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Generalized Anxiety Disorder: A Comparison of Symptom Change in Adults Receiving Cognitive-Behavioral Therapy or Applied Relaxation

Eleanor Donegan
Concordia University

Michel J. Dugas
Concordia University and Hôpital du Sacré-Coeur de Montréal

Objective: Generalized anxiety disorder (GAD) is characterized by excessive worry and somatic symptoms of anxiety (e.g., restlessness, muscle tension). Several psychological treatments lead to significant reductions in GAD symptoms by posttreatment. However, little is known about how GAD symptoms change over time. Our main goal was to examine how GAD symptoms changed in relation to one another during 2 distinct but efficacious psychological treatments: cognitive-behavioral therapy (CBT) and applied relaxation (AR). Specifically, we asked whether change in worry accounted for change over time in somatic anxiety (or the reverse) to the same degree in CBT and AR. ***Method:*** We examined data from 57 individuals with GAD enrolled in a randomized controlled trial. Self-report measures of worry and somatic anxiety were obtained daily during treatment. ***Results:*** Although the direction of influence between changes in worry and somatic anxiety was bidirectional to some extent in both treatments, a significant difference was also observed: Change in worry accounted for subsequent change in somatic anxiety to a greater extent in CBT than in AR. ***Conclusions:*** These findings allowed us to identify differences in a mechanism of change in GAD symptoms during 2 treatments and to provide some support for the idea that similarly efficacious treatments may produce symptom change via different mechanisms in a manner that is consistent with the theoretical rationales on which the treatments are based.

Keywords: generalized anxiety disorder, cognitive-behavioral therapy, mechanisms of symptom change, treatment process research

Generalized anxiety disorder (GAD) is characterized by excessive and uncontrollable worry and anxiety according to the current *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM–IV–TR*; American Psychiatric Association, 2000). Fortunately, several psychological treatments are efficacious in reducing GAD symptom severity (e.g., Borkovec & Costello, 1993; Öst & Breitholtz, 2000). Despite some success in treating GAD, however, we know surprisingly little about what occurs *during* efficacious treatments. Treatment process research has begun to address this question by identifying the rates of symptom change in particular treatments and putative mechanisms of change in other anxiety disorders (e.g., Aderka, Foa, Applebaum, Shafran, & Gilboa-Schechtman, 2011; Moscovitch, Hofmann, Suvak, & In-Albon, 2005). This research has the potential to help us better understand how symptoms change during efficacious treatments and whether symptoms change in a manner that is consistent with the theoretical models on which treatments are based.

In this study we examined how symptoms changed in two psychological treatments for GAD: cognitive-behavioral therapy (CBT) and applied relaxation (AR). The CBT protocol (see Dugas & Robichaud, 2007) is based on a cognitive theory that proposes that negative beliefs about uncertainty play an important role in the development of excessive worry; consequently, the treatment targets worry by addressing intolerance of uncertainty. AR, in contrast, is an anxiety reduction strategy that emerged from learning theories of anxiety, although its use in the treatment of GAD is appropriate given that GAD is reliably associated with muscle tension (Andrews et al., 2010). In AR, clients are taught to identify the physiological manifestations of anxiety and to apply muscle relaxation techniques to reduce these symptoms (e.g., Öst, 1987).

Despite differences in rationales and interventions, CBT and AR are often comparable in efficacy. The CBT protocol examined here has received empirical support and leads to significant reductions in worry and anxiety by posttreatment (e.g., Gosselin, Ladouceur, Morin, Dugas, & Baillargeon, 2006; Ladouceur et al., 2000). However, Dugas et al. (2010) compared this CBT to AR and found the treatments to be relatively similar in efficacy.[1] This is consistent with research demonstrating that cognitive therapy (CT) and AR are similar in efficacy when combined in the same protocol (e.g., CT vs. CT+AR; Borkovec & Costello, 1993) or as standalone treatments (e.g., CT vs. AR; Öst & Breitholtz, 2000). The availability of more than one efficacious treatment is encouraging.

