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Running Head: Long-Term Change in Defenses and Coping

Change in Defense Mechanisms and Coping Patterns over the Course of Two-Year-Long  
Psychotherapy and Psychoanalysis for Recurrent Depression: A Pilot Study of a Randomized

Controlled Trial

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## Abstract

Very little research has been conducted so far to study the potential mechanisms of change in long-term active psychological treatments of recurrent depression. The present pilot randomized controlled trial aimed to determine the feasibility of studying the change process occurring in patients over the course of two-year long dynamic psychotherapy, psychoanalysis and cognitive therapy, as compared to clinical management. In total,  $N = 8$  outpatients presenting with recurrent depression, 2 patients per treatment arm, were included. All patients were randomly assigned to one of the four treatment conditions. Defense mechanisms and coping patterns were assessed by using validated observer-rated methodology based on transcribed, semi-structured follow-along independent dynamic interviews. Results indicated that, whereas some patients in the active treatments changed on the symptomatic levels, some others remain unchanged over the course of their two-year long treatment. However, with regard to potential mechanisms of change in these patients, changes in defense mechanisms and coping patterns were revealed to be important processes over time in successful therapies, and to a lesser extent in less successful treatments. No change was found either on outcome or on the process measure for the control condition, *i.e.*, clinical management. These results are discussed along with previous data comparing change in defense mechanisms and coping over the course of treatments.

Key-Words: Defense Mechanisms; Coping; Cognitive Therapy; Psychodynamic Psychotherapy; Psychoanalysis; Recurrent Depression; Observer-Rated Methodology

CHANGE IN DEFENSE MECHANISMS AND COPING PATTERNS OVER THE  
COURSE OF TWO-YEAR-LONG PSYCHOTHERAPY AND PSYCHOANALYSIS FOR  
RECURRENT DEPRESSION: A PILOT STUDY OF A RANDOMIZED CONTROLLED  
TRIAL

**Introduction**

Recurrent depression is one of the most frequently diagnosed mental disorders in psychiatry, with several well-studied therapy options, such as cognitive, psychodynamic, and interpersonal therapies, as well as pharmacological treatment. One of the problems in psychotherapy research on depression is the recurrent course of the disorder. According to Arnow and Constantino (2003), 75% of all patients diagnosed with depression present a recurrent course, and 20%, a chronic course. This ubiquity of recurrence suggests the importance of longer treatments aiming at recovery from depression. According to APA Practice Guidelines for Depression (2000), three treatment phases are identified for depression: (1) acute treatment aiming at symptom reduction or reduction of etiological factors such as interpersonal conflicts (e.g., Elkin et al., 1989; Hayes, Feldman, Beevers, Laurenceau, & Cardaciotti, 2007; Schramm, et al., 2007); (2) continuation aiming at relapse prevention (e.g., Bondolfi, et al., 2010; Teasdale, Segal, Williams, Ridgeway, Soulsby, & Lau, 2000); and (3) maintenance of gains, aiming at full recovery from depression (e.g., Klein et al., 2004; Mueller et al., 1999). Regarding this latter treatment option, very little empirical data exists concerning long-term intensive treatment of recurrent depression, and in particular, concerning the notion of recovery. This encompasses not only significant symptom reduction (by 50% of the symptom level at intake, usually called treatment response), but also the attainment of a sustained time below the clinical cut-off of the measure. More relevant to the current inquiry, long-term psychodynamic treatments and psychoanalysis of recurrent

depression have very rarely been studied in a randomized controlled design. This lack of research also applies to long-term cognitive therapy.

