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The search for universal transdiagnostic and trans-therapy change processes: evidence for intolerance of uncertainty

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Abstract

The search for universal processes associated with symptom change across emotional disorders and different forms of psychotherapy offers hope of increased theoretical parsimony and treatment efficiencies. This study investigated whether intolerance of uncertainty (IU) is a universal process by examining whether changes in IU were associated with changes in symptoms across three different cognitive behavior therapy protocols for depression (n = 108), social anxiety disorder (n = 88), or generalized anxiety disorder (n = 62) in a community mental health clinic. IU was associated with reductions in repetitive negative thinking in all treatments, which is consistent with IU being a transdiagnostic and 'trans-therapy' process of change. Changes in IU were also associated with symptom relief in the social anxiety disorder and generalized anxiety disorder groups, but not in the depression group. Implications of these findings are discussed within the broader literature of transdiagnostic approaches to emotional disorders.

Key Words: transdiagnostic, intolerance of uncertainty, generalized anxiety disorder, social anxiety disorder, depression, cognitive behavior therapy

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The search for transdiagnostic mechanisms that maintain emotional disorders has gathered considerable momentum over the last decade. Transdiagnostic approaches seek to identify and target factors that breach traditional diagnostic boundaries and perpetuate more than one disorder (McEvoy, Nathan, & Norton, 2009; Norton & Paulus, in press). The rationale for targeting transdiagnostic factors across emotional disorders is compelling, including common genetic heritabilities (Kendler, Neale, Kessler, Heath, & Eaves, 1992) and underlying latent structures (Barlow, 2002; Brown, Chorpita, & Barlow, 1998; Carragher, Krueger, Eaton, & Slade, 2015), similar efficacy of pharmacological and psychotherapeutic interventions (Norton, 2008), high rates of comorbidity (Brown, Campbell, Lehman, Grisham, & Mancill, 2001), and evidence that comorbid disorders can remit during treatment for a primary disorder (Borkovec, Abel, & Newman, 1995). A range of transdiagnostic cognitive and behavioral processes have been identified (Harvey, Watkins, Mansell, & Shafran, 2004), and the race is on to develop psychological treatments that efficiently and effectively alleviate suffering by targeting these factors (e.g., Barlow et al., 2011; Dear et al., 2015; Norton, 2012; Titov et al., 2015). The potential advantages of transdiagnostic treatments include the ability to simultaneously treat multiple comorbid disorders, and the ease and cost-effectiveness of dissemination compared to a vast range of diagnosis-specific treatments (Addis, Wade, & Hatgis, 1999; Barlow, Allen, & Choate, 2004; Norton & Philipp, 2008). Given that transdiagnostic factors are theorized to maintain emotional disorders, changes in these mechanisms during treatment should be associated with symptom reduction for multiple disorders.

The search of transdiagnostic factors can be extended to ‘trans-therapy’ factors, defined here as those that are (a) directly or indirectly modified and (b) associated with

symptom relief *across* more than one bona fide therapy. One example of a transdiagnostic factor that may not necessarily be a trans-therapy factor is negative metacognitions. Negative metacognitions include beliefs that repetitive negative thinking (RNT, e.g., worry or rumination) is uncontrollable and potentially harmful (Wells & Cartright-Hatton, 2004), which are associated with symptoms of multiple disorders including depression (Papageorgiou & Wells, 2003), generalized anxiety disorder (GAD, Thielsch et al., 2015), and social anxiety disorder (SAD, McEvoy & Perini, 2009). Whereas metacognitive therapy directly targets negative metacognitive beliefs in therapy (Wells, 2009, e.g., "*I can't stop worrying* about making a fool of myself"), other cognitive approaches focus on challenging negative automatic thoughts (e.g., Rapee, Gaston, & Abbott, 2009, "*I will make a fool of myself*"), and thus targeting metacognitive beliefs may not be considered a trans-therapy factor. In contrast, reducing withdrawal by scheduling rewarding activities is a component in multiple therapies for depression, including behavioural activation (Dimidjian, Barrera, Martell, Munoz, & Lewinsohn, 2011) and cognitive therapy (Beck, Rush, Shaw, & Emery, 1979), and thus may be considered a trans-therapy factor. However, pleasant and achievement-based activity scheduling is most commonly prescribed for depression and thus may not be considered transdiagnostic.

Although transdiagnostic and trans-therapy factors are conceptually separable, it is likely that different treatments directly or indirectly modify many of the same transdiagnostic mechanisms. It is difficult to demonstrate, for instance, that *in vivo* exposure modifies arousal via habituation without modifying cognition (i.e., negative beliefs about the stimulus). Likewise, most cognitive interventions include behavioral components (e.g., imaginal exposure, behavioral experiments), which leaves open the possibility that behavioral processes are at least in part responsible for symptom change. Impacts across different processes occur not only between techniques deriving from alternative theoretical

frameworks, but also from a single technique. For instance, a behavioral experiment, whereby an individual confronts a feared situation in order to directly test a feared consequence (e.g., *"I will be ridiculed"*), has the potential to modify a range of cognitive processes in addition to the negative prediction. To successfully complete a behavioral experiment and test a prediction, the individual must override automatic attentional biases towards threatening information and also direct attention towards non-threatening information. Constructs such as coping self-efficacy and perceived control are also likely to be modified. This scattergun approach of most psychological techniques makes it difficult to demonstrate that a particular intervention is exclusively acting to modify a specific mechanism described within the particular theoretical framework from which it derives, or to rule out that alternative theoretical accounts are responsible for change. Collateral effects on multiple potentially therapeutic variables are therefore almost inevitable.

Identifying both transdiagnostic and trans-therapy factors is important for determining the most critical treatment foci for achieving successful outcomes from psychotherapy regardless of a patient's diagnostic profile or a therapist's preferred brand of psychotherapy, and may help to integrate the evidence-supported treatment and common factors literatures (e.g., Laska, Gurman, & Wampold, 2014). If a subset of transdiagnostic and trans-therapy change factors are identified (henceforth referred to as 'universal factors'), research and therapeutic efficiencies could be further increased beyond targeting transdiagnostic factors, as the search for the most effective ways of modifying critical processes is prioritized above the model or school of psychotherapy from which a technique derives. This study examined whether intolerance of uncertainty (IU) could be a candidate universal factor associated with symptom change across anxiety disorders and depression (i.e., transdiagnostic) and across different treatment protocols (i.e., trans-therapy).

Intolerance of uncertainty as a transdiagnostic process

IU has been defined as a dispositional fear of the unknown (Carleton, 2012) and as the tendency to consider the possibility of negative events as unacceptable and threatening regardless of the actually probability of the event occurring (Carleton, Sharpe, & Asmundson, 2007; Dugas, Gosselin, & Ladouceur, 2001). Carleton (2012) argues that uncertainty is inherent in anxiety, where future negative events are anticipated and uncertain (e.g., worry about future harm), but not fear, where negative events are more certain or current (e.g., imminent physical threat). IU is argued to be a risk factor for anxiety symptoms and anxiety disorders, such that individuals higher on this dimension are more likely to experience negative emotions in response to ambiguity or uncertainty (Koerner & Dugas, 2008). Associations have also been found between IU and depression, which may be a function of comorbidity with anxiety, the relationship between IU and RNT (e.g., depressive rumination), or a preference for pessimistic certainty rather than tolerating uncertainty (Carleton, 2012).

