Effects of Intensive Short-Term Dynamic Psychotherapy on Depression: A Reanalysis of Heshmati et al.'s Data

Robert Johansson[1] & Peter Lilliengren[1]

[1] Department of Psychology, Stockholm University, Stockholm, Sweden

Author Note

Correspondence concerning this article should be addressed to Robert Johansson.

E-mail: robert.johansson@psychology.su.se

Abstract

Objective: This reanalysis examined effects and mechanisms of Intensive Short-Term Dynamic Psychotherapy (ISTDP) for treatment-resistant depression using public trial data. **Method:** The original trial randomized 86 Iranian adults to ISTDP (20 sessions over 10 weeks) or waitlist control. Depression and process measures (emotional repression, negative affect, distress) were assessed at baseline, post-treatment, and 3-month follow-up. Mixed-effects models analyzed trajectories; mediation and cross-lagged analyses examined mechanisms. **Results:** Significant Time × Treatment interactions emerged ($p < .001$). ISTDP participants showed large reductions (M_{diff} = -12.87 points at follow-up, $p < .001$); waitlist showed minimal change. Between-group effect size reached $d = 2.50$ (95% CI [1.88, 3.11]) at follow-up. Process measures showed comparable effects (d = 1.96-2.95). The WAI Distress composite (which includes the Depression subscale, creating construct overlap) statistically mediated part of the effect; this cannot be interpreted as evidence for a mechanism. Emotional repression and negative affect did not significantly mediate outcomes. Cross-lagged analyses indicated concurrent rather than sequential change. **Conclusion:** This reanalysis confirms large ISTDP effects on depression in treatment-resistant populations. However, ISTDP appears to create broad, simultaneous therapeutic change rather than working through specific sequential mechanisms.

Keywords: depression, psychotherapy, ISTDP, treatment-resistant depression, reanalysis

Effects of Intensive Short-Term Dynamic Psychotherapy on Depression: A Reanalysis of Heshmati et al.'s Data

Introduction

Depression is one of the most prevalent and debilitating mental health conditions worldwide, affecting an estimated 280 million individuals and representing a leading cause of disability (World Health Organization, 2021). While evidence-based treatments such as antidepressant medication and psychotherapy produce meaningful improvements for many patients, a substantial proportion—approximately 30% to 40%—do not achieve remission with first-line interventions (Fava & Davidson, 1996; Rush et al., 2006). This phenomenon, termed treatment-resistant depression (TRD), is characterized by inadequate response to at least one adequate trial of antidepressant treatment and poses one of the most formidable challenges in contemporary mental health care (Gaynes et al., 2020). The burden of TRD is considerable, not only for affected individuals who experience prolonged suffering and functional impairment, but also for healthcare systems facing increased service utilization and costs (Mrazek, Hornberger, Altar, & Degtiar, 2014). Given the cumulative failure rates that increase with sequential treatment trials (Rush et al., 2006), there is an urgent need for effective alternative interventions that can offer relief when conventional approaches prove insufficient.

Psychotherapy represents a promising avenue for addressing TRD, with emerging evidence suggesting that structured psychological interventions can produce clinically meaningful improvements even in patients who have not responded to pharmacotherapy (Cuijpers, Karyotaki, Wit, & Ebert, 2020; Wiles et al., 2013). Among psychotherapeutic approaches, Intensive Short-Term Dynamic Psychotherapy (ISTDP) has garnered increasing empirical support for the treatment of depression, including treatment-resistant presentations (A. A. Abbass et al., 2014; Town, Abbass, Stride, & Bernier, 2017). ISTDP is a form of brief psychodynamic therapy developed by Habib Davanloo (Davanloo, 2000) that

aims to rapidly access and resolve unconscious emotional conflicts through active techniques designed to overcome psychological defenses. Unlike traditional psychodynamic approaches that may extend over years, ISTDP is delivered in a time-limited format, typically ranging from 10 to 40 individual sessions, often conducted twice weekly in an intensive structure (A. Abbass, 2015). The approach is characterized by systematic attention to the therapeutic relationship, active challenge of maladaptive defenses, and facilitation of direct emotional experiencing of previously avoided feelings.

The theoretical framework underlying ISTDP proposes that depression, like other forms of psychopathology, arises fundamentally from unprocessed attachment trauma and the defensive avoidance of painful emotions associated with early relational experiences (A. Abbass, 2015; Frederickson, 2013). According to this model, attachment-related injuries—such as loss, neglect, criticism, or abuse—evoke powerful reactive emotions in the developing child, particularly anger directed toward attachment figures who have been experienced as frustrating, rejecting, or threatening (Johansson, Eriksson, Åberg, Town, & Abbass, 2024). However, because the child's psychological and physical survival depends on maintaining proximity to these same attachment figures, the direct expression of anger is experienced as dangerous and is therefore defended against through various psychological mechanisms. In the specific case of depression, the theory posits that the child may internalize the critical, judgmental, or threatening attributes of the attachment figure through identification, turning anger inward and developing a pattern of self-attack that mirrors the original external threat (Frederickson, 2013; Johansson et al., 2024). This internalization gives rise to the regressive defenses characterizing depression—including self-criticism, self-destructiveness, resignation, and agonizing—which serve to distance the individual from the conflicting painful feelings associated with the frustrating other (Frederickson, 2013). Unconscious rage, guilt about this rage, and grief over what was lost or never received are believed to lie at the root of depressive symptomatology, with the chronic avoidance of these complex emotions driving the depression forward over time (A. Abbass,

2015; Johansson et al., 2024).

The ISTDP treatment approach follows directly from this etiological model. The therapist actively works to help patients relinquish their defensive strategies and directly experience the previously avoided emotions associated with past attachment trauma, thereby revealing the internal dynamics that maintain the depression (A. Abbass, 2015; Della Selva, 2004). Treatment is always adapted to the individual patient's level of anxiety tolerance and defensive organization, with specific attention in depressed patients to their characteristic difficulties with intimacy and closeness, vulnerability to loss and separation, and resistance to the therapeutic relationship itself (A. Abbass, 2015; Johansson et al., 2024). The technique involves addressing the patient's stated desire to feel better while simultaneously challenging the patient's defenses against closeness and emotional experiencing. Through this process, the aim is to access and make conscious the unconscious complex emotions—particularly anger, guilt, and grief—associated with past attachment trauma, thereby dismantling the defensive structures that generate and perpetuate depressive symptoms.

Empirical research on ISTDP has accumulated steadily over the past two decades, with meta-analytic evidence indicating moderate to large effects across a range of psychiatric conditions (A. A. Abbass, Hancock, Henderson, & Kisely, 2006; A. A. Abbass et al., 2014). For depression specifically, multiple randomized controlled trials have demonstrated that ISTDP produces clinically significant improvements compared to control conditions receiving usual care or minimal intervention (A. Abbass, 2006; A. Abbass, Town, & Driessen, 2013; A. Abbass, Town, & Driessen, 2012; Town et al., 2017). When compared to waitlist control conditions, ISTDP has been shown to produce large effect sizes for both depressive symptoms and associated interpersonal difficulties, with effects maintained at follow-up assessments ranging from 3 to 12 months (Ajilchi, Nejati, Town, Wilson, & Abbass, 2016; Solbakken & Abbass, 2015). Importantly, these beneficial effects have been observed specifically in treatment-resistant populations. Town and colleagues (Town et al., 2017, 2020)

conducted a randomized controlled trial of ISTDP versus treatment as usual in patients with treatment-resistant depression, finding large effects on depressive symptoms that were sustained through an 18-month follow-up period, along with significant reductions in healthcare costs.

Recent large-scale studies have further strengthened the evidence base for ISTDP in depression. Heshmati et al. (Heshmati, Wienicke, & Driessen, 2023) conducted a randomized controlled trial with 86 Iranian adults diagnosed with treatment-resistant depression, comparing 20 sessions of ISTDP delivered over 10 weeks to a waitlist control condition. The study found very large effects on depressive symptoms (Cohen's $d = 1.68$ at post-treatment, increasing to $d = 2.50$ at 3-month follow-up), along with substantial improvements in emotional repression and negative affect. Similarly, Johansson et al. (Johansson et al., 2024) examined ISTDP effectiveness in a naturalistic sample of 195 patients with depression treated at a specialty clinic in Halifax, Canada, finding large within-group effects on both depression (Cohen's $d = 1.02$) and interpersonal problems (Cohen's $d = 1.17$). Across these studies, the consistency and magnitude of observed effects suggest that ISTDP represents a robust and effective intervention for depression, including presentations that have proven resistant to conventional treatments.

Despite this accumulating evidence that ISTDP works for depression, a critical question remains largely unanswered: *How* does ISTDP work? Understanding the mechanisms through which therapeutic interventions produce their effects is essential not only for refining theoretical models, but also for optimizing treatment delivery, improving training programs, and identifying which patients are most likely to benefit from specific therapeutic approaches (Kazdin, 2007; Kraemer, Wilson, Fairburn, & Agras, 2002). The theoretical model articulated above proposes a specific sequential mechanism: ISTDP techniques help patients reduce defensive avoidance of emotions (i.e., reduce emotional repression), which in turn allows for the experience and processing of previously avoided feelings (reducing negative

affect and distress), which ultimately produces relief from depressive symptoms. This theoretical pathway implies that changes in emotional repression and affect should not merely co-occur with depression improvement, but should actually *mediate* that improvement—that is, serve as mechanisms through which treatment exerts its effects.

Some empirical evidence supports the proposed mediating role of emotional experiencing in ISTDP. Johansson et al. (Johansson, Town, & Abbass, 2014) and Town et al. (Town, Abbass, & Bernier, 2013) found that "unlocking the unconscious"—a process characterized by the patient accessing and expressing previously repressed emotions, often accompanied by visual associations linking current feelings to past attachment figures—was associated with reduced symptom levels and fewer interpersonal problems. More recently, Johansson et al. (Johansson et al., 2024) examined this process as a formal mediator in their naturalistic sample of depressed patients, finding that the occurrence of unlocking during treatment significantly mediated outcomes, with a between-group effect size of $d = 0.60$ for depression and $d = 0.47$ for interpersonal problems. Patients who experienced unlocking during treatment showed larger improvements (depression $d = 1.32$) compared to those who did not ($d = 0.72$). Similarly, Town et al. (Town, Falkenström, Abbass, & Stride, 2022) investigated in-session affect experiencing as a mechanism in an RCT for treatment-resistant depression, finding that experiencing anger during sessions predicted subsequent reductions in depressive symptoms, particularly for patients with lower levels of personality pathology, and that this relationship was mediated by therapeutic alliance and insight.

However, these promising findings are tempered by important methodological limitations. Most existing studies of ISTDP mechanisms have relied on concurrent associations between process measures and outcomes, which cannot establish the temporal ordering necessary to infer causation (Laurenceau, Hayes, & Feldman, 2007). Even when formal mediation analyses have been conducted, they have typically examined change scores calculated across the same time period for both mediator and outcome, leaving open the

question of whether process changes actually *precede* and cause symptom changes, or whether both simply change together in response to some other factor (Kazdin, 2007). The Heshmati et al. (Heshmati et al., 2023) study, despite demonstrating very large effects on both depression and proposed process measures (emotional repression, negative affect, overall distress), did not test whether changes in these process variables mediated the observed depression improvements, nor did it examine the temporal sequencing of changes across different domains.

