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Thought-action fusion across anxiety disorder diagnoses: Specificity and treatment effects

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Abstract

Thought-action fusion (TAF) is a cognitive error that has been frequently investigated within the context of obsessive-compulsive disorder (OCD). However, evidence suggests that this error may also be present in disorders other than OCD, indicating that TAF is related to higher-order factors rather than a specific diagnosis. We explored TAF in a sample of patients with mixed diagnoses undergoing treatment with a transdiagnostic CBT protocol. Elevated TAF levels at baseline were not specific to patients with OCD. However, the presence of any generalized anxiety disorder (GAD) diagnosis was unexpectedly the strongest predictor of likelihood TAF. Likelihood TAF, a particular component of TAF, was reduced after transdiagnostic treatment, and this reduction was not affected by the presence of a GAD diagnosis. Results indicate that TAF is responsive to treatment and should be assessed and, perhaps, treated in disorders beyond OCD.

Keywords

Anxiety; obsessive-compulsive disorder; generalized anxiety disorder; cognition; transdiagnostic

1. Introduction

1.1 Overview of thought-action fusion

Thought-action fusion (TAF) is a cognitive variable that has been heavily researched in the past 10 or 15 years, particularly in relation to obsessive-compulsive disorder (OCD). Cognitive processes such as TAF are thought to form an intermediate step between the occurrence of an intrusive obsessional thought and the performance of a compulsion, because they represent underlying beliefs about the meaning or effect of obsessions. Rachman's model (1997, 1998) proposes that attributions of significance are critical in transforming normal intrusive thoughts into obsession. TAF is currently considered one of several misinterpretations of thought that may underlie OCD, but it is not necessarily a factor for all OCD patients, nor is it sufficient alone to account for OCD symptoms (Obsessive Compulsive Cognitions Working Group, OCCWG, 1997).

TAF is divided into two components. “Likelihood TAF” is the belief that simply having a thought about an event makes that event more likely to occur. This has been further broken down into “likelihood-self TAF” (“If I think about getting into a car accident, it makes it

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more likely that I will get into a car accident") and "likelihood-other TAF" ("If I think about my brother getting into a car accident, it makes it more likely that he will get into a car accident"). In addition to likelihood TAF, there is "Moral TAF," which is the belief that thinking about an action or behavior is morally equivalent to actually performing that behavior ("Thinking about hitting my girlfriend is as morally wrong as actually hitting her").

The Thought-Action Fusion Scale (TAFS; Shafran, Thordarson, & Rachman, 1996) has been the measure that is most widely used in the study of TAF. The scale consists of 12 items measuring moral TAF and 7 items measuring likelihood TAF. In clinical samples, a two-factor solution (likelihood and moral) has been supported, with the two factors together accounting for up to 71% of the variance (Shafran et al., 1996; Rassin et al., 2001b). In a student sample, a three-factor solution (moral, likelihood-self, and likelihood-other) was optimal (Shafran et al., 1996). Most studies have found moderate correlations between the moral and likelihood subscales (Shafran & Rachman, 2004). The scale has been found to have good predictive validity for patients selected to be high in TAF (Rachman et al., 1996), and good internal consistency (Shafran et al., 1996). However, an assessment of test-retest reliability was somewhat disappointing, with 3-month correlations between scores of 0.52 and a substantial decrease in scores indicating levels of TAF over time (Rassin et al., 2001). TAF has also been assessed in adolescents and children (Barrett & Healy, 2003; Muris et al., 2001).

1.2 TAF and OCD symptoms

Because TAF was first discussed in the context of OCD patients, and because it has been so central to one of the most prominent theories of OCD (Rachman, 1997; 1998), many have assumed that TAF had a unique association with obsessional symptoms. Indeed, several studies have found a relationship between TAF (particularly likelihood TAF) and OCD symptom level (Amir, Freshman, Ramsey, Neary, & Brigidi, 2001; Rassin, Diepstraten, Merckelbach, & Muris, 2001; Shafran, Thordarson, & Rachman, 1996). However, other studies have produced conflicting results. Some researchers have found that after controlling for depression, TAF is no longer significantly correlated with OCD symptoms (Jonsson et al., 2011; O'Leary et al., 2009). A study specifically aimed at determining whether all OCD patients have high levels of "dysfunctional OC beliefs" such as inflated responsibility and TAF found that OCD patients could be divided into a "low OC-beliefs" group and a "high OC-beliefs" group, indicating that these beliefs are not necessarily critical to OCD per se (Taylor et al., 2006). In general, moral TAF has not been found to be reliably related to OCD symptoms, though it has been found to be related to depression (Abramowitz et al., 2003; Shafran and Rachman, 2004). Overall, few of these studies have considered possible higher-order factors that may account for (some of) the elevations in TAF scores and their relationship to OCD symptomatology.

1.3 TAF and other disorders

There is reason to believe that TAF may also play a role in non-OCD pathology. Patients with generalized anxiety disorder (GAD) have been shown to hold beliefs that their worries can influence events in the world (Borkovec, Hazlet-Stevens, & Diaz, 1999). Significant associations between measures of worry and likelihood TAF have been found in an undergraduate sample (Hazlett-Stevens, Zucker, & Craske, 2002). In that study, subjects with partial/full GAD also scored higher on likelihood TAF than subjects without partial/full GAD.

