

Attention Bias Modification in Remitted Depression Is Associated With Increased Interest and Leads to Reduced Adverse Impact of Anxiety Symptoms and Negative Cognition

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Abstract

Using a computational network approach, we reanalyzed data from a randomized controlled trial of attention bias modification (ABM) tasks on residual depression symptoms. The main aim was to characterize the symptom-to-symptom changes following ABM. ABM was associated with improvements in interest, which was in turn associated with improvements in other depression symptoms. Although there were no changes in the global network strength following ABM, the comparison with symptom change in the ABM and control group suggests that ABM leads to a reduction of the association between anxiety, depressed mood, and guilt. Findings suggest that reduction in depression symptoms following ABM may have been set in motion by increased interest and involvement in everyday activities, leading to a reduction of the adverse impact of anxiety and negative cognition. ABM may be more effective in patients in whom these symptoms are prominent.

Keywords

attention bias modification, depression, network analysis, attentional bias

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Depression is among the most prevalent and severe mental disorders and is related to substantial individual suffering (Cuijpers & Schoevers, 2004; Demyttenaere et al., 2004). Both currently (Gotlib, Krasnoperova, Yue, & Joormann, 2004) and previously depressed patients (Joormann & Gotlib, 2007) show an *attentional bias* toward negative stimuli. This negative attentional bias can be demonstrated in visual probe tasks, in which depressed individuals are faster to respond when probes replace negative stimuli compared with when probes replace positive stimuli (Peckham, McHugh, & Otto, 2010). This is also the case for never-depressed individuals who are at high risk because of a family history of depression (Joormann, Talbot, & Gotlib, 2007; Kujawa et al., 2011), implying attentional bias as a causal risk factor for depression debut, recurrence,

and consolidation (Beck & Bredemeier, 2016; Disner, Beevers, Haigh, & Beck, 2011; Mathews & MacLeod, 2005).

Attention bias modification (ABM) tasks aim to reduce attentional bias by automatically directing attention toward more positive stimuli (Hakamata et al., 2010). The sparse studies in depression have shown that ABM has the potential to reduce residual symptoms after depression (Beevers, Clasen, Enock, & Schnyer, 2015; Browning, Holmes, Charles, Cowen, & Harmer, 2012; Wells & Beevers, 2010; Yang, Ding, Dai, Peng, &

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Zhang, 2015). Because residual symptoms after a depressive episode are among the strongest predictors for recurrence (Paykel, 2008), ABM might prove effective in preventing relapse. However, as argued in a number of meta-analyses (see e.g., Cristea, Kok, & Cuijpers, 2015; Mogoase, David, & Koster, 2014), effect sizes are small and definitive conclusions are limited because of small sample size and poor trial methodology employed in many studies. Even so, emotional vulnerability seems to decrease in studies in which ABM is followed by a successful shift in attention bias (Grafton et al., 2017; but see also Cristea et al., 2017).

Recently, in a large preregistered randomized clinical trial of effects of ABM on residual symptoms in depression (Jonassen et al., 2018), a two-week ABM program resulted in a positive change in attentional bias and a small but statistically significant effect on depression symptoms as measured by the clinician-rated interview Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960). Importantly, the degree of symptom improvement increased with degree of positive bias modification within the ABM group, in line with the mechanism emphasized by Grafton et al. (2017).

To increase the effect of ABM, researchers are urged to explore novel ways to conceptualize and modify attentional bias in depression (Koster & Bernstein, 2015; Mogg, Waters, & Bradley, 2017). Another approach is to reconsider the model of depression vulnerability and how outcome of ABM is assessed. Clinical effects of ABM for depression commonly have been evaluated on the basis of sum scores on depression rating scales, such as the HRSD or the Beck Depression Inventory-II (Beck, Steer, & Brown, 1996). These scales have high construct validity and are extensively used in the clinic, but aggregating symptoms into a sum can conceal potentially important insights (Fried & Nesse, 2015). Individual depression symptoms are differentially related to risk factors, impaired psychological functioning, biomarkers, and antidepressant efficacy (for a review, see Fried, Nesse, Zivin, Guille, & Sen, 2014). It is likely that ABM is associated with changes in specific depression symptoms but that global assessments of symptom change masks this effect. Obtaining this knowledge may inform further development of ABM. Along these lines, the present study reexamines data demonstrating the effect of ABM in residual depression (Jonassen et al., 2018) on the symptom level using recent innovations in theoretical and computational network analysis (Borsboom, 2017; Borsboom & Cramer, 2013; Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012).

Over the past two years, the network approach has been increasingly used to understand mental disorders as systems of interacting symptoms (Hofmann & Curtiss, 2018; Hofmann, Curtiss, & McNally, 2016; McNally,

2016). According to this approach, each depressive symptom is not reflective of a “depressive disorder” but possesses independent causal powers that influence other symptoms (e.g., insomnia causes fatigue); symptoms are not merely passive indicators of an underlying disease (Borsboom & Cramer, 2013). Thus, a depressive episode emerges because of the pairwise associations between depression symptoms. These associations can be visualized in a *network model*, in which symptoms are represented by *nodes* and associations are represented by *edges* between the nodes (Borsboom, 2017).

Network analysis can determine which symptoms are the most *central* (i.e., influential) on the basis of the amount of influence that flows from one symptom to another (Borgatti, 2005; Valente, 2012). Central symptoms are those that have many strong relations with other symptoms, and the presence of a central symptom easily spreads to other symptoms, potentially producing a *cascade* of activation (Borsboom & Cramer, 2013; Valente, 2012). In depression, low mood and loss of interest appear to be the most central symptoms (Fried et al., 2017), along with anxiety (Fried, Epskamp, Nesse, Tuerlinckx, & Borsboom, 2016). Networks with many strong connections between symptoms (dense networks) are more likely pathogenic than networks characterized by weaker connections—that is, more “vulnerable” (Borsboom, 2017; Borsboom & Cramer, 2013; Fried et al., 2017; Heeren & McNally, 2018).

