THEORY

MEMORY RECONSOLIDATION UNDERSTOOD AND MISUNDERSTOOD

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Abstract

Memory reconsolidation is the brain's natural, neural process that can produce transformational change: the full, permanent elimination of an acquired behavior or emotional response. This article identifies and examines 10 common misconceptions regarding memory reconsolidation research findings and their translation into clinical practice. The research findings are poised to drive significant advancements in both the theory and practice of psychotherapy, but these benefits depend on an accurate understanding of how memory reconsolidation functions, and misconceptions have been proliferating. This article also proposes a unified model of reconsolidation and extinction phenomena based on the brain's well-established requirement of memory mismatch (prediction error) for reconsolidation to be triggered. A reinterpretation of numerous studies published without reference to the mismatch requirement shows how the *mismatch requirement and mismatch relativity* (MRMR) model can account for diverse empirical findings, reveal unrecognized dynamics of memory change, and generate predictions testable by further research.

Keywords: memory reconsolidation, psychotherapy, memory mismatch, prediction error, erasure

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Extensive research by neuroscientists since the late 1990s has found that the brain is innately equipped with a potent process, known as memory reconsolidation, that can fundamentally modify or erase a targeted, specific learning, even complex human emotional learnings formed subcortically, outside of awareness (Pine, Mendelsohn, & Dudai, 2014; for reviews see, e.g., Agren, 2014; Reichelt & Lee, 2013). Such learnings are found to underlie and drive most of the problems and symptoms addressed in psychotherapy and counseling (Toomey & Ecker, 2007; Ecker & Toomey, 2008), so the relevance and value of memory reconsolidation for the clinical field are profound.

To describe a particular learning as "erased" means that its behavioral, emotional, cognitive, and somatic manifestations disappear completely, and no further effort of any kind is required to maintain this nullification permanently. Such lasting, transformational change is the therapeutic ideal. There is growing evidence that in erasure, the neural encoding of the target learning is nullified (Clem & Huganir, 2010; Debiec, Díaz-Mataix, Bush, Doyère, & LeDoux, 2010; Díaz-Mataix, Debiec, LeDoux, & Doyère, 2011; Jarome et al., 2012). The discovery of an erasure process was something of an upheaval, reversing a firmly established conclusion, based on nearly a century of research, that subcortical emotional learnings were indelible for the lifetime of the individual (LeDoux, Romanski, & Xagoraris, 1989; Milner, Squire, & Kandel, 1998).

I began studying reconsolidation research findings in 2005, at about the 20-year point of my psychotherapy practice. Neuroscientists' densely technical accounts of their studies have been comprehensible to me, for the most part, thanks to my first career of 14 years as a research physicist, and it quickly became apparent to me that knowledge of reconsolidation could drive the evolution of the field of psychotherapy in major ways. The process that brings about erasure is so fundamental for potent, effective psychotherapy, and so sweeping in the advances that it delivers to the clinical field, that I refocused my clinical career on translating reconsolidation research into clinical practice. This has produced a versatile, integrative methodology of psychotherapy and a conceptual framework that maps out how knowledge of reconsolidation creates four major advances for the clinical field (Ecker, 2011; Ecker, Ticic, & Hulley, 2012, 2013a,b). These advances are: a new level of effectiveness for individual clinicians, the deep unification of seemingly diverse methods and systems of psychotherapy, clarification of the much-debated role of attachment in the therapeutic process, and a decisive breakthrough beyond nonspecific common factors theory and the almost 80-year-long "dodo bird verdict" that has appeared to limit all therapy systems to the same modest level of efficacy.

Understanding memory reconsolidation involves learning some new ways of thinking that differ from familiar concepts of psychotherapeutic change and may even seem counterintuitive initially. Therefore, various aspects of the reconsolidation framework are susceptible to misconceptions. I have been observing misconceptions as they have developed for nearly a decade as of this writing, and they are increasing as awareness of the importance of reconsolidation builds at an accelerating pace. In fact, sizable conceptual errors are being propagated widely in articles by science journalists in the popular media, in articles by psychologists in peer-reviewed journals, in posts by psychotherapists in online clinical discussion groups, and, surprisingly, even in articles and talks by some neuroscientists involved in reconsolidation research (Ecker, 2014).