The Intolerance of Uncertainty Model was originally developed to explain the maintenance of uncontrollable and excessive worry within the context of GAD (Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994). Consistent with this model, IU is elevated in individuals with GAD compared to non-anxious controls and is a cognitive vulnerability factor for worry and GAD (Dugas, Gagnon, Ladouceur, & Freeston, 1998; Koerner & Dugas, 2008; Dugas et al., 2007; Laugesen, Dugas, & Bukowski, 2003; Sexton, Norton, Walker, & Norton, 2003). However, evidence has since accumulated that IU is common across multiple emotional disorders, including obsessive compulsive disorder (OCD, Holaway, Heimberg, & Coles, 2006; Tolin, Abramowitz, Brigidi, & Foa, 2003), social anxiety (Boelen & Reijntjes, 2009; Carleton, Collimore, & Asmundson, 2010; Riskind, Tzur, Williams, Mann, & Shahar, 2007), panic disorder and agoraphobia (Carleton, Hackl, Fetzner, & McEvoy, 2013; Carleton et al., 2007; Mahoney & McEvoy, 2012a), and depression (Buhr & Dugas, 2002; Dugas, Schwartz, & Francis, 2004; McEvoy & Mahoney, 2012; van der Heiden et al., 2010).

Various forms of RNT that have been historically investigated within the context of different disorders, such as worry (GAD), rumination (depression), and post-event processing (SAD), have also been shown to be more similar than different (Ehring & Watkins, 2008; Harvey et al., 2004; McEvoy & Brans, 2013). In a comprehensive review of the literature, Ehring and Watkins (2008) concluded that the process of RNT is identical across different disorders and is characterized as being repetitive, difficult to control, negative in content, predominantly verbal, relatively abstract, and related to metacognitions (e.g., beliefs that engaging in RNT is helpful for preventing catastrophes, or that RNT is harmful and uncontrollable). The only differences in RNT across disorders was argued to be the content of the thoughts (e.g., threat themes in GAD, hopelessness themes in depression, and social-evaluative themes in SAD) and temporal orientation, with anxiety-linked RNT being more future-focused and depression-linked RNT being more past-focused (although not exclusively so). IU and RNT are therefore two key constructs in the Intolerance of Uncertainty Model (Freeston et al., 1994) that have been demonstrated to be transdiagnostic.

Intolerance of uncertainty as a potential 'trans-therapy' processes

The search for trans-therapy processes of change is predicated on the assumption that interventions deriving from a particular theoretical model will rarely exclusively modify processes identified within that model. Recent evidence that IU may be modified by treatments deriving from alternative cognitive behavioural frameworks suggests that IU could be a candidate trans-therapy mechanism of change. van der Heiden, Muris, and van der Molen (2012) compared IU therapy based on the Intolerance of Uncertainty Model to metacognitive therapy to and delayed treatment groups for GAD (N = 126). These therapies derive from distinct theoretical frameworks and there are key differences between them, but van der Heiden et al.'s (2012) findings were intriguing. While both treatments were highly effective, metacognitive therapy was associated with larger reductions on a measure of IU

compared to IU therapy. This finding is particularly striking given that demand effects for self-report measures should be strongest for a treatment that explicitly targets the construct. Presumably IU was not mentioned throughout metacognitive therapy and thus patients should feel less obliged to report favorable outcomes on this measure to please the clinicians, compared to patients receiving IU therapy.

One explanation for this finding is that techniques in metacognitive therapy indirectly reduce IU. For instance, worry postponement is a metacognitive therapy task designed to challenge metacognitive beliefs about worry being helpful (e.g., “worry keeps me safe”) or uncontrollable (e.g., “I cannot control my worry”), where patients are encouraged to nominate a regular time of day to exclusively engage in worry. Outside of the nominated ‘worry time’ patients are required to postpone their worry and observe whether they are able to control their engagement in worry and whether harm befalls them as a consequence. Importantly, when clients learn that they are in fact able to disengage from worry, they are also (implicitly) required to tolerate uncertainty that harm will befall them whilst postponing worry, and thus they might simultaneously learn that they can cope with uncertainty, nothing bad happens when they are uncertain, and the anxiety about uncertainty passes.

Another study by Mahoney and McEvoy (2012b) also found that IU reduced during traditional group cognitive behaviour therapy (CBT) for social phobia, and these reductions were associated with symptom improvement, despite the fact that IU was not explicitly mentioned in the protocol. We are not aware of any treatment studies that report the relationship between changes in IU and symptoms in a sample with primary depressive disorders, regardless of whether or not the treatment explicitly targeted IU. However, Boswell, Thompson-Hollands, Farchione, and Barlow (2013) reported that reductions in IU were correlated with reductions in comorbid depression symptoms in individuals with primary anxiety disorders who completed a unified (transdiagnostic) treatment that did not

explicitly target IU. Overall, these findings suggest that treatments designed to target cognitive mechanisms based on a particular theoretical framework can have collateral effects on mechanisms from alternative models. Identifying mechanisms associated with symptom change across treatments may provide clues about the most important and efficient strategies for promoting change regardless of the theoretical framework from which they derive.

The Current Study

The vast literature demonstrating that IU is transdiagnostic, together with evidence that IU changes across different therapies (trans-therapy), suggests that IU may be a universal process associated with change across different emotional disorders and treatments. However, further research demonstrating that changes in IU are associated with symptom improvement during psychotherapy for different disorders and whilst using different evidence-supported treatments is required to build the case for IU as a universal change process. The aim of this study was to examine whether changes in IU were associated with changes in RNT and symptom relief for individuals with different emotional disorders who received different group treatment protocols. The relationships between IU, RNT, and symptom change were examined for patients receiving group metacognitive therapy for GAD, imagery-enhanced CBT for SAD, and traditional CBT for depression. Changes in IU were hypothesized to be associated with changes in RNT and symptoms across these disorders and treatment groups. It was further expected that these relationships would remain significant after controlling for change in negative affect. Finally, it was expected that patients with higher IU would be more likely to drop out of treatment due to the inherent exposure to uncertainty in all three treatments. Support for these hypotheses would strengthen the case for IU as a universal change process.

Method

2.1 Participants

Patients were referred by General Practitioners, Psychiatrists, or Clinical Psychologists to a community mental health clinic for psychological treatment of anxiety disorders and/or depression. A structured diagnostic interview (Mini International Diagnostic Interview, MINI, Lecrubier et al., 1997; Sheehan et al. 1997a, b, 1998) was used to establish the presence of DSM-IV (American Psychiatric Association, 1994) anxiety and/or depressive disorders. Up to three disorders were recorded in the database.

Patient's demographic information is summarized in Table 1. The worry and rumination group sample comprised 62 patients with a diagnosis of GAD, two-thirds of which were diagnosed with more than one disorder. The most common primary diagnosis was GAD (n=45, 72.6%), followed by Major Depression (n=9, 14.5%). A small number of patients were diagnosed with primary Dysthymia, Agoraphobia without Panic Disorder (n=2 each, 3.4%), Panic Disorder without Agoraphobia, OCD, Major Depression in Partial Remission, or Type II Bipolar Disorder (n=1 each, 1.6%). All patients had a primary or comorbid diagnosis of GAD, and almost a third (31%) had a primary or comorbid depressive disorder. The median time since the onset of each patient's mental health problems was 10 years (IQR: 5 years to 15 years). Nearly the entire sample (97%) had received previous psychiatric treatment, but not responded adequately. A quarter had been hospitalized for a mental health problem, and about two-thirds were taking psychotropic medication. The median time taking medication was two years (IQR: 6 months to 5 years).

