Cogn Behav Ther. Author manuscript; available in PMC 2010 June 25.

Published in final edited form as:
Cogn Behav Ther. 2009 ; 38(1): 29–41. doi:10.1080/16506070902980745.

Cognitive Behavioural Therapy and Applied Relaxation for Generalized Anxiety Disorder: A Time Series Analysis of Change in Worry and Somatic Anxiety

Michel J. Dugas[1,2], **Kylie Francis**[1], and **Stéphane Bouchard**[3]

[1] Department of Psychology, Concordia University, Montreal

[2] Clinique des Troubles Anxieux, Hôpital du Sacré-Cœur de Montreal, Montreal

[3] Department of Psychoeducation and Psychology, University of Québec in Outaouais, Outaouais, Quebec, Canada

Abstract

The present study examined symptom change profiles in patients with generalized anxiety disorder (GAD) receiving either cognitive behavioural therapy (CBT) or applied relaxation (AR). It was hypothesized that (a) changes in worry would uniquely predict changes in somatic anxiety for most participants receiving CBT and (b) changes in somatic anxiety would uniquely predict changes in worry for most participants in the AR condition. Twenty participants (CBT n=10; AR n=10) completed daily ratings of worry and somatic anxiety during therapy, and multivariate time series analysis was used to assess the causal impact of each variable on the other. The hypotheses were not supported because we found no evidence of a match between individual symptom change profiles and treatment condition. Rather, a bidirectional relationship between worry and somatic anxiety was observed in 80% of participants receiving CBT and 70% of participants receiving AR. When only treatment responders were considered, 83% of participants receiving CBT and 86% of those receiving AR had such a bidirectional effect. The findings are discussed in terms of models of psychopathology that posit dynamic interactions between symptom clusters and in terms of the value of examining treatment mechanisms at the individual level.

Keywords

generalized anxiety disorder; treatment mechanisms; symptom change profiles; single-case designs

Correspondence address: Michel J. Dugas, Department of Psychology, Concordia University, 7141 Sherbrooke Street West, Montreal, Quebec H4B 1R6, Canada. Tel: +514 848 2424, ext. 2215; Fax: +514 848 4537. Michel.Dugas@concordia.ca.

Research into the psychological processes involved in generalized anxiety disorder (GAD) has led to the development of a number of empirically supported and clinically useful theories of this condition. For example, the avoidance theory of GAD (Borkovec, Alcaine, & Behar, 2004) and the intolerance of uncertainty theory of GAD (Dugas & Koerner, 2005) have both received considerable empirical support and led to the development of cognitive behavioural treatments that have been tested in randomized controlled trials (e.g. Borkovec & Costello, 1993; Borkovec, Newman, Pincus, & Lytle, 2002; Dugas, Ladouceur, Léger, Freeston, et al., 2003; Ladouceur et al., 2000). Other psychological theories of GAD that have received substantial empirical support include the metacognitive theory of GAD (Wells & Carter, 2001) and the emotion dysregulation theory of GAD (Mennin, Heimberg, Turk, & Fresco, 2002). Although these theories focus on different aspects of psychological functioning, they all fit under the broadly defined cognitive or cognitive behavioural umbrella.

Besides furthering our understanding of the processes involved in the cause of GAD, the aforementioned theories have the potential to (a) increase our ability to successfully treat patients with GAD and (b) further our understanding of the mechanisms that underlie successful treatments at both the collective and individual levels. In terms of the potential of the theories of GAD to improve treatment outcomes, the data thus far are equivocal. On the one hand, the treatments that have been derived from the avoidance and intolerance of uncertainty models of GAD have produced positive outcomes (e.g. Borkovec & Costello, 1993; Borkovec et al., 2002; Dugas, Ladouceur, Léger, Freeston, et al., 2003; Ladouceur et al., 2000). On the other hand, the few controlled trials that have compared CBT based on the GAD theories with interventions based on theories of anxiety in general have not produced data that are particularly convincing. Although there is some evidence of the superiority of the GAD-specific treatments (e.g. Dugas et al., 2004), it is fair to say that only minor increments in treatment efficacy have been reported thus far.

With regard to the potential for the GAD theories to increase our understanding of the mechanisms that underlie successful treatments, research in this area is still in its infancy. At the group level, some data suggest that cognitive change may be a prerequisite to successful treatment. For example, when controlling for common therapy factors, pre-to posttreatment change in beliefs about uncertainty predicted GAD symptom scores at posttreatment and at 6- and 24-month follow-ups (Dugas, Ladouceur, Léger, Langlois, et al., 2003). At the individual level, we are aware of no research examining symptom response profiles in individuals receiving treatment for GAD. For instance, it may be that some GAD treatments tend to produce initial change in a particular subset of symptoms, whereas others are more likely to generate initial change in another symptom subset. In addition, there may be important individual differences in treatment mechanisms for the same treatment. In other words, dissimilar sequences of change may be observed in different individuals receiving the same treatment. Obviously, only analyses at the individual level can address such issues.

Time series analysis is a statistical tool that is ideally suited to examine treatment mechanisms on an individual level. This statistical technique can be used to assess the impact of variables on each other over time as well as the mutual interaction of these variables. For example, time series has previously been used to examine the temporal

relationship between variables such as mood and compulsive behaviour in obsessive–compulsive disorder (Junginger & Head, 1991). More recently, time series analysis has been used to examine the relationship between cognitive vulnerability and anxious symptoms. In a study of panic disorder, Bouchard et al. (2007) examined the temporal relationship between panic-related beliefs and panic apprehension in individuals receiving treatment. The authors found that change in panic-related beliefs preceded change in panic apprehension for the majority of patients. Important individual differences were observed, however, in the contribution of the different panic-related beliefs to the prediction of panic apprehension. Thus, by using multivariate time series analyses, Bouchard et al. were able to show a general pattern of cognitive mediation as well as individual differences in specific types of beliefs involved in predicting panic apprehension.