Thus there is a growing need for a clear map of the new territory, showing where the path of understanding branches off into the various misunderstandings of memory reconsolidation. This article is an attempt to provide such a guide. For the clinical field to fully utilize the potential of memory reconsolidation for major advances, a clear and accurate understanding of it is necessary. Knowledge communities such as the clinical field can and historically do make collective errors in the development of new knowledge, locking onto limiting, polarized, or oversimplified notions that become unchallengeable for decades until, finally, a corrective movement forms. Reconsolidation is too important to fumble and delay in that way.

Understanding how memory reconsolidation can be utilized in psychotherapy (Ecker et al., 2012) is considerably simpler than understanding memory reconsolidation research findings, so many clinicians may choose to focus on the former and pass on the latter. The explanations of research findings in this article are for those with an appetite for more rigorous insights into how memory reconsolidation works. Though memory reconsolidation is a complex phenomenon, and there is still much for researchers to discover about the fine points of how it functions, its main features now appear to be fairly well established, particularly as regards its behavioral and experiential aspects, which are of primary interest to mental health clinicians.

This article covers the following common misconceptions regarding the major features of reconsolidation research findings and their translation into clinical practice:

Misconception 1. The reconsolidation process is triggered by the reactivation of a target learning or memory.

Misconception 2. The disruption of reconsolidation is what erases a target learning.

Misconception 3. Erasure is brought about during the reconsolidation window by a process of extinction. Reconsolidation is an enhancement of extinction.

Misconception 4. Anxiety, phobias and PTSD are the symptoms that memory reconsolidation could help to dispel in psychotherapy, but more research must be done before it is clear how reconsolidation can be utilized clinically.

Misconception 5. Emotional arousal is inherently necessary for inducing the reconsolidation process.

Misconception 6. What is erased in therapy is the negative emotion that became associated with certain event memories, and this negative emotion is erased by inducing positive emotional responses to replace it.

Misconception 7. The much older concept of corrective emotional experience already covers everything now being described as reconsolidation and erasure.

Misconception 8. To induce memory reconsolidation, therapists must follow a set protocol derived from laboratory studies.

Misconception 9. A long-standing emotional reaction or behavior sometimes ceases permanently in psychotherapy without guiding the steps that bring about erasure through reconsolidation, and this shows that reconsolidation isn't the only process of transformational change.

Misconception 10. Carrying out the steps required for reconsolidation and erasure sometimes fails to bring about a transformational change, which means that the reconsolidation process isn't effective for some emotional learnings.

The discussion of those topics will at some points (such as in the section on Misconception 3) go beyond a review of research findings to propose a new interpretation of the findings. Before delving into the misconception topics, however, a short overview is needed to provide the context that will make discussion of the misconceptions meaningful. In attempting to clarify both the reconsolidation research findings and their application to clinical work, this article spans a wide range of material, which in places may be more technical and laboratory focused than some clinical readers find useful. Clinical readers can skip ahead at such points.

Memory Reconsolidation in Context

Memory reconsolidation is the brain's innate process for fundamentally revising an existing learning and the acquired behavioral responses and/or state of mind maintained by that learning. In the reconsolidation process, a target learning is first rendered revisable at the level of its neural encoding, and then revision of its encoding is brought about either through new learning or chemical agents (for reviews see Agren, 2014; Reichelt & Lee, 2013). Through suitably designed new learning, the target learning's manifestation can be strengthened, weakened, altered in its details, or completely nullified and canceled (erased). Erasure through new learning during the reconsolidation process is the true unlearning of the target learning. When erasure through new learning is carried out in psychotherapy, the client experiences a profound release from the grip of a distressing acquired response (Ecker et al., 2012). The use of chemical agents to produce erasure is described later in this article.

In order to see the full significance of memory reconsolidation for psychotherapy, it is necessary to recognize the extensive role of learning and memory in shaping each person's unique patterns of behavior, emotion, thoughts, and somatic experience. Among the many types of learning and the many types of memory, the type responsible for the great majority of the problems and symptoms that bring people to psychotherapy is implicit emotional learning—especially the implicit learning of vulnerabilities and sufferings that are urgent to avoid, and how to avoid them. These learnings form usually with no awareness of learning anything, and they form in the presence of strong emotion, which greatly enhances their power and durability (McGaugh, 1989; McGaugh & Roozendaal, 2002; Roozendaal, McEwen, & Chattarji, 2009).