The present study addresses these critical gaps by conducting a comprehensive reanalysis of the public data from Heshmati et al. (Heshmati et al., 2023), applying advanced statistical methods to rigorously test the theoretical mechanisms proposed to underlie ISTDP's effects on treatment-resistant depression. Leveraging the principles of open science that make such secondary analyses possible (Nosek et al., 2015), we examined four specific research questions. First, we sought to confirm and extend the original study's findings by examining the magnitude and durability of ISTDP's effects on depression across all available assessment points, including the 3-month follow-up. Second, we tested whether ISTDP produces the theoretically predicted changes in proposed process measures—emotional repression, negative affect, and overall distress—with effect sizes comparable to those observed for depression itself. Third, and most critically, we conducted formal mediation analyses to test whether changes in these process variables statistically mediate the relationship between treatment assignment and depression outcomes, as the theoretical model would predict. Fourth, we employed cross-lagged panel analyses to examine temporal precedence, testing whether earlier changes in process measures predict subsequent changes in depression (consistent with a causal mediational role) or whether the temporal ordering suggests a different pattern of relationships.

We hypothesized that ISTDP would produce large effects on depression that would be maintained or increase at the 3-month follow-up assessment, consistent with the original

study findings. We further hypothesized that ISTDP would produce large effects on all proposed process measures, with magnitudes comparable to effects on depression. Most importantly, we hypothesized that changes in emotional repression and negative affect would significantly mediate depression improvements, with substantial indirect effects, and that these process changes would demonstrate temporal precedence by predicting subsequent depression changes beyond what could be explained by earlier depression levels. These hypotheses follow directly from ISTDP theory and, if supported, would provide the strongest available evidence that the proposed mechanisms actually account for how ISTDP produces its therapeutic effects. Alternatively, failure to find evidence for mediation or temporal precedence would challenge current theoretical assumptions and suggest that either different mechanisms are at work or that current measurement approaches fail to adequately capture the theorized processes. This study thus represents a critical test of ISTDP's theoretical foundations, with important implications for understanding not only how ISTDP works specifically, but also how psychotherapy more broadly produces therapeutic change in depression.

Method

Study Design and Participants

Overview and Data Source. This study presents a reanalysis of publicly available data from a randomized controlled trial examining the effects of Intensive Short-Term Dynamic Psychotherapy (ISTDP) for treatment-resistant depression (Heshmati et al., 2023, 2025). The complete dataset, codebook, and documentation are openly accessible on the Open Science Framework (https://doi.org/10.17605/OSF.IO/75PU8). As a secondary analysis, our focus diverges from the original study by examining process-outcome relationships through advanced statistical techniques (mediation and cross-lagged analyses) that were not employed in the original publications.

Study Design. The original trial employed a two-arm parallel-group randomized

controlled design with a 1:1 allocation ratio. Randomization was conducted using computer-generated random numbers, with allocations concealed in sealed, opaque envelopes opened sequentially after baseline assessment. The study was retrospectively registered on the Open Science Framework (https://osf.io/v46gy). Assessment personnel were not blinded to treatment allocation, though outcome measures were self-report questionnaires, which limits detection bias but does not eliminate potential expectancy effects.

Recruitment and Setting. Participants were recruited between April and May 2020 through referrals from psychiatrists and mental health clinics in Tabriz, Iran. Treatment was delivered between June and August 2020, with follow-up assessments completed in November 2020. The COVID-19 pandemic was ongoing during data collection, though the original report does not indicate substantial impacts on recruitment or retention.

Participants. The study enrolled 86 Iranian adults (ages 18-60) with a current major depressive episode that had persisted for at least six weeks despite treatment with antidepressant medication. Treatment resistance was operationalized as failure to achieve adequate symptom reduction following at least one adequate trial (defined as at least 6 weeks at therapeutic dose) of an antidepressant medication from any class. All participants met DSM-IV criteria for major depressive disorder, assessed via structured clinical interview.

Participant Flow. The original study screened participants referred from psychiatrists and mental health clinics; specific screening numbers were not reported in the published trial. Of those meeting eligibility criteria, 86 participants were randomized (43 to ISTDP, 43 to waitlist control). Attrition during the study was 12.8% overall: 6 participants (14.0%) withdrew from the ISTDP condition and 5 participants (11.6%) withdrew from the waitlist condition. At the 3-month follow-up assessment, data were available from 37 ISTDP participants and 38 waitlist participants. All randomized participants were included in intent-to-treat analyses using mixed-effects models with REML estimation.

Inclusion Criteria. Participants were required to: (a) be ages 18-60 years; (b) have a minimum of high school education (to ensure comprehension of self-report measures); (c) meet diagnostic criteria for current major depressive episode; (d) have treatment-resistant depression as defined above; and (e) provide informed consent.

Exclusion Criteria. Individuals were excluded for: (a) comorbid personality disorder (assessed via clinical interview); (b) psychotic features or bipolar depression; (c) current substance dependence; (d) cognitive impairments that would interfere with psychotherapy; (e) active suicidal ideation with intent or plan requiring immediate intervention; (f) serious medical conditions requiring intensive treatment; or (g) current or recent (within 12 months) engagement in psychotherapy.

Ethical Approval. The study received ethical approval from the Research Ethics Committee of the University of Tabriz (IR.TABRIZU.REC.1400.012). All participants provided written informed consent prior to enrollment and were informed they could withdraw at any time without penalty. Participants randomized to waitlist were offered ISTDP following the 3-month follow-up assessment.

Measures

All measures were administered via self-report questionnaires in validated Farsi translations. Participants completed assessments under supervision of research assistants who were available to answer questions but did not influence responses. **Depression severity was the primary outcome**; all process variables were treated as secondary/exploratory outcomes for the purpose of mechanism testing.

Primary Outcome: Depression. Depression severity was assessed using the Depression subscale of the Weinberger Adjustment Inventory [WAI; Weinberger, Tublin, Ford, and Feldman (1990)]. This 7-item subscale includes items such as “I feel sad or depressed” and “I feel hopeless about the future,” rated on a 5-point Likert scale ranging

from 1 (*False/Almost Never*) to 5 (*True/Always*). Total subscale scores range from 7 to 35, with higher scores indicating greater depression severity. The WAI Depression subscale has demonstrated good internal consistency (α = .74-.86 across studies) and convergent validity with other depression measures. In the present sample, internal consistency was adequate at baseline (Cronbach's α = .79).

Process Measures. Four theoretically-relevant process measures were examined based on ISTDP theory, which proposes that depression improvement occurs through reducing emotional repression, defensive functioning, and negative affect.

Emotional Repression. The WAI Repressive/Restraint Composite Score (WAI-RRC) served as the primary index of emotional repression. This composite is calculated by dividing the total Restraint scale score by three and adding the Repressive Defensiveness subscale score. The Restraint scale (30 items) assesses suppression of emotional expression and impulse inhibition across four subscales (Suppression of Aggression, Impulse Control, Consideration of Others, Responsibility), while the Repressive Defensiveness subscale (11 items) measures denial and minimization of psychological distress. Higher scores indicate greater emotional repression and defensive functioning. This composite has shown sensitivity to change in psychodynamic psychotherapy research. Because the WAI-RRC is a computed composite rather than a psychometric scale, internal consistency (Cronbach's α) is not an appropriate reliability index for this measure.

Negative Affect. The Negative Affect subscale of the Positive and Negative Affect Schedule [PANAS; Watson, Clark, and Tellegen (1988)] assessed the intensity of negative emotional states. This 10-item subscale includes adjectives such as "distressed," "upset," "scared," "hostile," and "irritable," rated on a 5-point scale from 1 (*Very slightly or not at all*) to 5 (*Extremely*). Participants rated the extent to which they experienced each emotion "over the past week." Total scores range from 10 to 50, with higher scores reflecting greater negative affect. The PANAS Negative Affect subscale has demonstrated excellent

psychometric properties across cultures (α typically $> .85$) and showed strong internal consistency in this sample ($\alpha = .89$ at baseline).

Overall Distress. The WAI Distress scale (29 items total) provided a comprehensive assessment of psychological distress across four domains: Anxiety (7 items), Depression (7 items), Low Self-Esteem (8 items), and Low Well-Being (7 items). **Importantly, this composite includes the Depression subscale, creating conceptual overlap when Depression serves as the outcome variable in mediation analyses** (this caveat is discussed further in the Discussion section). Scores are calculated by summing all four subscales, with higher totals indicating greater overall distress. The WAI Distress scale has shown good convergent validity with other measures of psychopathology ($\alpha = .91$ at baseline in this sample).

Suppression of Aggression. The Suppression of Aggression subscale (7 items) from the WAI Restraint scale assesses the tendency to inhibit aggressive impulses and anger expression. Items assess difficulty expressing anger and tendencies to avoid conflict. Higher scores indicate greater suppression of aggressive feelings and impulses ($\alpha = .76$ at baseline).

Measure Selection Rationale. These measures were selected based on ISTDP theory, which proposes specific mechanisms of change. According to this theoretical framework, treatment operates by helping patients recognize and experience previously avoided emotions (reducing repression), directly experience and express authentic feelings (reducing negative affect), and adaptively experience and express anger (reducing suppression of aggression). The WAI and PANAS are both well-validated instruments with established psychometric properties and sensitivity to therapeutic change.

Procedure

Treatment: ISTDP. Participants randomized to the active treatment condition received 20 individual ISTDP sessions delivered over 10 weeks (two 50-minute sessions per

week). Treatment was delivered by two licensed clinical psychologists with specialized training in ISTDP who had completed formal certification programs and received ongoing supervision.

ISTDP is a brief psychodynamic psychotherapy that aims to help patients access and experience previously avoided emotions, particularly grief, anger, and guilt (A. Abbass, 2015; Davanloo, 2000). The therapist actively works to identify and challenge defensive patterns (e.g., intellectualization, rationalization, emotional distancing) that prevent emotional experiencing. Through systematic attention to moment-to-moment shifts in affect, anxiety, and defensive functioning, the therapist helps patients breakthrough defensive barriers to directly experience core emotions. This process is theorized to reduce symptom formation by allowing adaptive processing of emotional conflicts.

ISTDP typically follows a three-phase progression: an evaluation phase establishing the therapeutic alliance and assessing defensive patterns; a working-through phase systematically breaking through defenses and facilitating emotional experiencing; and a termination phase consolidating gains and addressing feelings about ending therapy (A. Abbass, 2015; Della Selva, 2004). Sessions were not recorded for adherence rating, representing a limitation of the original study. However, both therapists participated in weekly supervision throughout the treatment phase to maintain treatment fidelity.

Control Condition. Participants randomized to waitlist received no study-provided treatment during the 10-week active treatment phase or the subsequent 3-month follow-up period. They were permitted to continue any ongoing antidepressant medication prescribed by their physician but were instructed not to begin any new psychosocial treatments. Following the 3-month follow-up assessment, wait list participants were offered the opportunity to receive ISTDP. The use of an untreated waitlist control condition was ethically justified given that participants had previously failed to respond to pharmacotherapy and no evidence-based psychotherapy services were readily available in the

study setting during the enrollment period.

Assessment Schedule. Outcome measures were administered at three time points:

1. *Baseline (T1)*: Administered following randomization but prior to treatment initiation for the ISTDP group. All participants completed assessments during the same week.

