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Anxiety in older adults: prevalence and low-threshold psychological interventions

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MEDIATORS AND PREDICTORS OF CHANGE IN COGNITIVE BEHAVIORAL THERAPY AND ACCEPTANCE AND COMMITMENT THERAPY FOR ANXIETY SYMPTOMS

Manuscript under review: Witlox M, Kraaij V, Garnefski N, Bohlmeijer ET, Spinhoven P. Mediators and predictors of change in Cognitive Behavioral Therapy and Acceptance and Commitment Therapy for anxiety symptoms.

Abstract

Background: Research suggests that Cognitive Behavioral Therapy (CBT) and Acceptance and Commitment Therapy (ACT) are equally effective in the treatment of anxiety symptomatology. So far, little empirical evidence is available on the working mechanisms of both treatments.

Objective**:** This study examined multiple candidate mechanisms of change in CBT and ACT for anxiety in terms of their prospective and/or mediational role. It was hypothesized that reappraisal mediated change in anxiety symptom severity in CBT. Acceptance, rumination, distraction and suppression were hypothesized to be ACT-specific mediators. Furthermore, behavioral avoidance, therapeutic alliance and treatment expectancies were hypothesized to be prospectively predictive of anxiety symptom severity in both treatments.

Methods: Data were collected as part of a randomized controlled trial comparing the effects of CBT and ACT in a sample of 314 older adults (aged 55-75 years) with anxiety symptomatology. Participants filled in self-report questionnaires assessing anxiety symptom severity (Generalized Anxiety Disorder-2) and the candidate mechanisms a total of five times over the course of treatment. Random intercept-cross lagged panel models were used to model the hypothesized prospective and mediational relationship on the within-person level.

Results: None of the candidate mechanisms were found to be mediators or prospective predictors of anxiety symptom severity over the course of the CBT and ACT intervention.

Conclusions**:** The examined candidate mechanisms were not found to be predictors or mediators of anxiety symptom change in CBT and ACT. The discrepancy with previous positive findings may be attributed to earlier studies not using a longitudinal design and analysis on the within-person level.

Introduction

Anxiety disorders and symptoms form the most common class of adult psychological problems [287,288]. Over the past decades, Cognitive Behavioral Therapy (CBT) has become the most empirically supported psychological treatment for anxiety [289-291]. More recent studies have demonstrated that Acceptance and Commitment Therapy (ACT) has similar effects as CBT in the treatment of anxiety symptoms in adults [53,243,292]. Our research team recently conducted the first large-scale randomized controlled trial (RCT) comparing CBT and ACT in a large sample of *older* adults with anxiety symptomatology [250] and also found no important differences between the two interventions regarding their effects on anxiety symptom severity and related clinical outcomes. Significant reductions of anxiety symptom severity (effect sizes d $\geq$.96) were observed in both the CBT and the ACT condition between baseline and posttreatment and were sustained at the one year follow up. Research so far thus suggests that CBT and ACT do not differ regarding their effectiveness in treating adults with anxiety.

Although the effectiveness of CBT and ACT for anxiety has been demonstrated, relatively little research has been conducted into the mechanisms through which these treatments lead to anxiety symptom change. Investigating the mechanisms that might be responsible for psychotherapeutic change will lead to a better understanding of the theoretical underpinnings of the treatments and provide directions for treatment augmentation strategies [227]. Convincingly demonstrating *the causal role* of proposed mechanisms of change is complicated and requires a series of studies and experiments. An important first step in understanding mechanisms of change in psychotherapy is the identification of treatment mediators: variables that statistically account for the relationship between treatment and treatment outcome [227].

One of the most common shortcomings of studies into treatment mediators is that they lack the establishment of a timeline that shows that the candidate mediator precedes the outcome, which is a necessary (but not sufficient) condition for mediation [227, 293]. Most studies into treatment mediation only assess the putative mediator(s) and outcome variable(s) at baseline and after treatment. Such studies cannot distinguish whether change in the putative mediator indeed *precedes* symptom change, *co-occurs* with symptom change, or *follows* symptom change. Rigorous examination of treatment mediators requires a study design in which the proposed mechanism of change and the outcome variable are repeatedly assessed during treatment. That is why in the current study we used data from multiple timepoints during treatment to

examine the temporal relationships between candidate mechanisms of change and anxiety symptom improvement in a brief CBT intervention and a brief ACT intervention for older adults with anxiety symptoms.

Theories of change in CBT and ACT

Both CBT and ACT are developed with an explicit theoretical notion of how the treatment leads to change. The two treatments can be most clearly distinguished by how they proposedly influence cognitive emotion regulation strategies. On the one hand, CBT aims to reduce the frequency and intensity of anxiety symptoms by identifying and adapting anxiety related cognitions. Through a process of reappraisal, unrealistic negative thoughts concerning the threat posed by certain situations, events or bodily sensations are replaced with more nuanced and adaptive thoughts [44]. Meta-analyses concluded that CBT for anxiety disorders indeed leads to improvements in threat reappraisal [294] and that these improvements are associated with reductions in anxiety symptom severity [295], but that there is not enough evidence yet to conclude that changes in threat reappraisal *cause* symptom improvement in CBT

Contrary to CBT, ACT does not directly focus on changing or reducing anxious feelings and thoughts, but instead stimulates active acceptance of all internal experiences, including those we tend to label as 'negative', 'unwanted' or 'harmful'. A more accepting stance towards internal experiences is theorized to lead and to less use of cognitive and behavioral strategies aimed at changing or controlling emotions or thoughts, that actually sustain or exaggerate anxiety (e.g., rumination, distraction, suppression, behavioral avoidance) [296]. Two review articles on the working mechanisms of ACT concluded that changes in constructs related to the acceptance of inner experiences seem to occur prior to changes in psychological symptoms, but strong causal evidence is lacking [64,297].

So far, three studies have directly compared cognitive emotion regulation strategies as mediators in CBT and ACT for anxiety, using session-by-session data [298-300]. These studies present mixed findings: on the one hand, they confirmed that ACT achieved its effect specifically through an increased acceptance of feelings, while change in CBT was mediated by increased use of strategies to change feelings [300]. On the other hand, the studies indicated that treatment outcomes in ACT and CBT were equally associated with changes in negative and dysfunctional thinking (expected to be CBT-specific mediators) and cognitive defusion (the process of distancing oneself from the literal meaning of anxiety-related cognitions; expected to be an ACT specific

mediator) [298,299]. Taken together, these results suggest that CBT and ACT may have both similar and distinct cognitive mechanisms of change.

While CBT and ACT differ regarding the cognitive emotion regulation strategies they assumedly promote, they also share an important theorized mechanism of change in the treatment of anxiety: both treatments aim to reduce anxiety related avoidance behavior through exposure. Although the rationale behind exposure differs between CBT and ACT, it can be expected that reductions in behavioral avoidance contribute to anxiety symptom improvement in both interventions. To date only Forman et al. [300] have examined the role of (self-reported) behavioral avoidance in ACT and CBT in a sample of students with an anxiety or mood disorder. This study indeed found that reductions in self-reported behavioral avoidance was associated with improvement of treatment outcome, irrespective of treatment group.

Common factors

Contrary or supplementary to the idea that treatments exert their effects through (specific) theorized mechanisms is the idea that treatments work through so called common factors: mechanisms of change that most or all psychotherapies share. One of the most well-known and well-developed common factor theories is the contextual model [301,302]. This model states that psychotherapies achieve their effects through two common pathways: 1) the therapeutic relationship and 2) creating positive expectations/hope. First, an empathic, genuine and caring connection between the client and therapist is assumed to be beneficial in itself, especially for those patients that do not have such connections in their everyday lives. Second, the model states that psychotherapies elicit positive expectancies in the clients by providing them with an explanation about their psychological problems and how the treatment will help them in reducing these problems. Clients' expectancies regarding their ability to successfully complete the treatment –also called treatment self-efficacy- are also stimulated. The clients thus come to believe that completing the treatment will help them in coping with their problems and are provided with a sense of control over their own distress, as they contribute their therapeutic progress to their own efforts.

