Change and sustained recovery in defense mechanisms and depression in a pilot study of antidepressant medications plus up to 18 months of psychotherapy for recurrent major depression

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Citation: Perry, J. C., Starrs, C. J., Semeniuk, T., Banon, E., & Westreich, R., (2025). Change and sustained recovery in defense mechanisms and depression in a pilot study of antidepressant medications plus up to 18 months of psychotherapy for recurrent major depression. *Research in Psychotherapy: Psychopathology, Process and Outcome, 28*(3), 885. doi: 10.4081/ripppo.2025.885

Contributions: JCP and CJS, conceptualization, methodology, investigation, formal analysis, writing – original draft, writing – review & editing; EB and TS, conceptualization, methodology, investigation, formal analysis, writing – review & editing; RW, conceptualization, methodology, investigation, formal analysis. All authors have read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of interest: the authors declare that they do not have any conflict of interest.

Ethics approval and consent to participate: ethical approval for the current study was obtained from the Jewish General Hospital Ethics Committee (protocol #04-013, 2004). Written consent to participate was obtained from all participants.

Availability of data and materials: the datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Funding: this study was supported by a grant from the Fund for Psychoanalytic Research of the American Psychoanalytic Association.

Received: 23 October 2025.
Accepted: 2 December 2025.

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Licensee PAGEPress, Italy
Research in Psychotherapy:
Psychopathology, Process and Outcome 2025; 28:885
doi:10.4081/ripppo.2025.885

ABSTRACT

Treatment studies of major depression commonly focus on symptoms rather than changes in psychological risk factors. This pilot study examines the relationship between changes in eight defenses specifically related to depression, called *depressive defenses*, and depressive symptoms. Thirty adults with acute, recurrent major depression were given antidepressant medications (ADM) and randomized to up to 18 months of either cognitive behavioral therapy (CBT), dynamic, or supportive psychotherapy, and followed for 4.5 years. Defenses were assessed using the observer-rated Defense Mechanism Rating Scales (DMRS) at intake and 18 months. The Hamilton Rating Scale for Depression-17 item version (HRSD-17) and the Beck Depression Inventory-2nd version (BDI-II) assessed depression periodically. Depressive symptoms decreased significantly on both the HRSD-17 (effect size [ES]=−1.03) and BDI (ES=−1.90). Overall defensive functioning (ODF) increased significantly (ES=.85), improving in 76% of participants. Similarly, the mean proportion of depressive defenses decreased significantly by termination (ES=−.62), although the overall mean remained above that generally seen in healthy adults. Twenty-four percent of participants attained this threshold at termination. After controlling for initial levels, at termination depressive defenses correlated significantly with HRSD-17 (r_s=.44, p=.02), and BDI (r_s=.33, p=.095). Although causal relationships were not established, depressive defenses were consistently related to changes in depressive symptoms, suggesting that they are promising mediators of treatment effects for major depression. Clinically, defenses are readily identifiable and can serve as important foci in treatment. Finally, levels of depressive defenses that exceed healthy norms may reflect a continuing level of risk for current or future depressive symptoms or episodes.

Key words: defense mechanisms, depression, psychotherapy, follow-up.

Introduction

Major depression has a lifetime prevalence of up to 19% of the adult population, with 4.4% affected at any given time, and is one of the leading causes of disability worldwide (Kessler & Bromet, 2013; GBD, 2018; WHO, 2017). Depression is associated with substantial limitations in functioning and well-being (Kessler, 2012; Solomon *et al.*, 2004), risk for completed suicide

(Gilman *et al.*, 2017; Tidemalm *et al.*, 2008), burdensome economic costs (König *et al.*, 2020), substantial risk for slow and/or incomplete recovery (Judd *et al.*, 1998; Keller *et al.*, 1992), and increasing risk for subsequent episodes following each additional occurrence (Mueller *et al.*, 1999). Considered together, these findings offer a clear rationale to identify and ameliorate any potential underlying modifiable risk factors for recurrence. A recent review (Minges *et al.*, 2017) supported reflective functioning, insight, defense mechanisms, and therapeutic alliance as potential mediators of improvement in psychotherapy. The current report examines improvement in defense mechanisms over the course of psychotherapy in relation to the long-term course of recurrent depression.

Defense mechanisms

Defense mechanisms are one of the most durable constructs in psychoanalysis, dynamic psychiatry and psychology, spanning theory, therapy, and research, since Freud first described them (Freud 1894/1962). A half-century of research has established that there is a hierarchy of defenses based on their usual level of adaptiveness (Cramer, 2006; Perry, 1993; Vaillant, 1993). Individuals learn to use a range of defenses, constituting their own personal defense repertoire, which tends to progress over time by a gradual process of replacing lower-level defenses with mid-level, then more mature, higher adaptive defenses (Vaillant, 1993). Psychotherapy studies (Babl *et al.*, 2019; Johanson *et al.*, 2010; Kramer *et al.*, 2010; Perry & Bond, 2012; Perry *et al.*, 2020) have shown this process, with increased adaptive defenses over the course of treatment, in lieu of less immature defenses. Furthermore, these improvements in overall defensive functioning (ODF), in turn, are associated with decreased symptoms at termination and/or follow-up in both dynamic and cognitive behavioral therapy (CBT) psychotherapies.