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Eleanor Donegan, Department of Psychology, Concordia University, Montreal, Quebec, Canada; Michel J. Dugas, Department of Psychology, Concordia University, and Hôpital du Sacré-Coeur de Montréal, Montreal, Quebec, Canada.

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Correspondence concerning this article should be addressed to Michel J. Dugas, Department of Psychology, Concordia University, 7141 Sherbrooke Street West, Montreal, Quebec H4B 1R6, Canada. E-mail: michel.dugas@concordia.ca

[1] Dugas et al. (2010) found that CBT was superior to AR on a measure of global clinical improvement and was associated with higher diagnostic remission rates at posttreatment (70% vs. 55%) and at 2 years following treatment (77% vs. 61%). However, most direct comparisons of GAD symptom measures revealed non-significant differences in efficacy.

However, the similarity in efficacy in treatments that differ in rationales and interventions is intriguing. Symptoms may change in identical ways across treatments, or different treatments may yield similar degrees of change via different pathways.

Only one study has compared the nature of symptom change in the current CBT protocol and AR for GAD. Dugas, Francis, and Bouchard (2009) used time-series analysis to examine the temporal precedence of daily ratings of worry and somatic anxiety (i.e., restlessness, muscle tension) during treatment. Their sample included 20 individuals enrolled in a larger clinical trial by Dugas et al. (2010). Significant effects in both directions were found: Prior levels of worry predicted subsequent levels of somatic anxiety, and the reverse, for 80% of participants in CBT and 70% in AR. The authors concluded that symptom change was bidirectional in both treatments. What these findings did not reveal was whether the direction of influence between changes in worry and somatic anxiety was *greater* in one condition or the other. In CBT, worry is explicitly targeted (unlike somatic anxiety) on the assumption that reductions in worry will lead to reductions in somatic anxiety. AR explicitly targets somatic symptoms of anxiety (but not worry), with the expectation that this will lead to reductions in worry. Thus, although some degree of change may be bidirectional, we asked whether the main direction of influence between changes in worry and somatic anxiety differed across two distinct treatments.

To address this question, we conducted mediation analyses to examine how GAD symptoms changed in relation to one another in a larger sample of individuals in CBT or AR. Our sample combined the 20 participants examined by Dugas et al. (2009) with an additional 37 participants who had completed the trial by its conclusion. GAD symptom ratings were obtained on a daily basis from pre- to posttreatment and assessed the percentage of each day that participants spent worrying and experiencing somatic anxiety. We tested three hypotheses. First, consistent with previous pre–post analyses on trait GAD symptom measures (e.g., Dugas et al., 2010), we hypothesized that mean decreases in daily ratings of worry and somatic anxiety would be observed in CBT and in AR (*Hypothesis 1*). Our next analyses related to the direction of influence between changes in worry and somatic anxiety. Thus, our second hypothesis was that change in worry would account for (or mediate) change in somatic anxiety to a greater extent in CBT than in AR (*Hypothesis 2*). Third, we expected that change in somatic anxiety would account for (or mediate) change in worry to a greater extent in AR than in CBT (*Hypothesis 3*).

Method

Participants

Participants were 57 treatment-seeking adults in a randomized controlled trial at the Anxiety Disorders Clinic of the Hôpital du Sacré-Coeur de Montréal (see Dugas et al., 2010). To ensure valid initial diagnoses, participants were assessed by trained assessors using two different diagnostic interviews (see Table 1 for pretreatment characteristics). Inclusion criteria were as follows: a primary diagnosis of GAD; 18–64 years of age; stable psychoactive medication; no suicidal intent; no current substance abuse; and no current or past schizophrenia, bipolar disorder, or organic mental disorder. A total of 83 individuals were assessed, and 19 were excluded prior to treatment (see Figure 1). An additional seven participants were excluded, as they did not receive all components of their assigned treatment. The remaining 57 participants received CBT ($n = 31$) or AR ($n = 26$).

