Mechanism Engagement as a Potential Evidence-Based Approach to Personalized Treatment Termination

Shannon Sauer-Zavala[1]

Matthew W. Southward

Doug R. Terrill

Stephen A. Semcho

Nicole E. Stumpp

University of Kentucky

[1]Corresponding Author. Department of Psychology, University of Kentucky, Lexington, KY 40506, ssz@uky.edu, 859-218-4082

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Abstract

Objective: This study explores whether early change on a putative mechanism maintaining symptoms can serve as a proximal indicator of response to prompt discontinuation. **Method:** Patients (N = 70; M_{age} = 33.74, 67% female, 74% white) with heterogeneous anxiety and depressive disorders completed a sequential multiple assignment randomized trial (SMART). Patients received 6 sessions of skill modules from the Unified Protocol and then underwent a second-stage randomization to either receive the remaining 6 sessions (Full duration) or discontinue treatment (Brief duration). All participants completed weekly self-report measures of anxiety and depressive symptoms and distress aversion for the full 12-week treatment window. We used structural equation modeling to test (1) if distress aversion demonstrated significant variability during the first-stage randomization and (2) if distress aversion during the first-stage randomization predicted second-stage changes in anxiety and depression. **Results**: Participants demonstrated significant variability in first-stage distress aversion. Latent distress aversion slopes significantly predicted latent second-stage anxiety slopes, whereas latent distress aversion intercepts significantly predicted latent second-stage depression slopes. **Conclusions:** These results suggest that early mechanism engagement may have potential as a trigger to prompt personalized termination. Shorter courses of care may reduce patient costs and increase the mental health service system's capacity.

Keywords: personalization; transdiagnostic; SMART; Unified Protocol, termination

Core Mechanism Engagement as an Evidence-Based Approach to Treatment Termination

Cognitive behavioral therapy (CBT) is an efficacious treatment for anxiety and depressive disorders (Hofmann & Smits, 2008; van Straten et al., 2010). However, despite the existence of proven CBT protocols, prevalence rates for these conditions remain persistently high (Bandelow & Michaelis, 2015), perhaps in part because over 20 million US adults with a mental illness do not receive treatment each year (Center for Behavioral Health Statistics and Quality [CBHSQ], 2016).

Long waitlists at clinics that provide evidence-based care represent an important barrier to easing the burden of mental illness (Beck et al., 2015; Southward et al., 2020). Although an insufficient number of providers relative to growing mental health needs contributes to difficulty accessing care (Markit, 2018), wait times may also be exacerbated by patients remaining in treatment longer than necessary. Indeed, patients demonstrate substantial variability in the number of sessions required to produce clinically significant improvements. In a systematic review of the dose-response effect, the optimal dose of psychotherapy needed to produce symptom improvement ranged from 4 to 26 sessions (Robinson et al., 2020a). These results underscore the need to personalize treatment discontinuation decisions so that patients receive a sufficient dose of care, but not more than is necessary.

When to discontinue care is a critical, yet rarely addressed, question in psychosocial treatment research. Protocol-based approaches, often used in clinical trials, encourage the provision of all components included in a treatment manual, regardless of patient improvement. Routine outcome monitoring allows clinicians and patients to end treatment when symptom-based goals have been reached (Boswell et al., 2015), though symptom change itself may be a relatively distal indicator of success. Recent advances in treatment development research

underscore the need to specify and target mechanistic processes that maintain symptoms rather than the symptoms themselves (Sauer-Zavala et al., 2017; Hayes & Hofmann, 2019). Kazdin, (2007) defines a "mechanism" as a process with 1) a strong association with the outcome of interest, 2) temporal precedence wherein change in the mechanism of action precedes change in the outcome, and 3) a dose-response relationship where greater change in a mechanism of action leads to better outcomes. Thus, if a treatment targets core psychopathological processes, it is possible that symptom change is actually a delayed indicator of a patient's response or the robustness of their improvements. For example, Strunk and colleagues (2014) describe degree of improvement on putative mechanisms of cognitive therapy as a potential early indicator of relapse. It is also possible that a more proximal indicator of eventual response during treatment may be change in the targeted core process, which could be used in termination decisions.

Investigations into factors associated with treatment response have found that the rate of symptom reduction is associated with ultimate treatment response. For example, rapid symptom reduction early in treatment has predicted more positive treatment outcomes (Lewis et al., 2012). Sudden gains, clinically relevant decreases in symptoms between consecutive treatment sessions, have consistently predicted better treatment outcomes regardless of when they occur in treatment (Tang & DeRubeis, 1999; Shalom & Aderka, 2020). Early investigations into optimized treatment length examined the relationship between the quantity or concentration (i.e., "dose") of treatment and the subsequent probability of clinical improvement (Howard et al., 1986). In these studies, clinical improvement is typically defined as a statistically reliable reduction in psychiatric symptoms using Jacobson and Truax's (1991) definition of reliable and clinically significant improvement (RCSI). Dose-response studies most often aim to determine the number of treatment sessions needed for 50% of patients to reach RCSI (Robinson et al., 2020a). This

number is often determined after data collection is complete, as dose-response study designs have not historically discontinued treatment when patients reach this threshold of improvement (Robinson et al., 2020a; Robinson et al., 2020b). After RCSI is reached, researchers have generally found a negatively accelerated relationship between dose and clinical improvement. That is, patients often continue to improve after reaching RSCI but do so at an increasingly slower rate (Robinson et al., 2020a; Stulz et al., 2013). Because sessions continue after RCSI is achieved, typical dose-response designs do not allow researchers to examine whether patients would have continued to improve if treatment was withdrawn.

In contrast to the dose-response model, the "Good Enough Level" model (GEL; Barkham et al., 2006), proposes that patients discontinue treatment after reaching an adequate, or good enough, level of improvement (Owen et al., 2016). This model suggests that rates of symptom improvement vary by dose of treatment, such that patients who have fewer sessions demonstrate faster symptom reduction and those with greater sessions demonstrate slower symptom reduction (Barkham et al., 2006). Thus, treatment dose may be considered an indicator of treatment response, rather than a predictor of treatment response (Lee et al., 2021). As patients discontinue treatment in the GEL model after reaching their individual good enough level, researchers conducting treatment studies in naturalistic settings are unable to determine if patients continue to improve after they have reached this level.