Studies have identified eight immature defenses that are strongly associated with depression (Babl *et al.*, 2019; Bloch *et al.*, 1993; DeFife & Hilsenroth, 2005; Høglend & Perry, 1998; Perry & Cooper, 1989). These include passive-aggression, acting out, help-rejecting complaining, splitting of self and others' images, projective identification, projection, and devaluation. A recent meta-analysis found that depressed patients scored higher on non-mature defenses than did controls (Fiorentino *et al.*, 2024). Di Giuseppe *et al.* (2024) found that passive aggression was a central node relating to several other depressive defenses, and that 7 depressive defenses were significantly associated with depressive symptoms. Babl *et al.* (2019) found that depressed patients had a higher proportion of depressive defenses compared to anxiety patients. Høglend and Perry (1998) showed that when present at the outset of treatment for major depression, depressive defenses predicted poorer response at six months. Self-report assessments have also demonstrated a stronger relationship for depressive (Di Giuseppe *et al.*, 2020) or immature defenses (Prout *et al.*, 2022) with depressive symptoms than with anxiety or other symptoms, including in a large survey of 19,860 participants across 6 countries (Békés *et al.*, 2024).

Recent meta-analyses have demonstrated that various therapy types, including CBT, interpersonal therapy (IPT), and dynamic therapy, are associated with greater improvement in depression than control conditions (Casacalenda *et al.*, 2002; Cuipers *et al.*, 2013; Driessen *et al.*, 2015; Leichsenring *et al.*, 2015; Steinert *et al.*, 2017). Furthermore, psychotherapy may also delay recurrence (Biesheuvel-Leliefeld *et al.*, 2015; Cuijpers *et al.*, 2013), even after successful treatment with antidepressant medications (ADM; Guidi & Fava, 2020). Given that these effects have been demonstrated across therapy types, it is likely that there may be one or more mechanisms of change mediating these improvements.

In depressed patients, depressive defenses improve more than most other defenses during psychotherapy (Babl *et al.*, 2019; Perry *et al.*, 2020), but not in anxiety patients (Babl *et al.*, 2019). In addition, change in depressive defenses correlates highly with change in depression (Perry *et al.*, 2020), as well as in other symptoms and general functioning (Perry & Bond, 2012). Both Kramer *et al.* (2010) and Babl *et al.* (2020) found evidence consistent with a causal role of improvement in defenses and improvement in symptoms. In our previous study (Perry *et al.*, 2020), with 20 sessions of either CBT or dynamic therapy *plus* ADM, patients with recurrent major depression showed significant decreases in depressive defenses by termination (effect size [ES]=.97). However, a mean of 12.17% (standard deviation [SD]=10.60) depressive defenses remained and only five subjects (50%) attained normative defensive levels. Furthermore, at 1-year follow-up, depressive defenses had returned somewhat. Overall, these findings suggest some trait and some, less stable, state changes in defenses.

Aims of the current study

The present study extends our previous work to determine whether longer-term treatment and follow-up might produce more durable improvement in both depressive defenses and depression. We report here on a sample of 30 individuals with acute, recurrent major depressive disorder who received ADM *plus* either CBT, dynamic, or supportive psychotherapy for up to 18 months. Participants were then followed for up to 4.5 years. This study examines the following hypotheses:

H1: Depressive symptoms will improve over the course of treatment, from intake to 4.5 years, as assessed by i) the Hamilton Rating Scale for Depression-17 item version (HRSD-17), our primary outcome, and ii) the Beck Depression Inventory-II (BDI-II).

H2: Defense mechanisms will improve over the course of treatment. This will be evidenced in two ways: i) ODF will improve, with changes in individual defense levels generally following the hierarchy of adaptiveness, and ii) specifically, depressive defenses will have decreased by 18-month termination.

H3: After controlling for initial depression and defense scores, i) the degree of change in ODF and depressive defenses will be associated with improvement in depressive symptoms at 4.5 years, and ii) those attaining either a normative ODF or with a normative proportion of depressive defenses (8%) will demonstrate sustained recovery from depression at 4.5 years.

Finally, although not a hypothesis given our small sample size, we will explore whether improvement in depressive defenses and depression is consistent across all three treatment conditions (trans-theoretical), or only present within dynamic, but not CBT or supportive therapy.

Materials and Methods

We conducted a randomized pilot study to demonstrate the feasibility of comparing up to 18 months of CBT, dynamic, or supportive psychotherapy, with ADM for acute recurrent major depression (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* [DSM-IV]; APA, 1994). We did not examine

the role of ADM alone. Following our previous 20-session treatment comparison (Perry *et al.*, 2020), we chose an 18-month treatment period, as originally suggested by Arieti & Bemporad (1978), anticipating that with longer treatment and follow-up, we might detect sustained recovery in up to half of the sample. Follow-up continued over 4.5 years.

Procedures and participants

We recruited participants from hospital outpatient sources. All participants gave written informed consent. The study was approved by the Jewish General Hospital Research & Ethics Committee (protocol #04-013, 2004). Inclusion criteria were acute, recurrent DSM-IV major depression, and an HRSD-17 score of 17 or higher. Exclusion criteria included a diagnosis of psychotic or bipolar type I disorders, current problematic substance use/dependence, and an effective response to ADM in the prior 4 weeks.