Procedure

Following the diagnostic interviews, eligible participants were invited to participate, signed a consent form, and completed pretreatment trait symptom measures. Participants were randomly assigned to treatment condition and received 12 weekly individual sessions, although clinicians could increase or decrease the number of sessions by up to two sessions as needed. Participants completed daily ratings of worry and somatic anxiety from pre- to posttreatment.

Table 1
Sample Characteristics at Pretreatment (N = 57)

Variable	CBT	AR	Significance test	*p*	*d*
Sex, *n* (% female)	20/31 (64.52)	18/26 (69.23)	$\chi^2(1) = 0.14$	.707	0.05†
Age, *M* ± *SD*	40.35 ± 12.79	36.38 ± 11.97	$F(1, 55) = 1.44$	.235	0.32
Years of education, *M* ± *SD*	15.16 ± 3.85	15.77 ± 3.34	$F(1, 55) = 0.40$	.531	−0.17
Employed full-time, *n* (%)	18/31 (58.06)	15/26 (57.69)	$\chi^2(2) = 3.97$	.138	0.26†
One or more comorbid conditions, *n* (%)	15/31 (48.39)	18/26 (69.23)	$\chi^2(1) = 2.52$	.112	−0.21†
Taking psychoactive medication, *n* (%)	18/31 (58.06)	17/26 (65.38)	$\chi^2(1) = 0.32$	.572	−0.08†
ADIS (CSR), *M* ± *SD*	5.48 ± 1.18	5.00 ± 1.89	$F(1, 55) = 1.40$	.241	0.30
PSWQ, *M* ± *SD*	60.45 ± 9.08	57.66 ± 6.48	$F(1, 55) = 1.72$	.196	0.35
BDI-II, *M* ± *SD*	13.10 ± 7.80	13.35 ± 6.75	$F(1, 55) = 0.02$	.898	−0.03
WAQ-Som, *M* ± *SD*	20.84 ± 4.00	20.96 ± 4.68	$F(1, 55) = 0.01$	.915	−0.03
WAQ-Som (restlessness, muscle tension), *M* ± *SD*	7.23 ± 1.54	7.00 ± 1.81	$F(1, 55) = 0.27$	.603	0.14
Daily Self-Monitoring Booklet (% of 1st day)					
Worry, *M* ± *SD*	39.84 ± 23.83	31.54 ± 18.37	$F(1, 55) = 2.10$	.153	0.39
Somatic anxiety, *M* ± *SD*	38.81 ± 23.66	41.73 ± 23.66	$F(1, 55) = 0.22$	.644	0.21

Note. CBT = cognitive-behavioral therapy; AR = applied relaxation; ADIS (CSR) = Clinician's Severity Rating of the Anxiety Disorders Interview Schedule for *DSM–IV*; PSWQ = Penn State Worry Questionnaire; BDI-II = Beck Depression Inventory–II; WAQ-Som = Worry and Anxiety Questionnaire, Somatic subscale.
† Phi coefficients were used to assess effect sizes for chi-square tests; $\alpha = .05$.