Adaptive Treatment Trial Designs

Adaptive treatment designs offer useful features to test hypotheses about treatment decision-making, including when to terminate care (Almirall et al., 2012). For example, an adaptive treatment design might include an initial randomization to condition (e.g., assignment to an experimental intervention vs. treatment as usual) followed by a second randomization after a

specified number of weeks. In one example of this approach, Chronis-Tuscano and colleagues (2016) first randomized mothers to receive either stimulant medication or behavioral training to address maternal and child ADHD symptoms. Mothers were then re-randomized eight weeks later to either continue with their initial treatment or receive the alternative intervention as a supplement. Because patients are randomized to receive treatment adaptations, researchers can draw stronger conclusions about optimal treatment planning decisions (e.g., is additional time needed for the initial intervention to work or is it better to add an adjunctive treatment?).

In addition to randomizations based solely on time (e.g., re-randomizing all patients at session eight), it is also possible to use *tailoring variables* to determine whether to adapt treatment. For instance, patients whose anxiety symptoms do not reach a pre-determined threshold by a particular point in treatment may be re-randomized to continue with current care or receive more intensive treatment (e.g., Gunlicks-Stoessel et al., 2016). Using symptoms as a tailoring variable, a secondary randomization in which patients are assigned to either continue or discontinue treatment would allow researchers to test whether and how symptom improvement can be used to prompt termination (e.g., below a certain cutoff, degree of absolute change). Randomizing patients to receive the same treatment for different durations allows for comparisons between individuals who received the full dose and those that ended early. If differences in outcomes exist as a function of treatment length, such a design would allow researchers to determine the degree of symptom change that predicts comparable symptom improvement at a subsequent follow-up assessment for those ending treatment early relative to those that received the full dose of care.

Moreover, researchers could also consider putative mechanisms of action as the trigger for a treatment decision (i.e., re-randomization). Since improvements in mechanisms may

precede symptom changes, randomizing patients to discontinue care (or continue with the full treatment protocol) would allow researchers to test whether a hypothesized mechanism is indeed important for subsequent symptom change in a robust experimental design. In other words, if a psychological process is truly a mechanism maintaining symptoms, early mechanism engagement (indicated by scores above or below a particular threshold or by degree of change in a specified process) should predict continued symptom improvement even after treatment is withdrawn. This design would allow researchers to determine whether it is possible to use mechanistic change to make treatment discontinuation decisions.

Present Study

The present study is a secondary data analysis of Sauer-Zavala et al. (2022) in which we conducted an adaptive treatment trial, specifically a sequential multiple assignment randomized trial (SMART; Southward & Sauer-Zavala, 2020), to garner initial evidence as to whether early change in putative mechanisms of action could serve as an indicator of eventual treatment response, thus prompting discontinuation of care. Our study treatment was the Unified Protocol (UP; Barlow et al., 2018), a transdiagnostic CBT with demonstrated efficacy for a variety of psychiatric disorders (Cassiello-Robbins et al., 2020; Sakiris & Berle, 2019). The developers of the UP describe a functional model of mood, anxiety, and related disorders whereby aversive reactions to frequently occurring negative emotions are conceptualized as a mechanism that maintains and exacerbates symptoms of a wide range of emotional disorders (Bullis et al., 2019; Sauer-Zavala et al., 2017). Thus, the UP includes core skills aimed at reducing aversive reactions to negative emotions (e.g., psychoeducation on the adaptive nature of emotions, mindfulness training, cognitive flexibility, exposure to emotions). Improvements in aversive reactivity to emotions, an umbrella term that includes several related constructs (e.g., anxiety sensitivity,

experiential avoidance, distress intolerance; Semcho et al., 2022), has been shown to predict reductions in anxiety symptoms in CBT generally (Dalrymple & Herbert, 2007; Eustis et al., 2016; 2020; Kocovski et al., 2009; cf. Kocovski et al., 2015), and the UP specifically (Sauer-Zavala et al., 2012; Eustis et al., 2020), however research with more frequent assessment is needed to suggest temporal precedence.

Patients participating in our SMART underwent two randomizations. In the first-stage randomization, patients with primary anxiety, depressive, or related disorders were randomized to receive the five core skill modules from the UP (Wilamowska et al., 2010) in a personalized or standardized order. The second-stage randomization occurred at mid-treatment (i.e., after 6 sessions), with patients assigned to either discontinue care immediately or receive the remaining six sessions. This design, particularly our secondary randomization (i.e., early termination versus full course of care), is well-suited to (1) determine whether treatment outcomes vary as a function of treatment length (6 vs. 12 sessions) and (2) evaluate variables that may predict continued improvement after the withdrawal of care and could subsequently be used to make termination decisions. In order to use a psychological process as a trigger for a personalized treatment decision, it is first important to show that patients demonstrate change in this construct over the course of treatment and that these changes differ from patient to patient. Thus, Aim 1 of the present study was to determine (a) the average rate of change in aversive reactivity improvement (the putative mechanism of the UP) and (b) individual differences in this rate of change that occur during the first-stage randomization (i.e., across the first six sessions).

Next, in Aim 2, we examined whether the degree of early improvement in aversive reactivity, could predict maintenance or continued symptom improvement from week 6 to week 12. We were particularly interested in collecting initial evidence on whether mechanism

engagement (i.e., change in aversive reactivity to emotions) could serve as an early treatment response, prompting termination and signifying that a patient would ultimately continue to demonstrate symptom improvement.

Methods & Materials

Participants

A sample of treatment-seeking adult patients was recruited from Kentucky. Individuals were eligible for the study if they met Diagnostic and Statistical Manual (5th ed.; American Psychiatric Association, 2013) criteria for at least one of the following emotional disorders as determined by a clinician-rated structured clinical interview (Tolin et al, 2016): panic disorder (PD), generalized anxiety disorder (GAD), social anxiety disorder (SAD), obsessive-compulsive disorder (OCD), posttraumatic stress disorder (PTSD), major depressive disorder (MDD), or persistent depressive disorder (PDD). Individuals were excluded if they met criteria for diagnoses or symptoms requiring clinical prioritization or hospitalization, including acute suicide risk (i.e., imminent intent), mania within the past year (i.e., uncontrolled bipolar disorder), severe substance use disorder within the last three months (i.e., those with clinical severity ratings of $\geq$ 5 for SUD on our diagnostic measure; Tolin et al., 2016), or psychotic features. Individuals were also excluded if they had received five or more sessions of CBT within the last five years. Individuals included in the study agreed to discontinue other psychotherapy focused on emotional disorder symptoms prior to participating. Individuals taking psychotropic medication were asked to maintain their current dosages during study participation. The study was approved by the local university Institutional Review Board, and informed consent was obtained prior to any research activity.

