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PH.D. THESIS SUMMARY

COGNITIVE BIASES IN EMOTIONAL AND PSYCHOSOMATIC DISORDERS

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Notes: _____

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(2) All the Tables and Figures are numbered within the corresponding chapter or subchapter of the thesis.

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Key words: cognitive-behavioral psychotherapy, cognitive biases, cognitive bias modification (CBM) paradigm, attention bias modification (ABM), attention bias, depression, anxiety, gastrointestional condition

CHAPTER I

THEORETICAL BACKGROUND

Emotional disorders (i.e., clinical and subclinical dysfunctional feelings) and psychosomatic disorders have a high prevalence and a high comorbidity rate, as well as high associated costs (Alonso et al., 2004; Cash, Sullivan, & Barghout, 2005; Johnston, Westerfield, Momin, Phillippi, & Naidoo, 2009; Talley, 2008; Wittchen et al., 2011; Wu, 2012). Cognitive behavioral therapy (CBT) has been shown to be one of the best scientifically supported treatment option for both emotional disorders (see NICE guidelines at www.nice.org.uk) and psychosomatic disorders (Bothwell, 2003). However, the number of those who suffer from emotional and/or psychosomatic conditions *and* receive CBT treatment is rather small for a variety of reasons including treatment accessibility and high associated costs. Even among those who receive adequate CBT treatment, relapse rate is quite high, indicating the need for constant innovation and treatment optimization (David & Szentagotai, 2006). Therefore, we need on the one hand to increase access to the evidence-based psychological treatment, and on the other hand to innovate existing procedures in order to enhance treatment efficacy and/or decrease its associated costs.

The cognitive bias modification (CBM) paradigm has set recently the framework for such an approach. Cognitive biases are information processing strategies reflected in representing and/or appraising information (i.e., selective information processing), that are due to systematic errors in mental heuristics and can affect conscious and/or unconscious information processing.

Building on cognitive-experimental science, CBM states that we can modify the automatic cognitive biases through cognitive training and this has notable clinical effects (see, for example, Hallion & Ruscio, 2011). Cognitive biases are assumed to be important factors involved in the onset and maintenance of psychopathology in cognitive theories of psychopathology that underpin CBT. Depending on the cognitive processes with which cognitive biases interfere, they can be classified in three major categories: attentional biases, interpretation biases, and memory biases (Cisler & Koster, 2010; Everaert, Koster, & Derakshan, 2012; Eysenck & Keane, 2010). However, when clinicians speak about cognition, they usually refer to thought streams and mental images that patients are aware of and can report. Although self-report of cognitive factors are important for understanding the onset and maintenance of psychopathology, and we can infer from them certain cognitive process, they do not in themselves reveal too much about the processes that led up to that content appearing in consciousness (Mathews, 2006).

Within cognitive behavioral therapy, only explicit biases are addressed directly, despite the fact that implicit cognitive processing has been incorporated in the ABC model underlying CBT intervention (and thus the importance of implicit cognition is clearly acknowledged; David et al., 2009). Therefore, targeting the implicit biases has the potential to improve the current CBT treatment protocols.

The entire thesis is aimed to contribute to the development and testing of new therapeutic strategies, building on the knowledge from clinical field *and* experimental cognitive science. After carefully considering the theoretical and empirical evidence of cognitive biases in emotional and psychosomatic disorders, in this introductory chapter we point out the main problems identified in the field, namely:

1. Due to the fact that CBM and CBT have developed rather in parallel than in tandem, despite their common cognitive grounds, there is conceptual overlapping between them

and we did not have a comprehensive definition of cognitive biases which apply to both cognitive science research and clinical field. We proposed a work definition, stating that cognitive biases are descriptive, inferential, and/or evaluative automatic cognitive processes that operates at an implicit and/or explicit level to favor the processing of one type of information over the other.

2. Although CBM stirred a huge amount of interest among researchers and initial studies reported promising results, there is a lack of systematic investigation of its mechanisms of change. In addition, the parallel development of CBM and CBT resulted in no investigation of the impact that CBM could theoretically have on the cognitive factors targeted as mechanisms of change within classical CBT.
3. Regarding psychosomatic disorder, despite the theoretical and evidence of cognitive bias, the role played by the cognitive bias in relation to symptoms is unclear.
4. Cognitive biases are not routinely measured within CBT clinical practice. The available methods used to assess cognitive biases are experimental and have limited ecological validity. Moreover, some of them have been criticized for poor psychometric properties. Current CBM procedures are modifications of the methods used for experimental bias assessment and proved to be boring for participants.
5. In the absence of systematic research efforts, informed by sound theoretical models, the CBM paradigm risks to undermine the CBT very defining feature that is an evidence-based approach to mental health. We think this is likely to happen as long as, despite the lack of firm proves that it works and in the context of underinvestigating its mechanisms, CBM is still marketed by researchers involved in the field (see www.managingyouranxiety.com).

In the light of the theoretical and empirical data related to the role of cognitive biases in emotional and psychosomatic disorders, and considering the problems identified in the field, we finally highlighted the relevance of our research topic that is articulated from two complementary directions: (1) investigating CBM efficacy within a CBT framework, and (2) investigating CBM mechanisms of change.

In the next chapter we outlined the research objectives and the overall methodological approach entailed to reach these objectives, in an effort to create the general framework for the understanding of the studies included in this research project.

CHAPTER II

RESEARCH OBJECTIVES AND OVERALL METHODOLOGY

The general goal of this research project was to investigate the role that cognitive biases play in emotional and psychosomatic disorders within a CBT framework. In the field of emotional disorders we focused on investigating a new experimental treatment approach (i.e., CBM) in terms of its efficacy and mechanisms of change. In the field of psychosomatic disorders, we aimed to extend the available empirical evidence regarding the existence of cognitive bias by means of investigating the role that attentional bias plays in relation with gastrointestinal symptoms maintenance (as the prevalence of gastrointestinal disorders is notably high and gastrointestinal symptoms are among the most frequent subjective health complaints).

The **first** major objective of our research was to quantitatively review the data available in the literature regarding the clinical efficiency of attentional bias modification, one of the most popular CBM interventions. This objective aimed to contribute to the empirical evidence regarding the overall ABM clinical efficacy and was pursued by means of a quantitative meta-analysis (Study 1).

The **second** major objective of our research was to replicate and extend the previously reported results regarding ABM clinical utility, in terms of its efficiency and mechanisms of change. This objective aimed at conceptual and theoretical innovations, having at the same time practical implications regarding the utility of ABM as a clinical strategy. To accomplish this objective, we run one clinical study and two experimental studies. We investigated the role of exposure as a potential factor contributing to the efficiency of ABM in reducing anxiety symptoms in a controlled randomized study (Study 3). In addition, we tested the role that expectancies (Study 3, Study 4) and attentional control, as a general ability (Study 4), or expressed in relation to the processing of affective contents (Study 5), could play in supporting the ABM efficiency.

The **third** major objective of our research was to integrate the CBM interventions with the classical cognitive-behavioral therapeutic interventions. In doing that, we specifically investigated if CBM interventions have any impact on the dysfunctional beliefs known to be involved in the onset and maintenance of psychopathology (Study 2, Study, Study 4, and Study 5). Similar to the second objective, this objective aimed at theoretical and conceptual innovations.

Finally, our **fourth** major objective was to investigate the role that attentional bias plays in relation to the maintenance of gastrointestinal symptoms. This objective aimed at theoretical innovation (Study 6 – a cross-sectional, correlational study).

The **structure of the Ph.D. project** is closely molded on these objectives. Most of the conducted studies are fundamental research studies, aimed to advance the current understanding of the cognitive biases functions in emotional and psychosomatic conditions. However, two of the studies we conducted (Study 2 and study 3) have important implications in terms of clinical practice, as they tested the clinical utility of two CBM training procedures for reducing anxious symptoms (training procedure tested: ABM; Study 3) and depressive symptoms (training procedure tested: concreteness training, CNT; Study 2). In addition, our study that investigated attentional bias in gastrointestinal patients was aimed to advance our current understanding about the relevance of attentional bias for the clinical management of gastrointestinal patients.

CHAPTER III

ORIGINAL RESEARCH

PART 1. EMPIRICAL ANALYSIS OF THE DATA AVAILABLE REGARDING ATTENTION BIAS MODIFICATION

STUDY 1. Clinical efficacy of attention bias modification (ABM) procedures: a comprehensive meta-analysis[1]

Introduction

In recent years, an extensive body of research on attentional bias modification (ABM) procedures has accumulated. Several reviews have examined ABM effects on symptoms and AB change. However, two main issues of these syntheses of the ABM literature should be noted: namely, (1) their reports are inconsistent, both in terms of change in AB and symptoms, and (2) none of them included any of the recent negative findings reported with the ABM, although all of them reported publication bias.

This study was aimed to comprehensively examine the clinical efficacy of ABM, both in terms of outcome (i.e., reducing and/or preventing symptoms of subjective distress, dysfunctional behaviors, and biological markers of psychopathology) and in terms of the presumed mechanism of change (i.e., AB). To this end, we considered studies that trained attention *away* from disorder relevant stimuli (congruent with the theory-specified direction of clinical improvement) and compared this intervention with an adequate control group (i.e., no training of attention). This allowed us to provide a global estimate of ABM effect size on symptoms and AB. Second, we aimed to investigate the degree to which ABM yields therapeutic benefits for different symptom categories. Third, we aimed to test possible moderators of the ABM effect. Finally, we were interested to investigate the relationship between pre-existent AB and the reduction in AB and symptoms.

Based on the potential moderators considered in the previous reviews and on the theoretically-derived assumptions about the factors affecting ABM efficacy, we considered the following **potential moderators**: (1) Type of psychopathology; (2) Clinical status of the sample; (3) Training methodology; (4) Type of outcomes; and (5) Participants' age.

We investigated the relationship between AB and symptom change in two ways. First, we examined pre-existent AB and the reduction in AB and symptoms given that the reduction in AB is the presumed mechanism of change of ABM interventions. Yet, none of the previous meta-analyses considered the role of the pre-existent AB in relation with ABM efficacy. Second we examine the relation between AB change and symptom changes. Previous meta-analyses failed to find a statistically significant relationship between reduction in AB and reduction in symptoms following ABM (Hakamata et al., 2010; Hallion & Ruscio, 2011). This may be due to the possibility that ABM lowers symptoms via AB reduction only in persons with a pre-existing AB, where this could be an important selection criteria to enroll in training (see Eldar et al., 2012).

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[1] This study is under review at *Journal of Clinical Psychology:* Mogoaş e, C., David, D., & Koster, E.W.H. (2013). Clinical Efficacy of Attentional Bias Modification Procedures: An Updated Meta-analysis.
The authors contributed to the manuscript as follows: Mogoaş e, C. – study design, study implementation (including data analysis), writing the manuscript; David, D. – study design, structuring the manuscript, consultation for writing the manuscript; Koster, E.W.H. – structuring the manuscript, consultation for writing the manuscript

This meta-analysis has both theoretical and practical key implications. From a theoretical point of view, as compared to previous meta-analyses in the field, it brings the following new innovations: it includes newer studies that failed to replicate the original positive findings with ABM; it assesses the pre-existing bias levels; and, it looks for differential ABM effect within anxiety studies. All of these have the potential of further inform the research work. From a practical point of view this meta-analyses is timely because ABM seems to be promoted sometimes based on "good marketing" and "the new halloo effect", all stimulated by a combination of "simple intervention strong effects"; therefore, to rigorously guide the clinical practice of psychology we need a comprehensive meta-analysis, organized innovatively from a theoretical point of view and which try to delineate clear practical implications and future directions of development.

Method

Literature search

Potential relevant studies were identified through a systematic search of the ISI Web of Science, Scopus, and Medline databases through July 2013, using the following search terms: "attentional bias modification", and "attention bias" combined with "attentional (re)training" and "experimental manipulation". We also systematically searched the references from the empirical papers, meta-analyses, and reviews on the topic.

Inclusion criteria

The following criteria were applied for inclusion in the meta-analysis: (a) the study was designed to manipulate AB to reduce symptoms and/or to emotional vulnerability (in the latter case, to be included a study should have included at least one measure of distress); (b) the study assessed clinically-relevant symptoms; (c) participants were randomized to training conditions; (d) a control condition (defined as sham training) existed; (e) the study was written in English and published/accepted for publication in a peer-reviewed journal; (f) sufficient data to compute effect size were available.

Selection of the comparison groups

Some studies included a third group (trained to attend to threat stimuli; e.g., Heeren, Reese, McNally, & Philippot, 2012; Klumpp & Amir, 2010), but we focus exclusively on comparing the ABM group trained towards the neutral/positive stimuli to the control group. We chose to do this because we were specifically interested in the clinical implications of AB reduction rather than in the consequences of experimental induction of AB.

Coding procedures

For every eligible study we retained the following variables: study identification data (author, year of publication), symptoms category, clinical status of the sample, sample size, participants' mean age, type of ABM procedure, type of stimuli, position of stimuli during training, number of training trials, number of ABM sessions, temporal separation of ABM sessions, ABM treatment duration, follow-up interval, and outcome measures.

The dependent variables were classified as follows:

1. *Primary outcomes* (or measures of the core symptoms related to the investigated condition) versus *secondary outcomes* (or measures of general distress/nonspecific symptoms).
2. *Self-report, clinician-rated, and bio-behavioral measures*. The same outcomes classified previously as being primary or secondary were classified here based on how they were

measured (for example, for bio-behavioral measures we considered the cortisol level or indicators of heart rate variability).

Statistical analyses

We calculated Hedges's *g* effect sizes for every outcome measure for which sufficient data were reported. All the effect sizes were coded such that a positive value of Hedges's g indicated greater improvement in the experimental group compared with the control group. For all sets of the calculated effect sizes we used the random effects model (based on the assumption that studies come from populations where the effect sizes varies). To address publication bias, we calculated a fail-safe *N* (Rosenthal, 1991). In addition, we generated and visually examined funnel plots, which plot standard error for each study (determined by sample size) against the effect size computed for that study. In case of an asymmetrical funnel plot, Duval and Tweedie's trim-and-fill procedure (Duval & Tweedie, 2000) estimates the likely number of the missing studies that would correct for the publication bias. All analyses were run using Comprehensive Meta-Analysis, Version 2.2.046 (Borenstein, Hedges, Higgins, & Rothstein, 2005).