The social anxiety group sample comprised 88 patients with a diagnosis of SAD. The majority (81%) were diagnosed with more than one mental disorder. The most common primary disorder was SAD (n=73, 83.0%), followed by Major Depression (n=12, 13.6%), Dysthymia, GAD, and Bipolar Disorder Type I (n=1 each, 1.1%). Half the sample (n=44) had a primary or secondary depressive diagnosis. Patient's psychiatric problems were longstanding, with a median duration of 10 years (IQR: 6.5 years to 17 years). About a

quarter of the sample had attempted suicide. Like the worry and rumination group, the majority of social anxiety group patients had previously sought help but failed to respond adequately. Two-thirds were taking psychotropic medication, and had been doing so for a median duration of 11 months (IQR: 3 months to 4 years).

The mood management group sample comprised 106 patients with a depressive disorder. Again, the vast majority (85%) were diagnosed with more than one mental disorder. The primary diagnoses were Major Depression (n=96, 90.6%), Dysthymic Disorder (n=5, 4.7%), SAD (n=2, 1.9%), GAD (n=2, 1.9%), and PTSD (n=1, 0.9%). The most common comorbidities were SAD and GAD. The median duration of patients' psychiatric problems was 12 years (IQR: 10 years to 20 years). More than one third had attempted suicide. Like the other groups, nearly all patients had previously sought treatment but had an inadequate response. The median duration of medication use was two years (IQR: 3 months to 10 years). Overall, the patients in the three samples can be described as suffering from chronic and highly comorbid disorders that had proven difficult to treat.

2.2 Outcome Measures

2.2.1 Intolerance of Uncertainty Scale-12 (IUS-12, Carleton et al., 2007). The IUS-12 is a 12-item version of the original 27-item IUS (Buhr & Dugas, 2002; Freeston et al., 1994) that measures negative beliefs about and reactions to uncertainty. The 12-item version has been found to be highly correlated (r = .96) with the full version in undergraduate (Carleton et al., 2007; Khawaja & Yu, 2010) and clinical (McEvoy & Mahoney, 2011) samples. The IUS-12 comprises two subscales, Prospective IU (cognitive anticipation, "*I always want to know what the future has in store for me*") and Inhibitory IU (behavioral, "*when it's time to act, uncertainty paralyses me*"), although a total score is commonly used. The IUS-12 is associated with symptoms of multiple anxiety disorders and depression even when controlling for neuroticism (Boelen, Vrinssen, & van Tulder, 2010; Carleton et al.,

2010; Mahoney & McEvoy, 2012c; McEvoy & Mahoney, 2011, 2012; Norton & Mehta, 2007), and has been shown to be dimensional rather than taxonic (Carleton et al., 2012). Items are rated on a 5-point Likert scale from not at all characteristic of me (1) to entirely characteristic of me (5). Sum scores on the IUS-12 can range between 7 and 35 (Prospective IU), 5 and 25 (Inhibitory IU) and 12 and 60 (Total Score). Internal consistencies in the current study were high for the subscales (Prospective IU $\alpha = .88$, Inhibitory IU $\alpha = .87$), and for the total score ($\alpha = .92$).

2.2.2 Repetitive Negative Thinking Questionnaire (RTQ-10, McEvoy, Mahoney, & Moulds, 2010; McEvoy, Thibodeau, & Asmundson, 2014). The RTQ is a transdiagnostic measure of RNT that was developed by modifying items from the Penn State Worry Questionnaire (Meyer, Miller, Metzger, & Borkovec, 1990), Ruminative Responses Scale (Treynor, Gonzalez, & Nolen-Hoeksema, 2003) and revised Post-Event Processing Questionnaire (McEvoy & Kingsep, 2006) to remove diagnosis-specific content, such as references to worry and depression symptoms (Mahoney, McEvoy, & Moulds, 2012; McEvoy et al., 2010). The 10-item trait version of the RTQ used in this study has a unidimensional structure, distinguishes well between clinical and non-clinical populations, and correlates very highly ($r = .95$) with a longer 27-item version of the scale (McEvoy et al., 2010; McEvoy et al., 2014). Example items are "*Once I start thinking about the situation, I can't stop*" and "*I think about the situation all the time*." Items are rated with respect to respondents' experience when they are distressed or upset on a 5-point Likert scale from not true at all (1), somewhat true (3), to very true (5). RTQ-10 (henceforth RTQ) total scores can fall between 10 and 50. Internal consistency in the current study was high ($\alpha = .88$).

2.2.3 Positive and Negative Affect Scale (PANAS, Watson, Clark, & Tellegen, 1988). The PANAS features 10-item positive (PANAS-POS) and negative (PANAS-NEG) affect subscales, although only the PANAS-NEG subscale was used in this study. The 10

negative emotions (e.g., *distressed, upset, guilty ashamed*) are rated on a 5-point response scale to indicate the extent to which the respondent generally feels this way: very slightly or not at all (1), a little (2), moderately (3), quite a bit (4) or extremely (5). Total scores can range between 10 and 50. Crawford and Henry (2003) have provided evidence of high internal consistency (αs = .90-.95) and construct validity (including convergent and divergent validity). Internal consistency in the current study was high (α = .84).

2.2.4 Social Interaction Anxiety Scale (SIAS) and Social Phobia Scale (SPS, Mattick & Clarke, 1998). The SPS and SIAS (Mattick & Clarke, 1998) are 20-item measures of performance and interaction anxiety, respectively. The SPS assesses situations in which the person is the focus of attention and observed by others (e.g., "*I become anxious if I have to write in front of other people*", "*I get nervous that people are staring at me as I walk down the street*"). The SIAS contains items reflecting cognitive, affective, and behavioral reactions to interaction situations (e.g., "*I have difficulty making eye-contact with others*", "*When mixing socially I am uncomfortable*"). The 5-point response scale for both scales is not at all (0), slightly (1), moderately (2), very (3), or extremely (4) characteristic of me. Total scores for both measures can range from 0 to 80. These scales have demonstrated high 12-week test-retest reliabilities (SIAS *r* = .92; SPS *r* = .93, Mattick & Clarke, 1998) and sensitivity to change (Cox, Ross, Swinson, & Direnfeld, 1998). Internal consistencies in the current study were high for the SIAS (α = .85) and the SPS (α = .90).

2.2.5 Beck Depression Inventory (BDI-II, Beck, Steer, & Brown, 1996). The BDI-II is a 21-item measure of depression symptoms experienced over the previous two weeks. Items are rated on a 4-point scale and total scores can range from 0 to 63. Internal consistency and test–retest reliability (*r* = .93 over 1 week) are well established (Beck et al., 1996), and evidence for construct validity has been demonstrated (Dozois et al., 1998). Support for

convergent and discriminant validity has also been reported (Steer et al., 1997). Internal consistency in the current sample was high (α = .88).

2.2.6 Penn State Worry Questionnaire (PSWQ, Meyer et al., 1990). The PSWQ is a 16 item trait-based questionnaire commonly used to measure pathological worry in GAD (e.g., "*I am always worrying about something*"). Respondents rate the extent to which each item applies to them on a 5-point scale ranging from not at all typical of me (1) to very typical of me (5). Five negatively worded items are reverse scored before summing to form a total score ranging from 16 to 80 with higher scores reflecting greater levels of worry. The PSWQ has demonstrated good validity and reliability (Brown, Antony, & Barlow, 1992; Meyer et al., 1990). Internal consistency in the current study was high (α=.92).

