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The Potential Role of Learning Capacity in Cognitive Behavior Therapy for Depression: A Systematic Review of the Evidence and Future Directions for Improving Therapeutic Learning

Sanne J. E. Bruijniks[1,2], Robert J. DeRubeis[3], Steven D. Hollon[4], and Marcus J. H. Huibers[1,2,3]

[1]Department of Clinical Psychology, Vrije Universiteit Amsterdam; [2]Amsterdam Public Health Research Institute, Vrije Universiteit Amsterdam; [3]Department of Psychology, University of Pennsylvania; and [4]Department of Psychology, Vanderbilt University

Abstract

Insight into how cognitive behavioral therapy (CBT) works is urgently needed to improve depressive outcome. First, we discuss the role of learning in CBT for depression by reviewing evidence for learning processes involved in the development and maintenance of depression. Second, we investigate the role of learning capacity as a moderator by reviewing empirical evidence for the relation between (a) CBT procedures and learning processes, (b) learning processes and CBT treatment processes, and (c) learning processes and CBT outcome. We propose that learning capacity moderates the relation between CBT procedures and change in CBT treatment processes and explains why therapeutic procedures lead to process change and long-term success in some but not all patients. Third, we identify procedures that lead to successful therapeutic learning and describe how experimental studies help to better explain causal mechanisms of change and the role of learning capacity in CBT for depression.

Keywords

depression, cognitive behavioral therapy, mechanisms of change, learning, process, therapeutic procedure

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Cognitive behavioral therapy (CBT) is one of the most effective and best tested treatments for depression (Beck, Rush, Shaw, & Emery, 1979; Cuijpers, Berking, et al., 2013), but its exact working mechanisms are unknown. Previous studies have focused on specific (i.e., cognitive change, behavioral activation, and cognitive skill acquisition; Lorenzo-Luaces, German, & DeRubeis, 2015; Manos, Kanter, & Busch, 2010; Strunk, DeRubeis, Chiu, & Alvarez, 2007) and nonspecific (i.e., therapeutic alliance; Huibers & Cuijpers, 2015) processes that might play a role in the effects of CBT, but the findings are mixed. Despite decades of mechanism research, it remains largely unclear which processes or factors are responsible for the reduction of depression or how the different processes might be related to each other. Insight into how CBT works is urgently needed to improve depressive treatment outcome because only about half of all patients respond to CBT (i.e., with response defined as a 50% reduction in symptoms) and at least a quarter of the patients who respond to treatment relapse within a year (i.e., a return of symptoms associated with the treated episode; Cuijpers et al., 2014; Lemmens et al., 2019). These rates are as good (with respect to acute response) or better (with respect to the prevention of subsequent relapse or recurrence) as found for medication but still fall short of what would be desired because less than half of all patients respond to CBT and stay free from relapse (Dobson et al., 2008).

Corresponding Author:
Sanne Bruijniks, Vrije Universiteit Amsterdam, Department of Clinical Psychology, van der Boechorststraat 7, 1081BT Amsterdam
E-mail: s.j.e.bruijniks@vu.nl

Recent studies have focused on the role of memory and learning as essential ingredients for therapeutic success and tried to identify the processes that might benefit or harm learning (Ecker, Ticic, & Hulley, 2012; Harvey et al., 2014; Lane, Ryan, Nadel, & Greenberg, 2015). Moreover, neurobiological processes that are related to learning have been related to the success of psychological treatments (Brunoni, Lopes, & Fregni, 2008; Shimizu et al., 2003; Tadić et al., 2011). The role of learning and memory in CBT is eminently sensible because CBT is a skills-based approach, and unlike more traditional approaches to psychotherapy, those skills must be acquired if they are to work. However, the exact role of learning in relation to CBT is unclear. In the present article, we review evidence for the role of CBT's most researched procedures and processes and investigate the potential role of learning in CBT for depression. This review is not intended to be exhaustive. Rather, its aim is to integrate theory and the relevant empirical literature relating to CBT and learning to advance research on CBT for depression.

First, we will review what cognitive and neurobiological processes are disrupted in depressed patients that might affect learning during treatment. We will refer to these processes as *learning processes* and give an overview of their role in the development and maintenance of depression. Subsequently, we will distinguish between therapeutic procedures and treatment processes and suggest that together they form the causal mechanisms of change of a certain treatment. Because earlier studies have defined learning as "the process by which changes in behavior arise as a result of experiences interacting with the world" (Harvey, Watkins, Mansell, & Shafran, 2004, p. 163), learning in CBT will be defined as the process that may lead to changes in behavior during CBT procedures for depression. We will review CBT's most investigated mechanisms of change by discussing evidence for the potential effects of CBT procedures on change in CBT's hypothesized treatment processes.

Second, we will introduce the concept of learning capacity as the extent to what learning processes are present and affect the success of learning in a particular individual (thus, the less disturbed the learning processes, the better the individual's capacity to learn). We will review how limits to learning processes might interfere with the effect of CBT procedures for depression on their target processes and subsequent outcome by conducting a systematic review on the relation between: (a) CBT procedures and learning processes, (b) learning processes and CBT processes, and (c) learning processes and CBT outcome.

Third, we will propose an integrated CBT mechanism model for depression that suggests that learning capacity moderates the success of CBT procedures in bringing about change in CBT treatment processes. In other words, how much is learned (i.e., the degree to what the procedure is successful in bringing change in the process) by the patient will depend on his or her capacity to learn. Although CBT procedures can result in reduced depression on average (outcome), it does not explain how those therapeutic procedures lead to process change and (long-term) treatment success in some patients but not others. Individual differences in learning capacity might explain why one patient responds to or relapses after therapy and another does not (moderation).

Fourth, we identify CBT procedures that may improve or lead to successful therapeutic learning and describe how current findings can direct future research. In addition, we describe how experimental studies and upcoming technologies might help increase insight into the causal mechanisms underlying change in CBT and its moderation by learning capacity. Hopefully, these recommendations will help researchers explore new avenues of research that improve initial treatment response and reduce relapse rates.

Learning in CBT for Depression

Learning processes in the development and maintenance of depression

Depression is related to numerous impairments in cognitive and neurobiological processes that have been related to learning (Disner, Beevers, Haigh, & Beck, 2011; Harvey et al., 2004; McClintock, Husain, Greer, & Cullum, 2010). Compared with healthy individuals, depressed patients show impairments in executive functioning (Beevers, 2005; Snyder, 2013), suffer from repetitively negative thinking focused on symptoms or meanings of distress (i.e., also referred to as rumination; Nolen-Hoeksema, 2000), and show attentional and memory biases toward negative information (Dalgleish & Yiend, 2006; Elliott, Zahn, Deakin, & Anderson, 2011; Ellis, Wells, Vanderlind, & Beevers, 2014; Fu et al., 2008; Harvey et al., 2004; Joormann & Gotlib, 2008). Depressed patients have reported an overgeneral autobiographical memory (Köhler et al., 2015; Sumner, Griffith, & Mineka, 2010; Sutherland & Bryant, 2007) and deficits in recollecting memories (MacQueen, Galway, Hay, Young, & Joffe, 2002) and suffer from intrusive memories of traumatic or negative interpersonal events (Harvey et al., 2004; Reynolds & Brewin, 1999; Williams & Moulds, 2007). Moreover, dysfunctional thinking has been related to deficits in reflective processing (i.e., also known as rule-based, explicit, controlled, conscious, and rational processing) and the impaired correction of biases in associative processing (i.e., quick and effortless information processing on a preconscious level). It has been suggested that associative processing is the default mode of thinking and that reflective

processing can be used to change biases in associative thinking (Beevers, 2005). Besides impairments in cognitive processes, studies have focused on impairments in neurobiological processes and related them to impairments in cognitive processes. For example, cognitive biases have been related to hyperactivity in the amygdala and reduced activity in the dorsolateral prefrontal cortex (DLPFC; Disner et al., 2011), whereas stress and depression have been linked to decreased neurogenesis (Brunoni et al., 2008; Duman, 2004; Fuchikami et al., 2011; Gersner, Gal, Levit, Moshe, & Zangen, 2014; Groves, 2007; Karege et al., 2002); specific pathways of neurotransmitters (Nutt, 2008; Werner & Coveñas, 2010); reduced gray matter in the brain, including loss of hippocampal volume, an area that is critically involved in learning (Deng, Aimone, & Gage, 2010; Duman, 2004; Lener & Losifescu, 2015; McKinnon, Yucel, Nazarov, & MacQueen, 2008); and abnormal brain activity patterns (Lener & Losifescu, 2015). Other studies reviewed the role of different brain structures and functionalities as potential biomarkers that predict treatment outcome, but further research is yet warranted (Lener & Losifescu, 2015).

Several models suggested that cognitive and neurobiological impairments are more than concurrent with depression and suggest that these processes may be diatheses that lead to the onset or maintenance of depressive symptoms. For example, reviews have suggested that impairments in cognitive and neurobiological processes can result in a negative feedback loop that allows the process of dysfunctional thinking to persist (Banich et al., 2010; Beevers, 2005). More specifically, disturbances in neurobiological processes may lead to cognitive errors and impairments in executive functioning. Subsequently, impairments in executive functioning may lead to a limited control of emotional responses and an impaired ability to correct biases in associative thinking, regulate behavior toward rewarding experiences, or distract from negative information as appropriate, leading to worsened mood (Banich et al., 2010; Beevers, 2005; Gotlib & Joormann, 2010; Harvey et al., 2004, Chapter 3; Heatherton & Wagner, 2011). Cognitive errors might also limit a person's access to the underlying specific memories, thereby preventing change of unhelpful beliefs (Harvey et al., 2004, Chapter 3), and longitudinal studies have indicated that deficits in cognitive functioning might interact with stressful life events to predict the onset of depression (Hankin, Abramson, Miller, & Haeffel, 2004; Robinson & Alloy, 2003; Scher, Ingram, & Segal, 2005). For example, early life stressors have been related to disturbances in brain development that in turn increase risk for depression later in life (Levin, Heller, Mohanty, Herrington, & Miller, 2007). Disner and colleagues (2011) integrated neural mechanisms into Beck's cognitive model of depression and suggested that, in general, negative cognitive biases in depression are facilitated by increased influence from subcortical emotion processing regions combined with attenuated top-down cognitive control in the prefrontal areas. Moreover, as the major goal of CBT is to target cognitive errors or biases, this may result in not only cognitive change but also subsequent reduced subcortical activity and enhanced cognitive control and activity in prefrontal areas (Disner et al., 2011). However, although these studies pointed to the role of cognitive or neurobiological processes in (the cognitive model of) depression, it is not yet clear how these processes directly relate to CBT for depression.

An overview of relevant literature on the role of learning processes in depression is given in Table 1.

The potential causal mechanisms of change during CBT for depression

Because studies have defined learning as "the process by which changes in behavior arise as a result of experiences interacting with the world" (Harvey et al., 2004, p. 163), the present article will define learning in CBT as the process that leads to stable changes in behavior during CBT for depression. Moreover, we propose that to advance our understanding of the underlying causal processes involved in CBT, a distinction should be made between therapeutic procedures and therapeutic processes. Whereas therapeutic procedures can be seen best as those techniques delivered by the therapist with the intent of producing change, treatment processes can be described as the mechanisms inside (the mind of) the patient that are mobilized by those procedures. A therapy might include different therapeutic procedures that are targeted at different treatment processes. We propose that to the degree that a therapeutic procedure is successful in changing the targeted process (i.e., the treatment process) and the extent to which change in that process results in subsequent change in the outcome, the procedure and process form a chain that can be seen as the causal mechanisms to change (see Fig. 1). Insight into the causal mechanisms of change of CBT will provide information about which treatment processes are changed as a result of CBT procedures to eventually change behavior and reduce depression. Note that a certain process can be a treatment process and a learning process at the same time, such as is the case in, for example, cognitive bias modification interventions (therapeutic procedure) that directly target change of attention to negative threat stimuli (i.e., the treatment process; MacLeod & Grafton, 2016). We will now review evidence for CBT's most often investigated potential causal mechanisms of change.

Cognitive change. Cognitive change is CBT's most investigated potential mechanism of change. Cognitive change

Table 1. Relevant Reviews on the Role of Cognitive and Neurobiological Processes in the Onset and Maintenance of Depression

Process	Relevant articles
Cognitive impairments	
Executive functioning	Beevers (2005)
Inhibition	Snyder (2013)
Attention	Harvey, Watkins, Mansell, and Shafran (2004)
Planning	Gotlib and Joormann (2010)
Working memory	McClintock, Husain, Greer, and Cullum (2010)
Shifting/updating	Elliott, Zahn, Deakin, and Anderson (2011)
Processing speed	Scher, Ingram, and Segal (2005)
Memory	Harvey et al. (2004)
	Köhler et al. (2015)
	Sumner, Griffith, and Mineka (2010)
Neurobiological impairments	
Brain activity	Disner, Beevers, Haigh, and Beck (2011)
Brain structure	Levin, Heller, Mohanty, Herrington, and Miller (2007)
Neurotransmission	McKinnon, Yucel, Nazarov, and MacQueen (2008)
Neurogenesis	Werner and Coveñas (2010)
	Nutt (2008)
	Groves (2007)
	Duman (2004)
	Brunoni, Lopes, and Fregni (2008)

can be distinguished into change in dysfunctional thinking (i.e., treatment process) and cognitive change work (i.e., the cognitive therapy procedure in CBT). Change in dysfunctional thinking has been defined as changes in automatic negative thoughts, dysfunctional attitudes, and core beliefs as well as the larger information-processing schemas that determine how an individual processes incoming information and retrieves memories and mostly has been measured with the use of explicit self-report questionnaires (Garratt, Ingram, Rand, & Sawalani, 2007). There is ample evidence to suggest that dysfunctional thinking is involved in the development and maintenance of depression (Dozois et al., 2009; Scher et al., 2005; Segal et al., 2006; Sheppard & Teasdale, 2000, 2004). During cognitive change work, the therapist and patient work together to change the pattern of dysfunctional thinking by evaluating and targeting these negative beliefs. Note that cognitive change work can be distinguished from other third-wave CBT therapeutic procedures that do not primarily aim to change the content of dysfunctional beliefs (i.e., cognitive change) but focus on changing the function of dysfunctional thinking by targeting treatment processes such as rumination (i.e., during rumination-based CBT; Hvenegaard et al., 2015) or self-compassion (i.e., during mindfulness-based CBT; Kuyken et al., 2010). Numerous studies have shown that change in dysfunctional thinking is associated with a reduction of depressive symptoms during procedures focused on cognitive change work (Garratt et al., 2007; Lemmens et al., 2017) and that the extent to which dysfunctional beliefs can be activated in formerly depressed patients predicts subsequent relapse after CBT (Segal et al., 2006). However, the causal role of cognitive change in CBT procedures can still be questioned (Lorenzo-Luaces et al., 2015), and although the field of depression already includes a few studies that did disentangle treatment packages (e.g., cognitive therapy vs. behavioral activation; Dimidjian et al., 2006), to our knowledge, only one experimental study so far has isolated a CBT procedure to investigate its direct effects on a CBT process and subsequent outcome (Teasdale & Fennell, 1982). In this study, it was shown that isolated cognitive change work led to direct improvements in idiosyncratic dysfunctional beliefs and mood, although a procedure focused on

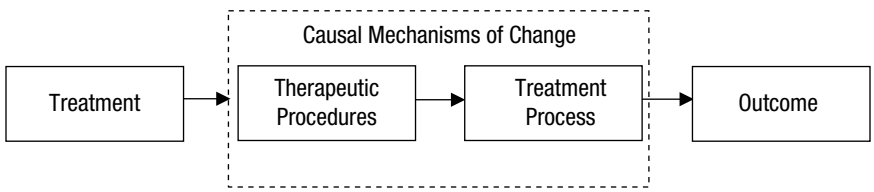

Fig. 1. Causal pathway hypothesis: the relation between treatment, therapeutic procedures, treatment processes, and outcome.

exploring but not changing these dysfunctional beliefs did not, hereby providing preliminary evidence for a causal pathway between cognitive change work and change in dysfunctional beliefs.