Individuals with depression show a more densely connected network compared with healthy subjects (Pe et al., 2015), and this network predicts difficulty recovering from a depressive episode (van Borkulo et al., 2015; but see also Schweren, van Borkulo, Fried, & Goodyer, 2018). Simulation studies show that in dense networks, only a minimal of worsening in a central symptom may trigger a downstream cascade of symptoms. This might lead to a “vicious cycle” of negative cognition and symptoms (Teasdale, 1988; Wichers, 2014), with a depressed state as the result (Cramer et al., 2016).

We used network analysis to characterize the symptom changes following ABM. We believe that traditional approaches for conceptualizing symptom changes (i.e., sum scores of depression scales; Fried et al., 2017) could be occluding symptom changes following ABM. Our main aim is thus to provide a comprehensive exploration of ABM’s impact on residual depression and evaluate whether ABM changes specific symptoms or other aspects of the symptom network. First, we estimated the initial symptom network before ABM and compared symptom centrality with previous network studies on depression to assess the generalizability of the current sample. Second, we estimated networks on the basis of symptom changes from pre to post ABM

and examined whether specific symptoms are improved or the symptom cascade is changed (i.e., the dynamic changes in symptom-symptom relations over time). We examined whether ABM is associated with improvements in individual symptoms and decreases the connectivity between symptoms. Finally, we examined symptom networks post ABM and hypothesized that ABM weakens connections among symptoms, that is, ABM reduces the overall network connectivity.

Method

Participants

We obtained data from a randomized double-blind placebo-controlled trial of ABM on residual depression symptoms among patients with remitted depression. The study was preregistered at ClinicalTrials.gov (NCT02648165). For details regarding design, methods, and results, see Jonassen et al. (2018). ABM training led to significantly greater decrease in clinician-rated but not subjective-rated symptoms of depression compared with the control condition.

The sample included 322 participants recruited mainly from an outpatient clinic in the Department of Psychiatry, Diakonhjemmet Hospital in Oslo. Participants were also recruited from other clinical sites, by local advertisements, and via social media. Inclusion criteria were the presence of a remitted major depressive disorder, age between 18 and 65 years, and fluency in Norwegian. Exclusion criteria were the presence of current or former neurological disorder, substance use disorder, attention-deficit disorder, head trauma, psychosis, or bipolar disorder. These criteria were assessed using the Mini-International Neuropsychiatric Interview (Sheehan et al., 1998). A total of 302 patients were included per protocol. Participants provided written informed consent, and the study was approved by the Regional Ethical Committee for Medical and Health Research for Southern Norway (2014/217/REK sør-øst D).

Measures and materials

Symptoms. Depression symptoms were measured using HRSD (Hamilton, 1960), which is an observer-rated interview with 17 items. The internal consistency reliability of HRSD was acceptable in the current sample ($\alpha = .77$).

Attention bias modification task. The ABM was a computerized, validated visual dot-probe procedure (Browning et al., 2012), presented on laptop computers (14" HP Elitebook 840, 1600 × 900, 8 GB, Intel Core i5-4310U). Paired images of faces (the stimuli) were presented followed by one or two dots (a probe), which appeared behind one

of the stimuli. Participants were required to press one of two buttons as quickly as possible to indicate the number of dots in the probe. Stimuli were pictures of emotional faces (Karolinska Directed Emotional Faces; Lundqvist, Flykt, & Öhman, 1998) of three valences: positive, neutral, or negative (angry and fearful). The task comprised 96 trials with equal numbers of the three stimulus pair types. There were equal numbers of trials in which the stimuli were randomly presented for 500 or 1,000 ms before the probe was displayed. Stimuli from two valences were displayed in each trial of the task in one of the following pairing types: positive-neutral, positive-negative, and negative-neutral. Probes were located behind positive (valid) emotional stimuli in 87% of the trials in the training condition. Thus, when completing training, participants should learn to deploy their attention toward positive stimuli and in this way develop a relatively more positive attentional bias. The control condition was identical in every respect other than the location of the probe, which was located behind the positive (valid) stimuli in only 50% of the trials.

Procedure

We explained to participants that the study aimed at examining “attention focus, how this changes over time, and how this is related to mood and depression symptoms” (the specific rationale underlying ABM was not explained). After providing written consent, participants were allocated randomly to either training or control condition by an independent lab technician (not involved in the day-to-day collection of data) who prepared laptop computers to deliver either training or control treatment according to a randomization list in a 1:1 ratio. A trained administrator, who was blind to condition allocation, assessed depression symptoms and demonstrated ABM. Participants were then instructed to do the task at home twice a day for two weeks (28 sessions in total), before returning for a second assessment of depression symptoms.

Network analysis

Network estimation. We used a graphical Gaussian model (GGM) to estimate the networks. In the presented networks, nodes represent depression symptoms and edges represent conditional independence relationships between nodes when controlling for the effects of all other nodes (Epskamp, Borsboom, & Fried, 2018). It is common to regularize GGMs via the graphical LASSO (least absolute shrinkage and selection operator). The graphical LASSO estimates a maximum likelihood solution in which the likelihood is penalized for the sum of absolute parameter estimates (Epskamp & Fried, 2018; Friedman, Hastie,

& Tibshirani, 2008). First, it computes regularized partial correlations between pairs of nodes, thereby limiting the sum of absolute partial correlation coefficients. This eliminates spurious associations (edges) attributable to the influence of other nodes in the network. Second, it shrinks trivially small associations to zero, thereby removing potentially “false positive” edges from the graph and producing a sparse graph comprising only the strongest edges. We used the R package *qgraph* (Epskamp et al., 2012), which automatically implements the graphical LASSO regularization, in combination with an extended Bayesian Information Criterion (EBIC) model selection (Foygel & Drton, 2011). In this approach, 100 different network models are estimated with different degrees of sparsity. Then, the model with the lowest EBIC value is selected given a certain value of the hyperparameter γ ; this procedure strikes a balance between including false-positive edges and removing true edges. The hyperparameter γ is usually set between 0 and 0.5 (Epskamp et al., 2018). As the value of γ nears 0.5, the EBIC will favor a simpler model that contains fewer edges. As the value of γ nears 0, the EBIC will favor a model with a greater number of edges. Given the exploratory nature of the study, we set γ to 0 to maximize the sensitivity.