2.3 Procedure

Patients referred for treatment of an anxiety disorder or depression were posted the questionnaire battery to complete prior to their initial assessment, at which the MINI was completed by a Clinical Psychologist experienced in both the assessment and treatment of emotional disorders. All cases, diagnoses, and treatment plans were presented and discussed at weekly clinic meetings. Patients with SAD, GAD, or depression nominated their most distressing disorder and on this basis were typically allocated to the social anxiety group, worry and rumination group, or mood management group, respectively. As is standard in clinical practice, in some instances the patient and assessing therapist determined that a comorbid disorder required treatment before the primary disorder, so the patient was allocated to a group targeting the comorbid disorder. For instance, patients with long-standing and temporally primary SAD may have completed a mood management group if their depression was severe and likely to interfere with treatment targeting SAD. During this assessment session patients were provided with general information about the content of the group program (e.g., "you will learn practical strategies for helping you to manage your

[social anxiety, depression, worry and rumination] more effectively"), which excluded any reference to increasing tolerance of uncertainty per se. All measures were completed following the final group session (post-treatment), and at a one-month follow-up appointment. Patients provided informed written consent for their clinical data to be used for the purposes of quality improvement, evaluation, and publication, and the procedures were approved for each group program by the Hospital's Human Research Ethics Committee (QI_2014_04, QI_2014_05, QI_2014_23).

2.4 Treatment

The worry and rumination group (McEvoy, Erceg-Hurn, Anderson, Campbell, & Nathan, 2015; McEvoy, Erceg-Hurn, Anderson, Campbell, Swan, et al., 2015), social anxiety group (McEvoy & Saulsman, 2014; McEvoy, Erceg-Hurn, Saulsman, & Thibodeau, 2015), and mood management group (McEvoy & Nathan, 2007) protocols used in this study have previously been evaluated and found to be highly effective and to meet international benchmarks (i.e. efficacy and effectiveness trials). All groups were co-facilitated by two senior clinicians (masters- or doctoral-level clinical psychologists), or one senior clinician and one clinical psychology trainee. All senior clinicians had participated in the published trials of the group protocols. Importantly, the concept of IU is not explicitly discussed in any of the protocols, thereby allowing an assessment of whether IU changes as a consequence of implementing the standard procedures within each treatment whilst minimizing social desirability biases. All three groups include a one-month follow-up session at which progress is reviewed, difficulties are problem-solved, and future management plans are developed.

The worry and rumination group protocol is based on Wells and Mathews' (1996) Self-Regulatory Executive Function model and Wells' (2009) metacognitive therapy for emotional disorders, although there are some departures from Wells' approach (see McEvoy, Erceg-Hurn, Anderson, Campbell, Swan et al., 2015, for more details). The 6-session worry

and rumination group (plus one month follow-up) includes psychoeducation and socialization to the metacognitive formulation, including negative beliefs about RNT, unhelpful behaviors used to avoid RNT (e.g., situational avoidance, suppression), attentional biases, and positive beliefs about RNT. Subsequent sessions included thought challenging and behavioral experiments targeting negative (e.g., RNT is uncontrollable and dangerous) and positive (e.g., RNT is helpful) metacognitive beliefs. Attentional flexibility was targeted through several attention training tasks that involved sustaining attention on present moment activity and mindfulness techniques. Active coping (structured problem-solving) was used as a technique to reduce maladaptive behavioral symptoms (e.g., situational avoidance) and to promote more adaptive approach behaviors. The final session involved a review of the key principles and the development of self-management plans.

The 12-week (plus one-month follow-up) imagery-enhanced social anxiety group (McEvoy, Erceg-Hurn, Saulsman, et al., 2015) is based on Rapee et al.'s (2009) CBT protocol and includes psychoeducation, cognitive monitoring and restructuring, in vivo exposure conducted as a series of behavioral experiments involving hypothesis testing, safety behavior elimination, video-feedback to correct distorted self-images, attention training, identification and challenging of negative core beliefs and relapse prevention (see also McEvoy, Nathan, Rapee, & Campbell, 2012). The protocol includes imagery techniques within each component (see McEvoy & Saulsman, 2014, for more details).

The 10-session mood management group (plus one month follow-up) is based on Beck et al.'s (1979) depression manual and incorporates elements of Barlow and Craske's (1994) anxiety manual to address comorbid anxiety. Key components of the manual are psychoeducation about depression and comorbid anxiety, behavioral activation, calming techniques to bring attention to the present moment and reduce arousal, behavioral experiments, and cognitive restructuring (see McEvoy & Nathan, 2007, for more details).

2.5 Analyses

All analyses were intent-to-treat, with all available data used in each analysis. Pearson correlations were used to examine the relationship between pre-treatment IUS-12 scores and attendance. Mixed-effects models were used to examine changes in each outcome over time, and to study associations between the IUS-12, PANAS, and symptoms. Within-treatment effect sizes (standardized mean changes) were calculated for each outcome by subtracting the estimated post-treatment (or follow up) mean from the pre-treatment mean, and dividing by the observed pre-treatment standard deviation (Morris, 2008). Effect sizes of .20, .50 and .80 were regarded as small, medium, and large, respectively (Cumming, 2012).

In order to test whether *changes* in intolerance of uncertainty and negative affect during treatment predicted symptoms, the IUS-12 and PANAS were modelled as time-varying covariates. A *time-invariant* covariate is a variable that does not change during treatment, such as gender, whereas a *time-varying* covariate can take on different values at each measurement occasion. An appealing aspect of fitting models with time-varying covariates is that it enables within- and between-person effects to be disentangled. For example, the Intolerance of Uncertainty Model predicts that at any given time point, patients with high IUS-12 scores should have more severe symptoms than patients with low IUS-12 scores. This is a between-person effect, as it describes how *differences in IUS-12 scores between individuals* are associated with symptoms. It also follows from the IUS-12 model that *the degree to which IU changes within an individual* should be related to changes in that individual's symptoms. This is a within-person effect. The focus of the present analyses was on the within-person effects – in other words, are changes within individuals on the IUS-12 during treatment associated with changes in their symptoms, and is this still true after controlling for within-person changes in negative affect?

We followed Hedeker and Gibbons' (2006) procedure for fitting mixed-effect models with time-varying covariates. Each patient had up to three IUS-12 scores (eg 28 pre-treatment, 20 post-treatment, 9 follow up). For each individual, we computed a mean IUS-12 score, for example (28+20+9)/3=19, and the deviations around this mean (28 - 19 = 9, 20 – 19 = 1, 9 – 19 = -10). The same procedure was followed for the PANAS.

For each outcome measure, two models were run. In the first model, the fixed effects were an intercept, time, each patient's IUS-12 mean, and their IUS-12 deviation scores. The PANAS mean and deviation scores were then added to the model. The regression coefficient for the IUS-12 deviation scores in these models indicates whether change on the IUS-12 is associated with changes on symptom measures. All models included random intercept terms for each patient, and a random slope for time if doing so provided a better fit to the data that the random-intercept only model. For some outcomes time was fit as a categorical rather than continuous variable, if doing so provided a better fit to the data (e.g., because change between post-treatment and follow-up did not follow the same trajectory as between pre- and post-treatment). All models were run with data from all participants within each group and then re-run including only participants with primary SAD (social anxiety group), primary GAD (worry and rumination group), and primary depression (mood management group). The pattern of effect sizes was almost identical so only results for the full samples are reported.