Besides limited knowledge on the causal relationship between CBT's procedures and CBT treatment processes, it remains unclear whether change in dysfunctional thinking is an actual change in the dysfunctional structures (accommodation model) or the deactivation of dysfunctional structures and activation of more functional structures that lead to less dysfunctional thinking (deactivation–activation model; Barber & DeRubeis, 1989; Brewin, 1989, 2006). In favor of the (de)activation hypothesis (Brewin, 1989, 2006), studies have shown that former depressed patients have a certain cognitive vulnerability (Segal et al., 2006; Segal, Gemar, & Williams, 1999) that might predict the onset of a (recurrent) depressive episode (Alloy et al., 1999) and have suggested that CBT may reduce relapse by changing relationships to negative thoughts rather than changing beliefs in thought content (Teasdale et al., 2002). Moreover, it was suggested that relapse would be hard to explain if the old dysfunctional structures have been permanently changed (Brewin, 1989). These findings seem to suggest that CBT procedures do not lead to the alteration but merely to the deactivation of dysfunctional thinking.

Can CBT procedures also lead to the change of dysfunctional structures (i.e., following the accommodation model)? Although old cognitive theories suggested that memories become permanent, more recent studies have shown that it might be possible to modify memory in both animals and healthy individuals (Nader & Einarsson, 2010; Nader, Schafe, & LeDoux, 2000; Tronson & Tayler, 2007). More specifically, when a memory is retrieved, it enters into a fragile and labile state and requires another consolidation period, referred to as *reconsolidation*, that enables change or disruption of memory (Nader et al., 2000). These findings were translated to the suggestion that change or disruption of emotional memories might be a way to increase treatment success for patients suffering from various mental illnesses, such as posttraumatic stress disorder (PTSD), negative autobiographical memories, dysfunctional thinking, compulsive drinking, or psychotic symptoms (Ecker et al., 2012; Köhler et al., 2015; Lane et al., 2015). Experimental studies that investigate whether disrupting reconsolidation can be helpful in the treatment of psychological disorders have been focusing mostly on the disrupting of fear memory. More specifically, it was shown that a procedure consisting of the combination of reactivating the original fear memory and providing a mismatch with the original fear memory (i.e., something to be learned, also called a *prediction error*), combined with administering a protein synthesis inhibitor, might disrupt reconsolidation (i.e., the process in which the original, reactivated memory is "resaved") and prevents the return of fear memory, compared with procedures that focused on the deactivation of emotional memories (i.e., extinction; Brunet et al., 2008; Kindt, Soeter, & Vervliet, 2009; Sevenster, Beckers, & Kindt, 2012). In addition, noninvasive procedures that do not disrupt the reconsolidation itself but utilize reconsolidation to incorporate information that contradicts the original memory were developed, and several studies already suggested that noninvasive procedures after retrieval of the memory may also lead to successful change of emotional memory (Björkstrand et al., 2016; Golkar, Tjaden, & Kindt, 2017; Schiller et al., 2010). Other experiments have shown that not only fear memory but also other forms of memory (e.g., declarative, episodic, or spatial memory) can be changed via use of reconsolidation. For example, experiments have shown that reconsolidation procedures can disrupt learned associations of word pairs (Forcato, Argibay, Pedreira, & Maldonado, 2009; Forcato et al., 2007; Forcato, Rodríguez, Pedreira, & Maldonado, 2010) or even memory for personal experiences (Schwabe & Wolf, 2009).

In relation to CBT, one of the goals of cognitive change work is to create a mismatch between a certain belief (dysfunctional thought/assumption/schema) and a certain experience, in the literature also referred to as *corrective experiences*, that repair previous negative experiences (Boswell, 2013). For example, a patient who believes he or she is not capable of initiating a conversation (i.e., dysfunctional thought, attitude, or schema) may create a mismatch with this belief by evaluating and targeting this negative belief using cognitive change work. Theoretically, it seems possible that reactivation of the memory structure of this negative belief in combination with the presence of a mismatch (i.e., by evaluation and targeting the belief) followed by providing new functional information can lead to reconsolidation and lead to change of the particular memory structure of this dysfunctional belief. However, empirical studies on the use of reconsolidation have focused mainly on the disruption of fear memory or have used healthy participants. Also, although there is substantial evidence in favor of the role of disruption or modification of memory during reconsolidation, alternative explanations (e.g., not protein synthesis–dependent cellular changes but contextual cues play a role in the retrieval of memory) and inconsistent findings remain (Elsey, Van Ast, & Kindt, 2018). Without the use of controlled studies that use an experimental framework to test the effects of reconsolidation (for this framework, see Elsey et al., 2018), conclusions about the role of reconsolidation in CBT for depression cannot be drawn.

Behavioral activation. Besides cognitive change, another mechanistic pathway that CBT may act through is that of behavioral activation. Behavioral strategies have always been a part of CBT but more recently have been stripped of their cognitive accompaniments to develop an approach to treatment called *behavioral activation* (BA). BA can be broken down into different therapeutic procedures, including activity monitoring, assessment of life goals and values, activity scheduling, skills training, relaxation training, contingency management, procedures targeting verbal behavior, and procedures targeting avoidance (Kanter et al., 2010). What it does not contain are any specific cognitive change strategies. Although each procedure in BA has its own specific focus, they all are hypothesized to target behavioral treatment processes that are disturbed during depression: a deficit in positive reinforcement as a consequence of not engaging in pleasant activities and a heightened aversiveness to negative events (Eshel & Roiser, 2010). Both sets of processes lead to decreased engagement in pleasant activities and increased engagement in negative activities (Dimidjian, Barrera, Martell, Muñoz, & Lewinsohn, 2011; Kanter et al., 2010), often measured by the use of self-report or observational instruments (Manos et al., 2010). There is support for the relation between the type and frequency of activities and depressive outcome, supporting the idea that the reinstatement of positive reinforcement and activities is responsible for a reduction in depression (Kanter et al., 2010; Manos et al., 2010). Moreover, it has been argued that changes in dysfunctional thinking do not play a role in BA because BA as a stand-alone procedure is as effective as cognitive change work in reducing depressive symptoms (Longmore & Worrell, 2007; Richards et al., 2016). However, equal effectiveness of cognitive therapy (CT) and BA does not inform us about the underlying causal mechanisms of change (Keefe & DeRubeis, 2016; Lorenzo-Luaces, Keefe, & DeRubeis, 2016), and the relation between BA and changes in dysfunctional thinking has been a controversial one. From early on, it has also been proposed that changes in dysfunctional thinking might be the treatment process of change underlying behavioral procedures (Beck, 1970; Beck & Haigh, 2010), and behavioral strategies are often used to test dysfunctional thoughts and beliefs during CBT (Hollon, 1999). For example, in his classic work on self-efficacy, Bandura (1977) suggested that enactive (behavioral) procedures work best but largely work through cognitive mechanisms (expectations). Moreover, activity monitoring has been used to facilitate cognitive restructuring (Hiebert & Fox, as cited in Kanter et al., 2010), and changes in attributional style early on in treatment predicted the change in depressive symptoms during the course of BA (Jacobson et al., 1996). These results suggest that increasing the amount of positive and rewarding events will lead the patient to reassess his or her beliefs and subsequently results in symptom relief. In other words, procedures focused on behavioral activation might be just another way to target the process of dysfunctional thinking and change beliefs.

CBT skills. A third potential mechanism of change is the development and use of cognitive and behavioral skills (Barber & DeRubeis, 1989; Strunk, Hollars, Adler, Goldstein, & Braun, 2014). Cognitive change work aims to impart a number of different skills, including the capacity to reevaluate the accuracy of one's own automatic thoughts (a cognitive skill), whereas BA aims to impart the capacity to engage proactively in pleasurable activities (a behavioral skill). So far, cognitive behavioral therapy skills have been measured by using self-report instruments or testing responses to hypothetical situations about negative thoughts (e.g., using the Ways of Responding questionnaire; Strunk et al., 2007). Both treatment processes (i.e., cognitive and behavioral therapy skills) are thought to result in a reduction of depressive symptoms and help patients cope more adaptively (Strunk et al., 2014). Evidence in support of their role as mechanisms of change include the finding that the ability to monitor dysfunctional thoughts is disrupted when depressed but restored after treatment (Sheppard & Teasdale, 2004), that CBT skill use during cognitive therapy predicts symptom reduction (Strunk et al., 2014) and subsequent freedom from relapse (Strunk et al., 2007), that CBT skill use mediates change in depression during guided Internet CBT (Forand et al., 2017), and that higher levels of cognitive skill use are related to lower levels of dysfunctional thinking (Adler, Strunk, & Fazio, 2015; Strunk et al., 2014). Moreover, compensatory skills might provide an alternative explanation for the enduring effects of CBT: Something is being changed that is not changed using antidepressants (Dobson et al., 2008), and the long-term effects of CBT might seem hard to account for if negative beliefs would be deactivated only.

In short, CBT procedures and changes in underlying CBT treatment processes seem to be associated with changes in depressive symptoms. However, research on direct relationships between procedures, processes, and outcome in the form of a causal pathway is lacking, and it is not clear what kind of process is responsible for the effects of CBT and whether this might differ for each procedure. In fact, without the direct manipulation of an isolated CBT procedure, it is impossible to detect a true causal relationship. In relation to cognitive change, it is not clear yet whether cognitive change is an actual change in the dysfunctional thought structures (accommodation model), the deactivation of dysfunctional thought structures and activation of more functional structures (deactivation model), or the use of compensatory skills that keep the depression at bay (compensation model). An overview of each procedure and its hypothesized treatment processes are given in Table 2.

Table 2. Therapeutic Procedures and Processes in Cognitive Behavior Therapy for Depression

Causal mechanisms of change	Therapeutic procedure	Treatment process	Relevant articles
Cognitive change	Cognitive change work	Deactivation of dysfunctional thinking	Garratt, Ingram, Rand, and Sawalani (2007)
		Accommodation of dysfunctional thinking	Lorenzo-Luaces, German, and DeRubeis (2015)
Cognitive and behavioral therapy skills	Cognitive change work	Cognitive behavioral therapy skills	Barber and DeRubeis (1989)
			Strunk, Hollars, Adler, Goldstein, and Braun (2014)
Behavioral activation	Activity monitoring	Increased positive activation and reward/ less aversiveness and unpleasant events	Dimidjian, Barrera, Martell, Munoz, and Lewinsohg (2011)
	Assessment of life goals and values		Kanter et al. (2010)
	Activity scheduling		
	Skills training		
	Relaxation training	Deactivation of dysfunctional thinking	Lorenzo-Luaces, Keefe, and DeRubeis (2015)
	Contingency management		
	Procedures targeting verbal behavior	Accommodation of dysfunctional thinking	Keefe and DeRubeis (2016)
	Procedures targeting avoidance		

The Role of Learning Capacity in CBT for Depression

As seen previously, depression has been associated with impairments in cognitive and neurobiological processes that have been related to learning and may be involved in the onset or maintenance of the depressive symptoms. However, it is yet unclear how the learning processes that are involved in the development and maintenance of depression are related to CBT's procedures and hypothesized treatment processes and may interfere with or benefit CBT outcome. More specifically, if learning plays a role in CBT for depression, we would expect that (a) patients with deficits in learning processes (i.e., worse learning capacity) would show less change in the underlying processes in CBT and exhibit worse outcome in CBT compared with patients whose learning processes are relatively unimpaired (i.e., well-functioning learning capacity) and (b) to the extent CBT procedures target learning processes directly, they will lead to better short- and long-term outcomes. The following section will address these questions by systematically reviewing empirical evidence that bears on the relation between (a) CBT procedures and learning processes, (b) learning processes and CBT processes, and (c) learning processes and CBT outcome (see Fig. 2).

During the systematic search, peer-reviewed articles published between January 1980 and April 2018 were searched using databases Pubmed, PsychInfo, and EMBASE. We included empirical studies that focused on a combination of (a) CBT procedures or CBT processes, (b) learning processes, and (c) depression. Excluded were (a) studies focusing on patients with depression plus a comorbid somatic disease (e.g., the presence of Parkinson, dementia, or multiple sclerosis), (b) studies on participants below the age of 18, (c) studies with nonhumans, and (d) studies on third-wave CBT procedures or third-wave CBT processes (e.g., mindfulness-based CBT). The presence of comorbid mental disorders was not excluded. Search terms were a variety of synonyms related to CBT (#1), learning processes (#2), and depression (#3). Both MeSH terms and text words were included in the search process, and the three sets of search terms were combined as follows: #1 AND #2 AND #3. Our initial selection was based on titles and abstracts. As a result of the search, we examined abstracts from PubMed (1,306), Embase (59), and PsychInfo (744). The exact search terms are presented in the Supplemental Material available online. From the 2,109 abstracts, we retrieved 159 full-text articles for possible inclusion, of which 104 were excluded because they did not meet the inclusion criteria. Reference lists of resulting systematic reviews and opinion articles were checked for empirical studies that satisfied the inclusion criteria. An overview of the resulting 55 studies that were considered relevant for evaluating the relation between learning processes, CBT procedures, and CBT processes can be found in Table S1 in the Supplemental Material. The quality of each study was taken into account by evaluating the levels of evidence for therapeutic and prognostic studies (Burns, Rohrich, & Chong, 2011; OCEBM Levels of Evidence Working Group, 2011; Shekelle, Maglione, & Luoto, 2013; Song & Chung, 2010).

Are CBT procedures related to learning processes?

With regard to cognitive processes, evidence from randomized-controlled trials (RCTs) showed that compared with control or active intervention groups, CBT led to

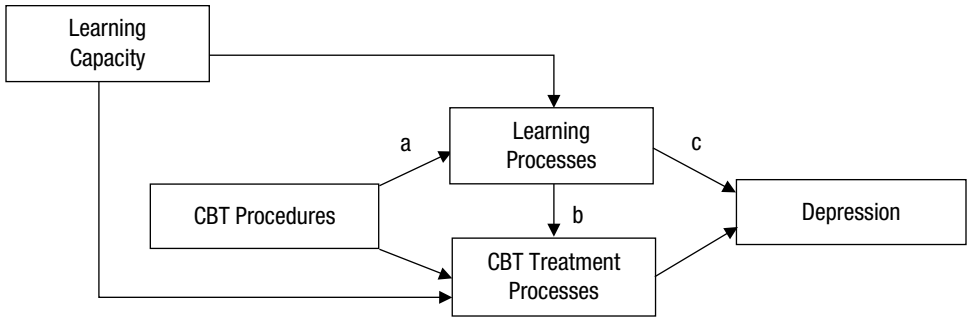

Fig. 2. A hypothetical model to investigate the role of learning capacity in cognitive behavior therapy.

improved visual sustained attention, spatial problem solving, and verbal fluency in depressed adults (Groves et al., 2015); diminished attentional bias related to infant distress in depressed pregnant women (Pearson et al., 2013); and better general cognitive functioning in elderly depressed patients (Hummel et al., 2017). Another but nonrandomized and uncontrolled trial pointed to improved accuracy and reaction times on a cognitive control task after CBT in patients with a mood disorder (Beard et al., 2015). In addition, RCTs and case-control studies have pointed to change in patterns in brain activity in relation to CBT's procedures. For example, CBT has been linked to improvements in areas linked to self-referential processing of negative emotional stimuli in depression (Yoshimura et al., 2017), enhanced capacity for "top-down" emotion regulation from the fronto-parietal network on the amygdala (Shou et al., 2017), and decreases or increases in areas linked to cognitive control or emotional processing (Fu et al., 2008; Goldapple, Segal, Garson, & Lau, 2004; Kennedy et al., 2007; Ritchey, Dolcos, Eddington, Strauman, & Cabezza, 2011). Other studies have specifically focused on the effects of CBT's BA procedures on changes in neurobiological processes related to reward (Dichter et al., 2009) or have shown that BA procedures, compared with their absence, led to reduced connectivity of brain areas that are related to self-referential processing such as rumination and cognitive control and the effortful regulation of emotion, leading to better control of attention toward positive stimuli in the external environment (Yokoyama et al., 2017). Not all studies supported a relation between CBT procedures and learning processes: Compared with treatment as usual, group CT did not differentially affect the specificity of autobiographical memories (Spinhoven et al., 2006), whereas in the study of Gollan et al. (2014), brain activity involved in motivation did not change after BA procedures.

