fig. 2. Surprisingly, while the healthy control group was not higher than the panic group at pre-treatment on $AC_{not me + panicked}$, $\Delta G^2(df = 1) < .01, p = .97, w < .01$, the healthy control group showed a stronger Not Me + Panicked association at post-treatment relative to the panic group, $\Delta G^2(df = 1) = 5.99, p = .01, w = .02$.

D was approximately equal for the healthy control group and the panic group at pre-treatment, $\Delta G^2(df = 1) = 1.06, p < .30, w < .01$. At post-treatment, D was higher for the panic group compared to the healthy control group, $\Delta G^2(df = 1) = 15.46, p < .01, w = .03$. Further, at pre-treatment, G was higher for the healthy control group compared to the panic group, $\Delta G^2(df = 1) = 8.71, p = .03, w = .03$, indicating a greater response bias toward “calm” in the healthy control group, as expected. However, there were no significant differences in the G parameter at post-treatment, $\Delta G^2(df = 1) = .66, p = .42, w < .01$. Finally, there were no significant group differences at pre-treatment ($\Delta G^2(df = 1) = .73, p = .39, w < .01$) or post-treatment ($\Delta G^2(df = 1) = .00, p = 1, w < .01$) on the OB parameter.

In sum, while $AC_{me + calm}$ and G were higher for the healthy control (versus panic) group prior to treatment, these differences were not statistically significant following treatment. In contrast, the healthy control group showed a stronger Not Me + Panicked

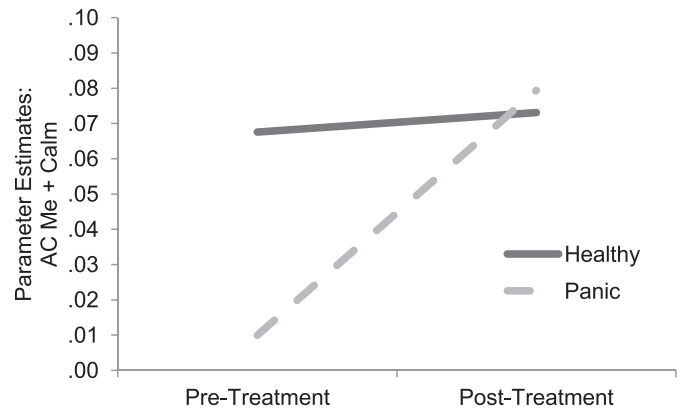


Fig. 2. Between-subjects comparisons of $AC_{me + calm}$ for the healthy control versus panic groups at pre- and post-treatment.

association at post-treatment relative to the panic group. Meanwhile, D was approximately equal across groups at pre-treatment, but higher for the panic (versus control) group following treatment. Finally, there were not significant group differences for OB at pre- or post-treatment.

Quad model application: trajectory of change over treatment, within-subjects comparisons within the panic group

To examine changes in model parameters over the course of treatment, the Quad model was applied to data across all five sessions (1, 3, 6, 9, and 12) for the panic group. The data were aggregated across all panic group participants for each assessment point before fitting the model. The model fits the data well, $G^2(df = 15) = 17.46, p = .29, w = .03$.

As expected, $AC_{me + calm}$ increased over the course of treatment, $\Delta G^2(df = 4) = 17.63, p < .01, w = .03$. See Fig. 3. Contrary to expectations, however, $AC_{not me + panicked}$ did not increase over the course of treatment, $\Delta G^2(df = 4) = 2.79, p = .59, w = .01$. As well, D did not change over the course of treatment, $\Delta G^2(df = 4) = 7.52, p = .11, w = .02$. Interestingly, G increased over the course of treatment, indicating that responses became more biased toward responding “Calm,” $\Delta G^2(df = 4) = 9.49, p = .05, w = .024$. Not surprisingly, given OB reflected an ability to override a calm association, there was no detectable change for OB over the course of treatment, $\Delta G^2(df = 4) = 2.33, p = .67, w = .01$.

In sum, the $AC_{me + calm}$ and G parameters increased over treatment, whereas there was no significant change for the $AC_{not me + panicked}$, D , or OB parameters within the panic group across treatment.

Table 1
Best fitting Quadruple Process (Quad) model parameter estimates and confidence intervals.