Results

3.1 Worry and Rumination Group

The mean number of sessions completed was 6.14 (*SD* = 1.39) out of a possible 7 (including follow-up), and the median was 7 sessions. Eleven percent of participants completed between 2 and 4 sessions, and 89% completed five or more. Pre-treatment IUS-12 total scores were not related to the number of sessions completed, $r < .01$, 95% CI [-.27, .27], $p = .98$. Changes over the course of treatment for each outcome measure can be found in

Table 2, and are plotted in the first column of Figure 1. There were very large reductions on the PSWQ and RTQ during treatment, and patients continued to improve over the follow up period. IUS-12 and PANAS scores also improved substantially, with effect sizes exceeding 1 *SD* by follow up. The figure suggests that change on the IUS-12 between post-treatment and follow up continued at the same rate as was observed during treatment, whereas the rate of change on other variables slowed somewhat. Within-person change on the IUS-12 was associated with PSWQ scores over the course of treatment, and this association persisted even after controlling for change on PANAS scores. The relevant regression coefficient (for the IUS-12 deviation term) was .47 for the model without the PANAS, 95% CI [.21, .72], p = .001, and .31 after controlling for the PANAS, 95% CI [.06, .56], p = .015. Change on the PANAS was also associated with PSWQ scores, controlling for change in IUS-12 scores, Est = .83, 95% CI [.42, 1.24], p < .001. The findings were similar when the dependent variable was the RTQ. Change on the IUS-12 was associated with change on the RTQ, Est = .35, 95% CI [.16, .53], p <.001, and the association was still present after controlling for PANAS, Est = .25, 95% CI [.06, .45], p = .011. Change on the PANAS was also uniquely associated with change on the RTQ, Est = .50, 95% CI [.17, .83], p = .003

3.2 Social Anxiety Group

The mean number of sessions completed was 9.98 (SD = 3.39) out of a possible 13 (including follow-up), and the median was 11. The majority of participants (77%) completed nine or more sessions; 11% completed between 1 and 4 sessions, and 11% completed between 5 and 8 sessions. Pre-treatment IUS-12 scores were unrelated to the number of sessions attended, r = -.02, 95% CI [-0.28, .19], p =.82. Changes over the course of treatment for each outcome measure can be found in Table 3, and are plotted in the middle column of Figure 1, except for the SPS (the trajectory for the SPS was practically identical to that for the SIAS). There were very large reductions on the SIAS and SPS, a large reduction on the

PANAS and RTQ, and moderate to large reductions on the IUS-12. Like in the worry and rumination group, patients continued to improve between post-treatment and follow up, particularly on the IUS-12. Change on the IUS-12 over the course of treatment was associated with change on the RTQ, Est = .35, 95% CI [.19 .51], $p < .001$. The magnitude of the association only slightly reduced after controlling for the PANAS, Est = .31, 95% CI [.14, .48], $p < .001$. Change on the PANAS was more weakly and not significantly associated with change on the RTQ, Est = .25, 95% CI [.00, .50], $p = .052$. Change on the IUS-12 was also found to be related to change on the SIAS, without (Est = .59, 95% CI [.33, .83], $p <.001$) and with (Est .47, 95% CI [. 26, .69], $p < .001$) the PANAS included in the model. Change on the PANAS was also uniquely predictive of change on the SIAS, Est = 1.20, 95% CI [.89, 1.51], $p < .001$. The results were similar for the SPS. Change on the IUS-12 was significantly associated with change on the SPS, Est = .57, 95% CI [.29, .85], $p <. 001$, and the relationship held after controlling for change on the PANAS, Est = .45, 95% CI [.20, .70], $p < .001$. Change on the PANAS was also associated with change on the SPS when controlling for the IUS-12, Est = 1.06, 95% CI [.69, 1.42], $p < .001$.

3.3 Mood Management Group

The mean number of sessions attended was 7.65 (*SD* = 2.99) out of a possible 11 (including follow-up), and the median was 8. Twelve percent of participants completed between 1 and 3 sessions; 28% completed between 4 and 7, and 60% completed 8 or more. Pre-treatment IUS-12 scores were not related to the number of sessions attended, $r = .06$, 95% CI [-.13, .25], $p = .55$. Changes in each outcome measure can be found in Table 4 and the right-hand column of Figure 1. There was a large change on the BDI-II, while changes on the RTQ and PANAS were more modest than for the other groups, and change on the IUS-12 was small. Unlike the other groups, there were no statistically or clinically significant changes on any outcome between post-treatment and follow up.

Over the course of treatment, change on the IUS-12 was associated with change on the RTQ, Est = .39, 95% CI [.23, .55], $p < .001$. This relationship was still evident after controlling for PANAS, Est = .39, CI [.24, .55], $p < .001$. The PANAS was also uniquely related to the RTQ, Est = .19, 95% CI [.04, .34], $p = .014$. In contrast, there was only a weak association between change in IUS-12 and BDI-II that was not statistically significant, Est = .19, 95% CI [-.13, .39], $p = .16$. When the PANAS was added to the model, the relevant coefficient was .16, 95% CI [-.08, .40], $p = .20$. Unlike the IUS-12, change in PANAS scores was uniquely associated with change in BDI-II, Est = .57, 95% CI [.34, .81], $p < .001$

3.4 IUS-12 Subscales

All analyses were rerun using the IUS-12 subscales rather than the total score. The pattern of results was practically identical in all cases except two. For the worry and rumination group, change in Prospective IU was a statistically significant predictor of PSWQ scores after controlling for the PANAS, Est = .50, 95% CI [,10, .89], $p = .014$, whereas Inhibitory IU just failed to achieve statistical significance, Est = .50, 95% CI [-.04, 1.04], $p = .071$. The same pattern of results was present when the dependent variable was the RTQ. Prospective IU was a statistically significant predictor after controlling for the PANAS (Est = .44, 95% CI [.14, .74], $p = .005$) while Inhibitory IU was not (Est = .35, CI [-.07, .77], $p = .102$. While the p-values for Inhibitory IU after controlling for the PANAS in both cases are above .05, the confidence intervals are wide and consist almost entirely of positive values. This suggest that changes in Inhibitory IU across therapy probably are associated with changes in RNT and worry after controlling for negative affect, but that the sample size in the current study was too small to demonstrate a statistically significant relationship.

Discussion

The aim of this study was to determine whether IU is associated with symptom change across different emotional disorders (GAD, SAD, depression) and different treatment

group protocols (metacognitive therapy, imagery-enhanced cognitive behavior therapy, and traditional cognitive therapy, respectively). It was hypothesized that changes in IU would be consistently associated with changes in RNT and symptoms across disorders and treatments, which was supported for RNT and partially supported for symptoms with the exception being depression symptoms. The significant relationships remained after controlling for the higher order construct of negative affectivity, thus supporting the discriminant validity of IU from negative affect and suggesting that changes in IU were incrementally associated with changes in RNT and symptoms for social anxiety and worry beyond reductions in general negative affect. These findings are broadly consistent with IU being a universal process of change, defined here as both a transdiagnostic and trans-therapy construct.

The prediction that IU would be associated with greater treatment dropout was not supported for any of the treatments and suggests that IU need not be an impediment to treatment engagement. We are not aware of any previous research investigating the relationship between IU and treatment attrition, although studies of treatments targeting IU have demonstrated low levels of dropout (<10%, Dugas et al., 2003). All three treatments in the current study titrated uncertainty by using a graded approach to cognitive and behavioural change (i.e., least to most challenging), which may have increased treatment acceptability for high IU individuals.

Our findings are consistent with an extensive literature demonstrating that IU is associated with a range of emotional disorders in cross-sectional, experimental, longitudinal, and treatment studies (Carleton, 2012; Norton & Paulus, in press), and with evidence that self-reported IU significantly reduces and is associated with symptom change during CBT protocols that do not explicitly target IU (Mahoney & McEvoy, 2012b; van der Heiden et al., 2012). It is arguable that techniques in all three protocols encouraged engagement with inherently uncertain tasks. For example, the metacognitive therapy protocol (worry and

rumination group) encouraged patients to regularly postpone engagement in RNT to test the controllability of their RNT and uncertain negative consequences of disengaging from RNT. The imagery-enhanced CBT protocol encouraged patients with SAD to regularly confront inherently uncertain social-evaluative situations to test their fears of evaluation. The traditional cognitive behavior therapy protocol (mood management group) encouraged individuals with depression to increase their engagement in a range of activities and to challenge their negative beliefs, despite uncertainty about whether these tasks would be helpful for improving their mood.