Some researchers have directly compared patients with OCD and patients with other anxiety disorders on TAF scores. Two studies by Rassin and colleagues (2001a; 2001b) found that TAF scores in OCD patients were not significantly higher than the scores of patients with

other anxiety disorders. Abramowitz and colleagues (2003) conducted a study of 25 healthy control participants, 20 OCD patients, and 75 patients with non-OCD anxiety disorders or depression. No differences between the groups in moral TAF were found, and elevated levels of likelihood TAF were not specific to OCD. Researchers have also found elevated TAF levels in children with non-OCD anxiety disorders (Barrett & Healy, 2003).

In patients with eating disorders, a concept termed “thought-shape fusion” (TSF) has been explored. TSF consists of beliefs such as “Thinking about eating a forbidden food can actually make me gain weight” or “Thinking about eating a forbidden food is almost as bad as actually eating it. These beliefs have been found to be associated with disordered eating beliefs and behaviors in both clinical and nonclinical samples (Radomsky, de Silva, Todd, Treasure, & Murphy, 2002; Shafran & Robinson, 2004; Shafran, Teachman, Kerry, & Rachman, 1999). One study measured TAF in patients with anorexia nervosa and found total scores of 39.2 (SD = 12.1), compared to previously reported TAF scores in OCD patients of 33.2 and 31.5 (Abramowitz et al., 2003; Rassin et al., 2001a; Shafran et al., 1999).

Based upon the previous work indicating that TAF might be related to broader constructs in anxiety and therefore was not necessarily an OCD-specific phenomenon, Abramowitz and colleagues (2003) examined whether the relationship between OCD diagnosis and TAF scores was mediated by negative affect. As described above, they compared patients with OCD to patients with other anxiety and mood diagnoses and control participants. Their results indicated that negative affect (as measured by the Beck Depression Inventory (Beck et al., 1961) and the State-trait Anxiety Inventory (Speilberger et al., 1983)) did mediate the relationship between diagnosis and TAF. This is an important finding that broadens our understanding of TAF and what factors may underlie the relationship of this cognitive bias to diagnostic status.

1.4 Changes in TAF across treatment

Studies have largely shown an effect of treatment on TAF (Emmelkamp, van Oppen, & van Balkom, 2002; Rassin et al., 2001a; Whittal, Woody, McLean, Rachman, & Robichaud, 2010, but see also McLean et al., 2001). Jonsson et al. (2011) found a small effect of treatment on moral TAF in their overall sample, and a moderate effect of treatment on likelihood TAF. However, when controlling for changes in depressive symptoms, changes in TAF were no longer associated with changes in OCD symptoms over treatment (Jonsson et al., 2011). Manos et al. (2010) found that, when controlling for symptoms of depression and anxiety, only the perfectionism/certainty subscale of the Obsessional Beliefs Questionnaire (OCCWG, 2001) predicted changes in OCD symptoms over treatment. The responsibility/threat estimation subscale (most similar to the concept of inflated responsibility (IR), or excessive beliefs that one is responsible for preventing negative outcomes) and the importance/control of thoughts subscale (most similar to the concept of TAF) did not predict OCD symptom change during treatment. In contrast, Solem et al. (2009) found that beliefs about the need to control thoughts and positive beliefs about the need to worry predicted post-treatment symptom severity after controlling for depressive symptoms.

These results again raise questions about the relationship between treatment effects and TAF. Several studies have found that TAF is reduced following successful treatment for OCD (as would be expected based on Rachman's model). However, some other studies have shown that when depressive symptoms are controlled for, changes in TAF and related beliefs are no longer predictive of OCD symptom change. To our knowledge, no studies examining treatment effects on TAF in other anxiety or mood disorders beyond OCD have been conducted prior to the present investigation.

Our goal in the present study was to investigate the construct of TAF across a variety of diagnostic groups and across treatment. We used data from the first randomized controlled trial (Farchione et al., 2012) of the Unified Protocol for Transdiagnostic Treatment of Emotional Disorders (UP; Barlow et al., 2011) in order to examine TAF levels as a function of diagnosis, the potential relationship between TAF and NA, and whether TAF levels decreased over the course of treatment using a transdiagnostic approach. Our hypotheses for the present study were: (1) Both OCD and GAD diagnoses (either as a principal or additional diagnosis) would be associated with higher levels of likelihood TAF, (2) baseline NA would significantly predict baseline likelihood TAF, and (3) TAF would be reduced following transdiagnostic treatment, and this reduction would not vary by diagnostic group.

2. Methods

2.1 Patients

Patients entering an outpatient clinic specializing in anxiety disorders were administered the Anxiety Disorders Interview Schedule for *DSM-IV* – Lifetime Version (ADIS-IV-L; DiNardo, Brown, & Barlow, 1994) in order to determine all current and past diagnoses. When using the ADIS, diagnoses are assigned a clinical severity rating (CSR) of 0-8, with diagnoses with a CSR of 4 or higher being considered to have reached the clinical significance threshold of the DSM. When assigning diagnoses from the ADIS, “principal” is used to refer to the patient's most interfering and distressing diagnosis (as identified by the baseline ADIS clinical severity rating (CSR)) and “additional” to refer to any clinical diagnoses that were also present at baseline at a lower CSR than the principal diagnosis. In cases where a patient was assigned two diagnoses of equal CSR and that CSR was the highest of any of the clinical diagnoses assigned at baseline (e.g., panic disorder (PDA) and OCD at a CSR of 6, specific phobia at a CSR of 4), the term “co-principal” is used.