Treatments and therapists

All therapies were planned for up to 18 months of sessions. CBT was based on Beck's cognitive theory model (Beck, 1995; DeRubeis *et al.*, 2001). Dynamic therapy followed Luborsky's Supportive-Expressive psychotherapy as applied to depression (Luborsky *et al.*, 1995; Luborsky *et al.*, 1996), supplemented by the approach of Arieti and Bemporad (1978). Supportive psychotherapy followed a manual previously validated by Novalis and colleagues (1993).

Participants were assigned to each treatment using computer-based urn randomization with two *a priori* factors, gender, and total number of episodes (<4 *vs.* >4) to balance the treatment groups. A clinician administered a 1-hour dynamic interview at intake and termination (Fowler & Perry, 2005; Perry *et al.*, 2005), which was subsequently rated for defenses. In addition, a research assistant administered the Relationship Anecdote Paradigm (RAP; Luborsky & Crits-Christoph, 1990) at intake and termination. The RAP is a semi-structured interview with open-ended questions that elicit two spontaneous recent life vignettes across three areas: occupation, intimate relationships, and psychotherapy/helping relationships (Beck & Perry, 2008). We used this approach to solicit participant *verbatim*, which was then rated for defenses.

Therapists were experienced clinicians who primarily worked within their respective models. Treatment guidelines were developed by each treatment team to delineate the focus of treatment and ensure fidelity through periodic supervision groups. Treatment adherence was examined by rating sessions using the Psychotherapy Process Q-sort (Pole *et al.*, 2008). Regarding adherence, dynamic treatments scored most highly on the dynamic factor, CBT scored most highly on the CBT factor, and supportive was lower on both (for details, see Grimm *et al.*, 2016).

All participants received medication prescribed by a psychiatrist following the guidelines for 18-month ADM management adapted from a previous depression treatment study (see Perry *et al.*, 2020 for details).

Measures

Diagnoses according to the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (DSM-IV-TR; APA, 2000) were made by a psychiatrist using the Guided Clinical Interview (Perry, 1992; Perry & Bond, 2009). This interview has shown high inter-rater reliability with median kappas of .86 for Axis I, .87 for Axis II, .92 for major depressive disorder, and .86 for dysthymic disorder (Perry *et al.*, 2017).

Depressive symptoms were assessed using two measures, and Cronbach's α included all available observations. The HRSD-17 (Hamilton, 1960) is a widely used 17-item clinician-rated assessment of the severity of depressive symptoms over the past week. Items are rated on a 3 or 5-point scale and summed to obtain a total score. Scores are interpreted as: severe >24, moderate 17-23, mild 7-16, absent <6. The HRSD-17 was administered twice during intake and monthly until 18-month termination, then bi-monthly over the follow-up period by research assistants and the prescribing physicians. Cronbach's α was .87. The BDI-II (Beck *et al.*, 1996) is a 21-item self-report scale that assesses the cognitive, affective, behavioral, and neurovegetative features of depression over the past two weeks. Items are rated from 0 to 3, with the total sum score ranging from 0 to 63. Scores are interpreted as: minimal 0-13; mild 14-19; moderate 20-28; and severe 29-63 (Beck *et al.*, 1996). Cronbach's α was .96. We applied the following cutoffs for recovery: HRSD-17 ≤6, and BDI-II ≤10.

Defense mechanisms were identified using the Defense Mechanism Rating Scales (DMRS), fifth edition (Perry, 1990). The DMRS is an observer-rated method for identifying defense mechanisms in *verbatim* transcripts. Defenses are arranged hierarchically into 7 defense levels based on their general level of adaptiveness (Perry, 1993). Three levels of scoring yield continuous, ratio scales for: 1) individual defenses, 2) seven defense levels, and 3) three defense categories. In addition, the immature defense category can be subdivided into depressive and non-depressive defenses (Høglend & Perry, 1998; Perry *et al.*, 2020). For additional scoring details, see Perry *et al.* (2020). The recovery cutoff for ODF is >5.38, which corresponds to half a standard deviation below the mean of a normative community sample of adult women (Perry *et al.*, 2015). The cutoff for recovery on depressive defenses is ≤.08 (Perry *et al.*, 2020). These values are rounded from the original study on which they are based (Perry *et al.*, 2015), in which a control sample of healthy women showed a mean proportion of depressive defenses of 0.079 (SD=0.084). We also previously employed this cutoff in Perry *et al.* (2020). Intake and 18-month dynamic and RAP interviews were blinded and rated by trained research assistant raters in random order, blinded to treatment type. Intraclass $R_{(2,1)}$ inter-rater reliability figures were: number of defenses identified I_R=.72; ODF I_R=.84; high adaptive (mature) category I_R=.68; neurotic category I_R=.55; immature category I_R=.65; depressive defenses I_R=.83; non-depressive defenses I_R=.45; defense levels median I_R=.68, range .32 to .72 (Perry *et al.*, 2020). These are acceptable to good, and similar to figures recently reported elsewhere (Babl *et al.*, 2019).