Dugas, Langlois, Rhéaume, and Ladouceur (1998) also used time series analyses to examine the connection between cognitive vulnerability and GAD symptoms over the course of treatment. Specifically, the authors used multivariate time series analyses to investigate the temporal relationship between negative beliefs about uncertainty and level of worry in GAD patients receiving CBT. In line with cognitive theory, the data revealed that change in beliefs about uncertainty preceded change in worry for 56% of participants, whereas change in worry preceded change in beliefs about uncertainty for only 6% of participants. Thus, the combination of single-subject designs and time series analysis has been successfully used to investigate temporal relationships between cognitive–emotional vulnerability and anxious symptoms over the course of treatment. To our knowledge, however, these procedures have never been used to examine idiographic symptom change profiles in anxious patients receiving treatment.

Generally, GAD is characterized by two main symptom clusters. The first is excessive and uncontrollable worry about a number of events and activities, whereas the second consists of six somatic symptoms: restlessness or feeling keyed up or on edge, being easily fatigued, difficulty concentrating or mind going blank, irritability, muscle tension, and sleep disturbance (*Diagnostic and Statistical Manual of Mental Disorders,* fourth edition, text revision [*DSM-IV-TR*], American Psychiatric Association, 2000). (Although the *DSM-IV-TR* uses the term “associated symptoms” to describe the second GAD symptom cluster, we prefer the term “somatic symptoms” because it is more informative and consistent with previous publications.) It is now acknowledged that GAD, like other anxiety disorders, involves a process of interacting cognitive, physiological, affective, and behavioural systems (Beck & Clark, 1997; Borkovec et al., 2002; Dugas & Koerner, 2005). In other words, change in one system typically leads to changes in other systems. Clinically, this implies that treatments for GAD do not necessarily have to target all GAD symptom clusters to ultimately achieve global change. In fact, although conventional wisdom suggests that treatments that target more than one symptom cluster (or system) have a greater chance of leading to holistic change, the evidence that this is indeed the case is weak. For example, Borkovec et al. (2002) found that cognitive therapy (targeting worry), applied relaxation (AR) and self-control desensitization (targeting somatic anxiety), and a combination of these methods were equally efficacious for the treatment of GAD. The authors hypothesized that this may have been the case because change in one GAD response system generalizes to other response systems, resulting in overall improvement within each treatment condition.

Therefore, although treatments that target different GAD symptom clusters may lead to overall improvement, the mechanisms that underlie global change may be treatment-specific. As mentioned, there may also be considerable differences in treatment mechanisms for individuals receiving the same treatment.

The goal of the current study was, therefore, to examine individual symptom change profiles in patients receiving one of two psychological treatments for GAD: CBT or AR. Specifically, we were interested in exploring the temporal relationship between change in worry and change in somatic anxiety in each treatment condition. The CBT protocol used in this study, which is based on the intolerance of uncertainty theory of GAD, targets excessive and uncontrollable worry and does not directly address somatic anxiety. AR, which is based on general theories of anxiety, targets somatic anxiety and does not directly address excessive and uncontrollable worry. Accordingly, we predicted (a) that changes in worry would uniquely predict changes in somatic anxiety for most participants receiving CBT and (b) that changes in somatic anxiety would uniquely predict changes in worry for most participants in the AR condition.

Method

Participants

The sample was made up of primary GAD patients who completed treatment in a randomized clinical trial of CBT and AR at the Anxiety Disorders Clinic of Sacré-Cœur Hospital of Montreal. Each participant was independently assessed by a clinic psychiatrist using the Mini-International Neuropsychiatric Interview-Version 4.4 (MINI) and by a graduate student trained in the administration of the Anxiety Disorders Interview Schedule for *DSM-IV* (ADIS-IV). Participants were included in the study if they met the following criteria: (a) a primary diagnosis of GAD with a severity score of at least 4/8 on the ADIS-IV (moderate clinical severity); (b) a severity score difference of at least 2 points on the ADIS-IV between GAD and all comorbid conditions; (c) age between 18 and 64 years; (d) no change in medication type or dose during 4 to 12 weeks before assessment (4 weeks for benzodiazepines, 12 weeks for antidepressants and hypnotics); (e) no evidence of suicidal intent; (f) no evidence of current substance abuse; and (g) no evidence of current or past schizophrenia, bipolar disorder, or organic mental disorder.

The final sample was made up of 20 participants—10 in the CBT condition and 10 in the AR condition—who were randomly allocated (using a random numbers sequence) to CBT or AR. Allocation concealment and implementation procedures were as follows: (a) MINI and ADIS-IV assessments were discussed at weekly team meetings; (b) a decision was reached to include or exclude the patient; (c) when a patient was accepted into the study, the research coordinator applied the random allocation sequence; and (d) after the meeting, a team psychiatrist contacted the patient to inform him or her of the decision and of the result of randomization if the patient was accepted into the study. The CBT condition included six women and four men (mean age=40.7 years, *SD*=11. 4), and the AR condition was made up of eight women and two men (mean age=33.1, *SD*=13.2). There were no significant between-group differences for either gender composition or age. Participants in both conditions were also comparable in terms of GAD severity (CBT: *M*=6.0, *SD*=0.9; AR:

M=5.3, *SD*=2.2) and number of comorbid conditions (CBT: *M*=0.5, *SD*=0.7; AR: *M*=0.4, *SD*=0.7). Finally, seven of 10 participants in each treatment condition were taking antidepressant or anxiolytic medication.