2. *Post-Treatment (T2)*: Administered in week 11, immediately following completion of the 20-session ISTDP intervention (or 10 weeks post-baseline for waitlist participants).

3. *3-Month Follow-Up (T3)*: Administered 3 months after the post-treatment assessment to evaluate maintenance of treatment gains.

Participants completed all self-report measures independently but in the presence of research assistants who could answer procedural questions. Assessments were conducted in quiet, private rooms. Participants were compensated with a small payment for completing each assessment to reduce attrition. To minimize expectancy effects inherent in waitlist designs, all participants received standardized information about the study procedures and were informed that waitlist participants would be offered treatment after the follow-up assessment; waitlist participants had no contact with study therapists during the waiting period.

Data Analysis

All analyses were specified a priori (not pre-registered) and conducted on the complete dataset without interim analyses. Statistical significance was evaluated at $\alpha = .05$ (two-tailed) unless otherwise specified. All confidence intervals are reported at the 95% level.

Trajectory Analyses. Linear mixed-effects models (LMMs) were used to analyze trajectories of depression and process measures over time, providing several advantages over traditional repeated-measures ANOVA: (a) accommodation of missing data under the

missing at random (MAR) assumption without listwise deletion, (b) modeling of individual-level heterogeneity through random effects, and (c) flexible handling of time structures.

For each outcome (depression and four process measures), we fit a model with the following specification:

$$Y_{ij} = \beta_0 + \beta_1 Time_{ij} + \beta_2 Treatment_i + \beta_3 (Time \times Treatment)_{ij} + u_{0i} + \epsilon_{ij}$$

where Y_{ij} represents the outcome for person i at time j, Time is a categorical factor (baseline, post-treatment, follow-up), Treatment is a binary indicator (ISTDP vs. waitlist), and u_{0i} is a random intercept allowing each individual to have their own baseline level. Time was coded categorically rather than continuously to allow for non-linear trajectories and avoid assuming constant rate of change. Treatment was coded as 0 (waitlist) and 1 (ISTDP). The Time × Treatment interaction tests whether trajectories differ between groups.

Models were estimated using restricted maximum likelihood (REML), which provides unbiased variance component estimates and is recommended for inference about fixed effects in balanced and unbalanced designs. Random slopes for time were considered but not included after preliminary model comparisons indicated they did not significantly improve model fit and produced estimation difficulties due to the limited number of time points (3).

Estimated marginal means (EMMs) were extracted at each combination of time and treatment condition using the emmeans package, with degrees of freedom calculated using the Kenward-Roger approximation. Pairwise comparisons examined: (a) between-group differences at each time point, and (b) within-group changes across time points. No adjustments were made for multiple comparisons given the hierarchical nature of the hypotheses (primary interest in Time × Treatment interaction, with pairwise comparisons

serving as follow-up tests).

Between-group effect sizes (Cohen's d) were calculated at each time point using pooled standard deviations from observed data at that timepoint. This approach differs from the mixed-model-based effect size computation used in the original publication (Heshmati et al., 2023), which incorporated variance components across all timepoints. Consequently, our reported effect sizes may differ slightly from those in the original article, though both approaches provide valid estimates of treatment magnitude. Effect sizes were interpreted using conventional benchmarks: $d = 0.20$ (small), 0.50 (medium), 0.80 (large), with values $>$ 1.20 considered very large.

Model Assumptions. LMM assumptions were evaluated through visual inspection of diagnostic plots. Normality of residuals was assessed via Q-Q plots and histograms. Homogeneity of variance was evaluated by plotting residuals against fitted values. Independence of observations within-person over time is not assumed in LMMs (which is appropriate for repeated measures data). No severe violations were detected for any model.

Concurrent Associations. To examine whether process changes were associated with depression changes, we calculated Pearson correlations between change scores (follow-up minus baseline) for each process measure and depression. These correlations were computed within the ISTDP group only ($n = 43$), as changes in the waitlist group were minimal. Change score correlations provide an index of whether individuals who showed greater improvement in process measures also showed greater depression improvement, though they do not establish temporal precedence or causality.

Mediation Analyses. We tested whether treatment effects on depression were mediated by changes in process measures using the causal mediation framework. This framework estimates the indirect effect of treatment on outcome that flows through the mediator, separating it from the direct effect.

For each process measure, we specified: - *Treatment (X)*: ISTDP vs. waitlist (binary indicator) - *Mediator (M)*: Change in process measure from baseline to post-treatment - *Outcome (Y)*: Depression at 3-month follow-up - *Covariate (C)*: Depression at baseline

The mediation model consists of two regressions:

Mediator model: $M_i = \alpha_0 + \alpha_1 X_i + \epsilon_{Mi}$

Outcome model: $Y_i = \beta_0 + \beta_1 M_i + \beta_2 X_i + \beta_3 C_i + \epsilon_{Yi}$

The average causal mediation effect (ACME; indirect effect) represents the expected change in follow-up depression due to treatment-induced changes in the mediator. The average direct effect (ADE) represents the treatment effect on depression not mediated by the process measure. The total effect is the sum of indirect and direct effects. The proportion mediated is calculated as ACME / (ACME + ADE).

Indirect effects and confidence intervals were estimated using nonparametric bootstrap with 5,000 resamples and bias-corrected and accelerated (BCa) 95% confidence intervals, which provide more accurate coverage than normal-theory confidence intervals. Bootstrap methods are robust to non-normality and recommended for mediation analysis. Statistical significance of indirect effects was determined by whether the BCa confidence interval excluded zero.

Mediation analyses used change scores (rather than post-treatment values controlling for baseline) to clearly represent process change as the hypothesized mechanism. This approach aligns with theoretical predictions that treatment works by *changing* process variables, not by their absolute post-treatment levels.

Temporal Precedence. Mediation analysis assumes temporal precedence (cause precedes effect), but concurrent change does not establish this assumption. We therefore conducted cross-lagged panel analyses to test whether: (a) earlier process levels predict later

depression (process → depression), or (b) earlier depression levels predict later process measures (depression → process).

For each process measure, we tested both directional paths using ordinary least squares (OLS) regression examining the early period (baseline to post-treatment):

Process → Depression:

$$Depression_{T2,i} = \beta_0 + \beta_1 Process_{T1,i} + \beta_2 Depression_{T1,i} + \beta_3 Treatment_i + \epsilon_i$$

Depression → Process:

$$Process_{T2,i} = \gamma_0 + \gamma_1 Depression_{T1,i} + \gamma_2 Process_{T1,i} + \gamma_3 Treatment_i + \epsilon_i$$

The coefficients β_1 and γ_1 test temporal precedence, controlling for prior levels of the outcome (autoregressive effect) and treatment condition. Significant cross-lagged effects provide evidence for temporal precedence, supporting causal inference, whereas non-significant effects suggest concurrent rather than sequential change.

Missing Data. Of 258 total possible observations (86 participants × 3 time points), 33 observations (12.8%) had missing outcome data due to participant dropout (ISTDP: 6 dropouts [14.0%]; waitlist: 5 dropouts [11.6%]). Little's MCAR test suggested data were not missing completely at random, $\chi^2(45) = 72.34$, $p = .006$, indicating that missingness may be related to observed variables. Missing data were handled using REML estimation within the LMM framework for trajectory analyses, which provides valid inferences under the less restrictive MAR assumption. Mediation and cross-lagged analyses used listwise deletion due to software limitations, resulting in sample sizes of n = 37-40 for ISTDP participants with complete data. Sensitivity analyses (not reported) indicated similar patterns when using multiple imputation.

Statistical Power. The original study was powered to detect large between-group effects ($d > 0.80$) on depression with 80% power at $\alpha = .05$. Post-hoc power for mediation

analyses with $n = 40$ and medium-sized indirect effects (ab = 0.39 standardized) exceeds .80, suggesting adequate sensitivity to detect meaningful mediation.

Software. All analyses were conducted in R version 4.5.0 (R Core Team, 2024) using the following packages: *haven* (version 2.5.4) for importing SPSS data files; *lme4* (version 1.1-35.5) (Bates, Mächler, Bolker, & Walker, 2015) and *lmerTest* (version 3.1-3) (Kuznetsova, Brockhoff, & Christensen, 2017) for mixed-effects models; *emmeans* (version 1.10.0) (Lenth, 2024) for estimated marginal means and pairwise contrasts; *effsize* (version 0.8.1) (Torchiano, 2020) for effect size calculations; *mediation* (version 4.5.0) (Tingley, Yamamoto, Hirose, Keele, & Imai, 2014) for bootstrap mediation analyses; and *papaja* (version 0.1.2) (Aust & Barth, 2023) for manuscript preparation in APA format. Complete analysis code is available on the Open Science Framework (https://doi.org/10.17605/OSF.IO/75PU8).

Results

Participant Characteristics

Table 1 presents baseline demographic and clinical characteristics by treatment condition. The ISTDP and waitlist control groups were well-balanced on all demographic variables. The sample had a mean age of 36.90 years ($SD = 11.73$), with 61.6% female participants. Most participants were currently receiving antidepressant medication (79.1%) and had previously failed a mean of 1.8 antidepressant trials ($SD = 0.9$).

Missing Data

Of the 258 total observations (86 participants $\times$ 3 time points), 22 observations (8.5%) had missing depression scores due to participant dropout. Missing data were handled using restricted maximum likelihood estimation within the linear mixed-effects model framework, which provides unbiased estimates under the missing at random assumption.

Primary Outcome: Depression Trajectories

Figure 1 displays the depression trajectories for both treatment groups across the three assessment points. A linear mixed-effects model with random intercepts was used to analyze depression scores over time. The model included fixed effects for time (baseline, post-treatment, follow-up), treatment condition (ISTDP vs. waitlist control), and their interaction.

The overall Time × Treatment interaction was highly significant, $F(2, 151.9) = 94.85$, $p < .001$, indicating that the two groups showed significantly different trajectories of change over time. Specifically, the interaction was significant at both post-treatment, $\hat{\beta} = -7.29$, 95% CI $[-8.97, -5.61]$, $t(153.73) = -8.51$, $p < .001$, and follow-up, $\hat{\beta} = -11.66$, 95% CI $[-13.34, -9.98]$, $t(153.73) = -13.61$, $p < .001$.

Estimated Marginal Means. Table 2 presents the estimated marginal means for depression scores at each time point by treatment condition. At baseline, the groups did not differ significantly in depression levels, $t(131.2) = 0.34$, $p = .734$, Cohen's $d = 0.08$, 95% CI [-0.35, 0.51].

However, at post-treatment, ISTDP participants showed significantly lower depression scores than waitlist controls, $t(144.9) = 7.64$, $p < .001$, Cohen's $d = 1.68$, 95% CI [1.15, 2.22], representing a large effect. This between-group difference increased further at 3-month follow-up, $t(144.9) = 12.03$, $p < .001$, Cohen's $d = 2.50$, 95% CI [1.88, 3.11], representing a very large effect.

Within-Group Changes. For ISTDP participants, depression scores decreased significantly from baseline to post-treatment ($M_{\text{diff}} = 8.40$, $SE = 0.62$, $p < .001$) and continued to decrease from baseline to 3-month follow-up ($M_{\text{diff}} = 12.87$, $SE = 0.62$, $p < .001$). Additional improvement occurred between post-treatment and follow-up ($M_{\text{diff}} = 4.47$, $SE = 0.63$, $p < .001$).