Assessment point	Healthy control group		Panic group				
	Pre	Post	Pre	3	6	9	Post
Parameter							
$AC_{me + calm}$.07 (.04–.09)	.07 (.04–.10)	.01 (.00–.03)	.03 (.01–.05)	.03 (.01–.06)	.04 (.02–.06)	.08 (.05–.11)
$AC_{not me + panicked}$.05 (.02–.07)	.08 (.05–.11)	.05 (.02–.07)	.03 (.01–.05)	.03 (.01–.05)	.02 (.00–.04)	.03 (.01–.05)
D	.94 (.92–.95)	.93 (.91–.95)	.95 (.93–.96)	.97 (.96–.98)	.97 (.95–.98)	.97 (.96–.98)	.97 (.95–.97)
G	.63 (.52–.73)	.52 (.41–.63)	.38 (.28–.51)	.51 (.35–.68)	.43 (.28–.62)	.67 (.51–.84)	.59 (.43–.74)
OB	.91 (.59–1.0)	1.00 (.86–1.0)	.67 (.00–1.0)	1.00 (.76–1.0)	.94 (.46–1.0)	.89 (.47–1.0)	1.00 (.88–1.0)

Note. Parameter estimates were calculated for participants in the panic group across each treatment session where the IAT was administered (1, 3, 6, 9, and 12), and at matched times when the IAT was administered in the healthy control group (sessions 1 and 12). Parameter estimates are probabilities, ranging from 0 to 1, with 95% confidence intervals presented in parentheses. To keep the confidence intervals within the acceptable probability interval (0–1), confidence intervals were obtained from $N = 1000$ non-parametric bootstrap samples. Non-parametric bootstrapping involves resampling from the data with replacement, fitting the model to the resampled data and repeating the process a large number of times (see Moshagen, 2010 for further detail).

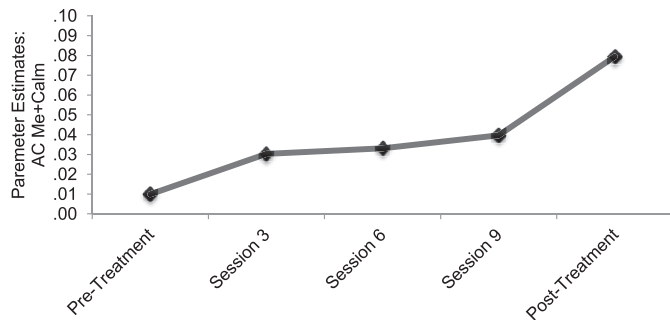


Fig. 3. Trajectory of change for $AC_{me + calm}$ across assessment points for the panic group.

Quad model application: relationships among change in individual parameter estimates and panic symptoms within the panic group

We evaluated the extent to which the change in slope for $AC_{me + calm}$ was associated with the change in slope for panic symptoms (assessed with the PDSS), within participants in the panic group. Specifically, for each individual participant, we fit the Quad model to estimate individual parameter estimates [across participants, the mean G^2 ($df = 15$) was 8.68 ($p > .05$ for 27 out of 28), indicating an acceptable fit]. Next, we calculated a growth coefficient across sessions 1, 3, 6, 9, and 12 for the panic symptoms and the $AC_{me + calm}$ parameter estimates. The $AC_{me + calm}$ estimates were logit transformed before the calculation in order to help account for the skewed distribution. These growth coefficients reflected the linear change of the panic symptoms and the transformed $AC_{me + calm}$ in slope across treatment. The relationship between the slopes for $AC_{me + calm}$ and PDSS was in the anticipated direction and indicated a small effect, with greater increase of Me + Calm being associated with greater decrease of panic symptoms on the PDSS ($r = -.20$, $p = .31$); however, the effect did not reach significance. Note, it was not possible to evaluate the relationship between the slopes of the G parameter (the other parameter showing within-subjects change over treatment) and PDSS symptoms because nearly every participant had many curves in their trajectory of the G parameter over treatment. Hence, a linear growth model was unable to account for the change in the G parameter because the slope of the G parameter was not linear (slope = 0).

Next, we conducted a time-lagged analysis that evaluated the temporal precedence of the Association Activation for Me + Calm predicting panic symptoms. Specifically, we computed a linear growth coefficient across sessions 1, 3, and 6 for the $AC_{me + calm}$ parameter estimate. This provided us with an estimate of change over the first half of therapy. Again, the $AC_{me + calm}$ estimates were logit transformed before the calculation. Next, we conducted a partial correlation between the slope of $AC_{me + calm}$ across the first half of treatment and an average of panic symptom scores at sessions 9 through 12, controlling for baseline panic symptom scores. We took an average of panic scores at sessions 9 through 12 to have a more reliable indicator of panic symptom outcomes. In line with earlier analyses, there was a small, non-significant relationship between the slope of change in $AC_{me + calm}$ over the first half of treatment and later panic symptoms ($r = -.19$, $p = .36$).

Discussion

This research represents the first study to our knowledge that systematically applies the Quad model to a clinically anxious sample undergoing treatment, which allowed us to simultaneously

explore the underlying processes implicated in implicit panic responses.

Specifically, the present study applied the Quad model to evaluate four distinct processes that may influence implicit task performance: Association Activation, Detection, Guessing, and Overcoming Bias. To provide convergent evidence, we examined both between-subjects (panic vs. healthy control group differences in parameter estimates) and within-subjects (changes in parameter estimates over the course of panic treatment for individuals in the panic group) effects. As well, we explored the extent to which the slope of $AC_{me + calm}$ within individual participants in the panic group was associated with and predicted the slope of symptom change.