For the current trial, patients with a principal diagnosis of an anxiety disorder as assessed by the ADIS were eligible to participate. Patients were excluded if they had had a previous adequate trial of CBT, if they had a serious mental illness that required prioritization in treatment (e.g., bipolar disorder, schizophrenia), current suicide risk, or current or recent substance abuse or dependence (a more detailed overview of study methodology can be found in Farchione et al., 2012). Thirty-seven patients were consented and randomized to either immediate or delayed (16-week waitlist) treatment with the UP. Principal diagnoses were GAD (n=7), social anxiety disorder (SAD, n=8), OCD (n=8), PDA (n=8), anxiety disorder NOS (n=2), and post-traumatic stress disorder (PTSD, n=1). Three patients had co-principal diagnoses; these were SAD and anxiety disorder NOS, GAD and SAD, and OCD and PDA. The average number of clinical-level diagnoses per patient in the trial was 2.16 at pre-treatment (SD=1.19; range 1 to 5 diagnoses, see Table 1). Patients ranged in age from 19 to 52 years (M = 29.67, SD = 9.43), and the majority of the patients were female (58%) and Caucasian (97%).

2.2 Treatment

The UP treatment utilizes core cognitive-behavioral components, including psychoeducation, motivation enhancement, cognitive reappraisal, and in vivo and interoceptive exposures. Rather than focusing on symptoms of a particular disorder (i.e., social fears in social anxiety disorder), the UP utilizes a broad-based approach to encourage patients to tolerate emotions (both pleasant and unpleasant) without avoiding or suppressing them (for details of the protocol development and pilot data, see Ellard et al., 2010). The UP was designed to be used for the treatment of all anxiety and unipolar mood disorders, and based on theory, may also prove to be effective in the treatment of bipolar disorder, somatoform disorders, and dissociative disorders (Barlow et al, 2004; Fairholme et al,

2010). There is no "TAF-specific" element in the UP; rather, all of a patient's dysfunctional beliefs are targets for reappraisal based upon more rational evaluation. Patients then directly test their fears and their ability to tolerate discomfort by engaging in exposure practice.

2.3 Procedure

The UP treatment was conducted the Center for Anxiety and Related Disorders (CARD) in Boston. Treatment consisted of a maximum of eighteen 60-minute individual sessions. Three doctoral students and one licensed doctoral-level psychologist served as therapists for the study. All therapists had previous experience in administering CBT and underwent extensive training and certification prior to beginning the study. Twenty-six patients were randomized to immediate treatment with the UP and 11 patients were randomized to a waitlist delayed-treatment condition. Patients in the delayed treatment condition were placed on a waitlist for 16 weeks, after which they were reassessed and then completed UP treatment. Groups did not differ by age or sex. Patients who initiated treatment completed an average of 15.26 sessions (SD=4.60; range 2 to 18 sessions). A total of 18 patients were seen for all possible sessions (18) and 6 were seen for 17 sessions. All treatment completers (n = 32) received all treatment modules.

2.4 Measures

TAF was measured using the TAFS (Shafran et al., 1996). Negative affect was measured using the Positive And Negative Affective Scale (Watson, Clark, & Tellegen, 1988). The PANAS has been shown to have acceptable levels of internal consistency (alpha = .87 for NA scale), test-retest reliability, and internal and external validity (Watson et al., 1988). General anxiety and depression symptoms were measured using the Hamilton Anxiety Rating Scale (HARS; Hamilton, 1959) and the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960), among other measures. Both Hamilton scales were administered by independent evaluators following the Structured Interview Guide for the Hamilton Anxiety and Depression Rating Scale (SIGH-A and SIGH-D; Shear, Vander Bilt, & Rucci, 2001; Williams, 1988).

3. Results

3.1 Relationship of TAF to diagnosis

The three patients with co-principal diagnoses were not included in any of the calculations that were broken down by principal diagnosis. Across the entire sample, moral TAF scores ranged from 0-40 and likelihood TAF scores ranged from 0-21. Mean values of TAF scores across the four most common principal diagnoses (OCD, GAD, SAD, and PDA) are reported in Table 2. The two patients who had received a principal diagnosis of Anxiety Disorder NOS were included in the GAD group, as it had been determined at the initial diagnostic assessment that GAD was the disorder that would most closely capture their symptom presentation. In order to examine whether particular principal diagnoses were uniquely associated with high levels of TAF at baseline, we ran ANOVAs to compare patients with the four most common principal diagnoses (OCD, SAD, PDA, and GAD) on both moral and likelihood TAF scores. The results for moral TAF indicated no significant differences among the four groups ($F(3, 29)= 0.97$, *ns*). The results for likelihood TAF indicated a nearly significant difference across groups ($F(3, 30)= 2.81$, $p = .057$), and post-hoc t-tests revealed that only patients with a principal diagnosis of GAD had significantly higher baseline likelihood TAF scores compared to the other three diagnostic groups ($t(35)= 2.14$, $p<.05$).

We also chose to examine whether the presence of any clinical-level OCD or GAD diagnosis (whether as a principal or additional, non-principal diagnosis) was related to level

of TAF. We created a dummy variable to indicate the presence of any clinical-level OCD diagnosis at baseline versus no clinical OCD diagnosis, and ran t-tests to compare the TAF scores (moral and likelihood) of the two groups at baseline. We then created a second dummy variable to indicate the presence of any clinical-level GAD diagnosis at baseline versus no GAD diagnosis, and repeated the t-tests between groups. As in the previous analysis, patients who had received any diagnosis of Anxiety Disorder NOS (most resembling GAD) were included in the GAD group.

Results indicated that baseline moral TAF was not significantly related to diagnostic status. The comparisons of moral TAF levels between the any OCD diagnosis group versus not, as well as between the any GAD diagnosis group and not were non-significant (any OCD $t(35)= 1.32$, *ns*, any GAD $t(35)= 0.87$, *ns*). The effect sizes for these comparisons were small-to-medium level, with the any OCD versus no OCD comparison yielding an effect of $d=0.45$ and the any GAD versus no GAD yielding an effect of $d=0.29$ (see Table 3).

