cognitive neuroscience are extremely welcome. It is likely that the many varieties of transformation that patients undergo will never be explained by any one mechanism, whether reconsolidation or retrieval competition. Basic research on memory change does, however, constitute one of the most promising lines of enquiry presently available, and findings from both animal and human research have the capacity to deliver new insights into what makes therapy effective. Although I agree with the authors that different forms of memory, such as semantic and episodic memory, are strongly interlinked, this does not mean that they are all equally important in different types of psychopathology. There are also perceptual forms of memory that appear to be distinct from episodic memory and may become relatively disconnected within conditions such as posttraumatic stress disorder (Brewin 2014; Brewin et al. 2010). It seems highly likely that our understanding of different change mechanisms needs to develop in tandem with a more differentiated view of human memory systems.

Clinical applications of counterfactual thinking during memory reactivation

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Felipe De Brigard[a,b,c] and Eleanor Hanna[b,c]
[a]*Department of Philosophy, Duke University, Durham, NC 27708;* [b]*Center for Cognitive Neuroscience, Duke University, Durham, NC 27708;* [c]*Duke Institute for Brain Sciences, Duke University, Durham, NC 27708.*
felipe.debrigard@duke.edu **eleanor.hanna@duke.edu**
www.felipedebrigard.com

Abstract: The Integrative Memory Model offers a strong foundation upon which to build successful strategies for clinical intervention. The next challenge is to figure out which cognitive strategies are more likely to bring about successful and beneficial modifications of reactivated memories during therapy. In this commentary we suggest that exercising emotional regulation during episodic counterfactual thinking is likely to be a successful therapeutic strategy to bring about beneficial memory modifications.

Without a doubt, the Integrative Memory Model (IMM) offered by Lane et al. constitutes a parsimonious and elegant framework in which to understand the affective and cognitive processes underlying therapeutic success. Moreover, if we consider the therapeutic session a suitable context for reactivating injurious memory traces and modifying them into healthier ones after reconsolidation, then we have a strong foundation on which to build successful strategies for therapeutic intervention. The next task for researchers and therapists is to identify the cognitive processes that are more likely to generate successful and beneficial modifications of reactivated memories during therapy. In this commentary we want to put forth the hypothesis that exercising emotional regulation during episodic counterfactual thinking is likely to be a successful therapeutic strategy to bring about beneficial memory modifications.

Episodic counterfactual thinking (ECT) refers to our psychological tendency to mentally simulate alternative ways in which past personal events could have occurred but did not (De Brigard & Giovanello 2012; De Brigard et al. 2013a). As such, ECT is a subclass of our more general capacity to entertain thoughts about ways in which both personal and nonpersonal events could have been (Roese 1997). ECT is ubiquitous, and the past two decades have seen an explosion of research on its psychological mechanisms and effects on emotion and behavior. One consistent result is that engaging in counterfactual simulation amplifies emotions, which can be either negative, like the regret we feel when the counterfactual involves a better outcome than the one obtained (i.e., upward counterfactual), or positive, like the relief we feel when the counterfactual involves a worse outcome than the one obtained (i.e., downward counterfactual; Kahneman & Miller 1986). Accordingly, it has been suggested that counterfactual thinking serves two different functions, depending on the direction of the mutation. Upward counterfactuals are said to serve a *preparative* function in anticipation to similar events that may occur in the future, whereas downward counterfactuals are said to serve an *affective* function that helps agents feel better about their experienced outcomes (Roese 1994; Roese and Olson 1995).

That view fails to account for the fact that sometimes we entertain upward counterfactuals about events that we know will not ever be repeated, however, as well as the fact that some downward counterfactuals elicit regret rather than relief. As a result, it was recently suggested that the function of counterfactual thoughts may differ depending on whether one focuses on comparing the simulated alternative against the actual event (evaluative alone) or simply on reflecting about the simulated alternative alone (reflective mode; Markman & McMullen 2003). Indeed, in an updated modified version of the functional view, Epstude and Roese (2008) suggest that the content and the emotion associated with the simulation are two different routes by means of which counterfactual thoughts can affect subsequent behavior. If so, it is worth wondering how one's affective response to a mental simulation interacts with the representational content of the counterfactual thought – or, to put it in terms of IMM, how is it possible that the emotion associated with ECT can affect one's subsequent reappraisal of the experienced event?