For example, if a small child consistently receives frightening anger from a parent in response to the child expressing needs, the child learns not to express

or even feel needs or distress and not to expect understanding or comfort from others. This learning can occur with no representation in conscious thoughts or conceptualization, entirely in the implicit learning system. The child configures him- or herself according to this adaptive learning in order to minimize suffering in that family environment. Later in life, however, this same learned pattern has life-shaping, extremely costly personal consequences. The learnings in this example are very well-defined, yet they form and operate with no conscious awareness of the learned pattern or its self-protective, coherent emotional purpose and necessity. From outside of awareness these learnings shape the child's and later the adult's behavior, so the individual is completely unaware of living according to these specific learnings. The neural circuits encoding these learnings are mainly in subcortical regions of implicit memory that store implicit, tacit, emotionally urgent, procedural knowledge, not mainly in neocortical regions of explicit memory that store conscious, episodic, autobiographical, declarative knowledge (Schore, 2003).

As in the example above, the vast majority of the unwanted moods, emotions, behaviors, and thoughts that people seek to change in psychotherapy are found to arise from implicit emotional learnings, not in awareness (Toomey & Ecker, 2007). Common clinical phenomena that express implicit emotional learnings include insecure attachment patterns, family of origin rules and roles, unresolved emotional issues, compulsive behaviors or emotional reactions in response to an external or internal trigger, panic and anxiety attacks, depression, low self-esteem, fear of intimacy, sexual inhibition, traumatic memory and posttraumatic stress symptoms, procrastination, and many others.

Of course, some psychological and behavioral symptoms are not caused by emotional learnings—for example, hypothyroidism-induced depression, autism, and biochemical addiction—but it is implicit emotional learnings that therapists and their clients are working to overcome in most cases. There are also genetic or biochemical factors that may contribute to mood disturbances, but it is nevertheless the individual's implicit emotional learnings that are largely responsible for triggering specific bouts of emotional instability (Toomey & Ecker, 2009).

It is the tenacity of implicit emotional learnings, more than their ubiquity, that is the real clinical challenge. On a daily basis, psychotherapists encounter the extreme durability of original emotional learnings that fully maintain their chokehold decades after they first formed. Researchers too have observed that "A unique feature of preferences [the authors use that term to denote compelling, emotionally complex avoidances and attractions] is that they remain relatively stable over one's lifetime. This resilience has also been observed experimentally, where . . . acquired preferences appear to be resistant to extinction training protocols" (Pine et al., 2014, p. 1). The life-constraining grip of such patterns is the bane of psychotherapists and their clients, yet that very tenacity is a survival-positive result of natural selection. In the course of evolution, selection pressures crafted the brain so that any learning accompanied by strong emotion becomes encoded by enhanced, exceptionally durable synapses due to the emotion-related hormones that influence synapse formation (McGaugh, 1989; McGaugh & Roozendaal, 2002; Roozendaal et al., 2009).

So durable are implicit emotional learnings that they continue to function and drive responses even during states of amnesia and are only temporarily suppressed, not erased, by the process of extinction (nonreinforcement of a reactivated, learned expectation). Psychologists and neuroscientists have amassed extensive evidence that even after complete extinction of an emotionally learned response, the extinguished response is easily retriggered in various ways. This revealed that extinction training does not result in the unlearning, elimination, or erasure of the suppressed, original learning (making the term "extinction" something of a misnomer, suggesting as it does a permanent disappearance). Rather, the research found that extinction training forms a separate, second learning that competes against, but does not change, the original learning (see, e.g., Bouton, 2004; Foa & McNally, 1996; Milner et al., 1998; Myers & Davis, 2002). The learning formed by extinction training of a fear response is encoded in the brain's prefrontal cortex, a region that can suppress and temporarily override the nearby subcortical amygdala, which plays a central role in storing and reactivating fear-based learnings (Milad & Quirk, 2002; Phelps, Delgado, Nearing, & LeDoux, 2004; Santini, Ge, Ren, de Ortiz, & Quirk, 2004; Quirk, Likhtik, Pelletier, & Pare, 2003).