As for likelihood TAF, it was not significantly related to OCD diagnostic status at baseline ($t(35)= 0.63$, *ns)*, but there was a significant difference in scores between patients with any GAD diagnosis at baseline and patients without that diagnosis ($t(35)= 3.30$, $p<.01$) (see Table 3). Furthermore, the contrast of likelihood TAF scores between patients with any GAD versus no GAD yielded a large effect size of $d=1.10$. Given our results regarding the impact of a principal diagnosis of GAD on likelihood TAF scores, we ran a t-test to compare baseline likelihood TAF scores between patients with a principal diagnosis of GAD (n=9) versus patients with a co-principal or additional diagnosis of GAD (n=10). This test revealed no significant differences between the two groups ($t(17)= 0.42$, *ns*).

We conducted t-tests to examine whether levels of NA differed among patients in the different diagnostic categories. The comparison of baseline NA between patients who had any clinical diagnosis of OCD at baseline versus those who did not was not significant ($t(35)= 0.74$, *ns*, $d= 0.26$). However, the comparison of baseline NA between patients who had any clinical diagnosis of GAD at baseline versus those who did not was significant, with a large effect size ($t(35)= 3.26$, $p< .01$, $d= 1.07$). Across the entire sample, baseline NA was significantly correlated with baseline likelihood TAF ($r= .29$, $p<.05$) but not with baseline moral TAF ($r= .09$, *ns*).

We then performed regression analyses to determine how much variance in TAF scores was accounted for by the presence of any GAD diagnosis after accounting for NA. The regression of baseline likelihood TAF onto baseline NA approached but did not reach significance ($B= 0.32$ (SE 0.18), $p= .078$). The R^2 for this variable was .086, indicating that NA accounted for approximately 8.6% of the variance in likelihood TAF scores. In step 2, diagnostic status (any GAD diagnosis at baseline versus none) was added, and this variable was significant ($B= 5.36$ (SE 2.03), $p = .01$), indicating that a diagnosis of GAD significantly predicted likelihood TAF scores at baseline above and beyond what was predicted by baseline NA. The addition of diagnostic status into the equation only accounted for an additional 16% of the variance in likelihood TAF scores (R^2 in step 2= .242).

3.2 Effect of treatment on TAF

We examined the pre- and post-treatment TAF scores of all patients who completed active treatment (immediate and delayed conditions) to examine the effect of the UP on TAF. Paired t-tests revealed that treatment resulted in a significant reduction of likelihood TAF scores ($t(28)= 2.06$, $p<.05$, $d=.41$) from pre-treatment (M=3.97, SD=6.21) to post-treatment (M=2.24, SD=4.48). Moral TAF scores did not show a significant change across treatment ($t(28)= 1.07$, *ns,* $d=.20$) from pre-treatment (M=11.72, SD=10.78) to post-treatment (M=9.93, SD=8.88).

ANOVAs comparing change in TAF subscale scores across the four most common principal diagnoses revealed no significant differences in the amount of change across treatment for either moral TAF ($F(3, 23)= 0.31$, *ns*) or likelihood TAF ($F(3, 23)= 0.64$, *ns*). We also examined whether the presence of any GAD diagnosis had an effect on the level of change in TAF subscales across treatment. We found no such effect for any baseline clinical-level GAD diagnosis on either moral TAF change in treatment ($F(1, 27)= 0.18$, *ns*) or likelihood TAF change in treatment ($F(1, 27)= 2.99$, *ns*). We examined whether changes on either TAF subscale across treatment were related to changes in ratings of anxiety and depression, as measured by the SIGH-A and SIGH-D. Correlations among these change scores were not significant; this could be due to the relatively low levels of TAF endorsed at baseline across the sample, which may have created a floor effect in our measurement.

Finally, we examined whether baseline TAF scores predicted responder status across the sample using logistic regression. To meet the definition of a “responder,” patients had to achieve a 30% or greater improvement on at least two measures including the ADIS CSR for their principal diagnosis, the Work and Social Adjustment Scale (a measure of functioning; WSAS; Marks, Connolly, & Hallam, 1973; Mundt, Marks, Shear, & Greist, 2002), or the corresponding diagnosis specific self-report measure for their principal diagnosis. This calculation is a conservative adaptation of responder status algorithms used in similar trials of CBT for anxiety (for further details regarding the calculation of responder status in this sample, see Farchione et al., 2012). Across the sample, 55% of patients were classified as responders (Farchione et al., 2012). We found that the regression of responder status onto baseline moral TAF scores was not significant, $\chi^2 (1) = .02$, *ns*. The regression of responder status onto baseline likelihood TAF scores was also not significant, $\chi^2 (1) = 3.29$, *ns*.

4. Discussion

The results of this study expand our understanding of the relationship between TAF and particular diagnoses, as well as the impact of treatment on TAF. We found that baseline moral TAF was not uniquely related to any of our four main principal diagnostic categories, nor was it related to the presence of any clinical-level OCD or GAD diagnosis. This was unsurprising given previous research, which has connected moral TAF much more closely to depressive symptoms than to anxiety.

The finding that patients with a principal diagnosis of OCD did not differ significantly from any of the other three main principal diagnostic groups in their level of baseline likelihood TAF parallels other researchers' findings when examining TAF in mixed patient groups (Rassin et al., 2001a; 2001b). Our results provide further evidence that TAF is not uniquely related to a principal diagnosis of OCD despite the fact that the concept was initially proposed as an important etiological and maintaining component of that disorder.