Although most of these findings support the hypothesis that CBT procedures affect several different learning processes, two factors limit the interpretation of these findings. First, the direction of these effects on cortical activity was not always consistent. For example, whereas one study reported a decrease in activity in the cerebral cortex following CBT (dorsal, medial, ventral frontal cortex; Goldapple et al., 2004), others reported an increase in activity (i.e., ventromedial prefrontal cortex, left inferior temporal cortex, ventromedial frontal cortex, and right occipital-temporal cortex activation; Kennedy et al., 2007; Ritchey et al., 2011). A possible interpretation for the decrease in cortical activity might be that CBT reduces the retrieval and encoding of maladaptive memories, rumination, or the overprocessing of information (Goldapple et al., 2004), whereas an explanation for an increase in cortical activity was an enhanced engagement of processes involved in modulating responses to affect-laden stimuli (Ritchey et al., 2011). Second, it is unclear whether the changes in brain activity are independent of symptom level beyond (change in) depression. Whereas some studies did not find a correlation between changes in brain activity and change in depressive symptoms (Yang et al., 2018; Yokoyama et al., 2017) or find that CBT led to cognitive changes beyond change in depressive symptoms (Groves et al., 2015), other studies showed that controlling for depression nullified the change in brain activity (Goldapple et al., 2004) or reported a correlation between the changes in activation of brain areas and changes in depressive symptoms (Yoshimura et al., 2017).

Are learning processes related to change in CBT processes?

Several studies have related cognitive processes to the process of dysfunctional thinking (Johnco, Wuthrich, & Rapee, 2015; Romero, Sanchez, & Vazquez, 2014; Sheppard & Teasdale, 2000, 2004; Wenzlaff & Bates, 1998). For example, processing under reduced cognitive control has been associated with biases in the recall of negative self-referent information in formerly depressed individuals when controlled for current depressive symptoms (Romero et al., 2014), and

cognitive load directly led to increased dysfunctional thinking in participants identified as at risk for depression (Wenzlaff & Bates, 1998). In addition, depressed patients have shown less metacognitive monitoring of dysfunctional statements (Sheppard & Teasdale, 2000), whereas partially remitted patients resembled nondepressed controls on metacognitive monitoring of dysfunctional cognitions but acutely depressed patients on access to dysfunctional schemas (Sheppard & Teasdale, 2004). These findings seem in line with the hypothesis that associative processing is a default mode of thinking, reflective processing can be used to change biases in associative thinking (Beevers, 2005), and improvement in depression is related to better metacognitive monitoring of dysfunctional thinking rather than a reduced access to dysfunctional schemas (Sheppard & Teasdale, 2004). However, in contrast, the study of Gemar, Segal, Sagrati, and Kennedy (2001) did not find evidence for the relation between dysfunctional thinking and associative processing, suggesting these are two different processes.

Besides its relation with cognitive control processes, the process of dysfunctional thinking has been linked to greater brain activity in areas involved in attention processing and episodic memory retrieval (Sankar et al., 2015) and showed a strong positive correlation with reduced levels of serotonin in patients with major depressive disorder (Meyer et al., 2004). More specific, extreme, but not regular responses on the scale measuring dysfunctional thinking (Dysfunctional Attitude Scale, or DAS) were related to greater activation in areas related to the processing of negative information, attention, working memory of emotional stimuli, and visual processing (i.e., the left parahippocampal gyrus, inferior parietal lobe, and precuneus) but not greater activity in the amygdala in patients with major depressive disorder compared with healthy controls. In addition, activation in the left parahippocampal gyrus did attenuate less in depressed patients after CBT relative to healthy controls, suggesting a persistent vulnerability to dysfunctional thinking (Sankar et al., 2015).

Deficits in reward and reinforcement learning have been linked to brain functioning in depressed patients that is different from healthy controls (Knutson, Bhanji, Cooney, Atlas, & Gotlib, 2008; McCabe, Cowen, & Harmer, 2009; Pizzagalli et al., 2009; Wang, Zhou, Dai, Ji, & Feng, 2017; Yang et al., 2015). For example, abnormal brain functioning and deficits in areas involved in reward and response have been linked to self-report measures of behavioral activation and inhibition (i.e., approach/avoidance motivation; DelDonno et al., 2017; Gollan et al., 2014). Whereas self-reported levels of behavioral activation and inhibition modulated brain activity during the reappraisal of emotional pictures differently compared with healthy controls (Wang et al., 2017). Also, compared with healthy controls, depressed patients showed a different neural response in reward-related brain structures involved in cost-benefit decision making during a decision-making task (Yang et al., 2015), abnormalities in the neural representation of reward to the sight of rewarding stimuli (McCabe et al., 2009), and reduced positive affect to reward stimuli and less arousal following gains that were mirrored by differences in brain activity (Pizzagalli et al., 2009).

Other studies have focused on the role of brain functioning in reward and reinforcement by measuring the neurobiological responses to (negative) feedback (Elliott, Sahakian, Michael, Paykel, & Dolan, 1998; Santesso et al., 2011; Tavares et al., 2008; Tucker, Luu, Frishkoff, Quiring, & Poulsen, 2003) or associations with the monitoring of errors (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008; Ruchsow et al., 2004). For example, responses to (negative) feedback about the performance of a cognitive task were associated with attenuation of activation within a network implicated in reward mechanisms (Elliott et al., 1998), whereas depressed participants showed lower accuracy and hyperactivation in areas related to affective processing and cognitive aspects of self-evaluations after committing errors (Holmes & Pizzagalli, 2008).

In sum, these studies support the hypothesis that a variety of learning processes (i.e., cognitive control, activity in different areas of the brain) are related to processes that are targeted during CBT (i.e., dysfunctional thinking, reward, reinforcement). It should be noted that except for one study that manipulated cognitive load experimentally to investigate its direct effects on depressive thinking (Wenzlaff & Bates, 1998), studies on the relation between CBT treatment processes and learning processes provide only preliminary evidence of correlational nature.

Are learning processes related to CBT outcome?

Studies have pointed to a relationship between measures of cognitive processes and CBT outcome (Ekeblad, Falkenstrom, & Holmqvist, 2015; Goodkind et al., 2015; Hummel et al., 2017; Yang et al., 2018). For example, higher pretreatment scores on self-report measures of reflective functioning (Ekeblad et al., 2015) and poor pretreatment performance on behavioral measures of executive functioning (Goodkind et al., 2015) have been related to improvements in depressive symptoms. In contrast, errors on a cognitive control task at baseline predicted self-reported attentional control and rumination but not the levels of depression after treatment (Beard et al., 2015); there seems to be no relation

between measures of executive functioning and CBT outcome (Goodkind et al., 2015; Thompson et al., 2015), and autobiographical memory and neurocognitive variables at pretreatment did not predict relapse or recurrence of depression (Spinhoven et al., 2006).

Studies on the association between neurobiological processes and response to CBT point to the following results. First, it appears that patients who subsequently respond to treatment may be more comparable with healthy participants in baseline neural processes related to cognitive control (Crowther et al., 2015; Fu et al., 2008; Ritchey et al., 2011; Webb et al., 2018). For example, depressed patients who showed the most clinical improvement had similar patterns of activation in an area (anterior cingulate cortex) that is activated during tasks using the potential loss of reward to healthy participants (Fu et al., 2008), whereas less impairment in areas hypothesized to be involved in cognitive control during emotional processing was related to better response (Ritchey et al., 2011). Possibly, depressed patients who have higher ventromedial prefrontal cortex (vmPFC) activity before treatment may have a better functional circuitry that is necessary to effectively use reappraisal strategies and improve on outcome, whereas patients with low activity might have more trouble processing the information that is provided in the session or applying CBT procedures in daily life (Ritchey et al., 2011). This hypothesis is in line with an earlier finding that showed that vmPFC activity is associated with learned resilience in rats (Maier, Amat, Baratta, Paul, & Watkins, 2006). Moreover, in the study by Crowther et al. (2015), brain connectivity differed in healthy controls compared with depressed patients, but connectivity between different brain areas that predicted better treatment response were more similar to healthy participants, whereas in another study, larger pretreatment right rostral anterior cingulate cortex (rACC) volume (i.e., reduced volume has been related to maladaptive forms of self-referential processing) was a significant predictor of greater depressive symptom improvement in Internet CBT (Webb et al., 2018).

Second, studies indicate that CBT might be most useful for individuals who show elevated cognitive and emotional biases at baseline (Burkhouse et al., 2016; Ritchey et al., 2011; Siegle, Carter, & Thase, 2006; Stange, MacNamara, Kennedy, et al., 2017). For example, patients with stronger negative bias, enhanced attention to aversive stimuli, and increased reactivity in the amygdala in response to negative words showed the largest improvement in depression during CBT (Ritchey et al., 2011; Siegle et al., 2006; Stange, MacNamara, Barnas, et al., 2017). Another example showed that depressed patients with less neural reactivity toward rewarding stimuli at baseline were more likely to respond and showed greater pre- to posttreatment reduction in depression (Burkhouse et al., 2016).

Third, changes in brain activity during CBT have been related to better treatment outcomes. For example, greater reduction in brain areas related to emotion regulation during CBT was associated with better treatment outcome (Rubin-Falcone et al., 2018), whereas greater increase in brain-derived neurotrophic factor was related to greater improvement in depression (Gourgouvelis, Yielder, Clarke, Behbahani, & Murphy, 2018). Fourth, brain connectivity has been used to distinguish drug (antidepressant) versus CBT responders, pointing to the possibility that change patterns in brain activity differ between unique treatments (Seminowicz et al., 2004).

These studies suggest that learning processes are related to CBT outcomes. However, not all results were consistent. For example, some studies did not find a relation between cognitive performance and CBT outcome (Goodkind et al., 2015; Spinhoven et al., 2006; Thompson et al., 2015). Also, because only a few studies focused on the relation between changes in brain activity from pre- to posttreatment and treatment outcome or the differential change between different treatments, definitive conclusions are premature. Note, too, that not all studies had an adequate sample size ($n >$ 50), and evidence was of only correlational nature.

In conclusion

The systematic search resulted in a set of studies that by and large supported the presence of an association between learning processes, CBT procedures, CBT processes, and CBT outcome. First, CBT procedures improve cognitive functioning in some areas (different aspects related to executive functioning) but not others (the specificity of autobiographical memories). In addition, CBT and BA procedures were related to change in areas of the brain related to emotional and cognitive processing and the experience of reward but not to activity in areas related to motivation. Second, CBT's treatment processes have been linked to learning processes: (a) Dysfunctional thinking has been related to deficits in metacognitive monitoring and reduced levels of serotonin and brain activity in areas related to, among other things, the processing of (negative) information, whereas (b) disturbances in reinforcement, response to reward, and loss or (negative) feedback have been linked to brain activity different from that of healthy controls. Third, most but not all studies showed that better performance at baseline on a variety of cognitive tasks (reflective functioning, executive functioning, cognitive and emotional control tasks) predicted better outcome of CBT. Moreover, patients with fewer

impairments in areas of the brain involved in cognitive control processes and higher activity in brain areas related to emotional biases at baseline were more likely to show improvement during CBT. Linking these findings together, they suggest that CBT procedures target learning processes and that change in CBT's treatment processes is made more difficult by impairments in learning processes, which then results in less change in the processes involved in CBT and subsequent worse outcome.

Nonetheless, there are limitations. For example, studies varied greatly in their methodologies: Whereas several studies used an experimental design, the majority of studies were case-control studies and only resulted in correlational evidence for the hypothesized relationships. Also, it is yet unknown how learning processes affect CBT treatment processes and outcome in the long term. In addition, most sample sizes were small (n < 50), and not all studies used a control group, whereas other studies used an active intervention control group that may complicate interpretation of the findings. Differences in study characteristics, such as a focus on different areas of the brain, different populations, different doses of CBT, and the use of different tasks during the measurement of brain activity, warrant caution in interpretation. Moreover, these differences may explain the variability in the results (e.g., the relation between cognitive processes and CBT outcome or the different directions of change in brain activity in areas related to cognitive control after CBT procedures). Finally, for some of the studies, it was not clear whether change in neurobiological processes are just nonspecific consequences of reductions in depression or whether neurobiological processes independently affect the effect of CBT procedures or change in CBT treatment processes.

An Updated Model on the Causal Mechanisms in CBT for Depression

Despite the aforementioned limitations, evidence so far points to an association between learning processes, CBT procedures, CBT processes, and CBT outcome. Therefore, we propose that learning capacity might be an essential element in the causal mechanisms underlying change in CBT for depression that explains (in part) variability in response to treatment (see Fig. 3). In essence, we propose that learning capacity moderates the relation between CBT procedures and change in CBT processes. Individual differences in learning capacity might affect change in CBT processes as a consequence of the application of CBT procedures and explains (in part) individual differences in the effect of

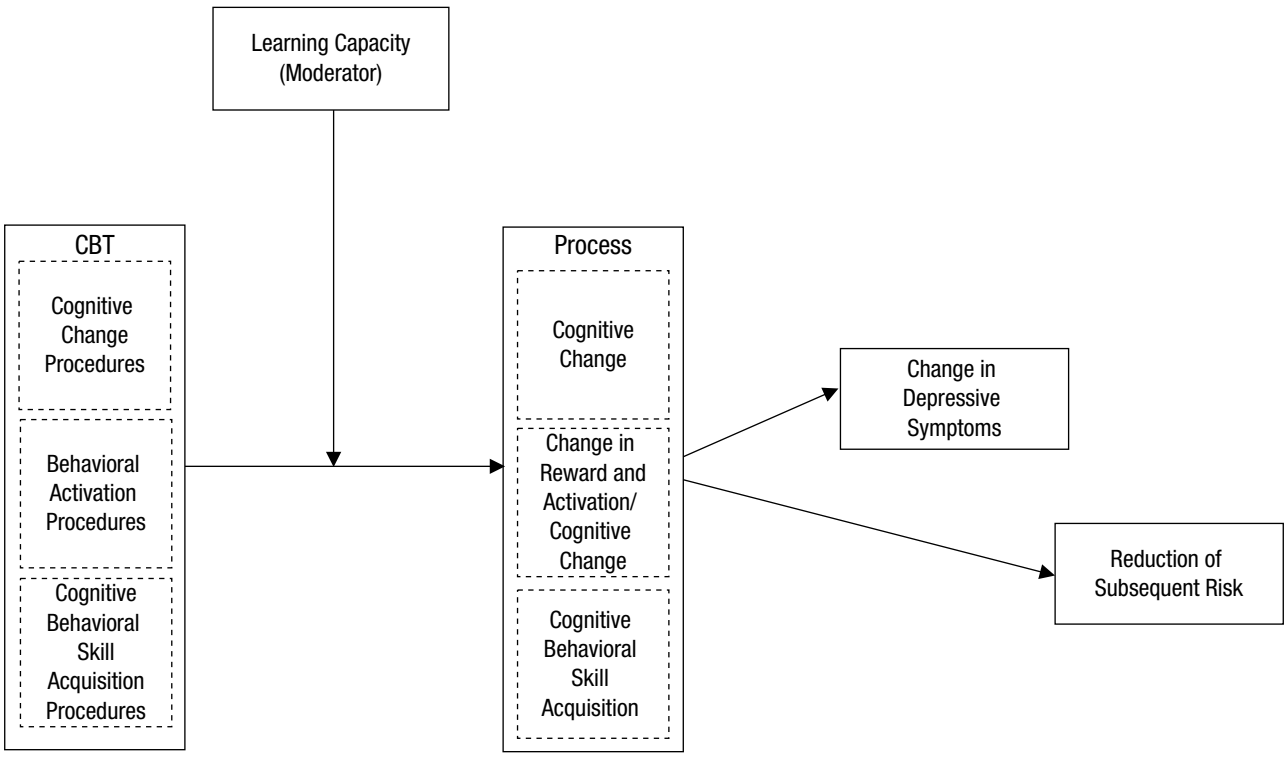

Fig. 3. Learning capacity as a moderator of the effect of cognitive behavior therapy (CBT) procedures on CBT process change in CBT for depression.

