# Negative mood regulation expectancies as a mechanism of change in cognitive therapy for depression

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**Abstract**

The present study investigates the effects of negative mood regulation expectancies (NMRE) on symptom severity of depression in two cognitive therapies for depression. The sample included the first 146 consecutively recruited patients from a randomized controlled trial. Patients received 22 sessions of either cognitive behavioral therapy (CBT) or exposure based cognitive therapy (EBCT). They completed the Beck Depression Inventory and Negative Mood Regulation Scale at baseline and treatment termination, as well as after sessions seven and 14. Multilevel modeling was applied. We found a significant between-patient effect of NMRE on symptom severity of depression, when NMRE within-patient effects were set to random. There was no significant interactive effect of the between-patient NMRE with type of treatment. However, a significant moderation effect of the within-patient NMRE effect by treatment condition on depression severity was detected, with patients receiving CBT benefiting more from improvements in NMRE. Together, these results empirically support NMRE as a relevant mechanism of change in cognitive therapy for depression.

*Keywords*: Negative mood regulation expectancies, change mechanisms, depression, exposure-based cognitive therapy, cognitive-behavioral therapy.

## Clinical Impact Statement

In the present study, we aimed to determine if negative mood regulation expectancies (NMRE) might be a meaningful mechanism of change in cognitive therapy for depression. Improvements in NMRE over the course of treatment were associated with lower depression severity, when controlling for NMRE average level during therapy. This preliminary evidence for NMRE as a mechanism of change in the treatment of depression suggests that cognitive-behavioral therapists should try to target and enhance NMRE during therapy. Future studies will need to replicate the results with larger and balanced patient and therapist samples in order to reliably account for therapist effects.

Cognitive-behavioral therapy (CBT) has proven to be effective in the treatment of depression, even outperforming pharmacotherapy in the long run. However, a meaningful number of patients does not show beneficial effects (Cuijpers, 2017, Cuijpers et al., 2018). While CBT is the treatment presenting the greatest empirical support, its overall impact remains modest. Some patients only partially respond, some completely fail to respond, and many experience relapses (Moras, 2006; Wojnarowski, Firth, Finegan, & Delgadillo, 2018).

One strategy to improve CBT for depression is the identification of outcome-relevant change mechanisms that can be targeted in treatment (Crits-Christoph, Gibbons, & Mukherjee, 2013; Doss, 2004). In the last years, there have been several theoretical and empirical attempts to identify such processes and mechanisms of change, especially of cognitive nature: Clark and Beck (2010) suggest that the reduction of dysfunctional attitudes and negative beliefs in mental schemes is one of the primary mechanisms of change in CBT for depression. Also, patients' improvement in cognitive restructuring has been associated with better long-term outcome after cognitive therapies for depression (Gómez Penedo et al., 2020). Furthermore, reducing cognitive-behavioral avoidance such as denial of stressful situations (Ottenbreit & Dobson, 2004) and increasing patients' self-efficacy (Bandura, 1989; Backentrass et al., 2016; Bigman et al., 2016) have been supported as relevant change mechanisms in CBT for depression (Gómez Penedo et al., 2020). Fitzpatrick, Whelen, Falkenström, and Strunk (2019) found that in patients with interpersonal problems, social anxiety, or social-skill deficits, changes in negative cognitions were associated with greater therapeutic gains. In patients with negative evaluations of their social skills, changes in cognitions were further strongly related to symptom change (Fitzpatrick, Whelen, Falkenström, & Strunk, 2019).

Additionally, some emotion-related variables, such as emotion regulation have been postulated as the main mechanism of CBT for depression (Pascual-Leone, 2009). Several