We did find that baseline likelihood TAF was uniquely related to the presence of a GAD diagnosis at any level (principal or additional). Although previous work in patients with high levels of worry has supported the presence of TAF among this group, we believe these are the first results to suggest a particular relationship between a GAD diagnosis and levels of likelihood TAF. Patients with any level of GAD diagnosis had significantly higher levels of NA at baseline than patients without a GAD diagnosis; nonetheless, the presence of GAD anywhere in the diagnostic picture did add significantly to the prediction of baseline likelihood TAF, beyond the prediction of NA.

The finding that a GAD diagnosis is predictive of likelihood TAF scores can perhaps be understood by considering the worry process. When patients worry, they imagine multiple potential negative outcomes of a given situation; however, their worry prevents them from attending fully to any one negative thought, and reduces their overall autonomic response to

the negative thoughts (Borkovec, 1994). Thus, worry functions as a type of avoidance strategy. Researchers who have conducted experimental manipulations of TAF in the laboratory have found that evoking TAF leads to increases in intrusive thinking, and subsequently to discomfort and attempts to avoid/suppress/neutralize (Berman, Wheaton, Fabricant, Jacobson, & Abramowitz, 2011; Bocci & Gordon, 2007; Rassin, Merkelbach, Muris, & Spaan, 1999). Studies have also shown that the relationship between TAF and OC symptom severity is mediated by efforts to suppress the unwanted thoughts (Marcks and Woods, 2007; Rassin, Muris, Schmidt, & Merckelbach, 2000). A similar relationship may hold for worry symptoms in the case of GAD, with high levels of TAF leading to efforts to suppress thoughts and a corresponding increase in worry.

It is also possible that the relationship between GAD diagnosis and baseline likelihood TAF scores reflects the belief among some GAD patients that worry can be an adaptive process and, similarly, that failing to worry can result in disaster (Freeston et al., 1994; Rapee, 1991). This almost “magical thinking” quality of worry in GAD is somewhat analogous to the likelihood TAF construct. Indeed, studies of the relationship between likelihood TAF and magical thinking have indicated that the former may be a specific instance of the latter, and that TAF scores are only related to OCD symptomatology via their relationship to magical thinking (Einstein & Menzies, 2004a, 2004b; Lee, Cougle, & Telch, 2005). Worry, of course, is restricted to unpleasant outcomes; it is unknown whether patients with GAD would agree that this sort of magical thinking also applies to positive outcomes, or if their endorsement of likelihood TAF beliefs is limited to negative thoughts. “Positive TAF” has been reported in one study of undergraduate students with high levels of OCD symptoms, but has not been explored in GAD (Amir, Freshman, Ramsey, Neary, & Brigidi, 2001).

In considering the effects of treatment on TAF, we found that moral TAF did not change significantly across treatment, although it was associated with a small effect size. Likelihood TAF showed a significant reduction across treatment and treatment was associated with a medium effect. This resembles the finding of Jonsson et al. (2011), who reported a small effect of treatment on moral TAF scores but a moderate effect on likelihood TAF scores.

Across many studies, among patients with OCD moral TAF scores on the TAFS have been reported ranging from 20.35-25.70 at pre-treatment, and from 15.03-19.97 at post-treatment; likelihood TAF scores have been reported from 9.40-12.20 at pre-treatment and 6.50-8.19 at post-treatment (Jonsson, Hougaard, & Bennedsen, 2011; Shafran & Rachman, 2004). Among patients with other anxiety disorders, moral TAF scores have been reported from 19.60 to 22.00 and likelihood TAF scores from 5.10-10.50 (Shafran & Rachman, 2004). Among subjects with no psychiatric disorder, TAF scores range quite widely across reported studies, with moral TAF from 1.50-18.10 and likelihood TAF from 1.30-7.00 (Shafran & Rachman, 2004). The average levels of TAF endorsed by our sample were not as high as have been reported in other clinical populations (although the range of scores across the sample was quite broad). It is unclear why our sample endorsed relatively low levels of TAF on average. However, previous studies have reported overlap between clinical and non-clinical samples' scores on measures of TAF (Abramowitz, Whiteside, Lynam, & Kalsy, 2003; Shafran et al., 1996), and the construct of TAF is generally agreed to be a continuous rather than categorical construct (Berle & Starcevic, 2005).

In our study, changes in both likelihood and moral TAF across treatment were similar across the four main principal diagnostic groups. Although the absolute level of change in TAF scores across treatment was small, we were able to see statistically significant changes in likelihood TAF with treatment. As was true of baseline TAF scores across the sample, change in TAF TAF across treatment varied widely. Changes in likelihood TAF (post-treatment minus baseline) ranged from -12 to 3, indicating that some participants saw a

substantial drop in their likelihood TAF scores over the course of treatment. Furthermore, there was no difference in the effect of treatment on likelihood TAF scores based on the presence or absence of GAD at baseline. This indicates that, despite GAD being associated with higher levels of likelihood TAF pre-treatment, this association did not translate into poorer treatment outcome. Patients both with and without any GAD experienced similar levels of likelihood TAF reduction across treatment. Baseline TAF scores on either subscale did not predict patients' responder status at post-treatment.

A primary methodological strength of our study is our use of the diagnostic information from the intake ADIS. We believe that our use of full diagnostic information (both principal and additional diagnosis) is an advance over previous work examining TAF. Given the growing understanding that TAF is a continuous, rather than categorical, construct that is present at varying degrees across diagnoses (Berle & Starcevic, 2005), we looked beyond simply principal diagnoses in our analyses. Furthermore, while earlier treatment studies have either focused on highly TAF-specific interventions (Marino-Carper et al, 2010; Zucker et al., 2002) or disorder-specific CBT protocols (Rassin, Diepstraten et al., 2001), our results indicate that a broad-based transdiagnostic approach seems to be effective.