The most robust finding in the present study was the change in the $AC_{me + calm}$ parameter. First, compared to healthy participants, individuals in the panic group had a weaker association between Me + Calm at baseline. However, there was no significant group difference in $AC_{me + calm}$ following session 12. Moreover, within-subjects findings indicated that the association between Me + Calm strengthened over treatment for participants in the panic group. Together, these findings provide one of the cleanest empirical demonstrations to date that it is possible to change automatically activated associations in a clinical sample via therapy. While previous research has demonstrated change in overall IAT effects across therapy (see Teachman et al., 2008), it is not clear to what extent those effects reflected change in activation of associations and/or only change in the ability to override the biased activation. Applying the Quad model is therefore valuable because it allows a more nuanced test of the underlying processes driving changes in IAT effects.

Critically, automatic associations may share some similarities with anxious schemas in the sense that they represent interconnected, relatively automatic associations in memory (see Clerkin & Teachman, 2010; Teachman et al., 2008; Teachman & Woody, 2004). Moreover, cognitive behavioral models of panic predict that anxious schemas, or fear networks, guide the ways in which people screen, code, and process information (e.g., Beck, Emery, & Greenberg, 1985). Hence, consistent with Beck and Clark's (1997) influential theory, the capacity to change these automatically activated associations is both theoretically and clinically meaningful.

It is important to highlight that while the $AC_{me + calm}$ parameter did appear to meaningfully change in the panic group, this change in slope was not significantly associated with or predictive of panic symptoms (as assessed with the PDSS). This finding warrants consideration given previous work demonstrating that changes in overall panic-relevant IAT scores [operationalized as associations between Me (vs. Not Me) + Calm (vs. Panicked)] predicted changes in panic symptoms (Teachman et al., 2008). There are a few possible ways to interpret this difference across studies, as well as the non-significant finding more generally. First, in the present study, the relationship between change in the $AC_{me + calm}$ parameter and change in symptoms was in the anticipated direction, with a small effect size ($r = -.20$). As well, in the time-lagged analysis, there was also a small effect for the slope in the $AC_{me + calm}$ parameter across the first half of treatment to predict later panic symptoms ($r = -.19$). It is possible that these effects are meaningful, but difficult to detect given our relatively small sample size. Indeed, our sample size of 28 for the panic group was considerably smaller than the sample size of 43 reported in Teachman et al. (recall that the reduced sample size in the current study was due to the differing aims of these projects; namely, we were interested in evaluating trajectories of change in Quad model parameters over treatment, so we wanted to insure that participants had received an adequate dose of therapy). It is also important to point out that maximum

likelihood estimation can over-estimate individual variability (e.g., Farrell & Ludwig, 2008; Wetzels, Vandekerckhove, Tuerlinckx, & Wagenmakers, 2010), which may have the effect of attenuating a possible correlation between the parameters and outcome variables.

Of course, it is also possible that $AC_{me + calm}$ is simply not strongly and/or reliably related to changes in self-reported panic symptoms. When considering this possibility, it is helpful to recall that the effect discussed in Teachman et al. was for the overall IAT effect, which was not decomposed into its component processes. Hence, it is possible that while the relatively pure form of automatic activation (in this case, the Association Activation for Me + Calm) may be influenced by treatment, the impact that this purer measure of automatic processing has on predicting degree of symptom reduction is less robust than the impact of the full IAT D score (which reflects the contributions of multiple processes in addition to association activation). When considered in this light, the current research is valuable because it highlights why simply evaluating the overall IAT effect may be less informative than exploring the underlying processes contributing to that effect.

Consistent with dual process theories (e.g., Wilson, Lindsey, & Schooler, 2000), it is also possible that change in the activation of automatic associations will be most predictive of those symptoms of panic that are less amenable to conscious control or awareness (e.g., psychophysiological markers of panic), as opposed to those that are more strategic in nature (e.g., a self-report measure of symptoms, as assessed in this study). Finally, there have been several recent studies that cast doubt on some tenets of cognitive models of anxiety and obsessive compulsive spectrum disorders. For example, recent work suggests that change in cognitions related to obsessive-compulsive disorder may not be causally related to symptom reduction (Woody, Whittal, & McLean, 2011; see also Olatunji et al., 2013). Clearly, more work is needed to understand the conditions under which cognitive change will predict symptom change, and perhaps equally importantly, the conditions under which cognitive change will not predict symptom change.