Results

ABM effect on AB

Regarding the ***overall change in AB***, as measured *post-intervention*, results showed a small and statistically significant effect size, g = 0.312, p = 0.003, 95% CI = [0.216; 0.409], $Q(34)$ = 33.966, p = 0.469, I^2 = 0.000. For *follow-up measurements*, the average effect size was non-significant, g = 0.553, p = 0.137, 95% CI = [-0.177; 1.282], $Q(4)$ = 26.349, p = 0.000, I^2 = 84.819. The average effect size computed for ***anxiety studies*** was 0.329, p = 0.000, 95% CI = [0.183; 0.474], $Q(17)$ = 19.304, p = 0.311, I^2 = 11.937. For ***depression*** it was 0.217, p = 0.099, 95% CI = [-0.040; 0.475], $Q(5)$ = 2.188, p = 0.823, I^2 = 0.000, for ***pain*** it was 0.202, p = 0.379, 95% CI = [-0.248; 0.651], $Q(1)$ = 0.372, p = 0.542, I^2 = 0.000, and for ***substance abuse*** it was 0.340, p = 0.070, 95% CI = [-0.027; 0.707], $Q(4)$ = 8.569, p = 0.70, I^2 = 53.806); for ***healthy participants*** the effect size was 0.378, p = 0.003, 95% CI = [0.125; 0.631], $Q(3)$ = 2.370, p = 0.499, I^2 = 0.000.

Only training setting was found to significantly moderate the ABM effect for change in AB in the overall data set, $Q(1)$ = 4.770, p = 0.029, with studies conducted in laboratory yielding significant lager effects (g = 0.371, p = 0.000, 95% CI = [0.261; 0.480], $Q(25)$ = 24.720, p = 0.478, I^2 = 0.000) than studies conducted out of the laboratory (g = 0.116, p = 0.259, 95% CI = [-0.085; 0.317], $Q(8)$ = 4.476, p = 0.812, I^2 = 0.000). The same was true for *anxiety studies*, $Q(1)$ = 5.202, p = 0.023, studies conducted in laboratory being found to yield significant larger effect size (g = 0.407, p = 0.000, 95% CI = [0.254; 0.561], $Q(14)$ = 13.281, p = 0.505, I^2 = 0.000) compared with studies conducted out of the laboratory (g = 0.032, p = 0.824, 95% CI = [-0.251; 0.315], $Q(2)$ = 0.821, p = 0.663, I^2 = 0.000). In addition, in anxiety study subsample participants' age significantly moderated the ABM effect on bias, with younger participants benefiting more (slope = - 0.021, p = 0.01).

ABM effect on symptoms

Overall change in symptoms. The results computed on *data collected post-intervention* showed a small, yet statistically significant effect size, g = 0.160, p = 0.003, 95% CI = [0.055; 0.265], $Q(39)$ = 70.079, p = 0.002, I^2 = 44.349. For studies reporting outcome measures *following a stressor*, the average effect size 0.375, p = 0.000, 95% CI = [0.246; 0.504], $Q(12)$ = 8.794, p = 0.720, I^2 = 0.000. For studies reporting *follow-up measures* the average effect size wasnon-significant, g = 0.227, p = 0.087, 95% CI = [-0.033; 0.488], $Q(8)$ = 19.190, p = 0.014, I^2 = 58.313.

Change in symptoms across disorders. We computed separate effect sizes for different disorder categories and different time points. ABM yielded reliable effects at post-intervention and following a stressor only for *healthy (g* = 0.211, 95% CI = [0.046; 0.375], *Q*(3) = 1.420, *p* = 0.701) and *anxious participants* (*g* = 0.260, 95% CI = [0.132; 0.388], *Q*(21) = 95.678, *p* = 0.000). No statistically significant effect sizes were obtained for follow-up measures, except for one study conducted in healthy participants that had a 2-week follow-up period (*g* = 0.273, 95% CI = [0.033; 0.513]).

Moderators of the ABM effect on symptom reduction. We ran the moderation analyses based on data collected *post intervention*, and *post-stressor*, respectively, considering only *anxiety studies*. When symptoms were measured post intervention, ABM yielded a small but significant effect on symptoms in social anxiety, and a medium effect in generalized anxiety. The effect size for other anxiety disorder (e.g., phobias, post-traumatic stress disorder) was not significant. Similarly, studies using the modified dot-probe task as well as those conducted in laboratory yielded significant larger effects compared with studies using spatial cueing task, or conducted via Internet, at home. The effect sizes were significant only for primary outcomes, regardless of the time of measurement (i.e., post intervention or post stressor). No other significant categorical moderators were identified. Participants' age was found to significantly moderate the ABM effect when symptoms were measured post-intervention, with younger individuals benefiting most from ABM (slope = -0.034, *p* = 0.000).

Relationship between the pre-existent AB, change in AB, and change in symptoms

In the overall dataset, the pre-existent AB was significantly related to the change in AB, *r*(34) = .519, *p* = 0.002, and the change in AB correlated significantly with the change in symptoms, *r*(34) = .342, *p* = 0.048. However, there was no direct relationship between the pre-existent AB and change in symptoms, *r*(32) = -.005, *p* = 0.977. We tested in a mediation model the assumption that the pre-existent AB (predictor) influenced symptom reduction (outcome) via change in AB (mediator). For mediation analysis we used bootstrapping tests with 1000 re-samples and corrected confidence interval (Preacher & Hayes, 2008). The results indicated no significant direct or indirect effects of the pre-existent AB on change in symptoms (for the direct effect, 95% CI = [-0.384; 0.181]; for the indirect effect, 95% CI = [-0.039; 0.234]). The same results pattern was observed within the anxiety sample of studies.

Publication bias

Publication bias for change in symptoms. There was evidence of publication bias in the overall data set for change in symptoms as measured post-intervention: fail-safe *N* was 133, smaller than $5K+10$[2]. In addition, Duval and Tweedie's trim-and-fill procedure (Duval & Tweedie, 2000) estimated 10 missing studies with effect sizes smaller than the mean effect size, which would have reduced the mean effect size to non-significance, *g* = 0.031, 95% CI = [-0.080, 0.147]. However, there was no evidence of publication bias in the overall data set for change in symptoms as measured post-stressor: fail-safe *N* was 127, larger than $5K+10$, and trim-and-fill procedure estimated no missing studies. Similarly, for anxiety studies, there was some evidence of publication bias for change in symptoms when post intervention data were considered: fail-safe *N* was 119, and trim-and-fill procedure estimated 3 missing studies, which would have reduced the effect size to 0.205, 95% CI = [0.072, 0.339]. However, when post-stressor data were considered, there was no evidence of publication bias (fail-safe *N* = 63; no missing studies were estimated).

[2] According to Rosenthal (1991), the computed fail-safe *N* should be larger than $5K+10$, where *K* is the number of studies included in meta-analysis

Publication bias for change in AB. No evidence of publication bias was found for change in AB as measured post intervention in the overall data set (fail-safe *N* was 740, no missing studies were estimated). However, in the anxiety studies subset there was some evidence of publication bias: fail-safe *N* was 101, and trim-and-fill procedure 4 missing studies which would have reduced the effect size to 0.229, 95% CI = [0.066, 0.393].

Discussion

This meta-analysis aimed to assess the clinical utility of ABM procedures. For this purpose we performed a quantitative review of studies that included a procedure aimed to reduce AB and examined effects on symptoms or features of psychopathology. The obtained results indicated that: (1) ABM successfully reduces AB and symptoms/emotional vulnerability in anxious individuals and healthy participants; and (2) although the pre-existent AB was significantly related to change in AB, and change in AB was positively related with change in symptoms both in the overall data set and in the anxiety subsample of studies, no direct or indirect effect of the pre-existent AB on the change in symptoms was observed. Therefore, ABM seems to have a small clinical impact (about 58%-62% of participants in the control group would have more symptoms compared with the average participant in the experimental group) (McGough & Faraone, 2009), but how it works is not fully clear.

These results have two main research and clinical implications. First, we need more powerful ABM procedures, to strengthen the ABM effect: we should strive to improve the existing ABM procedures, and/or to develop and test new theory-driven training procedures.

The second implication of these results concerns the need to clarify the ABM mechanism of change. As ABM therapeutic benefits are rather limited, we believe it is clearly premature to speak of ABM treatment (see Bar-Haim, 2010; Hakamata et al., 2010). The results of the present work urges for more adequately powered, randomized controlled clinical trials, conducted by different research groups, and aimed to rigorously assess ABM impact on both AB and symptoms.

PART 2. COGNITIVE BIAS MODIFICATION IN EMOTIONAL DISORDERS

STUDY 2. Can concreteness training alone reduce depressive symptoms and overgeneral autobiographical memory bias? A randomized pilot study using an Internet-delivered protocol[3]

Introduction

The overgeneralization bias is regarded as a crucial cognitive mechanism in depression (Carver & Ganellen, 1983). In relation to depression, overgeneralization is conceptualized as a cognitive distortion (i.e., the tendency to make faulty inferences or to draw inaccurate conclusions; Beck, 1976), as an irrational belief (i.e., global rating/evaluation of self-worth; David, 2006; Ellis, 1962, 1994) or as a functional aspect of autobiographical memory that is common among individuals who are depressed or at risk (Sumner, Griffith, & Mineka, 2010; Williams et al., 2007).

Drawing upon research in social and cognitive psychology (i.e., construal levels theory; Trope & Liberman, 2003), Watkins and colleagues suggested that overgeneralization involves abstract processing of self-relevant information (Watkins, Baeyens, & Read, 2009; Watkins & Moberly, 2009; Watkins, Moberly, & Moulds, 2008).

Watkins and colleagues proposed "concreteness training" (CNT) as an intervention to modify the abstract information processing mode observed in depressed individuals (Watkins et al., 2009; Watkins & Moberly, 2009). CNT consists of a guided relaxation procedure, followed by repeated concrete processing exercises that prompt participants to use mental imagery in order to generate detailed step-by-step descriptions of different scenarios.

Our study aimed to replicate previous findings on the effectiveness of CNT in ameliorating depressive symptoms and the depressogenic cognitive mechanisms (rumination and overgeneralization), while also eliminating the possible confounding factors that may have accounted for CNT effects in prior studies. Similar to prior studies conducted by Watkins and colleagues (Watkins et al., 2009; Watkins & Moberly, 2009), the training protocol used in our study included exercises that trained participants to think in more concrete and specific terms about both positive and negative events. However, our training protocol differed from the one developed by Watkins et al. (2009) in three ways. First, we included hypothetical scenarios and not autobiographical events because our intent was to determine if the intervention effects could

[3]This study has been published: Mogoaş e, C., Brăilean, A., & David, D. (2013). Can concreteness training alone reduce depressive symptoms? A randomized pilot study using an Internet-delivered protocol. *Cognitive Therapy and Research*, 37(4). Doi: 10.1007/s10608-012-9514-z

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be generalized to untrained contents. Second, we used CNT as a standalone intervention, eliminating specific factors known to contribute to expected effects (i.e., relaxation, problem solving) and nonspecific factors (i.e., therapeutic relationship, success expectancies). In order to eliminate nonspecific factors, we delivered training exclusively online and chose not to present it as an intervention designed to reduce depressive symptoms. Third, although our training sessions had the same duration as those of Watkins et al. (2009), our intervention was designed to include fewer scenarios per session, as participants were asked to write down detailed descriptions of imagined events, with the aim of ensuring compliance with instructions.

Thus, our goal was to test the assumption that an increase in concrete processing is the crucial mechanism in ameliorating depressive symptoms. We expected concrete processing training to reduce depressive symptoms, rumination, and global evaluation in the CNT group compared to the control group, while increasing the concreteness of thinking as well as autobiographical memory specificity.

Method

Participants

Ninety-six undergraduate students were initially recruited using email advertisements that invited participation in exchange for course credits. We included only participants who showed stable dysphoria, operationalized as a score of at least 12 on the Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996), at two consecutive assessments (i.e., initial screening and pre-training assessment). The interval between the two consecutive assessments varied from 8 days to 15 days (M = 11.15, SD = 3.17). The 42 participants were randomly allocated to one of two experimental conditions (CNT or waiting list control). There were no differences between groups on any baseline measures.

The intervention

Concrete processing training involved repeated exercises designed to facilitate concrete thinking. We generated hypothetical scenarios based on the examples provided by Watkins et al. (2008) that we considered relevant to our participant sample. Similar to the training protocol of Watkins et al. (2009), our study protocol included seven scheduled daily sessions designed to last about 15 minutes. We used a total of five positive and five negative written scenarios. For example, one positive scenario was "It is your birthday. Your family organized a great surprise party for you at home" and one negative scenario was "You are on a trip in a foreign country, when you suddenly realize that your wallet has been stolen". Every scenario was presented to participants on a standard form. Instructions were similar to those used by Watkins and Moberly (2009): ''Focus on how the event happened and imagine in your mind, as vividly and concretely as possible, a 'movie' of how the event unfolded. As you imagine the event, see it through your own eyes, from your own viewpoint, as if you were looking out on the scene. Imagine the event in the present tense, as if you were there right now." Participants' answers were guided by the following questions, elaborated from a first-person perspective: (1) Space and time details: Where am I? What moment of the day is it?; (2) Sensory-focused details: What do I see? What do I hear? What do I feel? What does it smell like? What does it taste like?; (3) Comparison time details: What is different now from other times?; and (4) Processing details: How does the movie unfold? These questions provided on the standard form along with the scenario were meant to help them vividly visualize the hypothetical event. After they spent at least two minutes

imagining the event, they were asked to fill in the form, providing written details of the representation they had experienced.

Measures

Concreteness of thinking. Concrete thinking was measured using a methodology similar to a "Problem Elaboration Questionnaire" (PEQ; Stöber & Borkovec, 2002).

Autobiograpical memory specificity. We used a version of Autobiographical Memory Test (AMT; Williams & Broadbent, 1986) as an additional measure of concrete thinking.

Depressive symptomatology. We used BDI-II (Beck, Steer, & Brown, 1996), a widely used 21-item self-report scale that indexes the severity of depressive symptoms over a two-week measuring period[4].

Rumination. We used the Ruminative Response Scale (RRS; Nolen-Hoeksema & Morrow, 1991), a 22-item instrument that assesses the tendency to ruminate in response to depressive mood.

Global evaluation/self-downing. We used the self-downing subscale of the Attitude and Belief Scale (ABS-II; DiGiuseppe, Leaf, Exener, & Robin, 1988). ABS-II is a 72-item instrument that measures irrational cognitions (i.e., global evaluation/self-downing, as well as demandingness, low frustration tolerance, and awfulizing) assumed to be involved in the onset and maintenance of emotional disorders.

Procedure

The study was conducted entirely online, with no direct contact between participants and researchers. Participants were given a study rationale similar to that used by Watkins et al. (2008), in which researchers investigated the interplay between imagination, emotion and cognition, and training exercises were designed to increase imaginative skills.

Results

Effects of training on concreteness of thinking and autobiographical memory specificity

A 2 (Time: pre- versus post-testing) x 2 (Group: training versus control) repeated measures analysis of variance (ANOVA) on PEQ scores showed a significant main effect of time, $F(1, 39) = 26.40$, $p < .01$, $\eta^2 = .40$, and no significant main effects of group, $F(1, 39) = 1.16$, $p = .28$, $\eta^2 = .02$. The Time x Group interaction, however, was significant, $F(1, 39) = 23.75$, $p = .000$, $\eta^2 = .37$. Paired samples *t*-tests revealed that participants in the CNT group offered significantly more concrete descriptions of problems from pre- to post-intervention, $t(19) = 7.62$, $p < .01$, Cohen's $d = 0.46$, while no significant differences were evident in the control group, $t(20) = .17$, $p = .86$, Cohen's $d = 0.01$. The two groups significantly differed at post-test with regard to the level of concreteness of their descriptions, $t(39) = 1.97$, $p < .05$, Cohen's $d = 0.61$, although no differences were evident at pre-test, $t(39) = .31$, $p = .75$, Cohen's $d = 0.02$.