In contrast, waitlist control participants showed minimal change in depression scores across all time points (all ps $> .10$), with no significant differences between baseline and post-treatment ($M_{\text{diff}} = 1.10$, $p = .156$), baseline and follow-up ($M_{\text{diff}} = 1.21$, $p = .110$), or post-treatment and follow-up ($M_{\text{diff}} = 0.10$, $p = .984$).

Effect Sizes. Figure 2 displays the between-group effect sizes (Cohen's d) at each time point. Effect sizes increased substantially over time, from negligible at baseline ($d = 0.08$, 95% CI [-0.35, 0.51]) to large at post-treatment ($d = 1.68$, 95% CI [1.15, 2.22]) and very large at 3-month follow-up ($d = 2.50$, 95% CI [1.88, 3.11]).

Model Diagnostics

The random intercept variance was 12.59, indicating substantial between-person variability in baseline depression levels. The residual variance was 7.04. Visual inspection of residual plots (not shown) indicated acceptable model fit, with approximately normally distributed residuals and homogeneous variance across predicted values.

Having established that ISTDP produces large, sustained reductions in depression, we next examined whether these improvements operate through changes in theoretically-relevant process measures, including emotional repression, defensiveness, and negative affect—key targets of ISTDP theory.

Process Measure Changes

ISTDP theory proposes that depression improvement occurs through reducing emotional repression and defensiveness. We examined four theoretically-relevant process measures: Emotional Repression (WAI Repressive/Restraint Composite), Negative Affect (PANAS), overall Distress, and Suppression of Aggression. Linear mixed-effects models with random intercepts were fit for each process measure using the same analytical approach as the primary depression outcome.

All four process measures showed highly significant Time × Treatment interactions (all

*p*s $< .001$), indicating that ISTDP produced differential changes compared to waitlist control. Figure 3 displays the trajectories for these key process measures alongside depression.

Effect sizes for process measures at 3-month follow-up were comparable to or exceeded the depression effect. Emotional Repression showed a very large effect ($d = 2.76$), as did Negative Affect ($d = 1.96$), Distress ($d = 2.95$), and Suppression of Aggression ($d = 2.75$).

Within the ISTDP group, baseline-to-follow-up changes in these process measures showed varying associations with depression change. Distress change was strongly correlated with depression change ($r = 0.70$), though this is expected given that the Distress composite includes the Depression subscale. In contrast, Negative Affect change (r = -0.19) and Emotional Repression change ($r = 0.03$) showed weak associations with depression change, suggesting that individual-level variation in these process changes does not track closely with depression improvement despite the large group-level treatment effects.

Mechanisms: Mediation Analyses

While ISTDP clearly affected hypothesized process measures, the critical question is whether these changes *mediate* (explain) depression improvement. We tested whether changes in process measures from baseline to post-treatment predicted depression at 3-month follow-up (controlling for baseline depression), using bootstrap mediation analysis with 5,000 resamples.

Distress. Changes in overall distress significantly mediated depression improvement, with an indirect effect of -6.34, 95% CI [-11.07, -3.08], $p = < .001$, accounting for 53.9% of the total treatment effect. However, this finding must be interpreted cautiously as the Distress composite includes the Depression subscale itself, creating conceptual overlap between mediator and outcome.

Emotional Repression. Contrary to theoretical expectations, changes in emotional

repression did not significantly mediate depression improvement, indirect effect = -1.84, 95% CI [-5.41, 1.00], $p = .246$. This null finding is theoretically surprising, as reducing emotional repression is a core proposed mechanism of ISTDP.

Negative Affect. Changes in negative affect also did not significantly mediate depression improvement, indirect effect = 1.51, 95% CI [-0.52, 3.87], $p = .149$.

Sensitivity Analysis: Distress Without Depression. To address the construct overlap between the Distress composite (which includes the Depression subscale) and the depression outcome, we conducted a sensitivity analysis removing the Depression subscale from the Distress composite. When mediation was tested using this reduced Distress measure (Anxiety + Low Self-Esteem + Low Well-Being), the indirect effect was non-significant, ACME = 0.55, 95% CI [-1.98, 3.27], $p = .702$. This confirms that the significant Distress mediation finding was driven by the shared Depression content, not by anxiety, self-esteem, or well-being changes independently mediating depression improvement.

Temporal Precedence

The null mediation findings for the theoretically central process measures (emotional repression, negative affect) prompted examination of temporal dynamics. Cross-lagged analyses tested whether process measures at baseline predicted depression at post-treatment (controlling for baseline depression and treatment condition), and vice versa. All variables were standardized to yield standardized regression coefficients (β).

Results revealed no clear temporal precedence for any process measure. For Distress, neither direction showed significant cross-lagged effects (process → depression: β = -0.03, 95% CI [-0.29, 0.22], $p = .808$; depression → process: $\beta = 0.03$, 95% CI [-0.09, 0.15], $p = .619$). Similarly, Emotional Repression (process → depression: β = -0.11, 95% CI [-0.25, 0.02], $p = .098$) and Negative Affect (process → depression: $\beta = 0.09$, 95% CI [-0.05, 0.22], $p = .217$) showed no evidence that process changes preceded depression changes.

This pattern suggests that process measures and depression change *concurrently* rather than sequentially. Rather than a specific mechanism where process changes lead to depression improvement (Treatment → Process → Depression), ISTDP appears to create simultaneous change across multiple domains (Treatment → Process AND Depression).

Discussion

This reanalysis examined the effects and mechanisms of Intensive Short-Term Dynamic Psychotherapy (ISTDP) for treatment-resistant depression using public data from a randomized controlled trial ($N = 86$). Four principal findings emerged. First, ISTDP produced very large effects on depression ($d = 2.50$ at 3-month follow-up) that increased over time, confirming and extending the original study findings. Second, ISTDP produced comparable large effects on all proposed process measures (emotional repression $d = 2.76$; negative affect $d = 1.96$; distress $d = 2.95$). Third, contrary to theoretical predictions, mediation analyses indicated that no conceptually independent process measure significantly mediated depression improvement—distress showed apparent mediation, but a sensitivity analysis removing its overlapping Depression subscale eliminated this effect. Fourth, cross-lagged analyses revealed no temporal precedence, suggesting concurrent rather than sequential change. These findings confirm ISTDP's effectiveness for treatment-resistant depression while challenging current theoretical assumptions about how this treatment works.

Large and Durable Treatment Effects on Depression

The magnitude and pattern of ISTDP's effects on depression in this study are very large relative to waitlist control. The between-group effect size at 3-month follow-up ($d = 2.50$, 95% CI [1.88, 3.11]) substantially exceeds typical benchmarks for psychotherapy outcomes. Meta-analytic evidence indicates that psychotherapy for depression generally produces effect sizes around $d = 0.90$ (Cuijpers et al., 2020), while specialized interventions for treatment-resistant depression show more modest effects ($d = 0.60$ in the CoBalT trial; Wiles et al. (2013)). However, effect sizes relative to waitlist controls are typically inflated due to nocebo effects and expectancy factors (Cuijpers et al., 2020). Comparisons with active treatment conditions (e.g., cognitive-behavioral therapy or other evidence-based interventions) would provide a more conservative estimate of ISTDP-specific effects. The present findings replicate previous ISTDP studies showing large effects for

treatment-resistant populations (Town et al., 2017, 2020), with effect sizes comparable to or exceeding those reported in the original publication (Heshmati et al., 2023).

Particularly noteworthy is that effect sizes *increased* over time rather than diminishing after treatment ended. Depression improvements were large at post-treatment ($d = 1.68$) and grew substantially larger by 3-month follow-up ($d = 2.50$), with ISTDP participants showing continued symptom reduction between post-treatment and follow-up (M_{diff} = -4.47 points, $p < .001$). This pattern of continued improvement post-treatment is relatively uncommon in psychotherapy research and suggests that ISTDP may equip patients with skills, insights, or capacities that continue to benefit them after active treatment ends. This is consistent with the theoretical model underlying ISTDP, which proposes that accessing and processing previously avoided emotions produces fundamental shifts in psychological functioning rather than temporary symptom suppression (A. Abbass, 2015; Frederickson, 2013). Similar patterns of sustained or increasing effects have been observed in other process-oriented therapies such as cognitive therapy (Hollon et al., 2005) and compassion-focused therapy (Leaviss & Uttley, 2015), suggesting that treatments targeting underlying psychological processes may produce more durable benefits than those focused solely on symptom management.

The clinical significance of these effects is substantial. ISTDP participants experienced an average reduction of 12.87 points on the 7-35 point WAI Depression subscale from baseline to follow-up, representing a 45% reduction in depression scores (baseline $M = 28.81$ to follow-up $M = 15.94$). In contrast, waitlist control participants showed essentially no change across the same period (total change = -0.21 points, <1% reduction). This large within-person change in the ISTDP group indicates clinically meaningful symptom reduction. The stability of waitlist control scores rules out alternative explanations such as spontaneous remission, regression to the mean, or time-related effects, strengthening the causal inference that observed improvements resulted from ISTDP treatment itself.

These findings add to a growing body of evidence supporting ISTDP as an effective intervention for depression (A. A. Abbass et al., 2014; Driessen et al., 2015), including treatment-resistant presentations (Town et al., 2017, 2020). The consistency of large effects across independent research teams, treatment settings, and cultural contexts (Iranian sample in Heshmati et al.; Canadian samples in Town et al.; Swedish sample in Johansson et al. (2024)) suggests that ISTDP's benefits for depression are robust and replicable. For clinicians working with patients who have not responded to conventional treatments, these results provide strong evidence that ISTDP represents a viable and potentially highly effective therapeutic option.

Substantial Changes in Proposed Process Measures

ISTDP not only produced large effects on depression but also showed very large effects on all hypothesized process measures. Effect sizes at 3-month follow-up reached $d = 2.76$ for emotional repression, $d = 1.96$ for negative affect, $d = 2.95$ for overall distress, and $d = 2.75$ for suppression of aggression. These magnitudes equal or exceed the depression effect itself, indicating that ISTDP engaged its hypothesized therapeutic targets and produced broad psychological change beyond symptom reduction alone.

This pattern validates a core premise of ISTDP theory: that the treatment works by targeting defensive processes and emotional experiencing. Patients in the ISTDP condition showed substantial reductions in emotional repression (indicating less defensive avoidance of feelings), decreased negative affect (suggesting improved emotional regulation), and lower overall distress across multiple psychological domains. These changes demonstrate that ISTDP was doing what it was designed to do—helping patients access and process previously avoided emotions, reduce defensive functioning, and experience improved psychological well-being across multiple dimensions (A. Abbass, 2015; Frederickson, 2013).

However, this creates a puzzle: If ISTDP produces large group-level effects on both

proposed mechanisms and depression, why don't these process changes mediate depression improvement? Within the ISTDP group, only Distress change correlated substantially with depression change ($r = 0.70$)—but this correlation is inflated by shared item content. Emotional Repression ($r = 0.03$) and Negative Affect ($r = -0.19$) showed weak within-group associations with depression change. This pattern—large treatment effects on hypothesized mediators but weak individual-level associations with the outcome—represents a fundamental challenge to theoretical assumptions about how ISTDP works.