Interestingly, the $AC_{not\ me +\ panicked}$ parameter was significantly higher for participants in the healthy control (versus panic) group following session 12. This finding was surprising given the pattern of findings for $AC_{me +\ calm}$. One might suspect that the pattern of findings for the two AC parameters would be identical. That is, as people have a stronger activation of Me + Calm (which we saw in the present study), they might correspondingly have a stronger activation of Not Me + Panicked. This would reflect the notion that “I am calm, not panicked.” Although speculative, one reason why the AC parameters might have diverged in the present study concerns the possible construal of “Not me” by the panic group. Individuals undergoing group treatment had just learned how common panic actually is, and they spent a considerable amount of time with others who experienced panic. So, it is plausible that they conceptualized “not me” as “other people,” given the stimuli are designed to denote other people (e.g., “them”). In other words, activation of Not Me + Panicked associations may have been less about the negation of panic tied to the self (e.g., “I am not someone who panics”), and more about an increased recognition that panic affects others as well (e.g., “Other people are very likely to panic”). Regardless, it is clear that the relative nature of the IAT—and the corresponding category labels—can be particularly challenging to interpret in the context of clinical phenomena.

The challenges that come with the relative nature of the IAT were most highlighted by our investigation of the OB parameter. The OB parameter is conceptualized as a relatively controlled, self-regulatory process that prevents automatically activated associations from leading to an incorrect response. For example, the OB parameter might allow someone to overcome a habitual response,

like automatically equating White + Good, on an IAT that measures automatic associations of White (versus Black) with Bad (versus Good). In the present study, however, the OB parameter represented the ability to overcome the tendency to associate oneself with *calm* (a positive association). Hence, for OB to reduce one's implicit panic associations in this context, participants would have needed to *lose* their self-regulatory abilities across treatment, as opposed to *gaining* self-regulatory abilities across treatment. This seems implausible given that other research suggests that inhibitory learning, and developing the ability to overcome one's unhelpful automatic associations, is a critical component in recovering from anxiety problems (e.g., Craske et al., 2008). As well, the Panic Control Treatment used in this study is partly designed to enable individuals to develop self-regulatory skills, like explicitly restructuring unhelpful cognitions (Barlow & Craske, 1994). Finally, it is unlikely that self-regulation skills during IAT performance would diminish over time as one gained more experience performing the task.

In terms of the Detection or D parameter, healthy control and panic participants were approximately equal in terms of their ability to discriminate (i.e., determine the correct answer) at the beginning of treatment, but those in the panic (versus healthy control) group had a stronger D parameter at session 12. Meanwhile, within-subjects analyses revealed that the D parameter did not significantly change over the course of treatment for those in the panic group. We suspect that the increased detection for those in the panic (versus healthy control) group reflects the fact that this parameter is especially susceptible to practice effects, and the panic group had extra practice completing the IAT. Moreover, the within-subjects effect for the panic group presumably did not reach significance because those in the panic group started at such a high level in terms of their ability to detect (.95), pointing to a ceiling effect. According to Conrey et al. (2005), the D parameter is sensitive to motivation and the desire to succeed on the task.

Finally, the G parameter reflects a more general response bias in the absence of an automatically activated association or knowledge of the correct answer (Conrey et al., 2005; Sherman et al., 2008). Interestingly, at baseline, healthy (versus panic) participants were relatively more biased toward the calm category, whereas this group difference was not apparent following session 12 of treatment. Furthermore, within the panic group, it appeared that the relative bias toward the calm category increased over the course of treatment. This finding is intriguing, and is consistent with the notion that patients with anxiety problems generally have a bias toward negative or threatening information (e.g., Cloitre & Liebowitz, 1991; Maidenberg, Chen, Craske, Bohn, & Bystritsky, 1996; Teachman et al., 2012; Williams et al., 1997). Hence, this finding—that the relative bias toward calm increased for the panic group—may be meaningful. That said, it is important to clarify that the G parameter did not indicate that patients diagnosed with panic disorder had an initial, absolute bias toward panic. As well, we must be cautious in interpreting this finding given that when evaluated at the individual level, nearly every participant had many curves in their trajectory of change. This made it impossible to obtain a reliable growth coefficient for the G parameter at the individual level. Thus, it is possible that the aggregate-level data masked some unreliability and variability in this data.

Limitations

These data must be interpreted in light of several limitations. First, as noted, we were unable to evaluate the extent to which OB represented a self-regulatory process of overcoming a *negative* bias. Second, the healthy versus panic comparisons in this study are confounded by the number of times the IAT was performed, with

panic participants completing the IAT more than healthy control participants. Given that learning and practice can influence IAT effects, it is possible that the between-group differences we saw were influenced by the different number of times that the IAT was completed in the two groups. That being said, we did evaluate our data in several ways (e.g., within- and between-groups effects) in order to provide convergent evidence for change in the Quad model parameters. As well, while there is evidence that the magnitude of the overall IAT effect is reduced if an individual has prior experience completing an IAT, research suggests that this difference is being driven by whether an individual has 0–1 versus 2 + previous IAT completions (Greenwald, Nosek, & Banaji, 2003). In fact, when investigating the question of whether practice influences IAT performance, Greenwald et al. note: “Little or no further reduction in IAT scores occurred for two or more previous uses” (p. 203). Thus, given that both groups completed the IAT more than once, previous data suggest that there is likely not a large difference in practice effects across groups.