Data analysis

As the HRSD-17 was administered monthly and the BDI on multiple but fewer occasions, we modeled intent-to-treat outcomes by calculating individual simple linear regression models for each participant, for each measure. From these, we estimated: slope (rate of change), intercept (predicted intake score), and predicted score at the last observed follow-up. We then calculated raw change and ES, as in prior studies (Perry & Bond, 2009, 2012; Perry & Fowler, 2021). These models smooth out extreme scores, producing highly conservative and robust estimates. The same procedure was completed for defenses. Of note, effect sizes are interpreted using Cohen's *d* (1988) criteria: .20 small, .50 medium, and .80 large. Change was examined with a *t*-test or a signed-rank test. Non-parametric Spearman correlations examined the rela-

tionships between defense change and depression outcomes, partialing out initial levels of each. Exploratory analyses were conducted by treatment group, for which the magnitude of the findings may be more heuristically meaningful than the nominal p-values (Kraemer *et al.*, 2002).

Given the small sample size, we considered the risk of both type I and II errors. As our depression estimates were modeled using multiple observations, we considered the precision obtained as a partial guard against false findings. This is further strengthened whenever findings across measures are consistent in direction. Furthermore, as defense levels are ordered in a hierarchy, the degree to which the findings follow this pattern is also a partial assurance against error. We present the nominal p-values, but given multiple comparisons within each table of analyses, we also note the Bonferroni corrected alpha for each independent variable that is not a composite variable (8 levels). It should be noted that the Bonferroni corrected alpha is overly conservative, as defenses are intercorrelated, as are the depression outcomes.

Results

Descriptive statistics

We enrolled 30 adults, 19 (63%) identified as female and the sample mean age was 43 (range 23-62). Intake depressive episodes had a mean duration of 5.20 months (range 1-20), and the median lifetime episode number was 3, distributed as 2 (11; 37%), 3 (7; 23%), 4 (4; 13%), 5 or more (8; 17%) episodes. Mean age of first episode was 26.10 (range 11-55). The group median lifetime major psychiatric disorders was 5 (2.5 current), including 14 (47%) with dysthymic disorder and 16 (53%) with an Axis II personality disorder. At intake, the mean HRSD-17 was 24.70 (SD=4.52, range 17-36) and BDI-II was 32.93 (SD=10.73; range 13-51). Mean intake global assessment of functioning (GAF) was 48.90 (SD=4.20), and mean best usual GAF in the past year was 62.30 (SD=5.80).

Treatment assignment, attrition, and duration

No subjects refused the treatment they were randomly assigned. Assignment yielded: 13 CBT, 10 dynamic, 7 supportive. Attrition was defined as stopping prior to 18 months, without patient-therapist agreement. Overall, 4 participants (13.33%) dropped out: CBT 3 (23%), dynamic 0 (0%), and supportive 1 (14%). On average, participants received 33.97 (SD=25.73) sessions, which differed significantly by treatment group (Kruskal-Wallis $\chi^2(2)=8.83$, p=.02): CBT 21 (SD=10.44), dynamic 62.70 (SD=23.43), supportive 17 (SD=9.04). On average, only the dynamic group was treated for 18 months, whereas CBT and supportive therapy averaged a year in treatment.

Improvement in depression

For the HRSD-17, the median duration of follow-up was 4.56 years (mean 4.20, SD=1.49), with a median of 27 observations (mean=24.93, SD=8.89). Scores were not normally distributed as 5 participants completed less than two years of observations, while the others gave >4 years. The HRSD-17 demonstrated significant improvement over the study period (p<.001). Twenty-four (80%) participants were improving, and 9 (30%, confidence interval [CI]: 12.60-47.40) attained a sustained recovery (HRSD-17<6). Among the 25 who gave 4 or more years of follow-up data, the percentage entering sustained recovery rose to 36% (CI: 15.78-56.22). This figure does not differ significantly from the hypothesis that 50% would attain sustained recovery (n=25, t=1.423, p=.17). No one with fewer than 2 years of follow-up entered sustained recovery.

For the BDI-II, the median duration of follow-up was 4.40 years (mean 3.77, SD=1.76), with a median of 6.50 observations (mean 5.97, SD=2.68). Of note, 2 participants gave only an initial BDI and were not included in the models. BDI-II scores improved significantly over this period (p<.001). Twenty-two (78.47%) were improving, while 18 (64.29%, CI: 45.37-83.21) attained a sustained recovery (BDI<10). For the 5 participants with fewer than 2 years of follow-up, 2 had a final estimate in the recovered range, but there were too few observations and time to detect sustained recovery.

The modeled scores for HRSD-17 and BDI-II were positively but non-significantly correlated at initial (r_s=.33, n=28, p=.08) and significantly at final values (r_s=.84, n=28, p<.001). A 2×2 analysis (recovered *vs.* not recovered by HRSD-17 *vs.* BDI-II) yielded a non-significant odds ratio (OR=7.20, CI: .75-69.38, n=28, p=.098). This was primarily due to the larger number recovered on the BDI. The positive predictive value (PPV) of BDI recovery for HRSD recovery was 44.44%, and the negative predictive value (NPV) was 90.00% (Table 1).

Improvement in defensive functioning

Changes in the 7 defense levels ranged from −3.70% to 3.65%. The four immature and hysterical defense levels showed small to moderate decreases (ES: −0.17 to −0.63). Equally, mature, obsessional and other neurotic level defenses also demonstrated small to moderate increases (ES: .34 to .53). The three defense categories reflected a similar pattern with both mature (ES= .53) and neurotic (ES= .41) defenses showing moderate increases, and immature defenses large decreases (ES=−.83). Within the immature category, depressive defenses also exhibited moderate decreases (ES=−.62), more than non-depressive defenses (ES=−.47). Reflecting these improvements, ODF increased significantly (ES=.85). ODF improved in 20 (76%) participants, not in 9 (24%) (Table 2).