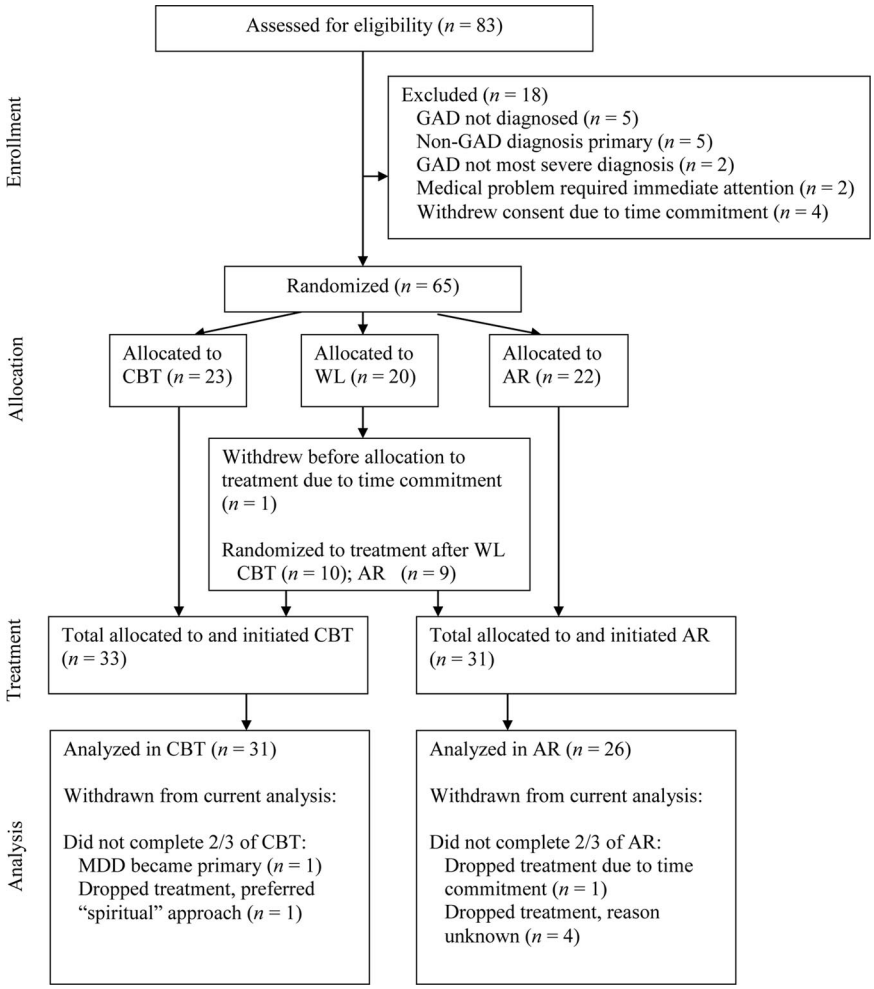

Figure 1. Participant flow diagram. GAD = generalized anxiety disorder; CBT = cognitive-behavioral therapy; WL = waitlist; AR = applied relaxation; MDD = major depressive disorder.

Treatment and Therapists

In CBT, participants received psychoeducation and worry awareness training, uncertainty recognition and behavioral exposure, re-evaluation of the usefulness of worry, problem-solving training, and imaginal exposure (see Dugas & Robichaud, 2007). In AR, participants received psychoeducation and tension awareness training, tension-release training, relaxation by recall, relaxation by counting, and conditioned relaxation training, which involved applying relaxation techniques in everyday situations (see Bernstein & Borkovec, 1973; Öst, 1987). Therapy was administered by licensed psychologists who were trained in these protocols.

Measures

Initial diagnoses were determined using the Mini International Neuropsychiatric Interview, Version 4.4 (MINI; Sheehan et al., 1994) and the Anxiety Disorders Interview Schedule for *DSM–IV* (ADIS-IV; Di Nardo, Brown, & Barlow, 1994). The Clinician's Severity Rating (CSR) of the ADIS-IV was used to assess symptom severity for MINI and ADIS-IV diagnostic interviews. Trait symptom measures were administered to ensure comparable symptom levels at pretreatment. The Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) is well-validated measure of excessive worry with high internal consistency. The Worry and Anxiety Questionnaire (WAQ; Dugas et al., 2001) assesses *DSM–IV–TR* GAD symptom severity and has fair test–retest reliability. The Somatic subscale (WAQ-Som) assesses non-worry symptoms (i.e., restlessness, fatigue, poor concentration, irritability, muscle tension, sleep disturbance). The Beck Depression Inventory–II (BDI-II; Beck, Steer, & Brown, 1996) has excellent internal consistency as well as convergent and divergent validity.