Another limitation is that we did not include a waitlist control group; hence, it is unknown whether the within-subjects effects seen in this study would have occurred naturally over time, or whether they were caused by the specific treatment. However, there is an extensive literature attesting to the specific effects of cognitive-behavioral therapy for panic disorder (e.g., meta-analyses: Gould et al., 1995; Mitte, 2005), including effects of therapy on cognition (e.g., Casey, Oei, & Newcombe, 2005; Clark et al., 1999, 1997; Hofmann et al., 2007). Further, a previous test with these data provided numerous indications that treatment had a positive impact on symptoms (including catastrophic misinterpretations, panic attack frequency, distress/apprehensions, and avoidance behavior; Teachman, Marker, & Clerkin, 2010). Together, this gives us reasonable confidence in the inference that treatment influenced the observed changes in the $AC_{me + calm}$ parameter. Finally, the small sample size and correspondingly low power were a limitation, particularly for the analyses investigating individual-level relationships, though the repeated measures design and small number of focused tests that were run helped in this regard. As well, the predominantly White sample is a limitation insofar as it weakens the external generalizability of this work.

Future directions and conclusions

Taken together, this research provides a valuable and novel—albeit preliminary—examination into the underlying processes of implicit cognition among patients with panic disorder. Future research using different category labels will be needed to determine the extent to which the expression of an undesirable panic association may be overcome by self-regulatory processes (e.g., rather than directly contrasting calm with panicked, one could examine associations tied to panic intensity or frequency, such as the category “panic” with the categories “often” vs. “rare”). Future research might also utilize a full waitlist control group to more clearly determine whether effects were caused by the specific treatment, and it will be valuable to replicate the between-groups effects when the panic and control groups complete an equal number of IATs. As well, it will be critical to replicate and extend these findings to a larger and more diverse sample. Given the non-significant relationship between the $AC_{me + calm}$ parameter and panic symptoms, it will be especially important to clarify the conditions under which change in the activation of automatic associations will meaningfully predict or be associated with symptom change.

Ultimately, the current research takes us one step closer to testing tenets from cognitive models of anxiety about the role of automatic processing. These findings emphasize the viability of

changing the actual activation of automatic associations in the context of treatment for panic, versus only changing a person's ability to use reflective processing to overcome biased automatic processing. For instance, the findings offer thought-provoking evidence in light of claims that, in order for fear extinction to occur, new learning and memories must compete with and inhibit the original fear memories (e.g., Bouton, 1993; see discussion in Craske, Liao, Brown, & Vervliet, 2012). Interestingly, data from the current study suggest that it may be possible to directly change the actual Association Activation for Me + Calm, even in the absence of evidence that strategic override of the old fear associations occurred (at least as measured by the OB parameter). This is in line with recent work suggesting that it is possible to actually erase fearful memory traces by disrupting the reconsolidation of that memory (e.g., Agren et al., 2012). That said, future work will be needed to determine whether the stimuli are activating a different set of associations after treatment (i.e., whether the associations are actually qualitatively changing), or whether the same associations are still activated, but to a greater degree (suggesting more quantitative change).

Further, future work is critical to determine whether changing the $AC_{me + calm}$ parameter is also a *clinically*—and not just *theoretically*—meaningful finding. Along these lines, an exciting potential avenue for future research is to apply mathematical modeling techniques like the Quad Model to the ongoing research designed to directly manipulate relatively automatic forms of cognition (e.g., attention bias training: Amir, Weber, Beard, Bomyea, & Taylor, 2008; conditioning of implicit rejection associations: Clerkin & Teachman, 2010). Utilizing an experimental design in conjunction with mathematical modeling techniques would allow researchers to more directly test causality and mechanisms in cognitive models of anxiety by exploring the underlying processes driving changes in relatively automatic forms of cognition.

Acknowledgments

This research was supported by NIMH R03MH068756 and NIAR01AG033033 grants to Bethany Teachman, and a NIAAA R21AA021151 grant awarded to Elise Clerkin. We are thankful for the statistical consultation provided by Yueqin Hu, a quantitative at Texas State University.