Parallel to the dearth of research into outcomes of different forms of long-term psychotherapy for recurrent depression, there is also very little research investigating the question of potential mechanisms of change in treatments aiming at recovery from depression (Perry & Bond, 2009, 2012). The over-arching concept of adaptational process, encompassing defense mechanisms and coping processes, may be noted here (Cramer, 1998; Kramer, 2010a). Despite some conceptual overlap, both concepts represent operationalizations of the notion of adaptation: for coping, skills-based competencies; for defense mechanisms, the protection of the individual from possible intra-psychic conflicts (Kramer, 2010b). It is therefore worthwhile to use both concepts jointly to study the over-arching notion of adaptational processes. Similarly, the notion of adaptive skills (Badgio, Halperin, & Barber, 1999) encompasses a set of change mechanisms in patients found in both cognitive-behavioral and psychoanalytic treatment forms. From a comparative research perspective, defenses and coping have been studied jointly in only a few recent studies. Using a questionnaire-approach on pooled data sets, Connolly Gibbons et al. (2009) showed that change in compensatory or coping skills over the course of both psychodynamic and cognitive therapy predicted symptom decrease over treatment. However, as hypothesized, change in self-understanding was specific to dynamic psychotherapy. As underlined by the authors, their study suffers from its sole reliance on self-report measures, and the pooled sampling procedure, which is prone to biases. Using an observer-rated methodology to address some of the criticisms raised, Kramer, de Roten, Michel, and Despland (2009) showed in a naturalistic study focusing on early change in Short-Term Dynamic Psychotherapy for patients presenting with adjustment disorder ( $N = 32$ ) that, overall, no change in either coping skills or defense mechanisms was found over the initial 12 sessions of psychodynamic therapy. However, if the therapeutic

alliance was introduced into the model, the authors found an interaction effect: the better the therapeutic alliance over time, the better the coping over the initial sessions. This same effect was not present for defense mechanisms, which remained unchanged over the early sessions. In a study based on the same therapies, Kramer, Despland, Michel, Drapeau, and de Roten (2010) showed in psychodynamic psychotherapy processes that there was neither any change in the coping variable over the course of efficient 40-session psychodynamic psychotherapy, nor any effect in terms of the linkage with therapeutic outcome. However, overall defensive functioning, which did increase significantly over the course of psychodynamic psychotherapy, predicted outcome. The same methodology was applied on very brief psychodynamic intervention (four sessions) for a sample of  $N = 61$  ( $N = 50$ ), yielding both significant change in defensive functioning (Drapeau, de Roten, Perry, & Despland, 2003) and coping functioning (Kramer, de Roten, Drapeau, & Despland, submitted). Using the same methodology, Perry, Beck, Constantinides, and Foley (2009) showed a large effect for change in coping functioning in a single case of a severely depressed outpatient over the course of psychodynamic psychotherapy. Finally, in a psychopathology interview-study, Kramer (2010a) showed in a small sample of inpatients ( $N = 18$ ) in crisis intervention presenting with Bipolar Disorder, overall defensive functioning remained stable between crisis and post-crisis, whereas overall coping functioning collapsed during the crisis, before increasing in adaptiveness again at post-crisis assessment. All these studies are limited to short-term treatment or treatment durations of up to one year. Therefore, we do not know if we may validly generalize these conclusions to longer treatments. Perry (2001) showed for a single case a steady increase in overall defensive functioning over the course of psychodynamic treatment taking place over several years. Recently, Perry and Bond (2012) reported a study of long-term dynamic psychotherapy in which a moderate improvement in defensive functioning at 2.5 years of therapy was highly associated with the degree of improvement in

symptoms and functioning after 5 years. This was consistent with the potential role of defenses as mediators of change.

Other than these, to our knowledge, no data exist concerning the long-term evolution of defense mechanisms, in particular in comparison with coping skills on the one hand and other forms of psychotherapy, such as cognitive therapy or treatment as usual, on the other. We would expect that in long-term treatments, patients' coping and defensive functioning might increase even further than that observed in short-term treatments.

The present pilot study aims at addressing this gap in knowledge. As the study was conducted on a very small sample of patients presenting with recurrent depression, we did not formulate specific hypotheses other than the general oriented assumption that defense mechanisms and coping would improve over the course of active treatments and that these improvements would be associated with improvement on measures of symptoms and functioning, to the degree the latter improved.

## **Method**

### **Design**

The present pilot study is the first step in demonstrating the feasibility of a randomized controlled trial for recurrent depression. After the patients were screened and gave written informed consent for the study, they were randomly assigned to one of four treatment conditions: (1) Psychoanalysis, (2) Psychodynamic Psychotherapy, (3) Cognitive Therapy, or (4) Clinical Management. Randomization was performed using a computerized program. In total,  $N = 12$  patients were screened for the study;  $N = 8$  accepted the study and started treatment.