The *Daily Self-Monitoring Booklet* was used to examine the direction of influence between changes in worry and somatic anxiety during treatment. Participants recorded the percentage of each day they spent worrying and experiencing somatic anxiety between the first and final treatment session. Comparable ratings of worry correlate significantly with other valid measures of worry (e.g., PSWQ) in non-clinical and GAD samples (Dupuy, Beaudoin,

Rhéaume, Ladouceur, & Dugas, 2001). To minimize overlap, worry was defined as "a chain of upsetting thoughts about something bad that could happen to you or to others." Somatic anxiety was "a physiological reaction that includes responses such as muscle tension, restlessness, and feeling keyed up or on edge." Although GAD symptoms also include fatigue, poor concentration, irritability, and sleep disturbance, these were excluded as they are not specific to anxious responding (Andrews et al., 2010).

Data Analysis Strategy

The longitudinal data produced a multilevel structure and main analyses were conducted using multilevel modeling (HLM, Version 6.06; Raudenbush, Bryk, & Congdon, 2004). This method is appropriate, as it can accommodate missing data and adjusts for bias due to non-independence in repeated measures. All analyses involved two-level models, with repeated measurements of daily symptoms at Level 1 and participants at Level 2. The direction of influence between changes in worry and somatic anxiety was assessed in two multilevel mediation models (see Figure 2). Time (i.e., days from the start of treatment) was the initial predictor in each model. Model 1 assessed whether change in worry (mediator) accounted for change in somatic anxiety (outcome). Model 2 assessed reverse mediation or whether change in somatic anxiety (mediator) accounted for change in worry (outcome). Our approach followed recommendations by Kenny, Korchmaros, and Bolger (2003) and has been used to identify the direction of influence among changing symptoms in other anxiety disorders (e.g., Aderka et al., 2011; Moscovitch et al., 2005). Treatment condition was added at Level 2, making these analyses moderated mediation, or when groups differ on one or more components of a mediated relation (e.g., the *a* or *b* paths in Figure 2; Preacher, Rucker, & Hayes, 2007). Finally, one criterion for mediation is that the mediator precedes the outcome temporally. We created a 1-day lag between each mediator and outcome to test this temporal relation directly. Thus, we asked if change in the mediator at Time *t* accounted for change in the outcome at Time $t + 1$ (see Aderka et al., 2011, for a similar approach).[2,3]

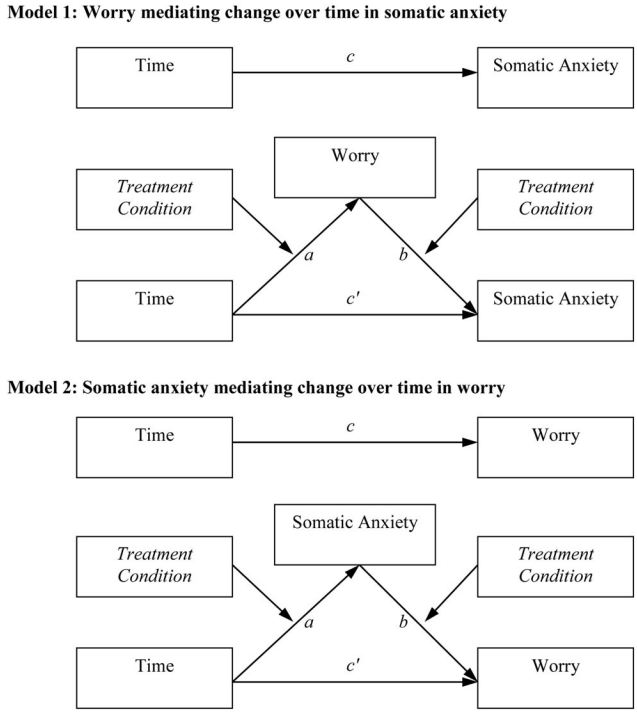

Figure 2. Hypothesized moderated mediation models. Model 1: Worry will mediate change in somatic anxiety to a greater extent in cognitive-behavioral therapy (CBT) than in applied relaxation (AR; *Hypothesis 2*). Model 2: Somatic anxiety will mediate change in worry to a greater extent in AR than in CBT (*Hypothesis 3*). A lag of 1 day was introduced such that mediators at Time *t* accounted for variance in outcome variables at Time $t + 1$.