CBT procedures on depressive outcome. Moreover, individual differences in learning capacity might predict differences in risk for relapse after CBT. This implies that one way to optimize CBT procedures to improve their effect on treatment outcome is not only to find out what processes are changed as a result of which procedures (i.e., insight into the causal mechanisms of change) but also to find ways to increase learning capacity and investigate whether different procedures work better for subgroups of patients with different cognitive profiles.

The next section will describe methods for empirical tests of these proposals.

Directions for Future Research to Improve CBT

Increasing the effects of treatment: procedures to improve therapeutic learning

Procedure 1: optimize session frequency. One way to improve therapeutic learning and subsequent therapeutic outcome might be by optimizing the frequency of therapy sessions. Both neurobiological and psychological studies point to the presence of a certain timeframes wherein learning is optimal. First, neurobiological studies have shown that repeating a learning task within a certain timeframe can increase the survival of newborn cells. For example, Gould, Beylin, Tanapat, Reeves, and Shors (1999) reported that engaging animals in learning activities requiring the hippocampus (e.g., spatial water maze training) but not those not requiring the hippocampus 1 week after labeling of newborn cells led to the increased survival of the new cells that would normally diminish. In addition, Epp, Spritzer, and Galea (2007) found that providing a learning task when cells are 6 to 10 days old (but not younger than 6 days or older than 11 days) increased cell survival, whereas in the study by Döbrössy et al. (2003), training at Days 4 to 8 (compared with Days 1–4) led to increased cell proliferation and survival and decreased the newly born cells produced during the early phase, a process that was linked to better performance. Thus, the process underlying learning seems to be more complex than just survival of new cells: Learning tasks might induce the death of the (younger) less relevant cells within certain timeframes to trigger the survival and proliferation of other cells and in this way increase learning (Döbrössy et al., 2003; Dupret et al., 2007).

Second, observational, naturalistic studies have pointed to the importance of the frequency of therapy sessions (Erekson, Lambert, & Eggett, 2015; Reardon, Cukrowicz, Reeves, & Joiner, 2002; Reese, Toland, & Hopkins, 2011). In the field of anxiety, randomized studies highlighted the potency of a greater session frequency by showing that a higher intensity of treatment leads to faster recovery and/or better treatment outcomes (Bohni, Spindler, Arendt, Hougaard, & Rosenberg, 2009; Ehlers et al., 2014; Foa et al., 2018; Herbert, Rheingold, Gaudiano, & Myers, 2004). For example, 12 weekly sessions of cognitive behavior therapy for social anxiety disorder resulted in better outcome than the same number of sessions extended over 18 weeks (Herbert et al., 2004), whereas a massed 3-week cognitive behavioral therapy for panic disorder led to faster recovery but was as effective as a 13-week cognitive behavioral therapy schedule (Bohni et al., 2009). For depression, a metaregression analysis found a positive association between the number of sessions per week and the magnitude of the effect of psychotherapy for adult depression (Cuijpers, Huibers, Ebert, Koole, & Andersson, 2013). Importantly, it was not the total number of sessions or the duration of therapy but the number of sessions per week (i.e., session frequency) that positively predicted positive response to psychotherapy (Cuijpers, Huibers, et al., 2013). In our recent randomized trial, we investigated whether twice-weekly compared with once-weekly sessions of CBT or interpersonal psychotherapy (IPT) for depression led to better treatment outcomes 6 months after the start of treatment (Bruijniks et al., 2015). Results have been analyzed, and we hope to publish these findings as soon as possible.

Procedure 2: investigate procedures to increase change in dysfunctional thinking. Anxiety research has provided us with specific procedures that might disrupt or utilize reconsolidation to change or erase dysfunctional structures or improve the extinction of fear memories. Procedures focused on the disruption or use of reconsolidation start with the destabilization of a certain memory structure by reactivation of the memory structure in combination with providing a mismatch between the expectation of what would happen according the target learning versus what actually happens during the reactivation. Subsequently, the memory structure can be changed by using a more invasive (administration of a protein synthesis inhibitor) or noninvasive procedure (e.g., a single extinction trial that provides a second mismatch with the target learning). Other procedures in the field of anxiety have focused on ways to optimize inhibitory learning (i.e., optimize the learning of a new association that arises beside the old dysfunctional associations; Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014) and pointed to procedures such as including aversive events (Culver, Stevens, Fanselow, & Craske, 2017), multiple stimuli (Waters, Kershaw, & Lipp, 2018), or limiting the use of safety cues while maximizing retrieval cues

(i.e., cues associated with fear extinction; Blakey & Abramowitz, 2019) to optimize the strength and generalization of new learned associations.

However, as already described, the precise conditions under which reconsolidation will occur or might lead to extinction are under debate, and alternative explanations have to be ruled out (Dunsmoor, Niv, Daw, & Phelps, 2015). Also, these studies have focused mostly on the effects of procedures on disrupting reconsolidation or enhancing the effects of extinction in samples with anxiety, whereas these procedures might work differently for different populations. For example, although a prediction error might be important for the success of procedures using reconsolidation, there have been studies that suggest that prediction error might be impaired in depression because of symptoms of anhedonia or reward system dysfunction (Beevers, 2005; Gradin et al., 2011), implying that the procedure to create a mismatch may be different for anxious versus depressed patients.

It is possible that CBT procedures already lead to the reactivation of dysfunctional memory structures and subsequent change of memory (in contrast to the development of a new memory) but that there are differences between procedures (e.g., the presence of multiple mismatches), between patients differing in learning capacity, or between therapists differing in the ability to formulate and retrieve the most relevant dysfunctional memory structure or the ability to modulate arousal. This may account for differences in memory change versus (de)activation of memory structures and subsequent outcome. The use and effects of the different procedures on dysfunctional thinking in CBT for depression have to be tested.

Procedure 3: should we modulate emotional arousal?

Early studies have shown that too little or too much arousal leads to less efficient learning (Yerkes & Dodson, 1908), and more recent studies focused on the role of emotional arousal in the success of psychotherapy (Lane et al., 2015). Studies have shown that a sufficient amount of emotional arousal might lead to better cognitive performance (e.g., better declarative memory or executive functioning; Bos, Schuijer, Lodestijn, Beckers, & Kindt, 2014; Kofman, Meiran, Greenberg, Balas, & Cohen, 2006; Lambourne & Tomporowski, 2010), whereas too much stress seems to impair memory (Salehi, Cordero, & Sandi, 2010). In addition, (a moderate amount of) heightened arousal, as measured by both self-report or activity in brain areas related to emotional reactivity, seems to predict the success of psychotherapies for depression (Carryer & Greenberg, 2010; Ritchey et al., 2011; Siegle et al., 2006). For example, an optimal frequency (i.e., 25% of the session) of high expressed emotional arousal was found to relate to outcome during experiential therapy for depression (Carryer & Greenberg, 2010).

Although the role of emotion has always been recognized in CBT (Beck, 1971; Hofmann, Asmundson, & Beck, 2013; Samoilov, 2000), it is not clear how emotional arousal affects learning and outcome in CBT for depression, and clear guidelines for how patients should manage arousal in session or outside of the session in regular CBT for depression are lacking (Samoilov, 2000). One potential way to increase emotional arousal in depressed patients is the use of experiential procedures, such as the two-chair technique (i.e., a technique drawn from Gestalt psychotherapy that is often used in schema therapy, a therapy that expands on CBT; Young, Klosko, Weishaar, & Temple, 2003), to help activate affect and incorporate it into cognitive change work (Greenberg, 1979; Samoilov, 2000). Hayes and colleagues (2007) found that an exposure-based CBT resulted in a transient worsening of depressive symptoms (i.e., depression spikes) that was related to more cognitive emotional processing. Also, depression spikes were related to less depression at the end of treatment when they occurred during the part of treatment that actively focused on activating, exploring, and questioning the thoughts, affect, behavior, and somatic responses related to depression (Hayes et al., 2007). These findings are in line with our findings that CBT might be most useful for individuals who show increased cognitive and emotional biases and higher activity in the brain regions subserving emotional processing (Burkhouse et al., 2016; Ritchey et al., 2011; Siegle et al., 2006; Stange, MacNamara, Kennedy, et al., 2017). Possibly, patients with increased cognitive and emotional biases are better able than patients with lower levels of cognitive and emotional biases in activating depressed feelings, leading to more depression spikes, more exploration, and questioning of material related to depression and subsequent better outcomes.

Nevertheless, a recent study showed that CBT that included more emotion-focused procedures did not lead to better outcomes compared with regular CBT (Grosse Holtforth et al., 2017). In addition, in contrast to patients with anxiety disorders, depressed patients without anxiety showed an impaired ability to modulate arousal-related cortical structures in the right hemisphere (Moratti, Rubio, Campo, Keil, & Ortiz, 2008). This suggests that among depressed patients who do not suffer from anxiety, the challenge is to ensure that there is enough emotional arousal in the session, instead of preventing the occurrence of too much emotional arousal. Studies in the field of anxiety have investigated the role of fear as an indicator of learning during exposure treatment and showed that it is not the reduction of fear but the success of inhibitory learning (i.e.,

learning of new information that competes with the original learning that is thought to be central to extinction) that mediates subsequent treatment success (Craske et al., 2008). Although emotional arousal might have been linked to better cognitive performance and success in psychotherapy, it might thus not reflect whether therapeutic learning took place, at least in the context of exposure for anxiety. In addition, it is yet unclear whether emotional arousal is (equally) important for procedures focused on using reconsolidation or extinction.

Procedure 4: adapt existing procedures by anticipating learning capacity. Multiple studies have tried to enhance the effects of treatment by developing therapeutic procedures that improve patients' cognitive functioning (Baert, De Raedt, Schacht, & Koster, 2010; Lang, Blackwell, Harmer, Davison, & Holmes, 2012; Lang, Moulds, & Holmes, 2009; Lester, Mathews, Davison, Burgess, & Yiend, 2011), but these cognitive procedures do not seem to lead to better treatment success and might be even less effective (Cristea, Kok, & Cuijpers, 2015). Therefore, a better way to improve treatments might be to adapt existing therapeutic procedures to optimize learning. Harvey and colleagues (2014) argued that deficits in cognitive functioning might impair the degree to which patients can incorporate, remember, and practice the content of the therapy sessions and proposed different methods that can be used to provide cognitive support within the therapy session. For example, cue-based reminders appear to enhance recall of new information or the transfer of learning to other contexts, whereas increasing attention might help patients to encode new information. Moreover, additional interventions integrated into CBT, on a biological or a cognitive level (e.g., deep brain stimulation or attention training), may help to deal with cognitive biases and improve outcomes (Disner et al., 2011; Wiers, Boffo, & Field, 2018). Even more interesting, a recent small pilot study by Dong et al. (2017) suggested that adding a memory-support intervention to cognitive therapy might lead to better absolute recall, better absolute treatment outcome, and lower rates of relapse and recurrence, whereas one of our own experiments showed that retrieval tests following sessions of problem-solving therapy might lead to better recall of the sessions compared with rehearsal of the session, but only in patients with low scores on a working memory task (Bruijniks, Sijbrandij & Huibers, 2019). Cognitive therapy versus cognitive therapy integrated with memory support for depression is currently being tested (Harvey et al., 2017).

Another way to optimize learning in existing treatments might be to account for individual differences in learning capacity. For example, patients with deficits in executive functioning might benefit from cognitive support strategies that help them attend to or remember the contents of the session (e.g., by reducing the length of the session or increasing session frequency), whereas patients with less activity in areas related to emotional biases might benefit from strategies that benefit from the reactivation of (emotional) memory structures or activation of a sufficient amount of emotional arousal that is necessary for changing, activating, or deactivating memory. In line with this suggestion, a recent study pointed to the importance of individual differences by showing that participants with better working memory showed better fear extinction compared with those with worse working memory (Stout et al., 2018). It might also be beneficial for CBT to distinguish between individual differences in memory structure(s) that maintain the depression by measuring the idiosyncratic dysfunctional beliefs of the patient and adapting CBT procedures to this specific memory. For example, negative autobiographical memory is strongly related to depression, which might influence the focus of cognitive change procedures (Hankin et al., 2004; Köhler et al., 2015; Taft, Resick, Watkins, & Panuzio, 2009). A high percentage of depressed patients suffer from traumatic memories (Campbell et al., 2007; Green et al., 2006), and comorbid PTSD predicts worse outcomes through 1-year follow-up after CBT for depression (Green et al., 2006) and higher suicide risk (Cougle, Resnick, & Kilpatrick, 2009), and a history of childhood trauma has been associated with a lack of success in treatment for depressed patients (Nanni, Uher, & Danese, 2012) and chronicity of depression (Wiersma et al., 2009). Thus, for a subgroup of patients, it might be more efficient to focus on traumatic memory instead of focusing on the dysfunctional semantic structures. On the other hand, patients without traumatic memory might benefit from procedures that focus on changing other emotional memories. Moreover, as previous studies have related changes in different levels of cognitive change (automatic negative thoughts, dysfunctional attitudes, and core beliefs) in response to CBT (see pp. 228–229 in Garratt et al., 2007) but also suggested that these constructs may change independently from each other (Kwon & Oei, 1994, 2003; Whisman, Miller, Norman, & Keitner, 1991; Zettle & Rains, 1989), it would be interesting to find out whether the success of change of the superficial versus the deeper structures of dysfunctional thinking predicts the extent of remission and relapse after CBT for depression.

Experimental research and upcoming technologies

Experimental research. Unless we can conduct independent manipulations of the purported mediator, we can never know whether it was a true mechanism or just a statistical mediator. Most of the studies that have

investigated mechanisms of change within the context of RCTs have measured potential mediators (proxy variables that statistically account for the relation between the independent variable and the dependent variable) across the course of treatment to better understand the underlying causal processes (Lemmens et al., 2015; Lemmens, Müller, Arntz, & Huibers, 2016; Quilty, McBride, & Bagby, 2008). However, in the case of mediation, there are always three possible explanations for covariation: (a) direct causality (X→Y), (b) reverse causality (Y→X), and (c) third variable causality (Z→X and Z→Y in the absence of any direct causal link between X and Y). Thus, tests of mediation in the course of experimental trials can provide a guide as to what "active" ingredients and "causal" mechanisms to subject to further disconfirmation, but experimental manipulation always gives more certainty than pure statistical inference.

We believe that experimental research may benefit CBT in at least three different ways. First, experimental research can increase insight into the underlying causal mechanisms of change by isolating therapeutic procedures and investigating the direct effects of therapeutic procedures on hypothesized treatment processes and outcome (Bruijniks, Sijbrandij, Schlinkert, & Huibers, 2018). Future studies should isolate CBT procedures described in Table 2 to investigate their direct effects on treatment processes. An example would be to isolate activity monitoring (i.e., a BA procedure) and measure its direct effect on activation and reward processes (self-report or measures of brain activity). In addition, experimental studies should investigate the potential for combining cognitive support procedures (Dong et al., 2017), exposure-based procedures (Hayes et al., 2007), or procedures focused on using reconsolidation with CBT procedures and should investigate their effects on change in CBT's treatment processes and CBT outcomes compared with regular CBT procedures. Future studies should test whether different procedures lead to change or deactivation of dysfunctional memory structures. Also, studies should investigate the direct effects of CBT procedures on cognitive or neurobiological processes while controlling for (change in) depressive symptoms (or the other way around: isolate or manipulate cognitive processes to investigate their effect on the effect of CBT procedures on CBT processes). An example might be the manipulation of the patient's attention to CBT procedures.