studies support this idea, showing that emotion regulation is associated with better psychotherapy outcomes (Gómez Penedo et al., 2020; grosse Holtforth et al, 2012). Considering that negative mood is a core symptom of depression and therefore particularly relevant for its treatment, patients' ability to regulate and cope with emotions might be a particularly relevant change mechanism for the treatment of depression. During the last years, several studies with nonclinical samples have shown that negative mood regulation plays an important role for a number of psychological variables. Also NMRE has shown an important role in variables such as substance use, coping strategies, mood, anxiety, and stress symptoms (Backentrass et al., 2006). NMRE are defined as people's beliefs that they can alleviate the negative moods they experience by themselves (Catanzaro & Mearns, 1990). Such beliefs vary across individuals and are associated with different coping strategies for bad mood (Catanzaro & Greenwood, 1994, Altan-Atalay, 2018). Previous studies have shown that positive NMRE predicted the use of more adaptive coping strategies as well as a lower negative impact of stress (Catanzaro, Wasch, Kirsch, & Mearns, 2000; Thorberg & Lyvers, 2006; DiMauro, Renshaw & Kashdan, 2016). Kassel and colleagues (2007) found that NMRE predicted subsequent depression and anxiety in a student sample. Additionally, Fergus and Bardeen (2016) report that NMR expectancies moderated the association between unrealistic emotional goals and depressive symptoms, with the emotional goal of happiness being significantly related to depressive symptoms only in the context of low NMRE.

So far, only few studies investigated the association between NMRE and outcome in clinical samples. Backentrass and colleagues (2006) found that changes in NMRE correlated significantly with changes in depression severity during therapy. Siegel et al. (2017) found that changes in NMRE during treatment correlated with changes in depressive symptoms in a sample of war veterans. Both studies suggest NMRE to be a promising mechanism of change in the treatment of depression, however, they also present some methodological shortcomings

which should be addressed in future research: First, they did not account for the nested structure of the data (Raudenbush & Bryk, 2002). As repeated measures of the outcome variables were nested within patients, the assumption of independence of observations is violated, which is a necessary precondition for parametric analysis. To counter these shortcomings, multilevel models are recommended for an unbiased estimation of the outcome variables' evolution and the mechanisms' effects rather than correlation analysis (Hox & Maas, 2005). Second, it is worth noting that a mechanism of change can be associated with outcome in at least two ways (Zilcha-Mano & Errázuriz, 2015): One, when it is generally higher or lower for some patients relative to others and two, as it shifts in individual patients over time. The former describes between-patient effects, while the latter describes within-patient effects (Curran & Bauer, 2011).

Taking these considerations into account, the present study examines the effects of NMRE in two cognitive therapies for depression (EBCT and CBT). We aim to determine if there are significant between- and within-patient effects of NMRE on depression severity, using multilevel analysis. We hypothesize that: (hypothesis #1) there is a significant between-patient effect of NMRE on outcome, i.e., lower NRME will be associated with greater overall severity, (hypothesis #2) there is a significant within-patient effect of NMRE on outcome, i.e., positive variations in NMRE will be associated with better outcome, (hypothesis #3) there is a significant between-patient and within-patient interactive effect of NMRE on outcome, i.e., patients with a lower overall level of NMRE will benefit more from an increase of NMRE during treatment, and (hypothesis #4) treatment condition will neither moderate the between-patient effect nor the within-patient effect of NMRE on depression severity.

## Methods

### Participants

The sample included the first 146 consecutively recruited patients from a randomized controlled trial (grosse Holtforth et al., 2019). Patients were treated free of charge (due to local ethical regulations) in a psychotherapeutic outpatient clinic of a Swiss University. For being eligible, patients had to be at least 18 years old and meet diagnostic criteria for major depression. This meant that all participants had to score above 14 points on the German version of the Beck Depression Inventory-II (BDI-II; Hautzinger, Keller, & Kühner, 2006) and below 13 points on the World Health Organization Well-Being Questionnaire (WHO-5; Henkel et al., 2003). Patients gave their written informed consent to participate in the study before being randomized to receive either CBT (n=71) or EBCT (n=75). They were each assigned to the next therapist on a time capacity list.

The majority of patients were female (57%) with an average age of 40.6 years (SD=11.44). They reported not to be in a romantic relationship (either single, separated, divorced or widowed; 58.7%) at the beginning of treatment. Most participants had either received professional training (42.8%) or university education (41.3%) as their highest level of education. There were no significant differences in patients' sociodemographic or clinical characteristics between the two treatment conditions.