References

- Agren, T., Engman, J., Frick, A., Björkstrand, J., Larsson, E. M., Furmark, T., et al. (2012). Disruption of reconsolidation erases a fear memory trace in the human amygdala. *Science*, 337(6101), 1550–1552. <http://dx.doi.org/10.1126/science.1223006>.
- Amir, N., Weber, G., Beard, C., Bomyea, J., & Taylor, C. T. (2008). The effect of a single session attention modification program on response to a public-speaking challenge in socially anxious individuals. *Journal of Abnormal Psychology*, 117, 860–868. <http://dx.doi.org/10.1037/a0013445>.
- Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and non-anxious individuals: a meta-analytic study. *Psychological Bulletin*, 133, 1–24. <http://dx.doi.org/10.1037/0033-2909.133.1.1>.
- Barlow, D. H., & Craske, M. G. (1994). *Mastery of your anxiety and panic, II*. Albany, NY: Graywind.
- Beck, A. T., & Clark, D. A. (1997). An information processing model of anxiety: automatic and strategic processes. *Behaviour Research and Therapy*, 35, 49–58. [http://dx.doi.org/10.1016/S0005-7967\(96\)00069-1](http://dx.doi.org/10.1016/S0005-7967(96)00069-1).
- Beck, A. T., Emery, G., & Greenberg, R. L. (1985). *Anxiety disorders and phobias: A cognitive perspective*. New York: Basic Books.
- Bosson, J. K., Swann, W. B., Jr., & Pennebaker, J. W. (2000). Stalking the perfect measure of implicit self-esteem: the blind men and the elephant revisited. *Journal of Personality and Social Psychology*, 79, 631–643. <http://dx.doi.org/10.1037/0022-3514.79.4.631>.
- Bouton, M. E. (1993). Context, time, and memory retrieval in the interference paradigms of Pavlovian learning. *Psychological Bulletin*, 114, 80–99. <http://dx.doi.org/10.1037/0033-2909.114.1.80>.