Second, experimental research might help us to investigate which procedures work for whom. In other words, studies should focus on whether individual differences in learning capacity moderate or predict the effects of procedures on treatment processes and outcome and whether different procedures might work better for different subgroups of patients. Different memory structures underlying the depression (i.e., traumatic, autobiographical, or semantic memory), or profiles of learning capacity, might require a different or another frequency of procedures to reach the same levels of remission. Also, it might be that for patients with few impairments in learning capacity, it may not matter what you do, whereas for the ones who have impairments in learning capacity, you need a treatment with a specific effect to get them well (i.e., as was found in relation to depression severity; Driessen, Cuijpers, Hollon, & Dekker, 2010). Future research may test whether CBT would benefit from a structured assessment of the central memory structures or impairments in learning processes (i.e., learning capacity) before the start of treatment and an individualized matching procedure (from a personalized mental health perspective) that also takes learning processes into account (DeRubeis to et al., 2014; Huibers et al., 2015).

Third, an experimental design would be incredibly suitable to investigate the conditions under which a treatment is most optimal. Experimental studies should find out whether different levels of (emotional) arousal or session frequency can lead to differences in change in treatment processes, under what circumstances CBT procedures might lead to the deactivation or use of reconsolidation when targeting memory structures, and how we can make these procedures suitable for clinical practice.

Upcoming technologies. Although experimental manipulation seems to always give better confidence than statistical inference, recent developments in statistical methods have resulted in new ways to test the direction of causal change or rule out potential third variable confounders as alternative explanations for the relationship between the mediator and the outcome. For example, advances in structural equation modeling have pointed to the possibility to model change in processes over time to predict change in other processes or subsequent treatment outcomes while also controlling for reverse causality (Grimm, An, McArdle, Zonderman, & Resnick, 2012; Hamaker, Kuiper, & Grasman, 2015; Lemmens et al., 2017). In these models, comparisons of the strength of different parameters representing change from one variable to the other or the fit of models that represent different causal relationships are used to determine causal predominance. To gather multiple measurements of both the mediator and outcome over time, studies should consider the use of ecological momentary assessment (i.e., collecting data in the "real" world with use of tablets, cellphones, or watches) to collect a large amount of data in a short time (Armey, Schatten, Haradhvala, & Miller, 2015; Connolly & Alloy, 2017). In addition, studies pointed to the importance of disentangling the effects between (changes that occur between individuals) versus within (changes that happen within the individual) persons. More specifically,

this distinction can be used to rule out potential third variables (control for the stable between-person variance) when the main interest is to investigate causal relationships of change within persons (Falkenström, Finkel, Sandell, Rubel, & Holmqvist, 2017; Hamaker et al., 2015) or the separate roles of between versus within variance in predicting treatment outcomes (Sasso, Strunk, Braun, DeRubeis, & Brotman, 2016; Zilcha-Mano, Lipsitz, & Errázuriz, 2018; Zilcha-Mano, Muran, Eubanks, Safran, & Winston, 2017). However, the choice of different models or methods is large and may depend on the specific research question. In addition, studies that focus on ruling out reverse causality while also controlling for stable third variables have been scarce and may still have to deal with statistical difficulties that may complicate their interpretation (Falkenström et al., 2017). Moreover, controlling for stable between-group differences will not exclude the possibility of third variables that vary over time (Sasso et al., 2016). Thus, these sophisticated tests of mediation are promising for investigating causal mechanisms of change in psychological treatments but also need further development and have yet to be widely adopted by process-outcome researchers.

Summary and Conclusions

We have reviewed major theories about causal mechanisms in CBT for depression and investigated the role of learning capacity in CBT for depression. First, we concluded that although CBT is the predominant treatment for depression and provides a clear model of procedures, processes, and mechanisms of change, evidence on its mechanistic structure is mixed and mostly associational. Moreover, existing models do not provide an explanation of why CBT works for some people and not others. Second, available studies that show that depression is accompanied by impairments in cognitive and neurobiological learning processes that are related to CBT procedures, CBT processes, and CBT outcomes indicate that learning capacity might be related to CBT success. We propose that learning capacity is an essential element in the complex mechanism of change system in CBT for depression, meaning that it moderates the relation between CBT procedures and processes and might explain why therapeutic procedures lead to process change and (long-term) treatment success in some but not all patients. We integrated theory and literature relating to CBT and (therapeutic) learning and have proposed how to advance future research aimed to improve the outcomes of CBT for the depressed individual.

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Author Contributions

S. J. E. Bruijniks conducted the literature review. S. J. E. Bruijniks drafted the manuscript with contributions from R. J. DeRubeis, S. D. Hollon, and M. J. H. Huibers. All the authors approved the final manuscript for submission.

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References

Adler, A. D., Strunk, D. R., & Fazio, R. H. (2015). What changes in cognitive therapy for depression? an examination of cognitive therapy skills and maladaptive beliefs. *Behavior Therapy*, *46*, 96–109. doi:10.1016/j.beth.2014.09.001

Alloy, L. B., Abramson, L. Y., Whitehouse, W. G., Hogan, M. E., Tashman, N. A. L., Steinberg, D., . . . Donovan, P. (1999). Depressogenic cognitive styles: Predictive validity, information processing and personality characteristics, and developmental origins. *Behaviour Research and Therapy*, *37*, 503–531. doi:10.1016/S0005-7967(98)00157-0

Armey, M. F., Schatten, H. T., Haradhvala, N., & Miller, I. W. (2015). Ecological momentary assessment (EMA) of depression-related phenomena. *Current Opinion in Psychology*, *4*, 21–25. doi:10.1016/j.copsyc.2015.01.002.Ecological

Baert, S., De Raedt, R., Schacht, R., & Koster, E. H. W. (2010). Attentional bias training in depression: Therapeutic effects depend on depression severity. *Journal of Behavior Therapy and Experimental Psychiatry*, *41*, 265–274.

Bandura, A. (1977). Toward a unifying theory of behavioral change. *Psychological Review*, *84*, 191–215. doi:10.1037/0033-295X.84.2.191

Banich, M. T., Mackiewicz, K. L., Depue, B. E., Whitmer, A. J., Miller, G. A., & Heller, W. (2009). Cognitive control mechanisms, emotion and memory: a neural perspective with implications for psychopathology. *Neuroscience & Biobehavioral Reviews*, *33*, 613–630.

Barber, J. P., & DeRubeis, R. J. (1989). On second thought: Where the action is in cognitive therapy for depression.

Cognitive Therapy and Research, *13*, 441–457. doi:10.1007/BF01173905

Beard, C., Donahue, R. J., Dillon, D. G., Van't Veer, A., Webber, C., Lee, J., . . . Pizzagalli, D. A. (2015). Abnormal error processing in depressive states: A translational examination in humans and rats. *Translational Psychiatry*, *5*(5). doi:10.1038/tp.2015.54

Beck, A. T. (1970). Cognitive therapy: Nature and relation to behavior therapy. *Behavior Therapy*, *1*, 184–200. doi:10.1016/S0005-7894(70)80030-2

Beck, A. T. (1971). Cognition, affect, and psychopathology. *Archives of General Psychiatry*, *24*, 495–500. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/5578094

Beck, A. T., & Haigh, E. A. P. (2010). Advances in cognitive theory and therapy: The generic cognitive model. *Annual Review of Clinical Psychology*, *10*, 1–24. doi:10.1146/annurev-clinpsy-032813-153734

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York, NY: Guilford.

Beevers, C. G. (2005). Cognitive vulnerability to depression: A dual process model. *Clinical Psychology Review*, *25*, 975–1002. doi:10.1016/j.cpr.2005.03.003

Björkstrand, J., Agren, T., Åhs, F., Frick, A., Larsson, E. M., Hjorth, O., . . . Fredrikson, M. (2016). Disrupting reconsolidation attenuates long-term fear memory in the human amygdala and facilitates approach behavior. *Current Biology*, *26*, 2690–2695. doi:10.1016/j.cub.2016.08.022

Blakey, S. M., & Abramowitz, J. S. (2019). Dropping safety aids and maximizing retrieval cues: Two keys to optimizing inhibitory learning during exposure therapy. *Cognitive and Behavioral Practice*, *26*, 166–175. doi:10.1016/j.cbpra.2018.03.001

Bohni, M. K., Spindler, H., Arendt, M., Hougaard, E., & Rosenberg, N. K. (2009). A randomized study of massed three-week cognitive behavioural therapy schedule for panic disorder. *Acta Psychiatrica Scandinavica*, *120*, 187–195. doi:10.1111/j.1600-0447.2009.01358.x

Bos, M. G. N., Schuijer, J., Lodestijn, F., Beckers, T., & Kindt, M. (2014). Stress enhances reconsolidation of declarative memory. *Psychoneuroendocrinology*, *46*, 102–113.

Boswell, J. F. (2013). Intervention strategies and clinical process in transdiagnostic cognitive-behavioral therapy. *Psychotherapy*, *50*, 381–386. doi:10.1037/a0032157

Brewin, C. R. (1989). Cognitive change processes in psychotherapy. *Psychological Review*, *96*, 379–394. doi:10.1037/0033-295X.96.3.379

Brewin, C. R. (2006). Understanding cognitive behaviour therapy: A retrieval competition account. *Behaviour Research and Therapy*, *44*, 765–784. doi:10.1016/j.brat.2006.02.005

Bruijniks, S. J. E., Bosmans, J., Peeters, F. P. M. L., Hollon, S. D., van Oppen, P., van den Boogaard, M., . . . Huibers, M. J. H. (2015). Frequency and change mechanisms of psychotherapy among depressed patients: Study protocol for a multicenter randomized trial comparing twice-weekly versus once-weekly sessions of CBT and IPT. *BMC Psychiatry*, *15*(1). doi:10.1186/s12888-015-0532-8

Bruijniks, S., Sijbrandij, M., Schlinkert, C., & Huibers, M. (2018). Isolating therapeutic procedures to investigate mechanisms of change in cognitive behavioral therapy for depression An experimental paradigm to investigate how psychotherapy works. *Journal of Experimental Psychopathology*. Advance online publication. doi:10.1177/2043808718800893

Brunet, A., Orr, S. P., Tremblay, J., Robertson, K., Nader, K., & Pitman, R. K. (2008). Effect of post-retrieval propranolol on psychophysiologic responding during subsequent script-driven traumatic imagery in post-traumatic stress disorder. *Journal of Psychiatric Research*, *42*, 503–506. doi:10.1016/j.jpsychires.2007.05.006

Brunoni, A. R., Lopes, M., & Fregni, F. (2008). A systematic review and meta-analysis of clinical studies on major depression and BDNF levels: Implications for the role of neuroplasticity in depression. *The International Journal of Neuropsychopharmacology*, *11*, 1169–1180. doi:10.1017/S1461145708009309

Burkhouse, K. L., Kujawa, A., Kennedy, A. E., Shankman, S. A., Langenecker, S. A., Phan, K. L., & Klumpp, H. (2016). Neural reactivity to reward as a predictor of cognitive behavioral therapy response in anxiety and depression. *Depression and Anxiety*, *33*, 281–288. doi:10.1002/da.22482

Burns, P., Rohrich, R., & Chong, K. (2011). The levels of evidence and their role in evidence-based medicine. *Plastic and Reconstrive Surgery*, *128*, 305–310. doi:10.1097/PRS.0b013e318219c171.The

Campbell, D. G., Felker, B. L., Liu, C. F., Yano, E. M., Kirchner, J. E., Chan, D., . . . Chaney, E. F. (2007). Prevalence of depression-PTSD comorbidity: Implications for clinical practice guidelines and primary care-based interventions. *Journal of General Internal Medicine*, *22*, 711–718. doi:10.1007/s11606-006-0101-4

Carryer, J. R., & Greenberg, L. S. (2010). Optimal levels of emotional arousal in experiential therapy of depression. *Journal of Consulting and Clinical Psychology*, *78*, 190–199. doi:10.1037/a0018401

Chiu, P. H., & Deldin, P. J. (2007). Neural evidence for enhanced error detection in major depressive disorder. *American Journal of Psychiatry*, *164*, 608–616. doi:10.1176/ajp.2007.164.4.608

Connolly, S. L., & Alloy, L. B. (2017). Rumination interacts with life stress to predict depressive symptoms: An ecological momentary assessment study. *Behaviour Research and Therapy*, *97*, 86–95. doi:10.1016/j.brat.2017.07.006

Cougle, J. R., Resnick, H., & Kilpatrick, D. G. (2009). PTSD, depression, and their comorbidity in relation to suicidality: Cross-sectional and prospective analyses of a national probability sample of women. *Depression and Anxiety*, *26*, 1151–1157. doi:10.1002/da.20621

Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, *46*, 5–27. doi:10.1016/j.brat.2007.10.003

Craske, M. G., Treanor, M., Conway, C. C., Zbozinek, T., & Vervliet, B. (2014). Maximizing exposure therapy: An inhibitory learning approach. *Behaviour Research and Therapy*, *58*, 10–23. doi:10.1016/j.brat.2014.04.006

Cristea, I. A., Kok, R. N., & Cuijpers, P. (2015). Efficacy of cognitive bias modification interventions in anxiety

and depression: Meta-analysis. *The British Journal of Psychiatry*, *206*, 7–16. doi:10.1192/bjp.bp.114.146761

Crowther, A., Smoski, M. J., Minkel, J., Moore, T., Gibbs, D., Petty, C., . . . Dichter, G. S. (2015). Resting-state connectivity predictors of response to psychotherapy in major depressive disorder. *Neuropsychopharmacology*, *40*, 1659–1663. doi:10.1038/npp.2015.12

Cuijpers, P., Berking, M., Andersson, G., Quigley, L., Kleiboer, A., & Dobson, K. S. (2013). A meta-analysis of cognitive-behavioural therapy for adult depression, alone and in comparison with other treatments. *Canadian Journal of Psychiatry*, *58*, 376–385.

Cuijpers, P., Huibers, M., Ebert, D. D., Koole, S. L., & Andersson, G. (2013). How much psychotherapy is needed to treat depression? A metaregression analysis. *Journal of Affective Disorders*, *149*, 1–13. doi:10.1016/j.jad.2013.02.030

Cuijpers, P., Karyotaki, E., Weitz, E., Andersson, G., Hollon, S. D., & van Straten, A. (2014). The effects of psychotherapies for major depression in adults on remission, recovery and improvement: A meta-analysis. *Journal of Affective Disorders*, *159*, 118–126. doi:10.1016/j.jad.2014.02.026

Culver, N. C., Stevens, S., Fanselow, M. S., & Craske, M. G. (2017). Building physiological toughness: Some aversive events during extinction may attenuate return of fear. *Journal of Behavior Therapy and Experimental Psychiatry*, *58*, 18–28. doi:10.1016/j.jbtep.2017.07.003

Dalgleish, T., & Yiend, J. (2006). The effects of suppressing a negative autobiographical memory on concurrent intrusions and subsequent autobiographical recall in dysphoria. *Journal of Abnormal Psychology*, *115*, 467–473. doi:10.1037/0021-843X.115.3.467

DelDonno, S. R., Jenkins, L. M., Crane, N. A., Nusslock, R., Ryan, K. A., Shankman, S. A., . . . Langenecker, S. A. (2017). Affective traits and history of depression are related to ventral striatum connectivity. *Journal of Affective Disorders*, *221*, 72–80. doi:10.1016/j.jad.2017.06.014

Deng, W., Aimone, J. B., & Gage, F. H. (2010). New neurons and new memories: How does adult hippocampal neurogenesis affect learning and memory? *Nature Reviews. Neuroscience*, *11*, 339–350. doi:10.1038/nrn2822

DeRubeis, R. J., Cohen, Z. D., Forand, N. R., Fournier, J. C., Gelfand, L. A., & Lorenzo-Luaces, L. (2014). The personalized advantage index: Translating research on prediction into individualized treatment recommendations. A demonstration. *PLOS ONE*, *9*(1), Article e83875. doi:10.1371/journal.pone.0083875

Dichter, G. S., Felder, J. N., Petty, C., Bizzell, J., Ernst, M., & Smoski, M. J. (2009). The effects of psychotherapy on neural responses to rewards in major depression. *Biological Psychiatry*, *66*, 886–897. doi:10.1016/j.biopsych.2009.06.021.The

Dimidjian, S., Barrera, M., Martell, C., Muñoz, R. F., & Lewinsohn, P. M. (2011). The origins and current status of behavioral activation treatments for depression. *Annual Review of Clinical Psychology*, *7*, 1–38. doi:10.1146/annurev-clinpsy-032210-104535

Dimidjian, S., Hollon, S. D., Dobson, K. S., Schmaling, K. B., Kohlenberg, R. J., Addis, M. E., . . . Jacobson, N. S. (2006). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *Journal of Consulting and Clinical Psychology*, *74*, 658–670. doi:10.1037/0022-006X.74.4.658

Disner, S. G., Beevers, C. G., Haigh, E. A., & Beck, A. T. (2011). Neural mechanisms of the cognitive model of depression. *Nature Reviews Neuroscience*, *12*, 467–477. doi:10.1038/nrn3027

Döbrössy, M. D., Drapeau, E., Aurousseau, C., Le Moal, M., Piazza, P. V, & Abrous, D. N. (2003). Differential effects of learning on neurogenesis: learning increases or decreases the number of newly born cells depending on their birth date. *Molecular Psychiatry*, *8*, 974–982. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/14647395

Dobson, K. S., Hollon, S. D., Dimidjian, S., Schmaling, K. B., Kohlenberg, R. J., Gallop, R. J., . . . Jacobson, N. S. (2008). Randomised trial of behavioural activation, cognitive therapy, and antidepressant medication in the prevention of relapse and recurrence in major depression. *Journal of Consulting and Clinical Psychology*, *76*, 468–477. doi:10.1037/0022-006X.76.3.468.