Treatment was provided by 25 therapists, most of whom were female (92%) with a mean age of 31.4 years (SD = 5.14). Each therapist saw an average of 5.8 patients within the trial (SD = 2.8). All therapists were master-level psychologists who were either in postgraduate psychotherapy training or had already completed it.

To limit the potential confounding effect of the treatment condition and therapist, all therapists delivered both treatments and were assigned an equal number of patients per condition. This trial was registered at Clinical-Trials.gov (identifier: NCT01012856) and approved by the Cantonal Ethics Committee.

**Measures**

**Negative Mood Regulation Scale.** The Negative Mood Regulation Scale (NMRS; Catanzaro & Mearns, 1990) is a 30-item self-report questionnaire to assess affect regulation, including the frequency and success of attempts to terminate negative moods. It comprises five different types of items: general, cognitive, behavioral-alone, behavioral-social, and behavioral-unspecified. Responses are reported on a 5-point Likert scale from 1 (strongly disagree) to 5 (strongly agree), with higher scores reflecting more positive expectancies about the ability to regulate moods during times of upset. Analyses indicate that the scale is unifactorial with an excellent internal consistency (α= .88; Catanzaro & Mearns, 1990). In the current sample, the NMRS presented good internal consistency at baseline (α= .74).

**Beck Depression Inventory.** The Beck Depression Inventory (BDI-II; Hautzinger et al., 2006) is a self-assessment tool consisting of 21 items representing different depressive symptoms (e.g., feeling sad, guilty, hopeless, etc.) rated on a 4-point Likert scale from 0 to 3, with higher scores reflecting more depression severity. The German version of the BDI-II has previously shown satisfactory reliability, validity, and sensitivity to change (Kühner, Bürger, Keller, & Hautzinger, 2007). In this sample, Cronbach's alpha of the BDI-II total score was .88 at baseline.

NMRS and BDI-II were applied at baseline, after seven and 14 sessions, and at post-treatment.

**Treatments**

Patients were randomly assigned to 22 weekly sessions of either CBT or ECBT. Treatment duration was predefined by the RCT and based on the usual length of cognitive-behavioral therapy in the outpatient setting in Switzerland. After termination, they were offered three optional booster-sessions at 3-, 6-, and 12-months follow-up.

**CBT.** This treatment condition was based on Beck's cognitive therapy (Beck, Rush, Shaw, & Emery, 1979). Therapists used techniques such as psychoeducation, treatment

socialization, behavioral activation, and cognitive restructuring by means of identifying and challenging maladaptive cognitions and assumptions of the patient. Finally, they focused on consolidating therapeutic gains and preventing relapses. To stress differences between the treatment conditions, therapists were explicitly asked not to use techniques specific to emotion-focused therapy (see below) while providing treatments in the CBT condition.

**EBCT.** This treatment condition followed the principles of assimilative integration (Castonguay, Newman, & grosse Holtforth, 2018) in that emotion-focused aspects (e.g., emotional exposure) were integrated into the general CBT framework with the aim of fostering emotional processing (grosse Holtforth et al., 2012; Hayes, 2015). Greenberg and Pascual-Leone (2006) defined emotional processing (EP) by four central components: (1) awareness and arousal of emotion, (2) enhancement of emotion regulation, (3) reflection on emotion, and (4) transformation of emotion through activation of adaptive emotional experience. The general idea behind the fostering of EP is that the therapist helps the patient to (a) decrease avoidance and ruminative patterns, (b) decrease the fear of negative and positive emotions, (c) explore and disrupt depressive patterns, and (d) develop new, more adaptive patterns. As a result, the patient learns to uncouple negative emotions and cognitions and to develop more adaptive associations between stimuli, responses, and meaning (grosse Holtforth et al. 2019).