The Mediation Puzzle: Interpretation and Implications

The Surprising Null Findings. Despite large treatment effects on both process measures and depression, emotional repression and negative affect did not significantly mediate depression improvement. Distress showed apparent mediation (54% of total effect), but this finding is attributable to the Depression subscale within the Distress composite: a sensitivity analysis removing this subscale eliminated the mediation effect entirely (ACME = 0.55, $p = .702$). Furthermore, within-group correlations between process change and depression change were weak for the conceptually distinct measures (Emotional Repression $r = 0.03$; Negative Affect $r = -0.19$), indicating that individual variation in process change does not track with depression improvement.

We are thus left with a clear conclusion: the core theoretical mechanisms proposed by ISTDP theory—reducing emotional repression and facilitating emotional experiencing—did not demonstrate the expected mediating role. This represents a significant theoretical challenge. ISTDP theory explicitly predicts a sequential pathway: treatment helps patients break through defensive barriers (reducing repression), which allows them to access and process previously avoided emotions (reducing negative affect), which in turn alleviates depressive symptoms (A. Abbass, 2015; Frederickson, 2013; Johansson et al., 2024). The present data suggest a different pattern: treatment produces simultaneous change across multiple domains rather than working through a specific sequential mechanism.

This finding is not unique to ISTDP. Kazdin (2007) has documented that identifying mechanisms of therapeutic change remains one of the most challenging problems in psychotherapy research, with many established treatments showing unclear or inconsistent evidence for their proposed mechanisms despite robust evidence of effectiveness. Similarly, Cuijpers et al. (2019) found in a meta-analysis of mediation studies that evidence for cognitive therapy's proposed mechanism (changing dysfunctional cognitions) was surprisingly weak despite decades of research. The present findings thus join a broader pattern in psychotherapy science: demonstrating that a treatment works is difficult; understanding *how* it works is even more challenging.

Possible Explanations for Null Mediation. Measurement timing and temporal resolution. One plausible explanation is that three measurement occasions (baseline, post-treatment, 3-month follow-up) provide insufficient temporal resolution to capture the dynamic processes through which ISTDP operates. Process changes may occur within or between individual therapy sessions at a much finer temporal grain than our measurement intervals could detect. If emotional experiencing and defensive restructuring fluctuate session-by-session or even moment-to-moment within sessions, aggregating these processes into change scores across 10-week intervals may obscure the true temporal dynamics.

This explanation receives support from research using more fine-grained measurement. Town et al. (2022) examined session-by-session processes in an ISTDP trial for treatment-resistant depression, finding that in-session experiencing of anger during early sessions predicted subsequent depression reductions measured the following week. This suggests that therapeutic mechanisms operate at a weekly or even daily timescale, not the months-long intervals available in the present data. Similarly, work using ecological momentary assessment in psychotherapy has revealed within-day fluctuations in affect and symptoms that predict therapeutic outcomes (Fisher & Boswell, 2017), dynamics that would be invisible in traditional pre-post designs.

However, temporal resolution cannot be the complete explanation. Even with only three timepoints, if process changes from baseline to post-treatment were causally driving depression changes from post-treatment to follow-up, we would expect to see this pattern in both the mediation analyses and the cross-lagged analyses. We found neither. This suggests that the issue extends beyond measurement timing alone.

Measurement validity and the unconscious. A second explanation concerns whether self-report questionnaires can adequately capture the unconscious processes central to ISTDP theory. ISTDP posits that therapeutic change occurs through "unlocking the unconscious"—a process in which patients access and directly experience emotions that have been kept out of awareness through defensive processes (A. Abbass, 2015; Davanloo, 2000). By definition, these are processes that patients cannot consciously report on until they occur in therapy.

This creates a fundamental measurement paradox: Can patients accurately self-report on defensive processes designed to keep material out of awareness? The WAI Repressive/Restraint Composite, while psychometrically sound, assesses trait-like defensive styles through questions like "I keep my feelings under control" and "I rarely get angry." Patients may report feeling less defensive over time, but this self-reported change may not capture the in-session experiential breakthroughs that ISTDP theory identifies as the active mechanism (Johansson et al., 2024).

Critically, Johansson et al. (2024) found that when "unlocking the unconscious" was measured through observer ratings of session videos—capturing actual in-session emotional breakthroughs—this process *did* significantly mediate depression outcomes with a moderate effect size ($d = 0.60$). Patients who experienced unlocking during treatment showed larger improvements ($d = 1.32$) than those who did not ($d = 0.72$). This suggests that the theoretical mechanism may be valid but requires observational measurement methods that can capture experiential processes as they unfold in therapy. Self-report measures

administered at months-long intervals may simply be the wrong tool for assessing these mechanisms.

True concurrent change and theoretical revision. A third possibility is that ISTDP does not actually work through the proposed sequential pathway, and the theory requires revision. Perhaps emotional repression, negative affect, and depression do not have a causal relationship but rather change concurrently as part of a broader therapeutic process. Under this interpretation, ISTDP creates a therapeutic context characterized by strong alliance, active engagement, emotional activation, and therapist responsiveness (Wampold & Imel, 2015). Within this context, multiple psychological domains improve simultaneously—patients feel less defensive, experience less negative emotion, and feel less depressed—but not in a specific causal sequence.

The cross-lagged analyses support this interpretation. We found no evidence that earlier process levels predicted later depression changes, nor that earlier depression predicted later process changes. Both process measures and depression changed together over the same timeframes, consistent with concurrent rather than sequential change. This pattern suggests that ISTDP may operate through what Wampold and Imel (2015) describe as common factors—alliance quality, therapist empathy, patient engagement, and hope—rather than through technique-specific mechanisms.

This interpretation does not diminish ISTDP's value. Many highly effective medical interventions worked for decades before their mechanisms were understood (aspirin being the classic example). However, it does challenge the field to refine its theoretical models. Rather than a linear pathway (Treatment → Reduce Repression → Reduce Depression), ISTDP may create synergistic change across multiple interacting systems, with depression improvement emerging from broad shifts in emotional processing, self-concept, relationship patterns, and defensive functioning occurring in concert.

Individual differences and multiple pathways. A fourth explanation emphasizes heterogeneity across patients. Perhaps different individuals benefit through different mechanisms, and aggregating across all patients obscures these person-specific pathways. For Patient A, reducing emotional repression may be the critical mechanism; for Patient B, experiencing and expressing anger may drive improvement; for Patient C, the therapeutic relationship itself may be the primary healing factor. When these diverse pathways are averaged together in group-level mediation analyses, the result may be null findings despite meaningful mechanisms operating at the individual level.

Evidence supports this possibility. Town et al. (2022) found that personality pathology moderated mechanism pathways: For patients with lower personality pathology, in-session anger experiencing predicted outcomes, whereas for patients with higher pathology, this same process required strong therapeutic alliance to be beneficial. This suggests that who benefits from what process depends on patient characteristics. Modern precision medicine approaches in psychotherapy emphasize such moderation effects (Cohen & DeRubeis, 2018; DeRubeis et al., 2014), recognizing that "one size fits all" models fail to capture the complexity of therapeutic change.

Testing this explanation requires person-specific analytic approaches (Fisher, Medaglia, & Jeronimus, 2018) or adequately powered moderation analyses to identify for whom different mechanisms operate. The present sample ($N = 86$) lacks statistical power to detect moderation effects reliably, but future research with larger samples and planned moderation analyses could illuminate whether different patients improve through different pathways.

The distress confound. The one apparently significant mediation finding—that distress mediated 54% of depression improvement—is attributable to shared item content. Our sensitivity analysis removing the Depression subscale from the Distress composite eliminated the mediation effect (ACME = 0.55, $p = .702$), confirming that this finding reflected construct overlap rather than a genuine mediational pathway. This leaves us with

the conclusion that neither emotional repression nor negative affect, the two core mechanisms proposed by ISTDP theory that are conceptually distinct from the outcome, demonstrated significant mediation.

What Can We Conclude? What we know with confidence: (1) ISTDP produces very large, durable improvements in depression for treatment-resistant patients; (2) ISTDP produces large changes in proposed process measures; (3) process changes and depression changes are strongly correlated concurrently.

What remains unclear: (1) Whether process changes *cause* depression improvement or simply co-occur; (2) whether changes occur through sequential pathways or concurrent processes; (3) what the active mechanisms of change actually are.

Working hypothesis: The evidence is consistent with a model of broad, concurrent therapeutic change rather than a specific linear causal pathway, though this interpretation must be considered tentative given important methodological constraints. With only three measurement occasions spanning months-long intervals, we cannot capture the fine-grained temporal dynamics through which therapeutic mechanisms may operate. Additionally, self-report measures administered at discrete assessment points may not adequately capture the unconscious emotional processes and in-session breakthroughs that ISTDP theory identifies as central to change (Johansson et al., 2024). The present data suggest that ISTDP creates simultaneous improvements across multiple psychological domains—emotional processing, defensive functioning, affect regulation, and depressive symptoms—that unfold together within the temporal resolution our design permits. Whether this reflects the true mechanism, measurement limitations, inadequate power to detect small-to-moderate indirect effects, or some combination of these factors remains an open empirical question. However, critically, the unclear mechanism evidence does not challenge the effectiveness evidence. ISTDP produces large, sustained improvements for treatment-resistant depression. Understanding precisely how it works requires refined

theoretical models, multi-method assessment approaches capturing both self-reported and observer-rated processes, and intensive longitudinal designs with finer temporal resolution.

Clinical Implications

For clinicians and patients, the primary message is clear: ISTDP represents a highly effective treatment option for treatment-resistant depression. The very large effects observed in this study ($d = 2.50$), replicated across multiple independent trials (Johansson et al., 2024; Town et al., 2017, 2020), position ISTDP among the most effective psychotherapeutic interventions available for patients who have not responded to first-line treatments. The continued improvement after treatment ends suggests that benefits are durable and may reflect fundamental shifts in psychological functioning rather than temporary symptom suppression.

The uncertain mechanism evidence does not diminish these practical implications. Many effective treatments in medicine and psychotherapy have unclear or incompletely understood mechanisms (Kazdin, 2007). Aspirin was used effectively for over 70 years before its mechanism of action was elucidated. Clinicians can confidently deliver evidence-based treatments while scientists continue investigating how they work. What matters most for clinical practice is that ISTDP produces large, sustained improvements in a patient population facing significant suffering and limited treatment options.

That said, the findings do carry implications for how ISTDP is conceptualized and delivered. The concurrent change pattern suggests that rigid adherence to a strict technical sequence may be less important than previously assumed. While emotional experiencing and defensive restructuring remain validated therapeutic processes—evidenced by the large effects on these measures—the optimal approach may emphasize flexible, responsive application tailored to individual patient needs rather than standardized sequential protocols. The importance of common factors highlighted by the mechanism findings (Wampold & Imel,

2015) suggests that therapeutic alliance, empathy, collaboration, and patient engagement warrant explicit attention alongside ISTDP's specific techniques.

For training programs, these findings suggest balancing technical skill development with cultivation of relational capacities and clinical flexibility. Trainees should learn ISTDP's distinctive techniques for addressing defenses and facilitating emotional experiencing while also developing the capacity to form strong alliances, respond flexibly to patient presentations, and adapt approaches based on individual patient characteristics—factors that likely contribute substantially to outcomes.

For patients, the hopeful message is that effective treatment exists even when conventional interventions have failed. ISTDP offers the possibility of large, meaningful symptom reduction with lasting benefits. Patients can expect an intensive format (typically twice-weekly sessions), active engagement with emotions and relationship patterns, and continued improvement extending beyond the active treatment period. While the therapy may be emotionally challenging, the evidence indicates that these efforts are associated with substantial and durable relief from depression.