Looking at the empirical evidence for the common factors in the contextual model, a recent review article that included studies that accounted for temporality through the use of repeated assessments during treatment, concluded that improvement of the therapeutic alliance may indeed precede symptom reduction, which might point to a causal role of this common factor [303]. Considering client expectations, a large meta-

analysis including studies into different patient populations and treatment approaches found a small but statistically significant association between more optimistic early-therapy treatment expectancies and more favorable therapeutic outcomes [304]. Treatment expectancy is thus an empirically validated *correlate* of treatment outcome, but it is not clear if it should be considered a mechanism of action or merely a proxy of therapeutic improvement. Regarding evidence for a mechanistic function of this common factor, one elegant study found that changes in treatment expectancy during CBT for generalized anxiety disorder (GAD) mediated the relationship between baseline GAD severity and reliable change in this outcome at posttreatment [305].

The current study

The current study used data collected at multiple assessments during treatment to examine candidate mechanisms of change in a brief CBT and brief ACT intervention for older adults with anxiety symptomatology. The candidate mechanisms were divided into mechanisms related to the theoretical underpinnings of CBT and ACT and mechanisms assumed to drive change in psychotherapy in general (common factors). We hypothesized that increased use of cognitive reappraisal mediated treatment outcome in CBT. Furthermore, we expected that treatment outcome in ACT was mediated by an increase in the non-judgmental acceptance of feelings, decreased dwelling upon feelings (rumination) and decreased use of strategies aimed at avoidance of internal experiences (suppression, distraction). Lastly, we expected behavioral avoidance to be equally associated with anxiety symptom severity in the ACT and CBT group. Behavioral avoidance was thus not studied as a mediator in the strict sense of the term in the current study, as the study did not include a control condition in which behavioral avoidance was not targeted. Since behavioral avoidance was hypothesized to be a mechanism of change in both ACT and CBT, statistical analyses concerning the role of behavioral avoidance did not include treatment condition as an independent variable. Regarding common mechanisms, we followed the contextual model and hypothesized the therapeutic alliance and treatment expectancies (treatment outcome expectancy and treatment self-efficacy expectancy) to be associated with anxiety symptom severity across both treatments. Similarly to behavioral avoidance, these three factors could not be studied as mediators and were only expected to prospectively predict change in anxiety symptom severity during treatment across both conditions.

Methods

This study uses data collected in a cluster-randomized single blind controlled trial in the Netherlands. The trial evaluated the effectiveness of face-to-face CBT compared to a blended ACT intervention over a period of 12 months. The study was powered to detect a difference between the conditions on the primary outcome anxiety symptom severity as measured with the Generalized Anxiety Disorder-7 (GAD-7) [131]. Randomization took place at the level of the mental health counselors that provided the interventions (n=40). The mental health counselors were randomized to either provide only CBT (n=20) or only ACT (n=20) to study participants. Details about the study design and methods have been published [236]. The trial was registered in the Netherlands Trial Register (NL6131 (NTR6270)) and approved by the medical ethics committee of Leiden University Medical Center (LUMC; no. P16.248).

Participants and procedure

Participants were recruited from 38 general practices in the Netherlands between November 2017 and March 2019. Patients (aged 55 – 75) from the participating general practices were sent a letter containing information about the study and an invitation to participate. If people were interested in participation, they could register on a study website, after which they entered the screening procedure which consisted of an online questionnaire and a telephone interview. Inclusion criteria were: age between 55 and 75 years, presence of mild to moderate anxiety symptoms (GAD-7 score between 5 and 15 [131]), mastery of the Dutch language, internet access and the possibility to spend up to 30 min per day on the intervention. Exclusion criteria were: unstable severe medical condition(s); severe cognitive impairment; very high or low anxiety symptom severity (GAD-7 score < 5 / > 15 [131]); severe depressive symptoms (PHQ-9 [170] score $\geq$ 20); psychological or psychopharmacological treatment (stable benzodiazepine or SSRI use excepted) within the last 3 months; severe role impairment in at least 2 life areas (score of $\geq$8 on two or three items of the Sheehan Disability Scale (SDS) [171]; high suicide risk (M.I.N.I.-Plus [139]); substance use disorder (M.I.N.I.-Plus); lifetime diagnosis of bipolar disorder or schizophrenia (medical record and M.I.N.I.-Plus).

Eligible participants signed an online informed consent form and subsequently completed the baseline assessment, after which they were informed about their treatment allocation. Participants completed 4 main assessments: at baseline, posttreatment

(three months after baseline), 6 months after baseline and 12 months after baseline. In the current study, data from the 6- and 12-month follow up are not used in the analysis.

During treatment, participants were asked to complete a short questionnaire assessing anxiety symptom severity and potential mechanisms of change multiple times. Participants in the CBT group were sent the questionnaire after every session with their mental health counselor (4 times). Participants in the blended ACT group were asked to fill in the questionnaire at the beginning of each lesson of the online module (9 times). In order to compare the hypothesized temporal and mediational pathways in both groups, in the current study we did not include the data of all the 9 assessments in the ACT group, but only those completed after each face-to-face session. Furthermore, for both the CBT and ACT group, we excluded the data collected after the fourth (final) face to face session: only 62 participants completed this assessment and of these a majority of 37 did so one day prior or on the same day as the posttreatment assessment. Summarizing, in the current study we used data collected at baseline (T0), during treatment (T1-T3) and posttreatment (T4). To study the common factors we only used data collected during treatment (T1-T3), because participants could not rate the therapeutic alliance and expectations regarding treatment before having been introduced to their mental health counselor and the treatment approach in the first session. The data used for analyses in the current study were all collected using online self-report questionnaires.

Interventions

Therapists

Treatment was provided by mental health counselors working in general practices in the Netherlands. The counselors provide short term psychological treatment to patients with mild to moderately severe psychological complaints. The occupation is fulfilled by mental health professionals with varying educational backgrounds. Of the counselors participating in this study, most were graduates in psychology (n=13), social psychiatric nurses (n=14) or social workers (n=5). Years of experience with providing individual psychological treatment in the sample of mental health counselors ranged from 3 to 42, with a median of 16 years.

Cognitive Behavioral Therapy

Participants in the CBT condition attended 4 face-to-face sessions with the mental health counselor and completed homework exercises in between the sessions. The sessions took place in a timespan between 9 to 12 weeks. The sessions followed a

protocol (developed by authors N.G., M.W., V.K. and P.S.) that focused on identifying and challenging negative cognitions and reducing anxiety-related avoidance behavior. The protocol mainly consisted of worksheets and exercises related to specific types of anxiety (e.g., panic, worrying, social anxiety). Additionally, some worksheets/exercises focused on common side effects of anxiety (e.g., sleeping problems, physical tension). After the first session, which served as an intake, the counselor and client collaboratively set treatment goals. In the second and third session, homework was evaluated and prepared and key exercises/information were repeated. The last session focused on evaluating the progress of the client and formulating a relapse prevention plan.

Blended Acceptance and Commitment therapy

Participants in the Blended ACT condition were provided with the online ACT-module 'Living to the Full' [180,181] and 4 face-to-face sessions with the mental health counselor at their general practice. The online module contains 9 lessons that revolve around the 6 core processes of ACT: acceptance, cognitive defusion, contact with the present moment, self as context, personal values and committed action. Participants completed the module in 9 to 12 weeks, which required them to dedicate 15-30 minutes to the program on a daily basis. The 4 face-to-face sessions with the mental health counselor followed a protocol developed by the authors of Living to the Full and focused on increasing motivation, repeating key exercises, and discussing potential problems the client faced in working with the online module.

Measurements: outcome variable

Anxiety symptom severity

Anxiety symptom severity at baseline, during treatment and at posttreatment was measured with the Generalized Anxiety Disorder-2 [183]. The GAD-2 consists of the first two items of the Generalized Anxiety Disorder-7 ("Feeling nervous, anxious, or on edge" and "Not being able to stop or control worrying"). The GAD-2 is a reliable, valid, and sufficiently sensitive and specific instrument [183].

Measurements: candidate mechanisms of change

Cognitive emotion regulation strategies

The cognitive emotion regulation strategies reappraisal, acceptance, rumination, distraction, suppression and were each measured with one self-developed item. See

Table 1 for the items. Participants were asked to rate how often they used the strategy during the preceding week on a scale ranging from 0 (never) to 5 ((almost) always).

Behavioral avoidance

Behavioral avoidance was assessed with a self-developed item that is presented in Table 1. Participants indicated how often they avoided situations/activities due to their anxiety in the preceding week on a 0-5 scale.