[4] For the purposes of this study, participants were asked to rate their depressive symptoms on BDI-II over the most recent week.

Effect of training on depressive symptoms

We found no significant main effects of time, $F(1, 39) = 1.94$, $p = .17$, $\eta^2 = 0.04$, or group, $F(1, 39) = 0.83$, $p = .36$, $\eta^2 = 0.02$, and no significant interaction effects of Time x Group, $F(1, 39) = 0.72$, $p = .40$, $\eta^2 = 0.01$, on depressive symptoms.

Effect of training on rumination

We found no significant main effect of time, $F(1, 38) = 0.86$, $p = .35$, $\eta^2 = 0.02$, or group, $F(1, 38) = 2.43$, $p = .12$, $\eta^2 = 0.06$, and no significant interaction effect of Time x Group $F(1, 38) = 0.73$, $p = .39$, $\eta^2 = 0.01$, on rumination.

Effect of training on the global evaluation

No significant main effect of time, $F(1, 37) = 0.02$, $p = .88$, $\eta^2 = 0.00$, or group, $F(1, 37) = 0.32$, $p = .36$, $\eta^2 = 0.00$, were found. However, we found a significant interaction effect of Time x Group, $F(1, 37) = 9.52$ $p = .004$, $\eta^2 = 0.20$. GE showed a marginally significant decrease in the CNT group, $t(18) = 1.92$, $p = .07$, Cohen's $d = 0.46$, whereas the control group showed a significant increase in GE, $t(19) = 2.48$, $p < .05$, Cohen's $d = 0.61$. While there were no differences between the means of the two groups at pre-test, $t(38) = 1.34$, $p = .18$, the difference between the two groups became significant at post-test $t(38) = 2.01$, $p < .05$, Cohen's $d = 0.64$.

Discussion

This study compared the efficiency of a concreteness training intervention against a waitlist control in ameliorating symptoms of depression and in modifying related cognitive mechanisms: concreteness of thinking, autobiographical memory specificity, rumination, and global evaluation of self-worth.

As predicted, results showed an increase in concreteness of thinking in the CNT group. However, contrary to our hypothesis, we did not find evidence for the efficacy of CNT in increasing autobiographical memory specificity, suggesting that training effects did not generalize to untrained cognitive contents. Previous studies did not use the AMT to assess CNT effects on increasing concreteness of thinking (although overgeneral autobiographical memory was previously shown to be a marker for depression). As our training protocol did not include autobiographical memories, we cannot rule out the possibility that CNT effects might be task-dependent (i.e., the effect is visible only on the PEQ). However, given the methodological similarity between PEQ and AMT, we believe that a task-dependent effect is unlikely.

Contrary to our hypothesis, CNT was not effective in reducing depressive symptoms or rumination. Global evaluation demonstrated a marginally significant decrease from pre- to post-intervention in the experimental group, while it significantly increased in the control group.

Despite its inherent limitations, this online study provides a robust test for the clinical utility of CNT. Consistent with prior studies, our study demonstrates that CNT is effective in increasing the concreteness of thinking. More adequately powered studies are needed to demonstrate that increases in concrete processing mediate the amelioration of depressive symptoms. Our results signify the importance of further investigating CNT with regard to its efficacy and mechanisms of change before delivering it in a clinical context.

STUDY 3. Attention bias modification in social phobia: effects on symptoms and self-report cognitive factors

Introduction

Despite that CBT has been shown to be effective in decreasing symptoms of social anxiety disorder, many of the socially anxious individuals fail to receive treatment (Fehm et al., 2005). Even among those who receive treatment, a significant proportion remains symptomatic, indicating the need of refining and innovate the existing treatment in order to optimize it (McEvoy & Perini, 2009). Because most CBT-based intervention protocols for social anxiety disorder target the modifications of a variety of *conscious* cognitions and behaviors contributing to the symptomatology, one possibility of improving the available treatment protocols may lay in addressing automatic information processing occurring *outside of the conscious awareness* and being amenable to change by means of cognitive bias modification. If attentional bias has indeed a critical role in the onset and maintenance of the symptoms (as theoretically suggested), targeting it could have beneficial effects and could be more advantageous in terms of the associated costs of the intervention.

Although several studies indicated the efficacy of attention bias modification (ABM) in reducing social anxiety symptoms (e.g., Amir, Beard, Taylor, et al., 2009; Heeren et al., 2011; Klumpp & Amir, 2010; Li, Tan, Qian, & Liu, 2008; Schmidt et al., 2009), the mechanism behind ABM is far from being understood. It was suggested that ABM works through facilitating disengagement from negative stimuli (see Heeren et al., 2011). However, there is no clear evidence that facilitated disengagement explains the reduction in symptoms. Alternative explanations could count for ABM efficacy as well. For example, the simple exposure to the negative stimuli relevant for social anxious individuals could have a benefic effect.

On the other hand, it was suggested that ABM works through improving executive functioning (see, for example, Klumpp & Amir, 2010). Arguably, this possibility is rather complementary than concurrent to the possible effect due to the stimuli used for training.

In this context, we aimed to replicate the previous reported results regarding ABM efficacy in socially anxious individuals and investigate the role that stimuli used during training play in relation with ABM efficacy.

The second aim of the present study was to investigate the effect that ABM could have on negative cognitions known to be involved in the onset and maintenance of social anxiety (i.e., irrational beliefs; negative automatic thoughts, including self-statements in public speaking situations, and participants' expectancies related to the intervention).

Method

Design

We used a unifactorial experimental design with repeated measures. The independent variable was the intervention. Participants were randomly distributed in three groups:

(a) ABM with stimuli relevant for social anxiety (i.e., imagistic stimuli portraying neutral and negative human faces) (Group 1)

(b) Sham ABM (i.e., control condition using the same human faces stimuli used in the first group) (Group 2)
(c) ABM with stimuli irrelevant for social anxiety (i.e., imagistic stimuli portraying neutral scenes or physical injury) (Group 3)

The dependent variables were outcome and mechanism of change measures. The *primary interest outcome* was the social anxiety. The *secondary interest outcome* was the general distress, operationalized as (1) trait anxiety and (2) depressive symptoms measures. In terms of *mechanisms of change*, we considered: irrational cognitions; fear of negative evaluation; participants' expectancies regarding the intervention efficacy; negative automatic thoughts; positive and negative self-statements in public speaking situations

Participants

A total of 230 potential participants were recruited using email advertisements that invited participation in a study "investigating a promising experimental treatment for social phobia". We selected only participants with significant social anxiety symptoms, operationalized as scores equal or above 30 at Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987), both in the screening phase and pre-intervention (baseline measurement). The interval between screening and study enrollment varied between 8 and 17 days (M = 9.67, SD = 4.1). The exclusion criteria were: (a) current treatment for social anxiety symptoms, (b) substance abuse, (c) psychotic symptoms, and (d) suicidal ideation or attempts.

Measures

Social anxiety. We used the self-report version of Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987), to evaluate fear and avoidance associated to social situations. Because there is no instrument measuring social anxiety adapted for Romanian population, we used an additional social anxiety measure, namely Social Phobia Inventory (SPIN) (Connor et al., 2000).

Trait anxiety was measured using the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Luchene, Vagg, & Jacobs, 1983). We used the STAI-X2 version, adapted to the Romanian population by Pitariu and his collaborators in 1987 (Pitariu, Miclea, & Munteanu, 1987).

Depressive symptomatology was measured with the Beck Depression Inventory (BDI-II; Beck et al., 1996), the same instrument we used to measure depressive symptoms in the previous study.

Participants' expectancies regarding the intervention efficacy and the future anxiety in social situations were measured using visual analogue scale (VAS; Holstein & Luria, 1973; Williams, Morlock, & Feltner, 2010).

Irrational cognitions. We used the General Attitudes and Beliefs – Short Form (GABS-SV; Lindner, Kirkby, Wertheim & Birch, 1999).

Fear of negative evaluation. We used the Brief Fear of Negative Evaluation – Revised (BFNER; Carleton, McCreary, Norton, & Asmundson, 2006). The BFNER-R is a 12-item instrument used for measuring fear of negative evaluation (e.g., „I am afraid that others will not approve of me").

General negative automatic thoughts. We used the Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1980), a 15 items instrument that measures the frequency of negative self-statements associated with depression.

Positive and negative self-statements in social situations. We used the Self-Statements in Public Speaking Situations scale (SSPS; Hofmann & DiBartollo, 2000).

The intervention

We used the modified dot-probe task (MacLeod et al., 2002) to train biases in attention. Every trial began with a fixation cross presented for 500 ms in the middle of the screen. After that, two stimuli were simultaneously presented below and above the fixation cross for another 500 ms. One of the two stimuli was negative (i.e., disgusted facial expression), while the other one was neutral (i.e., neutral facial expression). Next, a neutral probe (e.g., letter F or E) appears at the location previously occupied by one of the two stimuli, i.e., at the location of either the negative stimulus (= congruent trials) or the neutral stimuli (= incongruent trials). Participants were asked to indicate as fast and correct as possible the cue type (i.e., E and F) by pressing the left or right button of the computer mouse. To train attention away from negative stimuli and towards neutral ones, for participants in the first group the target replaces the neutral stimulus in 80% of the trials. In contrast, to train attention away from neutral stimuli and towards the negative ones, for participants in the second group the target replaced the negative stimulus in 80% of the trials. For control condition, the target replaced negative or neutral stimulus with equal probability. Participants were presented with a total of 160 trials, consisting in all possible combinations of probe type (E or F), probe position (up or bottom), and cue type (neutral face or face expressing disgust).

The imagistic stimuli we used were selected from two sets of standardized images:

(a) **Human faces stimuli** were selected from Matsumoto & Ekman (1989) stimulus set; we selected 8 images depicting neutral human faces and another 8 images of the same persons, showing disgust. These stimuli were used for the active ABM condition with human faces and for the sham ABM (control condition).
(b) **Imagistic stimuli irrelevant for social phobia**, depicting neutral scenes and physical injury were selected from the International System of Affective Pictures (IAPS, Center for the Study of emotion and Attention – National Institute of Mental Health, 1999). For the purposes of this study, we selected 16 pictures (8 neutral and 8 negative). These pictures were used for the active ABM condition with stimuli irrelevant for the socially anxious individuals.

The training task were built after the specifications reported by Schmidt et al. (2009). The intervention protocol included a total of 8 sessions, with two sessions completed per week. The majority of the participants (85 %) completed all the 8 sessions. Each session lasted approximately 20 minutes, during which each participant performed the modified dot-probe task according to the experimental condition he/she was randomized in.

Results

Randomization check

No significant differences between groups were evident in baseline on any variables (all $p > .05$), except for social anxiety measured with SPIN, $F(2, 83) = 3.239$, $p = .044$, positive self-statements in public speaking situations, $F(2, 83) = 3.265$, $p = .043$, and negative self-statements in public speaking situations, $F(2, 83) = 3.964$, $p = .023$. Means and standards deviations for all the variables considered in the study (except for efficiency expectancies) are shown in Table 1.

ABM effect on outcome measures

ABM effect on social anxiety

A 2 (Time: pre and post intervention) x 3(Group: Active ABM with human faces, Sham ABM, and Active ABM with stimuli irrelevant for social anxiety) repeated measure analysis of variance (ANOVA) on LSAS scores showed a significant main effect of time, $F(1, 83) = 32.90$, $p = .00$, $\eta^2 = .28$, but no main significant effects of group were obtained, $F(2, 83) = 1.261$, $p = .289$, $\eta^2 = .029$, or significant effect of the interaction Time x Group, $F(2, 83) = .377$, $p = .687$, $\eta^2 = .009$. On SPIN scores, and obtained similar results: a significant main effect of time, $F(1, 83) = 53.78$, $p = .00$, $\eta^2 = .39$, no significant effect of group, $F(2, 83) = 2.226$, $p = .114$, , $\eta^2 = .05$, and no significant interaction effect of Time x Group, $F(2, 83) = .546$, $p = .482$, $\eta^2 = .01$.

ABM effect on general distress

ANOVA with repeated measures conducted on STAI-T scores revealed a significant main effect of time, $F(1, 83) = 25.54$, $p = .00$, $\eta^2 = .235$, but no significant main effect of group, $F(2, 83) = .633$, $p = .533$, $\eta^2 = .01$ and no significant Time x Group interaction, $F(1, 83) = 1.975$, $p = .145$, $\eta^2 = .04$.

ANOVA with repeated measures conducted on BDI-II scores showed a significant main effect of time, $F(1, 83) = 9.215$, $p = .003$, $\eta^2 = .100$ and a significant effect of Time x Group interaction, $F(2, 83) = 3.949$, $p = .023$, $\eta^2 = .08$, but no significant main effect of group, $F(2, 83) = 1.389$, $p = .255$, $\eta^2 = .03$.

ABM effect on the presumed mechanisms of change

ABM effect on irrationality

A 2 (Time: pre- and post-intervention) x 3 (Group: Active ABM with human faces stimuli, Sham ABM, and Active ABM with stimuli irrelevant for social phobia) on scores obtained at GABS irrationality subscale showed a significant main effect of time, $F(1, 83) = 172.90$, $p = .00$, $\eta^2 = .678$, but non-significant effects of Group, $F(2, 83) = .697$, $p = .501$, $\eta^2 = .017$, and Time x Group interaction, $F(2, 83) = .164$, $p = .849$, $\eta^2 = .004$.

ABM effect on rationality

Similarly, the ANOVA with repeated measures we performed on scores obtained at the GABS rationality subscale showed non-significant main effects of time, $F(1, 83) = 3.037$, $p = .085$, $\eta^2 = .036$, and group, $F(2, 83) = .777$, $p = .463$, $\eta^2 = .0019$, as well as non-significant Time x Group interaction, $F(2, 83) = .859$, $p = .427$, $\eta^2 = .021$.