Table 1. Change in depressive symptoms from intake to the end of follow-up by respective models.

Depression scale	Intake Mean or median (SD)	Follow-up Mean or median (SD)	Raw change Mean or median (SD)	ES	Slope Mean (SD)	Improved/ recoveredn (%)	Contrast W, p
HRSD-17 (n=30)	17.43 (6.00)	11.32 (8.36)	−6.12 (7.48)	−1.03	−3.31 (5.75)	24 (80)/9 (30)	−167.50, <.001
BDI-II (n=28)	23.28 (6.82)	9.77 (11.30)	−13.61 (11.51)	−1.90	−5.84 (9.84)	22 (78)/18 (64)	−173.00, <.001

SD, standard deviation; ES, effect size; W, Wilcoxon signed-rank test; HRSD-17, Hamilton Rating Scale for Depression-17 item version; BDI-II, Beck Depression Inventory 2nd version.

Improvement in defenses and depression

Table 3 presents the correlations between final values of defenses and the modeled final HRSD-17 value, after controlling for their initial values, for the whole sample. Final ODF demonstrated a significant negative correlation with final HRSD-17 (r_s=−.62, p<.001). All three defense categories correlated in the expected direction with final HRSD-17. Only the immature defenses were significant, driven entirely by depressive defenses (r_s=.44, p=.020). Whereas non-depressive defenses demonstrated no relationship. Mature defenses were trending positive. The general form of the third hypothesis (3i) was upheld for both ODF and depressive defenses.

Table 3 also displays the correlations between final values of defenses and BDI-II values at follow-up, after partialing out their initial values. In general, the findings for ODF, mature and immature defenses were significant and similar to those for the HRSD-17. The correlation with depressive defenses was slightly diminished in magnitude and significance compared to that with the HRSD-17.

For hypothesis 3ii, we first examined the 2×2 table of ODF recovery by HRSD recovery. Twenty-four participants were concordant on both measures (6 recovered, 17 not recovered), whereas 3 participants each were recovered on one measure but not on the other. The odds ratio was significant (OR=11.33, CI: 1.78-72.17, p=.006). Second, the 2×2 table of recovery on depressive defenses by HRSD-17 recovery yielded a non-significant odds ratio (OR=2.0, CI: .34-11.70, p=.445. Hypothesis 3ii was upheld for HRSD-17 recovery and ODF recovery. It was not upheld for HRSD-17 recovery and depressive de-

Table 2. Change in defenses from intake to 18 months (n=29, except as noted).

	Intake Mean (SD)	18-months Mean (SD)	Raw change Mean (SD)	Slope Mean (SD)	ES 1 *vs.* 2	Contrast t, p
Defense levels						
7. High adaptive* (n=27)	16.58 (6.91)	20.23 (10.12)	3.65 (12.63)	1.82 (4.30)	.53	1.50, .144
6. Obsessional	25.13 (8.64)	28.09 (9.74)	2.96 (11.82)	2.54 (10.28)	.34	182.50#, .074
5a. Hysterical	13.15 (7.31)	11.89 (6.91)	−1.26 (9.68)	−0.48 (6.42)	−.17	143.50#, .356
5b. Other neurotic	14.35 (6.22)	17.37 (9.20)	3.02 (5.90)	2.08 (3.82)	.49	2.76, .010
4. Minor image-distorting (n=28)	9.48 (6.41)	7.80 (5.97)	−1.69 (5.80)	−1.49 (4.34)	−.26	1.54, .136
3. Disavowal and fantasy	8.74 (6.02)	6.45 (4.03)	−2.28 (6.15)	−1.25 (4.84)	−.38	2.00, .055
2. Major image-distorting (n=23)	2.35 (2.77)	1.37 (1.32)	−.97 (3.46)	−0.78 (2.82)	−.35	124.00#, .477
1. Action	9.81 (5.83)	6.12 (4.84)	−3.70 (6.92)	−2.57 (5.25)	−.63	2.89, .008
Category scores						
High adaptive (level 7)	16.53 (6.91)	20.23 (10.12)	3.65 (12.63)	1.82 (4.30)	.53	1.50, .144
Neurotic (levels 5-6)	52.63 (12.00)	57.51 (10.84)	4.88 (11.78)	4.15 (9.47)	.41	2.23, .033
Immature (level 1-4)	30.10 (10.50)	21.34 (7.67)	−8.76 (11.11)	−6.19 (8.22)	−.83	4.25, <.001
Depressive defenses	17.52 (8.29)	12.40 (6.45)	−5.12 (10.04)	−3.61 (7.23)	−.62	2.75, .010
Non-depressive defenses	12.59 (7.69)	8.94 (4.51)	−3.64 (8.00)	−2.58 (5.75)	−.47	114.50#, .010
Summary scores						
Number of defenses	50.90 (18.89)	53.92 (21.26)	3.02 (12.96)	2.80 (10.10)	.16	1.25, .220
Overall defensive functioning	4.87 (.41)	5.22 (.39)	.35 (.56)	.23 (.39)	.85	3.35, .002

SD, standard deviation; ES, effect size; *a.k.a. mature defenses; #Wilcoxon signed-rank test; defenses were identified using the Defense Mechanism Rating Scale in the Relationship Anecdote Paradigm and dynamic interviews; the Bonferroni corrected alpha for each contrast is based on 8 defenses levels only (alpha=.01), the overall defensive functioning and categories are composites of these and not independent.