A baseline network was estimated for the whole sample in which edges represent the partial correlations between each of the 17 HRSD items before ABM. This network highlights possible pathways between depression symptoms—in other words, the symptomatologic landscape in which ABM in some way must operate. Node placement was determined by Fruchterman and Reingold’s (1991) algorithm, whereby nodes nearer to the center of the graph tend to have the strongest connections with other nodes. A thicker edge denotes a larger association. Green edges represent positive partial correlations, whereas red ones represent negative partial correlations.

We estimated two networks on the basis of symptom-change scores from baseline to post ABM (post ABM minus baseline). A positive symptom score indicates an improvement in symptom severity from pre to post, and a negative score indicates a worsening in symptom severity from pre to post. These networks give a visual presentation of how changes in one symptom relate to changes in other symptoms. First, we estimated a symptom-change network for the whole sample, including a node representing ABM condition (control = 0, training = 1). This network indicates whether ABM is related to changes in specific symptoms. Second, we estimated symptom-change networks for each group (training vs. control) separately, indicating whether ABM induces a changed symptom cascade from baseline to post ABM. Node placement in these training and control networks were determined by the node layout from the baseline network to ease comparison.

Finally, we estimated networks separately for each group (training vs. control), in which nodes represented symptom scores post ABM. Node positioning in these networks was also according to the baseline network layout. We compared these networks in global strength, symptom centrality, and specific edge differences.

Centrality indices. For each network, we computed centrality indices to quantify the importance of each node (Opsahl, Agneessens, & Skvoretz, 2010). We focused on *strength* centrality, defined as the sum of the weights of the edges attached to that node, because previous network research in psychopathology has indicated that this is the most stable and reliable centrality metric (e.g., Beard et al., 2016; Bernstein, Heeren, & McNally, 2017). Higher values reflect greater centrality in the network. We created centrality plots that depict these values as *z*-scores for ease of interpretation.

We evaluated the stability of the centrality indices by using the R package *bootnet* (Epskamp et al., 2018) by implementing a subset bootstrap procedure (Costenbader & Valente, 2003). The procedure is described in the Supplemental Material available online.

Networks comparisons. Network differences were evaluated on the basis of global strength, defined as the weighted sum of the absolute connections within a network (Barrat, Barthélemy, Pastor-Satorras, & Vespignani, 2004). Higher values reflect greater interconnectivity among nodes. We used the *NetworkComparisonTest* (NCT) to test for differences between the training and control network in global strength. The NCT is a two-tailed permutation-based test in which the difference between two groups is calculated repeatedly (10,000 times) for randomly regrouped individuals (van Borkulo, 2016). This produces a distribution of values under the null hypothesis (i.e., assuming equality between the groups) that enables one to test whether the observed difference in global network strength differs significantly ($p < .05$) between groups.

To clarify potential significant differences between groups, we also tested whether edge weights were significantly different between groups for each edge of the networks. To do so, we relied on permutation-based tests using NCT (van Borkulo, 2016) and applied the Holm-Bonferroni correction to control for the large number of tests (i.e., minimizing the risk of Type 1 error).

Results

Participants

The majority of the participants were women (70%). Mean age for the whole sample was 40.9 years ($SD = 13.2$), and participants had on average experienced 4.1 ($SD = 5$) depressive episodes. Mean HRSD score at

Table 1. Demographic and Clinical Characteristics

Variables	Control (<i>n</i> = 149)	Training (<i>n</i> = 153)
Age, years, <i>M</i> (<i>SD</i>)	41.5 (13.6)	40.2 (12.7)
Sex (females), <i>n</i>	103	109
Educational level (ISCED), <i>M</i> (<i>SD</i>)	5.9 (1.2)	6.0 (1.1)
Use of SSRI/SNRI medication, <i>n</i>	43	38
Rumination score (RRS), <i>M</i> (<i>SD</i>)	49.5 (12.2)	51.4 (12.1)
Stroop inhibition-switching, seconds to complete, <i>M</i> (<i>SD</i>)	59.9 (15.5)	60.1 (14.4)
Number of depressive episodes, <i>M</i> (<i>SD</i>)	4.1 (4.6)	4.1 (4.9)
Comorbidity rate, %	61	62
HRSD total score, <i>M</i> (<i>SD</i>)		
Baseline	8.3 (5.1)	9.2 (6.0)
Post	8.8 (5.9)	8.3 (6.0)
Pre-post change	-0.5 (4.6)	1.1* (5.3)

Note: SSRI/SNRI = serotonin specific/serotonin-norepinephrine reuptake inhibitor; HRSD = Hamilton Rating Scale for Depression; MDD = major depressive disorder; ISCED = International Standard Classification of Education; RRS = Ruminative Responses Scale.

* $p < .01$ versus control group. All other differences (t tests and chi-square tests) were not statistically significant.

baseline was 8.8 ($SD = 5.6$). Twenty-seven percent of the participants used a serotonin-specific/serotonin-norepinephrine reuptake inhibitor antidepressant. Demographic and clinical characteristics of the participants are presented in Table 1. There were no significant differences between the training and control group except that HDRS scores from baseline to post ABM improved by 1.1 points ($SD = 5.3$, corresponding to 12% improvement) in the training group and worsened by 0.5 points ($SD = 4.6$, corresponding to 6% worsening) in the control group (Cohen's $d = 0.32$), as reported by Jonassen et al. (2018).