**Adherence**. To check and to optimize treatment adherence, therapists completed intervention checklists, as well as session-report questionnaires after each session. One scale of the session report assessed the use of EBCT-specific interventions, whereas another scale assessed the use of CBT-specific interventions. In addition, this was assessed by two observers blind to treatment conditions on the basis of video recordings of single sessions (all sessions were recorded with the patients' prior consent). The adherence scale for CBT was based on the Collaborative Study Psychotherapy Rating Scale (CSPRS; e.g., Hill, O'Grady,

& Elkin, 1992) and EBCT-R-specific items were generated from the manual. The instrument contained 21 items across 2 subscales: CBT-specific interventions (13 items; α = .76) and EBCT-specific interventions (8 items; α = .64). In order to test inter-rater reliability, 21 randomly chosen videos were rated by two independent raters and showed acceptable adherence ratings, that is, ICC > .60 (ICC[2,1]: CBT = .78, EBCT-R = .66; Shrout, Spitzer, & Fleiss, 1987) (grosse Holtforth et al. 2019).

**Analytic strategy**

**NMRE main effects (Hypothesis #1 and Hypothesis #2).** Following recommendations to disaggregate between- and within-patient effects in psychotherapy (Falkenström, Finkel, Sandell, Rubel, & Holmqvist, 2017), we ran multilevel models with BDI-II as outcome variable including (i) NMRE average across treatment (grand-mean centered) as a level-2 predictor of the intercept (Hypothesis #1, between-patient effect), and (ii) NMRE as a level-1 random predictor (Hypothesis #2, patient-mean centered; within-patient effect). The between-patient effect represents the association of the general levels of NMRE and BDI, while the within-patient effect shows the association of variations in NMRE with outcome. This effect shows how each patient deviates at different times from his own mean, and how these deviations are associated with changes in BDI.

To test for therapist effects, we first ran a three-level (repeated measures nested within patients, nested within the therapist) unconditional model with BDI as outcome variable and calculated Intraclass Correlations Coefficients (ICC). The therapist accounted for less than 1% of the variance in the outcome variable (ICC= .001), suggesting that it is no meaningful source of variance (Westra, Constantino, & Antony, 2016). Based on this empirical finding and a recent simulation study showing that when studying mechanisms of change, three-level models do not outperform two-levels models, and might even bias the estimations in the context of unbalanced designs (Falkenström, Solomonov, & Rubel, 2020), we decided to use

a two-level structure (Level 1: within-patient effects; Level 2: between-patient effects) for the main analyses of the study. To estimate NMRE between- and within-patient effects we used the following equations:

*Level-1 Model*

$$BDI_{ij} = \beta_{0j} + \beta_{1j} * (\text{NMRE\_Within}_{ij}) + r_{ij}$$

*Level-2 Model*

$$\beta_{0j} = \gamma_{00} + \gamma_{01} * (\text{NMRE\_Between}_{j}) + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + u_{1j}$$

At Level 1, BDI scores at time *i* for patient *j* were predicted by patient *j*'s estimated BDI score in a session with his average NMRE score ($\beta_{0j}$) and by the effect of fluctuations around his own mean in NMRE ($\beta_{1j}$, NMRE within-patient effect). The random effect $r_{ij}$ allowed patient *j* to vary at time *i* from his estimated score in BDI. At Level 2, $\beta_{0j}$ was predicted by the estimated BDI score in a session with an average NMRE level for participants with an average NMRE across treatment ($\gamma_{00}$) and by the effect of the level of NMRE across treatment ($\gamma_{01}$), while $\beta_{1j}$ was predicted by the average sample within-patient effect of NMRE fluctuations ($\gamma_{10}$). The random effects $u_{0j}$ and $u_{1j}$ allowed the patients *j* to deviate from the average intercept and within-patient NMRE effect.

To control for possible temporal confounders of the within-patient effects, we followed recommendations by Wang and Maxwell (2015) as well as Falkenström et al. (2017), replicating the model adjusting for time effects by including time in sessions (centered at baseline) as an extra (fixed) level-one predictor. The reason why the detrending method is not the main analysis is that including time in within/between models is a very restrictive and conservative criterion (Falkenström et al. 2017). For this reason, Falkenström and colleagues (2017) suggest first running the model without including the time variable and then, as a sensitivity analysis, running the model again including time. If the effects remain

significant, there is more robust evidence in support of the mechanism studied (Falkenström et al. 2017).