Procedure

Participants who received a primary diagnosis of GAD on both structured interviews and who met the study's other inclusion criteria were invited to participate in the study. Before treatment, participants completed a battery of self-report measures, including the Penn State Worry Questionnaire (PSWQ) and the Worry and Anxiety Questionnaire (WAQ). At the first treatment session, the therapist explained how to use the daily self-monitoring booklet. Participants were asked to rate the percentage of the day they spent worrying, experiencing somatic anxiety, and feeling depressed on a continuous scale ranging from 0 to 100%. The final page of each booklet included daily monitoring of medication use to ensure stability of dose throughout treatment. Because ratings of depression and medication were not part of the present study, only worry and somatic anxiety are discussed. To maximize the validity of the ratings, participants were provided with simple definitions for each construct. Worry was defined as "a chain of upsetting thoughts about something bad that could happen to you or to others," whereas somatic anxiety was defined as "a physiological reaction that includes responses such as muscle tension, restlessness, and feeling keyed up or on edge." We chose to exclude the other GAD somatic symptoms from our definition because of their weak specificity to anxious responding (i.e. fatigue, difficulty concentrating, irritability, and sleep disturbance are often unrelated to anxiety). The therapist encouraged participants to make their ratings of worry and anxiety at the end of each day and record the subtle changes in each symptom that occurred from day to day rather than rounding off ratings (Dupuy, Beaudoin, Rhéaume, Ladouceur, & Dugas, 2001). Treatment consisted of 12 sessions of either CBT or AR, and these conditions were matched for therapist contact and number of between-session exercises. The therapist was trained using the treatment manuals, and weekly supervision meetings were held with Michel J. Dugas. After treatment, participants were asked to complete the same battery of questionnaires administered at intake.

Measures

The MINI (Sheehan et al., 1998) is a brief structured interview that assesses the major Axis I disorders according to *DSM-IV* and International Classification of Diseases (10th edition) criteria. Although the MINI takes only 20 to 30 min to administer, it has shown good validity compared with longer measures such as the Structured Clinical Interview for *DSM-III-R*–patient edition (Spitzer, Williams, Gibbon, & First, 1990). Although the interview typically does not provide severity ratings, we used the 9-point rating scale from the ADIS-IV (see later) to obtain information about the severity of MINI diagnoses.

The ADIS-IV (Di Nardo, Brown, & Barlow, 1994) is a detailed diagnostic interview that assesses a range of *DSM-IV* Axis I disorders, with a focus on differential diagnosis between the anxiety disorders. The interview provides information on the presence of disorders with severity ratings on a 9-point Likert scale ranging from 0 (*absent or none*) to 8 (*very severe or very severely disturbing/disabling*). The diagnostic reliability of the anxiety disorders

obtained with the ADIS-IV is good, with improvements over the ADIS-III-R (Brown, Di Nardo, Lehman, & Campbell, 2001).

The daily self-monitoring booklet consisted of a small booklet made up of four pages assessing worry, somatic anxiety, depression, and medication use. Each page contained a different question (e.g. “Today, I was worried for __ % of the day”) and a grid containing seven boxes, one for each day of the week. The booklet was based on those used in our previous clinical trials for GAD (Dugas & Ladouceur, 2000; Dugas, Ladouceur, Léger, Freeston, et al., 2003; Gosselin, Ladouceur, Morin, Dugas, & Baillargeon, 2006; Ladouceur et al., 2000). Research has shown that this type of daily self-rating of worry correlates significantly with scores on the PSWQ (Dupuy et al., 2001; Verkuil, Brosschot, & Thayer, 2007), a well-established and valid measure of worry.

The PSWQ (Meyer, Miller, Metzger, & Borkovec, 1990) is a 16-item measure of uncontrollable and excessive worry. The PSWQ has high internal consistency (α=.86–.95) and good test–retest reliability over 4 weeks (r=.74–.93). It shows evidence of convergent and divergent validity because it is more highly correlated with other measures of worry than with measures of anxiety and depression (Molina & Borkovec, 1994).

The WAQ (Dugas et al., 2001) is an 11-item measure that assesses *DSM-IV* diagnostic criteria for GAD. The WAQ has satisfactory test–retest reliability over 9 weeks (diagnostic sensitivity: 75%; diagnostic specificity: 82%) and good known-groups validity (Dugas et al., 2001). As a complement to the PSWQ, which assesses the tendency to worry, only the Somatic subscale of the questionnaire (WAQ-Som) was used for this study. The WAQ-Som is both valid and sensitive to changes over treatment (Dugas, Ladouceur, Léger, Freeston, et al., 2003; Ladouceur et al., 2000).

Therapist

The same therapist (Renée Leblanc) provided treatment to all study participants. The therapist was a licensed psychologist who had not received previous training in CBT; she was trained as a psychodynamic therapist. By using a therapist who had not received extensive training in CBT, we aimed to increase the external validity of the study (i.e. generalization to more therapists). The therapist had 5 years of clinical experience, which was the result of holding a part-time private practice for mood, anxiety, and adjustment disorders. She was trained using the AR and CBT treatment manuals, and she received weekly supervision from the senior study authors.

Treatment conditions

CBT consisted of 12 individual weekly sessions of a manualized treatment based on our previous clinical trials (Dugas & Ladouceur, 2000; Dugas, Ladouceur, Léger, Freeston, et al., 2003; Gosselin et al., 2006; Ladouceur et al., 2000). The main goal of CBT was to help patients develop a greater tolerance for uncertainty in their everyday lives. The treatment included the following components: (a) psychoeducation and worry awareness training; (b) uncertainty recognition and behavioural exposure; (c) reevaluation of the usefulness of worry; (d) problem-solving training; (e) imaginal exposure; and (f) relapse prevention. As mentioned previously, the treatment does not directly target the somatic symptoms of GAD.

A detailed account of the treatment's rationale and procedures can be found in Dugas and Robichaud (2007).

AR also consisted of 12 individual weekly sessions of a manualized treatment. The treatment was based on the Bernstein and Borkovec (1973) manual and on the AR techniques used by Borkovec and Costello (1993). The goal of AR was to help patients identify the first signs of somatic anxiety (in particular, muscle tension) and apply relaxation skills to decrease their anxious responding. The AR treatment included the following components: (a) psychoeducation and tension awareness training, (b) tension-release training, (c) relaxation by recall, (d) relaxation by counting, (e) conditioned relaxation, and (f) relapse prevention.