Many decades of studying extinction led researchers to the conclusion that implicit emotional learnings are permanent and indelible for the lifetime of the individual once they have been installed in long-term memory circuits through the process of *consolidation* (reviewed in McGaugh, 2000). There appeared to exist no form of neuroplasticity capable of unlocking the synapses of consolidated implicit memory circuits. The tenet of indelibility reached its peak influence with the publication of a research article on extinc-

tion studies by neuroscientists LeDoux, Romanski, and Xagoraris (1989) titled "Indelibility of Subcortical Emotional Memories." The indelibility model soon entered the literature of psychotherapy when van der Kolk (1994) published in the *Harvard Review of Psychiatry* his seminal article "The Body Keeps the Score: Memory and the Evolving Psychobiology of Posttraumatic Stress," in which there was a section titled "Emotional memories are forever." The conclusion that implicit emotional learnings persist for a lifetime meant that people could never become fundamentally free of flare-ups of childhood emotional conditioning. The worst experiences in an individual's past could at any time become reactivated and seize his or her state of mind or behavior in the present.

Then, several studies published from 1997 to 2000 suddenly overturned the model of irreversible memory consolidation and indelibility. Actually, a handful of earlier studies published from 1968 to 1982 had reported observations of the disappearance of well consolidated emotional learnings (Judge & Quartermain, 1982; Lewis, 1979; Lewis, Bregman, & Mahan, 1972; Lewis & Bregman, 1973; Mactutus, Riccio, & Ferek, 1979; Misanin, Miller, & Lewis, 1968; Richardson, Riccio, & Mowrey, 1982; Rubin, 1976; Rubin, Fried, & Franks, 1969). However, these unexplained challenges to the prevailing model of irreversible consolidation were seen as anomalies and received scant attention from memory researchers and clinicians at the time.

At the end of the 1990s, however, neuroscientists in several different laboratories resumed studying the effects of reactivating an established emotional learning (Nader, Schafe, & LeDoux, 2000; Przybyslawski, Roullet, & Sara, 1999; Przybyslawski & Sara, 1997; Roullet & Sara, 1998; Sara, 2000; Sekiguchi, Yamada, & Suzuki, 1997). Using sophisticated new techniques as well as the field's advanced knowledge of exactly where in the brain certain emotional learnings form and are stored in memory, researchers again demonstrated the full elimination of any expression of a target learning. In addition, they demonstrated that such erasure of the learning became possible because consolidated, locked memory synapses had returned to a deconsolidated, unlocked, unstable or "labile" state, allowing erasure of the learning by chemical agents that disrupt only synapses that are in an unstable, nonconsolidated condition. The longstanding tenet of irreversible consolidation was disconfirmed.

The destabilized state of deconsolidation was found to exist only soon after the target learning had been reactivated by a suitable cue or reminder. Yet, long after such a reactivation, an implicit learning is found to be once again in a stable, consolidated state. Thus the detection of a deconsolidated, destabilized state of memory soon after its reactivation implied the existence of a natural process of *reconsolidation*, the relocking of the synapses of a destabilized memory, returning the memory to stability. Subsequent studies found that the labile state of deconsolidation lasts for about five hours—a period widely known now as the reconsolidation window—during which the unstable target learning can be modified or erased (Duvarci & Nader, 2004; Pedreira, Pérez-Cuesta, & Maldonado, 2002; Pedreira & Maldonado, 2003; Walker, Brakefield, Hobson, & Stickgold, 2003).

If, following the reactivation and destabilization of a target learning, there is no new learning and no chemical treatment, then after its reconsolidation (that is, more than about five hours later) the target learning is found to have increased strength of expression (e.g., Forcato, Fernandeza, & Pedreira, 2014; Inda, Muravieva, & Alberini, 2011; Rossato, Bevilaqua, Medina, Izquierdo, & Cammarota, 2006; Stollhoff, Menzel, & Eisenhardt, 2005). For that reason, researchers regard reconsolidation as having two biological functions: (a) It preferentially strengthens recent learnings that are most frequently reactivated and destabilized, and (b) it allows new learning experiences to update (strengthen, weaken, modify, or nullify) an existing learning. The latter function is the one utilized for bringing about nullification and transformational change in psychotherapy. When a learned, unwanted emotional reaction is erased, there is no loss of memory of events in one's life (as shown by Kindt, Soeter, & Vervliet, 2009, and as illustrated by a clinical example later in this article). There is evidence that the destabilization/restabilization process and the updating/erasure process occur through different molecular and cellular processes (Jarome et al., 2012; Lee et al., 2008).