**Cross-level interaction (Hypothesis #3).** To test for the cross-level interaction of between-patient and within-patient NMRE effects, we included NMRE between-patient effects (grand-mean centered) as a level two predictor of the NMRE within-patient effect on BDI, using the following equations:

*Level-1 Model*

$$BDI_{ij} = \beta_{0j} + \beta_{1j} *(\text{NMRE\_W}_{ij}) + r_{ij}$$

*Level-2 Model*

$$\beta_{0j} = \gamma_{00} + \gamma_{01} *(\text{NMRE\_B}_{j}) + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11} *(\text{NMRE\_B}_{j}) + u_{1j}$$

Here, $\gamma_{11}$ represents the effect of the level of NMRE across treatment on the within-patient effect of NMRE on BDI.

**Interactive effects of NMRE with treatment (Hypothesis #4).** To test for interactive effects of NMRE with treatment, we included treatment condition (EBCT=0; CBT=1) as a predictor of the intercept and the NMRE within-patient effect. Furthermore, we included the interactive effect of NMRE between-patients with treatment condition on the intercept. For this model, we used the following equations:

*Level-1 Model*

$$BDI_{ij} = \beta_{0j} + \beta_{1j} *(\text{NMRE\_W}_{ij}) + r_{ij}$$

*Level-2 Model*

$$\beta_{0j} = \gamma_{00} + \gamma_{01} *(\text{NMRE\_B}_{j}) + \gamma_{02} *(\text{Treatment}_{j}) + \gamma_{03} *(\text{NMRE\_BxTreatment}_{j}) + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11} *(\text{Treatment}_{j}) + u_{1j}$$

Here, $\gamma_{02}$ represents the effect of the treatment condition on the intercept ($\beta_{0j}$), while $\gamma_{03}$ represents the interactive effect of NMRE between-patients with treatment condition on

the intercept. Finally, $\gamma_{11}$ represents the effect of treatment condition on the NMRE within-patient effect on outcome.

## Results

### Sample Descriptives

In Table 1, we present the sample descriptive statistics for both process (NMRE) and outcome (BDI) variables at baseline and post-treatment. For BDI, results show that on average patients had a depression score of 25.48 (*SD* = 9.01) at intake. When measured after session 22, patients had decreased their BDI scores to 9.45 (*SD* = 8.23), on average. Regarding NMRE, patients exhibited a pre-treatment general level of 2.81 (*SD* = 0.54), while their mean at post-treatment was 3.49 (*SD* = 0.61).

In order to test for baseline distribution differences in the subsamples of the two treatment conditions (i.e. CBT and ECBT), we conducted independent sample *t* tests for each variable (i.e. NMRE and BDI). As presented in Table 1, neither the NMRE nor the BDI general score levels showed evidence for differences between the groups.

### Unconditional time-as-only predictor models

To characterize NMRE and BDI evolution over the course of treatment, we conducted unconditional time-as-only-predictor (TAOP) models. These models include time in sessions (centered at session 11, i.e., middle of the time series) as the only level-1 predictor. We tried models including both, linear as well quadratic trajectories (both as fixed and random effects).

When using NMRE as outcome, a model with the linear time slope set as random outperformed a model with a fixed slope, $\chi^2(2) = 27.42$, $p < .001$. Furthermore, a model

including a quadratic term set as fixed presented a better model fit than the one only including the linear term, $\chi^2(1) = 18.37$, $p < .001$. A model including both the linear and quadratic term set as random did not converge. The final TAOP model (i.e., with a linear term as random and the quadratic term as fixed) showed both significant linear effects, $\gamma_{10} = 0.03$, SE= 0.003, $t(126) = 12.37$, $p < .001$, and quadratic effects, $\gamma_{20} = -0.001$, SE= 0.0003, $t(249) = -4.37$, $p < .001$, of time on NMRE. Thus, patients on average increased their NMRE by 0.03 units per session (linear time effect), while they decelerated those increases by 0.001 units per session (quadratic time effect. The comparisons of the ICC of the linear fixed effect model (ICC= .55) and the linear random effect model (ICC= .66) showed that an extra 8% of variance was explained by that random effect model. The model including the fixed quadratic term (ICC= .68) increased the explained variance by another 2% compared to the random linear effect model.