Results

Diagnostic reliability

Diagnostic reliability was assessed by calculating agreement on the severity of GAD on the MINI and ADIS-IV. Diagnostic agreement was defined as a difference of no more than 1 point on the 9-point severity scales of the structured interviews. Using this criterion, we obtained a value of κ=.636 for the sample of 20 participants.

Treatment integrity

Treatment integrity was calculated using intervention checklists adapted from our previous studies. The CBT and AR checklists, which closely followed each respective treatment manual, included items assessing the structure of each session as well as the information to be presented and discussed within each treatment component. A trained research assistant listened to the audiotapes of one randomly selected participant in each condition to assess treatment integrity. Specifically, the research assistant compared the therapist's interventions with those described on the intervention checklist and noted whether each item was properly addressed. Treatment integrity was 88.4% in the CBT condition and 95.6% in the AR condition.

Overview of data analysis

Although all participants received 12 sessions of therapy, some took longer to complete treatment as a result of delayed sessions. The total number of daily ratings, therefore, varied among participants, ranging from 77 to 134 data points (M=90.6, SD=19.0); this is an appropriate number of data points for time series analysis (McLeary & Hay, 1980). Accordingly, we used multivariate time series analysis to evaluate the process of change in each participant's ratings of worry and somatic anxiety over the course of therapy. Multivariate time series analysis is ideal for this type of data because it uses the serial dependency of scores to generate a vectorial model for each participant. The dependency of scores is specified in a model with two components: the *autoregressive* component and the *moving average* component. This technique is, therefore, known as vector ARMA modeling (Tiao & Box, 1981). A particular strength of multivariate time series analysis is that it allows the joint testing of the serial dependency within two variables as well as the evaluation of their impact on each other over time. Therefore, in a paradigm proposed by Granger (1969)

and Weiner (1956) and operationalized by Boudjellaba, Dufour, and Roy (1992), if the parameters defining the cross-lagged effects of one variable over the other are removed and the fit of the model is significantly compromised, it can be concluded that the model needs to take into account the lagged impact of that variable. Using this causality testing technique, therefore, a variable can be tested for its predictive impact on another variable over time.

Individual model-building

Individual models were identified using time series software by Scientific Computing Associates (Liu & Hudak, 1995). Model-building consisted of four basic steps, with both worry and somatic anxiety series tested jointly, as recommended by Tiao and Box (1981). The first phase of analysis involved identifying tentative models based on cross- and extended cross-correlations, stepwise autoregressions, and smallest canonical correlations. The second estimation step required fitting the best candidate models to the data using an exact likelihood function and obtaining indices of fit such as residuals after the fit. The third diagnostic step involved applying diagnostic indices to the residual series. The best-fitting model produces few or no significant time-lagged patterns in the residuals. In addition, because Lüktepohl (1985) has argued that the best-fitting model for a series of data also has the lowest Schwarz Bayesian criterion, this criterion was also used to select the best-fitting model. Finally, constraints were applied to nonsignificant parameters by setting them to zero; this was only done if parameter estimates were significantly smaller than their standard errors and when constraints resulted in an improved fit for this “restricted” model. After these steps were carried out, a final mathematical model for the data was generated, which was used to carry out causality testing. Table 1 summarizes the models identified for each participant, with indices of model adequacy and fit. As can be seen, it was possible to obtain a model for all participants, although some models were relatively complex. Although in most cases a small number of residuals remained after the final model fit, these were the best possible models for the data; furthermore, the residuals exhibited a random pattern.

Causality testing

In vector ARMA models, causality implies that change in a variable at one time predicts change in another variable at a later time. For the current study, causality testing evaluated the null hypotheses that levels of worry did not predict later levels of somatic anxiety and, conversely, that levels of somatic anxiety did not predict later levels of worry. As specified by Boudjellaba et al. (1992), causality testing can be carried out by setting to zero the parameters describing the time-lagged impact of a variable on a second variable. If setting this parameter to zero significantly compromises the fit of the model, that variable can be considered to have a causal impact on the second variable. A chi-square analysis is used to test the significant loss in the model’s fit following the constraining of each causal variable. Table 2 provides a description of the causality tests carried out for each participant, and Table 3 summarizes these effects for each treatment condition. As can be seen in Table 3, a majority of participants in each treatment condition (eight of 10 in CBT; seven of 10 in AR) showed a bidirectional effect between worry and somatic anxiety. In other words, for most participants in each treatment condition, changes in worry predicted changes in somatic anxiety and changes in somatic anxiety predicted changes in worry. For the remaining two participants receiving CBT, either changes in worry uniquely predicted changes in somatic

anxiety (n=1) or there was no predictive effect of change in worry or somatic anxiety (n=1). For the remaining three participants in the AR condition, either changes in worry uniquely predicted changes in somatic anxiety (n=1), changes in somatic anxiety uniquely predicted changes in worry (n=1), or there was no predictive effect of change in worry or somatic anxiety (n=1).

Because some researchers have argued that analysis of treatment mechanisms should exclude participants who do not respond fully to treatment (e.g. Hoffart, 1996), we reexamined the time series data for treatment responders only. Treatment response was assessed by examining pre- to posttreatment changes in the questionnaire measures of worry (PSWQ) and GAD somatic symptoms (WAQ-Som). Consistent with previous studies (e.g. Borkovec & Costello, 1993; Dugas, Ladouceur, Léger, Freeston, et al., 2003), treatment response was defined as a 20% or greater improvement on the GAD symptom measures; participants who had not attained a 20% improvement on both measures were considered nonresponders. Using this criterion, six participants in the CBT condition and seven participants receiving AR were considered treatment responders. Of the CBT responders, five had a bidirectional effect between worry and somatic anxiety, and one had no predictive effect of either change in worry or somatic anxiety. Among AR responders, six had a bidirectional effect between worry and somatic anxiety, whereas changes in worry uniquely predicted changes in somatic anxiety for one participant.