A total of 70 people consented to participate in the study. Participants were 33.74 (*SD* =

12.64) years old on average and the majority of the sample identified as female (*n* = 47; 67.1%), white (*n* = 52; 74.3%), and heterosexual (*n* = 52; 74.3%). The most common primary diagnosis (i.e., rated as most distressing/interfering) was GAD (*n* = 33; 47.1%), followed by MDD (*n* = 19; 27.1%) and SAD (*n* = 16; 22.9%). See Sauer-Zavala et al. (2022) for full demographic data. Eleven participants (15.7%) did not complete study procedures after being lost to contact following baseline assessment (*n* = 2; 2.9%), unable to attend regular therapy sessions (*n* = 6; 8.6%), or discontinuing due to telehealth limitations resulting from the COVID-19 pandemic (i.e., patient moved out-of-state and could not receive telehealth sessions; *n* = 3; 4.3%). Participants recruited prior to March 15, 2020 (*n* = 29; 41.4%) completed at least some of their study visits in-person, whereas those enrolled on or after March 15, 2020 (*n* = 41; 58.6%) participated in all aspects of the study remotely due to the COVID-19 pandemic.

Study Design & Treatment

We conducted a pilot SMART with a two-stage randomization. Patients received a personalized version of the UP based on the conditions two which they randomized. They either received all or a selection of the following UP modules: Understanding Emotions, Mindful Emotion Awareness, Cognitive Flexibility, Countering Emotional Behaviors[1], and Confronting Physical Sensations (see Payne and colleagues [2014] for a full description of the UP modules). Modules were delivered in weekly, individual, 45–60-minute sessions. All modules consisted of two individual sessions except for Countering Emotional Behaviors, which was delivered across four sessions.

The first-stage randomization occurred following a baseline diagnostic assessment and

[1] The Countering Emotional Behaviors module in this study consists of two modules from the standard UP: Countering Emotional Behaviors and Emotion Exposures. We linked these two modules because both address aversive reactions to emotions by engaging in behaviors explicitly designed to approach emotional experiences.

involved randomization to one of three module-sequencing conditions: Standard, strengths, and weaknesses. Twenty-five participants (35.7%) were randomized to complete UP modules in the standard order described by Barlow et al. (2018): Understanding Emotions, Mindful Emotion Awareness, Cognitive Flexibility, Countering Emotional Behaviors, and Confronting Physical Sensations. Twenty-four (34.3%) completed these modules in an order that prioritized their relative strengths, and 21 (30.0%) completed these modules in an order that prioritized their relative deficits. See Sauer-Zavala et al. (2022) for more details about the first-stage randomization.

At the start of their sixth session, patients were informed of their assignment for the second-stage randomization (Brief treatment or Full treatment). Those in the Brief treatment condition discontinued care after that session for a total of six sessions of UP treatment (n = 30; 50.8%), whereas those in the Full condition completed 12 sessions total (n = 29; 49.2%). Patients were informed about the timing of the second-stage randomization during consenting (i.e., they understood they would be alerted to whether they were in the Brief or Full condition at the start of session 6). Study therapists were also masked to duration condition until this point so that knowledge of treatment duration would not influence patient or therapist behaviors and to allow session time for wrap-up in the Brief condition. Participants in the Full condition received all five modules, whereas those in the Brief condition received either two UP modules, if one module received was Countering Emotional Behaviors, or three, if Countering Emotional Behaviors was not assigned during the first six sessions.

Four study therapists provided the treatment: a licensed clinical psychologist, a post-doctoral fellow, and two advanced clinical psychology graduate students who were certified in the provision of the UP by one of its developers. All sessions were audio recorded, and 20%

were randomly selected to be rated for competence on a 5-point scale, which consisted of fidelity to the treatment protocol and therapeutic skill (e.g., time management, empathy). Average competence was high (M = 4.26, SD = .54) and did not differ between study sessions completed in-person (M = 4.26, SD = .50) or via telehealth (M = 4.26, SD = .59), $t(44) = .05$, $p = .96$, 95% CI [–.33, .35].

Measures

Emotional Disorder Symptoms

The Overall Anxiety Severity and Impairment Scale (OASIS; Norman et al., 2006) is a 5-item self-report questionnaire designed to measure anxiety symptoms over the prior week. Total scores range from 0 to 20 with a clinical cutoff score of 8. Participants completed the OASIS at baseline and weekly before each session. In the current sample, OASIS items demonstrated good internal consistency at baseline (McDonald's ω = .84).

The Overall Depression Severity and Impairment Scale (ODSIS; Bentley et al., 2014) is a 5-item self-report questionnaire designed to assess depressive symptoms over the prior week. Total scores also range from 0 to 20 with a clinical cutoff score of 8. Participants completed the ODSIS at baseline and weekly before each session. In the current sample, ODSIS items demonstrated excellent internal consistency at baseline (ω = .94).

Aversive Reactivity to Emotions

The Multidimensional Experiential Avoidance Questionnaire – Distress Aversion subscale (MEAQ-DA; Gámez et al., 2011) is a 13-item subscale from the MEAQ. The MEAQ-DA specifically assesses perceptions of distress as unwelcome or intolerable (e.g., "If I could magically remove all of my painful memories, I would;" Gámez et al., 2011). Items are rated on

a Likert-type scale from 1 (*strongly disagree*) to 6 (*strongly agree*). The MEAQ-DA subscale was used in the present study to reflect the UP's putative mechanism – aversive reactivity to emotions. Participants completed the MEAQ-DA at baseline and weekly before each session. MEAQ-DA items exhibited good internal consistency at baseline (ω = .89).

Data Analytic Plan

We used structural equation modeling as implemented in Mplus Version 7.0 (Muthén & Muthén, 2008-2012) to estimate all parameters of interest. We estimated separate latent intercept and slope factors for OASIS and ODSIS total scores across the first- and second-stage randomizations, respectively, in addition to a latent intercept and slope factor for MEAQ-DA total scores across the first-stage randomization. This process creates latent person-specific intercepts and slopes. Because participants were randomized to different module sequencing (strengths, weaknesses, standard) and treatment duration conditions (brief, full) and were seen by different therapists, we included dummy-coded indicator variables representing each of these factors as covariates in all analyses. We then examined the means and variances of the latent intercept and slope factors. Specifically, to test Aim 1, we examined the mean and variance of the latent slope factor for MEAQ-DA scores.

To test Aim 2, we first regressed the latent second-stage slopes of OASIS scores on latent first-stage intercepts[2] and slopes of MEAQ-DA scores and latent second-stage OASIS intercepts, including the indicator variables above. We examined whether the duration condition indicator variable was a significant predictor of second-stage OASIS slopes. We then repeated this model, replacing OASIS factors with ODSIS factors, and, in each model, examined whether first-stage MEAQ-DA intercepts and slopes significantly predicted second-stage OASIS or ODSIS slopes.