Common factors

The Session Rating Scale (SRS) [219] was used to measure participant's rating of the therapeutic relationship. The SRS assesses 4 aspects of the working alliance during a therapeutic session, using one item per aspect: the relational bond, the degree to which desired goals and topics of the individual were discussed, the therapist's approach or working style, and an overall evaluation of the session. Items were answered on an 11-point, with '0' reflecting the most negative evaluation and '10' the most positive response. A sum score of the 4 items was calculated, with higher scores reflecting a better alliance according to the client. The SRS has high test-retest and internal consistency reliability, as well as acceptable validity [220, 221]. Treatment outcome expectancy was measured with one item, that was answered on a scale ranging from 1 (not at all) to 7 (very much). The item comes from the Treatment Credibility Questionnaire [207]. Treatment self-efficacy was assessed with one self-developed item, that used the same 7-point scale as the treatment outcome expectancy question. See Table 1 for the items.

Table 1. Overview of the examined candidate mechanisms of change and related hypotheses

Candidate mechanism	Measure	Hypothesis
Reappraisal	I tried to change how I think about the cause of my feelings	Mediates reduction of anxiety symptom severity in CBT
Acceptance	I tried to accept my feelings without judging them	Mediates reduction of anxiety symptom severity in ACT
Rumination	I could not stop thinking about my feelings	Mediates reduction of anxiety symptom severity in ACT
Distraction	I tried to distract myself from my feelings	Mediates reduction of anxiety symptom severity in ACT
Suppression	I tried to suppress my feelings	Mediates reduction of anxiety symptom severity in ACT
Behavioral avoidance	My anxiety made me avoid situations and/or activities	Prospectively predicts changes in anxiety symptom severity across CBT and ACT

Table 1. Continued

Candidate mechanism	Measure	Hypothesis
Therapeutic alliance	SRS	Prospectively predicts changes in anxiety symptom severity across CBT and ACT
Treatment outcome expectancy	How confident are you that the intervention will be helpful in reducing your anxiety complaints	Prospectively predicts changes in anxiety symptom severity across CBT and ACT
Treatment self-efficacy	How confident are you that you will do what is required to successfully follow and complete this intervention?"	Prospectively predicts changes in anxiety symptom severity across CBT and ACT

Statistical Analysis

Descriptive statistics were calculated in SPSS 25.0 [306]. All other statistical procedures were performed using Mplus v 6.11 [307]. To test whether the candidate mechanisms of change predicted anxiety symptoms over the course of treatment, Random Intercept Cross Lagged Panel Models (RI-CLPM) were used. A separate model was created for each candidate mechanism. RI-CLPM is an extension of the traditional Cross Lagged Panel Model that accounts for time-invariant, trait-like stability in the modelled variables by the inclusion of random intercepts [308]. Traditional CLPM assumes no stable intra-individual differences in the studied variables. This assumption is often untrue, as many psychological variables are trait-like to a certain extent. In RI-CLPM the variance of the observed score is divided into variance due to a between-person stable invariant trait (by adding a random intercept) and variance due to within-person fluctuation. By separating within-person variance from between-person variance, RI-CLPM allows for statements regarding within–person processes, which are more likely to reflect causal effects than between-person associations [308,309].

Prior to the RI-CLPM analyses, we calculated intra-class correlations (ICC) for all variables. ICC can be defined as the proportion of the variance explained by differences between subjects. Consequently, the RI-CLPM was created, by first regressing the observed scores for anxiety symptom severity and the candidate mechanism on their own latent factor (loading fixed to 1). Residual variances of the observed variables were set to zero, so that the latent factor structure captured the within- and between-person variance.

Next, two random intercepts (one for anxiety symptom severity, one for the candidate mechanism) were added to the model, with factor loadings constrained at one. These random intercepts reflect an individual's time-invariant deviation

from the grand means and therefore represent stable trait-like differences between participants with regard to the modelled variables. The correlation between the random intercepts represents the association between stable between-person differences in the candidate mechanism variable and stable between-person differences in anxiety symptom severity.

The latent factors were used to model autoregressive paths, cross-sectional paths and cross-lagged paths. Autoregressive paths are interpreted as the extent to which deviations from expected scores (based on the grand mean and random intercept) at one wave predicted deviations from expected scores for the same variable at the next assessment wave. The cross-sectional paths reflect the association between deviations from the expected scores on anxiety symptom severity and deviations from the expected scores on the candidate mechanism variable at each assessment wave. To test the hypotheses concerning the temporal precedence of the candidate mechanisms, the cross-lagged paths are of interest. These paths reflect the bidirectional relationship between anxiety symptom severity and the candidate mechanism. They indicate to what extent deviations from expected scores on the candidate mechanism variable are associated with deviations from expected anxiety symptom severity at the next measurement moment, and vice versa.

Lastly, for the subset of candidate mechanisms that we hypothesized to be treatment mediators (reappraisal, acceptance, rumination, distraction, suppression), we estimated the indirect effect of the intervention condition (CBT=0, ACT=1) on anxiety symptoms severity at assessment wave *t* via the hypothesized mediating variables at *t-1* using a bootstrapping procedure (n=5000). This resulted in 3 indirect effects in each mediation model (anxiety symptom severity at T4 via mediator at T3; anxiety symptom severity at T3 via mediator at T2; anxiety symptom severity T2 via mediator at T1). The mediation test required that we also added the direct effects of intervention condition on scores at all assessment waves after baseline to these models.

Analyses were performed on the basis of the intention-to-treat principle, including all randomized participants with baseline assessments. Full information maximum likelihood (FIML) estimations were used to handle missing data. We used 4 model fit indices to evaluate the fit of the models: the Root Mean Square Error of Approximation (RMSEA), the Standardized Root Mean squared residual (SRMR), the Comparative Fit Index (CFI) and the Tucker–Lewis index (TLI). For the RMSEA and SRMR values smaller than 0.08 and 0.05 were considered indicators of respectively acceptable and good model fit [42, 43]. For the CFI and TFI model fit was considered adequate for values higher than 0.90 and good for values higher than 0.95 [310,311].

Results

Assessments were completed by 314 participants at baseline (T0) (CBT n=164; ACT n=150), 238 after session 1 (T1) (CBT n=131; ACT n=107), 204 after session 2 (T2) (CBT n=102; ACT n=102), 153 after session 3 (T3), (CBT n=91; ACT n=62) and 222 at posttreatment (T4) (CBT n=121 ACT n=101). See Table 2 for the means and standard deviations of the observed scores for the total sample and two treatment conditions at each assessment wave. ICC's for the examined variables varied from 0.15 (reappraisal) to 0.41 (therapeutic alliance); all other ICC values fell in a range of 0.21 to 0.32. This indicates that for most variables between 21% and 32% of variance could be explained by differences *between* participants, while the rest (i.e., most) variance could be explained by fluctuations *within* participants.

Outcomes of the RI-CLPM are presented in Table 3 and in Figure 1-9 in Appendix 1. Model fit was acceptable or good for all RI-CLPM's (RMSEA: 0.00 – 0.04; SRMR: 0.02 – 0.05; CFI: 0.96 – 1.00; TLI: 0.90 – 1.00). At the between-person level, we found statistically significant associations between the random intercept of anxiety on the one hand and the random intercepts of the variables rumination, distraction, suppression and behavioral avoidance on the other hand. This indicates that participants who had higher anxiety symptom severity scores across the 5 measurement waves (i.e., higher trait-like anxiety) also reported higher levels of rumination, distraction, suppression and behavioral avoidance across the assessments. The random intercepts of the other predictor variables were not significantly associated with the random intercept of anxiety symptom severity.

Most auto-regressive paths were not statistically significant, indicating no consistent relation between within-person fluctuations at successive assessment waves. Regarding cross-sectional associations, reappraisal, rumination and behavioral avoidance showed a consistent positive relationship with anxiety symptom severity on a within-person level. This indicates that within-person change in anxiety symptom severity was related to within-person change in reappraisal, rumination and behavioral avoidance at the same assessment wave. Suppression scores were positively associated with anxiety symptom severity at the first three assessment waves, and distraction scores only at the third assessment. No other cross-sectional paths were statistically significant.