### **Participants**

Patients



All 8 patients were Caucasian, French-speaking outpatients who presented with DSM-IV (APA, 1994) recurrent major depression. Two were assigned to each treatment arm. Their mean age was 47.00 years ( $SD = 7.98$ ), 6 (75%) were female. Trained clinicians made the diagnoses using the Semi-Structured Clinical Interviews for DSM-IV (SCID-I and II; First et al., 2004; APA, 1994). Two patients presented with co-morbid anxiety disorders (Generalized Anxiety Disorder and Panic Attacks), and two patients presented with Paranoid Personality Disorder. The mean GAF at intake was 51.38 ( $SD = 4.69$ ).

### Therapists

Eight therapists participated in the study, two per treatment. Two (25%) were female, and all were Caucasian psychiatrists. Their clinical experience included basic psychiatric and psychotherapeutic training according to local norms and federal legislation, and specific training in their treatment model of recurrent depression.

### Raters

In total, 6 raters participated in the study. They received a formal training in the rating procedures employed; their final reliability after six months of training on 13 cases prior to the use of the rating for the present study was excellent, as presented below.

### Treatment Conditions

All treatments were carried out in accordance with a respective manual. Psychoanalysis was implemented using the couch at a frequency of three times a week (Robertson, 2002). Psychodynamic psychotherapy was conducted face-to-face, according the expressive-supportive form of psychodynamic psychotherapy (Luborsky, 1984) and took place twice a week. Cognitive therapy followed J. Beck's (1995) manual and was implemented at a frequency of once or twice a week. Finally, clinical management was implemented according to Csank (2002) and Novalis, Rojcewicz, and Peele (1993). We

considered the latter condition as the control condition. All patients were given pharmacological treatment according to the latter medical guidelines (Novalis et al., 1993).

### **Instruments**

*Defense Mechanism Rating Scales (DMRS; Perry, 1990; French translation: Perry, Guelfi, Despland, & Hanin, 2004).* The DMRS is an observer-rater scale assessing 28 defense mechanisms, in which the defenses are ordered based on the empirical hierarchical relationship to adaptation (Perry & Cooper, 1989). Seven levels, ordered according to the criteria of adaptiveness, are included, from the least adaptive to highly adaptive: (1) Action (acting out, passive aggression, hypochondriasis); (2) Borderline or major-image-distorting (splitting of self/object representations, projective identification); (3) Disavowal (denial, rationalization, projection) and autistic fantasy (for further computation, this defense is considered on level 3, even if conceptually distinct); (4) Narcissistic or minor image-distorting (omnipotence, devaluation, idealization); (5) Neurotic (repression, dissociation, reaction formation, displacement); (6) Obsessional (isolation of affect, intellectualization, undoing); and (7) Mature (affiliation, altruism, anticipation, self-assertion, humor, self-observation, sublimation, suppression). Quantitative scoring was used, yielding relative frequency scores (percentages) for each defense level, as well as an Overall Defense Functioning (ODF) score, computed by weighting the absolute frequency of the defenses by their level (see Perry & Henry, 2004). Validity and reliability for the DMRS was reported by Høglend and Perry (1998; see also Perry & Ianni, 1998; Perry, 2001). Hilsenroth, Callahan, and Eudell (2003) reported evidence for the reliability and validity of Overall Defensive Functioning. For the current study, reliability coefficients on 21% (7) of the ratings were established among raters and yielded satisfactory results in terms of intra-class correlation coefficients (ICC<sub>2,1</sub>; Shrout, & Fleiss, 1979), varying between .62 and .92 ( $M = .77$ ;  $SD = .11$ ). For these reliability analyses, the defensive level was the unit of analysis (7 categories).