When using BDI as outcome, a model with a linear time slope set as random presented a better fit than a model with a fixed linear slope, $\chi^2(2) = 9.52$, $p < .01$. The models failed to converge when including a quadratic term as either fixed or random. Thus, the final model (i.e., the TAOP with time as linear random effects) showed that patients significantly reduced their BDI scores by 0.71 units per session, $\gamma_{10} = -0.71$, SE= 0.04, $t(134) = -18.27$, $p < .001$. Comparing the ICC of the linear fixed effect model (ICC= .49) and the linear random effect model (ICC= .57), we found an extra 8% of variance explained by that random effect.

**Disaggregating between- and within-patient effects of NMRE on BDI**

**NMRE main effect models**. A conditional model, including both NMRE between- and within-patient effects as predictors of the BDI scores and with the within-patient effect set as fixed, significantly improved the model fit when compared to the fully unconditional model ($\chi^2$ (2) = 245.7, $p < .001$). When setting the within-patient effect as random,

improvement in the model fit approached significance ($\chi^2$ (2) = 5.29, $p$ = .07), when compared to the model with the fixed within-patient effect. Based on these results, we decided to keep the model with the random NMRE within-patient effect, as the final model of the analysis. This model showed a significant between-patient effect of NMRE on symptom severity of depression, $\gamma_{01}$ = -8.37, *SE* = 1.08, 95% CI [-10.48, -6.26], $t$(139) = -7.77, $p$ < .001. As an effect size measure, we calculated standardized coefficients for the significant effects (Lorah, 2018). This effect size measure suggested that a one-standard deviation greater NMRE level during treatment was associated with a 0.38 standard deviations lower BDI during treatment. Furthermore, the model showed significant within-patient effects of NMRE on BDI scores, $\gamma_{10}$ = -13.00, *SE* = 0.81, 95% CI [-14.59, -11.41], $t$(65) = -16.05, $p$ < .001. A one-standard deviation greater increase in patient's NMRE scores from their individual mean was associated with a 0.50 standard deviation lower score in the BDI, adjusting for the between-patient association between BDI and NMRE. We then ran the same models adjusted for time effects by including time in sessions as a predictor variable (i.e., detrending). This model showed a significant session effect on BDI, when adjusting for NMRE between- and within-patient effects, $\gamma_{20}$ = -0.50, *SE* = 0.04, 95% CI [-0.58, -0.43], $t$(374) = -12.94, $p$ < .001. On average, patients reduced their BDI score by .50 units per session. Additionally, this model showed that when adjusting for time effects, both the between-patient NMRE effects, $\gamma_{01}$ = -7.58, *SE* = 1.02, 95% CI [-9.59, -5.58], $t$(142) = -7.41, $p$ < .001, and the within-patient NMRE effects, $\gamma_{10}$ = -6.73, *SE* = 0.85, 95% CI [-8.39, -5.07], $t$(110) = -7.96, $p$ < .001, remained significant. At the same time, the detrended model demonstrated significant improvement in the model fit ($\chi^2$ (1) = 139.85, $p$ < .001), compared to the random-effects model.

**Cross-level interaction model.** The cross-level interaction model including general NMRE levels during treatment as the only level 2 predictor of the within-patient effect is

summarized in Table 2. Results showed that overall NMRE levels across treatment did not have a significant effect on the within-patient NMRE effect on BDI, $\gamma_{11}$ = -2.56, *SE* = 1.78, 95% CI [-6.05, 0.93], *t*(70) = -1.44, *p* = .15. Moreover, this model did not significantly improve the model fit of the main effect model including the within-patient NMRE effect as random ($\chi^2$ (1) = 1.96, *p* = .16).