Interestingly, after controlling for negative affectivity, Prospective IU more strongly predicted changes in worry than Inhibitory IU for patients completing the worry and rumination group. This finding is consistent with previous evidence that Prospective but not Inhibitory IU mediates the relationship between neuroticism and worry in mixed anxiety disorder samples (McEvoy & Mahoney, 2012). Prospective IU assesses concerns about future uncertainty (e.g., “I always want to know what the future has in store for me”), whereas Inhibitory IU assesses behavioral inhibition (e.g., “When it’s time to act, uncertainty paralyzes me”). The focus on the future and cognitive symptoms likely explains the stronger relationship between Prospective IU and the often future-oriented nature of worry (Ehring & Watkins, 2008). Overall, our findings suggest that patients in all three programs were less fearful of (Prospective IU) and paralyzed by (Inhibitory IU) the unknown following treatment, and these changes were associated with reductions in RNT and anxiety symptoms.

The lack of an association between changes in IU and depression symptoms during the mood management group was unexpected. RNT is a well-established vulnerability factor for depression symptoms (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Given that reductions in IU were associated with reductions RNT in the mood management group, it was expected that reductions in IU would also be associated with reductions in depression

symptoms. An alternative mechanism through which IU could maintain depression is pessimistic certainty (Carlton, 2012), whereby negative predictions are expected to be highly likely (almost certain) in preference to tolerating uncertainty about an outcome. Reductions in IU should therefore reduce the tendency to expect negative outcomes as being likely, which, in turn, should positively impact on depression symptoms.

One potential explanation for the lack of an association in this study is therefore that the impact of IU on depression symptoms is indirect, perhaps via other constructs such as negative thoughts about the certainty of negative outcomes and absence of positive outcomes (Miranda, Rontes, & Marroquín, 2008). These forms of depressive certainty may be acquired with increasing fluency via RNT and, in turn, lead to hopelessness and worsening symptoms. Miranda and colleagues (2008) found evidence that while both anxiety and depression are characterized by pessimistic certainty about negative events, pessimistic certainty about the absence of positive events is unique to depression, and that these beliefs may be more proximal than IU to depression symptoms. Therefore, while IU may increase vulnerability to anxiety and depression via RNT, additional intermediate processes may be particularly important for more fully understanding the relationship between IU and depression symptoms. CBT for depression may therefore require a process of directly testing pessimistic certainty (e.g., "I know I enjoyed soccer in the past, but I *won't* now"), rather than challenging IU per se ("I know I enjoyed soccer in the past, but I'm *unsure* if I will now").

Another potential explanation for the lack of association between changes in IU and changes in depression symptoms is that the magnitude of change in IU was smaller in the mood management group than for the other groups, which may have attenuated the association. It is plausible that behavioral activation within traditional CBT for depression involves a lower dose of exposure to uncertainty that the other treatments, as patients are encouraged to engage with activities *known* to be previously associated with pleasure and

achievement. In contrast, patients in the social anxiety program regularly engage in inherently uncertain social situations with unfamiliar conversational partners and where the outcomes of the interactions are largely unknown. Likewise, patients in the worry and rumination group are required to disengage from habitual RNT and overcontrol strategies, which may result in a larger dose of uncertainty than CBT for depression.

Our finding that changes in IU explained unique variance in RNT in all three groups, and symptoms in the social anxiety and worry and rumination groups, is consistent with previous studies (McEvoy & Mahoney, 2013; Sexton et al., 2003). In contrast, Talkovsky and Norton (2014) recently found that within their transdiagnostic clinical sample anxiety sensitivity and IU failed to explain unique variance in state anxiety beyond negative affect. The researchers interpreted their findings as evidence that transdiagnostic CBT achieves symptom change by modifying the higher order construct of negative affectivity, thus deemphasizing the role of IU and anxiety sensitivity. There are several potential explanations for the discrepancy between Talkovsky and Norton's (2014) findings and our study.

First, Talkovsky and Norton (2014) used the state anxiety scale of the State Trait Anxiety Inventory (STAI, Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 2003) as the outcome variable, which may have been differentially sensitive to disorder-specific symptom changes than the measures used in this study. Moreover, the STAI shared a substantial proportion of variance (74%) with the PANAS negative affect subscale in Talkovsky and Norton's (2014) study, leaving little variance to be explained by IU. Second, although there are commonalities in the techniques used across the treatments (e.g., forms of exposure and cognitive restructuring), Talkovsky and Norton's (2014) transdiagnostic intervention may operate more broadly on NA compared to the treatments in the current study. It may be that the disorder-specific foci of our treatments more specifically target IU within each problem area, and indeed there is evidence that disorder-specific IU explains unique variance in

symptoms above and beyond trait IU (Mahoney & McEvoy, 2012a; Thibodeau et al., 2015). Third, the inclusion of anxiety sensitivity within Talkovsky and Norton's (2014) model may have usurped variance that would have been captured by IU. Notwithstanding the differences in findings in relation to IU, it is noteworthy that negative affectivity was a unique predictor of symptom change (and the sole predictor for depression symptoms) in our study, which is consistent with Talkovsky and Norton's (2014) findings.

The correlational nature of this study is an important limitation, so we are unable to draw any causal conclusions about the relationship between changes in IU and outcomes. Changes in IU may be a cause, consequence, or epiphenomenon of RNT and symptom change. Although insufficient, the associations found in our study are necessary for a universal process and provide some empirical justification for further research in this area. The use of self-reported outcomes, which are vulnerable to social desirability biases, is another limitation. Patients who were more likely to report lower symptoms at post-treatment to please the clinicians may have been more likely to also report lower IU, even though IU was not explicitly mentioned as a treatment target. Experimental research and clinical studies using behavioral assessments of IU before, during, and after treatment would be useful for addressing these limitations. The absence of a control group is another limitation of this study. IU is generally considered a trait that is stable over time without intervention (Carleton, 2012; Mahoney & McEvoy, 2012a), but without a control group we are unable to definitively attribute changes in IU to the active components of the interventions. Randomised controlled trials using control groups without the purported active treatment components are therefore required. The treatment protocols in the current study were also limited to forms of cognitive behavior therapy and to a restricted range of emotional disorders, so the findings may not generalize to other treatment approaches or disorders. There is evidence, however, that acceptance-based approaches also impact on multiple

constructs including IU (Treanor, Erisman, Salters-Pedneault, Roemer, & Orsillo, 2011). Exposure to uncertainty may also be important for reducing relapse in emotional disorders (Arch & Abramowitz, 2015), so assessing the relationship between reductions in IU and longer-term outcomes is another important future research direction.

Another limitation was that IU, RNT, and NA are only a subset of potential universal change processes, which precluded an evaluation of the relative contribution of these factors to other potentially important change processes. Future research should simultaneously compare a broader array of candidate factors (e.g., perceived control, psychological flexibility, avoidance, metacognitive beliefs), to identify those that provide incremental explanatory power. The search for universal processes will not be immune to redundancy unless multiple candidate processes are simultaneously assessed so those that (a) explain the largest proportion of unique variance in emotional disorder symptoms, and (b) most powerful and efficient at ameliorating emotional disorder symptoms, can be identified.