Table 1

Means (M) and standard deviations (SD) pre- and post-intervention

	Group 1 (*n* = 30)		Group 2 (*n* = 28)		Group 3 (*n* = 28)	
	Pre: *M (SD)*	Post: *M (SD)*	Pre: *M (SD)*	Post: *M (SD)*	Pre: *M (SD)*	Post: *M (SD)*
LSAS	63.23 (21.06)	55.63 (24.79)	68.35 (20.00)	59.42 (21.62)	73.64 (21.23)	62.67 (23.95)
SPIN	52.20 (11.79)	44.60 (13.74)	53.28 (11.04)	46.07 (12.84)	58.89 (8.81)	49.00 (10.76)
STAI	52.33 (8.54)	50.93 (8.93)	52.17 (6.37)	48.17 (7.27)	53.85 (8.32)	51.10 (8.96)
BDI-II	9.40 (6.10)	9.53 (8.13)	10.89 (9.15)	9.25 (0.07)	14.75 (9.85)	10.75 (7.72)
GABS.Ir	61.86 (14.53)	48.33 (15.99)	57.92 (15.97)	44.33 (14.54)	62.03 (14.12)	47.17 (11.34)
GABS.R	16.26 (2.46)	16.36 (2.28)	16.62 (1.77)	16.88 (2.06)	15.78 (1.89)	16.53 (1.91)
ATQ	36.06 (13.86)	35.60 (14.61)	36.75 (12.62)	31.32 (11.98)	41.35 (12.36)	36.85 (11.29)
BFNER	44.06 (9.37)	40.06 (9.37)	45.10 (9.36)	41.14 (10.73)	48.14 (8.06)	44.46 (9.99)
SSPS.poz	15.46 (5.17)	15.53 (4.65)	13.78 (4.87)	14.57 (4.10)	12.28 (4.90)	13.85 (3.96)
SSPS.neg	11.70 (5.17)	10.43 (5.72)	11.03 (5.38)	11.14 (6.28)	14.82 (5.61)	11.60 (5.03)

Note: ATQ = *Automatic Thoughts Questionnaire* (Hollon & Kendall, 1980); BDI-II = *Beck Depression Inventory – II* (Beck et al., 1996); BFNER = *Brief Fear of Negative Evaluation – Revised* (Carleton et al., 2006); GABS.Ir = Irrationality subscale of the *General Attitudes and Beliefs Scale* (Lidner et al., 1999); GABS.R = Rationality subscale of the *General Attitudes and Beliefs Scale* (Lidner et al., 1999); LSAS = *Liebowitz Social Anxiety Scale* (Liebowitz, 1987); STAI = *State Trait Anxiety Inventory* (Spielberger et al., 1983); SPIN = *Social Phobia Inventory* (Connor et al., 2000); SSPS.poz = positive self-statements subscale of the *Self-Statements in Public Speaking Situation* (Hofmann & DiBartollo, 2000); SSPS.neg = negative self-statements subscale of *the Self-Statements in Public Speaking Situation* (Hofmann & DiBartollo, 2000).

ABM effect on negative automatic thoughts

ANOVA with repeated measures conducted on ATQ scores showed a significant main effect of time, $F(1, 83) = 10.607$, $p = .002$, $\eta^2 = .113$, no significant main effect of group, $F(2, 83) = 1.310$, $p = .275$, $\eta^2 = .031$, and non-significant effect of Time x Group interaction, $F(2, 83) = 2.094$, $p = .130$, $\eta^2 = .048$.

ABM effect on fear of negative evaluation

ANOVA with repeated measures conducted on BFNER scores indicated a significant main effect of time, $F(1, 83) = 23.224$, $p = .000$, $\eta^2 = .219$, no significant main effect of group, $F(2, 83) = 1.613$, $p = .205$, $\eta^2 = .037$, and non-significant effect of Time x Group interaction, $F(2, 83) = .012$, $p = .988$, $\eta^2 = .00$.

ABM effect on self-statements in public speaking situations

ANOVA with repeated measures conducted on SSPS scores, *positive statements subscale*, showed a non-significant main effect of time, $F(1, 83) = 3.031$, $p = .085$, $\eta^2 = .035$, no significant main effect of group, $F(2, 83) = 2.732$, $p = .071$, $\eta^2 = .062$, and non-significant effect of Time x Group interaction, $F(2, 83) = .886$, $p = .416$, $\eta^2 = .021$.

ANOVA with repeated measures conducted on SSPS scores, *negative statements subscale*, showed a significant main effect of time, $F(1, 83) = 10.260$, $p = .002$, $\eta^2 = .110$, and no significant main effect of group, $F(2, 83) = 1.637$, $p = .201$, $\eta^2 = .038$.However, the Time x Group interaction was significant, $F(2, 83) = .886$, $p = .416$, $\eta^2 = .021$. Because there were significant differences between groups on SSPS negative statements subscale in baseline, to follow up this interaction we performed an ANCOVA analysis, with scores in baseline as covariate and group as the between factor. Results showed anon-significant main effect of group, $F(2, 83) = 2.277$, $p = .109$, $\eta^2 = .053$, but a significant effect of the SPSS (negative statements subscale) scores obtained in baseline, $F(1, 83) = 84.367$, $p = .000$, $\eta^2 = .507$

ABM effect on expectancies

Means and standard deviations for efficiency expectancies per group are shown in Table 2. Repeated measures ANOVA revealed a main effect of time on expectancies, $F(1, 68) = 4.577$, $p = .036$, $\eta^2 = .063$, indicating a decrease of efficiency expectancies from pre- to post-intervention across groups. The main effect of group was also significant, $F(2, 68) = 3.298$, $p = .043$, $\eta^2 = .088$. However, the Time x Group interaction was non-significant, $F(2, 68) = .525$, $p = .594$, $\eta^2 = .015$.

Table 2

ABM efficiency expectancies: means (M) and standard deviations (SD)

	Group 1 (n = 22)	Group 2 (n = 25)	Group 3 (n = 24)
	M (SD)	*M (SD)*	*M (SD)*
1st Session	5.30 (1.94)	5.98 (2.25)	4.83 (2.00)
4th Session	4.26 (2.07)	5.80 (2.56)	4.34 (2.13)
Last session	4.21 (2.41)	5.65 (2.86)	4.28 (2.79)

Discussion

This study was aimed to replicate the finding that ABM is efficient in decreasing social phobia symptoms. At the same time, we aimed to extend the previous findings with respect to role played by type of stimuli used during training and the possible alteration of self-report cognitive factors (i.e., cognitive mechanisms targeted in classical CBT intervention for social phobia) following ABM. Our results did not replicate the previous findings regarding the clinical potential of ABM, i.e. we did not find any differences between training groups and control group. Social phobia symptoms seemed to decrease across groups from pre- to post-intervention. However, this decrease seemed to be clinically insignificant, as LSAS mean scores across groups remained in clinical range (i.e., above 50; see Table 1). Moreover, we did not find any evidence that ABM impacted general distress, or self-report cognitive factors. Expectancies regarding ABM efficiency progressively decreased within groups from pre- to post-intervention, with no differences between groups post-intervention. The results of this study suggest that exposure (to neutral and negative human faces photos) does not have any notable beneficial effect for social anxious individuals. It is possible that exposure in this format is too weak to induce anxiety, as the person is not in a real social situation and his/her performance is unlikely to be evaluated. Therefore, the level of stress and arousal during ABM exposure could be insufficient to promote any change. Future studies should examine more closely the therapeutic potential of this form of exposure.

Our results did not support ABM as a therapeutic intervention, nor do they indicate that stimuli used during training contribute to the previous reported beneficial ABM effect. These findings add to the current experimental data regarding ABM efficacy in anxiety and call for future clarifications regarding the extent in which ABM reliably modifies attention bias – the basic condition to seek for a modification of symptoms and/or other cognitive factors.

STUDY 4: How does attention bias modification work? Attentional control, negative automatic thoughts, and expectancies as possible mechanisms

Introduction

Initial findings with attention bias modification (ABM; e.g., Amir, Beard, Burns, et al., 2009; Amir, Beard, Taylor, et al., 2009; Schmidt et al., 2009) stirred a lot of interest, enthusiasm, and hope of innovating current treatment options. However, recently it has become evident that ABM efficiency in reducing anxiety symptoms are not always replicable (e.g., Boettcher et al., 2012; Carlbring et al., 2012; Julian et al., 2012; see also our findings in Study 3).

This situation may be due to the fact that it is unclear what ABM modifies, and when (under which conditions) does it work. The presumed mechanism of ABM is the reduction of negative attentional bias. However, the very notion of attentional bias (in terms of its components and processes involved) is not clear (for recent reviews aimed to integrate the available data in coherent theoretical models, see Cisler & Koster, 2009; Quimet et al., 2009). The way in which attentional bias contributes to the maintenance of anxiety may be explained from different theoretical perspectives: valence specific models and attentional control models of attentional bias.

Although both valence specific models and attentional control models of attentional bias in anxiety have empirical support, are intensively researched, and seem to be rather complementary than contradictory, the theoretical accounts of attentional bias are rarely linked to ABM (see also Heeren et al., 2013). ABM mechanism of change is crucially important in understanding whether it's worthy to use ABM as a clinical tool, when and for whom we can use it, and how we can maximize its clinical potential. What we know for now about ABM mechanisms is that it probably facilitates disengagement from negative (threatening) stimuli (see Amir et al., 2009; Heeren et al., 2011). However, alternative explanations are possible as well. For example, from a valence specific models perspective, ABM may simply train attentional avoidance rather than disengagement (see also Carlbring et al., 2012). If ABM trains attentional avoidance, the reduction in anxiety symptoms could be explained in terms of temporary "removing" the activating event. However, this effect would be a *feeling better*, and not a *getting better* (David, 2006; David et al., 2009), because in case the person cannot avoid the negative stimuli, the anxiety will rebound. Therefore, on the long term this could contribute to the maintenance of the anxiety rather than to anxiety reduction. By contrast, if ABM promotes attentional disengagement, the beneficial effect could be lasting, given that, even when a person cannot avoid a negative stimulus/situation, (s)he is still capable of disengaging from it.

But was does disengagement means? In order to successfully disengage from a negative stimulus, one should (1) engage with that stimulus and (2) be able to switch attention flexibly from that stimulus to another one, depending on his/her current goal(s). In order words, we believe that disengagement requires attentional control. If ABM truly facilitates disengagement, then it should improve attentional control.

In this context, this study was aimed to test the protective ABM effect in a stressful situation, while verifying at the same time ABM possible mechanisms of change. Specifically, we verified (1) if ABM induced the expected attentional bias according to the training condition, and (2) if ABM had any impact on the attentional control, negative automatic thoughts, and expectancies. We

expected ABM to modify attentional bias according to training condition. In addition, we expected participants in the neutral training group to report lower anxiety during the experimental stressor task compared with participants in the control group. No other hypotheses were formulated regarding the relationship between ABM and other variables in this study, i.e., we investigated exploratory these relationship.

Method

Design

We used an experimental design with repeated measures. The independent variable was the training condition. Participants were randomly distributed in three training conditions: Training toward neutral stimuli (Group 1); Training toward negative stimuli (Group 2); Sham ABM (control condition) (Group 3).

The dependent variables were outcome measures and presumed mechanism of change measures. The *primary interest outcome* was the self-reported anxiety during the experimental stressor task. The *secondary interest outcome* was the self-evaluated performance during the experimental stressor task. In terms of *mechanisms of change*, we considered: Attentional bias; Attentional control; Participants' expectancies for anxiety; Negative automatic thoughts.

Before experimental session, we measured also the following *trait variables* (as potential moderators of the ABM effect on the dependent variables): general irrationality, self-report attentional control, fear of negative evaluation, trait anxiety, and self-statements in public speaking situations.

Participants

A total of 87 potential participants were initially recruited from the participants at Junior Summer University, an educational project addressed to high school teenagers, organized by the Students' Organization in Babeş -Bolyai University, Cluj-Napoca. Our participants were volunteers who agreed to participate in order to have a glimpse of how an experimental psychology study looks like. Our final sample included 81 healthy participants (62% females; mean age = 18.82, standard deviation = 1.64.).

Materials and measures

Trait variables. The following trait variables were measured: *general irrationality* (measured with Attitudes and Beliefs Scale – II, ABS-II; DiGiuseppe, Leaf, Exener, & Robin, 1988), *self-report attentional control* (measured with Attentional Control Scale, ACS; Derryberry & Reads, 2002), *fear of negative evaluation* (measured with Brief Fear of Negative Evaluation – Revised, BFNER; Carleton, McCreary, Norton, & Asmundson, 2006), *trait anxiety* (measured with State-Trait Anxiety Inventory, STAI-X2; Spielberger, Gorsuch, Luchene, Vagg, & Jacobs, 1983), and *self-statements in public speaking situations* (measured Self-Statements in Public Speaking Situations scale, SSPS; Hofmann & DiBartollo, 2000).

Experimental tasks

***Attentional bias measurement task*.** To measure attentional bias, we used a spatial cueing task adaptated from Koster, Crombez, Verschuere, Van Damme, et al. (2006) and previously used by Cocia, Uscatescu, and Rusu (2012). Each trial began with a fixation cross presented for 500 ms in

the middle of the screen, flanked by two white rectangles (dimensions: 6 x 8 cm). The middle of each rectangle was 7.5 cm apart from the central fixation cross. Photos of neutral, fearful, or disgusted human faces selected from the NimStim Stimulus Set (Tottenham, Borscheid, Ellertsen, Marcus, & Nelson, 2002; Tottenham et al., 2009) were randomly presented in one of the rectangles, at the right/left side of the fixation cross, for 700 ms. These photos were used as cues. After cue disappearance, participant was presented with a target either at the cue location (i.e., valid trials) or at the opposite location (i.e., invalid trials). The cue onset and the target onset were separated by a 500 ms interval. The target was a small black square (dimensions; 1.1 x 1.1. cm). Participants were asked to indicate the location of the target by pressing one of two different keys.

Attentional control task. We used the Attentional Network Task (ANT, Fan et al., 2002). ANT is an experimental procedure designed to provide a behavioral measure of the attentional alerting, orienting, and conflict. Attentional conflict measure is considered to capture the executive function of attention and was the index in which we were interested for the purposes of the current study. ANT version used in this study was designed based on the parameters originally described by Fan et al. (2002). Every trial began with a fixation cross presented in the center of the screen. After a random interval (varying between 400 and 1600 ms) a cue (i.e., an asterix) was briefly presented (100 ms). There were four cue conditions[5] (no cue; central cue; double cue; spatial cue; see Figure 3). Following cue disappearance, the central fixation cross was presented alone for another 400 ms; then a combination of five stimuli (i.e., arrows *or* lines and arrows) was presented below or above the fixation cross and the participant was required to indicate the direction of the central arrow (i.e., to determine if the central arrow point to left or right) as soon as possible, by pressing a corresponding key. The target (i.e., the central arrow) and flankers remain on the screen until the participant gave his/her response or until 1700 ms elapsed.The stimuli were presented in black against a white background, 3 cm above or below the central fixation cross. There were 24 practice trials, followed by a total of 228 experimental trials. Response accuracy and reaction time were recorded for every trial.

ANT allows the derivation of three attention network scores: alerting, orienting, and attention control. For the purposes of this study, only attentional control scores were computed, by subtracting the mean reaction time for congruent trials from the mean reaction time for incongruent trials (see Figure 3 to understand what a congruent/incongruent trial is). Positive scores indicated attentional interference (i.e., poorer attentional control) in the presence of distracting stimuli (see Fan et al., 2002, 2005; Weslye, Grydeland, Walhovd, & Fjell, 2011).