Discussion

The study's hypotheses were not supported: change in worry did not uniquely predict change in somatic anxiety for most patients receiving CBT, and change in somatic anxiety did not uniquely predict change in worry for most patients receiving AR. Rather, 80% of participants receiving CBT and 70% receiving AR showed a bidirectional relationship in which change in worry and somatic anxiety equally predicted each other. Furthermore, when only treatment responders were considered, 83% of participants receiving CBT and 86% of those receiving AR had a bidirectional effect between worry and somatic anxiety. In retrospect, this pattern of findings is not entirely surprising. It may be that for most patients change in one symptom cluster leads to change in a second symptom cluster, which in turn "feeds forward" to the first symptom cluster, so that a bidirectional relationship is set in motion. As Bouchard et al. (2007) point out, the presence of a bidirectional relationship does not rule out the possibility that change in one variable may have preceded and initiated the bidirectional relationship that appears between two variables. However, the actual initiation of the process of interaction may be too subtle to be identified. Of course, the design of the present study does not permit the examination of these specific questions.

The interacting cognitive subsystems (ICS) model described by Teasdale (1993, 1997) offers an interesting perspective on the bidirectional findings of the present study. The ICS model includes two levels of meaning: a propositional level, relating to specific concrete meanings, and a holistic level, relating to higher order, emotional knowledge. Teasdale argues that emotion occurs at the holistic level, and only when certain patterns of stimuli are present at the propositional level (e.g. the presence of a snake + autonomic arousal + perception of self as in danger). One implication of Teasdale's model is that meaningful therapeutic change

occurs at the holistic level, through the pattern of activation of propositional subsystems. Furthermore, it is assumed that change in any one system will lead to change in other systems. More specifically, however, Teasdale's model proposes that it would be possible to create change at the holistic level by targeting only one element of the system at the propositional level (i.e. cognitive, physiological, or behavioural). One can easily see how these ideas can be applied to the bidirectional results of the current study. This interpretation makes sense on a clinical level: if change in one symptom of a disorder leads to change in a second symptom, it is difficult to imagine that change in the second symptom would not contribute to further change in the first. This may be particularly true among clients who are successfully treated and demonstrate clinically significant change: in other words, among treatment responders.

An alternative explanation for the prevalence of bidirectional effects in this study is that worry and anxiety may not have been sufficiently differentiated by participants. To explore this possibility, we calculated correlations between daily ratings of worry and anxiety for individual participants and found that all correlations were significant and ranged from .45 to .95. For 16 of the 20 participants, the correlation did not exceed .90, indicating that ratings of worry and anxiety were not multicollinear or singular. In other words, worry and anxiety were considered to be related but distinct constructs (Tabachnick & Fidell, 2001). Of the four participants for whom worry and anxiety were correlated at more than .90, three had a bidirectional relationship and one had no relationship. A point-biserial correlation showed that the degree of relationship between worry and anxiety was not related to the presence of a bidirectional relationship, $r(20)=.21$, $p=.37$. Overall, these rough estimates suggest that the prevalence of bidirectional results found in this study was not due to an insufficient differentiation between worry and anxiety.

In addition to the finding of a predominance of bidirectional effects between worry and somatic anxiety, a second noteworthy finding of this study is the presence of individual differences in symptom change profiles within each treatment condition. For example, one CBT responder showed no predictive effect of either change in worry or change in anxiety, suggesting that the mechanisms leading to successful CBT can vary from one individual to another. In the AR condition, changes in worry uniquely predicted changes in anxiety for one treatment responder. This latter finding is particularly intriguing because one might expect that successful AR would lead either to changes in somatic anxiety uniquely predicting changes in worry or to a bidirectional relationship between changes in worry and somatic anxiety. What this finding suggests, however, is that initial changes in cognition (in this case, worry) can lead to subsequent changes in somatic anxiety in at least some individuals receiving AR. Incidentally, as clinicians, we have often been surprised by the considerable cognitive change that can occur when anxious individuals begin using relaxation techniques. Thus, the finding that changes in worry led to changes in somatic anxiety for one study participant receiving AR is not inconsistent with our clinical experience.

In summary, the use of multivariate time series analysis to address individual symptom change profiles revealed (a) a bidirectional relationship between worry and somatic anxiety in most GAD patients receiving either CBT or AR and (b) individual variability in change

profiles within each treatment condition. It is worth noting that such results can be obtained only with single-subject designs and that individual variability cannot be captured with group or between-group designs. Although the study of individual differences in treatment mechanisms is certainly an ambitious endeavour, we believe that it is well worthwhile. By furthering our understanding of individual variability in how treatments exert their influence, we will be better able to appreciate the heterogeneity of treatment mechanisms and guard against “one-size-fits-all” conceptualizations of the processes that underlie therapeutic change. Ultimately, a better understanding of individual differences in treatment mechanisms has the potential to increase the efficacy of psychological treatments. Consequently, rather than pitting group designs against single-subject designs, we would do well to consider these as complementary research plans, with each one providing different information about the mechanisms and efficacy of treatment.

Although this study had a number of important strengths, such as the use of sophisticated analytical procedures in a carefully diagnosed sample, it also had a number of notable limitations. First and foremost, the study relied on subjective ratings of worry and somatic anxiety to examine symptom change profiles in participants. In particular, the self-reports of somatic anxiety would have been strengthened had we obtained convergent physiological evidence. Moreover, the reports of worry and somatic anxiety were obtained retrospectively at the end of each day. Although one could certainly argue that daily retrospective ratings are not particularly susceptible to memory biases given the relatively short time frame between assessments, these ratings are nonetheless not as valid as online ratings of worry and somatic anxiety. Thus, the subjective and retrospective nature of the main dependent variables represents an important limitation of the present study.