The search for universal processes of change offers hope for greater conceptual clarity and clinical parsimony. Hagger (2014) described the 'déjà-variable phenomenon' in the social psychology literature; "the feeling that one has seen a variable with the same definition and content before only referred to by a different term. And if not precisely identical, one can recognize considerable overlap and redundancy in the definition of variables making it difficult to establish whether constructs with different terms are appreciably different in content (p. 1)." Hagger argues that conceptual redundancies cause difficulties in statistical analysis (e.g. multicolinearity) and the interpretation of research findings, which ultimately "hinders the progress of psychological science (p. 1.)" We argue that the déjà-variable phenomenon is alive and well in the clinical psychology literature, which can result in considerable inefficiencies and obfuscations that retard the accumulation and synthesis of knowledge. Researchers investigating substantively identical constructs in parallel and

steadfastly maintaining that they are unique may be better off pooling resources and focusing instead on the vast commonalities (Castonguay, 2011). As recently noted by Mennin, Ellard, Fresco, and Gross (2013) in relation to traditional and recent CBTs, "…shining the light here [on whether recent third wave CBTs are novel] may inordinately focus the discourse on the fringes, thereby picking apart smaller differences at the boundaries while ignoring the substantial overlap and synergy of these approaches (p. 235)." We agree that much can be gained by focusing on the commonalities across disorders, theories, and therapeutic approaches to treating emotional disorders.

Mennin et al. (2013) recommended a shift from comparing the efficacy of similar therapeutic approaches towards the identification of common goals, change principles, and therapeutic processes across therapies. These researchers outlined a framework for considering these commonalities, where behavioral adaptation was the superordinate goal targeted by all CBTs and context engagement, attention change, and cognitive change were common change principles that facilitate goal attainment. Mennin et al. proposed that a range of techniques (e.g. exposure, cognitive reframing, defusion, acceptance, behavioral activation) may be emphasized to a greater or lesser extent across therapeutic approaches, but all target one or more of these common change principles. The findings from our study suggest that applying techniques to contexts, attentional biases, and cognitive content associated with IU may be important for promoting behavioral adaption for individuals high on this dimension. Treatments that increase engagement with uncertainty, modify attentional bias to threat within uncertain contexts, and modify cognitions relating to uncertainty (e.g., *I can't stand not knowing!*), may be associated with reductions in RNT and symptom relief in multiple emotional disorders. IU may therefore provide a useful framework within which to target these common processes in therapy. An individual with SAD who is uncertain about judgment, an individual with GAD who is uncertain about harm befalling a loved one, and an

individual with major depression who is uncertain about whether the future is hopeless may all benefit from engaging with contexts associated with their IU whilst learning to increase attentional flexibility by broadening it to non-threatening and hopeful aspects of the experience, and ultimately modifying the meaning of uncertainty within those contexts. For instance, therapeutic goals may be to engender a sense of curiosity in patients about whether uncertainty is in fact tolerable and often benign, and to help them discover that uncertain situations often provide important opportunities for new information and personal growth.

The most robust change processes are likely to be those that are associated with change across disorders and interventions. This study found evidence that IU was associated with reductions in RNT across different group treatment protocols for GAD, SAD, and depression, and with reductions in symptoms of GAD and SAD, even after controlling for negative affectivity. Further research is required to determine whether IU is separable from other transdiagnostic constructs, and to assess the unique contribution that targeting IU can make to relieving symptoms of emotional disorders.

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Table 1

Demographic Characteristics

Characteristic	**WRG** (N = 62)	**SAG** (N = 88)	**MMG** (N = 106)
Age	36.6 (12.3)	28.9 (10.2)	37.0 (12.7)
Female	69	55	74
Australian born	76	78	74
Employed	60	49	43
Marital Status[1]			
Single	47	80	52
Married/Defacto	44	17	27
Divorced/Separated	10	4	20
Education			
Less than High School	24	30	33
High School	9	33	27
Certificate/Diploma	10	5	20
Degree	57	32	20
Number of Diagnoses			

1	34	19	15
2	40	48	47
3 or more	26	33	38
Primary Diagnosis			
Major Depression / Dysthymia	18	15	95
Social Anxiety	0	83	2
Generalized Anxiety Disorder	73	1	2
Primary and Comorbid Diagnoses			
Major Depression / Dysthymia	31	50	100
Social Anxiety	19	100	44
Generalized Anxiety Disorder	100	28	42
Attempted Suicide	13	23	37
Previous Treatment	97	88	92
Psychiatric Hospitalisation	26	22	32
Currently Medicated	63	67	87

Note. The numbers are percentages except for age, which is a mean and standard deviation. [1] One patient in the MMG was widowed. WRG = worry and rumination group, SAG = social anxiety group, MMG = mood management group.

Table 2.

Worry and Rumination Group Outcomes

Measure and Time	*M*	SE	Mean Change from Pre-Treatment Est	95% CI		Standardized Mean Change *d*	95% CI	
IUS-12								
Pre	**39.96**	1.32						
Post	**32.99**	1.38	**6.97**	4.65	9.30	**.67**	.45	.89
Follow Up	**29.20**	1.46	**10.76**	8.37	13.16	**1.03**	.80	1.26
PANAS								
Pre	**28.26**	0.91						
Post	**20.59**	0.98	**7.67**	6.29	9.05	**1.08**	.88	1.27
Follow Up	**18.93**	0.99	**9.33**	7.90	10.76	**1.31**	1.11	1.51
RTQ								
Pre	**40.34**	0.90						
Post	**27.79**	1.25	**12.55**	10.30	14.79	**1.79**	1.47	2.11
Follow Up	**25.93**	1.37	**14.41**	11.87	16.95	**2.06**	1.70	2.42
PSWQ								

Pre	**66.53**	1.07						
Post	**51.16**	1.59	**15.37**	12.36	18.39	**1.82**	1.46	2.18
Follow Up	**47.96**	1.69	**18.57**	15.47	21.67	**2.20**	1.83	2.57

Note. Est = Estimated change from pre-to post-treatment, or pre-treatment to follow up. CI = Confidence interval. Pre-treatment *SD*s used to compute the standardized mean changes were 10.41 (IUS-12), 7.11 (PANAS), 7.00 (RTQ) and 8.44 (PSWQ). P-values for all changes in outcome during treatment were < .001. IUS-12 = Intolerance of Uncertainty Scale-12, PANAS = Positive and Negative Affect Scale, RTQ = Repetitive Thinking Questionnaire, PSWQ = Penn State Worry Questionnaire.

Table 3.

Social Anxiety Group Outcomes

Measure and Time	*M*	SE	Mean Change from Pre-Treatment			Standardized Mean Change		
			Est	95% CI		*d*	95% CI	
IUS-12								
Pre	**38.21**	1.09						
Post	**32.34**	1.29	**5.86**	3.73	7.99	**.57**	.36	.78
Follow Up	**29.16**	1.33	**9.05**	6.46	11.65	**.88**	.63	1.14
PANAS								
Pre	**28.50**	0.70						
Post	**21.78**	0.86	**6.72**	5.23	8.22	**1.03**	.80	1.25
Follow Up	**20.73**	0.91	**7.76**	6.11	9.42	**1.19**	.93	1.44
RTQ								
Pre	**38.22**	0.68						
Post	**32.61**	0.99	**5.61**	3.65	7.57	**.88**	.57	1.19
Follow Up	**30.30**	0.99	**7.92**	5.69	10.16	**1.25**	.89	1.60
SIAS								
Pre	**59.89**	1.12						
Post	**39.76**	1.64	**20.13**	16.85	23.41	**1.92**	1.61	2.23
Follow Up	**36.99**	1.91	**22.90**	19.22	26.58	**2.18**	1.83	2.53
SPS								
Pre	**43.15**	1.49						
Post	**22.92**	1.64	**20.23**	16.73	23.73	**1.50**	1.24	1.76
Follow Up	**20.96**	1.59	**22.19**	18.67	25.71	**1.65**	1.38	1.91

Note. Est = Estimated change from pre-to post-treatment, or pre-treatment to follow up. CI = Confidence interval. Pre-treatment *SD*s used to compute the standardized mean changes were 10.24 (IUS-12), 6.55 (PANAS), 6.36 (RTQ), 10.49 (SIAS), and 13.48 (SPS). P-values for all changes in outcome during treatment were < .001. IUS-12 = Intolerance of Uncertainty Scale-12, PANAS = Positive and Negative Affect Scale, RTQ = Repetitive Thinking Questionnaire, SIAS = Social Interaction Anxiety Scale, SPS = Social Phobia Scale.