Training task. We used the modified dot-probe task (MacLeod et al., 2002) to train biases in attention. Every trial began with a fixation cross presented for 500 ms in the middle of the screen. After that, two stimuli were simultaneously presented below and above the fixation cross for another 500 ms. One of the two stimuli was negative (i.e., disgusted facial expression), while the other one was neutral (i.e., neutral facial expression). Next, a neutral probe (e.g., letter F or E) appears at the location previously occupied by one of the two stimuli, i.e., at the location of either the negative stimulus (= congruent trials) or the neutral stimuli (= incongruent trials). Participants were asked to indicate as fast and correct as possible the cue type (i.e., E and F) by pressing the left or right button of the computer mouse. To train attention away from negative stimuli and towards neutral ones, for

[5] Cue condition is irrelevant for computing the conflict (i.e., attentional control) index; therefore, it will not be discussed further

participants in the first group the target replaces the neutral stimulus in 80% of the trials. In contrast, to train attention away from neutral stimuli and towards the negative ones, for participants in the second group the target replaced the negative stimulus in 80% of the trials. For control condition, the target replaced negative or neutral stimulus with equal probability. Participants were presented with a total of 160 trials, consisting of all possible combinations of probe type (E or F), probe position (up or bottom), and cue type (neutral face or face expressing disgust).

Stimuli used for the training task were selected from Matsumoto & Ekman (1989) stimulus set and included eight photos of males and eight photos of females. Half of these stimuli portrayed neutral facial expressions (four males, four females) while the other half portrayed disgust (four males, four females; every male/female character was represented with two photos, portraying one a neutral expression, and the other one a disgust expression). The dimensions of the photos used during the training phase were 6.3 x 4.4 cm.

Self-report state measures

Outcome variables.

Anxiety. We used Visual Analogue Scales (VAS; Holstein & Luria, 1973; Williams, Morlock, & Feltner, 2010) to measure how anxious participants felt before the impromptu speech. Participants were asked to mark a point on a 12 mm horizontal line to indicate how anxious they feel in that moment (i.e., just before starting to speak).

Self-reported performance. Following the speech, participants were asked to rate their performance using another 12 mm VAS.

Mechanisms of change.

Anxiety expectancies. We measured anxiety expectancy with a VAS. Before the impromptu speech, participants were asked to mark a point on a 12 mm horizontal line to indicate how anxious they expect to be during the speech.

Negative cognitions. In order to measure negative cognitions related to the specific experimental stressor, we used adapted versions of BFNER and SSPS described above.

Experimental stressor task

Participants were asked to give an impromptu 5-minute speech. This task was designed to maximize the probability of inducing stress. First, participants were asked about their opinion on a controversial topic (e.g., legalizing euthanasia). First, participants were asked about their opinion on a controversial topic (e.g., legalizing euthanasia; a complete list of the topics is available in Appendix 3). Next, they were told that they should give a 5-minute speech during which they should argue the opposing point of view (e.g., if the participant said she/he thinks that legalizing euthanasia is ok, she/he was asked to argue the point of view according to which euthanasia should not be legalized).

Results

Means and standard deviations for trait variables are shown in Table 1. No significant differences between groups were evidenced.

ABM effect on anxiety and self-reported performance

Means and standard deviations for outcome measures are shown in Table 2. One way analysis of variance (ANOVA) conducted on the anxiety ratings revealed no differences between groups, $F(2, 78) = 1.327$, $p = .271$. Similarly, one way ANOVA conducted on the performance ratings evidenced no differences between groups, $F(2, 78) = .455$, $p = .636$.

Table 1. *Means (M) and standard deviation (SD) for measures of trait variables*

	Group 1: Neutral training (n = 27)	Group 2: Negative training (n = 27)	Group 3: Sham training (n = 27)	One way Anova
STAI-X2	41.66 (9.53)	43.37 (12.70)	41.96 (9.59)	$F(2, 78) = .195$, $p = .823$
ABS-II	90.00 (35.49)	92.59 (45.14)	97.44 (44.25)	$F(2, 78) = .220$, $p = .803$
BFNER	34.11 (13.11)	36.96 (12.64)	34.00 (10.12)	$F(2, 78) = .526$, $p = .593$
SSPS.poz	18.07 (3.88)	17.22 (5.08)	16.74 (4.81)	$F(2, 78) = .576$, $p = .575$
SSPS.neg	9.62 (5.79)	10.00 (6.79)	9.25 (6.67)	$F(2, 78) = .089$, $p = .915$
ACS	53.33 (5.08)	50.48 (6.12)	51.51 (6.09)	$F(2, 78) = 1.679$, $p = .193$

Note: ABS = *Attitude and Beliefs Scale* (DiGiuseppe et al., 1988); ACS = *Attention Contrl Scale* (Derryberry & Read, 2002); BFNER = *Brief Fear of Negative Evaluation – Revised* (Carleton et al., 2006); STAI-X2 = *State Trait Anxiety Inventory* (Spielberger et al., 1983); SSPS.poz = positive self-statements subscale of the *Self-Statements in Public Speaking Situation* (Hofmann & DiBartollo, 2000); SSPS.neg = negative self-statements subscale of the *Self-Statements in Public Speaking Situation* (Hofmann & DiBartollo, 2000).

Table 2. *Means (M) and standard deviations (SD) for outcome measures*

	Group 1: Neutral training (n = 27)	Group 2: Negative training (n = 27)	Group 3: Sham training (n = 27)
Current anxiety	5.18 (2.90)	4.49 (3.53)	3.87 (2.19)
Self-reported performance	4.00 (2.28)	4.14 (3.28)	3.43 (3.06)

Note: Both current anxiety and the self-reported performance were measured using a 12-mm Visual Analogue Scale (VAS; Holstein & Luria, 1973; Williams, Morlock, & Feltner, 2010)

ABM effect on presumed mechanisms of change

ABM effect on attentional bias

Two types of attentional bias indicators were derived: engagement scores and disengagement scores. Engagement bias was computed by subtracting reaction time (RT) mean for the neutral valid trials from the RT mean for negative valid trials. Disengagement bias was computed by subtracting the RT mean for neutral invalid trials from the RT mean for the negative invalid trials. Means and standard deviations for AB indicators are shown in Table 3.

A 2 (Time: Pre and post ABM) x 3 (Group: Training towards neutral stimuli, Training towards negative stimuli, and control group) ANOVA with repeated measures conducted on the

attentional bias scores computed for engagement with disgust revealed a main effect of time, $F(1, 69) = 31.373$, $p = .000$, $\eta^2 = .313$, a non-significant main effect of group, $F(2, 69) = 1.468$, $p = .238$, $\eta^2 = .041$, and a non-significant effect of Time x Group, $F(2, 69) = .219$, $p = .804$, $\eta^2 = .006$. The same analysis conducted on the engagement with fear scores revealed no significant main effects of Time or Group (*ps* > .443), and no significant effect of Time x Group interaction, $F(2, 69) = 1.175$, $p = .177$, $\eta^2 = .049$.

A 2 (Time: Pre and Post ABM) x 3 (Group: Training towards neutral stimuli, Training towards negative stimuli, and control group) ANOVA with repeated measures conducted on the disengagement from disgust scores showed no main effect of Time, $F(1, 69) = 1.095$, $p = .299$, $\eta^2 = .016$, or Group, $F(2, 69) = 1.138$, $p = .326$, $\eta^2 = .032$. Although the groups' means were in the right direction (see Table 3), the Time x Group interaction failed to reach statistical significance, $F(2, 69) = 2.579$, $p = .083$, $\eta^2 = .070$.

A similar ANOVA with repeated measures conducted on disengagement from fear scores showed no main effect of Time, $F(1, 69) = .318$, $p = .575$, $\eta^2 = .005$, or Group, $F(2, 69) = .006$, $p = .994$, $\eta^2 = .00$. However, the Time x Group interaction was significant, $F(2, 69) = 4.366$, $p = .016$, $\eta^2 = .112$. To follow-up this interaction effect, we conducted separated one-way ANOVA on the disengagement scores measured in baseline and post ABM. Results showed no significant differences between groups, either in baseline, $F(2, 72) = 1.511$, $p = .228$, or following the intervention, $F(2, 72) = 1.736$, $p = .183$. Paired *t* tests showed significant changes within the trained groups, in the expected direction: for neutral training group, $t(23) = 2.172$, $p = .04$, Cohen's $d = .44$, and for negative training group, $t(23) = -2.025$, $p = .05$, Cohen's $d = .41$. No significant change was evident in the control group, $t(23) = -1.118$, $p = .27$.

ABM effect on attentional control

Due to some technical errors, attentional control data were available only from 54 participants. Based on protocol of data analysis reported in the literature (see Welsye et al., 2011), we excluded reaction times lower than 250 or higher than 1500. Attentional control index was computed by subtracting the mean reaction time for congruent trials from the mean reaction time for incongruent trials (higher values indicates poorer attentional control; Fan et al., 2002, 2005).

Means and standard deviations for attentional control index, measured pre and post ABM, are shown in Table 4. A 3 (Group: Neutral training, Negative training, Control group) x 2 (Time: Pre and Post ABM) ANOVA with repeated measures showed a main effect of time, $F(1, 51) = 38.00$, $p = .00$, $\eta^2 = .427$, indicating a significant decrease of attentional control from pre to post measurement across groups. No main effect of group, $F(2, 51) = .367$, $p = .695$, $\eta^2 = .014$, and no significant Time x Group interaction, $F(2, 51) = .363$, $p = .697$, $\eta^2 = .014$, were evident.

ABM effect on negative cognitions specifically related to the experimental stressor

Means and standard deviations for cognitive measures collected after the ABM training and before the experimental stressor are shown in Table 5.

One way ANOVA conducted on BFNER scores revealed no differences between groups, $F(2, 78) = .190$, $p = .827$. Similar results were obtained when one way ANOVA was conducted on SSPS scores (negative subscale), $F(2, 78) = .796$, $p = .455$, or anxiety expectancy, $F(2, 78) = .445$, $p = .642$.

Table 3. *Means (M) and standards deviations (SD) for attentional bias indicators, pre and post intervention*

		Group								
		Group 1: Neutral training			Group 2: Negative training			Group 3: Sham training		
Emotion	Attentional Bias	*n*	Pre: *M* (*SD*)	Post: *M* (*SD*)	*n*	Pre: *M* (*SD*)	Post: *M* (*SD*)	*n*	Pre: *M* (*SD*)	Post: *M* (SD)
Disgust	Engagement	24	-53.15 (69.70)	-5.52 (21.84)	24	-31.11 (75.64)	2.71 (25.22)	25	-39.54 (37.14)	3.35 (24.20)
	Disengagement	25	3.93 (30.77)	-6.50 (27.73)	25	-5.37 (40.83)	9.59 (37.83)	25	.40 (23.97)	14.38 (23.68)
Fear	Engagement	25	.31 (40.57)	-11.57 (20.0)	25	-15.37 (33.55)	2.30 (24.19)	25	.15 (41.14)	-5.04 (22.47)
	Disengagement	25	12.97 (40.85)	-6.27 (26.23)	25	-5.80 (37.02)	10.97 (47.52)	25	-4.17 (48.30)	8.52 (28.57)

Table 4. *Means (M) and standard deviations (SD) for attentional control index*

	Group 1: Neutral training (*n* = 17)		Group 2: Negative training (*n* = 18)		Group 3: Sham training (*n* = 19)	
	Pre: *M (SD)*	Post: *M (SD)*	Pre: *M (SD)*	Post: *M (SD)*	Pre: *M (SD)*	Post: *M (SD)*
Attentional Control	419.68 (237.81)	472.43 (271.30)	475.25 (142.29)	519.89 (169.22)	408.55 (222.27)	470.95 (275.20)

Table 5. *Means (M) and standard deviations (SD) for negative cognitions measured in the context of the experimental stressor*

	Group 1: Neutral training (n = 27)	Group 2: Negative training (n = 27)	Group 3: Sham training (n = 27)
	M (SD)	*M (SD)*	*M (SD)*
BFNER	25.59 (10.38)	27.25 (12.94)	27.00 (8.15)
SSPS.neg	7.22 (4.66)	8.85 (6.68)	7.29 (4.44)
Anxiety expectancy	6.25 (2.49)	6.77 (3.55)	6.03 (2.69)

Note: BFNER = *Brief Fear of Negative Evaluation – Revised* (Carleton et al., 2006); SSPS.neg = negative self-statements subscale of the *Self-Statements in Public Speaking Situation* (Hofmann & DiBartollo, 2000). Anxiety expectancy was measured using a 12-mm visual analogue scale (VAS; Holstein & Luria, 1973; Williams, Morlock, & Feltner, 2010).

Exploratory *post hoc* analyses

We exploratory investigated if ABM intervention had any effect on positive self-statement in public speaking situations. One way ANOVA conducted on SSPS scores (positive subscale) showed no differences between groups, $F(2, 78) = 2.354$, $p = .102$. Given that we found no ABM effect on attentional bias or on attentional control, we verified the consistency of measuring attentional bias and attentional control. In baseline, engagement with fearful faces correlated moderately, but non-significantly with engagement with disgust, $r(24) = .324$, $p = .124$. Post ABM, the correlation between engagement with fear and engagement with disgust was large and significant, $r(24) = .622$, $p = .001$. However, engagement with disgust measured pre ABM correlated trivially with engagement with disgust measured post ABM, $r(24) = -.058$, $p = .787$. Moreover, engagement with fearful faces measured pre ABM correlated moderately and negatively with engagement with fearful faces measured post ABM, $r(24) = -.348$, $p = .096$.

Similar results were obtained when we correlated disengagement scores obtained for baseline measure with disengagement scores obtained from post ABM measure. In contrast, the correlation between attentional control index measured in baseline and attentional control index measured post ABM was large and positive, $r(18) = .975$, $p = .000$.

Discussion

This study was aimed to investigate (1) the ABM efficiency in lowering anxiety associated with an experimental stressor, and (2) ABM possible mechanisms of change, i.e., attentional bias, attentional control, and negative cognitions. Contrary to our hypotheses, we found no ABM effect on anxiety, or on attentional bias. Our results are in strike opposition to some previous reported results (e.g., Amir, Weber, Beard, Bomyea, & Taylor, 2008; Dandeneau, Baldwin, Baccus, Sakellaropoulo, & Prussner, 2007; Heeren et al., 2012). However, they go along with other recent negative results reported in the literature (e.g., Julian et al., 2012).

First of all, ABM did not reliably modified attentional bias, the key element that it is expected to change. Importantly, our *a posteriori* analyses challenged this possibility, as the attentional bias measure seemed to be very unstable in time. Therefore, it is difficult to assess to what extend the observed changed in attentional bias score is due to the intervention or to random variations. This is a very big problem for ABM research. Future studies should explicitly investigate the attentional bias stability in various samples.

On the other hand it is possible that ABM had an effect on attentional bias, but we were unable to evidence it, because we used different tasks for ABM training and for bias assessment, respectively. Recent findings suggested the ABM effect might be task dependent (see Van Bockstaele, Koster, Verschuere, Crombez, & De Houwer, 2012). This issue should be investigated in subsequent research work.

Second, we find no differences between groups on outcome variables (not surprisingly, as no change was evident on attentional bias), or on the other presumed mechanisms of change. The lack of the ABM impact on anxiety may be due to the fact that our experimental stressor task seemed to induce only moderate anxiety, despite our efforts to maximize its stressful potential.