Table 3. Spearman correlations for ODF and defense category with depression, after controlling for initial levels.

	HRSD-17				BDI-II			
Defense category	Total sample Mean, p (n=29)	CBT Mean, p (n=13)	DYN Mean, p (n=10)	SUP Mean, p (n=6)	Total sample Mean, p (n=28)	CBT Mean, p (n=13)	DYN Mean, p (n=9)	SUP Mean, p (n=6)
ODF	−.62, <.001	−.76, .007	−.63, .093	−.74, .257	−.69, <.001	−.87, <.001	−.55, .199	−.97, .029
High adaptive*	−.34, .091	−.60, .090	−.66, .073	−.80, .202	−.54, .007 n=26	−.88, .002 n=11	−.84, .016	−.20, .803
Neurotic	.14, .488	.41, .216	.12, .771	−.21, .789	.32, .106	.52, .098	.45, .305	−.26, .743
Immature	.40, .037	.62, .041	.81, .015	.38, .620	.47, .015	.55, .082	.83, .021	.60, .395
Depressive	.44, .020	.61, .046	.71, .046	.15, .846	.33, .095	.38, .249	.19, .683	.97, .026
Non-depressive defenses	−.01, .957	.03, .927	.26, .537	−.35, .643	.04, .834	.05, .893	.53, .218	.19, .812

HRSD-17, Hamilton Rating Scale for Depression-17 item version; BDI-II, Beck Depression Inventory 2nd version; CBT, cognitive behavioral therapy; DYN, dynamic therapy; SUP, supportive therapy; ODF, overall defensive functioning; *a.k.a. mature defenses; Bonferroni corrected alpha for the three defense levels =.017; for depressive and non-depressive defenses =.025.

fenses recovery, although the finding was in the expected direction.

We also examined hypothesis 3ii for the BDI. For the 2×2 table of ODF recovery by BDI recovery, 19 participants were concordant on both measures (9 recovered, 10 not recovered), whereas 9 participants were not recovered on ODF but recovered on the BDI. None was recovered on ODF, but not recovered on BDI. The odds ratio was significant (OR=21.0, CI: 1.07-411.86, p=.008). The 2×2 table of final recovery on depressive defenses by BDI recovery yielded a non-significant odds ratio (OR=4.50, CI: .46-44.29, p=.179). This hypothesis was upheld for ODF recovery but not for recovery on depressive defenses and the BDI.

Finally, for exploratory purposes, Table 3 also displays the correlations by treatment group. For defenses and HRSD-17, the findings were mostly the same for the CBT group, more mixed for the dynamic group and less strong for the supportive therapy group. For defenses and BDI, the findings were more mixed across all three groups. Thus, although these findings are exploratory and should be interpreted with caution given the small size of each individual group, relationships between defenses and depression by group appeared more similar than different to the overall findings, suggestive of a trans-theoretical role for defenses as mechanisms of change.

Discussion

The premise of the current study is that underlying causal risk factors may explain, in part, the heterogeneity of outcomes in those with major depressive episodes. Outcomes comprised response to treatments, episode duration, symptom level, and long-term course, including recurrences, persistent symptoms, or sustained recovery (Perry & Fowler, 2021). We selected acute recurrent major depression in order to focus on those with greater liability for 1) a limited treatment response, 2) higher levels of residual symptoms, and 3) future recurrences. We chose a treatment duration of up to 18 months and hypothesized that up to 50% of the sample would attain sustained recovery. Our measurement model entailed multiple assessments over 4.5 years, which could reasonably detect sustained recovery in at least some patients (Keller *et al.*, 1992). Finally, we focused on defense mechanisms as a potential risk factor, which respond to treatment and, in turn, may influence the long-term course of depression, across treatment types.

Our sample fit the intended population description of acute depression, with moderate or greater severity of symptoms and other indicators of long-term liability, including prior episodes, dysthymic disorder, other co-occurring psychiatric syndromes and personality disorders. The best level of global functioning in the past year was also limited. Thus, our study group appears representative of the intended diagnosis and associated seriousness of distress and impairment.

Design choices

Randomization to three treatment types served multiple heuristic aims. All three treatment types are widely practiced in our department. While CBT has the largest number of published studies indicating treatment efficacy for depression, meta-analyses generally support the superiority of all specific psychotherapy types compared to wait-list controls or treatment-as-usual (Cuijpers *et al.*, 2023; Fukumori *et al.*, 2024). As a pilot study, our sample size was powered to detect overall medium effects for defenses, but the individual arms were not. Instead, our exploratory analyses sought to gain preliminary evidence for whether defense changes may be trans-theoretical, that is, potentially contributing to positive outcomes across treatment types.