The present study was also limited in a few important ways. The study had a small sample size, reducing our ability to detect small differences among groups, and mean levels of TAF were comparatively low. Although several of the comparisons of patients with GAD diagnoses were associated with strong effect sizes, these findings should be considered preliminary and will need to be replicated in larger studies. Furthermore, our sample was highly homogeneous with regard to race/ethnicity, limiting the applicability of our findings in minority groups. Future research should continue to investigate TAF in diverse, non-OCD populations, as well as focusing further on other possible moderator effects, the potential role of negative affect, and the effect of treatment on TAF.

5. Conclusion

Our study is the first to examine TAF in a mixed-diagnosis sample of patients who went on to receive transdiagnostic treatment. As such, we were able to explore questions regarding TAF across diagnostic groups and the effect of transdiagnostic treatment on TAF. While previous research has established that relatively elevated levels of TAF may be present in diagnostic groups other than OCD, we believe our study is the first to find a specific contribution of GAD diagnosis to likelihood TAF scores. This adds to our understanding that TAF is not an OCD-specific phenomenon (as it was originally proposed to be in models of OCD), and may help to direct future research on this construct.

It is widely acknowledged that cognitive errors are maintaining factors across the emotional disorders, not only in depression. Current manualized treatments for social phobia (Hope, Heimberg, & Turk, 2006), generalized anxiety disorder (Zinbarg, Craske, & Barlow, 2006), and panic disorder (Barlow & Craske, 2007) directly address the presence of biases such as black and white thinking, catastrophizing, probability overestimation, etc. Our results suggest that TAF is amenable to reduction with treatment and should also be included among these more widely recognized errors, rather than only being assessed and targeted in the treatment of certain principal disorders. Other researchers have found that a brief psychoeducational procedure can be effective in reducing TAF beliefs (Marino-Carper et al., 2010; Zucker et al., 2002). The UP does not contain any “TAF-specific” element, yet in the present study baseline TAF did not predict treatment outcome, suggesting that a transdiagnostic treatment can effectively address this cognitive bias. Furthermore, we believe that the finding that TAF-related treatment outcomes did not vary across the diagnostic groups is a valuable contribution to the literature, as the present study is the only

one thus far to include these four principal anxiety groups in one treatment study and report on TAF outcomes for each. Discovering the ways in which a phenomenon differs across diagnostic groups, as well as the ways in which it is the same for all groups, is important to truly understanding that phenomenon. Although attention to TAF beliefs has traditionally been limited to OCD treatments, our results suggest a need for exploration of and attention to this thinking error in other disorders where negative cognitions are known to serve a maintaining function, and considering the role that TAF may play in non-OCD pathologies.

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References

Abramowitz JS, Whiteside S, Lynam D, Kalsy S. Is thought-action fusion specific to obsessive-compulsive disorder? A mediating role of negative affect. Behav Res Ther. 2003; 41:1069–1079. [PubMed: 12914808]

Amir N, Freshman M, Ramsey B, Neary E, Brigidi B. Thought-action fusion in individuals with OCD symptoms. Behav Res Ther. 2001; 39:765–776. [PubMed: 11419608]

Barlow DH, Allen LB, Choate ML. Toward a unified treatment for emotional disorders. Behav Ther. 2004; 35:205–230.

Barlow, DH.; Craske, MG. Mastery of your anxiety and panic: Client workbook. 4th. New York: Oxford University Press; 2007.

Barlow, DH.; Farchione, TJ.; Fairholme, CP.; Ellard, KK.; Boisseau, CL.; Allen, LB.; Ehrenreich-May, J. The Unified Protocol for Transdiagnostic Treatment of Emotional Disorders: Therapist Guide. New York: Oxford University Press; 2011.

Barrett PM, Healy LJ. An examination of the cognitive processes involved in childhood obsessive-compulsive disorder. Behav Res Ther. 2003; 41:285–299. [PubMed: 12600400]

Beck, AT.; Emery, G.; Greenberg, RL. Anxiety disorders and phobias: A cognitive perspective. New York: Basic Books; 1985.

Beck AT, Ward CH, Mendelsohn M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiat. 1961; 4:561–571. [PubMed: 13688369]

Berle D, Starcevic V. Thought-action fusion: Review of the literature and future directions. Clin Psych Rev. 2005; 25:263–284.

Berman NC, Wheaton MG, Fabricant LE, Jacobson SR, Abramowitz JS. The effects of familiarity on thought-action fusion. Behav Res Ther. 2011; 49:695–699. [PubMed: 21835394]

Bocci L, Gordon PK. Does magical thinking produce neutralizing behaviour? An experimental investigation. Behav Res Ther. 2007; 45:1823–1833. [PubMed: 17403518]

Borkovec, TD. The nature, functions, and origins of worry. In: Davey, GCL.; Tallis, F., editors. Worrying: Perspectives on theory, assessment, and treatment. New York: Wiley; 1994.

Borkovec TD, Hazlett-Stevens H, Diaz ML. The role of positive beliefs about worry in generalized anxiety disorder and its treatment. Clin Psychol Psychot. 1999; 6:126–138.