Table 4.

Mood Management Group Outcomes

Measure and Time	*M*	SE	Mean Change from Pre-Treatment			Standardized Mean Change		
			Est	95% CI		*d*	95% CI	
IUS-12								
Pre	**38.58**	1.04						
Post	**34.93**	1.21	**3.64**	1.64	5.65	**.34**	.15	.52
Follow Up	**35.45**	1.36	**3.13**	0.95	5.31	**.29**	.09	.49
PANAS								
Pre	**29.56**	0.84						
Post	**24.42**	0.98	**5.14**	3.15	7.14	**.61**	.37	.84
Follow Up	**23.04**	1.05	**6.52**	4.41	8.62	**.77**	.52	1.02
RTQ								
Pre	**39.93**	0.74						
Post	**35.42**	1.04	**4.51**	2.72	6.30	**.60**	.36	.84
Follow Up	**34.76**	1.07	**5.16**	3.41	6.92	**.69**	.46	.92
BDI-II								
Pre	**34.33**	1.06						
Post	**22.61**	1.44	**11.73**	9.20	14.26	**1.09**	.85	1.33
Follow Up	**22.56**	1.69	**11.78**	8.51	15.04	**1.09**	.79	1.40

Note. Est = Estimated change from pre-to post-treatment, or pre-treatment to follow up. CI = Confidence interval. Pre-treatment *SD*s used to compute the standardized mean changes were 10.87 (IUS-12), 8.45 (PANAS), 7.49 (RTQ), and 10.76 (BDI). P-values for all changes in outcome during treatment were < .001 except for pre-treatment to follow-up change on the IUS-12 ($p = .005$). IUS-12 = Intolerance of Uncertainty Scale-12, PANAS = Positive and Negative Affect Scale, RTQ = Repetitive Thinking Questionnaire, BDI-II = Beck Depression Inventory-II.

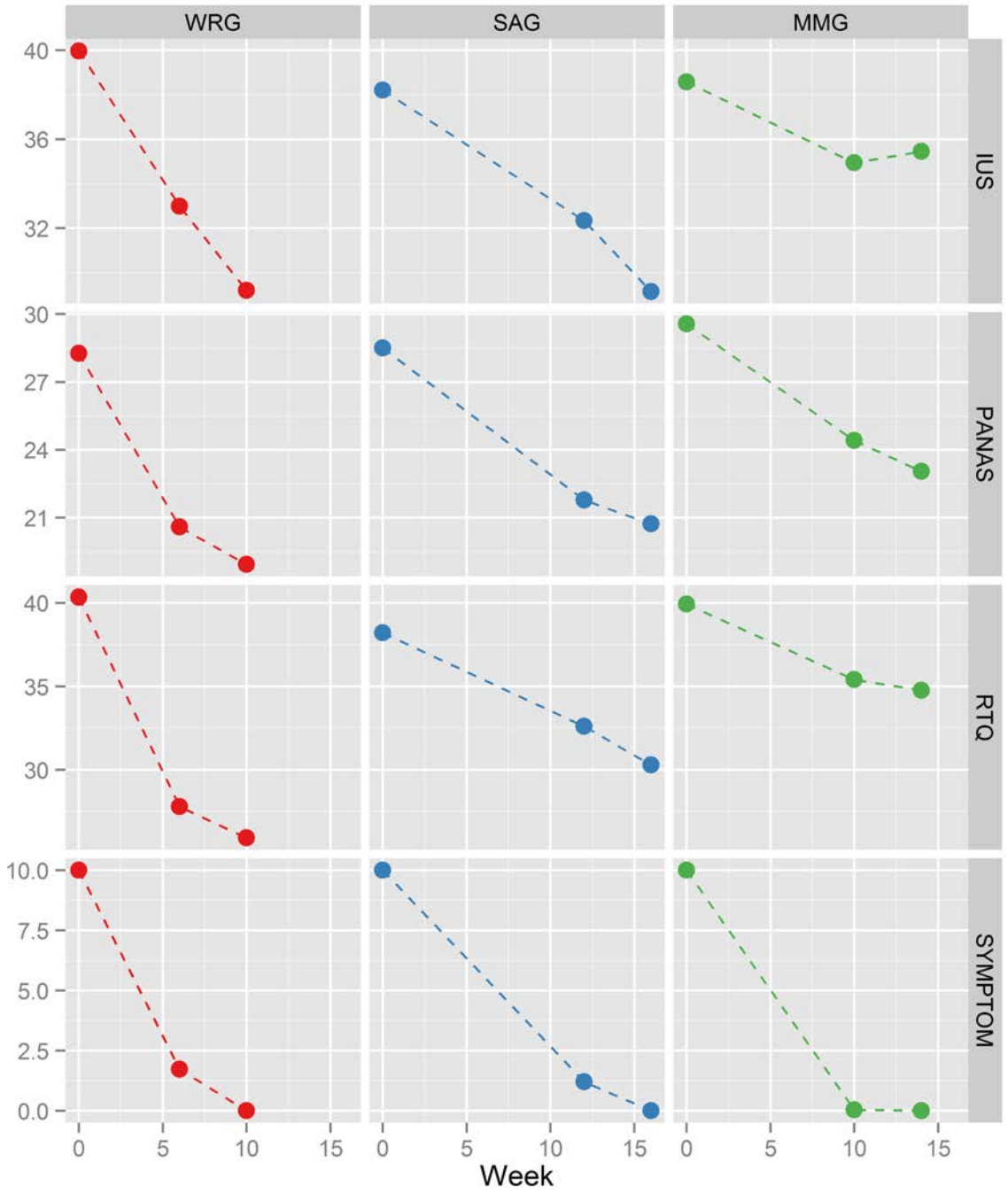

Figure 1. Mean Change Trajectories across each Treatment Program and Outcome Measure WRG = Worry and Rumination Group, SAG = Social Anxiety Group, MMG = Mood Management Group, IUS-12 = Intolerance of Uncertainty Scale-12, PANAS = Positive and Negative Affect Scale, RTQ = Repetitive Thinking Questionnaire. The bottom row contains trajectories for the symptom measures, which differed by group (Penn State worry

Questionnaire for WRG, Social Interaction Anxiety Scale for SAG, Beck Depression Inventory-II for MMG). Each measure uses a different scale, so they were rescaled to facilitate plotting. For each group, the pre-treatment symptom measure mean was rescaled to equal 10, and the follow-up mean score to equal 0. Rescaling the data allowed the trajectories to be plotted on a single axis. The rescaling only affects the numbers printed on the y-axis for these measures, not the shape of the trajectories.