Baseline network

The baseline network is presented in Figure S1 (in the Supplemental Material). Depressed mood, interest, guilt, and anxiety have many strong edges and are centrally located in the network. Insomnia symptoms and other vegetative symptoms (e.g., weight loss, agitation, psychomotor retardation) are located in the periphery and primarily connected to the rest of the network through anxiety. Centrality indices (Figure S2 in the Supplemental Material) indicate that anxiety (somatic = 1.84), depressed mood (1.70), guilt (0.81), and interest (0.70) have high strength centrality, meaning that they are strongly connected to the rest of the network.

Symptom-change networks

Figure 1 presents the symptom-change network for the whole sample and strength centrality for each node. The ABM node represents ABM condition (0 = control, 1 = training). The other nodes represent change scores

from baseline to post ABM (positive score = improvement, negative score = worsening). This network indicates that ABM condition covaries with improvement in interest, that is, when ABM condition changes from control to training, interest improves. Improvement in interest is in turn related to improvement in other symptoms. Strength centrality index indicate that ABM is a relatively unimportant node (-0.60) compared with most other nodes in the network, but strikingly, its connection is to what appears as the second most central node in the network (interest = 2.12).

We conducted additional network analyses (i.e., post hoc) to examine whether changes in attentional bias scores were associated with changes in symptoms (see Figures S9–S14 in Supplemental Material). Overall, results showed that there were edges appearing between, on the one hand, changes in positive versus negative attentional bias and, on the other hand, anxiety (somatic) and insight.

Figure 2 denotes symptom-change networks and strength centrality for each node in the training group (Fig. 2a) and the control group (Fig. 2b). Depressive mood, low interest, and anxiety are closely related in both networks. Global network strength was significantly weaker for the training network (0.09) than in the control network (2.28, $p < .05$). Anxiety (somatic) is the most central symptom in the control network (2.32) and markedly more central than in the training network (0.24). Permutation-based tests of edge differences revealed significant differences between the two networks. Indeed, five of the edges present in the control network were absent in the training network. Specifically, interest was connected to guilt ($p = .01$), anxiety (somatic) was connected to suicidal thoughts ($p < .01$) and somatic symptoms ($p < .01$), agitation was

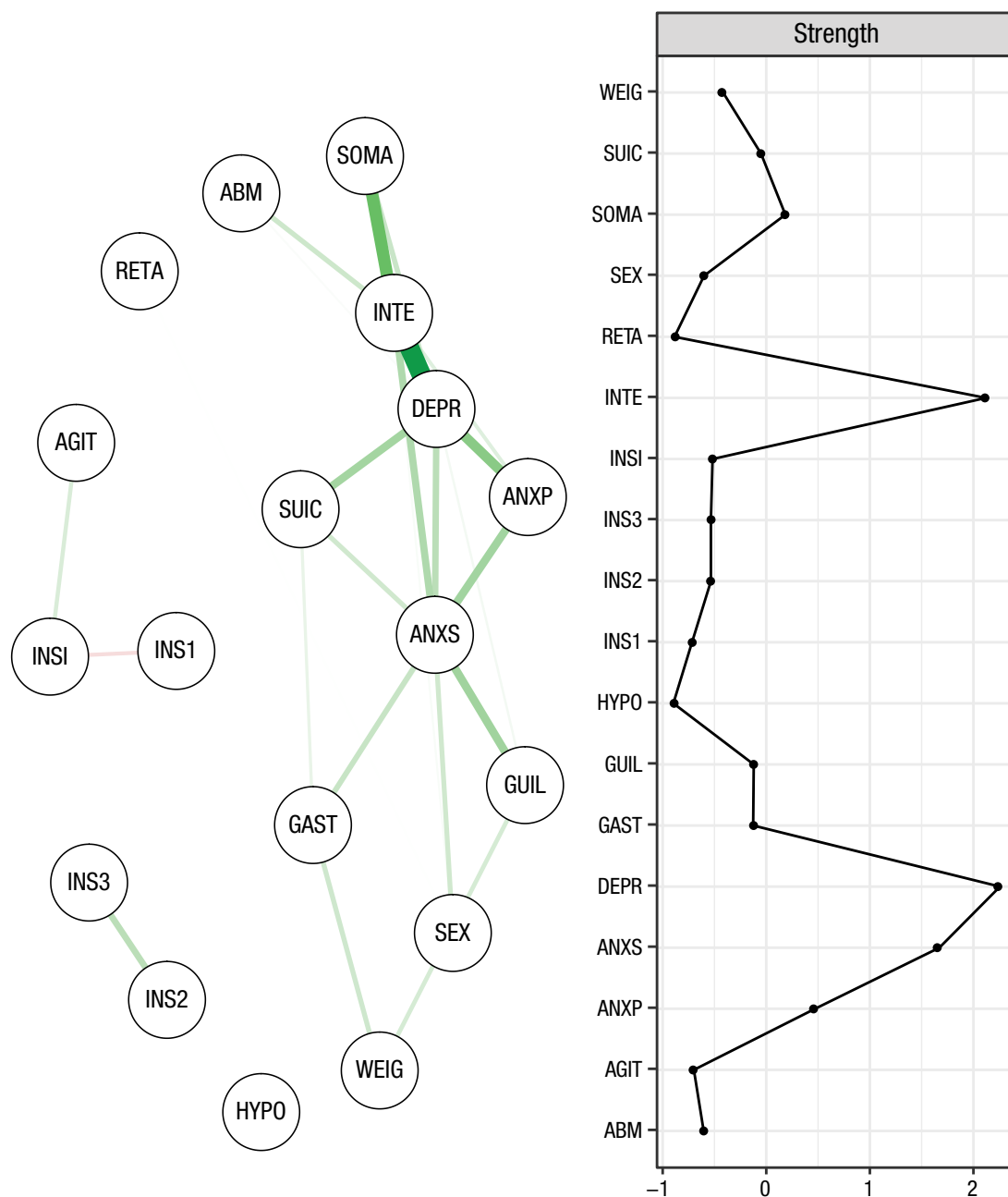


Fig. 1. Symptom-change network and strength centrality for the whole sample. Symptom nodes represent symptom severity changes on each Hamilton Rating Scale for Depression item from baseline to post ABM (post minus baseline). Associations are indicated by edge thickness between nodes (thicker edge = stronger association). ABM = attention bias modification condition (control = 0, training = 1); DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss.

connected to loss of insight ($p < .05$), and retardation was connected to loss of libido ($p < .05$). Guilt was also connected to anxiety (somatic) at borderline significance level ($p = .05$). No other edge was statistically different between the networks.