Results

Preliminary Analyses

Interrater agreement on initial diagnoses was good for the assessed sample ($\kappa = .69$; $N = 83$). Assessors agreed on GAD severity (within 1 point on the CSR) for 75.28% of the assessed sample and 80.70% of the final sample. Treatment integrity was high in CBT (84.9%) and AR (93.7%). The groups did not differ in demographic or clinical characteristics or in trait symptoms at pretreatment. Although baseline ratings of daily symptoms were not obtained, the groups did not differ significantly in the daily ratings obtained on the first day after the start of treatment, and they did not differ on the WAQ-Som items that specifically assessed restlessness and muscle tension prior to the start of treatment (see Table 1). On average, participants received 12.21 ($SD = 0.48$; range = 11–14) treatment sessions and provided 92.53 ($SD = 16.64$) daily symptom ratings (with a mean of 2.19% of missing data per participant; $SD = 3.95$). To ensure that group differences in the direction of symptom change were not due to differences in efficacy, we conducted a Group × Time repeated measures analysis of variance (ANOVA) on the CSR. The effect of Time was significant, $F(1, 55) = 132.59$, $p < .001$, $d = 1.57$, but the interaction was not, $F(1, 55) = 2.33$, $p = .132$, $d = 0.05$, and the treatments were comparable in efficacy. Remission rates were higher in CBT (74% vs. 65%), but this difference was not statistically significant, $\chi^2(1) = 0.52$, $p = .47$, $\varphi = .10$.

Mean Change in Daily Symptoms

Significant mean linear decreases were observed in daily ratings of worry, $B = -0.12$, $t(55) = -5.13$, $p < .001$, $pr = -.16$, and the effect of condition was not significant, $B = 0.01$, $t(55) = 0.23$, $p = .823$, $d = 0.06$. Mean linear decreases were also observed in somatic anxiety, $B = -0.14$, $t(55) = -5.84$, $p < .001$, $pr = -.19$, and the effect of condition was also non-significant, $B = 0.07$, $t(55) = 1.45$, $p = .154$, $d = 0.39$. Non-linear change was assessed with power polynomials, and quadratic time (Time^2) was a signif-

[2] Although we know of no standard or recommended time interval for assessing repeated anxiety symptoms, a 1-day interval was used to maximize the precision of our analyses while limiting the burden on clients during treatment.

[3] We wish to thank Michael K. Suvak, of Suffolk University, for his recommendations regarding our statistical analyses of multilevel moderated mediation.

Table 2
Summary of Level 1 Regression Analyses for Mediation Model 1

Path	Predictor	Outcome	*B*	SE_B	*t*	*df*	*p*	*pr*
			CBT					
c	Time	Som. Anx.	−0.11	0.03	−3.29	55	.002	−.12
a	Time	Worry	−0.12	0.03	−3.98	55	<.001	−.12
b	Worry	Som. Anx.	0.49_a	0.04	11.67	55	<.001	.72
c′	Time	Som. Anx.	−0.06	0.03	−2.37	55	.022	−.05
			AR					
c	Time	Som. Anx.	−0.18	0.03	−5.34	55	<.001	−.26
a	Time	Worry	−0.13	0.03	−3.63	55	.001	−.21
b	Worry	Som. Anx.	0.37_b	0.02	15.01	55	<.001	.62
c′	Time	Som. Anx.	−0.14	0.03	−5.20	55	<.001	−.18