[2] Models did not converge when we coded session 6 as the reference value for MEAQ-DA intercepts and slopes, so we retained session 1 as the reference value in all analyses.

We then conducted a series of Wald tests to determine if the size of the effect of these predictors significantly differed from one another.

Finally, we conducted two sets of robustness checks. In the first set, we re-ran our primary Aim 2 models, adding the product term between duration condition and first-stage MEAQ-DA slopes to test if the relations between first-stage changes in distress aversion and second-stage changes in anxiety and depression differed by whether participants discontinued treatment after session 6. In the second set, to evaluate if these results held when including latent first-stage OASIS (or ODSIS) intercepts and slopes in each of the two models above. We examined anxiety and depression outcomes in separate models due to errors estimating standard errors when trying to fit a single model with more parameters than participants.

For all models, we evaluated model fit using Hu and Bentler's (1999) recommendations (i.e., root-mean-square error of approximation [RMSEA; acceptable fit ≤ .10; good fit ≤ .06], comparative fit index [CFI] and Tucker-Lewis Index [TLI; acceptable fit ≥ .90; excellent fit ≥ .95]. Using the *pwrSEM* Shiny application (Version 0.1.2; Wang & Rhemtulla, 2021), we had 80% power to detect βs ≥ |.35| in OASIS models and βs ≥ |.32| in ODSIS models. To maximize power and account for missingness, we used full information maximum likelihood estimation, and we examined standardized estimates to ease interpretation. Code for analyses is available at https://osf.io/62mzw/?view_only=4c32fb7de6f341378fb5eea836c5ab90 and the parent study was registered at clinicaltrials.gov (NCT04584879).

Results

Aim 1

The model in which second-stage latent OASIS slopes were regressed on first-stage latent MEAQ-DA slopes and intercepts, second-stage latent OASIS intercepts, randomization

condition and therapist covariates demonstrated acceptable fit, $\chi^2(248) = 332.35$, $p < .01$; RMSEA = .071, 90% CI [.050, .090]; CFI = .908; TLI = .903; SRMR = .107. Similarly, the model in which second-stage latent ODSIS slopes were regressed on first-stage latent MEAQ-DA slopes and intercepts, second-stage latent ODSIS intercepts, randomization condition and therapist covariates demonstrated acceptable fit, $\chi^2(248) = 362.58$, $p < .01$; RMSEA = .083, 90% CI [.064, .101]; CFI = .900; TLI = .894; SRMR = .109 (Figure 3). To test Aim 1, we examined the mean and variability estimates of the latent MEAQ-DA slope factors. MEAQ-DA slopes were estimated to be significantly different from zero, $\hat{\mu} = -1.25$, $SE = .25$, $p < .01$, and to have significant variability, $\widehat{\sigma^2} = 2.55$, $SE = .69$, $p < .01$, indicating that roughly 68% of participants experienced average session by session changes in MEAQ-DA scores between –2.84 points per session and .35 points per session during the first-stage randomization (Table 1). Thus, patients, on average, demonstrated less distress aversion during the first 6 weeks of treatment with the UP; however, there was significant variability in the extent of that improvement across patients, suggesting that MEAQ-DA scores could serve as a tailoring variable.

Aim 2

We then examined if second-stage changes in OASIS and ODSIS scores differed by treatment duration condition. The slopes of OASIS scores during the second-stage randomization from participants in the Full treatment condition were numerically, but not significantly, more negative than those from participants in the Brief condition, $\beta = -.20.$, $SE = .16$, $p = .21$, 95% CI [–.51, .11]. By contrast, the slopes of ODSIS scores during the second-stage randomization from participants in the Brief treatment condition were numerically, but not significantly, more negative than those from participants in the Full treatment condition, $\beta = .02$, $SE = .16$, $p = .90$, 95% CI [–.29, .33]. Given that the effect of duration condition was non-significant in each

model, we collapsed second-stage OASIS and ODSIS slopes across treatment duration conditions to test the effects of MEAQ-DA intercepts and slopes on anxiety and depression changes in the second stage (Figure 2).

Steeper decreases in MEAQ-DA scores during the first-stage randomization significantly predicted continued steeper decreases in OASIS scores during the second-stage randomization, $\beta = .51$, $SE = .23$, $p = .03$, 95% CI [.06, .97]. By contrast, neither MEAQ-DA scores at session 1, $\beta = .26$, $SE = .18$, $p = .16$, 95% CI [–.10, .62], nor OASIS scores at session/week 7, $\beta = –.29$, $SE = .23$, $p = .22$, 95% CI [–.74, .17], significantly predicted second-stage changes in OASIS scores. Although the effect of first-stage MEAQ-DA slopes was significantly larger than that of MEAQ-DA scores at session 1, $\chi^2(1) = 4.40$, $p = .04$, the effect of first-stage MEAQ-DA slopes was not significantly larger than the absolute value of OASIS scores at session/week 7, $\chi^2(1) = 1.39$, $p = .24$. Using model-derived factor scores, participants demonstrating MEAQ-DA slopes $\leq –1.51$, corresponding to a 6-session decrease of 9.06 points, were likely to see continued decreases in anxiety during the second-stage randomization. In sum, early improvements in distress aversion (from session 1 to 6), predicted later improvements in anxiety symptoms (from session 7 to 12), whereas neither initial intensity of distress aversions nor severity of anxiety after 6 weeks of treatment, predicted anxiety improvement, although the effect of early improvements in distress aversion only significantly differed from initial intensity of distress aversion.

Lower MEAQ-DA scores at session 1 significantly predicted steeper decreases in ODSIS scores across the second-stage randomization, $\beta = .51$, $SE = .17$, $p < .01$, 95% CI [.18, .84]. Neither MEAQ-DA slopes, $\beta = –.26$, $SE = .22$, $p = .23$, 95% CI [–.69, .16], nor session/week 7 ODSIS scores, $\beta = .05$, $SE = .24$, $p = .83$, 95% CI [–.41, .52], significantly predicted changes in ODSIS scores across the second-stage randomization. However, the effect of MEAQ-DA scores

at session 1 was not significantly larger than the effect of first-stage MEAQ-DA slopes, $\chi^2(1) = .70$, $p = .40$, nor the effect of session/week 7 ODSIS scores, $\chi^2(1) = .18$, $p = .67$. Of note, MEAQ-DA intercepts were estimated to be significantly different from zero, $\hat{\mu} = 44.98$, $SE = 1.53$, $p < .01$, and to have significant variability, $\widehat{\sigma^2} = 145.65$, $SE = 27.00$, $p < .01$, indicating that roughly 68% of participants reported MEAQ-DA scores between 32.91 and 57.05 before the start of session 1 (Table 1). Using model-derived factor scores, participants with MEAQ-DA scores of 41.07 or below before session 1 were likely to see continued decreases in depression during the second-stage randomization. In sum, intensity of distress aversion at session 1 predicted whether patients' depressive symptoms continued to improve in weeks 6-12 of the study, whereas changes in and severity of depression after 6 weeks of treatment did not, although the size of these effects did not significantly differ from each other.