Dong, L., Lee, J. Y., & Harvey, A. G. (2017). Memory support strategies and bundles: a pathway to improving cognitive therapy for depression? *Journal of Consulting & Clinical Psychology*, *85*, 187–199. doi:10.1037/ccp0000167

Dozois, D. J., Bieling, P. J., Patelis-Siotis, I., Hoar, L., Chudzik, S., McCabe, K., & Westra, H. A. (2009). Changes in self-schema structure in cognitive therapy for major depressive disorder: A randomized clinical trial. *Journal of Consulting and Clinical Psychology*, *77*, 1078–1088. doi:10.1037/a0016886

Driessen, E., Cuijpers, P., Hollon, S. D., & Dekker, J. J. M. (2010). Does pretreatment severity moderate the efficacy of psychological treatment of adult outpatient depression? A meta-analysis. *Journal of Consulting and Clinical Psychology*, *78*, 668–680. doi:10.1037/a0020570

Duman, R. S. (2004). Depression: A case of neuronal life and death? *Biological Psychiatry*, *56*, 140–145. doi:10.1016/j.biopsych.2004.02.033

Dunsmoor, J. E., Niv, Y., Daw, N., & Phelps, E. A. (2015). Rethinking extinction. *Neuron*, *88*, 47–63.

Dupret, D., Fabre, A., Döbrössy, M. D., Panatier, A., Rodríguez, J. J., Lamarque, S., . . . Abrous, D. N. (2007). Spatial learning depends on both the addition and removal of new hippocampal neurons. *PLOS Biology*, *5*, 1683–1694.

Ecker, B., Ticic, R., & Hulley, L. (2012). *Unlocking the emotional brain: Eliminating symptoms at their roots using memory reconsolidation*. London: Routledge.

Ehlers, A., Hackmann, A., Grey, N., Wild, J., Liness, S., Albert, I., . . . Clark, D. M. (2014). A randomized controlled trial of 7-day intensive and standard weekly cognitive therapy for ptsd and emotion-focused supportive therapy. *American Journal of Psychiatry*, *171*, 294–304. doi:10.1176/appi.ajp.2013.13040552.A

Ekeblad, A., Falkenstrom, F., & Holmqvist, R. (2015). Reflective functioning as predictor of working alliance and outcome in the treatment of depression. *Journal of Consulting and Clinical Psychology*, *84*, 67–78. doi:10.1037/ccp0000055

Elliott, R., Sahakian, B. J., Michael, A., Paykel, E. S., & Dolan, R. J. (1998). Abnormal neural response to feedback on planning and guessing tasks in patients with unipolar depression. *Psychological Medicine*, *28*, 559–571. doi:10.1017/S0033291798006709

Elliott, R., Zahn, R., Deakin, J., & Anderson, I. (2011). Affective cognition and its disruption in mood disorders. *Neuropsychopharmacology*, *36*, 153–182. doi:10.1038/npp.2010.77

Ellis, A. J., Wells, T. T., Vanderlind, W. M., & Beevers, C. G. (2014). The role of controlled attention on recall in major depression. *Cognition & Emotion*, *28*, 520–529. doi:10.1080/02699931.2013.832153

Elsey, J. W. B., Van Ast, V. A., & Kindt, M. (2018). Human memory reconsolidation: A guiding framework and critical review of the evidence. *Psychological Bulletin*, *144*, 797–848. doi:10.1037/bul0000152

Epp, J. R., Spritzer, M. D., & Galea, L. A. M. (2007). Hippocampus-dependent learning promotes survival of new neurons in the dentate gyrus at a specific time during cell maturation. *Neuroscience*, *149*, 273–285.

Erekson, D. M., Lambert, M. J., & Eggett, D. L. (2015). The relationship between session frequency and psychotherapy outcome in a naturalistic setting. *Journal of Consulting and Clinical Psychology*, *83*, 1097–1107. doi:10.1037/a0039774

Eshel, N., & Roiser, J. P. (2010). Reward and punishment processing in depression. *Biological Psychiatry*, *68*, 118–124. doi:10.1016/j.biopsych.2010.01.027

Falkenström, F., Finkel, S., Sandell, R., Rubel, J., & Holmqvist, R. (2017). Dynamic models of individual change in psychotherapy process research. *Journal of Consulting & Clinical Psychology*, *85*, 537–549. doi:10.1037/ccp0000203

Foa, E. B., McLean, C. P., Zang, Y., Rosenfield, D., Yadin, E., Yarvis, J. S., . . . Peterson, A. L. (2018). Effect of prolonged exposure therapy delivered over 2 weeks vs 8 weeks vs present-centered therapy on PTSD symptom severity in military personnel: A randomized clinical trial. *Journal of the American Medical Association*, *319*, 354–364. doi:10.1001/jama.2017.21242

Forand, N. R., Barnett, J. G., Strunk, D. R., Hindiyeh, M. U., Feinberg, J. E., & Keefe, J. R. (2017). Efficacy of guided iCBT for depression and mediation of change by cognitive skill acquisition. *Behavior Therapy*, *49*, 295–307. doi:10.1016/j.beth.2017.04.004

Forcato, C., Argibay, P. F., Pedreira, M. E., & Maldonado, H. (2009). Human reconsolidation does not always occur when a memory is retrieved: The relevance of the reminder structure. *Neurobiology of Learning and Memory*, *91*, 50–57. doi:10.1016/j.nlm.2008.09.011

Forcato, C., Burgos, V. L., Argibay, P. F., Molina, V. A., Pedreira, M. E., & Maldonado, H. (2007). Reconsolidation of declarative memory in humans. *Learning & Memory*, *14*, 295–303. doi:10.1101/lm.486107.5

Forcato, C., Rodríguez, M. L. C., Pedreira, M. E., & Maldonado, H. (2010). Reconsolidation in humans opens up declarative memory to the entrance of new information. *Neurobiology of Learning and Memory*, *93*, 77–84. doi:10.1016/j.nlm.2009.08.006

Fu, C. H. Y., Williams, S. C. R., Cleare, A. J., Scott, J., Mitterschiffthaler, M. T., Walsh, N. D., . . . Murray, R. M. (2008). Neural responses to sad facial expressions in major depression following cognitive behavioral therapy. *Biological Psychiatry*, *64*, 505–512. doi:10.1016/j.biopsych.2008.04.033

Fuchikami, M., Morinobu, S., Segawa, M., Okamoto, Y., Yamawaki, S., Ozaki, N., . . . Terao, T. (2011). DNA methylation profiles of the brain-derived neurotrophic factor (BDNF) gene as a potent diagnostic biomarker in major depression. *PLOS ONE*, *6*(8), Article e23881. doi:10.1371/journal.pone.0023881

Garratt, G., Ingram, R. E., Rand, K. L., & Sawalani, G. (2007). Cognitive processes in cognitive therapy: Evaluation of the mechanisms of change in the treatment of depression. *Clinical Psychology-Science and Practice*, *14*, 224–239. doi:10.1111/j.1468-2850.2007.00081.x

Gemar, M. C., Segal, Z. V., Sagrati, S., & Kennedy, S. J. (2001). Mood-induced changes on the implicit association test in recovered depressed patients. *Journal of Abnormal Psychology*, *110*, 282–289. doi:10.1037/0021-843X.110.2.282

Gersner, R., Gal, R., Levit, O., Moshe, H., & Zangen, A. (2014). Inherited behaviors, BDNF expression and response to treatment in a novel multifactorial rat model for depression. *The International Journal of Neuropsychopharmacology / Official Scientific Journal of the Collegium Internationale Neuropsychopharmacologicum (CINP)*, *17*, 945–955. doi:10.1017/S1461145714000030

Goldapple, K., Segal, Z., Garson, C., & Lau, M. (2004). Modulation of cortical-limbic pathways in major depression: Treatment-specific effects of cognitive behavior therapy. *Archives of Generaly Psychiatry*, *61*, 34–41. doi:10.1001/archpsyc.61.1.34

Golkar, A., Tjaden, C., & Kindt, M. (2017). Vicarious extinction learning during reconsolidation neutralizes fear memory. *Behaviour Research and Therapy*, *92*, 87–93. doi:10.1016/j.brat.2017.02.004

Gollan, J. K., Hoxha, D., Chihade, D., Pflieger, M. E., Rosebrock, L., & Cacioppo, J. (2014). Frontal alpha EEG asymmetry before and after behavioral activation treatment for depression. *Biological Psychology*, *99*, 198–208. doi:10.1016/j.biopsycho.2014.03.003

Goodkind, M. S., Gallagher-Thompson, D., Thompson, L. W., Kesler, S. R., Anker, L., Flournoy, J., . . . O'Hara, R. M. (2015). The impact of executive function on response to cognitive behavioral therapy in late-life depression. *International Journal of Geriatric Psychiatry*, *31*, 334–339. doi:10.1002/gps.4325

Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: Current status and future directions. *Annual Review of Clinical Psychology*, *6*, 285–312. doi:10.1097/MPG.0b013e3181a15ae8.Screening

Gould, E., Beylin, A., Tanapat, P., Reeves, A., & Shors, T. J. (1999). Learning enhances adult neurogenesis in the hippocampal formation. *Nature Neuroscience*, *2*, 260–265. Retrieved from http://search.ebscohost.com/login.aspx?direct=true&AuthType=ip,uid,url&db=mdc&AN=10195219&lang=cs&site=ehost-live

Gourgouvelis, J., Yielder, P., Clarke, S. T., Behbahani, H., & Murphy, B. A. (2018). Exercise leads to better clinical outcomes in those receiving medication plus cognitive behavioral therapy for major depressive disorder. *Frontiers in Psychiatry*, *9*, 37. doi:10.3389/fpsyt.2018.00037

Gradin, V. B., Kumar, P., Waiter, G., Ahearn, T., Stickle, C., Milders, M., . . . Steele, J. D. (2011). Expected value and prediction error abnormalities in depression and schizophrenia. *Brain*, *134*, 1751–1764.

Green, B. L., Krupnick, J. L., Chung, J., Siddique, J., Kruase, E. D., Revicki, D., . . . Miranda, J. (2006). Impact of PTSD comorbidity on one-year outcomes in a depression trial. *Journal of Clinical Psychology*, *62*, 815–835. doi:10.1002/jclp

Greenberg, L. (1979). Resolving splits: Use of the two chair technique. *Psychotherapy: Theory, Research & Practice*, *16*, 316–324. doi:10.1037/h0085895

Grimm, K. J., An, Y., McArdle, J. J., Zonderman, A. B., & Resnick, S. M. (2012). Recent changes leading to subsequent changes: Extensions of multivariate latent difference score models. *Structural Equation Modeling*, *19*, 268–292. doi:10.1080/10705511.2012.659627

Grosse Holtforth, M., Krieger, T., Zimmermann, J., Altenstein-Yamanaka, D., Dörig, N., Meisch, L., & Hayes, A. M. (2017). A randomized-controlled trial of cognitive-behavioral therapy for depression with integrated techniques from emotion-focused and exposure therapies. *Psychotherapy Research*, *3307*, 1–15. doi:10.1080/10503307.2017.1397796

Groves, J. O. (2007). Is it time to reassess the BDNF hypothesis of depression? *Molecular Psychiatry*, *12*, 1079–1088. doi:10.1038/sj.mp.4002075

Groves, S. J., Porter, R. J., Jordan, J., Knight, R., Carter, J. D., McIntosh, V. V. W., . . . Joyce, P. R. (2015). Changes in neuropsychological function after treatment with metacognitive therapy or cognitive behavior therapy for depression. *Depression and Anxiety*, *32*, 437–444. doi:10.1002/da.22341

Hamaker, E. L., Kuiper, R. M., & Grasman, R. P. P. P. (2015). A critique of the cross-lagged panel model. *Psychological Methods*, *20*, 102–116. doi:10.1037/a0038889

Hankin, B. L., Abramson, L. Y., Miller, N., & Haeffel, G. J. (2004). Cognitive vulnerability-stress theories of depression: Examining affective specificity in the prediction of depression versus anxiety in three prospective studies. *Cognitive Therapy and Research*, *28*, 309–345. doi:10.1023/B:COTR.0000031805.60529.0d

Harvey, A. G., Dong, L., Lee, J. Y., Gumport, N. B., Hollon, S. D., Rabe-Hesketh, S., . . . Armstrong, C. C. (2017). Can integrating the memory support intervention into cognitive therapy improve depression outcome? Study protocol for a randomized controlled trial. *Trials*, *18*, 539. doi:10.1186/s13063-017-2276-x

Harvey, A. G., Lee, J., Williams, J., Hollon, S. D., Walker, M. P., Thompson, M. A., & Smith, R. (2014). Improving outcome of psychosocial treatments by enhancing memory and learning. *Perspectives on Psychological Science: A Journal of the Association for Psychological Science*, *9*, 161–179. doi:10.1177/1745691614521781

Harvey, A. G., Watkins, E., Mansell, W., & Shafran, R. (2004). *Cognitive behavioural processes across psychological disorders: A transdiagnostic approach to research and treatment*. New York, NY: Oxford University Press.

Hayes, A. M., Feldman, G. C., Beevers, C. G., Laurenceau, J. P., Cardaciotto, L. A., & Lewis-Smith, J. (2007). Discontinuities and cognitive changes in an exposure-based cognitive therapy for depression. *Journal of Consulting and Clinical Psychology*, *75*, 409–421. doi:10.1037/0022-006X.75.3.409

Heatherton, T. F., & Wagner, D. D. (2011). Cognitive neuroscience of self-regulation failure. *Trends in Cognitive Sciences*, *15*, 132–139. doi:10.1016/j.tics.2010.12.005

Herbert, J. D., Rheingold, A. A., Gaudiano, B. A., & Myers, V. H. (2004). Standard versus extended cognitive behavior therapy for social anxiety disorder. *Behavioural and Cognitive Psychotherapy*, *32*, 131–147. doi:10.1017/S1352465804001171

Hofmann, S. G., Asmundson, G. J. G., & Beck, A. T. (2013). The science of cognitive therapy. *Behavior Therapy*, *44*, 199–212. doi:10.1016/j.beth.2009.01.007

Hollon, S. D. (1999). Rapid early response in cognitive behavior therapy: A commentary. *Clinical Psychology: Science and Practice*, *6*, 305–309.