No ABM effect was found on attentional control. This could indicate that ABM has no impact at all on the executive attention. However, we used ANT to measure executive attention. ANT is a behavioral task using non-emotional stimuli. If the valence specific mechanisms act in concert with executive attention, then it is possible to evidence an eventual change on attentional control following ABM only when emotional stimuli are used. Future studies should investigate this possibility.

ABM had no impact on the negative cognitions. These findings replicated our results in the previous study, this time in the context of an activating event. However because we were unable to evidence any effect of the intervention, we cannot conclude that ABM does not impact negative cognitions at all. It is still possible that when ABM modifies attentional bias and has an effect on negative emotions, it might impact also on the negative cognitions. Future ABM studies should include cognitive measures on a regular basis in order to adequately test this possibility.

Our results do not support the ABM efficacy in lowering distress in stressful situations. Several implications for future research work can be derived based on our results. First, the nature of attentional bias (in terms of its stability) should be established, and the psychometric properties of attentional bias assessment should be more carefully investigated. Second, the pre-existent attentional bias should be used to select participants more likely to respond to ABM. Third, ABM impact on attentional control and negative cognitions should be further investigated.

In conclusion, despite its negative results, this study raised a series of inquiries and possible explanations that have the potential of advance our understanding of ABM clinical potential. Some of them will be followed up in the next study.

STUDY 5. Does attention bias modification improve the attentional flexibility of processing affective stimuli? Results from a randomized experimental study

Introduction

The reduction of negative attentional bias is assumed to be the mechanism of ABM. Negative findings with ABM seem to accumulate and challenge the clinical utility of this experimental intervention. There is no clear to what extent the ABM effect is task dependent or not, or when (and why) it works. Understanding its mechanisms is crucial for improving its efficacy and understanding the conditions in which it works.

Given that (1) the attentional bias reduction is not always achieved, and (2) decrease in attentional bias (when it is evidenced) following ABM is not always accompanied by symptoms decrease, there is possible that a third variable could influence both attentional bias and symptoms. Such a variable may be attentional control or flexibility, probably involved in flexible attending to and disengagement from emotional material. Actually, flexible affective processing (defined as the specific ability to switch back and forth between processing the affective and non-affective qualities of affective information) has been shown recently to be predictive of trait resilience (Genet & Siemer, 2011). As the core characteristic of successful disengagement is the ability to switch attention flexibly from a negative stimulus to another one, depending on the current goal(s), if ABM works through facilitating disengagement, it should improve attentional flexibility of processing emotional stimuli.

We investigated in the previous study the possibility that ABM change general attentional control and failed to find that. However, there we used a general attentional control task, with non-emotional stimuli. It is possible that the ABM effect on attention flexibility can be evidenced only when emotional stimuli are used. Similarly, in the previous study we failed to find an ABM effect on attentional bias. However, there we used different tasks and different stimuli for attentional bias assessment, and for ABM, respectively.

This study was designed (1) to replicate the ABM effect on attentional bias using the same procedure and stimuli for bias assessment and training task, and (2) to test the extent to which the modification in attentional bias is associated with an improved attentional flexibility of processing affective stimuli. We expected attentional bias to be modified in accordance with the experimental manipulation. More specifically, we expected that (1) participant in the neutral training group will exhibit significantly lower attentional bias following training compared with the participants in the control group; and (2) participants in the negative training group will exhibit significantly higher attentional bias following training compared with participants in the control group. We exploratory investigated if attentional bias manipulation has any effect on the attentional flexibility of processing affective stimuli.

Method

Design

We used an experimental design. The *independent variable* was the manipulation of attentional bias through ABM. Participants were randomized in three groups: one group was trained to preferentially process neutral stimuli (Group 1; neutral training), the other was trained to preferentially process negative stimuli (Group 2; negative training), and the third one was the control group (Group 3; sham training).

As *dependent variables*, we measured attentional bias and the attentional flexibility of emotional information. In addition, as potential moderators of the ABM effect on the dependent variables, we measured the following *trait variables*: trait anxiety, depressive symptoms, and irrational beliefs.

Participants

We included a total of 74 participants (63.5 % females; Mean age: 21.51, standard deviation: 2.95) recruited from students at Gent University, Belgium. They participated in the experiment as a partial fulfillment of the course requirements or in exchange for a financial reward. This study was approved by the Institutional Review Board of the Gent University and all the participants signed an informed consent form prior to the beginning of the experiment.

Instruments and materials

We use the same stimuli for the attentional bias assessment task and for the ABM training task. The stimuli were twenty four photos selected from the International Affective Pictures System (Lang, Bradley, & Cuthbert, 2005; IAPS has been validated for Dutch population – see Verschuere, Crombez, & Koster, 2001). Twelve out of the twenty four photos were neutral, while the other twelve were negatively valenced. Another six supplementary neutral photos were selected also from IAPS, to be used for practice phase (familiarization with task), before the attentional bias assessment task.

For the task measuring attentional flexibility of processing emotional information, sixty photos were selected from IAPS. Half of them had positive valence, while the other half had negative valence. Both the set of positive images and negative stimuli were selected in such a way that half of the positive photos (and half of the negative photos) depicted animated scenes (persons, group of people, etc.), while the other half of positive (and negative, respectively) images depicted inanimate things (guns, nature scenes, cars, etc.).

The experimental tasks are described below.

Attention bias assessment task and the ABM task. We used the dot probe task (MacLeod, Mathews, & Tata, 1986). For ABM training, we used the modified dot-probe task (MacLeod et al., 2002), with the difference that the contingency between the stimulus type and the cue presented following stimuli pair was manipulated in order to train attention, i.e., to encourage participants to process preferentially certain stimuli. More specifically, participants in neutral training group was presented with three blocks of trials, each consisting of 96 *incongruent* trials, 24 neutral trials, and 18 digit trials. Participants in the negative training group were presented with three blocks of trials, each consisting of 96 *congruent* trials, 24 neutral trials, and 18 digit trials. Participants in the control group were presented with the same number of trials, out of which 24 were neutral trials and 18 were digit trials, 48 were congruent trials, and the remaining 48 were incongruent trials. No feedback was provided during training phase.

Flexible affective processing task. To measure attentional flexibility of processing emotional stimuli, we adapted a task developed by Genet and Siemer (2011). Flexible affective processing task had two phases: accommodation phase and testing phase. Testing phase consisted in 240 trials during which the processing rule alternated randomly. The first 120 trials were consistent trials, while the other 120 were inconsistent trials (see Genet & Siemer, 2011). Whether consistent or inconsistent, trials could be repetitive trials (i.e., the current processing rule was the same as the processing rule active for the anterior trial) or switch trials (i.e., e current processing

rule was different from the processing rule active for the anterior trial. The measure of attentional flexibility of emotional processing stimuli was quantified in terms of reaction time costs associated with the change of the rule (i.e., switching costs). The switching costs were computed by subtracting the mean reaction time for the repetitive trials from the mean reaction time for the switch trials.

Self-report measures. We measured the following trait variables as potential moderators of the ABM effect on the dependent variables: *depressive symptoms* (measured Beck Depression Inventory-II, BDI-II; Beck, Steer, & Brown, 1996); *trait anxiety* (measured with State and Trait Anxiety Inventory, STAI-T; Spielberger, Gorsuch, Luchene, Vagg, & Jacobs, 1983); irrational beliefs (measured with the Belief Scale, BS; Malouff & Schuette, 1986).

Results

Means and standard deviations for the self-reported questionnaires are shown in Table 1. No differences were found between groups on any of these measures.

Table 1. *Means (M) and standard deviations (SD) for the self-report measures*

	Group			
	Neutral training (n = 26)	Negative training (n = 23)	Sham training (n = 25)	One way ANOVA
BDI-II	8.92 (8.61)	5.78 (5.35)	9.80 (7.11)	$F(2, 71) = 2.025$, $p = .140$
STAI-T	41.16 (10.89)	38.17 (9.14)	43.76 (11.48)	$F(2, 71) = 1.699$, $p = .190$
BS	59.48 (8.88)	59.30 (7.61)	61.57 (8.50)	$F(2, 71) = .579$, $p = .583$

Note: BDI-II = *Beck Depression Inventory – II* (Beck et al., 1996); STAI-T = *State Trait Anxiety Inventory* (Spielberger et al., 1983); BS = *The Belief Scale* (Malouff & Schuette, 1986).

ABM effect on attentional bias

Means and standard deviations for attentional bias measured pre- and post- ABM are shown in Table 2.

A 3 (Group: Neutral training, Negative Training, Sham training) x 2 (Time: Baseline, Post ABM) analysis of variance (ANOVA) with repeated measures showed a main effect of time, $F(1, 69) = 6.900$, $p = .011$, $\eta^2 = .091$, but no main effect of group, $F(2, 69) = 1.158$, $p = .320$, $\eta^2 = .032$. Time x Group interaction was not significant, $F(2, 69) = .060$, $p = .942$, $\eta^2 = .002$, indicating no differential response to ABM across groups.

Table 2. *Means (M) and standard deviations (SD) for the attentional bias index*

	Group		
	Neutral training (n = 24)	Negative training (n = 23)	Sham training (n = 25)
	M (SD)	*M (SD)*	*M (SD)*
Baseline measurement	-4.37 (22.02)	.08 (18.26)	-6.64 (21.08)
Post ABM measurement	8.80 (25.36)	13.00 (39.85)	3.24 (29.94)

ABM effect on flexible affective processing

Descriptive data for trials in consistent and inconsistent blocks, as well as *t* values for the paired *t* tests, and *F* values for the between groups differences are shown in Table 3.

Table 3. *Means (M) and standard deviations (SD) for consistent and inconsistent trials in flexible affective processing task*

	Neutral training (n = 25)	Negative training (n = 23)	Sham training (n = 25)	
Block Type	*M (SD)*	*M (SD)*	*M (SD)*	*One way ANOVA*
Consistent block	861.320 (179.88)	872.434 (195.39)	801.16 (211.67)	$F(2, 72) = .936$, $p = .397$
Inconsistent block	1165.44 (203.68)	1132.95 (166.51)	1135.04 (173.85)	$F(2, 72) = .244$, $p = .784$
Paired t test	$t(24) = -10.037$, $p = .000$	$t(22) = -9.325$, $p = .000$	$t(24) = -9.932$, $p = .000$	

Next, repetitive and switch trials were identified. Means and standard deviations per trial type are shown in Table 4. Paired *t* tests confirmed the existence of switching costs (i.e., higher reaction times were obtained when the rule switched across groups; see Table 4). No differences between groups were evident either on repetitive trials or on switch trials (see Table 4 for *F* statistics). Switching costs (which served as the attentional flexibility indicator) were computed by subtracting means for repetitive trials from means for switch trials. A one way ANOVA was conducted on the flexible affective processing scores, separate for consistent and inconsistent block. No significant differences between groups were observed either for consistent block, $F(2, 72) = 1.597$, $p = .210$., or for the inconsistent block, $F(2, 72) = .079$, $p = .924$.

Table 4. *Means (M) and standard deviations (SD) for repetitive and switch trials in flexible affective processing task*

		Neutral training (n = 25)	Negative training (n = 23)	Sham training (n = 25)	
		M (SD)	*M (SD)*	*M (SD)*	*One way ANOVA*
Trial type	Repetitive trials	931.20 (156.76)	907.65 (151.73)	892.24 (165.04)	$F(2, 72) = .385$, $p = .682$
	Switch trials	1079.72 (209.23)	1075.43 (193.48)	1024.24 (183.91)	$F(2, 72) = .594$, $p = .555$
Paired t test		$t(24) = -8.267$, $p = .000$	$t(22) = -9.576$, $p = .000$	$t(24) = -8.933$, $p = .000$	

Exploratory *post hoc* analyses

Given that previous results suggested that ABM effect may depend on the previous attentional bias, we selected only the participants that showed an attentional bias towards threat in baseline and re-run the ANOVA with repeated measures. Eight participants in the control group, 10 in the neutral training group, and 12 in the negative training group showed attentional bias in baseline. No significant main effects of Time or Group, and no significant interaction effect were obtained (all *p*s > .852).

Given that we obtained no ABM effect on bias, we tested the consistency of the bias measurement, by correlating attentional bias baseline scores with the attentional bias scores obtained after ABM in the sham training group. Results showed a small negative correlation, $r(25) = -.163$, $p = .435$, despite the fact that no change on attentional bias from pre to post assessment was expected in the control group.

We exploratory investigated the relationship between the attentional bias, attentional flexibility, and trait variables. For that purpose, we eliminated outliers (± two standard deviations) on every measure. As we found no differences between groups on any of the variables taken into consideration, we run the correlations on the whole sample. The correlation matrix is shown in Table 5.

Table 5. *Correlations between attentional bias, attentional flexibility, and trait variables*

	AB_1	AB_2	AF	BDI-II	STAI-T	BS
AB_1	1	-.055	-.087	-.049	-.162	-.042
AB_2		1	-.120	-.117	-.093	-.135
AF			1	.150	.244*	.259*
BDI-II				1	.730**	.505**
STAI-T					1	.589**
BS						1

Note: ** $p < .01$; * $p < .05$; AB_1 = Attentional bias measured in baseline; AB_2 = Attentional bias measured post ABM; AF = Attentional flexibility; BDI-II = *Beck Depression Inventory* (Beck et al., 1996); STAI-T = *State Trait Anxiety Inventory* (Spielberger et al., 1983); BS = *Belief Scale* (Malouff & Schuette, 1986).

Discussion

This study was aimed to test the ABM effect on attentional bias, and to investigate the extent to which the change in attentional bias is associated with an improved attentional flexibility of processing affective stimuli. Contrary to our hypothesis, we found no effect of ABM on attentional bias, i.e., the attentional bias scores increase from baseline to post measurement across groups. No effect could be evidenced either on flexible attentional processing of emotional information.

The lack of the ABM effect was unexpected, especially, because we used the same procedure and stimuli for bias assessment and training task. Moreover, attentional bias assessment task was administrated immediately before and after ABM. As our post hoc analyses revealed that the two measures of attentional bias were practically unrelated, questions arise regarding the adequacy of the measurement. The lack of correlation between the two measures may be due to task particularities and/or to the nature of the measured phenomenon. Future research should dismantle these two possibilities. No effect was found on flexible affective processing. As we found no effect on bias, this is not surprising.

Overall, our results suggest that the ABM is not powerful enough to induce reliable modifications on attentional processing of emotional information. Attentional bias in healthy individuals may have different characteristics that the attentional bias in clinical samples. Future studies should clarify the nature of attentional bias in different samples (nonclinical, subclinical, clinical) and should investigate the extent to which ABM effect is reliable in clinical samples.