Robustness Checks

Finally, we conducted two sets of robustness checks. We first tested if duration condition (i.e., 6 sessions of treatment or 12 sessions of treatment) moderated the relations between first-stage MEAQ-DA slopes and second-stage OASIS or ODSIS slopes in both primary Aim 2 models. Duration condition did not significantly moderate the relations between first-stage MEAQ-DA slopes and second-stage OASIS, $B = .10$, $SE = .07$, $p = .16$, or ODSIS, $B = –.07$, $SE = .09$, $p = .43$, slopes.

We then included latent OASIS or ODSIS intercepts and slopes, respectively, from the first-stage randomization in our primary Aim 2 models above. Although the size of the effect of first-stage randomization MEAQ-DA slopes on second-stage OASIS slopes was numerically larger in this model, it was not significant, $\beta = .56$, $SE = .31$, $p = .07$, 95% CI [–.04, 1.17]. Of note, neither first-stage OASIS intercepts nor slopes significantly predicted second-stage OASIS

slopes in this model, *p*s > .75. A similar pattern emerged when examining the ODSIS model. Here, latent MEAQ-DA intercepts at session 1 did not significantly predict second-stage OASIS slopes, β = .20, *SE* = .22, *p* = .35, 95% CI [–.23, .64], although neither did first-stage OASIS intercepts or slopes, *p*s > 65. In sum, when included latent intercepts and slopes for anxiety and depression in their respective models, early intensity and improvement in distress aversion no longer predicted later (study week 6-12) symptom improvement.

Discussion

The goal of the present study was to explore whether early changes in aversive reactivity to emotions—the putative core psychopathological mechanism targeted by the UP—could serve as a proximal indicator of treatment response that could prompt discontinuation prior to full symptom remission. To this end, participants were randomized to receive courses of the UP of varying lengths. We found no significant differences in the rates of symptom change between those who discontinued after session 6 and those who received the full UP treatment package (12 sessions). For all participants, steeper decreases in distress aversion during the first six weeks of treatment predicted steeper decreases in anxiety symptoms across weeks 7 to 12 regardless of whether patients discontinued treatment after their sixth session or continued for the full 12 sessions. Additionally, lower distress aversion at pre-treatment predicted steeper decreases in depressive symptoms during weeks 7 to 12. However, when severity and rate of improvement of anxiety and depressive symptoms were accounted for, early intensity and improvement in distress aversion no longer significantly predicted later (week 7-12) symptom improvement.

The lack of significant differences in second-stage symptom change between those in the Brief and Full duration conditions was surprising. However, these results are in line with previous research suggesting that patients may experience less pronounced symptom change

over time in treatment (Robinson et al., 2020a; Stulz et al., 2013). Our results extend these findings by suggesting that, for outpatients with transdiagnostic mood and anxiety disorders, the trajectory of symptoms after six sessions of treatment may not substantially differ whether patients continue in treatment or not. Shorter courses of care may be more feasible to administer in many settings and may address long waiting lists for services for acute symptoms. It is important to note, however, that other outcomes (e.g., quality of life) that are important to patient functioning were not evaluated here and longer courses of care may be required to address them (cf. Elhusseini et al., 2022).

Of course, these results regarding second-stage symptom change specifically apply to the group average – individual patients likely differ in their trajectories. In fact, we found significant between-patient variability in the slopes and intercepts of first-stage distress aversion which uniquely predicted second-stage changes in anxiety and depressive symptoms, respectively. The significant between-patient variability in first-stage distress aversion intercepts and slopes satisfies a necessary requirement for constructs to be used as tailoring variables. This indication was supported in the current study with unique effects on prospective anxiety and depressive symptom change.

Specifically, patients with steeper decreases in distress aversion during the first-stage randomization tended to demonstrate steeper decreases in anxiety during the second-stage randomization, regardless of whether they continued with treatment. When first-stage intercepts and slopes of anxiety were included in this model, the effect size of distress aversion slopes remained relatively unchanged, although it was rendered non-significant. These results replicate previous findings in which steeper decreases in distress aversion predicted subsequent decreases in anxiety over four-session blocks of the UP (Eustis et al., 2020). Our results extend these

findings by incorporating measures of distress aversion and anxiety at every session and demonstrating that such effects may hold regardless of whether patients continue in treatment. In addition to replicating the degree of change needed on the MEAQ-DA to predict continue improvement in anxiety symptoms, a next step in validating change on aversive reactivity as a tailoring variable for anxiety is to conduct a randomized trial in which patients are assigned to continue or discontinue treatment when they reach a specified level of improvement on the MEAQ-DA.

Whereas first-stage changes in distress aversion predicted second-stage changes in anxiety, first-stage distress aversion intercepts predicted second-stage changes in depression. This result suggests that patients who reported more unwillingness to experience emotions prior to their first therapy session would be more likely to experience decreases in depression during the second-stage randomization, regardless of whether they continued with treatment. Our result replicates previous research demonstrating that intensity of experiential avoidance, which is one aspect of aversive reactivity (Semcho et al., 2022), predicted subsequent decreases in depression over four-month intervals among women with borderline personality disorder in dialectical behavior therapy (Berking et al., 2009). Our results extend these findings to outpatients of multiple genders with emotional disorders more broadly, regardless of whether they continue with treatment. Together, these results suggest that higher initial levels of distress aversion may impede patients' depressive symptom improvement, perhaps by reducing their engagement with therapy processes intended to directly address depression (e.g., treatment skills, therapist support, etc.). This effect was significant above and beyond changes in distress aversion, suggesting that distress aversion may exhibit a trait-like effect on depressive symptom change that persists regardless of how much change distress aversion exhibits early in treatment.