*Coping Action Patterns (CAPRS; Perry, Drapeau, & Dunkley, 2005; French translation by Kramer & Drapeau, 2011)*. CAP is an observer-rating system assessing coping processes based on interview-transcripts. The rating scale encompasses 12 categories of coping (based on the comprehensive review by Skinner, Edge, Altman, & Sherwood, 2003). Three general domains have been identified (relatedness, competence, autonomy), encompassing each four categories (“families”) of coping. Also, according to Lazarus and Folkman’s (1984) distinction, six of the coping categories are conceived as coping with stress appraised as challenge (problem-solving, information-seeking, self-reliance, support-seeking, accommodation, negotiation), and the other six as coping with stress appraised as threat (helplessness, escape, delegation, isolation, submission, opposition). Each coping category may be broken down into three action levels (affective, behavioral, and cognitive). Therefore, a total of 36 coping processes are assessed by this instrument. For our study, we only used the 12 categories enumerated (*i.e.*, we did not utilize the affective, behavioral, and cognitive distinctions). Relative frequencies were computed for all coping processes. An Overall Coping Functioning (OCF) score can be computed (meaning the proportion of challenge-coping) which is the only score used for the present study. Empirical validation has been presented by D’Iuso, Blake, Fitzpatrick, and Drapeau (2009) and by Lewandowski et al. (in press) for the original English version, and by Kramer and Drapeau (2011), Kramer, de Roten, & Drapeau (2011), Kramer and Drapeau (2009), and Kramer, Drapeau, Khazaal, and Bodenmann (2009) for the French version used for this study. For the current study, reliability coefficients on 21% (7) of the ratings were established among trained raters and yielded satisfactory results in terms of intra-class correlation coefficients ( $ICC_{2,1}$ ; Shrout & Fleiss, 1979), varying between .62 and .87 ( $M = .74$ ;  $SD = .09$ ). These coefficients have been established on coping category as the unit of analysis (12 categories).

*Symptom Check-List-90-R* (SCL-90-R; Derogatis, 1994). This questionnaire includes 90 items measuring various psychological and somatic signs of distress; these items are scored using a Likert-type scaling ranging from 0 («not at all») to 4 («very much»). The present study only used the Global Severity Index (GSI, score ranging between 0 and 4), which is the mean of all symptoms scores. The clinical cut-off score is 0.80. The French validation study was carried out by Pariente and Guelfi (1990) and yielded satisfactory coefficients. For the present sample, Cronbach's alpha was  $\alpha = .98$  and the GSI at intake was on average 1.83 ( $SD = .72$ ); thus, the mean score is in the clinical range.

*Hamilton Depression Rating Scale* (HDRS; Hamilton, 1960; Guelfi, 1997, for the French version used in this study). This is a 21-item clinician-rated scale assessing depressive symptoms using a Likert-type scale ranging from 0 («symptom absent») to 4 («severe symptom»), or from 0 to 2 for some items. The sum score of 15 was defined as clinical cut-off representing clinical depression (Franck et al., 1991). French validation coefficients are sufficient for this scale (Guelfi, 1997). For the present sample, the sum score at intake was on average 17.25 ( $SD = 3.96$ ).

## **Procedure**

The questionnaires (SCL-90-R) were given to the patients at intake and after 6, 12, 18 and 24 months, as long as the treatment was carried on. In the case of termination, the assessment procedure was stopped. At each assessment, the patients underwent clinician-ratings of HDRS (see above) and a Dynamic Interview (DI; Fowler & Perry, 2005; Perry, Fowler, & Semeniuk, 2005) with a researcher-clinician not involved in the treatment (different from the therapists). The interviewer had five tasks to conduct a high quality DI: (1) Setting the interview frame, including work-enhancing strategies; (2) Offering support, including questions, support strategies, associations; (3) Affect exploration, including questions, reflections, clarifications, defense interpretations; (4) Trial interpretations,

including defense and transference interpretations; and (5) Formulating a synthesis. The DI lasted 50 minutes, with a focus on the «patient's life in general.» The DI as a research tool has developed from clinical practice of psychodynamic psychotherapy; these interviews benefit from high external validity, as patients tend to bring up similar processes as in an intake psychotherapy interview. The patients were given financial compensation equivalent to 70 USD per assessment.

All DI sessions were tape-recorded and transcribed according to the rules defined by Mergenthaler and Stigler (1997). Ratings were done based on the transcripts. Reliability of these ratings was established on 21% of all interviews, randomly chosen, for both scales. Both interviewers and raters were blind to the patient's treatment condition.