Brown TA. Temporal course and structural relationships among dimensions of temperament and DSM-IV anxiety and mood disorder constructs. J Abnorm Psychol. 2007; 116:313–328. [PubMed: 17516764]

Brown TA, Chorpita BF, Barlow DH. Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. J Abnorm Psychol. 1998; 107:179–192. [PubMed: 9604548]

Einstein DA, Menzies RG. The role of magical thinking in obsessive compulsive symptoms in an undergraduate sample. Depress Anxiety. 2004a; 19:174–179. [PubMed: 15129419]

Einstein DA, Menzies RG. The presence of magical thinking in obsessive compulsive disorder. Behav Res Ther. 2004b; 42:539–549. [PubMed: 15033500]

Emmelkamp, PMG.; van Oppen, P.; van Balkom, AJLM. Cognitive changes in patients with obsessive compulsive rituals treated with exposure in vivo and response prevention. In: Frost, RO.; Steketee, G., editors. Cognitive approaches to obsessions and compulsions: Theory, assessment, and treatement. Oxford: Elsevier; 2002.

Fairburn CG, Cooper Z, Doll HA, O'Connor ME, Bohn K, et al. Transdiagnostic cognitive-behavioral therapy for patients with eating disorders: A two-site trial with 60-week follow-up. Am J Psychiat. 2009; 166:311–319. [PubMed: 19074978]

Fairholme, CP.; Boisseau, CL.; Ellard, KK.; Ehrenreich, JT.; Barlow, DH. Emotions, emotion regulation, and psychological treatment: A Unified perspective. In: Kring, AM.; Sloan, AM., editors. Emotion regulation and psychopathology: A transdiagnostic approach to etiology and treatment. New York: Guilford Press; 2010. p. 283-309.

Farchione TJ, Fairholme CP, Ellard KK, Boisseau CL, Thompson-Hollands J, Carl JR, Barlow DH. The Unified Protocol for the Transdiagnostic Treatment of Emotional Disorders: A randomized controlled trial. Beh Ther. 2012; 43:666–678.

Foa, EB.; Kozak, MJ. Mastery of obsessive-compulsive disorder: Therapist guide. New York: Oxford University Press; 2004.

Freeston MH, Rhéaume JL, Letarte H, Dugas MJ, Ladouceur. Why do people worry? Pers Indiv Differ. 1994; 17:791–802.

Hamilton M. The assessment of anxiety states by rating. Brit J Med Psychol. 1959; 32:50–55. [PubMed: 13638508]

Hamilton M. A rating scale for depression. J Neurol, Neurosur Ps. 1960; 23:56–62.

Harvey, AG.; Watkins, ER.; Mansell, W.; Shafran, R. Cognitive behavioural processes across psychological disorders: A transdiagnostic approach to research and treatment. Oxford: Oxford University Press; 2004.

Hazlett-Stevens H, Zucker BG, Craske MG. The relationship of thought-action fusion to pathological worry and generalized anxiety disorder. Behav Res Ther. 2002; 40:1199–1204. [PubMed: 12375728]

Hope, DA.; Heimberg, RG.; Turk, CL. Managing social anxiety: A cognitive-behavioral therapy approac: Therapist guide. San Antonio, TX: Oxford University Press; 2006.

Jonsson H, Hougaard E, Bennedsen BE. Dysfunctional beliefs in group and individual cognitive behavioral therapy for obsessive compulsive disorder. J Anxiety Disord. 2011; 25:483–489. [PubMed: 21232914]

Lee HJ, Cougle JR, Telch MJ. Thought-action fusion and its relationship to schizotypy and OCD symptoms. Behav Res Ther. 2005; 43:29–41. [PubMed: 15531351]

Manos RC, Cahill SP, Wetterneck CT, Conelea CA, Ross AR, Riemann BC. The impact of experiential avoidance and obsessive beliefs on obsessive-compulsive symptoms in a severe clinical sample. J Anxiety Disord. 2010; 24:700–708. [PubMed: 20627655]

Marcks BA, Woods DW. Role of thought-related beliefs and coping strategies in the escalation of intrusive thoughts: An analog to obsessive-compulsive disorder. Behav Res Ther. 2007; 45:2640–2651. [PubMed: 17673167]

Marino-Carper T, Negy C, Burns G, Lunt RA. The effects of psychoeducation on thought-action fusion, thought suppression, and responsibility. J Behav Ther Exp Psy. 2010; 41:289–296.

Marks IM, Connolly J, Hallam RS. The psychiatric nurse as therapist. Brit Med J. 1973; 2:156–160. [PubMed: 4735516]

McLean PD, Whittal ML, Thordarson DS, Taylor S, Sochting I, Koch WJ, et al. Cognitive versus behavior therapy in the group treatment of obsessive-compulsive disorder. J Consult Clin Psych. 2001; 69:205–214.

Mundt JC, Marks IM, Shear MK, Greist JM. The work and social adjustment scale: A simple measure of impairment in functioning. Brit J Psychiat. 2002; 180:461–464.

Muris P, Meesters C, Rassin E, Merkelbach H, Campbell J. Thought-action fusion and anxiety disorders symptoms in normal adolescents. Behav Res Ther. 2001; 39:843–852. [PubMed: 11419614]

Obsessive Compulsive Cognitions Working Group. Cognitive assessment of obsessive-compulsive disorder. Behav Res Ther. 1997; 35:667–681. [PubMed: 9193129]

Obsessive Compulsive Cognitions Working Group. Development and initial validation of the obsessive beliefs questionnaire and the interpretation of intrusions inventory. Behav Res Ther. 2001; 39:987–1006. [PubMed: 11480839]

Rachman S. A cognitive theory of obsessions. Behav Res Ther. 1997; 35:793–802. [PubMed: 9299799]

Rapee RM. Generalized anxiety disorder: A review of clinical features and theoretical concepts. Clin Psych Rev. 1991; 11:419–440.