Of course, it is important to note the effects of distress aversion change on subsequent anxiety symptom improvement and baseline intensity on subsequent depressive symptom improvement were non-significant when early severity and changes in anxiety and depressive symptoms were included in their respective models. However, because the size of the effects remained stable when including these covariates even in the context of our relatively small sample, it remains possible that changes in distress aversion may be a functional mechanism on which treatment planning decisions may be made. At the same time, it is also possible that early symptom reduction (including sudden gains) may itself be a more direct indicator of eventual remission, especially since the size of the effects of distress aversion intensity and change did not significantly differ from the size of the effects of early changes in anxiety and depressive symptoms. Future researchers should continue to use adaptive treatment designs (i.e., randomization to different treatment lengths) with larger samples to draw stronger conclusions about the most useful information to draw on when making treatment planning decisions.

These results should be considered in light of the study's limitations. Although we were powered to detect medium-to-large sized effects, our sample was relatively small and homogeneous, especially when testing for differences between treatment conditions. We encourage future researchers to replicate these findings in larger and more diverse samples. Testing for treatment change after six compared to twelve sessions is also an arbitrary distinction, although one informed by the literature on symptom change (e.g., Niileksela et al., 2021; Sauer-Zavala et al., 2022; Southward & Sauer-Zavala, 2022) and useful for directly comparing symptom change over comparable time scales. Additionally, the primary measures in this study were self-reported outcomes administered by patients' therapists. Although therapists did not see these measures, it is possible that patients experienced demand characteristics that

may have biased their responses. Moreover, it is possible that, for patients in the brief condition, completing weekly questionnaires after discontinuation may have reminded them of weekly therapy and affected their scores on outcome measures. Additionally, with regard to real-world generalizability, it may be difficult to administer weekly measures of distress aversion in routine practice where assessment is already quite limited. Finally, although we chose aversive reactivity as our mechanism of action because it is theoretically linked to the UP, recent reviews indicate that psychotherapy research has yet to identify a single robust mechanism that is relevant to most patients (Carey et al., 2020).

Despite these limitations, we found some evidence that distress aversion may function as a tailoring variable for predicting subsequent change in anxiety and depressive symptoms among outpatients with transdiagnostic emotional disorders treated with the UP. Given the shortfall of mental health providers to address the growing demand of consumers, continuation of the line of research is paramount. It is imperative that our treatment protocols and our decisions about their delivery align with the clinical reality of routine practice. Too few providers necessitates moving patients through the therapeutic process as efficiently as possible so that individuals on the waitlist can access care sooner. Therapists using routine outcome monitoring may use these findings to determine how a patient's initial levels of and change in mechanisms and/or symptoms may indicate subsequent changes in functioning, prompting discontinuation before full remission. These results highlight the importance of assessing putative functional mechanisms, as they may act as leading indicators of subsequent symptom change, regardless of whether a patient continues treatment. We encourage future researchers to expand on these findings through replication and comparisons with other potential leading indicators to develop an optimal set of predictors for patient outcomes. Shorter courses of care based on data-driven

termination decisions may increase treatment efficiency, which has the potential to reduce patient costs and increase the mental health service system's capacity.

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Table 1

Estimated Means, Variances, and Correlations Among Latent Intercepts and Slopes

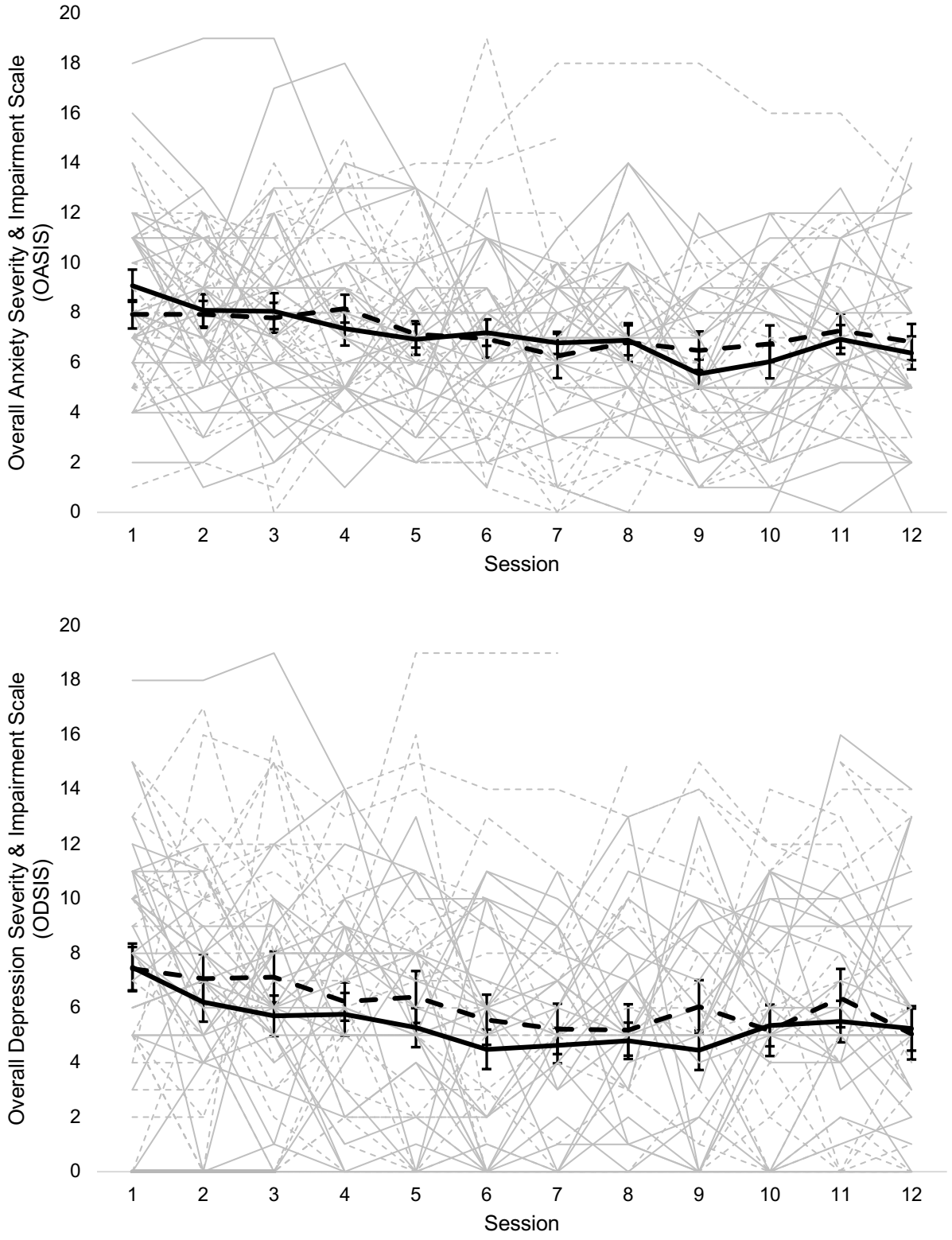
Variable	*M*	s^2	1	2	3	4	5	6	7	8	9
1. OASIS Intercepts - First Stage	8.50	9.25									
2. OASIS Slopes - First Stage	–.30	.28	–.71**								
3. OASIS Intercepts - Second Stage	6.63	9.30	.63**	–.15							
4. OASIS Slopes - Second Stage	.08	.24	–.32	.20	–.46**						
5. ODSIS Intercepts - First Stage	7.38	15.74	.72**	–.35	.48**	–.05					
6. ODSIS Slopes - First Stage	–.49	.20	–.67**	.74*	–.04	–.02	–.43*				
7. ODSIS Intercepts - Second Stage	4.94	15.14	.35**	.08	.56**	–.24	.66**	.49*			
8. ODSIS Slopes - Second Stage	.17	.29	.01	–.09	–.08	.77**	.17	–.49	–.26		
9. MEAQ-DA Intercepts - First Stage	45.21	148.72	.23	< .01	.23	.05	.32*	–.51*	.02	.42*	
10. MEAQ-DA Slopes - First Stage	–1.26	2.52	–.30	.27	–.36*	.55**	.03	.03	.08	–.24	–.20