Treatment with ADM for moderate to severe depression, alongside psychotherapy, is the standard of care in our department. Recent meta-analyses have developed consistent evidence that psychotherapy and ADM have additive effects, and that combined treatment is more effective than either alone, in both short- and longer-term interventions, and especially for moderate-to-severe depression (Cuijpers *et al.*, 2023; Fukumori *et al.*, 2024). Medication, as per our protocol (Perry *et al.*, 2020), was therefore applied to all participants, thus potentially improving outcomes and eliminating the potential design confound of medication *vs.* no medication.

Depression

Treatment was associated with significant improvement in depressive symptoms on both the HRSD-17 and the BDI. As the measurement models used many observations, which smooths out extreme values, they produced smaller, but highly robust, effect sizes compared to using a pre-post pair of individual scores, as previously demonstrated in Perry *et al.* (2020; see Table 1). Thus, as expected, the mean intake HRSD-17 (24.70) was greater than the mean modeled intake score (17.43).

Primary measure of depression: HRSD-17

Most participants (80%) showed improvements on the HRSD-17; however, only 30% attained sustained recovery. This number increased to 36% when examining only those who, per protocol, gave >4 years of follow-up. Furthermore, the confidence interval included the hypothesized figure of 50%. We suggest interpreting these numbers as 30% or 36% with long-term follow-up, definitely entering sustained recovery; however, additional studies with longer treatments and follow-up periods are required to determine if the final figure is closer to 50%.

Detecting sustained recovery requires two attributes: first, that a cut-off for recovery is attained, and second, that temporal factors indicate that it is sustained, *i.e.*, that future recurrences are unlikely. We chose an HRSD-17 cutoff <6 for recovery (Frank *et al.*, 1991). We relied on modeling many observations over the proposed timeframe (1.5 years of psychotherapy *plus* 3 years of follow-up) to delineate whether the participant was consistently in sustained recovery with scores <6. This modeling has performed well in previous samples, both in correctly predicting observations beyond the point of sustained recovery (Perry & Bond, 2009) and in converging with other measures of recovery (Perry & Fowler, 2021). Nonetheless, if either the number of observations is small or time is short, as is the case with the participants who completed less than two years of observations, the model produces less stable results. The large number of observations and length of follow-up in the other participants make our findings robust.

Secondary measure of depression: BDI-II

The BDI-II data mirrored the findings with the HRSD-17, except that 1) the effect size was nearly twice as large, and 2) a larger proportion of patients entered sustained recovery (64% *vs.* 30%). Recovery on the BDI-II did not significantly predict recovery on the HRSD-17, due to a low PPV (43%), despite a high NPV (90%).

The correlation between the two measures of depressive

symptoms at intake, where the ranges of scores was more restricted (*i.e.*, more similar scores across participants prior to treatment), was less than at end of follow-up (r_s=.33 *vs.* .84). Others have noted discrepancies between the two measures (Richter *et al.*, 1998), something we also reported in a previous study (Perry *et al.*, 2020). We conclude that the two measures are complimentary but not interchangeable, reflecting variance due to observer-rated *vs.* self-report perspectives. Regrettably, this is not considered in most meta-analyses, wherein effect sizes are combined as if all measures are comparable.

Improvement in defensive functioning

Our design protected against biasing our results by having defense raters, independent of and blinded to treatment, rate defenses from transcripts presented in random order, blinded to session number. The first author did not participate in ratings, except in so-called Super-consensus meetings to which raters brought difficult examples to think through the function and appropriate defense. Furthermore, depressive symptoms were rated by different, independent research assistants on a regular basis and by prescribing physicians.

As predicted, our study showed a large improvement in ODF (ES=.85). This was at the higher end compared to other psychotherapy studies using the DMRS: ES=.48 (Babl *et al.*, 2019), .64 (calculated from Table 1 in Kramer *et al.*, 2010), .71 (Perry & Bond, 2012), .76 (Roy *et al.*, 2009), .77 (Hersoug *et al.*, 2005), 1.13 (Perry *et al.*, 2020), and 1.31 (Johanson *et al.*, 2011). The decrease in depressive defenses was moderately large (ES=−.62), constituting most of the decrease in immature defenses. This effect was somewhat larger than in two other studies examining change in depressive defenses following short-term therapy for depression: −.48 (Babl *et al.*, 2020), and −.55 (Perry *et al.*, 2020). The pattern of changes we found across the defense categories and levels was consistent overall with the hierarchy of adaptation: mature and obsessional defenses increased, immature defenses decreased, and hysterical and other neurotic defenses remained in the middle.

Roy *et al.* (2009) reported that 71% of their psychoanalytic sample improved on ODF. Our current study found that 80% improved, and 72% had decreased depressive defenses. However, considered together, this means that 20-30% of participants remained resistant to improvement in defensive functioning within these treatments and timeframes. Future attention is needed to identify potential moderators of this resistance, such as co-occurring physical or psychiatric disorders, childhood traumata (Perry & Bond, 2012), and severe adverse life circumstances.