With that background, we can now examine the misconceptions of the reconsolidation process listed above.

Ten Common Misconceptions

Misconception 1: The Reconsolidation Process Is Triggered by the Reactivation of a Target Learning or Memory

As noted earlier, in the reconsolidation discovery studies of 1997 to 2000, a state of deconsolidation was found to exist only soon after the target learning had been reactivated by a suitable cue or reminder. This observation was interpreted by the researchers to

mean that each reactivation of a target learning deconsolidates its neural circuits, launching the reconsolidation process.

That conclusion may have been sensible based on the initial few studies, but it turned out to be incorrect. Pedreira, Pérez-Cuesta, and Maldonado (2004) were first to show that reactivation alone does not bring about deconsolidation and reconsolidation. They concluded, "at odds with the usual view, retrieval per se is unable to induce labilization of the old memory" (p. 581), and they demonstrated that what the brain requires to trigger the reconsolidation process is reactivation plus another critical experience, described below. Subsequently, this same two-step requirement has been demonstrated in at least 22 other studies that I have tallied as of this writing. They are listed in Table 1. In the discovery studies of 1997 to 2000, researchers had fulfilled this two-step requirement without awareness of doing so, as shown later in this section.

The early interpretation that reactivation by itself produces deconsolidation spread widely among both neuroscientists and science journalists and became a reconsolidation meme. Despite the post-2004 piling up of decisive evidence revealing that this original conclusion was incorrect, it has continued to be asserted in new writings by not only science journalists but also by some prominent researchers who were involved in the original studies, as well as by many later reconsolidation researchers. As of this writing, more than 10 years since the mismatch requirement was first detected and published, new research articles continue to be published that lack any consideration of the mismatch requirement's role in the reported results (e.g., Wood et al., 2015).

It is perhaps understandable that science journalists would latch on to and continue to spread the misconception that reactivation in itself destabilizes the reactivated learning, if they were unaware of what the ongoing research was revealing. It is less clear why the error would continue to be voiced by researchers. From my point of view as a clinician observer witnessing this situation unfold for almost a decade, I cannot escape the impression that many reconsolidation researchers appear unaware of sizable amounts of research published in their own area of specialization. Some of the more significant reconsolidation research articles, such as that of Schiller et al. (2010), assert that reactivation induces reconsolidation and reference none of the studies in Table 1 that have shown that view to be incorrect. Commenting here on this situation is hopefully warranted by the importance of assuring that research findings critically important for clinical application are not obscured.

What, then, is the second step that must accompany reactivation? Pedreira et al. (2004), followed by all of the studies listed in Table 1, have shown that in order to induce reconsolidation, reactivation must be accompanied or followed soon by what researchers term a *mismatch* experience or *prediction error* experience. This is an experience of something distinctly discrepant with what the reactivated target memory "knows" or expects—a surprising new learning consisting of anything from a superfluous but salient novelty element to a direct contradiction of what is known according to the target learning. It makes sense from an evolutionary perspective that deconsolidation and reconsolidation, being the brain's process for updating learnings and memories, would be triggered only by new information that is at odds with the contents of an existing learning (Lee, 2009). Lee wrote, "reconsolidation is triggered by a violation of expectation based upon prior learning, whether such a violation is qualitative (the outcome not occurring at all) or quantitative (the magnitude of the outcome not being fully predicted)" (p. 417). It would be biologically costly, with no benefit, if the brain launched the complex neurochemical process of reconsolidation when there is no new knowledge requiring a memory update. The studies listed in Table 1 have shown that the brain evolved so as to launch de/reconsolidation *only when an experience of something discrepant with a reactivated, learned expectation or model of reality signals the need for an update of that existing knowledge*. This empirical finding of a critical role of mismatch or prediction error can be regarded as a neurobiological validation of a central feature of the learning models of both Piaget (1955) and Rescorla and Wagner (1972).