The mediation hypotheses regarding the variables of reappraisal, acceptance, rumination, distraction and suppression were not confirmed: none of the modelled indirect paths were statistically significant. Results did indicate some statistically significant direct

effects: during treatment participants in the ACT group showed larger deviations from expected scores (based on the grand mean and random intercept) on the hypothesized ACT-mechanisms than participants in the CBT group. At T2 scores from participants in the ACT group scored showed larger deviations (in the expected direction) than participants in the CBT group on acceptance (t=3.45, p=.00), rumination (t=-2.33, p=.02), suppression (t=-2.80, p=.01) and distraction (t=-3.37, p=.02). At T3 this was still the case for suppression (t=-4.55, p=.00) and distraction (t=-2.74, p=.01). Furthermore, at T2 the c-path (from the independent variable to the dependent variable) was significant, with participants in the ACT group showing larger downward deviations from their expected anxiety symptom severity than participants in the CBT group (t=-2.99, p=.00).

Lastly, contrary to our hypotheses, none of the within-person cross-lagged paths from behavioral avoidance, therapeutic alliance, treatment outcome expectancy and treatment self-efficacy to anxiety symptom severity were statistically significant. This means that none of these variables prospectively predicted anxiety symptom severity over the course of the treatments.

Table 2. Mean scores and standard deviations for both conditions on all measurement waves

Variable	Condition	T0 (baseline), M (SD)	T1, M (SD)	T2, M (SD)	T3, M (SD)	T4 (posttreatment), M (SD)
Anxiety	CBT	2.40 (1.62)	2.33 (1.61)	2.22 (1.42)	1.65 (1.51)	1.48 (1.23)
	ACT	2.24 (1.52)	2.40 (1.51)	1.67 (1.19)	1.29 (0.98)	1.48 (1.21)
	Total	2.33 (1.57)	2.37 (1.55)	1.94 (1.33)	1.50 (1.33)	1.48 (1.22)
Reappraisal	CBT	1.44 (1.03)	1.68 (1.04)	2.05 (1.08)	2.30 (1.40)	2.38 (1.40)
	ACT	1.58 (1.11)	1.97 (0.99)	2.28 (1.20)	2.76 (1.35)	2.57 (1.43)
	Total	1.51 (1.07)	1.84 (1.02)	2.16 (1.14)	2.48 (1.30)	2.47 (1.41)
Acceptance	CBT	2.29 (1.22)	2.28 (1.20)	2.24 (1.28)	2.69 (1.25)	2.65 (1.36)
	ACT	2.23 (1.32)	2.24 (1.01)	2.83 (1.31)	3.05 (1.49)	2.80 (1.51)
	Total	2.26 (1.27)	2.26 (1.09)	2.53 (1.32)	2.84 (1.36)	2.72 (1.43)
Rumination	CBT	2.27 (1.26)	2.70 (1.36)	2.07 (1.32)	1.65 (1.16)	1.67 (1.31)
	ACT	2.06 (1.29)	2.61 (1.27)	1.69 (1.18)	1.36 (1.26)	1.42 (1.21)
	Total	2.12 (1.27)	2.65 (1.31)	1.88 (1.26)	1.53 (1.20)	1.56 (1.27)
Distraction	CBT	2.41 (1.11)	2.78 (1.17)	2.40 (1.19)	2.33 (1.24)	1.98 (1.30)
	ACT	2.40 (1.24)	2.70 (1.04)	1.99 (1.22)	1.53 (1.18)	1.76 (1.27)
	Total	2.41 (1.17)	2.74 (1.10)	2.20 (1.22)	2.01 (1.27)	1.88 (1.29)
Suppression	CBT	2.38 (1.16)	2.58 (1.25)	2.17 (1.28)	2.08 (1.17)	1.77 (1.28)
	ACT	2.29 (1.19)	2.60 (1.08)	1.66 (1.12)	1.11 (1.12)	1.30 (1.15)
	Total	2.33 (1.18)	2.59 (1.15)	1.92 (1.23)	1.69 (1.24)	1.56 (1.24)
Behavioral Avoidance	CBT	1.83 (1.15)	2.19 (1.30)	1.69 (1.29)	1.50 (1.11)	1.31 (1.22)
	ACT	1.77 (1.17)	2.21 (1.12)	1.36 (1.06)	0.79 (0.87)	1.02 (1.11)
	Total	1.80 (1.16)	2.20 (1.20)	1.52 (1.19)	1.21 (1.07)	1.18 (1.18)
Treatment expectancy	CBT	-	4.59 (1.19)	3.68 (1.00)	3.77 (1.19)	-
	ACT	-	4.69 (1.02)	3.86 (1.10)	4.3 (1.11)	-
	Total	-	4.64 (1.09)	3.77 (1.05)	3.99 (1.19)	-
Treatment self-efficacy	CBT	-	5.45 (1.36)	4.47 (0.98)	4.41 (1.28)	-
	ACT	-	5.41 (1.09)	4.46 (1.24)	4.55 (1.24)	-
	Total	-	5.43 (1.22)	4.46 (1.03)	4.46 (1.26)	-
Therapeutic Alliance	CBT	-	32.41 (6.06)	32.81 (6.89)	35.02 (5.77)	-
	ACT	-	31.81 (6.28)	32.54 (6.55)	35.15 (4.43)	-
	Total	-	32.08 (6.18)	32.68 (6.71)	35.07 (5.25)	-

Table 3. Outcomes of the random intercept cross-lagged panel models examining the temporal and mediational relationships of the candidate mechanisms of change and anxiety symptom severity

	Reappraisal	Acceptance	Rumination	Distraction	Suppression	Behavioral avoidance	Therapeutic alliance	Treatment expectancy	Treatment self-efficacy
Model fit									
RMSEA	0.04	0.03	0.00	0.02	0.03	0.02	0.00	0.03	0.01
CFI	0.96	0.98	1.00	1.00	0.99	1.00	1.00	1.00	1.00
TLI	0.90	0.96	1.00	0.99	0.97	1.00	1.01	0.97	1.00
SRMR	0.05	0.04	0.03	0.05	0.05	0.03	0.02	0.03	0.02
Random intercepts									
Variance RI anx	0.59 (0.12)***	0.56 (0.11)***	0.56 (0.11)***	0.58 (0.11) ***	0.59 (0.11)***	0.62 (0.11)***	0.45 (0.29)	0.46 (0.30)	0.51 (0.28)
Variance RI m	0.13 (0.06)*	0.29 (0.08)***	0.42 (0.08)***	0.36 (0.07)***	0.41 (0.06)***	0.42 (0.08)***	12.87 (3.8)**	0.43 (0.15)**	0.57 (0.21)**
RI anx with RI m	0.08 (0.06)	-0.07 (0.06)	0.35 (0.08)***	0.36 (0.07)***	0.37 (0.07)***	0.36 (0.07)***	0.53 (0.75)	-0.18 (0.13)	-0.12 (0.17)
Autoregressive paths									
anx1 on anx0	0.28 (0.08)***	0.26 (0.08)***	0.24 (0.08)**	0.25 (0.08)***	0.26 (0.08)**	0.28 (0.08)***	-	-	-
anx2 on anx1	0.14 (0.07)	0.14 (0.07)	0.12 (0.08)	0.10 (0.08)	0.11 (0.08)	0.12 (0.07)	0.19 (0.13)	0.16 (0.14)	0.20 (0.30)
anx3 on anx2	0.19 (0.12)	0.17 (0.14)	0.22 (0.13)	0.22 (0.13)	0.16 (0.13)	0.06 (0.13)	0.22 (0.19)	0.19 (0.21)	0.24 (0.19)
anx4 on anx3	0.15 (0.11)	0.13(0.11)	0.27 (0.10)**	0.19 (0.10)	0.17 (0.11)	0.14 (0.12)	-	-	-
m1 on m0	0.04 (0.08)	0.13 (0.08)	0.04 (0.08)	0.10 (0.08)	0.11 (0.08)	0.16 (0.09)	-	-	-
m2 on m1	0.15 (0.11)	0.04 (0.08)	-0.04 (0.10)	-0.03 (0.10)	-0.09 (0.11)	0.10 (0.09)	-0.16 (0.27)	-0.09 (0.30)	0.15 (0.16)
m3 on m2	0.23 (0.10)*	-0.02 (0.12)	0.27 (0.10)**	0.05 (0.10)	0.29 (0.10)**	0.35 (0.10)***	0.47 (0.13)***	0.60 (0.17)***	0.23 (0.11)
m4 on m3	0.49 (0.08)***	-0.09 (0.12)	0.23 (0.10)*	0.17 (0.11)	0.41 (0.10)***	0.25 (0.14)	-	-	-
Cross-sectional paths									
anx0 with m0	0.21 (0.10)*	-0.06 (0.11)	0.31 (0.11)*	0.12 (0.10)	0.21 (0.10)*	0.21 (0.10)*	-	-	-
anx1 with m1	0.34 (0.11)**	-0.05 (0.10)	0.34 (0.11)**	0.04 (0.10)	0.20 (0.10)*	0.34 (0.11)**	0.25 (0.83)	0.16 (0.16)	0.25 (0.18)
anx2 with m2	0.38 (0.09)***	-0.10 (0.10)	0.48 (0.10)***	0.10 (0.10)	0.20 (0.10)*	0.38 (0.09)***	-0.45 (0.76)	0.02 (0.15)	-0.00 (0.18)
anx3 with m3	0.42 (0.09)***	-0.10 (0.13)	0.42 (0.12)**	0.44 (0.12)***	0.16 (0.11)	0.42 (0.09)***	-0.49 (0.56)	-0.09 (0.10)	-0.13 (0.10)
anx4 with m4	0.29 (0.08)***	0.03 (0.09)	0.21 (0.09)*	0.09 (0.09)	0.14 (0.09)	0.29 (0.08)***	-	-	-