Holmes, A. J., & Pizzagalli, D. A. (2008). Spatiotemporal dynamics of error processing dysfunctions in major depressive disorder. *Archives of General Psychiatry*, *65*, 179–188. doi:10.1001/archgenpsychiatry.2007.19

Huibers, M. J., & Cuijpers, P. (2014). Common (nonspecific) factors in psychotherapy. In R. L. Cautin & S. O. Lilienfeld (Eds.), *The encyclopedia of clinical psychology* (pp. 1–6). Chichester, England: John Wiley & Sons. doi:10.1002/9781118625392.wbecp272

Huibers, M. J. H., Cohen, Z. D., Lemmens, L. H. J. M., Arntz, A., Peeters, F. P. M. L., Cuijpers, P., & DeRubeis, R. J. (2015). Predicting optimal outcomes in cognitive therapy or interpersonal psychotherapy for depressed individuals using the personalized advantage index approach. *PLOS ONE*, *10*(11), Article e140771. doi:10.1371/journal.pone.0140771

Hummel, J., Weisbrod, C., Boesch, L., Himpler, K., Hauer, K., Hautzinger, M., . . . Kopf, D. (2017). AIDE–acute illness and depression in elderly patients. Cognitive behavioral group psychotherapy in geriatric patients with comorbid depression: A randomized, controlled trial. *Journal of the American Medical Directors Association*, *18*, 341–349. doi:10.1016/j.jamda.2016.10.009

Hvenegaard, M., Watkins, E. R., Poulsen, S., Rosenberg, N. K., Gondan, M., Grafton, B., . . . Moeller, S. B. (2015). Rumination-focused cognitive behaviour therapy vs. cognitive behaviour therapy for depression: Study protocol for a randomised controlled superiority trial. *Trials*, *16*, 1–6. doi:10.1186/s13063-015-0875-y

Jacobson, N. S., Dobson, K. S., Truax, P. A., Addis, M. E., Koerner, K., Gollan, J. K., . . . Prince, S. E. (1996). A component analysis of cognitive-behavioral treatment for depression. *Journal of Consulting and Clinical Psychology*, *64*, 295–304.

Johnco, C., Wuthrich, V. M., & Rapee, R. M. (2015). The impact of late-life anxiety and depression on cognitive

flexibility and cognitive skill acquisition. *Depression and Anxiety*, *32*, 754–762. doi:10.1002/da.22375

Joormann, J., & Gotlib, I. H. (2008). Updating the contents of working memory in depression: Interference from irrelevant negative material. *Journal of Abnormal Psychology*, *117*, 182–192. doi:10.1037/0021-843X.117.1.182

Kanter, J. W., Manos, R. C., Bowe, W. M., Baruch, D. E., Busch, A. M., & Rusch, L. C. (2010). What is behavioral activation?. A review of the empirical literature. *Clinical Psychology Review*, *30*, 608–620. doi:10.1016/j.cpr.2010.04.001

Karege, F., Perret, G., Bondolfi, G., Schwald, M., Bertschy, G., & Aubry, J.-M. (2002). Decreased serum brain-derived neurotrophic factor levels in major depressed patients. *Psychiatry Research*, *109*, 143–148. doi:10.1016/S0165-1781(02)00005-7

Keefe, J. R., & DeRubeis, R. J. (2015). A critique of theoretical models of depression: commonalties and distinctive features. In A. Wells & P. Fisher (Eds.), *Treating depression: MCT, CBT, and third tave therapies* (pp. 242–264). Chichester, England: John Wiley & Sons.

Kennedy, S. H., Konarski, J. Z., Segal, Z. V., Lau, M. A., Bieling, P. J., McIntyre, R. S., & Mayberg, H. S. (2007). Differences in brain glucose metabolism between responders to CBT and venlafaxine in a 16-week randomized controlled trial. *American Journal of Psychiatry*, *164*, 778–788.

Kindt, M., Soeter, M., & Vervliet, B. (2009). Beyond extinction: Erasing human fear responses and preventing the return of fear. *Nature Neuroscience*, *12*, 256–258. doi:10.1038/nn.2271

Knutson, B., Bhanji, J. P., Cooney, R. E., Atlas, L. Y., & Gotlib, I. H. (2008). Neural responses to monetary incentives in major depression. *Biological Psychiatry*, *63*, 686–692. doi:10.1016/j.biopsych.2007.07.023

Kofman, O., Meiran, N., Greenberg, E., Balas, M., & Cohen, H. (2006). Enhanced performance on executive functions associated with examination stress: Evidence from task-switching and Stroop paradigms. *Cognition and Emotion*, *20*, 577–595. Retrieved from http://www.bgu.ac.il/%7B~%7Dnmeiran/index%7B_%7Dfiles/Kofman%5Cn2006.pdf

Köhler, C. A., Carvalho, A. F., Alves, G. S., McIntyre, R. S., Hyphantis, T. N., & Cammarota, M. (2015). Autobiographical memory disturbances in depression: A novel therapeutic target? *Neural Plasticity*, *2015*, 759139. doi:10.1155/2015/759139

Kuyken, W., Watkins, E., Holden, E., White, K., Taylor, R. S., Byford, S., . . . Dalgleish, T. (2010). How does mindfulness-based cognitive therapy work? *Behaviour Research and Therapy*, *48*, 1105–1112. doi:10.1016/j.brat.2010.08.003

Kwon, S. M., & Oei, T. P. S. (1994). The roles of two levels of cognitions in the development, maintenance, and treatment of depression. *Clinical Psychology Review*, *14*, 331–358. doi:/10.1016/0272-7358(94)90032-9

Kwon, S.-M., & Oei, T. P. S. (2003). Cognitive change processes in a group cognitive behavior therapy of depression. *Journal of Behavior Therapy and Experimental Psychiatry*, *34*, 73–85. doi:10.1016/S0005-7916(03)00021-1

Lambourne, K., & Tomporowski, P. (2010). The effect of exercise-induced arousal on cognitive task performance: A meta-regression analysis. *Brain Research*, *1341*, 12–24.

Lane, R. D., Ryan, L., Nadel, L., & Greenberg, L. (2015). Memory reconsolidation, emotional arousal, and the process of change in psychotherapy: New insights from brain science. *Behavioral and Brain Sciences*, *38*, 1–64. doi:10.1017/S0140525X14000041

Lang, T. J., Blackwell, S. E., Harmer, C. J., Davison, P., & Holmes, E. A. (2012). Cognitive bias modification using mental imagery for depression: Developing a novel computerized intervention to change negative thinking styles. *European Journal of Personality*, *26*, 145–157. doi:10.1002/per

Lang, T. J., Moulds, M. L., & Holmes, E. A. (2009). Reducing depressive intrusions via a computerized cognitive bias modification of appraisals task: Developing a cognitive vaccine. *Behaviour Research and Therapy*, *47*, 139–145. doi:10.1016/j.brat.2008.11.002

Lemmens, L. H. J. M., Arntz, A., Peeters, F. P. M. L., Hollon, S. D., Roefs, A., & Huibers, M. J. H. (2015). Clinical effectiveness of cognitive therapy v. interpersonal psychotherapy for depression: Results of a randomized controlled trial. *Psychological Medicine*, *45*, 2095–2110.

Lemmens, L. H. J. M., Galindo-Garre, F., Arntz, A., Peeters, F., Hollon, S. D., DeRubeis, R. J., & Huibers, M. J. H. (2017). Exploring mechanisms of change in cognitive therapy and interpersonal psychotherapy for adult depression. *Behaviour Research and Therapy*, *94*, 81–92. doi:10.1016/j.brat.2017.05.005

Lemmens, L. H. J. M., Müller, V. N. L. S., Arntz, A., & Huibers, M. J. (2016). Mechanisms of change in psychotherapy for depression: An empirical update and evaluation of research aimed at identifying psychological mediators. *Clinical Psychology Review*, *50*, 95–107. doi:10.1016/j.cpr.2016.09.004

Lemmens, L. H. J. M., van Bronswijk, S. C., Peeters, F., Arntz, A., Hollon, S. D., & Huibers, M. J. H. (2019). Long-term outcomes of acute treatment with cognitive therapy vs. interpersonal psychotherapy for adult depression: Follow-up of a randomized controlled trial. *Psychological Medicine*, *49*, 465–473.

Lener, M. S., & Losifescu, D. V. (2015). In pursuit of neuroimaging biomarkers to guide treatment selection in major depressive disorder: A review of the literature. *Annals of the New York Academy of Sciences*, *1344*, 50–65. doi:10.1111/nyas.12759

Lester, K. J., Mathews, A., Davison, P. S., Burgess, J. L., & Yiend, J. (2011). Modifying cognitive errors promotes cognitive well being: A new approach to bias modification. *Journal of Behavior Therapy and Experimental Psychiatry*, *42*, 298–308.

Levin, R. L., Heller, W., Mohanty, A., Herrington, J. D., & Miller, G. A. (2007). Cognitive deficits in depression and functional specificity of regional brain activity. *Cognitive Therapy and Research*, *31*, 211–233. doi:10.1007/s10608-007-9128-z

Longmore, R. J., & Worrell, M. (2007). Do we need to challenge thoughts in cognitive behavior therapy? *Clinical Psychology Review*, *27*, 173–187. doi:10.1016/j.cpr.2006.08.001

Lorenzo-Luaces, L., German, R. E., & DeRubeis, R. J. (2015). It's complicated: The relation between cognitive change procedures, cognitive change, and symptom change in cognitive therapy for depression. *Clinical Psychology Review*, *41*, 3–15. doi:10.1016/j.cpr.2014.12.003

Lorenzo-Luaces, L., Keefe, J. R., & DeRubeis, R. J. (2016). Cognitive-behavioral therapy: nature and relation to non-cognitive behavioral therapy. *Behavior Therapy*, *47*, 785–803. doi:10.1016/j.beth.2016.02.012

MacLeod, C., & Grafton, B. (2016). Anxiety-linked attentional bias and its modification: Illustrating the importance of distinguishing processes and procedures in experimental psychopathology research. *Behaviour Research and Therapy*, *86*, 68–86. doi:10.1016/j.brat.2016.07.005

MacQueen, G. M., Galway, T. M., Hay, J., Young, L. T., & Joffe, R. T. (2002). Recollection memory deficits in patients with major depressive disorder predicted by past depressions but not current mood state or treatment status. *Psychological Medicine*, *32*, 251–258. doi:10.1017/S0033291701004834

Maier, S. F., Amat, J., Baratta, M. V., Paul, E., & Watkins, L. R. (2006). Behavioral control, the medial prefrontal cortex, and resilience. *Dialogues in Clinical Neuroscience*, *8*, 397–406.

Manos, R. C., Kanter, J. W., & Busch, A. M. (2010). A critical review of assessment strategies to measure the behavioral activation model of depression. *Clinical Psychology Review*, *30*, 547–561. doi:10.1016/j.cpr.2010.03.008

McCabe, C., Cowen, P. J., & Harmer, C. J. (2009). Neural representation of reward in recovered depressed patients. *Psychopharmacology*, *205*, 667–677. doi:10.1007/s00213-009-1573-9

McClintock, S. M., Husain, M. M., Greer, T. L., & Cullum, C. M. (2010). Association between depression severity and neurocognitive function in major depressive disorder: A review and synthesis. *Neuropsychology*, *24*, 9–34. doi:10.1037/a0017336

McKinnon, M. C., Yucel, K., Nazarov, A., & MacQueen, G. M. (2008). A meta-analysis examining clinical predictors of hippocampal volume in patients with major depressive disorder. *Journal of Psychiatry & Neuroscience*, *34*, 41–54. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2612082&tool=pmcentrez&rendertype=abstract%5Cnhttp://www.ncbi.nlm.nih.gov/pubmed/19125212%5Cnhttp://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC2612082

Meyer, J. H., Houle, S., Sagrati, S., Carella, A., Hussey, D. F., Ginovart, N., . . . Wilson, A. (2004). Brain serotonin transporter binding potential measured with carbon 11–labeled DASB positron emission tomography. *Archives of General Psychiatry*, *61*, 1271–1279. doi:10.1001/archpsyc.61.12.1271

Moratti, S., Rubio, G., Campo, P., Keil, A., & Ortiz, T. (2008). Hypofunction of right temporoparietal cortex during emotional arousal in depression. *Archives of General Psychiatry*, *65*, 532–541. doi:65/5/532[pii]\r10.1001/archpsyc.65.5.532

Nader, K., & Einarsson, E. Ö. (2010). Memory reconsolidation: An update. *Annals of the New York Academy of Sciences*, *1191*, 27–41. doi:10.1111/j.1749-6632.2010.05443.x

Nader, K., Schafe, G. E., & LeDoux, J. E. (2000). The labile nature of consolidation theory. *Nature Reviews. Neuroscience*, *1*, 216–219. doi:10.1038/35044580

Nanni, V., Uher, R., & Danese, A. (2012). Childhood maltreatment predicts unfavorable course of illness and treatment outcomes in depression: A meta-analysis. *The American Journal of Psychiatry*, *169*, 141–151. doi:10.1176/appi.ajp.2011.11020335

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology*, *109*, 504–511. doi:10.1037/0021-843X.109.3.504

Nutt, D. J. (2008). Relationships of neurotransmittors to the symptoms of major depressive disorder. *Journal of Clinical Psychiatry*, *69*(Suppl E), 4–7.

OCEBM Levels of Evidence Working Group. (2011). *The Oxford levels of evidence*. Retrieved from https://www.cebm.net/2016/05/ocebm-levels-of-evidence/

Pearson, R. M., O'Mahen, H., Burns, A., Bennert, K., Shepherd, C., Baxter, H., . . . Evans, J. (2013). The normalisation of disrupted attentional processing of infant distress in depressed pregnant women following cognitive behavioural therapy. *Journal of Affective Disorders*, *145*, 208–213. doi:10.1016/j.jad.2012.07.033

Pizzagalli, D. A., Holmes, A. J., Dillon, D. G., Goetz, E. L., Birk, J. L., Bogdan, R., . . . Fava, M. (2009). Reduced caudate and nucleus accumbens response to rewards in unmedicated subjects with major depressive disorder. *American Journal of Psychiatry*, *166*, 702–710. doi:10.1176/appi.ajp.2008.08081201.Reduced

Quilty, L. C., McBride, C., & Bagby, R. M. (2008). Evidence for the cognitive mediational model of cognitive behavioural therapy for depression. *Psychological Medicine*, *38*, 1531–1541. doi:10.1017/S0033291708003772

Reardon, M. L., Cukrowicz, K. C., Reeves, M. D., & Joiner, T. E. (2002). Duration and regularity of therapy attendance as predictors of treatment outcome in an adult outpatient population. *Psychotherapy Research*, *12*, 273–285. doi:10.1080/713664390

Reese, R. J., Toland, M. D., & Hopkins, N. B. (2011). Replicating and extending the good-enough level model of change: Considering session frequency. *Psychotherapy Research*, *21*, 608–619. doi:10.1080/10503307.2011.598580

Reynolds, M., & Brewin, C. R. (1999). Intrusive memories in depression and posttraumatic stress disorder. *Behaviour Research and Therapy*, *37*, 201–215. doi:10.1016/S0005-7967(98)00132-6

Richards, D. A., Ekers, D., McMillan, D., Taylor, R. S., Byford, S., Warren, F. C., . . . Finning, K. (2016). Cost and outcome of behavioural activation versus cognitive behavioural therapy for depression (COBRA): A randomised, controlled, non-inferiority trial. *The Lancet*, *388*, 871–880. doi:10.1016/S0140-6736(16)31140-0

Ritchey, M., Dolcos, F., Eddington, K., Strauman, T., & Cabezza, R. (2011). Neural correlates of emotional processing in depression: Changes with cognitive behavioral therapy and predictors of treatment response. *Journal of Psychiatric Research*, *45*, 577–587. doi:10.1016/j.jpsychires.2010.09.007.Neural

Robinson, M. S., & Alloy, L. B. (2003). Negative cognitive styles and stress-reactive rumination interact to predict depression: A prospective study. *Cognitive Therapy and Research*, *27*, 275–291. doi:10.1023/A:1023914416469

Romero, N., Sanchez, A., & Vazquez, C. (2014). Memory biases in remitted depression: The role of negative cognitions at explicit and automatic processing levels. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*, 128–135. doi:10.1016/j.jbtep.2013.09.008

Rubin-Falcone, H., Weber, J., Kishon, R., Ochsner, K., Delaparte, L., Doré, B., . . . Miller, J. M. (2018). Longitudinal effects of cognitive behavioral therapy for depression on the neural correlates of emotion regulation. *Psychiatry Research: Neuroimaging*, *271*, 82–90. doi:10.1016/j.pscychresns.2017.11.002

Ruchsow, M., Herrnberger, B., Wiesend, C., Grön, G., Spitzer, M., & Kiefer, M. (2004). The effect of erroneous responses on response monitoring in patients with major depressive disorder: A study with event-related potentials. *Psychophysiology*, *41*, 833–840. doi:10.1111/j.1469-8986.2004.00237.x

Salehi, B., Cordero, M. I., & Sandi, C. (2010). Learning under stress: The inverted-U-shape function revisited. *Learning & Memory*, *17*, 522–530.