**Interactive effect model of NMRE with treatment.** As shown in Table 2, there was no significant interactive effect of NMRE between-patient with type of treatment, $\gamma_{03}$ = -0.47, *SE* = 2.18, 95% CI [-4.73, 3.79], *t*(136) = -0.22, *p* = .83. However, we found a significant effect of treatment condition on the within-patient NMRE effect on symptom severity of depression, $\gamma_{11}$ = -3.57, *SE* = 1.61, 95% CI [-6.72, -0.42], *t*(67) = -2.22, *p* < .01. Patients in the CBT condition had a 3.57-units larger within-patient NMRE effect on BDI (i.e., they benefited more from a greater positive fluctuation in NMRE). Nevertheless, when comparing this model to the main effect model (i.e., with within-patient NMRE effects as random), there was no significant improvement in model fit, $\chi^2$ (3) = 5.39, *p* = .15. Because NMRE and BDI-II were measured only four times during treatment, a cross-lagged model would only allow us to establish if, for example, the NMR levels in session 1 predict BDI-II in session 7. This does not appear to be a very intuitive association, which is why we decided to keep multilevel models as the analytical method of choice. Whereas they do not allow for inferences as robust as cross-lagged ones, they do present preliminary evidence for NMRE as a mechanism of change in therapy for depression (Falkenström et. al 2017).[1]

## Discussion

[1] Although the main analytic strategy was based on multilevel models, we also ran a random intercept cross-lagged panel model as a further sensitivity analysis (Falkenström et. al 2017). This model replicated the main results of our study, showing a significant effect of previous NMRE on subsequent BDI, Coefficient = -0.24, SE = 0.02, 95% CI [-0.28, -0.20], *p* <.001.

The aim of the current study was to examine the NMRE between-patient and within-patient effects on depression severity.

As expected, we found significant between-patient effects of NMRE on outcome, with higher NRME being associated with lower overall severity. This means that patients with a better NMRE showed lower levels of depression. This finding is consistent with previous research suggesting that overall problems in NMRE are associated with depressive symptoms (Catanzaro, Wasch, Kirsch, & Mearns, 2000; Kassel, Bornolova, & Mehta, 2007; Thorberg & Lyvers, 2006; DiMauro, Renshaw & Kashdan, 2016).

Results also confirmed a significant within-patient effect of NMRE on outcome, with positive variations in NMRE being associated with lower depression severity. This means that patients who improved their NMRE levels during treatment also reduced their depression levels over the course of treatment. These results are in line with previous studies associating NMRE changes with modifications in depression severity during treatment and follow-up (Backentrass et al., 2006; Siegel et al., 2017), and provide preliminary evidence in support of NMRE as a promising mechanism of change in cognitive therapy for depression. Although our models do not provide direct evidence of causality, including the between-patient component when estimating the within-patient effect allows for a better approximation of causality, since the stable characteristics of the patients are controlled for (Falkenström et al., 2017). Furthermore, the within-patient effects were replicated in the detrending models, remaining significant when controlling for time effects. This means that the effects were still significant after controlling for eventual temporal confounders (Falkenström et al., 2017).

Unexpectedly, there was a significant effect of treatment on the within-patient NMRE effect, suggesting that patients in the CBT condition benefited more than EBCT patients from improving there NMRE levels during treatment. We are unaware of any previous study comparing these effects in CBT versus other treatments (e.g., EBCT), however, some studies

found that changes in NMRE expectancies correlated with changes in depression during CBT treatment and could also predict further changes over the follow-up period (Backentrass et al., 2016). If these findings can be replicated in further research, future studies will need to analyze the mechanisms underlying these differential effects.

In addition to treatment, the within-patient effects of NMRE could vary depending on specific characteristics of the patients (i.e., patients with certain features benefiting more or less from improvements in NMRE; Gómez Penedo et al., 2019). Future research should explore patient characteristics as possible moderators to establish who, based on their characteristics, would particularly benefit from an improvement in NMRE. This could determine in which cases treatment should focus on the mechanism of NMRE, providing meaningful information for psychotherapy personalization via pre-treatment recommendations (Lutz et al., 2019).

This study presents several limitations that should be addressed in future research. First, all measures used were self-reported by the patient, which makes it more prone to social desirability (Ferrando & Anguiano-Carrasco, 2010) or shared-variance biases (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). It would be important to incorporate other sources of information (e.g., observer-coded process measures) in future studies. Moreover, in our study we did not include therapist effects as a third level in the model. However, as noted above recent simulation studies have shown that including therapist effect as a third level in these models, may represent a source of bias, in the context of an unbalanced design (i.e., when the number of patients per therapist is not constant; Falkenström et al., 2020). Second, our study lacks a cross-lagged analytical approach, and future research would benefit from gathering more frequent assessments of NMRE that would allow for cross-lagged models disaggregating within- between-patient effects. Third, future studies will need to replicate the results with larger and balanced patient and therapist samples in order to reliably

account for therapist effects. Fourth, although therapists were explicitly asked not to use specific emotion-focused therapy or EBCT techniques while providing treatments in the CBT only condition, both treatment conditions shared common techniques. Also, this way of providing interventions may result in a limited generalizability of our results to the original versions of CBT and ECBT. It would be relevant to continue studying NMRE effects in other treatment types.