Post-ABM networks

Symptom networks and strength centrality for training and control groups post ABM are shown in Figure 3. There was no statistical significant difference in network density

a

Training

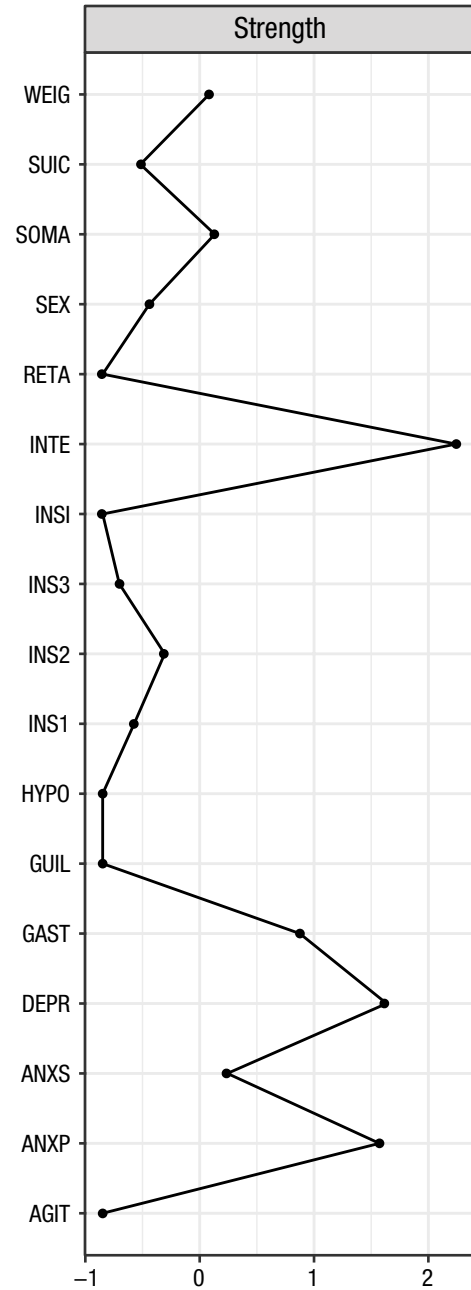
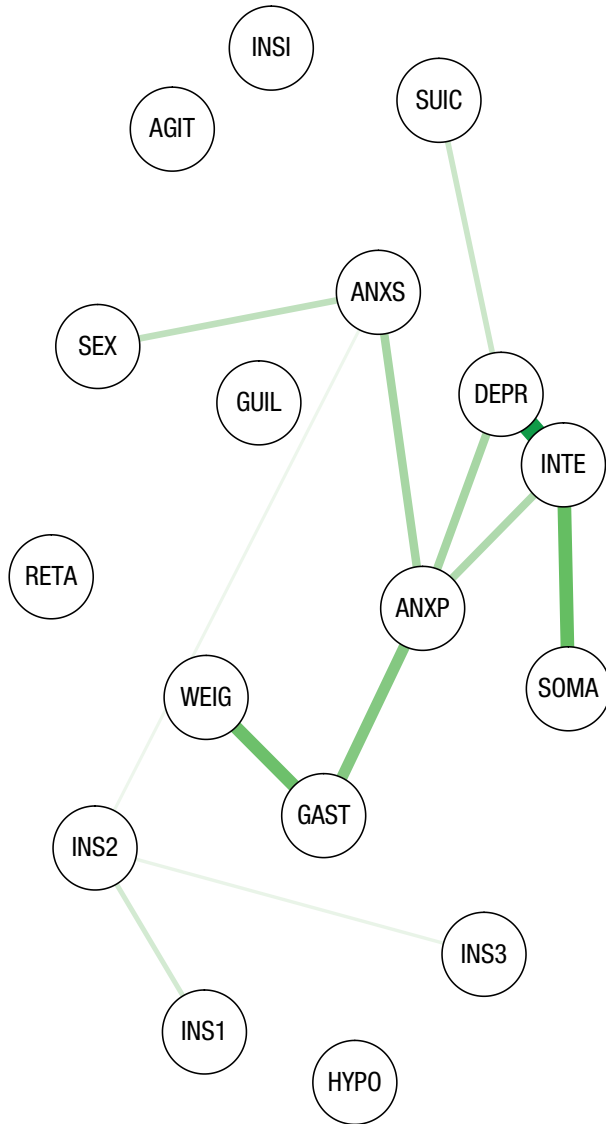


Fig. 2. (continued on next page)

($p = .62$) between the training network (global strength = 5.58) and the control network (global strength = 5.06). Centrality indices were highly similar. Permutation-based tests revealed several significant edge differences between the training and the control networks. In the control network, guilt was more strongly connected to anxiety (somatic; $p < .05$) and retardation ($p < .05$) and depressed mood more strongly connected to anxiety (psychic; $p < .05$). The training network had two edges present that

were not present in the control network: Interest was connected to gastro-intestinal symptoms ($p < .05$), and retardation was connected to late insomnia ($p < .05$). No other differences in edges were statistically significant.