Another limitation of the current study has to do with its sample size. Although one could argue that the study had a relatively large sample given the nature of the research design (almost 20 single-case replications), the small number of participants (by conventional standards) certainly limits the generalizability of the findings. Ideally, replication in a larger sample would allow one to be more confident about the generalizability and clinical implications of the current findings.

A third limitation relates to the use of time series analysis per se. Time series analysis requires considerable variability in the data for the generation of adequate models (McLeary & Hay, 1980). Although visual analysis of the data suggested adequate variability for all participants, there was a range of variability among participants, which may have influenced the results. A related concern is that no predictive effects of either worry or somatic anxiety were found for two participants in this study. Given the proposed connection between GAD response systems, this lack of a relationship was unexpected. A visual examination suggests that these participants have data variability comparable to the rest of the sample; however, one of these two participants was also classified as a treatment nonresponder. It might be speculated that a lack of relationship between worry and somatic anxiety reflects a lack of treatment response; however, this is conjectural and does not explain why the remaining participant, a treatment responder, also did not show a predictive impact of worry or somatic anxiety.

Another time series caveat relates to the nature of causality testing. Although this technique provides information about the predictive relationships between variables for each participant, it does not actually provide a definitive test of causality because a number of the conditions to establish causality cannot be addressed by simply examining precedence of change (for a discussion of causal risk, see Kraemer et al., 1997). Thus, although "causality" is used to describe the test that change in one variable predicts subsequent change in another, one could argue that this term is somewhat misleading given that only temporal antecedence is addressed via this technique.

Although the findings of the present study should be interpreted in light of the aforementioned limitations, one conclusion can certainly be drawn from the present study: the importance and benefit of examining therapeutic change at the individual level. As Hilliard (1993) pointed out, analysis of individual change is sadly lacking in the field of psychotherapy research. In the current study, the demonstration of a bidirectional relationship between worry and somatic anxiety in most participants in each treatment condition sheds additional light on the processes that may explain this type of response to treatment; this information would not have been obtained if group differences alone had been examined. Future psychotherapy research may benefit from adding analyses at the individual level to comparisons of group differences, thereby providing valuable information about individual differences and the mechanisms of change for different treatments.

Acknowledgments

Preliminary findings were presented at the 2008 conference of the British Association for Behavioural and Cognitive Psychotherapies, Edinburgh, UK. This study was supported by the Canadian Institutes of Health Research and the Fonds de la Recherche en Santé du Québec. We thank Naomi Koerner and Nina Laugesen for their advice on the statistical procedures used in this study and Marina Katerelos for her help with data entry.

References

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: Author; 2000. text rev

Beck AT, Clark DA. An information processing model of anxiety: Automatic and strategic processes. Behaviour Research and Therapy. 1997; 35:49–58. [PubMed: 9009043]

Bernstein, DA., Borkovec, TD. Progressive relaxation training. Champaign, IL: Research Press; 1973.

Borkovec, TD., Alcaine, OM., Behar, E. Avoidance theory of worry and generalized anxiety disorder. In: Heimberg, R.Turk, C., Mennin, D., editors. Generalized anxiety disorder: Advances in research and practice. New York: Guilford Press; 2004. p. 77-108.

Borkovec TD, Costello E. Efficacy of applied relaxation and cognitive-behavioral therapy in the treatment of generalized anxiety disorder. Journal of Consulting and Clinical Psychology. 1993; 61:611–619. [PubMed: 8370856]

Borkovec TD, Newman MG, Pincus AL, Lytle R. A component analysis of cognitive-behavioural therapy for generalized anxiety disorder and the role of interpersonal problems. Journal of Consulting and Clinical Psychology. 2002; 70:288–298. [PubMed: 11952187]

Bouchard S, Gauthier J, Nouwen A, Ivers H, Vallières A, Simard S, Fournier T. Temporal relationship between dysfunctional beliefs, self-efficacy and panic apprehension in the treatment of panic disorder with agoraphobia. Journal of Behavior Therapy and Experimental Psychiatry. 2007; 38:275–292. [PubMed: 17157264]

Boudjellaba H, Dufour JM, Roy R. Testing causality between two vectors in multivariate autoregressive moving average models. Journal of the American Statistical Association. 1992; 87:1082–1090.

Brown TA, Di Nardo PA, Lehman CL, Campbell LA. Reliability of DSM-IV anxiety and mood disorders: Implications for the classification of emotional disorders. Journal of Abnormal Psychology. 2001; 110:49–58. [PubMed: 11261399]

Di Nardo, PA., Brown, TA., Barlow, DH. Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV). San Antonio, TX: Psychological Corporation; 1994.

Dugas MJ, Freeston MH, Provencher MD, Lachance S, Ladouceur R, Gosselin P. Le Questionnaire sur l'Inquiétude et l'Anxiété. Validation dans des échantillons non cliniques et cliniques [The Worry and Anxiety Questionnaire: Validation in nonclinical and clinical samples]. Journal de Thérapie Comportementale et Cognitive. 2001; 11:31–36.

Dugas MJ, Koerner N. Cognitive-behavioral treatment for generalized anxiety disorder: Current status and future directions. Journal of Cognitive Psychotherapy: An International Quarterly. 2005; 19:61–81.

Dugas MJ, Ladouceur R. Treatment of GAD: Targeting intolerance of uncertainty in two types of worry. Behavior Modification. 2000; 24:635–657. [PubMed: 11036732]

Dugas MJ, Ladouceur R, Léger E, Freeston MH, Langlois F, Provencher M, Boisvert JM. Group cognitive-behavioral therapy for generalized anxiety disorder: Treatment outcome and long-term follow-up. Journal of Consulting and Clinical Psychology. 2003; 71:821–825. [PubMed: 12924687]

Dugas, MJ., Ladouceur, R., Léger, E., Langlois, F., Provencher, MD., Boisvert, J-M., Freeston, MH. Group CBT for generalized anxiety disorder: Does in tolerance for uncertainty predict symptom change beyond non-specific therapy factors?. Poster presented at the conference of the Association for Advancement of Behavior Therapy; Boston, MA. 2003 Nov.