Samoilov, A. (2000). Role of emotion in cognitive-behavior therapy. *Clinical Psychological Science and Practice*, *7*, 373–385. doi:10.1093/clipsy.7.4.373

Sankar, A., Scott, J., Paszkiewicz, A., Giampietro, V. P., Steiner, H., & Fu, C. H. Y. (2015). Neural effects of cognitive-behavioural therapy on dysfunctional attitudes in depression. *Psychological Medicine*, *45*, 1425–1433. doi:10.1017/S0033291714002529

Santesso, D. L., Steele, K. T., Bogdan, R., Holmes, A. J., Christen, M., Meites, T. M., . . . Behavior, M. (2011). Enhanced negative feedback responses in remitted depression. *NeuroReport*, *19*, 1045–1048. doi:10.1097/WNR.0b013e3283036e73

Sasso, K. E., Strunk, D. R., Braun, J. D., DeRubeis, R. J., & Brotman, M. A. (2016). A re-examination of process-outcome relations in cognitive therapy for depression: Disaggregating within-patient and between-patient effects. *Psychotherapy Research*, *26*, 387–398. doi:10.1080/10503307.2015.1026423

Scher, C. D., Ingram, R. E., & Segal, Z. V. (2005). Cognitive reactivity and vulnerability: Empirical evaluation of construct activation and cognitive diatheses in unipolar depression. *Clinical Psychology Review*, *25*, 487–510. doi:10.1016/j.cpr.2005.01.005

Schiller, D., Monfils, M.-H., Raio, C. M., Johnson, D. C., Ledoux, J. E., & Phelps, E. A. (2010). Preventing the return of fear in humans using reconsolidation update mechanisms. *Nature*, *463*(7277), 49–53. doi:10.1038/nature08637

Schwabe, L., & Wolf, O. T. (2009). New episodic learning interferes with the reconsolidation of autobiographical memories. *PLOS ONE*, *4*(10), Article e7519. doi:10.1371/journal.pone.0007519

Segal, Z. V., Gemar, M., & Williams, S. (1999). Differential cognitive response to a mood challenge following successful cognitive therapy or pharmacotherapy for unipolar depression. *Journal of Abnormal Psychology*, *108*, 3–10. doi:10.1037/0021-843X.108.1.3

Segal, Z., Kennedy, S., Gemar, M., Hood, K., Pedersen, R., & Buis, T. (2006). Cognitive reactivity to sad mood provocation and the prediction of depressive relapse. *Archives of General Psychiatry*, *63*, 749–755. doi:10.1001/archpsyc.63.7.749

Seminowicz, D. A., Mayberg, H. S., McIntosh, A. R., Goldapple, K., Kennedy, S., Segal, Z., & Rafi-Tari, S. (2004). Limbic-frontal circuitry in major depression: A path modeling metanalysis. *NeuroImage*, *22*, 409–418. doi:10.1016/j.neuroimage.2004.01.015

Sevenster, D., Beckers, T., & Kindt, M. (2012). Retrieval per se is not sufficient to trigger reconsolidation of human fear memory. *Neurobiology of Learning and Memory*, *97*, 338–345. doi:10.1016/j.nlm.2012.01.009

Shekelle, P. G., Maglione, M. A., Luoto, J., Johnsen, B., & Perry, T. R. (2013). NHMRC evidence hierarchy: Designations of "levels of evidence" according to type of research question. In *Global Health Evidence Evaluation*, (Table B.9). Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK121300/table/appb.t21itle

Sheppard, L. C., & Teasdale, J. D. (2000). Dysfunctional thinking in major depressive disorder: A deficit in metacognitive monitoring? *Journal of Abnormal Psychology*, *109*, 768–776. doi:10.1037/0021-843X.109.4.768

Sheppard, L. C., & Teasdale, J. D. (2004). How does dysfunctional thinking decrease during recovery from major depression? *Journal of Abnormal Psychology*, *113*, 64–71. doi:10.1037/0021-843X.113.1.64

Shimizu, E., Hashimoto, K., Okamura, N., Koike, K., Komatsu, N., Kumakiri, C., . . . Iyo, M. (2003). Alterations of serum levels of brain-derived neurotrophic factor (BDNF) in depressed patients with or without antidepressants. *Biological Psychiatry*, *54*, 70–75. doi:10.1016/S0006-3223(03)00181-1

Shou, H., Yang, Z., Satterthwaite, T. D., Cook, P. A., Bruce, S. E., Shinohara, R. T., & Sheline, Y. I. (2017). Cognitive behavioral therapy increases amygdala connectivity with the cognitive control network in both MDD and PTSD. *NeuroImage: Clinical*, *14*, 464–470. doi:10.1016/j.nicl.2017.01.030

Siegle, G. J., Carter, C. S., & Thase, M. E. (2006). Use of fMRI to predict recovery from unipolar depression with cognitive behavior therapy. *American Journal of Psychiatry*, *163*, 735–738. doi:10.1176/appi.ajp.163.4.735

Snyder, H. R. (2013). Major depressive disorder is associated with braod impairments on neuropsychological measures of evecutive function: A meta-analysis and

review. *Psychological Bulletin*, *139*, 81–132. doi:10.1037/a0028727.Major

Song, J. W., & Chung, K. C. (2010). Observational studies: Cohort and case-control studies. *Plastic and Reconstructive Surgery*, *126*, 2234–2242. doi:10.1097/PRS.0b013e3181f44abc.Observational

Spinhoven, P., Bockting, C. L. H., Schene, A. H., Koeter, M. W. J., Wekking, E. M., & Williams, J. M. G. (2006). Autobiographical memory in the euthymic phase of recurrent depression. *Journal of Abnormal Psychology*, *115*, 590–600. doi:10.1037/0021-843X.115.3.590

Stange, J. P., MacNamara, A., Barnas, O., Kennedy, A. E., Hajcak, G., Phan, K. L., & Klumpp, H. (2017). Neural markers of attention to aversive pictures predict response to cognitive behavioral therapy in anxiety and depression. *Biological Psychology*, *123*, 269–277. doi:10.1016/j.biopsycho.2016.10.009

Stange, J. P., MacNamara, A., Kennedy, A. E., Hajcak, G., Phan, K. L., & Klumpp, H. (2017). Brain-behavioral adaptability predicts response to cognitive behavioral therapy for emotional disorders: A person-centered event-related potential study. *Neuropsychologia*. Advance online publication. doi:10.1016/j.neuropsychologia.2017.06.027

Stout, D. M., Acheson, D. T., Moore, T. M., Gur, R. C., Baker, D. G., Geyer, M. A., & Risbrough, V. B. (2018). Individual variation in working memory is associated with fear extinction performance. *Behaviour Research and Therapy*, *102*, 52–59. doi:10.1016/j.brat.2018.01.002

Strunk, D. R., DeRubeis, R. J., Chiu, A. W., & Alvarez, J. (2007). Patients' competence in and performance of cognitive therapy skills: Relation to the reduction of relapse risk following treatment for depression. *Journal of Consulting and Clinical Psychology*, *75*, 523–530. doi:10.1037/0022-006X.75.4.523

Strunk, D. R., Hollars, S. N., Adler, A. D., Goldstein, L. A., & Braun, J. D. (2014). Assessing patients' cognitive therapy skills: Initial evaluation of the Competencies of Cognitive Therapy Scale. *Cognitive Therapy and Research*, *38*, 559–569. doi:10.1007/s10608-014-9617-9

Sumner, J. A., Griffith, J. W., & Mineka, S. (2010). Overgeneral autobiographical memory as a predictor of the course of depression: A meta-analysis. *Behaviour Research and Therapy*, *48*, 614–625. doi:10.1016/j.pestbp.2011.02.012. Investigations

Sutherland, K., & Bryant, R. A. (2007). Rumination and overgeneral autobiographical memory. *Behaviour Research and Therapy*, *45*, 2407–2416. doi:10.1016/j.brat.2007.03.018

Tadić, A., Wagner, S., Schlicht, K. F., Peetz, D., Borysenko, L., Dreimüller, N., . . . Lieb, K. (2011). The early non-increase of serum BDNF predicts failure of antidepressant treatment in patients with major depression: A pilot study. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *35*, 415–420. doi:10.1016/j.pnpbp.2010.08.011

Taft, C. T., Resick, P. A., Watkins, L. E., & Panuzio, J. (2009). An investigation of posttraumatic stress disorder and depressive symptomatology among female victims of interpersonal trauma. *Journal of Family Violence*, *24*, 407–415. doi:10.1007/s10896-009-9243-6

Tavares, J. V. T., Clark, L., Furey, M. L., Williams, G. B., Sahakiana, B. J., & Drevets, W. C. (2008). *NeuroImage*, *42*, 1118–1126. doi:10.1016/j.neuroimage.2008.05.049. Neural

Teasdale, J. D., & Fennell, M. J. V. (1982). Immediate effects on depression of cognitive therapy interventions. *Cognitive Therapy and Research*, *6*, 343–352.

Teasdale, J. D., Moore, R. G., Hayhurst, H., Pope, M., Williams, S., & Segal, Z. V. (2002). Metacognitive awareness and prevention of relapse in depression: Empirical evidence. *Journal of Consulting and Clinical Psychology*, *70*, 275–287. doi:10.1037/0022-006X.70.2.275

Thompson, D. G., Kesler, S. R., Sudheimer, K., Mehta, K. M., Thompson, L. W., Marquett, R. M., . . . O'Hara, R. M. (2015). FMRI activation during executive function predicts response to cognitive behavioral therapy in older, depressed adults. *American Journal of Geriatric Psychiatry*, *23*, 13–22. doi:10.1016/j.jagp.2014.02.001

Tronson, N., & Tayler, J. (2007). Molecular mechanisms of memory reconsolidation. *Nature Reviews. Neuroscience*, *8*, 262–275. doi:10.1038/nrn2090

Tucker, D. M., Luu, P., Frishkoff, G., Quiring, J., & Poulsen, C. (2003). Frontolimbic Response to negative feedback in clinical depression. *Journal of Abnormal Psychology*, *112*, 667–678. doi:10.1037/0021-843X.112.4.667

Wang, X., Zhou, X., Dai, Q., Ji, B., & Feng, Z. (2017). The role of motivation in cognitive reappraisal for depressed patients. *Frontiers in Human Neuroscience*, *11*, 1–12. doi:10.3389/fnhum.2017.00516

Waters, A. M., Kershaw, R., & Lipp, O. V. (2018). Multiple fear-related stimuli enhance physiological arousal during extinction and reduce physiological arousal to novel stimuli and the threat conditioned stimulus. *Behaviour Research and Therapy*, *106*, 28–36. doi:10.1016/j.brat.2018.04.005

Webb, C. A., Olson, E. A., Killgore, W. D. S., Pizzagalli, D. A., Rauch, S. L., & Rosso, I. M. (2018). Rostral anterior cingulate cortex morphology predicts treatment response to internet-based cognitive behavioral therapy for depression. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *3*, 255–262. doi:10.1016/j.bpsc.2017.08.005

Wenzlaff, R. M., & Bates, D. E. (1998). Unmasking a cognitive vulnerability to depression: How lapses in mental control reveal depressive thinking. *Journal of Personality and Social Psychology*, *75*, 1559–1571. doi:10.1037/0022-3514.75.6.1559

Werner, F.-M., & Coveñas, R. (2010). Classical neurotransmitters and neuropeptides involved in major depression in a multi-neurotransmitter system: A focus on antidepressant drugs. *International Journal of Neuroscience*, *120*, 455–470. doi:10.4172/2157-7099.1000266

Whisman, M. A., Miller, I. W., Norman, W. H., & Keitner, G. I. (1991). Cognitive therapy with depressed inpatients: specific effects on dysfunctional cognitions. *Journal of Consulting and Clinical Psychology*, *59*, 282–288. doi:10.1037/0022-006X.59.2.282

Wiers, R. W., Boffo, M., & Field, M. (2018). What's in a trial? On the importance of distinguishing between experimental lab studies and randomized controlled trials: the case

of cognitive bias modification and alcohol use disorders. *Journal of Studies on Alcohol and Drugs*, *79*, 333–343.

Wiersma, J. E., Hovens, J. G. F. M., Van Oppen, P., Giltay, E. J., Van Schaik, D. J. F., Beekman, A. T. F., & Penninx, B. W. J. H. (2009). The importance of childhood trauma and childhood life events for chronicity of depression in adults. *Journal of Clinical Psychiatry*, *70*, 983–989. doi:10.4088/JCP.08m04521

Williams, A. D., & Moulds, M. L. (2007). Cognitive avoidance of intrusive memories: Recall vantage perspective and associations with depression. *Behaviour Research and Therapy*, *45*, 1141–1153. doi:10.1016/j.brat.2006.09.005

Yang, X. H., Huang, J., Lan, Y., Zhu, C. Y., Liu, X. Q., Wang, Y. F., . . . Chan, R. C. (2015). Diminished caudate and superior temporal gyrus responses to effort-based decision making in patients with first-episode major depressive disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *64*, 52–59. doi:10.1016/j.pnpbp.2015.07.006

Yang, Z., Oathes, D., Linn, K. A., Bruce, S. E., Satterthwaite, T. D., Cook, P. A., . . . Sheline, Y. I. (2018). Cognitive behavioral therapy is associated with enhanced cognitive control network activity in major depression and posttraumatic stress disorder. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *3*, 311–319. doi:10.1016/j.bpsc.2017.12.006

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology and Psychology*, *18*, 459–482.

Yokoyama, S., Okamoto, Y., Takagaki, K., Okada, G., Takamura, M., Mori, A., . . . Yamawaki, S. (2017). Effects of behavioral activation on default mode network connectivity in subthreshold depression: A preliminary resting-state fMRI study. *Journal of Affective Disorders*, *227*, 156–163. doi:10.1016/j.jad.2017.10.021

Yoshimura, S., Okamoto, Y., Matsunaga, M., Onoda, K., Okada, G., Kunisato, Y., . . . Yamawaki, S. (2017). Cognitive behavioral therapy changes functional connectivity between medial prefrontal and anterior cingulate cortices. *Journal of Affective Disorders*, *208*, 610–614. doi:10.1016/j.jad.2016.10.017

Young, J. E., Klosko, J. S., Weishaar, M. E., & Temple, S. D. (2003). *Schema therapy: A practitioner's guide*. New York, NY: Guilford Press.

Zettle, R. D., & Rains, J. C. (1989). Group cognitive and contextual therapies in treatment of depression. *Journal of Clinical Psychology*, *45*, 436–445. doi:10.1002/1097-4679(198905)45:3<436::AID-JCLP2270450314>3.0.CO;2-L

Zilcha-Mano, S., Lipsitz, I., & Errázuriz, P. (2018). When is it effective to focus on the alliance? Analysis of a within-client moderator. *Cognitive Therapy and Research*, *42*(159–171). doi:10.1007/s10608-017-9867-4

Zilcha-Mano, S., Muran, J. C., Eubanks, C. F., Safran, J. D., & Winston, A. (2017). Not just a non-specific factor: Moderators of the effect of within- and between-clients alliance on outcome in CBT. *Cognitive Therapy and Research*, *42*, 159–171. doi:10.1007/s10608-017-9866-5