Stability of the centrality indices

Stability analyses of the strength centrality index for all networks are depicted in Figures S3 through S8 in the

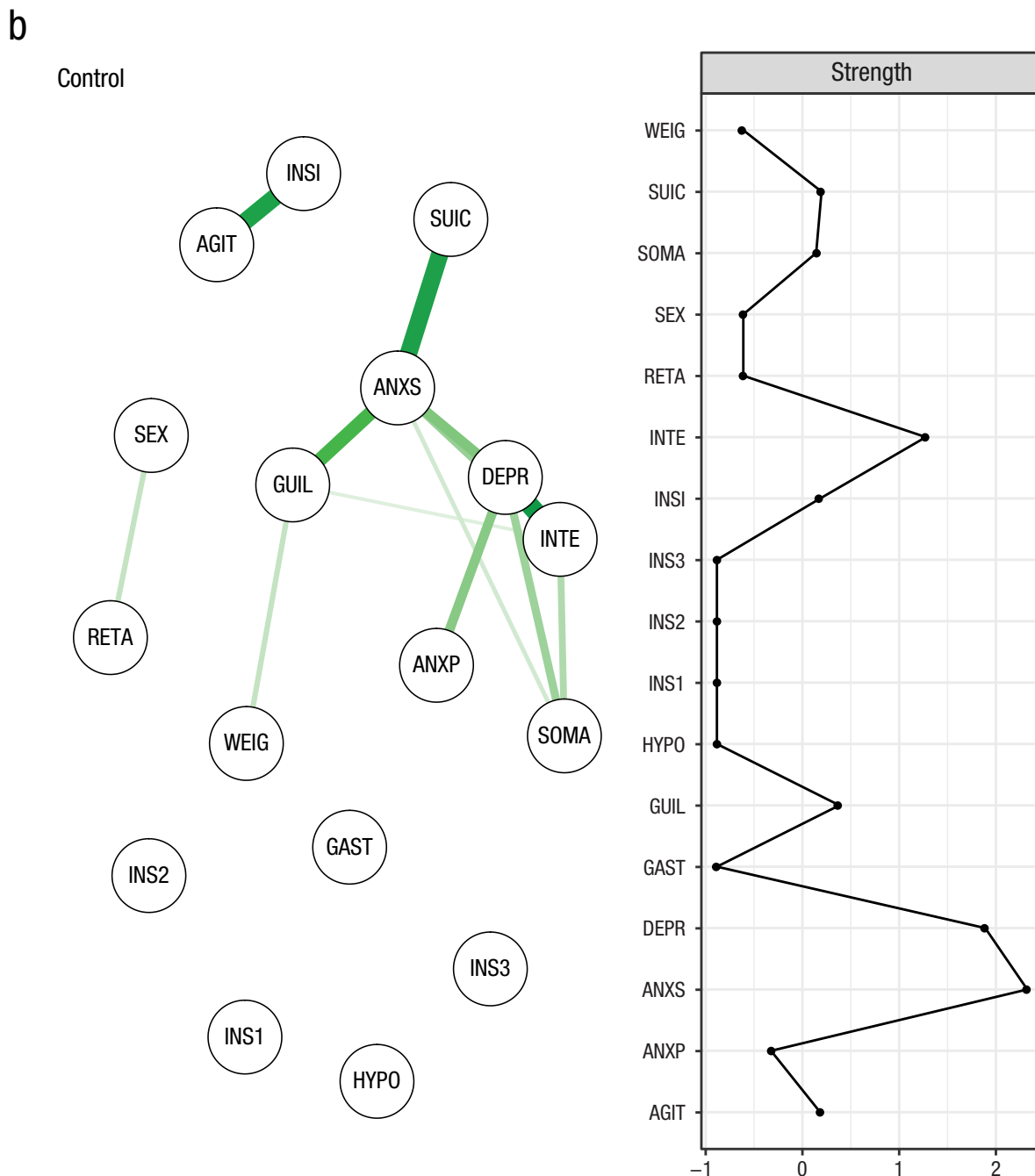


Fig. 2. Symptom-change networks and strength centrality for (a) the training group and (b) the control group. Nodes represent symptom severity changes on each Hamilton Rating Scale for Depression item from baseline to post ABM (post minus baseline). Associations are indicated by edge thickness between nodes (thicker edge = stronger association). ABM = attention bias modification; DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss.

Supplemental Material. The average correlation remained high after 50% of the cases were dropped, indicating stability in the centrality measure estimates (Epskamp et al., 2018).

Discussion

This is the first study examining a randomized clinical trial using network analysis and the first network