- Casey, L. M., Newcombe, P. A., & Oei, T. P. S. (2005). Cognitive mediation of panic severity: the role of catastrophic misinterpretation of bodily sensations and panic self-efficacy. *Cognitive Therapy and Research*, 29, 187–200. <http://dx.doi.org/10.1007/s10608-005-3164-3>.
- Clark, D. M., Salkovskis, P. M., Hackmann, A., Wells, A., Ludgate, J., & Gelder, M. (1999). Brief cognitive therapy for panic disorder. *Journal of Consulting and Clinical Psychology*, 67, 583–589. <http://dx.doi.org/10.1037//0022-006X.67.4.583>.
- Clark, D. M., Salkovskis, P. M., Öst, L.-G., Breitholtz, E., Koehler, K. A., Westling, B. E., et al. (1997). Misinterpretation of body sensations in panic disorder. *Journal of Consulting and Clinical Psychology*, 65, 203–213. <http://dx.doi.org/10.1037//0022-006X.65.2.203>.
- Clerkin, E. M., & Teachman, B. A. (2010). Training implicit social anxiety associations: an experimental intervention. *Journal of Anxiety Disorders*, 24, 300–308. <http://dx.doi.org/10.1016/j.jbrat.2008.08.002>.
- Clerkin, E. M., Teachman, B. A., & Smith-Janik, S. B. (2008). Sudden gains in group cognitive-behavioral therapy for panic disorder. *Behaviour Research and Therapy*, 46, 1244–1250. <http://dx.doi.org/10.1016/j.brat.2008.08.002>.
- Cloitre, M., & Liebowitz, M. R. (1991). Memory bias in panic disorder: an investigation of the cognitive avoidance hypothesis. *Cognitive Therapy and Research*, 15(5), 371–386. <http://dx.doi.org/10.1007/BF01173032>.
- Conroy, F. R., Sherman, J. W., Gawronski, B., Hugenberg, K., & Groom, C. (2005). Separating multiple processes in implicit social cognition: the Quad-model of implicit task performance. *Journal of Personality and Social Psychology*, 89, 469–487. <http://dx.doi.org/10.1037/0022-3514.89.4.469>.
- Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, 46, 5–27. <http://dx.doi.org/10.1016/j.brat.2007.10.003>.
- Craske, M. G., Liao, B., Brown, L., & Vervliet, B. (2012). Role of inhibition in exposure therapy. *Journal of Experimental Psychopathology*, 3, 322–345. <http://dx.doi.org/10.5127/jep.026511>.
- Farrell, S., & Ludwig, C. J. (2008). Bayesian and maximum likelihood estimation of hierarchical response time models. *Psychonomic Bulletin & Review*, 15(6), 1209–1217. <http://dx.doi.org/10.3758/PBR.15.6.1209>.
- Fazio, R. H., & Hilden, L. E. (2001). Emotional reactions to a seemingly prejudiced response: the role of automatically-activated racial attitudes and motivation to control prejudiced reactions. *Personality and Social Psychology Bulletin*, 27, 538–549. <http://dx.doi.org/10.1177/0146167201275003>.
- First, M. B., Spitzer, R. L., Gibbon, M., Williams, J. B. W., Davies, M., Borus, J., et al. (1995). The structured clinical interview for DSM-III-R Personality disorders (SCID-II): II. Multi-site test–retest reliability study. *Journal of Personality Disorders*, 9, 92–104. <http://dx.doi.org/10.1521/pedi.1995.9.2.92>.
- Gould, R. A., Otto, M. W., & Pollack, M. H. (1995). A meta-analysis of treatment outcome for panic disorder. *Clinical Psychology Review*, 15, 819–844. [http://dx.doi.org/10.1016/0272-7358\(95\)00048-8](http://dx.doi.org/10.1016/0272-7358(95)00048-8).
- Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. K. (1998). Measuring individual differences in implicit cognition: the implicit association test. *Journal of Personality and Social Psychology*, 74, 1464–1480. <http://dx.doi.org/10.1037//0022-3514.74.6.1464>.
- Greenwald, A. G., & Nosek, B. A. (2001). Health of the implicit association test at age 3. *Zeitschrift für Experimentelle Psychologie*, 48, 85–93. <http://dx.doi.org/10.1026/0949-3946.48.2.85>.
- Greenwald, A. G., Nosek, B. A., & Banaji, M. R. (2003). Understanding and using the implicit association test: I. An improved scoring algorithm. *Journal of Personality and Social Psychology*, 85, 197–216. <http://dx.doi.org/10.1037/0022-3514.85.2.197>.
- Hofmann, S. G., Meuret, A. E., Rosenfield, D., Suvak, M. K., Barlow, D. H., Gorman, J. M., et al. (2007). Preliminary evidence for cognitive mediation during cognitive-behavioral therapy of panic disorder. *Journal of Consulting and Clinical Psychology*, 3, 374–379. <http://dx.doi.org/10.1037/0022-006X.75.3.374>.
- Houck, P. R., Spiegel, D. A., Shear, M. K., & Rucci, P. (2002). Reliability of the self-report version of the panic disorder severity scale. *Depression and Anxiety*, 15(4), 183–185. <http://dx.doi.org/10.1002/da.10049>.
- Jacoby, L. L. (1991). A process dissociation framework: separating automatic from intentional uses of memory. *Journal of Memory and Language*, 30(5), 513–541. [http://dx.doi.org/10.1016/0749-596X\(91\)90025-F](http://dx.doi.org/10.1016/0749-596X(91)90025-F).
- Klauer, K. C., Voss, A., Schmitz, F., & Teige-Mocigemba, S. (2007). Process components of the implicit association test: a diffusion-model analysis. *Journal of Personality and Social Psychology*, 93(3), 353. <http://dx.doi.org/10.1037/0022-3514.93.3.353>.
- Maidenberg, E., Chen, E., Craske, M., Bohn, P., & Bystritsky, A. (1996). Specificity of attentional bias in panic disorder and social phobia. *Journal of Anxiety Disorders*, 10(6), 529–541. [http://dx.doi.org/10.1016/S0887-6185\(96\)00028-X](http://dx.doi.org/10.1016/S0887-6185(96)00028-X).
- Mathews, A., & MacLeod, C. (1994). Cognitive approaches to emotion and emotional disorders. *Annual Review of Psychology*, 45, 25–50. <http://dx.doi.org/10.1146/annurev.psych.45.1.25>.
- McArdle, J. J., & Nesselroade, J. R. (2002). Growth curve analysis in contemporary psychological research. In J. Schinka, & W. Velicer (Eds.), *Comprehensive handbook of psychology: Vol. 2. Research methods in psychology*. New York: Wiley.