PART 3. COGNITIVE BIASES IN GASTROINTESTINAL DISORDERS

STUDY 6: Attentional bias towards symptoms-related cues predicts analgesics use in patients with gastrointestinal conditions: results from a pilot study[6]

Introduction

Gastrointestinal conditions are highly costly in terms of health care utilization and debilitating in terms of medical care, work productivity and social well-being (Cash, Sullivan, & Barghout, 2005; Talley, 2008). Multiple factors, including genetic make-up, biological dysfunctions, environmental influences, psychological (cognitive, behavioral, emotional), and social factors are thought to be involved in the etiology and persistence of gastrointestinal conditions. The current evidence-based treatment approaches strive to considerate the influence and interplay of all these factors in gastrointestinal (GI) symptoms onset and persistence (Levy et al., 2006).

Arguably, within the mix of factors involved in the etiology and maintenance of GI symptoms, cognitive factors play a key role. In support of the critical role played by cognitive factors in relation with GI symptoms, cognitive-behavioral therapy (CBT) has been shown to be one of the most efficient treatments for functional gastrointestinal disorders (FGID) (Blanchard, 2005; Craske et al., 2011). CBT can be also a valuable adjunct of medical treatment in organic gastrointestinal conditions, by promoting well-being, adjusting to disease, and effective coping with (medical) symptoms. CBT is based on the premise that symptoms are dysfunctional/inadequate responses, learned during life experiences. A special emphasis is put on the capacity of human mind to process information, as information processing is the prerequisite of learning. CBT theory stipulates that people learn dysfunctional thinking patterns (cognitive schemas/beliefs) that predispose them toward distorted perception and interpretation of internal stimuli and external events in such a way that information incongruent with what the system has already learned is discredited, ignored or distorted. Thus, the information processing is biased toward certain kind of information. Cognitive biases are likely to negatively influence perception and appraisals of visceral sensations and consequently the behaviors (health-care seeking behaviors, avoidance of work/leisure activities, engaging in activities aimed to reduce pain etc.), causing symptoms appearance and persistence (Drossman et al., 1999; Levy et al., 2006).

Therefore, cognitive biases can play an active role in relation with gastrointestinal symptomatology, whether the symptoms are organic or functional (e.g., they can influence the patients' coping with their condition). However, the results are not unequivocal (Afzal, Potokar, Probert, & Munafò, 2006; Martin & Chapman, 2010). Moreover, as far as we know, there are no studies that investigated the role of attentional bias in relation with symptoms maintenance. This study was aimed to exploratory investigate attentional bias in relation to GI symptoms, whether they are functional or organic, while taking into consideration at the same time other psychological factors known to be involved in GI symptoms persistence.

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[6] This study is under review at *Journal of Clinical Psychology in Medical Settings*: Mogoaş e, C., David, D., Dumitraş cu, D. (2013). Attentional bias towards symptoms-related cues predicts analgesics use in patients with gastrointestinal conditions: results from a pilot study.

The authors contributed to this manuscript as follows: Mogoaş e, C. – study design and implementation, data analysis, writing the manuscript; David, D. – structuring the manuscript, consultation for study design and writing the manuscript; Dumitraş cu, D. – clinical supervision during data collection, consultation for study design and writing the manuscript.

Method

Design

We employed a cross-sectional design. To statistically analyze the data, we used correlations and linear regression.

Patients

Thirty-two voluntary patients diagnosed with gastrointestinal conditions were recruited from gastrointestinal ward of Adult Hospital from Cluj-Napoca, Romania. Patients were told that the study is aimed to investigate psychological factors related to GI symptoms and were invited to anonymously participate.

Measures

Attentional bias assessment task. We used a version of the dot-probe task (MacLeod, Mathews, & Tata, 1986) to measure attentional bias. We used linguistic stimuli, based on the stimuli used previously by Afzal et al. (2006). Similar to Afzal and colleagues, we selected our neutral stimuli from "household" Romanian words matched for length with the symptom-related words. We used a total 12 symptom-related words, and 12 neutral words. Participants completed a total of 144 trials.

Self-report measures

We measured ***self-report gastrointestinal symptoms*** (*Gastrointestinal Symptoms Questionnaire* , GSQ; Bovenshen et al., 2006), ***symptom-specific anxiety*** (*The Visceral Sensitivity Index*, VSI; Labus et al., 2004), ***pain catastrophising*** (*The Pain Catastrophizing Scale*; PCS; Sullivan, Bishop, & Pivik, 1995), ***irrational beliefs*** (*The Attitude and Belief Scale-II*, ABS-II; DiGiuseppe, Leaf, Exner, & Robin, 1988), ***general distress*** (*The Profile of Mood States-Short Version*, POMS-SV; Shacham, 1983), and ***pain intensity*** *(*measured with a *visual analogue scale*, VAS). Patients were asked also if they use analgesics (yes/no).

Results

Descriptive statistics for the variables included in this study are shown in Table 3. The correlation matrix is shown in Table 4.

Table 3. *Means and standard deviations for attentional bias and self-report measures*

Variable	*n*	Mean	Standard deviation
GSQ	30	37.86	20.16
AB	32	5.72	18.79
ABS-II: Rationality	32	113.87	17.47
ABS-II: Irrationality	31	62.45	30.38
VSI	31	48.29	17.79
PCS	32	24.21	13.53
POMS-SV	31	56.16	27.50

Note: GSQ = *Gastrointestinal Symptoms Questionnaire* (Bovenshen et al., 2006); AB = Attentional Bias; ABS-II = *Attitude and Belief Scale* (DiGiuseppe et al., 1983); VSI = *Visceral Sensitivity Index* (Labus et al., 2004); PCS = *Pain Catastrophizing Scale* (Sullivan et al., 1995); POMS-SV = *Profile of Mood States – Short Version* (Shacham, 1983)

Table 4. *Correlation Matrix*

	Bias	GSQ	Analgesics use	ABS-II: Irrationality	ABS-II: Rationality	VSI	PCS	POMS-SV	VAS pain intensity
Bias	1	$r(19) = -.057$, $p = .816$	$r(21) = .567$, $p = .007$	$r(20) = .300$, $p = .198$	$r(21) = -.209$, $p = .364$	$r(20) = .179$, $p = .449$	$r(21) = .061$, $p = .792$	$r(21) = .062$, $p = .794$	$r(16) = .268$, $p = .316$
GSQ		1	$r(20) = -.171$, $p = .472$	$r(19) = -.192$, $p = 431$	$r(20) = .165$, $p = .487$	$r(19) = .498$, $p = .030$	$r(20) = .555$, $p = .01$	$r(19) = .653$, $p = 0.02$	$r(17) = .368$, $p = .146$
Analgesics use			1	$r(20) = .275$, $p = .241$	$r(21) = -.317$, $p = .161$	$r(20) = .428$, $p = .06$	$r(21) = .042$, $p = .857$	$r(20) = -.118$, $p = .620$	$r(16) = .280$, $p = .293$
ABS-II: Irrationality				1	$r(20) = -.438$, $p = .05$	$r(19) = .255$, $p = .355$	$r(20) = .327$, $p = .159$	$r(19) = .240$, $p = .391$	$r(16) = .130$, $p = .644$
ABS-II: Rationality					1	$r(20) = .066$, $p = .683$	$r(21) = -.237$, $p = .300$	$r(20) = 0$, $p = 1$	$r(16) = -.188$, $p = .486$
VSI						1	$r(20) = .647$, $p = .00$	$r(20) = .442$, $p = .058$	$r(17) = .238$, $p = .359$
PCS							1	$r(20) = .601$, $p = .005$	$r(17) = .396$, $p = .104$
POMS-SV								1	$r(16) = .371$, $p = .130$
VAS pain									1

Note: GSQ = *Gastrointestinal Symptoms Questionnaire* (Bovenshen et al., 2006); AB = Attentional Bias; ABS-II = *Attitude and Belief Scale Scale* (DiGiuseppe et al., 1983); VSI = *Visceral Sensitivity Index* (Labus et al., 2004); PCS = *Pain Catastrophizing Scale* (Sullivan et al., 1995); POMS-SV = *Profile of Mood States – Short Version* (Shacham, 1983); VAS = *Visual Analogue Scale*

The attentional bias was a significant predictor of analgesics use, $F(1, 19) = 5.161$, $p = .035$, $R^2 = .214$, $\beta = .462$.

Discussion

This pilot study was aimed to investigate the role that selective attention to GI symptoms plays in relation with GI symptoms. The increased attention to symptom-related cues does not seem to be associated with increased self-report symptoms, but rather with pain intensity and behaviors aimed to reduce pain/pain-related behavior (i.e., analgesics use). At least two major explanations can account for the obtained results:

(1) The attention bias index as computed here reflected the difficulty to disengage from processing symptom-related information when it competes with neutral information. It is possible that GI patients do not have disengagement difficulties from symptom-related information, but rather hypervigilance/attentional avoidance for such information. Alternatively, it is possible that different components of attentional bias (i.e., hypervigilance, disengagement difficulties, and attentional avoidance; Cisler, Bacon, & Williams, 2009) act conjointly to influence GI symptoms. Future studies should investigate these possibilities.

(2) Attentional bias may be manifested in relation to a more diffuse abdominal *pain*, and not in relation with specific GI symptoms (see also Chapman & Martin, 2011). This possible interpretation is consistent with the positive (yet non-significant[7]) correlation we obtained between AB and pain intensity *and* the lack of AB association with self-report GI symptoms. Indeed, the lack of correlation between attentional bias index and self-report GI symptoms may be due to the fact that GI symptoms varied widely from patient to patient. It is conceivable that a certain patient may not manifest AB toward a symptom she/he did not experience. From this point of view, future studies could benefit from considering an individualized approach of AB assessment in GI patients.

This pilot study is the first that suggests AB is involved in the maintenance of GI symptoms, most probably through a non-specific mechanism (i.e., selective processing of stimuli associated with GI pain/discomfort, rather than selective processing of GI symptoms). Individuals who exhibit AB towards symptom-related cues seem to perceive more pain and report increased analgesics use. Future studies should test the robustness and replicability of these findings, and should clarify the potential causal relationship between AB and GI symptom maintenance. Similarly, future studies should further investigate how AB interacts with other psychological factors involved in the onset, maintenance and coping with gastrointestinal symptoms.

[7] The lack of statistical significance may be a direct consequence of the small sample size

CHAPTER IV

CONCLUSIONS AND IMPLICATIONS

The present project aimed to investigate the role of cognitive biases in emotional and psychosomatic (i.e., gastrointestinal disorders) within a cognitive-behavioral therapy framework. We aimed to bring back the experimental research line of cognitive bias (modification) within the cognitive behavioral framework and to investigate the cognitive biases importance against self-report cognitive factors that sustain psychopathology. To reach this goal, we oriented our efforts in two main directions: emotional disorders, and psychosomatic (i.e., gastrointestinal) disorders. Several theoretical and conceptual advances along with some methodological innovations deserve mentioning here.

At the end of our research work, the following main conclusions may be pointed out:

1. Evidence of the cognitive bias modification procedures' clinical efficacy is currently mixed. Cognitive bias modification (CBM) procedures do not seem to be powerful enough to have a significant clinical impact in emotional disorders. In addition, they do not seem to have any impact on the conscious cognitive factors known to be involved in the onset and maintenance of emotional disorders. Although this latter aspect is not an issue in itself, as cognitive biases can manifest at an implicit level of cognition, in order to assume that CBM procedures modifies implicit biases, these interventions should be proven to have a notable and replicable clinical effect. Therefore, it is premature to derive CBM as a standalone treatment option, even solely for self-help.
2. There is some evidence that attention bias modification training works in anxiety, having a small effect size. However, more and more recent studies challenge the initial findings in such a way that it becomes unclear to which extent we could speak about the clinical effect of attentional bias modification (ABM) intervention.
3. Mechanisms of change in attention bias modification remain largely unknown. This is at least partially responsible to the fail of reliably replicate ABM effect.
4. Attention bias assessment task seem to be highly unreliable. This could be due to the task particularities, to the nature of the measured phenomenon, or to a combination of these two factors. Currently it is unclear if attentional bias is a trait or a state characteristic. Moreover, we do not have clearly articulated theoretical models that integrate the data related to different aspects/components of the attentional bias. The available attentional bias assessment can hardly discriminate between different attentional bias components. Similarly, the variables that modulate attentional bias are not well established. We need a more sound understanding of the attentional bias notion, in order to design better assessment instruments. As long as we do not have psychometricaly sound instruments to reliable measure attentional bias, the attempt to replicate ABM effect resembles rather a lottery game than a scientifically sound approach.
5. Given the unreliability of attentional bias assessment task, it is questionable why all the available training procedures built on them. We need more powerful and psychometric sound procedures to modify attentional biases.
6. Cognitive bias modification research line should be informed by the classical cognitive-behavioral intervention protocols, as the late are the state of the art of evidence-based psychological interventions. As cognitive bias modification procedures aimed to be used as clinical tools, the *clinical significance* of their effect should be always considered along with their statistical significance and effect size.

Limitations and future directions

The research presented in this thesis has clear limitations. We discussed specific limitations related to every study in the Discussion section of that study. However, there are several general limitations that deserve to be mentioned here.

First of all, our samples included mainly non-clinical participants (except for the last study). Even when we recruited sub-clinical participants (see Study 3), we did not check their diagnostic status. This could have negatively influenced our results, as we have reasons to believe that the effect of cognitive bias modification procedures might be weaker and consequently more difficult to evidence in nonclinical samples.

Second, our participants were mainly women. Although we have no reasons to believe that cognitive biases can manifest differently in men and women, our findings should be carefully interpreted having in mind that they were derived mainly from women samples. Future studies should specifically investigate any eventual gender effect in relationship with cognitive biases.

Third, at least two of our studies (Study 3 and Study 6) clearly suffer from low statistical power. Therefore, our results should be replicated in adequately powered samples before drawing any definitive conclusions.

Fourth, we used only self-report instruments in order to assess symptoms. Future studies should consider other complementary measures (e.g., behavioral, clinician-rated) in order to strengthen our conclusions.

Despite its inherent limitations, we believe that the present research provided answers for some important questions regarding the clinical relevance of cognitive biases, as well as the clinical utility of modifying them through means of cognitive bias modification procedures. Our results did not indicate a notable clinical effect of the cognitive bias modification interventions. However, they raised several interesting future direction for subsequent research. We pointed out in our Discussion section, for specific studies, possible explanations of our findings that could inform future studies. We hope that this research stimulates dialogue and further research leading to a better understanding of cognitive biases in emotional and psychosomatic disorders, as well as to the improvement of the current inventory of intervention tools and clinical protocols.

SELECTIVE REFERENCES

Afzal, M., Potokar, J. P., Probert, C. S. J., & Munafò, M. R. (2006). Selective processing of gastrointestinal symptom-related stimuli in irritable bowel syndrome. *Psychosomatic Medicine*, *68*(5), 758–761. doi:10.1097/01.psy.0000232270.78071.28

Alonso, J., Angermeyer, M. C., Bernert, S., Bruffaerts, R., Brugha, T. S., Bryson, H., … Vollebergh, W. A. M. (2004). Prevalence of mental disorders in Europe: results from the European Study of the Epidemiology of Mental Disorders (ESEMeD) project. *Acta Psychiatrica Scandinavica. Supplementum*, (420), 21–27. doi:10.1111/j.1600-0047.2004.00327.x

Amir, N., Beard, C., Burns, M., & Bomyea, J. (2009). Attention modification program in individuals with generalized anxiety disorder. *Journal of Abnormal Psychology*, *118*(1), 28–33. doi:10.1037/a0012589

Amir, N., Beard, C., Taylor, C. T., Klumpp, H., Elias, J., Burns, M., & Chen, X. (2009). Attention Training in Individuals with Generalized Social Phobia: A Randomized Controlled Trial. *Journal of Consulting and Clinical Psychology*, *77*(5), 961–973. doi:10.1037/a0016685

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. International Universities Press.