### **Data Analytic Strategy**

As this is a pilot study with low power, we only report raw scale scores per person. In addition, as exploratory statistical analyses, we performed three sets of analyses. We examined bivariate relationships using a series of partial correlations between the variables (Spearman rank, partialling out the initial values of each variable). We conducted a series of univariate contrasts (Student's *t*-test or Wilcoxon signed rank test, as appropriate) to assess the significance of the change per variable. Finally, we conducted a series of linear regression analyses predicting the effect of active treatment (Psychoanalysis, Psychodynamic, Cognitive-Behavioral) on each variable, using a dummy variable for active versus control treatment (coded as 1 vs. 0). Because in long-term follow-along studies data collection is subject to irregularities of time, missing data and/or attrition, as previously detailed (Perry & Bond, 2009), we calculated individual simple linear regression models for each subject for each measure, using all available observations (*i.e.*, intake, intermediate and discharge). We estimated the slope (rate of change) and the predicted scores at intake and last observed follow-up, from which we calculated raw change. These predicted initial and final values

were used in our correlational analyses. The program SAS was used for these computations. Because of the very small sample size, and because we had prior hypotheses about the direction of changes, we were more concerned with falsely rejecting true findings (Type II errors) than accepting false findings (Type I errors) and therefore decided to discuss findings at  $\alpha = .10$ .

## **Results**

### **Preliminary results**

Cases A and B (both psychoanalysis) completed two years of treatment at the frequency of 3 times a week on the couch. Cases C and D (both psychodynamic psychotherapy) completed two years of treatment at the frequency of twice a week (face-to-face). Case E (cognitive-behavioral therapy) completed two years of treatment at the frequency of once a week, case F (cognitive-behavioral therapy) completed 1 year 3 months of treatment at the frequency of once a week, until this patient left the country and ended treatment. Case G (clinical management) completed two years of treatment at the frequency of once a month, while case H (clinical management) completed one year of treatment at the frequency of once every second month, until leaving the country and interrupting the treatment. Cases C and D (both dynamic psychotherapy) and G (clinical management) continued treatment after the end of the second year; however, we do not report the data beyond the second year of treatment. One case (D) developed breast cancer during the psychotherapy, requiring chemotherapy, which remitted again during treatment.

Change in GSI correlated negatively with change both in Overall Coping Functioning (OCF;  $r = -.71$ ;  $p = .05$ ); when both initial values were partialled out this correlation rose to  $r = -.93$ ;  $p = .006$ . The correlation between changes in GSI and Overall Defensive Functioning was  $r = -.33$ ;  $p = .42$ , remaining virtually the same when when both initial values were

partialled out ( $r = -.35, p = .49$ ). Partialling out initial values, changes in ODF and OCF were not significantly correlated (Spearman rank  $r = -.01; p = .97$ ).

We examined the final values at the end of study, partialling out the initial values of both variables. GSI correlated significantly with Overall Coping Functioning (Spearman rank  $r = -.85, p = .01$ ), but not with Overall Defensive Functioning ( $r = -.59, p = .17$ ). The partial correlation between ODF and OCF at discharge, controlling for GSI at intake, was also non-significant ( $r = .64, p = .12$ ). In Table 3 are presented these analyses using hierarchical linear regression, entering the respective initial value first, followed by the change variable. In both models 1 and 2, changes in ODF and OCF were significant predictors of GSI.

### **Symptomatic change over the course of Two-Year-Long Psychotherapy and Psychoanalysis**

As shown in Table 1, three cases out of eight (A, C, E) remitted from depression, in that their score was under the clinical cut-off of the scales towards the end of the two-year treatment. For the three active treatments (psychoanalysis, psychodynamic therapy, and cognitive-behavioral therapy), one patient per treatment group may be considered a responder (*i.e.*, showed a significant decrease in symptomatology), whereas three other patients (one per active treatment condition) did not seem to change over the course of treatment. No symptomatic change was shown in the clinical management treatment group for both patients involved.