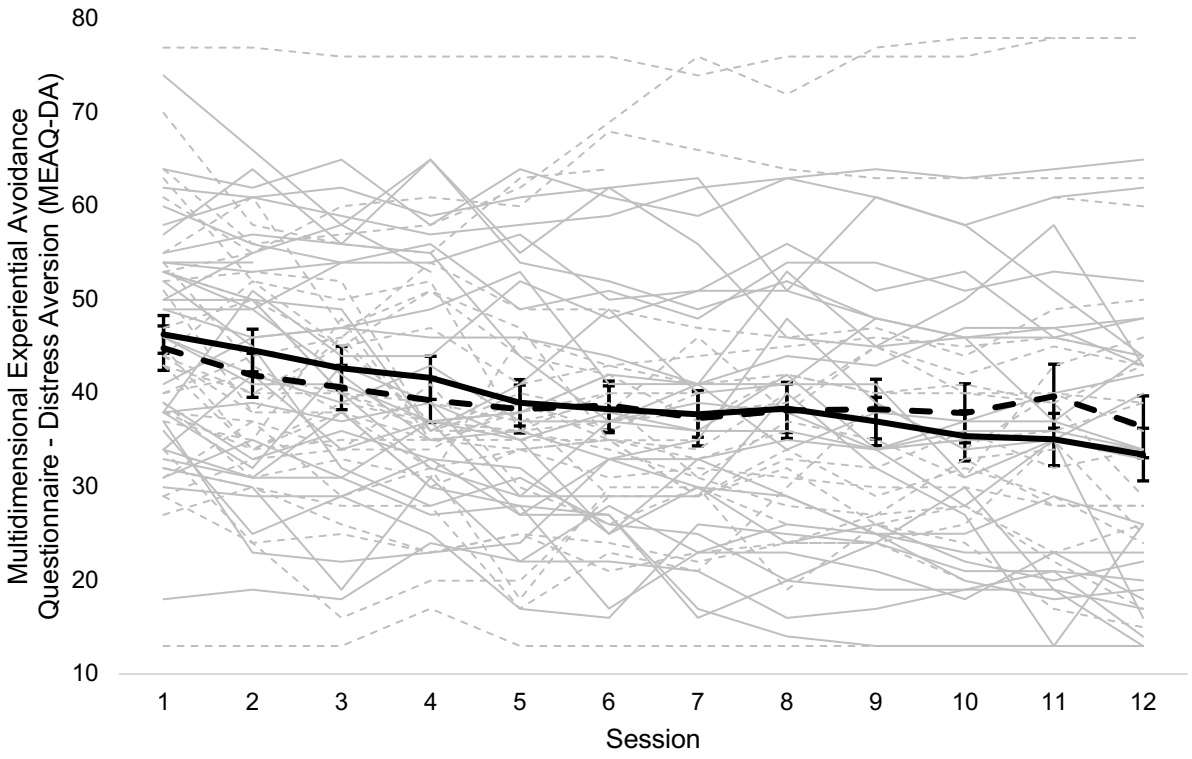
Note. Values generated from a structural equation model with only covariances among variables and no regressions and so may differ slightly from values generated by models reported in the text. OASIS = Overall Anxiety Severity and Impairment Scale. ODSIS = Overall Depression Severity and Impairment Scale. MEAQ-DA = Multidimensional Experiential Avoidance Questionnaire-Distress Aversion.

* $p < .05$, ** $p < .01$.

Figure 1

Change in Anxiety, Depression, and Distress Aversion by Treatment Duration Condition