Beck, A.T., & Clark, D. A. (1997). An information processing model of anxiety: Automatic and strategic processes. *Behaviour Research and Therapy, 35*(1), 49-58. doi:10.1016/S0005-7967(96)00069-1

Blanchard, E. B. (2005). A Critical Review of Cognitive, Behavioral, and Cognitive-Behavioral Therapies for Irritable Bowel Syndrome. *Journal of Cognitive Psychotherapy*, *19*(2), 101–123. doi:10.1891/jcop.19.2.101.66787

Boden, M. T., John, O. P., Goldin, P. R., Werner, K., Heimberg, R. G., & Gross, J. J. (2012). The role of maladaptive beliefs in cognitive-behavioral therapy: Evidence from social anxiety disorder. *Behaviour Research and Therapy*, *50*(5), 287–291. doi:10.1016/j.brat.2012.02.007

Boettcher, J., Berger, T., & Renneberg, B. (2012). Internet-Based Attention Training for Social Anxiety: A Randomized Controlled Trial. *Cognitive Therapy and Research*, 1–15. doi:10.1007/s10608-011-9374-y

Borenstein, M., Hedges, L., Higgins, J., & Rothstein, H. (2005). *Comprehensive meta-analysis, version 2*. Englewood, NJ: Biostat, Inc.

Bothwell, R. (2003). Cognitive behaviour therapy for psychosomatic disorders. *Continuing Medical Education*, *21*(3). Retrieved from http://www.ajol.info/index.php/cme/article/view/43884

Carlbring, P., Apelstrand, M., Sehlin, H., Amir, N., Rousseau, A., Hofmann, S. G., & Andersson, G. (2012). Internet-delivered attention bias modification training in individuals with social anxiety disorder - a double blind randomized controlled trial. *BMC Psychiatry*, *12*(1), 66. doi:10.1186/1471-244X-12-66

Carver, C.S., & Ganellen, R.J. (1983). Depression and elements of self-punitiveness: High standards, self criticism, and overgeneralization. *Journal of Abnormal Psychology, 92*(3), 330-337. doi: 10.1037/0021-843X.92.3.330

Cash, B., Sullivan, S., & Barghout, V. (2005). Total costs of IBS: employer and managed care perspective. *The American Journal of Managed Care*, *11*(1 Suppl), S7–16.

Chapman, S., & Martin, M. (2011). Attention to pain words in irritable bowel syndrome: increased orienting and speeded engagement. *British Journal of Health Psychology*, *16*(Pt 1), 47–60. doi:10.1348/135910710X505887

Cisler, J. M., & Koster, E. H. W. (2010). Mechanisms of attentional biases towards threat in anxiety disorders: An integrative review. *Clinical Psychology Review*, *30*(2), 203–216. doi:10.1016/j.cpr.2009.11.003

Connor, K. M., Davidson, J. R., Churchill, L. E., Sherwood, A., Foa, E., & Weisler, R. H. (2000). Psychometric properties of the Social Phobia Inventory (SPIN). New self-rating scale. *The British Journal of Psychiatry: The Journal of Mental Science*, *176*, 379–386.

Craske, M. G., Wolitzky-Taylor, K. B., Labus, J., Wu, S., Frese, M., Mayer, E. A., & Naliboff, B. D. (2011). A cognitive-behavioral treatment for irritable bowel syndrome using interoceptive exposure to visceral sensations. *Behaviour Research and Therapy*, *49*(6-7), 413–421. doi:10.1016/j.brat.2011.04.001

David, D., Lynn, S. J., & Ellis, A. (2009). *Rational and Irrational Beliefs:Research, Theory, and Clinical Practice: Research, Theory, and Clinical Practice*. Oxford University Press.

Derakshan, N., & Eysenck, M. W. (2009). Anxiety, Processing Efficiency, and Cognitive Performance. *European Psychologist*, *14*(2), 168–176. doi:10.1027/1016-9040.14.2.168

Drossman, D., Creed, F., Olden, K., Svedlund, J., Toner, B., & Whitehead, W. (1999). Psychosocial aspects of the functional gastrointestinal disorders. *Gut*, *45*(Suppl 2), II25–II30.

Duval, S., & Tweedie, R. (2000). Trim and Fill: A Simple Funnel-Plot–Based Method of Testing and Adjusting for Publication Bias in Meta-Analysis. *Biometrics*, *56*(2), 455–463. doi:10.1111/j.0006-341X.2000.00455.x

Ellis, A. (1962). *Reason and Emotion in Psychotherapy*. New York, NY: Lyle Stuart.

Ellis, A. (1994). *Reason and emotion in psychotherapy*. Carol Pub. Group.

Everaert, J., Koster, E. H. W., & Derakshan, N. (2012). The combined cognitive bias hypothesis in depression. *Clinical Psychology Review*, *32*(5), 413–424. doi:10.1016/j.cpr.2012.04.003

Eysenck, M. W., & Derakshan, N. (2011). New perspectives in attentional control theory. *Personality and Individual Differences*, *50*(7), 955–960. doi:10.1016/j.paid.2010.08.019

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: attentional control theory. *Emotion (Washington, D.C.)*, *7*(2), 336–353. doi:10.1037/1528-3542.7.2.336

Eysenck, M. W., & Keane, M. T. (2010). *Cognitive Psychology: A Student's Handbook, 6th Edition* (6th ed.). Psychology Press.

Fehm, L., Pelissolo, A., Furmark, T., & Wittchen, H.-U. (2005). Size and burden of social phobia in Europe. *European Neuro-psychopharmacology: The Journal of the European College of Neuro-psychopharmacology*, *15*(4), 453-462. doi:10.1016/j.euroneuro.2005.04.002

Gomborone, J. E., Dewsnap, P. A., Libby, G. W., & Farthing, M. J. (1993). Selective affective biasing in recognition memory in the irritable bowel syndrome. *Gut*, *34*(9), 1230–1233.

Hallion, L. S., & Ruscio, A. M. (2011). A meta-analysis of the effect of cognitive bias modification on anxiety and depression. *Psychological Bulletin*, *137*(6), 940-958. doi:10.1037/a0024355

Heeren, A., Koster, E. H. W., & Philippot, P. (2013). The (neuro)cognitive mechanisms behind attention bias modification in anxiety: proposals based on theoretical accounts of attentional bias. *Frontiers in Human Neuroscience*, *7*, 119. doi:10.3389/fnhum.2013.00119

Heeren, A., Lievens, L., & Philippot, P. (2011). How does attention training work in social phobia: Disengagement from threat or re-engagement to non-threat? *Journal of Anxiety Disorders*, *25*(8), 1108–1115. doi:10.1016/j.janxdis.2011.08.001

Heeren, A., Reese, H. E., McNally, R. J., & Philippot, P. (2012). Attention training toward and away from threat in social phobia: effects on subjective, behavioral, and physiological measures of anxiety. *Behaviour Research and Therapy*, *50*(1), 30–39. doi:10.1016/j.brat.2011.10.005

Johnston, K., Westerfield, W., Momin, S., Phillippi, R., & Naidoo, A. (2009). The direct and indirect costs of employee depression, anxiety, and emotional disorders--an employer case study. *Journal of Occupational and Environmental Medicine / American College of Occupational and Environmental Medicine*, *51*(5), 564–577. doi:10.1097/JOM.0b013e3181a1f5c8

Julian, K., Beard, C., Schmidt, N. B., Powers, M. B., & Smits, J. A. J. (2012). Attention training to reduce attention bias and social stressor reactivity: an attempt to replicate and extend previous findings. *Behaviour Research and Therapy*, *50*(5), 350–358. doi:10.1016/j.brat.2012.02.015

Klumpp, H., & Amir, N. (2010). Preliminary Study of Attention Training to Threat and Neutral Faces on Anxious Reactivity to a Social Stressor in Social Anxiety. *Cognitive Therapy and Research*, *34*(3), 263–271. doi:10.1007/s10608-009-9251-0

Levy, R. L., Olden, K. W., Naliboff, B. D., Bradley, L. A., Francisconi, C., Drossman, D., & Creed, F. (2006). Psychosocial aspects of the functional gastrointestinal disorders. *Gastroenterology*, *130*, 1447–1458. doi:10.1053/j.gastro.2005.11.057

Li, S., Tan, J., Qian, M., & Liu, X. (2008). Continual training of attentional bias in social anxiety. *Behaviour Research and Therapy*, *46*(8), 905–912. doi:10.1016/j.brat.2008.04.005

MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective attention and emotional vulnerability: assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of Abnormal Psychology*, *111*(1), 107–123.

Mahaffey, B. L., Wheaton, M. G., Fabricant, L. E., Berman, N. C., & Abramowitz, J. S. (2013). The contribution of experiential avoidance and social cognitions in the prediction of social anxiety. *Behavioural and Cognitive Psychotherapy*, *41*(1), 52–65. doi:10.1017/S1352465812000367

Malouff, J. M., & Schutte, N. S. (1986). Development and Validation of a Measure of Irrational Belief. *Journal of Consulting and Clinical Psychology*, *54*(6), 860-62.

Martin, M., & Chapman, S. C. E. (2010). Cognitive processing in putative functional gastrointestinal disorder: rumination yields orientation to social threat not pain. *European Journal of Pain*, *14*(2), 207–213. doi:10.1016/j.ejpain.2009.04.008

Mathews, A. (2006). Towards an experimental cognitive science of CBT. *Behavior Therapy*, *37*(3), 314–318. doi:10.1016/j.beth.2006.02.004

Matsumoto, D., & Ekman, P. (1989). *The Japanese and Caucasioan Facial Expressions of Emotions (JACFEE) and Neutrals (JACNeuF)*. San Francisco, CA: Intercultural and Emotion Research Laboratory, Department of Psychology, San Francisco State University.

McGough, J. J., & Faraone, S. V. (2009). Estimating the size of treatment effects: moving beyond p values. *Psychiatry*, *6*(10), 21–29.

Neubauer, K., von Auer, M., Murray, E., Petermann, F., Helbig-Lang, S., & Gerlach, A. L. (2013). Internet-delivered attention modification training as a treatment for social phobia: a randomized controlled trial. *Behaviour Research and Therapy*, *51*(2), 87–97. doi:10.1016/j.brat.2012.10.006

Posserud, I., Svedlund, J., Wallin, J., & Simrén, M. (2009). Hypervigilance in irritable bowel syndrome compared with organic gastrointestinal disease. *Journal of Psychosomatic Research*, *66*(5), 399–405. doi:10.1016/j.jpsychores.2008.09.020

Rosenthal, R. (1991). *Meta-Analytic Procedures for Social Research*. SAGE.

Schmidt, N. B., Richey, J. A., Buckner, J. D., & Timpano, K. R. (2009). Attention training for generalized social anxiety disorder. *Journal of Abnormal Psychology*, *118*(1), 5–14. doi:10.1037/a0013643

Sumner, J.A., Griffith, J.W., & Mineka, S. (2010). Overgeneral autobiographical memory as a predictor of the course of depression: a meta-analysis. *Behavior Research and Therapy, 48*(7), 614-625. doi: 10.1016/j.brat.2010.03.013

Talley, N. J. (2008). Functional gastrointestinal disorders as a public health problem. *Neurogastroenterology and Motility: The Official Journal of the European Gastrointestinal Motility Society*, *20 Suppl 1*, 121–129. doi:10.1111/j.1365-2982.2008.01097.x

Tottenham, N, Borsheid, A., Ellersten, K., Marcus, D. J., & Nelson, C. (2002). In *Categorization of facial expressions in children and adults. Establishing a larger stimlus set* (Vol. Journal of Cognitive Neuroscience, pp. 11-74). Presented at the Annual Meeting of the Cognitive Neuroscience Society, San Francisco, CA.

Tottenham, Nim, Tanaka, J. W., Leon, A. C., McCarry, T., Nurse, M., Hare, T. A., … Nelson, C. (2009). The NimStim set of facial expressions: judgments from untrained research. *Psychiatry Research*, 168(3), 242-249. doi: 10.1016/j.psychres.2008.05.006

Trope, Y., & Liberman, N. (2003). Temporal construal. *Psychological Review, 110*(3), 403-421. doi: 10.1037/0033-295X.110.3.403

Van Bockstaele, B., Koster, E. H. W., Verschuere, B., Crombez, G., & De Houwer, J. (2012). Limited transfer of threat bias following attentional retraining. *Journal of Behavior Therapy and Experimental Psychiatry, 43*(2), 794–800. doi:10.1016/j.jbtep.2011.11.001

Watkins, E.R., Baeyens, C.B., & Read, R. (2009). Concreteness training reduces dysphoria: proof-of-principle for repeated cognitive bias modification in depression. *Journal of Abnormal Psychology, 118*(1), 55-64. doi: 10.1037/a0013642

Watkins, E.R., & Moberly, N.J. (2009). Concreteness training reduces dysphoria: a pilot proof-of-principle study. *Behavior Research and Therapy, 47*(1), 48-53. doi: 10.1016/j.brat.2008.10.014

Watkins, E.R., Moberly, N.J., & Moulds, M.L. (2008). Processing mode causally influences emotional reactivity: distinct effects of abstract versus concrete construal of emotional response. *Emotion, 8*(3), 364-378. doi: 10.1037/1528-3542-8.3.364

Williams, J. M. G., Barnhofer, T., Crane, C., Hermans, D., Raes, F., Watkins, E., & Dalgleish, T. (2007). Autobiographical Memory Specificity and Emotional Disorder. *Psychological Bulletin, 133*(1), 122-148. doi:10.1037/0033-2909.133.1.122

Williams, J.M.G., & Broadbent, K. (1986). Autobiographical memory in suicide attempters. *Journal of Abnormal Psychology, 95*(2), 144-149. doi: 10.1037/0021-843X.95.2.144

Wittchen, H. U., Jacobi, F., Rehm, J., Gustavsson, A., Svensson, M., Jönsson, B., … Steinhausen, H.-C. (2011). The size and burden of mental disorders and other disorders of the brain in Europe 2010. *European Neuropsychopharmacology: The Journal of the European College of Neuropsychopharmacology*, *21*(9), 655–679. doi:10.1016/j.euroneuro.2011.07.018

Wu, J. C. (2012). Psychological Co-morbidity in Functional Gastrointestinal Disorders: Epidemiology, Mechanisms and Management. *Journal of Neurogastroenterology and Motility*, *18*(1), 13–18. doi:10.5056/jnm.2012.18.1.13