Table 3. Continued

	Reappraisal	Acceptance	Rumination	Distraction	Suppression	Behavioral avoidance	Therapeutic alliance	Treatment expectancy	Treatment self-efficacy
Cross-lagged paths									
anx1 on m0	-0.14 (0.10)	-0.03 (0.09)	0.06 (0.09)	0.07 (0.10)	-0.05 (0.10)	-0.09 (0.11)	-	-	-
anx2 on m1	-0.07 (0.10)	-0.15 (0.10)	0.01 (0.09)	-0.09 (0.10)	-0.01 (0.10)	0.04 (0.10)	0.00 (0.03)	-0.05 (0.19)	0.05 (0.21)
anx3 on m2	-0.19 (0.10)	-0.06 (0.09)	0.04 (0.12)	-0.08 (0.12)	-0.17 (0.12)	0.25 (0.13)	0.00 (0.03)	-0.03 (0.17)	-0.30 (0.24)
anx4 on m3	-0.08 (0.09)	-0.11 (0.08)	0.11 (0.11)	-0.18 (0.10)	-0.18 (0.12)	0.01 (0.15)	-	-	-
m1 on anx0	-0.03 (0.06)	-0.02 (0.06)	0.17 (0.07) ***	0.12 (0.06)*	0.14 (0.06)*	0.06 (0.06)	-	-	-
m2 on anx1	-0.08 (0.07)	-0.00 (0.07)	0.19 (0.07)**	0.04 (0.07)	0.05 (0.07)	0.14 (0.06)	-0.25 (0.44)	0.18 (0.10)	0.10 (0.12)
m3 on anx2	-0.23 (0.12)	-0.17 (0.12)	0.10 (0.13)	0.05 (0.10)	0.01 (0.11)	0.03 (0.09)	-0.87 (0.63)	-0.03 (0.11)	0.06 (0.11)
m4 on anx3	-0.11 (0.12)	-0.10 (0.11)	0.24 (0.11)*	-0.13 (0.10)	-0.05 (0.09)	0.16 (0.11)	-	-	-
Mediational paths									
cond to anx2 via m1	-0.02 (0.03)	0.01 (0.03)	0.00 (0.02)	0.00 (0.02)	0.00 (0.02)	-	-	-	
cond to anx3 via m2	-0.05 (0.04)	-0.04 (0.06)	-0.01 (0.05)	0.03 (0.05)	0.08 (0.07)	-	-	-	-
cond to anx4 via m3	-0.02 (0.03)	-0.01 (0.03)	0.03 (0.05)	0.11 (0.10)	0.15 (0.14)	-	-	-	-
cond to m2 via anx1	-0.01 (0.02)	0.00 (0.02)	0.03 (0.04)	0.01 (0.02)	0.01 (0.02)	-	-	-	-
cond to m3 via anx2	0.11 (0.08)	0.09 (0.07)	-0.05 (0.07)	-0.02 (0.08)	-0.01 (0.07)	-	-	-	-

Discussion

This study examined potential mechanisms of change in a brief CBT and a brief ACT intervention for adults aged 55-75 years with mild to moderately severe anxiety symptoms. These interventions were previously found to result in comparable reductions of anxiety symptom severity [8]. Data were collected at multiple assessments during treatment, which enabled the examination of the relationships between the candidate mechanisms and the outcome variable anxiety symptom severity on the within-person level.

Contrary to our hypotheses based on the theories of change in CBT and ACT, we did not find evidence that the treatments exert their effects on anxiety symptom severity through different cognitive emotion regulation strategies. The relationship between treatment condition and anxiety symptom severity during treatment was not mediated by previous levels of reappraisal, acceptance, rumination, distraction or suppression. Moreover, none of these variables prospectively predicted anxiety symptom severity during treatment across the two treatment conditions. We did find that after the second session participants in the ACT group on average scored higher (on the within-person level) on acceptance and lower on rumination, distraction and suppression than participants in the CBT group. For distraction and suppression this difference was also significant after the third session. At posttreatment however, the conditions did not differ on these variables. These findings may indicate that the ACT has a more direct impact on these cognitive processes than CBT, affecting them earlier during treatment. Lastly, behavioral avoidance did also not prospectively predict anxiety symptom severity over the course of treatment. The current results do therefore not indicate that the examined cognitive emotion regulation strategies and behavioral avoidance were mechanisms of action in the CBT and ACT intervention. This contradicts earlier studies testing the theories of change in CBT and ACT for anxiety. Those studies concluded that cognitive strategies aimed at changing thoughts mediate outcomes in CBT, that acceptance is an ACT-specific mediator [299,300] and that reductions in negative thinking, cognitive fusion and behavioral avoidance are equally associated with treatment outcome in CBT and ACT [298-300]. Importantly, these studies focused on (slightly) different variables and employed different statistical analyses than the current study, which hinders a straightforward comparison with the current findings.

Our hypotheses regarding the common factors were also not confirmed: ratings of the therapeutic alliance and treatment expectancies did not prospectively predict anxiety symptom severity over the course of the CBT and ACT intervention. The null-

finding regarding therapeutic alliance runs counter to earlier studies that found that within-person changes in the patient-rated alliance precede symptom reduction during treatment [303]. The most evident difference between the majority of those studies and the current one is the measurement of the therapeutic alliance. In the current study the Session Rating Scale (SRS) was used, while most other studies employed the Working Alliance Inventory (WAI) [303]. Psychometric evaluations of the SRS found that its concurrent validity with the WAI is moderate (r = .57–.65), which is lower than expected as both instruments aim to measure the working alliance in therapy [221,312]. The two scales might thus measure slightly different concepts, which may explain the discrepancy between the current findings and those from studies that used the WAI. A comprehensive discussion of the null-findings regarding treatment expectancy is precluded because rigorous studies into treatment expectancy as a mechanism of change in psychotherapy are largely lacking at the moment. Most previous studies have operationalized expectancy as a static construct and only assessed it once at the beginning of treatment [304]. To elucidate the role of treatment expectancy in psychological treatment, more studies are needed that –similar to the current study- consider expectancy as dynamic and malleable and measure it multiple times during treatment.

As is clear, we cannot easily compare the current findings to results from earlier studies, due to differences in research design, measurement and statistical procedure. Two features that have already been touched upon that most clearly distinguish the current study from many previous studies are its longitudinal design and the disentanglement of within- and between-person variance. These two features are important strengths of the current study, because they increase its weight in terms of potential causal inferences. Using data from multiple assessments during treatment enabled the establishment of a timeline, which is a requisite for inferring mediation or a causal relation. Furthermore, separating between-person and within-person variance is crucial in ascertaining whether associations reflect relatively stable differences *between* people (that can often be explained away by time-invariant third variables) or if they point to processes that occur over time *within* people and thus to possibly causal processes that might be useful targets for treatment augmentation strategies [313]. Unfortunately, the majority of studies into the mechanisms of psychotherapeutic change (of CBT and ACT) did not establish temporal precedence and/or did not separate within- and between-person variance in their analysis [64,294,295,297-300,303,304]. These studies have therefore mostly established cross-sectional

associations between candidate mechanisms and outcome variables on the between-person level. We replicated these findings and found that on the between-person level rumination, distraction, suppression and behavioral avoidance are indeed associated with anxiety symptom severity. However, such between-person associations do not allow for conclusions about a mechanistic role of the studied variables. To improve the examination and understanding of psychotherapeutic change, we prompt future research to use longitudinal designs and statistical procedures that separate between- and within-person variability. Only with such studies can we begin to elucidate whether hypothesized mechanisms of change indeed seem to play a causal role, or if they are merely correlates of treatment outcome [303].