Note. Different subscripts indicate a significant effect of condition at $\alpha = .05$. CBT = cognitive-behavioral therapy; AR = applied relaxation; Som. Anx. = daily ratings of the percentage of each day spent experiencing somatic anxiety from pre- to posttreatment; Worry = daily ratings of the percentage of each day spent worrying from pre- to posttreatment; *pr* = partial correlation coefficients used to assess the effect size for each estimated path.

icant predictor of somatic anxiety, although only in AR, $B = 0.003$, $t(55) = -3.36$, $p = .002$, $pr = -.13$. This effect was small, and change was linear for all other daily ratings; we therefore conducted all subsequent analyses using linear time.[4]

The Direction of Influence Between Changes in Worry and Somatic Anxiety

The statistical significance of mediated effects was assessed with asymmetrical confidence intervals and the program PRODCLIN, which provides a powerful test of mediation that takes into account covariance between *a* and *b* paths (MacKinnon, Fritz, Williams, & Lockwood, 2007). The magnitude of mediated effects was estimated with percent mediation, or the proportion of the total effect ($c' + ab + \text{Cov } ab$) accounted for by the mediator (*ab*) (Shrout & Bolger, 2002). In Model 1, we examined whether change in worry accounted for change in somatic anxiety. Significant partial mediation was found in both CBT (95% CI [−0.09, −0.03]) and AR (95% CI [−0.07, −0.03]; see Table 2). There was evidence of moderated mediation, with a significant effect of condition on the *b* paths, $B = 0.13$, $t(55) = 2.61$, $p = .012$, $d = 0.70$. Change in worry accounted for 49.45% of change in somatic anxiety in CBT and 25.87% of change in somatic anxiety in AR. In Model 2, we tested reverse mediation or whether change in somatic anxiety accounted for change in worry. We again found significant partial mediation in CBT (95% CI [−0.08, −0.03]) and AR (95% CI [−0.09, −0.04]; see Table 3), and the effect of condition on the *b* paths was significant, $B = 0.12$, $t(55) = 2.45$, $p = .018$, $d = 0.66$. Change in somatic anxiety accounted for 57.76% of change in worry in CBT and 48.57% of change in worry in AR.

Discussion

Our first goal was to examine mean change in daily ratings of GAD symptoms in adults receiving 12 sessions of CBT or AR. Consistent with *Hypothesis 1*, participants in both conditions experienced significant (and comparable) reductions in the amount of time spent worrying and experiencing somatic anxiety during treatment. At the start of treatment, participants spent over 1/3 of their waking hours worrying (35.69%) and feeling anxious (40.27%). If a typical day can be estimated to be about 15 hours (e.g., from 7 a.m. to 10 p.m.), participants were worrying or feeling anxious for 5–6 hours on the first day of treatment. This is consistent with other research indicating that individuals with GAD worry about 5 hours per day (Dupuy et al., 2001). By posttreatment, about 1/5 of each day or just over 3 hours was spent worrying (20.70%) and feeling anxious (22.45%). Thus, participants were spending less time each day experiencing GAD symptoms by posttreatment, although they were clearly not asymptomatic. Considering that Dupuy et al. (2001) found that individuals in non-clinical samples report about 1 hour of worry per day, an absence of worry and anxiety is an unrealistic treatment goal. Nonetheless, it will be important to continue to refine these treatments to enhance their efficacy in the future.

Our next analyses examined the direction of influence between changes in worry and somatic anxiety during treatment. First, a significant partial mediation effect was found in both directions and in both treatment conditions. Thus, change in worry occurs in part because of change in somatic anxiety, and vice versa, in both CBT and AR. This is encouraging from a clinical point of view, as it suggests that not all symptoms of a disorder must be targeted explicitly for changes in associated symptoms to occur.