Dugas, MJ., Langlois, F., Rhéaume, J., Ladouceur, R. Intolerance of uncertainty and worry: Investigating causality. In: Stoeber, J., editor. Worry: New findings in applied and clinical research; Symposium presented at the conference of the Association for Advancement of Behavior Therapy; Washington, DC: 1998 Nov.

Dugas, MJ., Robichaud, M. Cognitive-behavioral treatment for generalized anxiety disorder: From science to practice. New York: Routledge; 2007.

Dugas, MJ., Savard, P., Gaudet, A., Turcotte, J., Brillon, P., Leblanc, R., Ladouceur, R. Cognitive-behavioral therapy versus applied relaxation for generalized anxiety disorder: Differential outcomes and processes. In: Hazlett-Stevens, H., editor. New advances in the treatment of chronic worry and generalized anxiety disorder; Symposium presented at the conference of the Association for Advancement of Behavior Therapy; New Orleans, LA. 2004 Nov.

Dupuy JB, Beaudoin S, Rhéaume J, Ladouceur R, Dugas MJ. Worry: Daily self-report in clinical and non-clinical populations. Behaviour Research and Therapy. 2001; 39:1249–1255. [PubMed: 11579992]

Gosselin P, Ladouceur R, Morin CM, Dugas MJ, Baillargeon L. Benzodiazepine discontinuation among adults with GAD: A randomized trial of cognitive-behavioral therapy. Journal of Consulting and Clinical Psychology. 2006; 74:908–919. [PubMed: 17032095]

Granger CWJ. Investigating causal relations by econometric models and cross-spectral methods. Econometrica. 1969; 37:424–438.

Hilliard RB. Single-case methodology in psychotherapy process and outcome research. Journal of Consulting and Clinical Psychology. 1993; 61:373–380. [PubMed: 8326037]

Hoffart A. In vivo cognitive therapy of panic attacks. Journal of Cognitive Psychotherapy. 1996; 10:281–289.

Junginger J, Head S. Time series analysis of obsessional behaviour and mood during self-imposed delay and response prevention. Behaviour Research and Therapy. 1991; 29:521–530. [PubMed: 1759952]

Kraemer HC, Kazdin AE, Offord DR, Kessler RC, Jensen PS, Kupfer DJ. Coming to terms with the terms of risk. Archives of General Psychiatry. 1997; 54:337–343. [PubMed: 9107150]

Ladouceur R, Dugas MJ, Freeston MH, Léger E, Gagnon F, Thibodeau N. Efficacy of a new cognitive-behavioral treatment for generalized anxiety disorder: Evaluation in a controlled clinical trial. Journal of Consulting and Clinical Psychology. 2000; 68:957–964. [PubMed: 11142548]

Liu, L-M., Hudak, GB. The SCA statistical system reference manual for forecasting and time series analysis. Oak Brook, IL: Scientific Computing Associates; 1995.

Lüktepohl H. Comparison of criteria for estimating the order of a vector autoregressive process. Journal of Time Series Analysis. 1985; 6:35–52.

McLeary, R., Hay, RA. Applied time-series analysis for the social sciences. Thousand Oaks, CA: Sage; 1980.

Mennin DS, Heimberg RG, Turk CL, Fresco DM. Applying an emotion regulation framework to integrative approaches to generalized anxiety disorder. Clinical Psychology: Science and Practice. 2002; 9:85–90.

Meyer TJ, Miller ML, Metzger RL, Borkovec TD. Development and validation of the Penn State Worry Questionnaire. Behaviour Research and Therapy. 1990; 28:487–495. [PubMed: 2076086]

Molina, S., Borkovec, TD. The Penn State Worry Questionnaire: Psychometric properties and associated characteristics. In: Davey, GCL., Tallis, F., editors. Worrying: Perspectives on theory, assessment and treatment. New York: Wiley; 1994. p. 265-283.

Sheehan DV, Lecrubier Y, Sheehan KH, Amorim P, Janavs J, Weiller E, et al. The Mini-International Neuropsychiatric Interview (MINI): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. Journal of Clinical Psychiatry. 1998; 59(Suppl 20): 22–33.

Spitzer, RL., Williams, JBW., Gibbon, M., First, MB. Structured clinical interview for DSM-III-R–patient edition (SCID-P, Version 1.0). Washington, DC: American Psychiatric Press; 1990.

Tabachnick, BG., Fidell, LS. Using multivariate statistics. 4. Boston, MA: Allyn & Bacon; 2001.

Teasdale JD. Emotion and two kinds of meaning: Cognitive therapy and applied cognitive science. Behaviour Research and Therapy. 1993; 31:339–354. [PubMed: 8512536]

Teasdale, JD. The transformation of meaning: The interacting cognitive subsystems approach. In: Clark, DM., Fairburn, CG., editors. Science and practice of cognitive behaviour therapy. New York: Oxford University Press; 1997. p. 67-93.

Tiao GC, Box GEP. Modelling multiple time series with applications. Journal of the American Statistical Association. 1981; 76:802–816.

Verkuil B, Brosschot JF, Thayer JF. Capturing worry in daily life: Are trait questionnaires sufficient? Behaviour Research and Therapy. 2007; 45:1835–1844. [PubMed: 17382896]

Weiner, N. The theory of prediction. In: Breckenbach, EF., editor. Modern mathematics for engineers, Series 1. New York: McGraw-Hill; 1956. p. 165-190.

Wells A, Carter K. Further tests of a cognitive model of generalized anxiety disorder: Metacognitions and worry in GAD, panic disorder, social phobia, depression and non-patients. Behavior Therapy. 2001; 32:85–102.