Rassin E, Diepstraten P, Merckelbach H, Muris P. Thought-action fusion and thought suppression in obsessive-compulsive disorder. Behav Res Ther. 2001; 39:757–764. [PubMed: 11419607]

Rassin E, Merkelbach H, Muris P, Schmidt H. The thought-action fusion scale: Further evidence for its reliability and validity. Behav Res Ther. 2001; 39:537–544. [PubMed: 11341250]

Rassin E, Merkelbach H, Muris P, Spaan V. Thought-action fusion as a causal factor in the development of intrusions. Behav Res Ther. 1999; 37:231–237. [PubMed: 10087641]

Salkovskis P. Obsessional-compulsive problems: A cognitive-behavioral analysis. Behav Res Ther. 1985; 23:571–583. [PubMed: 4051930]

Shafran R, Rachman S. Thought-action fusion: A review. J Beh Ther Exp Psy. 2004; 35:87–107.

Shafran R, Robinson P. Thought-shape fusion in eating disorders. Brit J Clin Psych. 2004; 43:399–408.

Shafran R, Teachman BA, Kerry S, Rachman S. A cognitive distortion associated with eating disorders: Thought-Shape Fusion. Brit J Clin Psych. 1999; 38:167–179.

Shafran R, Thordarson DS, Rachman S. Thought-action fusion in obsessive-compulsive disorder. J Anxiety Disord. 1996; 10:379–391.

Shear MK, Vander Bilt J, Rucci P. Reliability and validity of a structured interview guide for the Hamilton Anxiety Rating Scale (SIGH-A). Depress Anxiety. 2001; 13:166–178. [PubMed: 11413563]

Solem S, Haland AT, Vogel PA, Hansen B, Wells A. Change in metacognitions predicts outcome in obsessive-compulsive patients undergoing treatment with exposure and response prevention. Behav Res Ther. 2009; 47:301–307. [PubMed: 19203749]

Taylor S, Abramowitz JS, McKay D, Calamari JE, Sookman D, Kyrios M, Wilhelm S, Carmin C. Do dysfunctional beliefs play a role in all types of obsessive-compulsive disorder? Anxiety Disord. 2006; 20:85–97.

Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: The PANAS scales. J Pers Soc Psychol. 1988; 54:1063–1070. [PubMed: 3397865]

Whittal ML, Woody SR, McLean PD, Rachman SJ, Robichaud M. Treatment of obsessions: A randomized controlled trial. Behav Res Ther. 2010; 48:295–303. [PubMed: 19969286]

Williams JB. A structured interview guide for the Hamilton Depression Rating Scale. Arch General Psych. 1988; 45:742–747.

Zinbarg, R.; Craske, M.; Barlow, DH. Therapist's guide for the mastery of your anxiety and worry program. New York, NY: Oxford University Press; 2006.

Table 1

Principal and additional diagnoses across subjects

		Additional dxs										
Principal dx	**n**	**None**	**GAD**	**SAD**	**OCD**	**PDA**	**Anx-NOS**	**PTSD**	**DD[b]**	**HC**	**SPEC**	**TOUR**
GAD	7	1	--	4	1	0	0	1	6	1	0	0
SAD	8	3	2	--	1	1	0	0	1	0	2	0
OCD	8	3	3	2	--	2	0	0	2	0	2	1
PDA	8	4	1	1	0	--	0	0	3	0	1	0
Anx-NOS	2	2	0	0	0	0	--	0	0	0	0	0
PTSD	1	0	1	0	0	0	0	--	0	0	0	0
Co-prin[a]	3	2	1	0	0	0	0	0	0	0	0	0
Totals	37	15	8	7	2	3	0	1	12	1	5	1

Note. GAD = Generalized anxiety disorder, SAD = Social anxiety disorder, OCD = Obsessive-compulsive disorder, PDA = Panic disorder with/without agoraphobia, Anx-NOS = Anxiety disorder not otherwise specified, PTSD = Post-traumatic stress disorder, DD = Depressive disorder, HC = Hypochondriasis, SPEC = Specific phobia, TOUR = Tourette's syndrome

[a]Co-principal diagnoses included

[b]Depressive disorders included major depressive disorder (n=8), dysthymia (n=2), and depressive disorder not otherwise specified (n=2)

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Table 2
Mean TAF subscale scores by principal diagnosis

Principal Diagnosis	n	Moral TAF	Likelihood TAF
GAD[a]	9	13.78 (7.77)	7.33 (7.14)
OCD	8	10.63 (10.28)	2.75 (5.54)
SAD	8	11.00 (12.48)	1.00 (0.65)
PDA	8	6.25 (4.13)	1.75 (3.28)

Note: GAD = generalized anxiety disorder; SOC = social anxiety disorder; PDA = panic disorder with agoraphobia; OCD = obsessive compulsive disorder.

[a]Includes patients with a principal diagnosis of Anxiety Disorder NOS (n=2)

Table 3

Relationship of baseline TAF to diagnosis

		mTAF (pre)			lTAF (pre)		
	n	**mean (SD)**	***t***	**d**	**mean (SD)**	***t***	**d**
Any OCD	11	14.00 (11.77)	1.31	.47	4.73 (7.60)	0.63	.23
No OCD	26	9.31 (9.04)			3.35 (5.36)		
Any GAD	18	12.78 (9.47)	1.24	.41	6.94 (7.26)	3.53[b]	1.19
No GAD	19	8.74 (10.34)			0.74 (1.79)		

[a] p = >.05,

[b] p = >.01