Our hypothesis – which is entirely consistent with Lane et al.'s IMM – is that, ordinarily, one of the reasons we engage in ECT is to "edit" the episodic autobiographical memories from which counterfactual simulations are construed. As Lane et al. point out, many studies have shown that, upon reactivation, memories are labile and prone to modification during reconsolidation (Nadel et al. 2012; Nader & Einarsson 2010; Schiller & Phelps 2011). Thus, because ECT requires the reactivation of a specific episodic autobiographical memory, we think it is likely that the experienced affect during reactivation, in addition to the direction of the mutation during counterfactual simulation, may alter the affective content of autobiographical memories upon subsequent reconsolidation.

Initial support for this hypothesis comes from a recent study showing that repeated reactivation of episodic counterfactuals of autobiographical memories decreases the subjective estimates of their perceived plausibility while increasing positive emotional valence (De Brigard et al. 2013b). In other words, the more we think about how a past event could have occurred, the less plausible it seems that such an event could have occurred as imagined, and the more positive our emotion is during the simulation. De Brigard et al. (2013b) hypothesized that this reduction in perceived plausibility as a result of repeated simulation is an adaptive feature of counterfactual thinking, as it helps to disregard events that did not occur to reduce our need to keep pondering about them. It seems as though ECT help us to come to grips with the past.

Now, when considered against the IMM, this result may actually be a manifestation of a more general effect. Ordinary instances of episodic counterfactual thoughts propitiate the healthy and often unassisted modification of an autobiographical memory trace by altering the original content and emotion associated with the past experience. The original content and emotion become new and better ones, elicited during the counterfactual simulation, a process that, for lack of a better pun, could be dubbed "memory mollification." Sadly, though, individuals suffering from anxiety and depression do not experience such relief (Nolen-Hoeksema 2000). In fact, their propensity to ruminate on negative thoughts associated with past events and to repetitively fixate on regret-provoking counterfactuals is not only a critical predictor (Roese et al. 2009), but also a debilitating component of both anxiety (Harrington & Blankenship 2002) and depression (Spasojevic & Alloy 2001; Thomsen 2006). This suggests that, at least in individuals suffering from these conditions, ECT fails to mollify their reactivated memories.

Given that ECT is theoretically well-situated to enact therapeutic change, further research is needed to fully understand how the affective attitude, as well as the direction of the mutation during counterfactual simulations, can affect the phenomenological characteristics of the autobiographical memory from which they are derived. Nonetheless, we hope to have offered good reason to believe that episodic counterfactual simulations may be particularly effective in bringing about the kinds of memory modifications (and mollifications) suggested by the IMM.

Changing maladaptive memories through reconsolidation: A role for sleep in psychotherapy?

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Susanne Diekelmann[a] and Cecilia Forcato[b]

[a]*Institute of Medical Psychology and Behavioral Neurobiology, University of Tübingen, Tübingen 72076, Germany; and* [b]*University of Buenos Aires, Institute of Physiology, Molecular Biology and Neuroscience (IFIByNE-CONICET), 1428 Capital Federal, Buenos Aires, Argentina*

susanne.diekelmann@uni-tuebingen.de cforcato@fbmc.fcen.uba.ar
http://www.medizin.uni-tuebingen.de/en/Research/Institutes/Medical+Psychology.html

Abstract: Like Lane et al., we believe that change in psychotherapy comes about by updating dysfunctional memories with new adaptive experiences. We suggest that sleep is essential to (re-)consolidate such corrective experiences. Sleep is well-known to strengthen and integrate new memories into pre-existing networks. Targeted sleep interventions might be promising tools to boost this process and thereby increase therapy effectiveness.

We greatly appreciate the target article by Lane et al. highlighting the importance of recent findings in the brain sciences for understanding and improving the mechanisms of action in psychotherapy. We believe that it is high time to incorporate this knowledge into psychotherapy research, as well as into practical psychotherapy and education. Lane et al. discuss compellingly the role of maladaptive emotional memories in psychopathology and the possibility to change dysfunctional memories through new corrective experiences in the therapy setting via processes of reconsolidation. Although we are in perfect agreement with this account, we want to highlight a potentially crucial factor in this process: the functional role of sleep.

Sleep is well-known to enhance the consolidation of freshly acquired memories, particularly emotional memories (Payne & Kensinger 2010; Rasch & Born 2013; Stickgold & Walker 2013). Delayed memory retrieval is typically enhanced if the initial acquisition of new memories is followed by a period of sleep compared with an equivalent wake period, with sleep occurring shortly after learning being more effective than delayed sleep (Gais et al. 2006). Some forms of memory even require sleep during the first night after learning, with the new memory being entirely lost if sleep is forgone (Stickgold et al. 2000). For many forms of memory, brief naps of 40 to 90 minutes are sufficient to promote consolidation processes (Diekelmann et al. 2012; Mednick et al. 2003; Tucker et al. 2006). One study suggests that even a very short nap of only 6 minutes can improve memory performance even though longer naps provide stronger improvements (Lahl et al. 2008).