Defenses and depression

While we found that both depression and defensive functioning improved significantly with treatment, our third hypothesis addressed the relationship between improvement and recovery. The correlations between the final values of each, after partialing out their initial values, were moderate for both ODF (−.62) and depressive defenses (.44), both in the predicted directions. These changes by 18 months preceded the overall change in depressive symptoms modeled over 4.5 years. As our HRSD-17 measurement model aimed to detect sustained recovery, we used all available observations. Therefore, we did not calculate and control for change in depressive symptoms from intake to 18 months, before examining subsequent change from 18 months to 4.5 years, which would be done in most mediational analyses (Kazdin, 2007). As noted above, prior studies have found that a change in ODF correlates with and predicts change in distress or depression after psychotherapy (Babl *et al.*, 2019; Johanson *et al.*, 2011; Kramer *et al.*, 2010; Perry *et al.*, 2020). Furthermore, Babl *et al.* (2020) and Perry *et al.* (2020) found consistent evidence that improvement in depressive defenses, and more broadly immature defenses, correlated with improvement in depressive symptoms among depressed patients. In addition, Babl *et al.* (2019) found this was not true in those with anxiety disorders, suggesting specificity for depression. Some authors have also found that mature defenses increase with treatment (Babl *et al.*, 2019; Johanson *et al.*, 2011; Perry *et al.*, 2020). Babl *et al.* (2020) also found a large, significant effect for change in mature defenses predicting change in depression, whereas Perry *et al.* (2020) found a small, insignificant effect. Thus, there is consistent evidence that improving ODF by decreasing depressive or overall immature defenses predicts decreases in depression, although the latter are based on fewer studies. Finally, the role of improvement in mature defenses warrants further research.

Only the present study and Perry *et al.* (2020) have examined recovery in ODF *vs.* recovery in depression, using clear cutoffs. Both found that recovery in ODF was associated with recovery or sustained recovery from depression. The present study found a large association for recovery in ODF (OR=11.33) but a lower and non-significant association for recovery in depressive defenses (OR=2.0). Each defense identified in an interview contributes to the calculation of ODF, but far fewer are specifically depressive defenses. The latter estimate, therefore, is less stable and more prone to a type II error. Having multiple assessments of depressive defenses over time would improve that stability and provide a better test. Finally, it is possible that the cutoff for recovery of depressive defenses needs further study and validation, and thus may benefit from refinement.

Future directions

A major future direction for this work is to determine whether improvement in defensive functioning mediates improvement in symptoms and functioning. Given that everyone uses defenses, regardless of diagnoses, studies should determine whether improvement in ODF mediates improvements in both heterogeneous and specific populations. For example, Minges (2019) showed that improvement in ODF mediated improvement in panic symptoms with either CBT or panic-focused psychodynamic psychotherapy. In addition, the issue of whether specific groups of defenses are mediators for specific symptom disorders is intriguing. The evidence for depressive defenses and depression is growing but not yet definitive. Future studies might include the DMRS-SR-30 (Di Giuseppe *et al.*, 2020) to control for potential effects of data perspective on the relationship to change in depressive symptoms. Theoretically, the neurotic defenses may correlate with specific anxiety disorders, which should be explored. Furthermore, both naturalistic cohort follow-up studies, wherein change is slow over longer periods of time, as well as psychotherapy studies, wherein change occurs more rapidly over shorter periods of time, are warranted. Individuals vary in the rate at which their defensive functioning changes due to their individual characteristics. Future studies should therefore examine moderators that affect the rate of change in ODF, such as past and ongoing adversity, financial and family burdens, and co-occurring psychiatric and medical disorders (Perry & Bond, 2012). These data would help predict which individuals might require early treatment modifications, such as a heavier emphasis on supportive in-

terventions, or those who might not respond adequately to shorter-term treatments. Finally, studies with very long follow-up (*e.g.*, 5 to 15 years or longer) may elucidate the timing and processes of development of healthy defensive functioning in relationship to important outcomes, such as symptoms, functioning, and life satisfaction, as other studies have demonstrated (Martin-Joy *et al.*, 2017; Perry & Bond, 2009; Perry & Fowler, 2021).

Limitations

Several limiting factors should be considered. Our design choice not to include a no-treatment *plus* placebo control group, although done on humanistic grounds, limits the interpretation of findings: we cannot disentangle naturalistic improvements from those brought about by psychotherapy and ADM. The small sample size also affected the stability of estimates for smaller groups of defenses, such as the defense levels and depressive defenses, compared to ODF, which includes all defenses. We did not examine participant moderators and predictors, therapist characteristics, or therapy-process variables potentially related to defense change. The list of such factors is likely to overlap with that of treatment-resistance in general, regrettably beyond the scope of this report. Our sample included participants with moderate to severe depressive profiles, limiting the generalizability of our findings to less severe cohorts. Finally, our exploratory examination of the relationship between changes in defenses and depression by treatment type was constrained by low power, affecting both type I and II error rates. Nonetheless, the relative consistency of the pattern of correlations from the treatment sub-groups to the whole sample indicates that it met heuristic aims, suggesting that defenses may play a role in depression outcome regardless of psychotherapy type.

Conclusions

Improvement across the hierarchy of defense levels, reflected in higher ODF, leads to a general positive effect on symptoms and other outcomes in a variety of studies (Johanson *et al.*, 2011; Kramer *et al.*, 2010; Perry & Bond, 2012; Perry & Fowler, 2021). While evidence is gathering that ODF may act as a mediator of change in some outcomes (Kramer *et al.*, 2010; Babl *et al.*, 2019), this study provides robust but only partial evidence that the group of eight depressive defenses plays a more specific mediating role in depression.

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