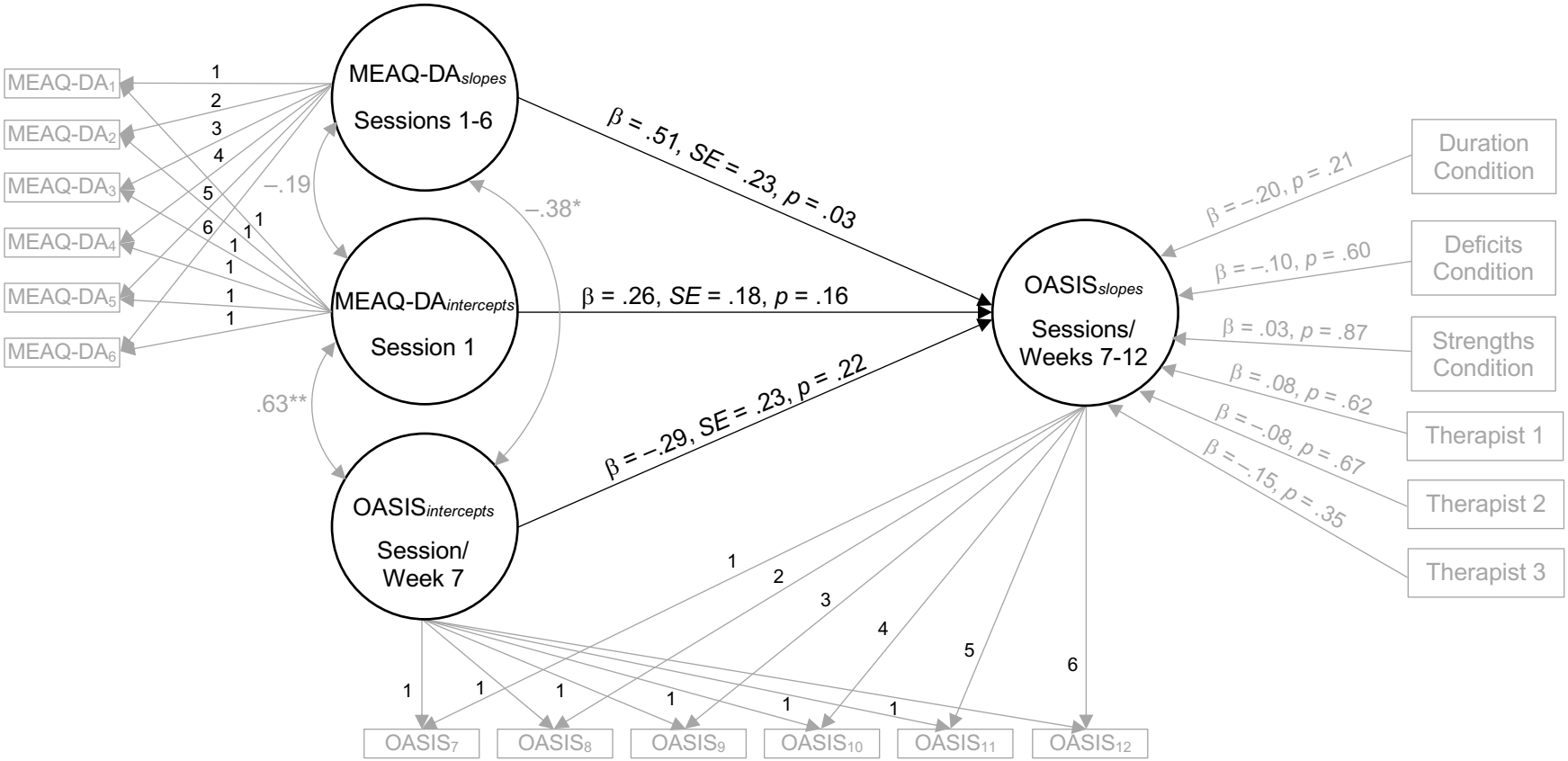




Note. Solid lines = Full treatment condition. Dashed lines = Brief treatment condition. Black lines = group average. Grey lines = individual trajectories. Error bars represent standard errors.

Figure 2

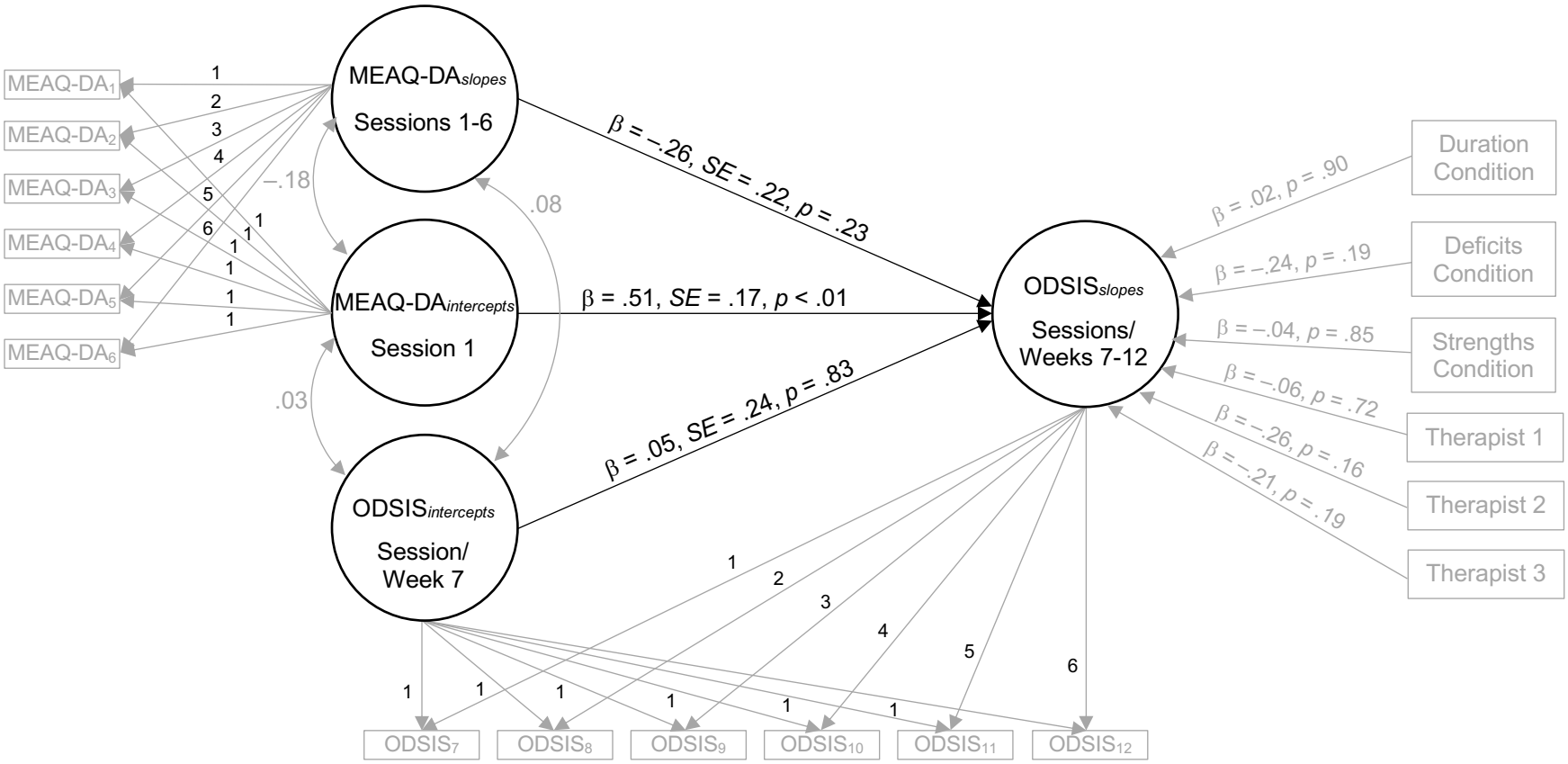
Structural Equation Model of First-Stage MEAQ-DA Latent Slopes and Intercepts and Second-Stage OASIS Intercepts Predicting Second-Stage OASIS Slopes



Note. * $p < .05$, ** $p < .01$.

Figure 3

Structural Equation Model of First-Stage MEAQ-DA Latent Slopes and Intercepts and Second-Stage ODSIS Intercepts Predicting Second-Stage ODSIS Slopes



Note. * $p < .05$, ** $p < .01$.