Apart from the strengthening and stabilization of memories, sleep also facilitates the integration of new memories into pre-existing schemas and semantic networks (Ellenbogen et al. 2007; Landmann et al. 2014; Tamminen et al. 2013), a function that seems to be of particular relevance in the context of changing and updating memories in psychotherapy. Reconsolidation of memories after reactivation during wakefulness (e.g., via retrieval) has likewise been suggested to benefit from sleep (Walker et al. 2003). It is generally believed that the consolidating function of sleep for memory relies on the neuronal reactivation ("replay") of new in conjunction with older memory representations during sleep, possibly in concurrence with a selective downscaling process, such that the respective memories are stronger and better integrated after sleep (Diekelmann & Born 2010; Lewis & Durrant 2011; Tononi & Cirelli 2014).

Apart from this memory-improving effect of normal sleep, recent studies suggest that specific characteristics of sleep can be directly targeted to enhance sleep's beneficial effect (Diekelmann 2014; Spiers & Bendor 2014). For example, facilitating memory reactivation by presenting olfactory or auditory cues during sleep that have previously been associated with the learning experience enhances memory consolidation (Oudiette & Paller 2013; Rasch et al. 2007). Such targeted memory reactivations can specifically enhance those memories that are cued during sleep while leaving uncued memories unaffected (Rudoy et al. 2009; Schonauer et al. 2014). Re-exposure of olfactory context cues during sleep that had been present during prior fear conditioning might even induce extinction of the conditioned fear response (Hauner et al. 2013; but see Barnes & Wilson 2014; Rolls et al. 2013).

Increasing slow oscillations (<1 Hz, the hallmark brain oscillation of slow wave sleep) by electrical transcranial direct current stimulation (tDCS) or auditory stimulation is another promising method to enhance sleep-dependent memory processing (Marshall et al. 2006; Ngo et al. 2013). Applying electrical currents that oscillate at the same frequency as natural slow oscillations intensifies endogenous slow oscillations and improves memory consolidation (Marshall et al. 2006). Similar increases in slow oscillations and associated memory performance are observed following timed auditory stimulation of slow oscillations (Ngo et al. 2013). A third way to manipulate sleep and memory relates to pharmacological interventions. Several drugs targeting different neurotransmitter systems have been proven effective to enhance memory during sleep, such as drugs manipulating neurotransmission of noradrenaline (Gais et al. 2011), dopamine (Feld et al. 2014), glutamate (Feld et al. 2013), and GABA (Kaestner et al. 2013).

Many psychiatric disorders are associated with impaired sleep and memory dysfunctions, such as post-traumatic stress disorder (PTSD) (Germain 2013), depression (Steiger et al. 2013), and schizophrenia (Lu & Goder 2012). Improving sleep in these patients might generally ameliorate disorder-related symptoms and improve cognitive performance. Patients with schizophrenia, for example, show reduced sleep-dependent memory consolidation (Goder et al. 2004), while electrical slow oscillation stimulation during sleep increases memory functions in these patients (Goder et al. 2013). Apart from a generally positive effect of restoring normal sleep patterns, we want to suggest that sleep can specifically support the strengthening and integration of emotional memories that have been updated during prior psychotherapy. Two recent studies provide first evidence that sleep after exposure therapy improves therapy outcome in spider phobia (Kleim et al. 2013; Pace-Schott et al. 2012). Patients underwent a virtual reality exposure session and were allowed to sleep for 90 minutes after the treatment (Kleim et al. 2013). At a follow-up test one week later, these patients reported significantly reduced fear and spider-related cognitions compared with a group of patients that had stayed awake after the treatment. It remains to be elucidated whether targeted sleep manipulations, such as cued memory reactivation and slow oscillation stimulation, can boost this effect further.

Based on this evidence, we suggest that sleep and specific sleep interventions can facilitate memory updating and thereby improve therapy gain in memory-related psychopathology. Future research should test whether certain sleep interventions are more effective for certain types of psychotherapy and how sleep interventions can best be incorporated into the therapy setting to optimize outcome. We believe that sleep interventions are highly