Despite this bidirectionality, our analyses revealed that the main direction of influence in symptom change differed significantly across treatments. We expected that change in worry would account for subsequent change in somatic anxiety to a greater extent in CBT than in AR (*Hypothesis 2*), and the data supported this hypothesis. Change in worry accounted for 49.45% of subsequent change in somatic anxiety in CBT but only 25.87% in AR. How-

[4] To ensure that a linear function provided the most robust estimate of change over time, we also compared models containing a linear and a true non-linear (natural log) function for each symptom variable. We compared models using the deviance statistic and model chi-square tests obtained in the HLM analyses. A linear model provided a superior fit for all daily ratings, including ratings of somatic anxiety in AR.

Table 3
Summary of Level 1 Regression Analyses for Mediation Model 2

Path	Predictor	Outcome	*B*	SE_B	*t*	*df*	*p*	*pr*
			CBT					
c	Time	Worry	−0.11	0.03	−3.74	55	.001	−.13
a	Time	Som. Anx.	−0.12	0.03	−3.40	55	.002	−.12
b	Som. Anx.	Worry	0.47_a	0.04	12.28	55	<.001	.72
c′	Time	Worry	−0.07	0.02	−3.36	55	.002	−.06
			AR					
c	Time	Worry	−0.12	0.03	−3.51	55	.001	−.20
a	Time	Som. Anx.	−0.19	0.04	−5.30	55	<.001	−.26
b	Som. Anx.	Worry	0.34_b	0.03	10.75	55	<.001	.61
c′	Time	Worry	−0.06	0.03	−2.13	55	.037	−.05

Note. Different subscripts indicate a significant effect of condition at $\alpha = .05$. CBT = cognitive-behavioral therapy; AR = applied relaxation; Worry = daily ratings of the percentage of each day spent worrying from pre- to posttreatment; Som. Anx. = daily ratings of the percentage of each day spent experiencing somatic anxiety from pre- to posttreatment; *pr* = partial correlation coefficients used to assess the effect size for each estimated path.

ever, we had also expected that change in somatic anxiety would account for subsequent change in worry to a greater extent in AR than in CBT (*Hypothesis 3*). Although change in somatic anxiety did account for a greater proportion of subsequent change in worry in the expected direction (i.e., 57.76% in AR vs. 48.57% in CBT), the difference in percent mediation between conditions was only 9.19%. Consequently, it may be prudent to conclude that change in somatic anxiety accounted for change in worry to a similar degree in both treatments. Nonetheless, we suggest that the finding that change in worry is a stronger predictor of subsequent change in somatic anxiety in CBT is consistent with the possibility that different treatments may produce a similar degree of symptom change via different mechanisms.

This study involved several limitations. First, the sample was relatively homogenous (i.e., 91% White/Caucasian; 100% Francophone). Although representative of the population from which it was drawn, this lack of diversity limits our ability to generalize our findings somewhat. Second, the distribution of comorbid conditions was somewhat atypical. The most common secondary conditions at pretreatment were panic disorder and specific phobia, rather than the more common social anxiety or major depressive disorders. Thus, it remains to be seen whether the direction of influence between changing symptoms is similar in samples with more typical comorbidity profiles. Finally, we did not assess the stability of daily symptom ratings prior to treatment and therefore cannot be sure that observed changes in symptoms would not have occurred without treatment. However, we suggest that the observed differences between treatments make this possibility somewhat less likely, although future studies should nonetheless include baseline ratings to ensure their stability prior to treatment.

Despite these limitations, the analyses presented here contribute to a growing body of treatment process research in which investigators are gaining a better understanding of the processes that occur during the pre- to posttreatment interval. Although future research should explore further why differences in the direction of influence of changing symptoms might be observed across the treatments examined here (e.g., by exploring the action of other theoretically relevant mechanisms of change), we suggest that our analyses provide support for the possibility that even when two treatments are for the most part similar in efficacy by posttreatment, differences may nonetheless exist in how these treatments bring about symptom change.

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