- McNally, R. J. (1995). Automaticity and the anxiety disorders. *Behaviour Research and Therapy*, 33, 747–754. [http://dx.doi.org/10.1016/0005-7967\(95\)00015-P](http://dx.doi.org/10.1016/0005-7967(95)00015-P).
- Mitte, K. (2005). A meta-analysis of the efficacy of psycho- and pharmacotherapy in panic disorder with and without agoraphobia. *Journal of Affective Disorders*, 88(1), 27–45. <http://dx.doi.org/10.1016/j.jad.2005.05.003>.
- Moshagen, M. (2010). multiTree: a computer program for the analysis of multinomial processing tree models. *Behavior Research Methods*, 42, 42–54. <http://dx.doi.org/10.3758/BRM.42.1.42>.
- Olatunji, B. O., Rosenfield, D., Tart, C. D., Cottraux, J., Powers, M. B., & Smits, J. A. (2013). Behavioral versus cognitive treatment of obsessive-compulsive disorder: an examination of outcome and mediators of change. *Journal of Consulting and Clinical Psychology*, 81, 415–428. <http://dx.doi.org/10.1037/a0031865>.
- Otto, M. W., Pollack, M. H., Penava, S. J., & Zuckerman, B. G. (1999). Group cognitive-behavior therapy for patients failing to respond to pharmacology for panic disorder: a clinical case series. *Behaviour Research and Therapy*, 37, 763–770. [http://dx.doi.org/10.1016/S0005-7967\(98\)00176-4](http://dx.doi.org/10.1016/S0005-7967(98)00176-4).
- Payne, B. K. (2008). What mistakes disclose: a process dissociation approach to automatic and controlled processes in social psychology. *Social and Personality Psychology Compass*, 2(2), 1073–1092. <http://dx.doi.org/10.1111/j.1751-9004.2008.00091.x>.
- Roefs, A., Huijding, J., Smulders, F. T. Y., MacLeod, C. M., de Jong, P. J., & Wiers, R. W. (2011). Implicit measures of association in psychopathology research. *Psychological Bulletin*, 137(1), 149–193. <http://dx.doi.org/10.1037/a0021729>.
- Shear, M. K., Brown, T. A., Barlow, D. H., Money, R. A., Sholomskas, D. E., Woods, S. W., et al. (1997). Multicenter collaborative panic disorder severity scale. *American Journal of Psychiatry*, 154, 1571–1575.
- Sheehan, D., Lecrubier, Y., Sheehan, K., Amorim, P., & Janavs, J. (1998). The Mini-International Neuropsychiatric Interview (MINI): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, 59, 22–33.
- Sherman, J. W. (2009). Controlled influences on implicit measures: confronting the myth of process-purity and taming the cognitive monster. In R. E. Petty, R. H. Fazio, & P. Briñol (Eds.), *Attitudes: Insights from the new wave of implicit measures* (pp. 391–426). Hillsdale, NJ: Erlbaum.
- Sherman, J. W., Gawronski, B., Gonsalkorale, K., Hugenberg, K., Allen, T. J., & Groom, C. J. (2008). The self-regulation of automatic associations and behavioral impulses. *Psychological Review*, 115, 314–335. <http://dx.doi.org/10.1037/0033-295X.115.2.314>.
- Sherman, J. W., Klauer, K. C., & Allen, T. J. (2010). Mathematical modeling of implicit social cognition: the machine in the ghost. In B. Gawronski, & B. K. Payne (Eds.), *Handbook of implicit social cognition: Measurement, theory, and applications* (pp. 156–175). New York: Guilford Press.
- Teachman, B. A. (2005). Information processing and anxiety sensitivity: cognitive vulnerability to panic reflected in interpretation and memory biases. *Cognitive Therapy and Research*, 29, 483–503. <http://dx.doi.org/10.1007/s10608-005-0627-5>.
- Teachman, B. A., Joormann, J., Steinman, S. A., & Gotlib, I. H. (2012). Automaticity in anxiety disorders and major depressive disorder. *Clinical Psychology Review*, 32, 575–603. <http://dx.doi.org/10.1016/j.cpr.2012.06.004>.
- Teachman, B. A., Marker, C. D., & Clerkin, E. M. (2010). Catastrophic misinterpretations as a predictor of symptom change during treatment for panic disorder. *Journal of Consulting and Clinical Psychology*, 78(6), 964. <http://dx.doi.org/10.1037/a0021067>.
- Teachman, B. A., Marker, C. D., & Smith-Janik, S. B. (2008). Automatic associations and panic disorder: trajectories of change over the course of treatment. *Journal of Consulting and Clinical Psychology*, 76, 988–1002. <http://dx.doi.org/10.1037/a0013113>.
- 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, 1791–1811. <http://dx.doi.org/10.1016/j.brat.2007.01.009>.
- Teachman, B. A., & Woody, S. (2003). Automatic processing among individuals with spider phobia: change in implicit fear associations following treatment. *Journal of Abnormal Psychology*, 112, 100–109. <http://dx.doi.org/10.1037/0021-843X.112.1.100>.
- Teachman, B. A., & Woody, S. (2004). Staying tuned to research in implicit cognition: relevance for clinical practice with anxiety disorders. *Cognitive and Behavioral Practice*, 11, 149–159. [http://dx.doi.org/10.1016/S1077-7229\(04\)80026-9](http://dx.doi.org/10.1016/S1077-7229(04)80026-9).
- Wetzels, R., Vandekerckhove, J., Tuerlinckx, F., & Wagenmakers, E. J. (2010). Bayesian parameter estimation in the expectancy valence model of the Iowa gambling task. *Journal of Mathematical Psychology*, 54(1), 14–27. <http://dx.doi.org/10.1016/j.jmp.2008.12.001>.
- Williams, J. M., Watts, F. N., MacLeod, C., & Mathews, A. (1997). *Cognitive psychology and emotional disorders* (2nd ed.). Chichester, UK: John Wiley & Son.
- Wilson, T. D., Lindsey, S., & Schooler, T. Y. (2000). A model of dual attitudes. *Psychological Review*, 107(1), 101–126. <http://dx.doi.org/10.1037/0033-295X.107.1.101>.
- Woody, S. R., Whittall, M. L., & McLean, P. D. (2011). Mechanisms of symptom reduction in treatment for obsessions. *Journal of Consulting and Clinical Psychology*, 79(5), 653. <http://dx.doi.org/10.1037/a0024827>.