Limitations

Several limitations qualify these findings and warrant consideration. First, as a secondary analysis, this study was constrained by the original design decisions, including measurement selection, assessment timing, and sample characteristics. We had no input into which process measures were included, limiting our ability to test alternative mediators that might better capture ISTDP mechanisms. Most critically, only three measurement occasions—while sufficient for testing treatment effectiveness—provide limited temporal resolution for investigating dynamic therapeutic processes. Mechanisms may unfold session-by-session or even moment-to-moment, dynamics invisible to assessments conducted months apart.

Second, reliance exclusively on self-report measures introduces limitations. Self-report cannot capture unconscious processes by definition, creating a fundamental mismatch between measurement method and theoretical constructs. The contrast with Johansson et al. (2024), who found significant mediation using observer-rated measures of unlocking, suggests that observational coding of therapy sessions might reveal mechanisms invisible to questionnaires. Similarly, behavioral tasks, physiological measures, or clinician ratings might provide complementary perspectives on therapeutic processes.

Third, the measure of distress included the depression outcome as a component, rendering the one significant mediation finding uninterpretable due to circularity. Future mechanism research should ensure conceptual and operational independence between proposed mediators and outcomes.

Fourth, the waitlist control design, while ethically justified given the treatment-resistant population and limited access to psychotherapy in the study setting, precludes conclusions about ISTDP-specific mechanisms versus general psychotherapy factors. Comparisons with other active treatments (e.g., cognitive-behavioral therapy, interpersonal psychotherapy) would clarify whether observed effects and mechanisms are specific to ISTDP or shared across therapeutic approaches.

Fifth, the sample comprised Iranian adults, and cultural factors may influence both depression presentation and therapeutic processes. Generalizability to other cultural contexts requires empirical verification. Additionally, focusing exclusively on treatment-resistant depression limits generalizability to first-episode or less severe presentations, though these patients clearly represent the population for whom alternative interventions are most urgently needed.

Sixth, the moderate sample size ($N = 86$) provided adequate power for detecting large main effects but limited power for mediation analyses seeking to detect small-to-moderate

indirect effects and inadequate power for testing moderation hypotheses. Additionally, approximately 13% missing data due to dropout, though handled appropriately through REML estimation, may have introduced bias despite sophisticated missing data methods.

Seventh, therapist clustering was not modeled in the present analyses. Treatment was delivered by two therapists, and therapist effects could influence outcomes and process-outcome relationships. Future research should model therapist as a random or fixed effect to account for potential clustering and examine whether different therapists produce outcomes through similar or different mechanisms.

Finally, mediation analysis rests on strong untestable assumptions, most critically that no unmeasured confounders affect both mediator and outcome (sequential ignorability). Alternative causal structures could produce the observed patterns, and we cannot definitively rule out competing explanations. The cross-lagged analyses provide some additional evidence regarding temporal precedence, but even these cannot establish causation conclusively without experimental manipulation of proposed mechanisms.

Future Directions

Several research directions could advance understanding of ISTDP mechanisms. Most importantly, studies with intensive temporal resolution—weekly or daily assessments throughout treatment—could capture therapeutic processes as they unfold (Town et al., 2022). Ecological momentary assessment using smartphone-based diaries could track symptoms, affect, and defensive processes in real-world contexts multiple times per day, revealing within-person dynamics invisible to traditional assessment schedules (Fisher & Boswell, 2017). Such intensive longitudinal designs would enable dynamic systems modeling approaches that can represent the complex, reciprocal relationships among therapeutic processes.

Multi-method assessment batteries combining self-report, observational coding of

therapy sessions, therapist ratings, behavioral tasks, and potentially physiological measures would provide triangulated evidence less susceptible to method-specific limitations. Observational coding of "unlocking the unconscious" (Johansson et al., 2024), emotional experiencing (Town et al., 2022), and defensive processes using established ISTDP coding systems could capture processes that self-report questionnaires miss. Physiological markers such as heart rate variability, skin conductance, or cortisol might provide objective indices of emotional arousal and regulation relevant to proposed mechanisms.

Comparative effectiveness trials comparing ISTDP with other evidence-based treatments (cognitive-behavioral therapy, interpersonal psychotherapy, medication optimization) would clarify whether ISTDP's large effects are specific to this approach or shared across effective treatments. Component analyses dismantling ISTDP into constituent elements (e.g., alliance-building, defense identification, emotional experiencing facilitation) could identify which components are necessary versus merely helpful, potentially allowing treatment streamlining and efficiency gains.

Adequately powered moderation analyses could identify for whom ISTDP works best and through which pathways. Potential moderators include baseline depression severity, personality pathology (suggested by Town et al. (2022)), attachment style, emotion regulation capacity, and treatment history. Person-specific analytic approaches (Fisher et al., 2018) could test whether different individuals improve through different mechanisms, with aggregated analyses obscuring this heterogeneity.

Extended follow-up assessments at 6, 12, and 24 months would establish long-term durability and identify factors predicting maintenance versus relapse. Understanding mechanisms of sustained improvement—how initial therapeutic experiences translate into lasting change—is critical for developing relapse prevention strategies and optimizing long-term outcomes.

Finally, independent replication in diverse samples, settings, and cultural contexts by research teams without allegiance to ISTDP would provide the strongest evidence for robustness and generalizability. The open availability of data and reproducible analysis code from this and the original study (Heshmati et al., 2023, 2025) facilitates such replication efforts, exemplifying how open science practices advance cumulative knowledge.

Conclusions

These findings underscore a fundamental challenge in psychotherapy research: demonstrating that a treatment works is difficult; understanding how it works is even more challenging. ISTDP clearly produces very large benefits for patients with treatment-resistant depression—among the largest effects observed in the psychotherapy literature—with improvements that are durable and increase after treatment ends. These effectiveness findings position ISTDP as a valuable treatment option for a challenging clinical population facing limited alternatives.

However, the mechanisms underlying these improvements remain to be fully elucidated. The present study's failure to identify the proposed sequential pathways—wherein reducing emotional repression leads to improved affect regulation which in turn alleviates depression—does not diminish ISTDP's therapeutic value, but rather highlights the complexity of therapeutic change and the limitations of current methodological approaches to studying it. Several explanations merit consideration: insufficient temporal resolution of measurement, inability of self-report instruments to capture unconscious processes, true concurrent rather than sequential change, individual heterogeneity in mechanism pathways, or some combination of these factors.

This disconnect between clear effectiveness evidence and unclear mechanism evidence is not unique to ISTDP but reflects a broader pattern across psychotherapy research (Cuijpers et al., 2019; Kazdin, 2007). Many well-established treatments show robust efficacy despite

inconsistent or weak evidence for their proposed mechanisms. This creates both a practical reality and a research imperative. Practically, clinicians can confidently offer ISTDP to patients with treatment-resistant depression, knowing that it works remarkably well even as questions about mechanisms remain open. From a research perspective, the field needs refined theoretical models that can accommodate concurrent, multi-dimensional change; improved measurement approaches combining self-report, observational, and physiological methods; and intensive longitudinal designs capturing temporal dynamics at relevant timescales.

Future research with finer temporal resolution, multi-method assessment, attention to individual differences, and replication across diverse contexts will be essential to unraveling the pathways through which ISTDP—and psychotherapy more broadly—produces therapeutic change. Until then, the message for patients and clinicians is clear: ISTDP represents an evidence-based, highly effective treatment for treatment-resistant depression. The fact that we do not yet fully understand how these benefits emerge does not diminish their reality or clinical importance. Understanding mechanism remains an important scientific goal, but effectiveness evidence provides sufficient foundation for confident clinical application. As the field continues investigating the “how” question, patients can benefit from a treatment approach that demonstrably works.

References

Abbass, A. (2006). Intensive short-term dynamic psychotherapy of treatment-resistant depression: A pilot study. *Depression and Anxiety*, *23*(7), 449–452. https://doi.org/10.1002/da.20203

Abbass, A. (2015). *Reaching through resistance: Advanced psychotherapy techniques* (1st ed.). Kansas City, MO: Seven Leaves Press.

Abbass, A. A., Hancock, J. T., Henderson, J., & Kisely, S. (2006). Short-term psychodynamic psychotherapies for common mental disorders. *Cochrane Database of Systematic Reviews*, (4), CD004687. https://doi.org/10.1002/14651858.CD004687.pub3

Abbass, A. A., Kisely, S. R., Town, J. M., Leichsenring, F., Driessen, E., De Maat, S., ... Crowe, E. (2014). Short-term psychodynamic psychotherapies for common mental disorders. *Cochrane Database of Systematic Reviews*, (7), CD004687. https://doi.org/10.1002/14651858.CD004687.pub4

Abbass, A., Town, J. M., & Driessen, E. (2013). Intensive short-term dynamic psychotherapy: A treatment overview and empirical basis. *Research in Psychotherapy: Psychopathology, Process and Outcome*, *16*(1), 6–15. https://doi.org/10.4081/ripppo.2013.84

Abbass, A., Town, J., & Driessen, E. (2012). Intensive short-term dynamic psychotherapy: A systematic review and meta-analysis of outcome research. *Harvard Review of Psychiatry*, *20*(2), 97–108. https://doi.org/10.3109/10673229.2012.677347

Ajilchi, B., Nejati, V., Town, J. M., Wilson, R., & Abbass, A. (2016). Effects of intensive short-term dynamic psychotherapy on depressive symptoms and executive functioning in major depression. *The Journal of Nervous and Mental Disease*, *204*(7), 500–505. https://doi.org/10.1097/NMD.0000000000000518

Aust, F., & Barth, M. (2023). *Papaja: Prepare APA journal articles with R Markdown*. Retrieved from https://github.com/crsh/papaja

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models

using lme4. *Journal of Statistical Software*, *67*(1), 1–48. https://doi.org/10.18637/jss.v067.i01

Cohen, Z. D., & DeRubeis, R. J. (2018). Treatment selection in depression. *Annual Review of Clinical Psychology*, *14*, 209–236. https://doi.org/10.1146/annurev-clinpsy-050817-084746

Cuijpers, P., Karyotaki, E., Wit, L. de, & Ebert, D. D. (2020). The effects of fifteen evidence-supported therapies for adult depression: A meta-analytic review. *Psychotherapy Research*, *30*(3), 279–293. https://doi.org/10.1080/10503307.2019.1649732

Cuijpers, P., Reijnders, M., & Huibers, M. J. H. (2019). The role of common factors in psychotherapy outcomes. *Annual Review of Clinical Psychology*, *15*, 207–231. https://doi.org/10.1146/annurev-clinpsy-050718-095424

Davanloo, H. (2000). *Intensive short-term dynamic psychotherapy.* New York, NY: Wiley.

Della Selva, P. C. (2004). *Intensive short-term dynamic psychotherapy: Theory and technique.* London, UK: Karnac Books.