We examined the partial correlations between change on each pair of variables related to symptomatic distress, partially out their initial values. Because change on the Global Severity Index (GSI) correlated highly with change on depression (HDRS;  $r = .81, p = .01$ ), due to this high redundancy, only the GSI will be reported in the subsequent sections (correlation analyses using the HDRS found very similar results as the ones presented on the GSI). Univariate testing (paired *t*-tests) showed significant decrease for GSI over the course

of all treatments ( $t = -2.83$ ;  $p = .03$ ;  $d = .48$ ), but no change for HDRS ( $t = -0.90$ ;  $p = .40$ ;  $d = .38$ ). These data were normally distributed.

### **Change in Defense Mechanisms and Coping Patterns over the course of Two-Year-Long Psychotherapy and Psychoanalysis**

On average at intake, the patients presented an Overall Defensive Functioning score of 3.14 (SD = .53; model-based predicted value = 3.50; SD = .54) which is in the defensive range of disavowal (denial, projection, rationalization, and autistic fantasy), bordering on minor image-distorting defense (devaluation, idealization, omnipotence). On average at intake, the patients presented an Overall Coping Functioning score of .34 (SD = .19; model-based predicted value = 0.35; SD = .17), indicating a predominance of stress appraisal as threat.

Univariate testing of the amount of change in ODF change over the course of all treatments ( $t = 2.02$ ;  $p = .08$ ;  $d = 1.33$ ), but no change for OCF ( $S = 10.5$ ;  $p = .16$ ;  $d = .93$ ). These data were normally distributed, except for OCF, for which Signed Rank testing was used.

As shown in Table 2, three cases out of eight (A, C, E) changed with regard to their overall defensive and coping functioning. For the three active treatments, one patient per treatment type demonstrated a large amount of change in both coping and defensive functioning over the course of treatment. No change in these variables was found in the clinical management condition. In addition, for two patients in the active treatment types (case D in psychodynamic psychotherapy and case B in psychoanalysis), there was substantial (case D) or slight (case B) change in defensive functioning, but no change in overall coping functioning in these patients.

### **Prediction of change in defenses and coping by active treatments**



Regression analysis of change in OCF and ODF yielded different pictures: active treatments (psychoanalysis, psychodynamic, and cognitive-behavioral) vs. control treatment did not predict change in Overall Coping Functioning ( $\beta = .137$ ;  $F(1, 6) = .32$ ;  $p = .59$   $R^2 = .05$ ). However, active treatments compared to control treatment predicted change in Overall Defensive Functioning:  $\beta = 1.36$ ;  $F(1, 6) = 7.38$ ;  $R^2 = .56$ ,  $p = .03$ , amounting to 1.36 ODF points greater change. Active treatment remains predictive of a similar magnitude of change in ODF after controlling for initial level of ODF ( $\beta = 1.36$ ;  $F(2, 5) = 5.85$ ;  $R^2 = .50$ ,  $p = .06$ ).

### Discussion

Although our sample was very small, we have found tentative evidence of significant symptomatic improvement in this small sample over the 2-year course for three forms of active psychotherapy together compared to our control therapy. Three of six active cases—one in each condition—made large gains in most or all variables, whereas the clinical management control cases showed very few changes. Each active treatment produced remission on symptom dimensions in one case out of two, whereas the symptoms of patients in clinical management diminished to a lesser extent. For these cases change on the GSI was substantial—all 3 successful cases remitted by the end of treatment—while that on the HRSD was less so. We may hypothesize that the latter changes at a slower rate than the GSI in the two-year time frame of the study; such observations were also made in early empirical studies comparing different clinician-rated scales of depression (Montgomery & Asberg, 1979). This is also consistent with a study of long-term dynamic psychotherapy that found that the GSI recovered earlier than the HRSD, *i.e.* 25<sup>th</sup> percentile attaining recovery at 2.8 vs. 4.3 years, respectively (Perry & Bond, 2009).