Table 1

Summary of ARMA models for each participant with indices of model adequacy

Participant	Condition	Autoregressive parameters	Moving average parameters	SBC	Residuals exceeding critical χ^2
1	CBT	4	1, 6	746.70	1
2	CBT	4	1, 8	964.76	2
3	CBT	1	3, 14	911.92	0
4	CBT	1, 3	4	760.03	2
5	CBT	1, 5, 8, 10	8	483.14	3
6	CBT	1, 4, 6, 8, 10	—	595.43	3
7	CBT	1	9	1042.35	0
8	CBT	1	2, 4	1327.35	0
9	CBT	1, 3, 6, 7, 9	1	1136.46	4
10	CBT	1, 3	1	535.12	5
11	AR	1	1, 3, 5	1080.22	3
12	AR	1	1	803.45	3
13	AR	2, 4, 6, 8	—	768.34	0
14	AR	1, 4, 6	2, 3	1567.63	1
15	AR	1, 2, 3, 4, 5, 6, 7, 8	—	663.29	7
16	AR	1, 3, 9	3	468.59	3
17	AR	2	1	957.34	1
18	AR	1	1	883.62	2
19	AR	1, 9	11	1368.45	1
20	AR	1, 2	2, 9	1026.28	4

Note. For Participants 9, 10, 15, and 20, these were the best possible models for the data despite a slightly high number of residuals. ARMA=autoregressive/moving average; SBC=Schwarz's Bayesian criterion, reported for the final restricted model; CBT=cognitive behavioural therapy; AR=applied relaxation.

Table 2

Causality testing for each participant

Participant	Condition	Null hypothesis	Parameter(s) constrained	χ^2	*df*	*p*
1	CBT	Worry −/→ anxiety	$\varphi^4_{21}=0$	9.66	1	.002
		Anxiety −/→ worry	$\varphi^4_{12}=0$	4.81	1	.030
2	CBT	Worry −/→ anxiety	$\theta^8_{21}=0$	7.63	1	.010
		Anxiety −/→ worry	$\theta^8_{12}=0$	13.32	1	.000
3	CBT	Worry −/→ anxiety	$\varphi^1_{21}=0$	10.30	1	.001
		Anxiety −/→ worry	$\theta^3_{12}=0$	11.92	1	.000
4	CBT	Worry −/→ anxiety	$\varphi^4_{21}=0$	18.61	1	.000
		Anxiety −/→ worry	$\varphi^3_{12}=0$	6.31	1	.010
5	CBT	Worry −/→ anxiety	$\varphi^{10}_{21}=0$	24.67	1	.000
		Anxiety −/→ worry	$\varphi^{10}_{12}=0$	14.28	1	.000
6	CBT	Worry −/→ anxiety	$\varphi^1_{21}=0$	15.78	1	.000
		Anxiety −/→ worry	$\varphi^6_{12}=0$	16.13	1	.000
7	CBT	Worry −/→ anxiety	*ns*			
		Anxiety −/→ worry	*ns*			
8	CBT	Worry −/→ anxiety	$\theta^4_{21}=0$	20.83	1	.000
		Anxiety −/→ worry	*ns*			
9	CBT	Worry −/→ anxiety	$\varphi^9_{21}=0$	5.98	1	.020
		Anxiety −/→ worry	$\theta^1_{12}=0$	13.84	1	.000
10	CBT	Worry −/→ anxiety	$\theta^1_{21}=0$	8.05	1	.004
		Anxiety −/→ worry	$\theta^1_{12}=0$	10.96	1	.000
11	AR	Worry −/→ anxiety	$\theta^1_{21}=0$	16.65	1	.000
		Anxiety −/→ worry	*ns*			
12	AR	Worry −/→ anxiety	*ns*			
		Anxiety −/→ worry	$\varphi^1_{12}=0$	9.59	1	.002
13	AR	Worry −/→ anxiety	$\varphi^8_{21}=0$	4.53	1	.030

Participant	Condition	Null hypothesis	Parameter(s) constrained	χ^2	*df*	*p*
		Anxiety −/→ worry	$\varphi^{3}_{12}=0$	4.80	1	.030
14	AR	Worry −/→ anxiety	$\varphi^{1}_{21}=0$	20.83	1	.000
		Anxiety −/→ worry	$\varphi^{1}_{12}=0$	56.45	1	.000
15	AR	Worry −/→ anxiety	$\varphi^{2}_{21}=0$	31.54	1	.000
		Anxiety −/→ worry	$\varphi^{1}_{12}=0$	12.55	1	.000
16	AR	Worry −/→ anxiety	$\theta^{3}_{21}=0$	10.81	1	.001
		Anxiety −/→ worry	$\varphi^{3}_{12}=0$	12.57	1	.000
17	AR	Worry −/→ anxiety	*ns*			
		Anxiety −/→ worry	*ns*			
18	AR	Worry −/→ anxiety	$\theta^{1}_{21}=0$	7.87	1	.005
		Anxiety −/→ worry	$\theta^{1}_{12}=0$	16.86	1	.000
19	AR	Worry −/→ anxiety	$\theta^{1}_{21}=0$	132.81	1	.000
		Anxiety −/→ worry	$\theta^{1}_{12}=0$	4.94	1	.030
20	AR	Worry −/→ anxiety	$\varphi^{2}_{21}=0$	6.60	1	.010
		Anxiety −/→ worry	$\varphi^{2}_{12}=0$	43.33	1	.000

Note. CBT=cognitive behavioural therapy; AR=applied relaxation; −/→=does not predict; φ=autoregressive parameter; θ=moving parameter; *ns*=a parameter that was not significant at any lag (a test was not carried out because causality was not demonstrated).

Table 3

Summary of effects demonstrated by causality testing in cognitive behavioural therapy (n=10) and applied relaxation (n=10)

Direction of change	No. cases	Percentage of total
Cognitive behavioural therapy		
Worry → anxiety	1	10%
Anxiety → worry	0	0%
Worry ↔ anxiety	8	80%
No causal relationship	1	10%
Applied relaxation		
Worry → anxiety	1	10%
Anxiety → worry	1	10%
Worry ↔ anxiety	7	70%
No causal relationship	1	10%