Limitations notwithstanding, this study provides preliminary evidence for between- and within-patient NMRE effects on symptom severity of depression in cognitive therapy for depression. In particular, NMRE improvements during treatment were associated with lower depression severity, supporting NMRE as a promising mechanism of change to be targeted in CBT. Improvements in NMRE would be particularly desirable in patients receiving CBT only as opposed to ECBT, considering the significant effect of treatment on the within-patient NMRE effect,.

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Table 1

*Mean, Standard Deviation, and Treatment Comparison of the Relevant Variables at Baseline and Posttreatment*

| | Total sample | CBT | EBCT | Comparison CBT vs. EBCT | | |
|---|---|---|---|---|---|---|
| *Variables* | M(SD) | M(SD) | M(SD) | *t* | *d.f.* | *p* |
| *BDI* | | | | | | |
| Baseline score | 25.48(9.01) | .37(9.26) | 24.66(8.75) | -1.15 | 143 | .25 |
| Posttreatment score | 9.45(8.23) | | | | | |
| *NMRE* | | | | | | |
| Baseline score | 2.81(0.54) | 79(0.56) | 2.83(0.52) | 0.40 | 138 | .69 |
| Posttreatment score | 3.59(0.61) | | | | | |

*Note.* BDI = Beck Depression Inventory; NMRE = Negative Mood Regulation Expectancies; CBT = Cognitive Behavioral Therapy; EBCT = Exposure-based cognitive therapy.

Table 2

*Results of the Unconditional Model, Negative Mood Regulation Main Effect Model, Cross-level Interaction Model, and Interactive Effects Model of NMRE by Treatment*

| | BDI level | | NMRE within-patient effect on BDI scores | | BDI rate of change during treatment | |
|---|---|---|---|---|---|---|
| Fixed Model Effects | γ | *SE* | γ | *SE* | γ | *SE* |
| *Fully unconditional model* | | | | | | |
| Intercept | 16.85** | 0.59 | | | | |
| *Main effects model* | | | | | | |
| Intercept | 16.91** | .51 | -13.00** | .81 | | |
| NMRE level (between-patient) | -8.37** | 1.08 | | | | |
| *Model comparison* | | | $X^2(4) = 1.96, p < .001$ | | | |
| *Main effects model detrended* | | | | | | |
| Intercept | 21.88** | .62 | -6.73** | .85 | -0.50** | 0.04 |
| NMRE level (between-patient) | -7.58** | 1.02 | | | | |
| *Model comparison* | | | $X^2(1) = 139.85, p < .001$ | | | |
| *Cross-level Interaction Model* | | | | | | |
| Intercept | 16.90** | .51 | -12.85** | .83 | | |
| NMRE level (between-patient) | -8.09** | 1.10 | -2.56 | 1.78 | | |
| *Model comparison* | | | $X^2(1) = 1.96, p = .16$ | | | |
| *Interactive Effects Model of NMRE by Treatment* | | | | | | |
| Intercept | 16.37** | .71 | -11.24** | 1.12 | | |
| Treatment | 1.08 | 1.01 | -3.57* | 1.61 | | |
| NMRE level (between-patient) | -8.19** | 1.41 | | | | |
| NMRE level X Treatment | -0.47 | 2.18 | | | | |
| *Model comparison* | | | $X^2(1) = 1.96, p = .16$ | | | |

*Note.* We only reported the random effects model for the Main Effects of NMRE, as it was selected as the final model. Treatment: EBCT=0, CBT=1; NMRE = Negative Mood Regulation. ** $p < .001$, * $p < .05$