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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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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

promising new therapy tools as they do not only bear a strong potential to increase therapy success but at the same time are cost-effective and have no to little side effects.

Minding the findings: Let's not miss the message of memory reconsolidation research for psychotherapy

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Abstract: That memory reconsolidation is the process underlying decisive, lasting therapeutic change has long been our proposal, and the recognition of its critical role by Lane et al. is a welcome development. However, in our view their account has significant errors due to neglect of research findings and neglect of previous work on the clinical application of those findings.

Lane et al. provide masterful coverage of learning and memory as relevant to psychotherapy. However, we take issue with their account of memory reconsolidation. Despite their central focus on reconsolidation, and despite affirming (rightly, in our view) that "clinical change occurs through the process of memory reconsolidation" (sect. 1, para. 8), their article provides no account of (a) abundant research findings that have identified the specific process of memory reconsolidation, or (b) extensive previous development by others of the article's main themes – the use of reconsolidation for psychotherapy and for a new framework of psychotherapy integration – or of the challenge that reconsolidation poses to nonspecific common factors theory (Ecker 2008; 2011; 2013; Ecker & Toomey 2008; Ecker et al. 2012, 2013; Welling 2012).

Throughout the twentieth century, myriad studies of extinction demonstrated that the memory circuits of a conditioned (learned) response are suppressed temporarily, but never erased, by extinction. Researchers concluded therefore that the brain lacks any neuroplastic process that could truly delete a learning that has been installed in long-term memory by the process of consolidation (whereas new learnings are unstable and disruptable prior to consolidation). Consolidation was believed irreversible, and consolidated memory circuits were believed to be stable and indelible for the individual's lifetime (e.g., LeDoux et al. 1989).

Then, during the late 1990s, several studies, culminating with that of Nader et al. (2000), found that the neural circuitry encoding a consolidated learning transformed into a deconsolidated, destabilized, disruptable state following a reactivation of the learning by cues that were salient features of the original learning experience. The existence of deconsolidation meant that memories also reconsolidate, and that the target learning could be completely eliminated while destabilized, not just suppressed temporarily. Erasure occurs either endogenously, through new learning that re-encodes the unlocked neural circuitry, or exogenously, as when chemical agents prevent circuits from reconsolidating, destroying them.

However, it was not until 2004 that the brain's inherent rules for launching deconsolidation/reconsolidation were identified (Pedreira et al. 2004), with subsequent confirmation by many other studies (for a list, see http://tiny.cc/7yutfx, Ecker 2015 or Ecker et al. 2012, p. 21). Those studies, taken together, have clarified what the brain requires for deconsolidating the neural encoding of a target learning or schema: (1) The target learning or schema has to be reactivated, vivifying its expectations of how the world or self will operate, and (2) concurrently the subject also has to experience something saliently novel or discrepant in relation to what the target learning expects or "knows" according to its schematic or semantic content or model.

Those two concurrent conditions constitute what reconsolidation researchers term a "mismatch experience" or "prediction error experience," and what we have termed a "juxtaposition experience" in the clinical context (e.g., Ecker 2008; Ecker et al. 2012; 2013). Reactivation without concurrent discrepancy fails to induce deconsolidation, and the memory remains stable (e.g., Sevenster et al. 2012). Lane et al. contend every reactivation of a memory is destabilizing, which has already been disproved. Neuroscientists view reconsolidation as the brain's process for updating memories because it launches only if discrepant experience accompanies schema reactivation. Lane et al.'s central message appears to be that emotional arousal is necessary for inducing memory reconsolidation. The research shows otherwise. The mismatch requirement has been detected for many types of memory ranging from cortical, factual learnings having no emotional content (e.g., changed set of syllable pairings; Forcato et al. 2009) to subcortical, intensely emotional learnings (e.g., change of safety position in animal studies; Morris et al. 2006). The brain clearly does not require emotional arousal per se for inducing deconsolidation. That is a fundamental point. If the target learning happens to be emotional, then its reactivation (the first of the two required elements) of course entails an experience of that emotion, but the emotion itself does not inherently play a role in the mismatch that then deconsolidates the target learning, or in the new learning that then rewrites and erases the target learning (discussed at greater length in Ecker 2015). Naturally, target learnings or schemas in psychotherapy usually are emotional, and the observable emotion accompanying their reactivation is a key marker of adequate reactivation. For those reasons, emotional arousal is usually present during moments of deep therapeutic change, but Lane et al. conflate that phenomenology of emotion with the mismatch phenomenology that deconsolidates the reactivated learning and allows transformational change.

The same considerations imply that "changing emotion with emotion" (stated three times by Lane et al.) inaccurately characterizes how learned responses change through reconsolidation. Mismatch consists most fundamentally of a direct, unmistakable perception that the world functions differently from one's learned model. "Changing model with mismatch" is the core phenomenology. Emotions then change as a derivative effect of change in semantic structures (models, rules and attributed meanings).

Lane et al. propose a psychotherapy integration scheme based on the structure of memory. We have proposed a psychotherapy integration framework centered on the brain's required steps that induce schema destabilization and erasure (Ecker 2011; Ecker et al. 2012, pp. 126–56), and have shown that the diverse systems of therapy can be unified by identifying how their distinctive methodologies do, or do not, facilitate those critical, universal steps. This approach creates "a shared, empirically based frame of reference and a shared vocabulary, allowing these practitioners to discuss their methods in a manner meaningful to each other and to practitioners of yet other clinical systems" (Ecker et al. 2012, p. 152). We predicted that the sequence of experiences required for schema destabilization and erasure could be found in any therapy sessions that produce deep, lasting change.

Furthermore, we argued (Ecker et al. 2012, pp. 153–55; Ecker 2013) that if transformational change of acquired responses indeed requires the specific behavioral steps that induce deconsolidation, then those steps constitute specific factors that are responsible and indispensable for decisive therapeutic change. This would mean that memory reconsolidation challenges the assertion of nonspecific common factors theory that specific factors can never be a major determinant of clinical outcome (e.g., Wampold 2001).

In short, reconsolidation research findings have far-reaching ramifications for psychotherapy, warranting close attention and nuanced understanding.