However, even with optimal research designs it remains highly challenging to understand psychotherapeutic change. Psychotherapy is a complex and multi-level process that is likely to work through a complex chain of changes: different mechanisms of change (at either the physiological, cognitive, behavioral or affective level or on multiple levels) occur at different time points and rates during treatment and certain changes might occur suddenly instead of gradually [314]. Furthermore, it may be the case that treatment components and the mechanisms of action associated with them work differently at different points of treatment and that their workings differ between subgroups of people receiving treatment. Therefore, we may never be able to explain psychotherapeutic change using the relatively simplistic (causal) models of change and associated research designs that psychological science has relied upon so far.

This study has some limitations that are important to discuss. First, although the use of longitudinal data is an important advantage of this study, it is plausible that the data (based on 5 measurement moments) was not sufficiently fine-grained to accurately model mechanisms of therapeutic change. The current null-findings might have resulted from the measurement waves being too far apart to adequately capture changes in the measured constructs during treatment. Future studies should therefore focus on establishing a more fine-grained analysis of the shape of therapeutic change. Experience Sampling Methods (ESM) are promising in this regard [315]. Second, like most studies in this field of research, all data in the current study came from self-report instruments. Self-report relies on people's ability to identify and remember their own mental processes – an ability that might be far from perfect [316]. A combination and integration of data collected with different types of measurement instruments (e.g., clinician rating scales, physiological measures, behavioral tasks, neuroimaging) is preferable over relying upon one assessment method [317]. A third shortcoming is

that we used self-developed one item assessments for most candidate mechanisms. We opted for this type of measurement to avoid placing too large a burden on the participants, because too many demands for data can lead to measurement artefacts as a result of study drop-out or unreliable completion of the measurements. Although we used straight-forward items mostly based on questions from validated instruments, we cannot be certain that the self-developed items reliably measure the intended constructs and are sufficiently sensitive to change. Fifth, the generalizability of our findings might be limited because we tested our hypotheses in a sample of adults aged 55-75 years. The findings may not generalize to younger adult samples, although there is currently no strong theoretically or empirically valid reason to assume that CBT and ACT might work through different processes in older patient populations.

Summarizing, the current study examined multiple putative mechanisms of change of a CBT intervention and an ACT intervention for older adults with anxiety symptoms. The cognitive emotion regulation strategies reappraisal, acceptance, rumination, distraction and suppression were expected to mediate treatment outcome, but hypotheses were not confirmed. Furthermore, contrary to our hypotheses, behavioral avoidance, therapeutic alliance and treatment expectancies did not prospectively predict anxiety symptom severity during treatment. The current study positively distinguishes itself from many previous studies in the field, because it used data collected at multiple time points during treatment and a statistical approach that examined the hypothesized relationships on the within-person level. Future studies are encouraged to use longitudinal designs that allow for a more fine-grained analyses of therapeutic change and to analyze the associations between potential mechanisms of change and treatment outcome on the within-person level.