b

Control

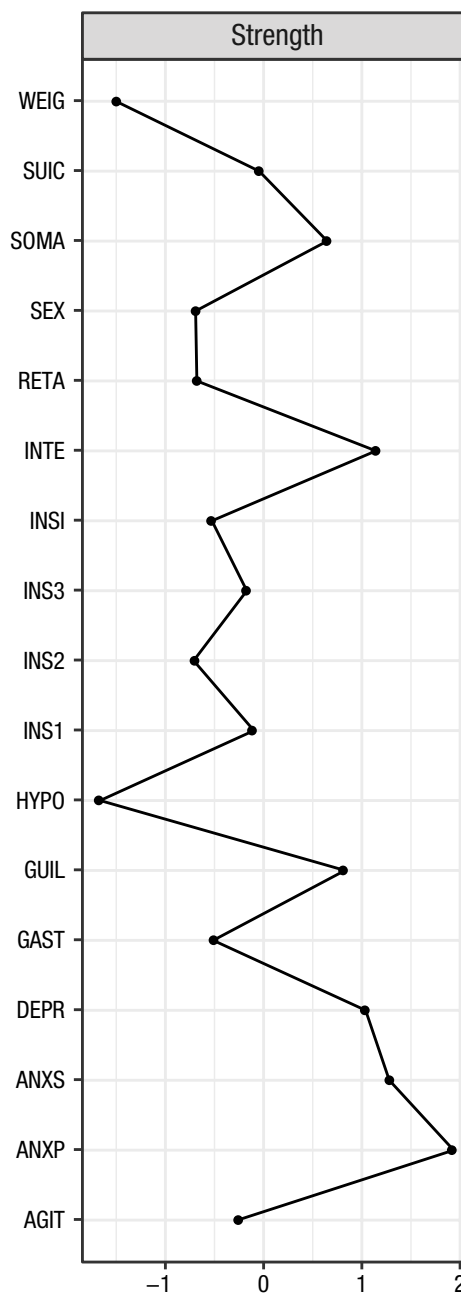
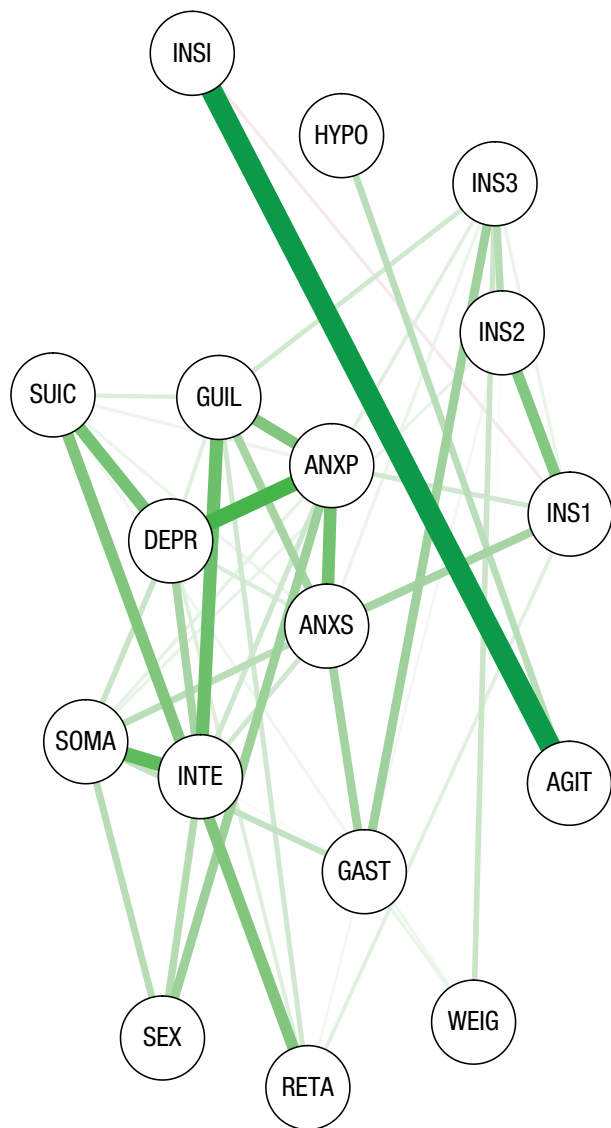


Fig. 3. Post ABM symptom networks and strength centrality for the training group (a) and the control group (b). Nodes represent Hamilton Rating Scale for Depression item scores post ABM (post minus baseline). Associations are indicated by edge thickness between nodes (thicker edge = stronger association). ABM = attention bias modification; DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss.

The aim of the present ABM procedure was to render patients more focused on positive social stimuli. It has been hypothesized that this mechanism may set in motion an implicit relearning of a range of emotional

associations in which ambiguous events or stimuli are perceived more positively and thus increase patients' motivation to engage in their social environment (Harmer, Duman, & Cowen, 2017). Thus, as participants'

attention is nudged toward positive aspects of everyday situations (e.g., a smiling face, an encouraging comment), this may increase the probability for prosocial interaction, which in turn may increase the frequency of positive feedback and ultimately reinforce approach behavior toward social interaction (for a review, see Fox, 2005). Although this interpretation remains speculative, the association between ABM and interest may reflect the endpoint of this mechanism.

On the other hand, post hoc analyses showed that changes in attentional bias (as measured by the dot-probe task) were not associated with interest. However, like most extant procedures for assessing attentional bias, the dot-probe task exhibits poor psychometric properties (for a review, see McNally, 2018). Recently developed experimental paradigms enabling optimal assessment of attentional bias might be more appropriate in future research agendas (e.g., Price et al., 2015; Sanchez-Lopez, Vanderhasselt, Allaert, Baeken, & De Raedt, 2018; Zvielli, Bernstein, & Koster, 2014).

An alternative interpretation is that interest is a moderator of the ABM effect. This hypothesis is in keeping with Shiroma, Thuras, Johns, and Lim (2014), who found that antidepressant response and remission was predicted by early changes in emotional processing of faces when considered along with perceived social support. Thus, helping patients to approach social situations during training may increase the effect of ABM.

Guilt has been found to be the most important symptom in explaining differences in overall network connectivity between depression persisters and remitters (van Borkulo et al., 2015). We found that ABM reduces connectivity between guilt and other depression symptoms. The guilt item in HRSD refers to “self-reproach and ideas of guilt or rumination over past errors” and is thematically similar to the brooding facet of rumination (Treyner, Gonzalez, & Nolen-Hoeksema, 2003). Rumination has been identified as a critical mechanism of depression (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). It is related to negative emotional processing (Gotlib & Joormann, 2010) and decreased cognitive control (Yang, Cao, Shields, Teng, & Liu, 2017). Specifically, brooding is characterized by attention being more firmly held on negative compared with positive information (Southworth, Grafton, MacLeod, & Watkins, 2017). Moreover, rumination has been found to mediate the effect of ABM on depression symptoms (Yang et al., 2015). Thus, the decoupling of guilt may represent a reduced impact of rumination in the context of other depression symptoms.