When we examined hypothetical mechanisms of change in the eight cases, according to the models on adaptational processes (Cramer, 1998; Badgio et al., 1999), we found a similar pattern for change in defense mechanisms and coping, observed in the same 3 – as

above - out of 6 cases in the active treatments . For these three patients, it may also be said that the joint assessment of defenses and coping did not yield any discrepancies, suggesting that large improvement in one is associated with large improvement in the other. This result is in accordance with theoretical elaborations (Cramer, 1998) suggesting moderate to substantial overlap between defenses and coping. It is further supported by findings that summary variables OCF and ODF (Kramer, 2010a; Kramer, de Roten, Michel, & Despland, 2009) demonstrate moderate correlations ( $.31 < r < .45$ ). However, the current study found no inter-correlation—when controlling for the one outlier—between defenses and coping. This surprising result should be interpreted very cautiously, as the number of observations is very low. Furthermore, the process data related to defenses and coping may present high intra-individual variance, consistent with the conceptual underpinnings positing fluctuating processes over time. Our data may also support the hypothesis of the greater volatility of mood-related coping, as compared to personality-based defenses (Cramer, 1998; Kramer, 2010a/b). Other research suggests that as individuals improve substantially, these fluctuations may decrease and the trends become more stable, which appears to require more time to assess than allowed by the two-year time frame of our study (Perry, Beck, Constantinides & Foley, 2009; Perry & Bond, 2012/in press). More frequent assessment may help to control for this, but this was not feasible in the current long-term study. However, high (negative) correlations were found between symptom change and change in the adaptational indices OCF and ODF over time, even though not all of these correlations were significant. The correlational approach and the small sample size did not allow us to disentangle the probable interaction effect between early outcome and change in adaptational processes, which may be addressed using formal mediation analysis.

Finally, for the active treatments, we need to acknowledge that coping change was not related to the active vs. control condition ( $R^2 = .14$ , ns), whereas defensive change was ( $R^2 =$

.56,  $p = .03$ ). The fact that the majority of treatments included in the study were psychodynamic/psychoanalytic (4 out of 6 active treatments) may account for some of these results; these treatments explicitly address defensive functioning. However, ODF improved on both CBT cases as well. A different line of explanation may be that change in defensive functioning may result from a general change process found across psychotherapy approaches. More particularly, in two patients enrolled in either psychoanalysis or dynamic psychotherapy (cases B and D), there was some increase in overall defensive functioning, whereas no change was found for either coping or symptoms in these patients. Case D in particular had the serious event of diagnosis and chemotherapeutic treatment for Breast Cancer occurring in the second year of treatment. It can be hypothesized that even if recovery was not attained in these two cases over the course of therapy, some characteristics of personality functioning — in particular, the psychodynamic concept of overall defensive functioning — changed over the active psychodynamic/psychoanalytic treatment types. Nonetheless, the final values attained were still well below the neurotic range (*i.e.*,  $ODF < 5.0$ ), so though improved, they were not dynamically well yet. This fact that the final value of ODF was still in a range associated with depression (Hoglend and Perry, 1998) is consistent with the finding that there was not more overall improvement in HRSD. This is an important empirical divergence between specifically-treated defensive functioning in these treatments and the coping functioning, which in these cases remained unchanged. Alternately, the greater sensibility to change on ODF associated with psychodynamic/psychoanalytic treatments may also be due to specifics related to psychodynamic interview format; these patients were more familiar with such psychodynamic interventions than the patients in the cognitive therapy, and might have been able to use more adaptive defenses as time unfolded. This explanation is questionable, given the amount of change in ODF among those patients receiving CBT

treatments. Further research into the relationship between defensive and coping functioning is definitely warranted.

Our pilot study has some major shortcomings. First, we included very few patients, which prevented us from conducting higher-order statistics. Dosage (sessions) per unit of time was not the same across treatments which complicates formal between-group comparison in terms of effects. Also, the information gathered in the psychodynamic interview might be skewed favoring the expression of defense mechanisms, vs. coping. In order to increase feasibility and because most of the treatments stopped at the end of the second year of treatment, we did not report the data on the patients who pursued treatment after this time point. However, we need to underline that the present study shows the feasibility of long-term assessment, from the outcome and process perspective, of long-term treatments of recurrent depression. Maintenance of gains may be assessed using self-report questionnaires, but we were also able to show that specific personality-based process measures may help to refine our understanding of the potential mechanisms of change involved in the treatments studied.