Appendix 1

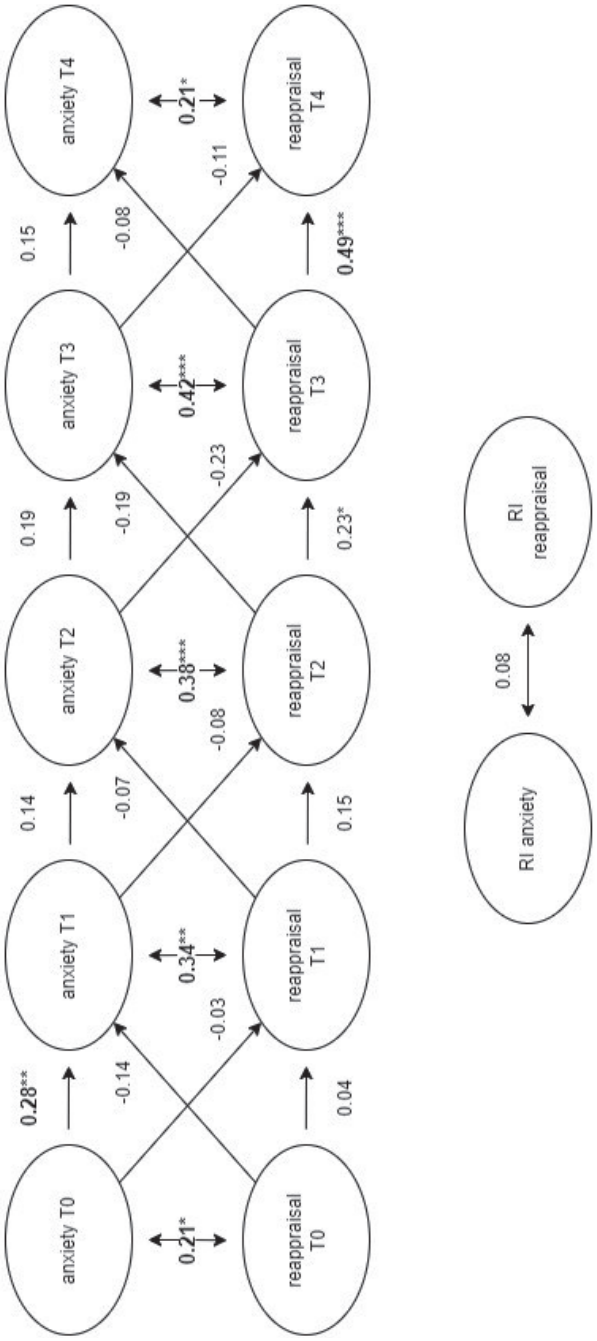

Figure A1. RI-CLPM reappraisal

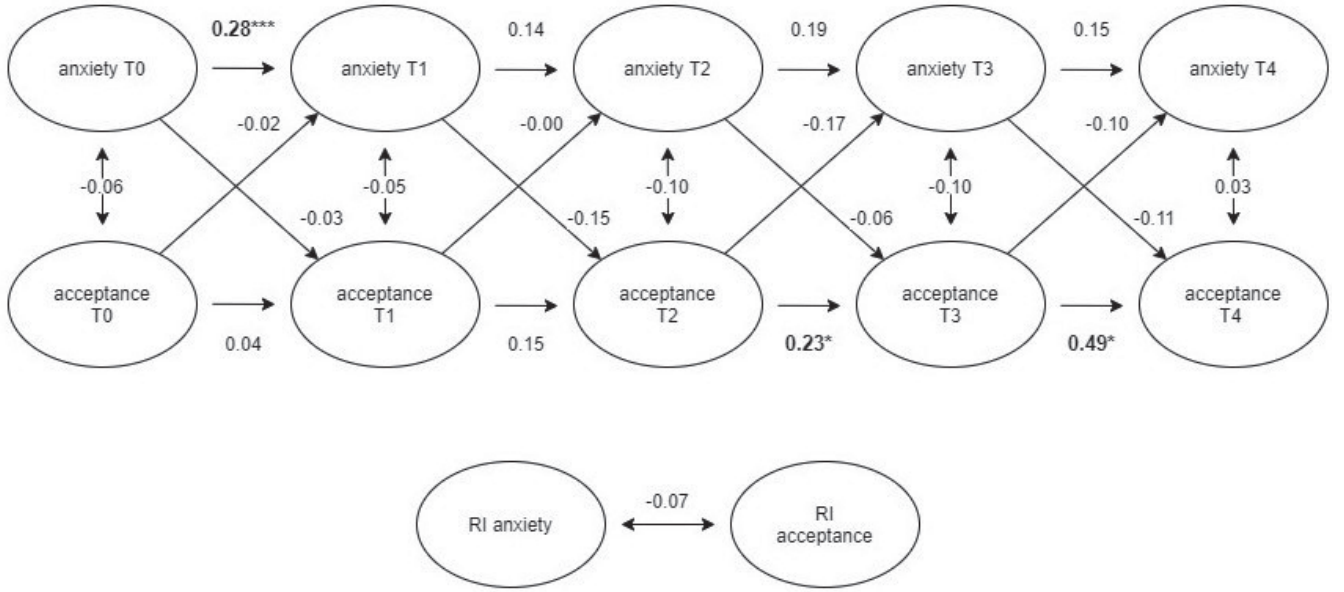

Figure A2. RI-CLPM acceptance

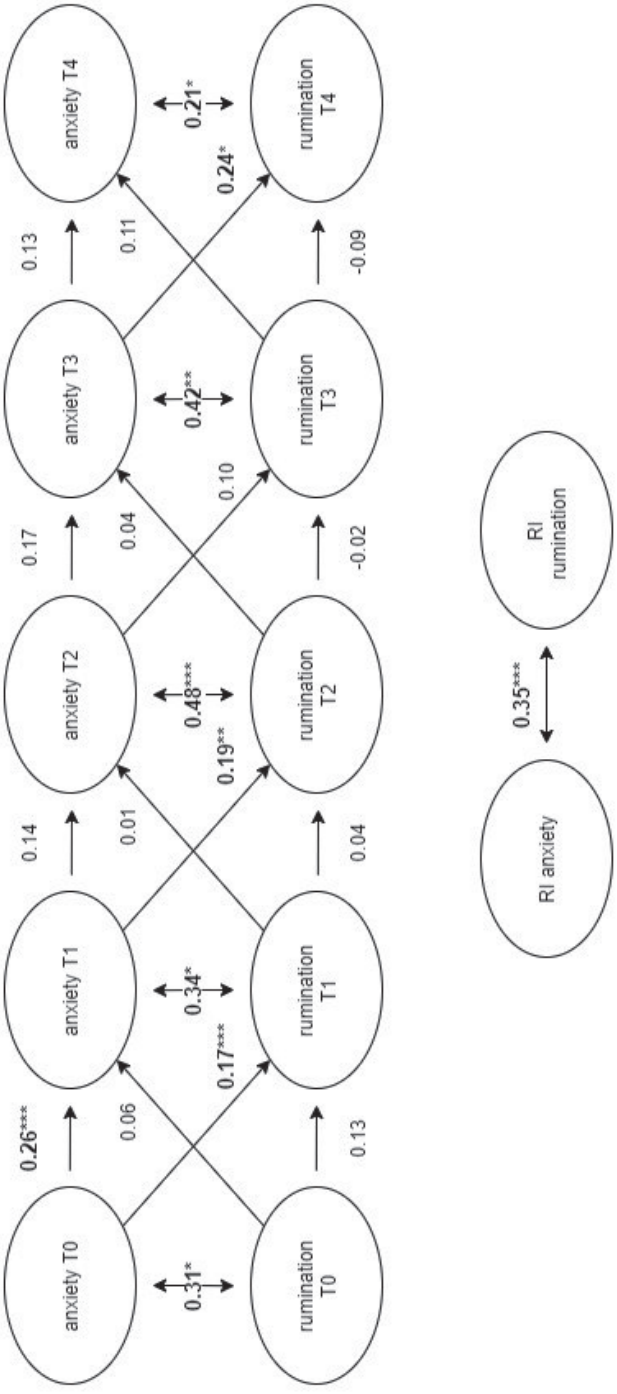

Figure A3. RI-CLPM rumination

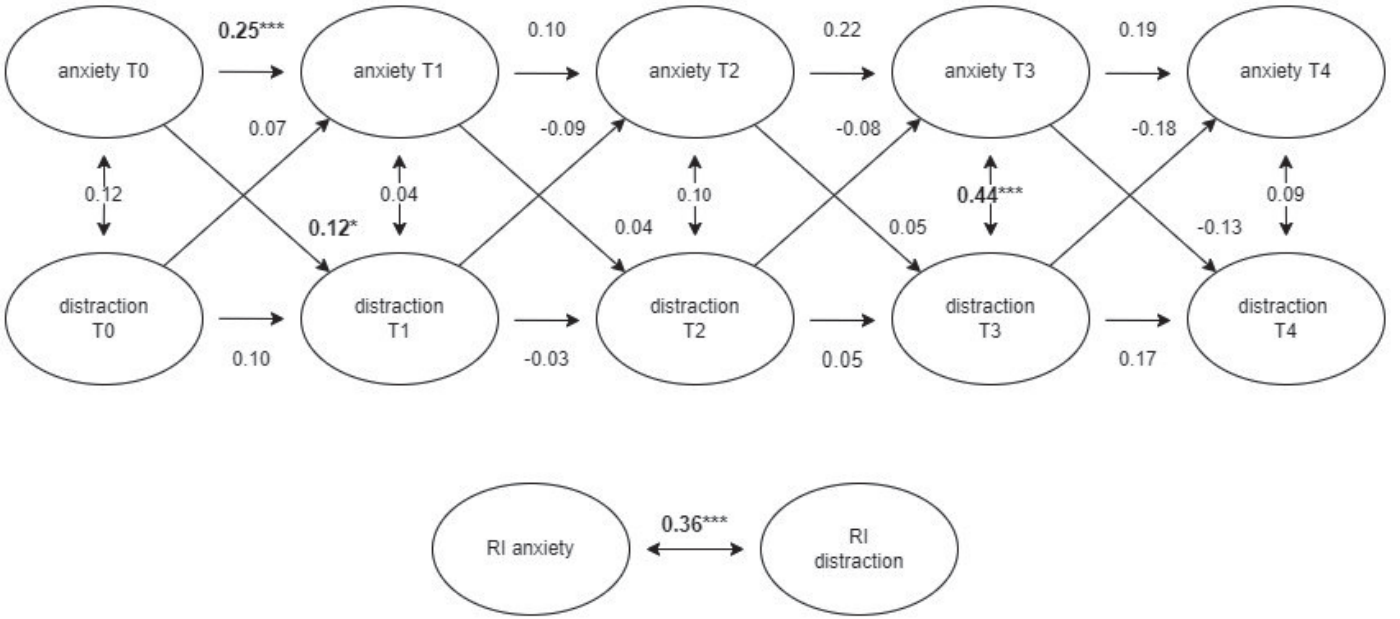

Figure A4. RI-CLPM distraction

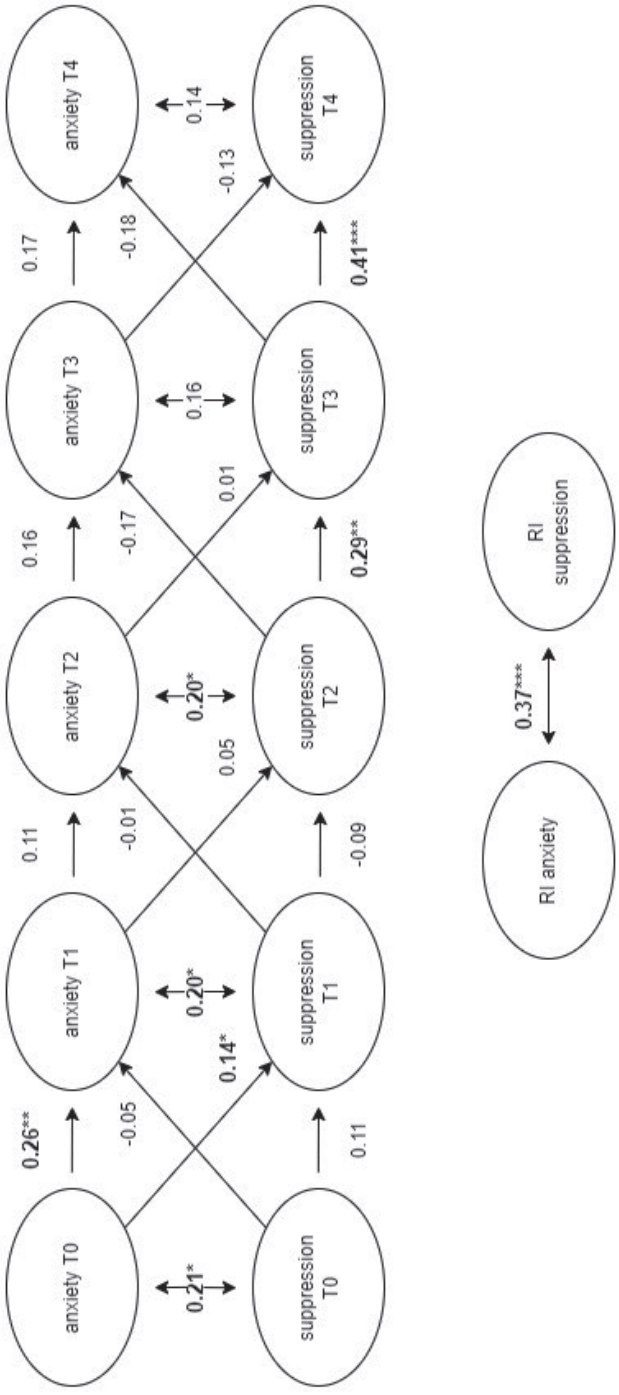