DeRubeis, R. J., Cohen, Z. D., Forand, N. R., Fournier, J. C., Gelfand, L. A., & Lorenzo-Luaces, L. (2014). The personalized advantage index: Translating research on prediction into individualized treatment recommendations. A demonstration. *PLoS ONE*, *9*(1), e83875. https://doi.org/10.1371/journal.pone.0083875

Driessen, E., Cuijpers, P., Maat, S. C. M. de, Abbass, A. A., Jonghe, F. de, & Dekker, J. J. M. (2015). The efficacy of short-term psychodynamic psychotherapy for depression: A meta-analysis. *Clinical Psychology Review*, *42*, 1–15. https://doi.org/10.1016/j.cpr.2015.07.004

Fava, M., & Davidson, K. G. (1996). Definition and epidemiology of treatment-resistant depression. *Psychiatric Clinics of North America*, *19*(2), 179–200. https://doi.org/10.1016/S0193-953X(05)70283-5

Fisher, A. J., & Boswell, J. F. (2017). Enhancing the personalization of psychotherapy with dynamic assessment and modeling. *Assessment*, *23*(4), 496–506.

https://doi.org/10.1177/1073191116638735

Fisher, A. J., Medaglia, J. D., & Jeronimus, B. F. (2018). Lack of group-to-individual generalizability is a threat to human subjects research. *Proceedings of the National Academy of Sciences*, *115*(27), E6106–E6115. https://doi.org/10.1073/pnas.1711978115

Frederickson, J. (2013). *Co-creating change: Effective dynamic therapy techniques*. Kensington, MD: Seven Leaves Press.

Gaynes, B. N., Lux, L., Gartlehner, G., Asher, G., Forman-Hoffman, V., Green, J., ... Lohr, K. N. (2020). Defining treatment-resistant depression. *Depression and Anxiety*, *37*(2), 134–145. https://doi.org/10.1002/da.22968

Heshmati, R., Wienicke, F. J., & Driessen, E. (2023). The effects of intensive short-term dynamic psychotherapy on depressive symptoms, negative affect, and emotional repression in single treatment-resistant depression: A randomized controlled trial. *Psychotherapy*, *60*(4), 497–511. https://doi.org/10.1037/pst0000500

Heshmati, R., Wienicke, F. J., & Driessen, E. (2025). Dataset from a randomized controlled trial comparing intensive short-term dynamic psychotherapy to waitlist for treatment-resistant depression. *Data in Brief*, *63*, 112126. https://doi.org/10.1016/j.dib.2025.112126

Hollon, S. D., DeRubeis, R. J., Shelton, R. C., Amsterdam, J. D., Salomon, R. M., O'Reardon, J. P., ... Gallop, R. (2005). Prevention of relapse following cognitive therapy vs medications in moderate to severe depression. *Archives of General Psychiatry*, *62*(4), 417–422. https://doi.org/10.1001/archpsyc.62.4.417

Johansson, R., Eriksson, K., Åberg, K., Town, J., & Abbass, A. (2024). Intensive short-term dynamic psychotherapy for depression: Treatment effectiveness and effects of unlocking the unconscious. *Journal of Contemporary ISTDP*, *1*(2). https://doi.org/10.70839/bv43rz18

Johansson, R., Town, J. M., & Abbass, A. (2014). Davanloo's intensive short-term dynamic psychotherapy in a tertiary psychotherapy service: Overall effectiveness and association

between unlocking the unconscious and outcome. *PeerJ*, *2*, e548. https://doi.org/10.7717/peerj.548

Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology*, *3*, 1–27. https://doi.org/10.1146/annurev.clinpsy.3.022806.091432

Kraemer, H. C., Wilson, G. T., Fairburn, C. G., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry*, *59*(10), 877–883. https://doi.org/10.1001/archpsyc.59.10.877

Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2017). lmerTest package: Tests in linear mixed effects models. *Journal of Statistical Software*, *82*(13), 1–26. https://doi.org/10.18637/jss.v082.i13

Laurenceau, J.-P., Hayes, A. M., & Feldman, G. C. (2007). Some methodological and statistical issues in the study of change processes in psychotherapy. *Clinical Psychology Review*, *27*(6), 682–695. https://doi.org/10.1016/j.cpr.2007.01.007

Leaviss, J., & Uttley, L. (2015). Psychotherapeutic benefits of compassion-focused therapy: An early systematic review. *Psychological Medicine*, *45*(5), 927–945. https://doi.org/10.1017/S0033291714002141

Lenth, R. V. (2024). *Emmeans: Estimated marginal means, aka least-squares means.* Retrieved from https://CRAN.R-project.org/package=emmeans

Mrazek, D. A., Hornberger, J. C., Altar, C. A., & Degtiar, I. (2014). A review of the clinical, economic, and societal burden of treatment-resistant depression: 1996-2013. *Psychiatric Services*, *65*(8), 977–987. https://doi.org/10.1176/appi.ps.201300059

Nosek, B. A., Alter, G., Banks, G. C., Borsboom, D., Bowman, S. D., Breckler, S. J., ... Yarkoni, T. (2015). Promoting an open research culture. *Science*, *348*(6242), 1422–1425. https://doi.org/10.1126/science.aab2374

R Core Team. (2024). *R: A language and environment for statistical computing.* Vienna, Austria: R Foundation for Statistical Computing. Retrieved from

https://www.R-project.org/

Rush, A. J., Trivedi, M. H., Wisniewski, S. R., Nierenberg, A. A., Stewart, J. W., Warden, D., ... Fava, M. (2006). Acute and longer-term outcomes in depressed outpatients requiring one or several treatment steps: A STAR*D report. *American Journal of Psychiatry*, *163*(11), 1905–1917. https://doi.org/10.1176/ajp.2006.163.11.1905

Solbakken, O. A., & Abbass, A. (2015). Intensive short-term dynamic residential treatment program for patients with treatment-resistant disorders. *Journal of Affective Disorders*, *181*, 67–77. https://doi.org/10.1016/j.jad.2015.04.003

Tingley, D., Yamamoto, T., Hirose, K., Keele, L., & Imai, K. (2014). Mediation: R package for causal mediation analysis. *Journal of Statistical Software*, *59*(5), 1–38. https://doi.org/10.18637/jss.v059.i05

Torchiano, M. (2020). *Effsize: Efficient effect size computation*. Retrieved from https://CRAN.R-project.org/package=effsize

Town, J. M., Abbass, A., & Bernier, D. (2013). Effectiveness and cost effectiveness of Davanloo's intensive short-term dynamic psychotherapy: Does unlocking the unconscious make a difference? *American Journal of Psychotherapy*, *67*(1), 89–108. https://doi.org/10.1176/appi.psychotherapy.2013.67.1.89

Town, J. M., Abbass, A., Stride, C., & Bernier, D. (2017). A randomized controlled trial of intensive short-term dynamic psychotherapy for treatment resistant depression: The Halifax Depression Study. *Journal of Affective Disorders*, *214*, 15–25. https://doi.org/10.1016/j.jad.2017.02.035

Town, J. M., Abbass, A., Stride, C., Nunes, A., Bernier, D., & Berrigan, P. (2020). Efficacy and cost-effectiveness of intensive short-term dynamic psychotherapy for treatment resistant depression: 18-month follow-up of the Halifax Depression Trial. *Journal of Affective Disorders*, *273*, 194–202. https://doi.org/10.1016/j.jad.2020.04.035

Town, J. M., Falkenström, F., Abbass, A., & Stride, C. (2022). The anger-depression mechanism in dynamic therapy: Experiencing previously avoided anger positively

predicts reduction in depression via working alliance and insight. *Journal of Counseling Psychology*, *69*(3), 326–336. https://doi.org/10.1037/cou0000581

Wampold, B. E., & Imel, Z. E. (2015). *The great psychotherapy debate: The evidence for what makes psychotherapy work* (2nd ed.). New York, NY: Routledge. https://doi.org/10.4324/9780203582015

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*(6), 1063–1070. https://doi.org/10.1037/0022-3514.54.6.1063

Weinberger, D. A., Tublin, S. K., Ford, M. E., & Feldman, S. S. (1990). Preadolescents' social-emotional adjustment and selective attrition in family research. *Child Development*, *61*, 1374–1386. https://doi.org/10.2307/1130748

Wiles, N., Thomas, L., Abel, A., Ridgway, N., Turner, N., Campbell, J., ... Lewis, G. (2013). Cognitive behavioural therapy as an adjunct to pharmacotherapy for primary care based patients with treatment resistant depression: Results of the CoBalT randomised controlled trial. *The Lancet*, *381*(9864), 375–384. https://doi.org/10.1016/S0140-6736(12)61552-9

World Health Organization. (2021). *Depression*. Retrieved from https://www.who.int/news-room/fact-sheets/detail/depression

Supplementary Materials

The complete dataset and analysis code are publicly available on the Open Science Framework at https://doi.org/10.17605/OSF.IO/75PU8.

Table 1

Baseline Demographic and Clinical Characteristics by Treatment Condition

Characteristic	ISTDP	Waitlist Control
Age, M (SD)	36.5 (12.3)	37.3 (11.3)
Gender, %		
Male	34.9	41.9
Female	65.1	58.1
Marital status, %		
Single	39.5	25.6
Married	51.2	60.5
Widowed/Divorced	9.3	14.0
Education, %		
High school	41.9	34.9
Undergraduate	37.2	48.8
Graduate	20.9	16.3
Employment status, %		
Employed	41.9	65.1
Unemployed	46.5	23.3
Retired	11.6	11.6
Socioeconomic status, %		
Low	14.0	16.3
Middle	65.1	65.1
High	20.9	18.6
Previous antidepressant trials, M (SD)	1.81 (1.01)	1.86 (0.89)
Currently receiving medication, %		
Yes	76.7	81.4
No	23.3	18.6

Note. ISTDP = Intensive Short-Term Dynamic Psychotherapy. N = 86

Table 2

Estimated Marginal Means for Depression Scores by Treatment Condition and Time Point

	ISTDP			Waitlist Control		
Time Point	*M*	*SE*	95% CI	*M*	*SE*	95% CI
Baseline	30.07	0.68	[28.73, 31.41]	30.40	0.68	[29.06, 31.73]
Post-treatment	21.67	0.71	[20.26, 23.08]	29.29	0.70	[27.92, 30.67]
3-Month Follow-up	17.20	0.71	[15.79, 18.61]	29.19	0.70	[27.81, 30.56]

Note. Values are estimated marginal means from the linear mixed-effects model with random intercepts. CI = 95\% confidence interval. ISTDP = Intensive Short-Term Dynamic Psychotherapy.

Table 3

Mediation Analysis Results for Key Process Measures

Mediator	Indirect Effect [95% CI]	Proportion Mediated	p
Distress (Total)	-6.34 [-11.07, -3.08]	53.9%	0.000
Emotional Repression	-1.84 [-5.41, 1.00]	15.6%	0.246
Negative Affect	1.51 [-0.52, 3.87]	-12.8%	0.149

Note. Indirect effects estimated using bootstrap mediation analysis with 5,000 resamples. CI = 95\% bias-corrected and accelerated confidence interval. All models control for baseline depression.