We may tentatively conclude that active long-term treatments produce symptomatic change to some extent, but more importantly, change in fundamental psychological process characteristics, such as defense mechanisms and coping. These may, in turn, hypothetically operate as potential mechanisms leading to symptomatic change in these treatments. An absence of symptomatic increase in patients presenting with recurrent depression over the course of treatment was evident, but this absence does not necessarily imply that the process characteristics remain stable. In fact, the level of ODF at termination was still below neurotic or healthy-neurotic levels. Changes in the psychological processes represented by defensive functioning, for example, are important to take into account in the assessment of a patient's evolution, in particular with regard to prognostic purposes. In this regard, it may be hypothesized that such process changes in defensive functioning precede symptomatic

changes over treatment (Johansson & Høglend, 2007). The potential for symptomatic change in very symptomatic samples may be more evident in even later phases of therapy, if indeed preceded by sufficient change in defensive functioning.

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Table 1: *Symptomatic Change over the Course of Long-Term Psychotherapy and Psychoanalysis (N = 8)*

	intake	6 m	12 m	18 m	24 m
Psychoanalysis					
Case A					
GSI	0.86	1.44	0.08	0.02	.01
HDRS	14	18	3	2	0
Case B					
GSI	3.32	3.53	3.13	2.98	3.13
HDRS	18	16	13	15	17
Psychodynamic					
Case C					
GSI	1.78	1.18	1.12	1.30	0.91
HDRS	22	24	25	21	13
Case D					
GSI	1.52	1.42	1.13	0.72	1.59
HDRS	10	10	9	8	18
Cognitive-Behavioral					
Case E					
GSI	1.41	0.80	0.22	0.15	0.11
HDRS	15	4	1	10	1
Case F					
GSI	2.53	2.66			
HDRS	20	34			
Clinical Management					
Case G					
GSI	1.42	0.78	1.17	0.59	1.41

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HDRS	20	18	9	12	17
Case H					
GSI	1.84	1.55			
HDRS	19	15			

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*Note.* GSI: Global Severity Index from SCL-90-R. HDRS: Hamilton Depression Rating Scale.

Table 2: *Overall Coping and Defensive Functioning over the Course of Long-Term**Psychotherapy and Psychoanalysis (N = 8)*

	intake	6 m	12 m	18 m	24 m
<hr/> Psychoanalysis					
Case A					
ODF	3.15	4.33	5.59	4.75	4.19
OCF	.21	.39	.52	.64	.89
Case B					
ODF	3.22	4.29	3.46	3.76	3.67
OCF	.40	.25	.25	.38	.38
<hr/> Psychodynamic					
Case C					
ODF	3.40	5.07	4.40	4.61	5.10
OCF	.08	.67	.65	.36	.91
Case D					
ODF	2.00	3.82	4.33	3.55	4.64
OCF	.47	.36	.33	.25	.33
<hr/> Cognitive-Behavioral					
Case E					
ODF	3.61	4.47	4.59	4.60	4.88
OCF	.67	.67	.53	.62	.82
Case F					
ODF	2.63	3.02			
OCF	.18	.21			
<hr/> Clinical Management					
Case G					
ODF	3.39	3.90	3.96	3.40	3.44

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OCF	.50	.69	.62	.50	.55
Case H					
ODF	3.70	3.00			
OCF	.17	.33			

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*Note.* ODF: Overall Defensive Functioning; OCF: Overall Coping Functioning

Table 3: *Hierarchical Linear Regression predicting raw change in GSI*

Models	$\beta$	df	$F$	$p$	$R^2$
Model 1		2, 5	9.17	.02	.79
Step 1: Initial ODF	-.73	1, 6	15.20	.01	.47
Step 2: Change in ODF	-.23	2, 5	7.46	.04	.32
Model 2		2, 5	4.97	.06	.67
Step 1: Initial OCF	-1.59	1, 6	4.21	.09	.03
Step 2: Change in OCF	-1.51	2, 5	9.43	.03	.63

*Note.* GSI: Global Severity Index; ODF: Overall Defensive Functioning; OCF: Overall

Coping Functioning