Figure A5. RI-CLPM suppression

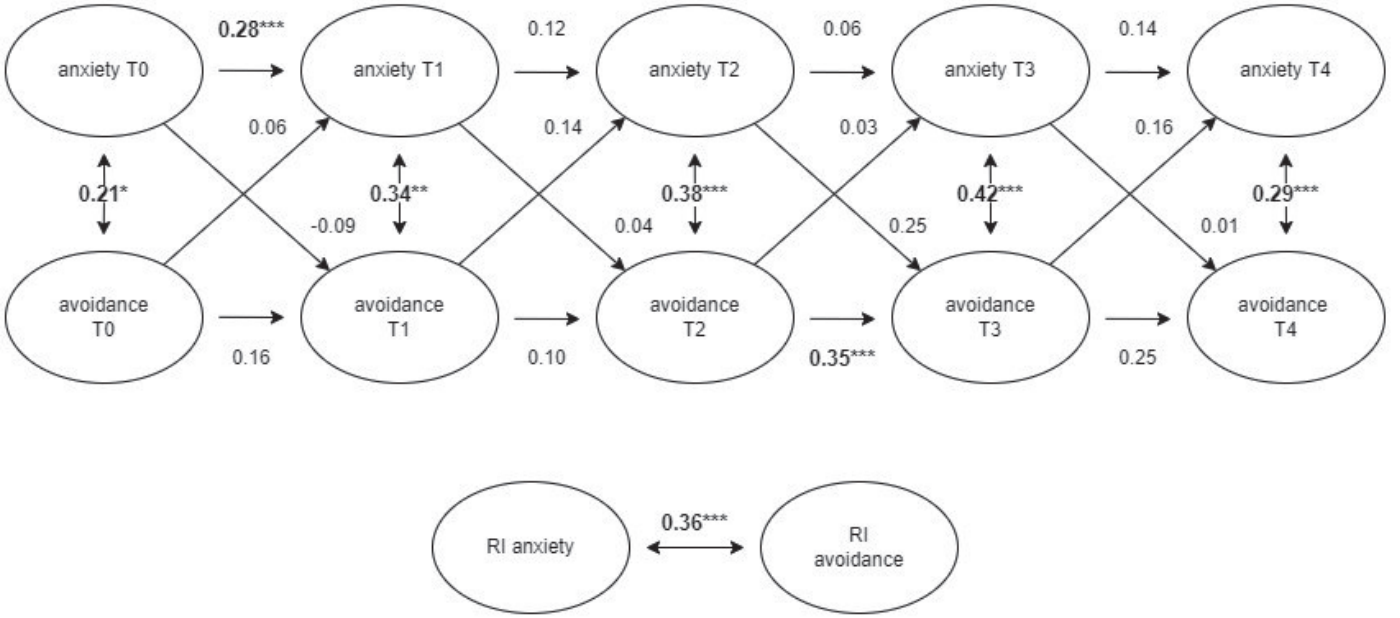

Figure A6. RI-CLPM avoidance

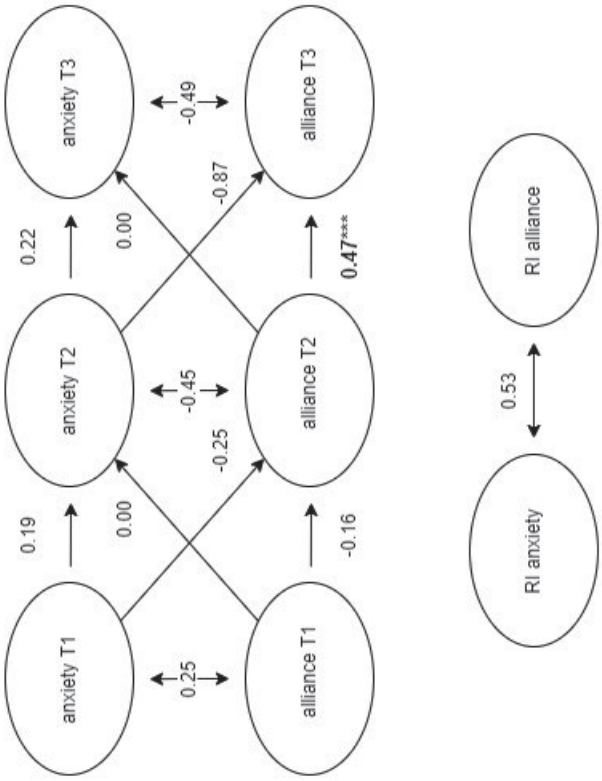

Figure A7. RI-CLPM therapeutic alliance

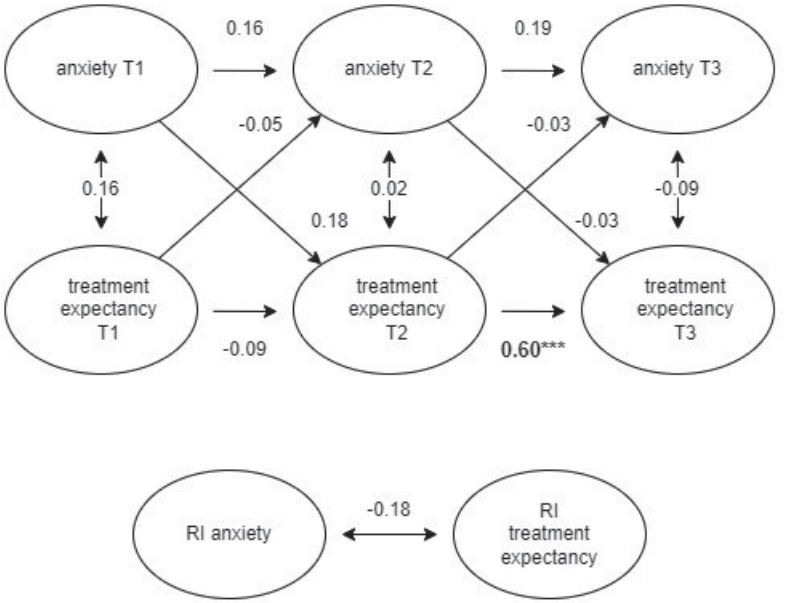

Figure A8. RI-CLPM treatment expectancy.

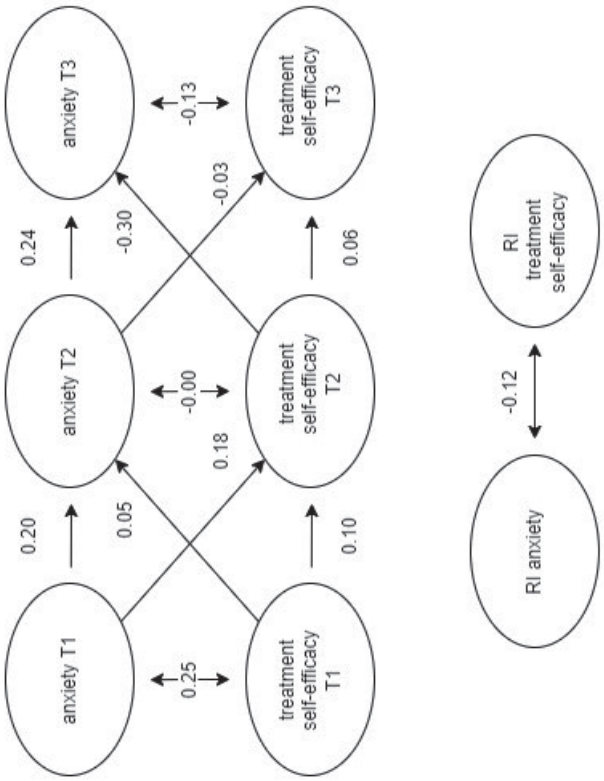

Figure A9. RI-CLPM treatment self-efficacy