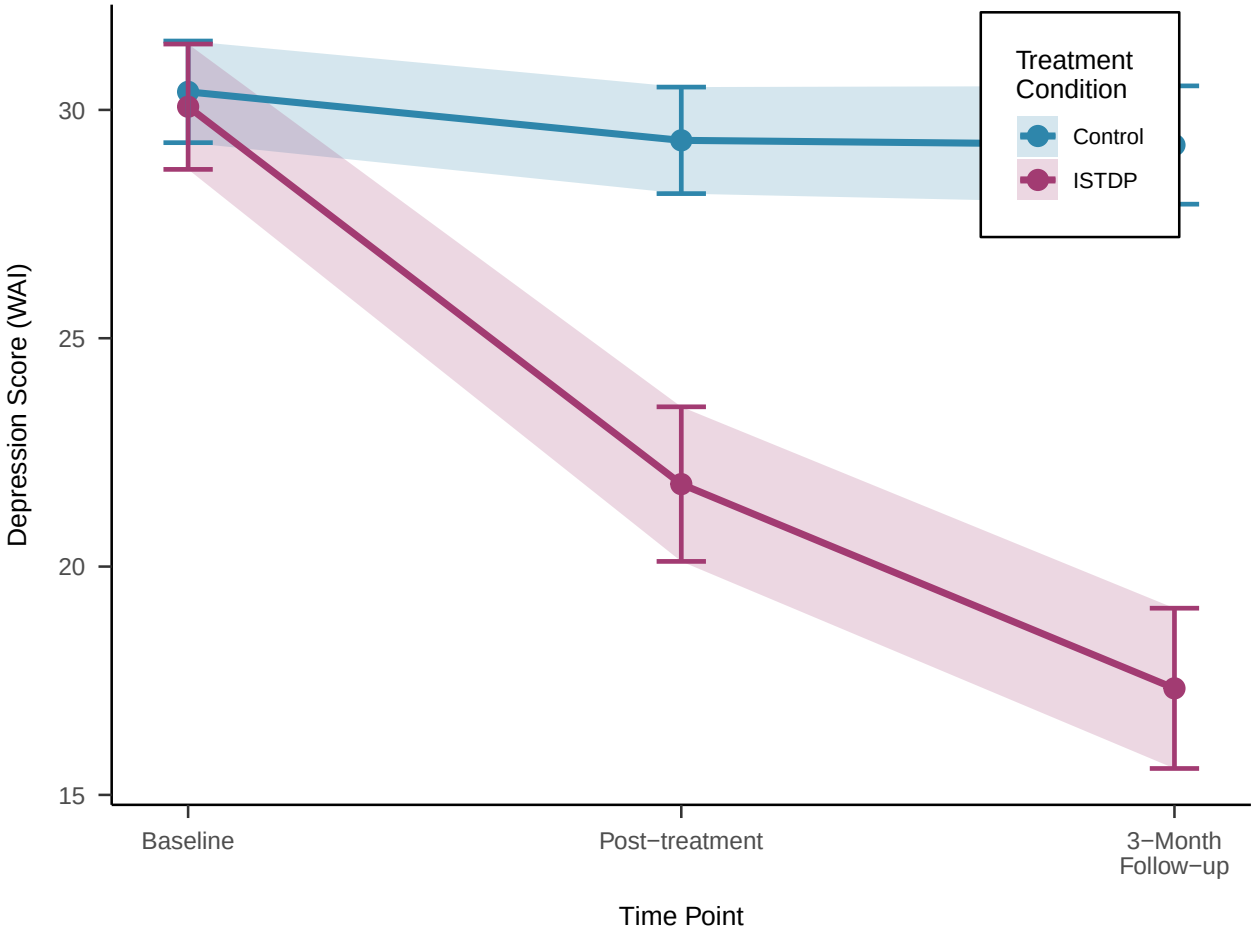

Figure 1. Mean depression trajectories by treatment condition. Error bars represent 95% confidence intervals. ISTDP = Intensive Short-Term Dynamic Psychotherapy.

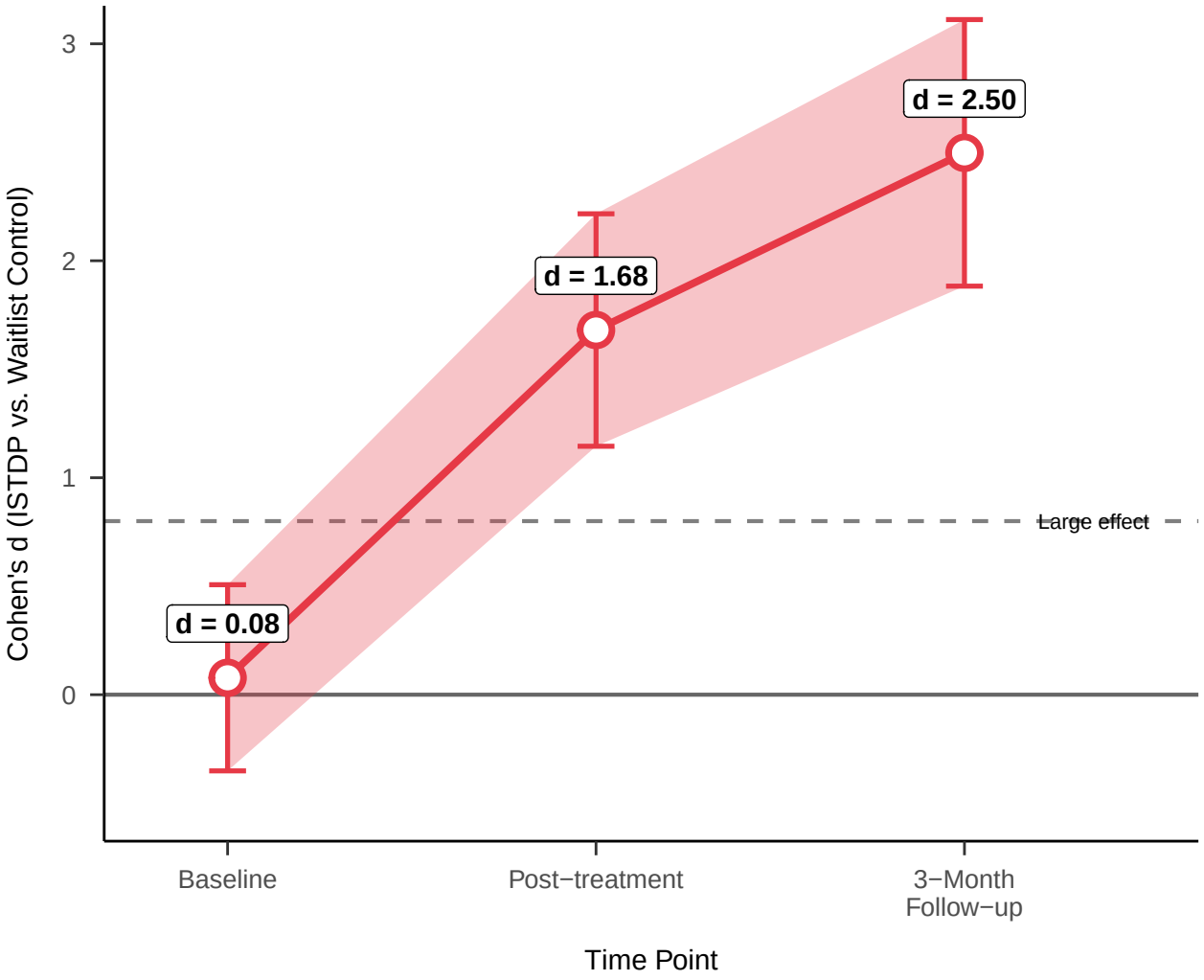

Figure 2. Between-group effect sizes (Cohen's d) over time with 95% confidence intervals. Positive values indicate lower depression in the ISTDP group compared to waitlist control. The dashed line represents the threshold for a large effect (d = 0.80).

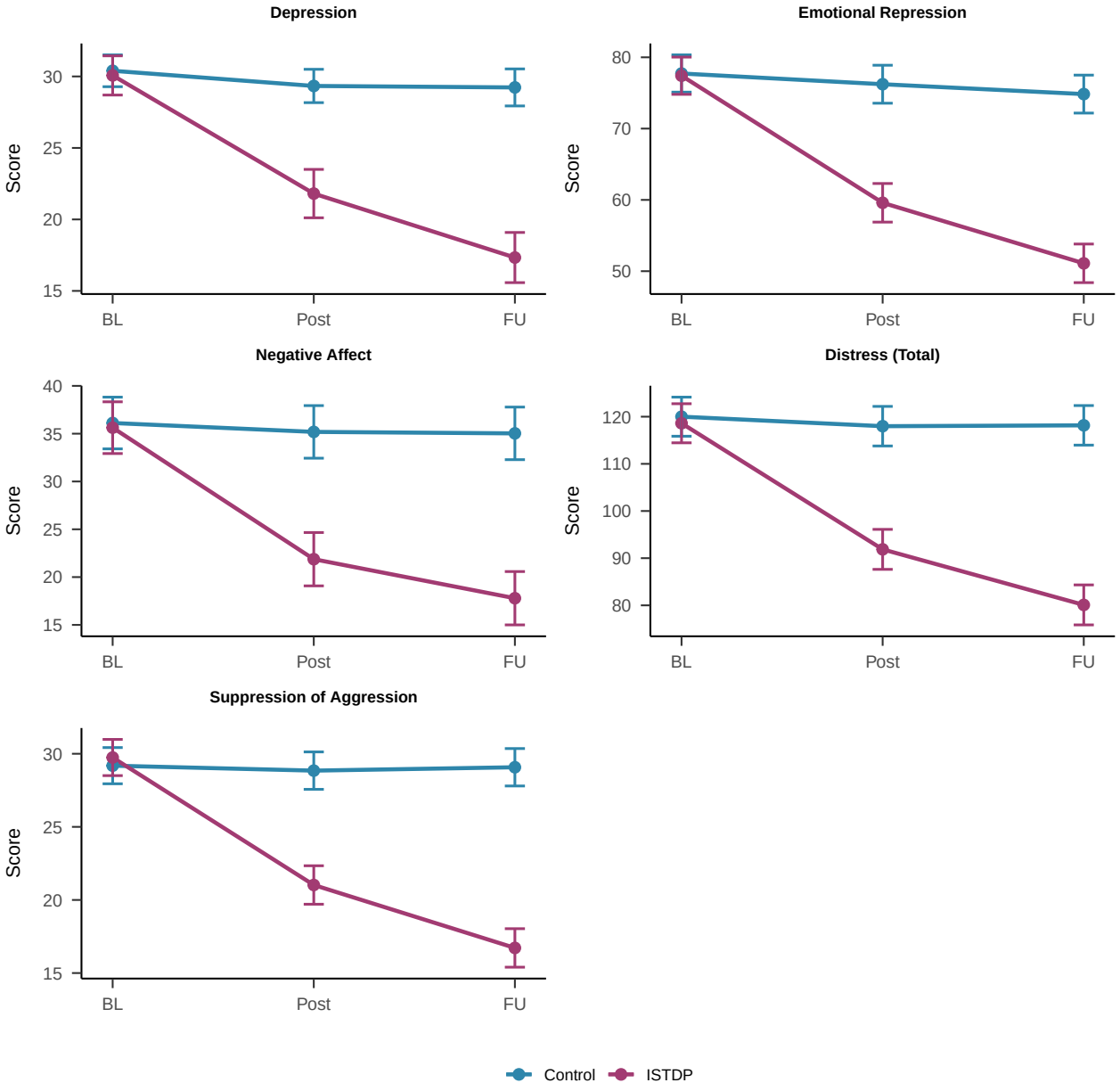

Figure 3. Trajectories for depression and key process measures by treatment condition. Error bars represent 95% confidence intervals. ISTDP = Intensive Short-Term Dynamic Psychotherapy; WAI-RRC = Weinberger Adjustment Inventory Repressive/Restraint Composite; PANAS = Positive and Negative Affect Schedule.