The present study provides an important basis to formulate new hypotheses on how ABM may alter depression symptoms. Our findings suggest that ABM reduces residual depression by increasing interest and motivation for social interaction, which in turn buffers

against anxiety symptoms and negative cognitions. Whether these changes are of a sufficient magnitude to prevent relapse of a depressive episodes needs to be examined in follow-up studies.

Compared with changes involving anxiety symptoms, ABM resulted in fewer changes related to depressed mood. This may be explained by the fact that attention was trained toward positive stimuli in the context of threatening or anxiety-related stimuli (i.e., angry and fearful faces). This observation is further corroborated by the complementary post hoc analyses of changes in attentional bias scores, showing that fostering attentional bias for positive stimuli in the presence of negative stimuli was associated with a reduction of anxiety symptoms. Training in a context of negative mood-related stimuli (e.g., sad faces) may result in more pronounced changes involving depressed mood. Because depressed mood is the most central symptom, it is possible that this training can increase the overall effect of ABM.

On the other hand, post hoc analyses showing that changes in anxiety were related to changes in attentional bias toward angry/fearful faces also point to anxiety as a potential mediator of ABM’s beneficial impacts. Thus, a reduction in the orientation toward threatening stimuli may have improved anxiety symptoms, which in turn may have led to improvements in other depression symptoms. Unfortunately, the exact sequence of change in the symptom cascade cannot be inferred from the present study, and future experiments are clearly needed to do so.

The network approach offers a promising conceptual framework to personalized treatment (Fried et al., 2017). The present study invites the hypothesis that ABM may be more effective in patients in whom interest, anxiety symptoms, and negative cognitions dominate the symptom network and less effective in patients in whom other symptoms are central (e.g., sleep problems and vegetative symptoms). Estimating individual symptom networks on the basis of intensive time-series data can provide a more fine-grained evaluation of ABM effects in individuals (see e.g., Fisher, Reeves, Lawyer, Medaglia, & Rubel, 2017). On the basis of these estimations, one can identify symptom profiles for which ABM might be the most effective treatment approach.

Including nonsymptom variables that are assumed to play a causal role in depression in the network models has the potential to bring more knowledge of how and for whom ABM can alter depression course (Jones, Heeren, & McNally, 2017). Relevant variables here are rumination and attentional control because these are regarded as core causal mechanisms of depression (De Raedt & Koster, 2010; Nolen-Hoeksema et al., 2008) and

are related to attentional bias change (Arditte & Joormann, 2014; Basanovic, Notebaert, Grafton, Hirsch, & Clarke, 2017) and symptom relief (Yang et al., 2015) after ABM.

The present study has several limitations. First, to the best of our knowledge, this is the first study considering an experimental condition within a network of continuous measures. Thus, the statistical adequacy of this approach has yet to be established. Emerging computational tools enabling the combination of categorical and continuous variables within a network, such as mixed graphical network modeling (Haslbeck & Waldorp, 2015), may therefore be more appropriate for future research in the field. Second, the edges were calculated with cross-sectional data, precluding strong inference regarding cause-effect relationships among the variables (Maurage, Heeren, & Pesenti, 2013). Cross-sectional edges represent both within- and between-subjects effects that cannot be disentangled, and the network trajectory over time may vary across individuals. Third, symptom-change networks were estimated on the basis of individual difference scores, making the assumption that there would be no symptom changes in the absence of intervention. An alternative model would be to assume that symptom scores at post ABM is a linear function of the symptom scores at baseline (i.e., residual change scores; for a discussion, see Gollwitzer, Christ, & Lemmer, 2014). However, to best capture the within- and between-person temporal dynamics of individual networks, one would need to apply graphical vector autoregressive modeling approaches on intensive time-series data from individual participants (e.g., Wichers, Groot, & Psychosystems, 2016; Wild et al., 2010). In this way, one can track change in symptoms and attentional bias over time to optimally model the continuous dynamics of the interplay between the different changes occurring during ABM. Fourth, although a sample size of 302 patients is usually not regarded as a small sample for a clinical study in depression, network models estimate a very large number of parameters, and cross-sample validations in larger samples are thus required to draw firm conclusions. On the other hand, results from the non-parametric bootstrap approach reinforce the generalizability of the present findings in similar samples. Fifth, as highlighted by Jonassen et al. (2018), uncertainty still abounds regarding the best way to interpret the relevance of the rather small improvement in HRSD score in the present sample (i.e., among patients who are not currently fulfilling diagnostic criteria for depression). We cannot exclude that the present effects on symptom networks would have been different with larger effects size. Finally, this study cannot determine whether the observed

network changes are specific to ABM or could be the result of any form of intervention. Other forms of treatments (i.e., psychological, pharmacological) may yield very different impacts on the symptoms network. Moreover, each patient's particular network structure may point to different treatments or a combination of treatments. Future research is thus clearly needed to clarify these issues.

In conclusion, by applying a computational network approach, we found that ABM improved depression by reducing the adverse impact of anxiety symptoms and negative cognitions on other depression symptoms. This change was associated with increased interest and involvement in everyday activities.

Action Editor

Stefan G. Hofmann served as action editor for this article.

Author Contributions

B. Kraft developed the study concept. B. Kraft and R. Jonassen collected the data. B. Kraft performed the data analysis and interpretation under the supervision of A. Heeren. B. Kraft and A. Heeren drafted the manuscript, and R. Jonassen, C. Harmer, T. Stiles, and N.I. Landrø provided critical revisions. All the authors approved the final manuscript for submission.

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Declaration of Conflicting Interests

C. Harmer has received consultancy fees from Johnson and Johnson Inc., P1vital, and Lundbeck. N.I. Landrø has received consultancy fees and travel expenses from Lundbeck. The remaining author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

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Supplemental Material

Additional supporting information can be found at <http://journals.sagepub.com/doi/suppl/10.1177/2167702618822480>

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