Thus, what shifts a particular learning into a deconsolidated, destabilized state, allowing its expression to be modified or erased by new learning during an approximately five-hour window, is not simply reactivation of that learning, but the experience of that reactivated learning encountering a mismatch or prediction error. As stated by Agren (2014) in reviewing research on reconsolidation of emotional learnings in humans, "it would appear that prediction error is vital for a reactivation of memory to trigger a reconsolidation process" (p. 73). Likewise, Delorenzi et al. (2014) commented, "strong evidence supports the view that reconsolidation depends on detecting mismatches between actual and expected experiences" (p. 309). Exton-McGuinness, Lee, and Reichelt (2015) review the role of prediction errors in memory reconsolidation studies and sum up their position by stating, "We propose that a prediction error signal . . . is necessary

Table 1

Studies demonstrating that both memory reactivation and memory mismatch (prediction error) are necessary for inducing memory destabilization (deconsolidation) and reconsolidation, and that memory reactivation alone is insufficient.

Year	Authors	Species	Memory type	Design and findings
2004	Pedreira et al.	Crab	Contextual fear memory	Reactivated learned expectation of visual threat must be sharply disconfirmed for memory to be disrupted by cycloheximide.
2005	Frenkel et al.	Crab	Contextual fear memory	New experience modifies memory expression only if preceded by a memory mismatch experience.
2005	Galluccio	Human	Operant conditioning	Memory is erased only by being reactivated along with a novel contingency.
2005	Rodriguez-Ortiz et al.	Rat	Taste recognition memory	Novel taste following reactivation allows memory disruption by anisomycin.
2006	Morris et al.	Rat	Spatial memory of escape from danger	Reactivation allows disruption of original memory by anisomycin only if learned safe position has been changed, creating mismatch of expectation.
2006	Rossato et al.	Rat	Spatial memory of escape from danger	Reactivation allows disruption of original memory by anisomycin only if learned safe position has been changed, creating mismatch of expectation.
2007	Forcato et al.	Human	Declarative memory	Memory of syllable pairings learned visually is destabilized and impaired by new learning only if, after reactivation by presentation of context, presentation of a syllable to be paired does not occur as expected, creating mismatch.
2007	Rossato et al.	Rat	Object recognition memory	Memory is disrupted by anisomycin only if reactivated in presence of novel object.
2008	Rodriguez-Ortiz et al.	Rat	Spatial memory of escape from danger	Reactivation allows disruption of original memory by anisomycin only if learned safe position has been changed, creating mismatch of expectation.
2009	Forcato et al.	Human	Declarative memory	Memory of syllable pairings learned visually is labilized and lost only if reactivation is followed by learning revised novel pairings.
2009	Pérez-Cuesta & Maldonado	Crab	Contextual fear memory	Reactivated learned expectation of visual threat must be sharply disconfirmed for memory to be disrupted by cycloheximide.
2009	Winters et al.	Rat	Object recognition memory	Memory is disrupted by MK-801 only if reactivated in presence of novel contextual features.
2010	Forcato et al.	Human	Declarative memory	Memory of syllable pairings learned visually destabilizes and incorporates new information only if, after reactivation, the expected opportunity to match syllables does not occur, creating mismatch.
2011	Coccoz et al.	Human	Declarative memory	Memory of syllable pairings learned visually destabilizes, allowing a mild stressor to strengthen memory, only if, after reactivation, the expected opportunity to match syllables does not occur, creating mismatch.
2012	Caffaro et al.	Crab	Contextual fear memory	New experience modifies memory expression only if preceded by a memory mismatch experience.
2012	Sevenster et al.	Human	Associative fear memory (classical conditioning)	Reactivated fear memory is erased by propranolol only if prediction error is also experienced.
2013	Balderas et al.	Rat	Object recognition memory	Only if memory updating is required does reactivation trigger memory destabilization and reconsolidation, allowing memory disruption by anisomycin.
2013	Barreiro et al.	Crab	Contextual fear memory	Only if memory reactivation is followed by unexpected, mismatching experience is the memory eliminated by glutamate antagonist.

Table 1 cont.

2013	Díaz-Mataix et al.	Rat	Associative fear memory (classical conditioning)	Reactivated fear memory is erased by anisomycin only if prediction error is also experienced.
2013	Reichelt et al.	Rat	Goal-tracking memory	Target memory reactivated with prediction error was destabilized and then disrupted by MK-801, but not if brain's prediction error signal was blocked.
2013	Sevenster et al.	Human	Associative fear memory (classical conditioning)	Reactivated fear memory is destabilized, allowing disruption by propranolol, only if prediction-error-driven relearning is also experienced.
2014	Exton-McGuinness et al.	Rat	Instrumental memory (operant conditioning)	Memory for lever pressing for sucrose pellet was disrupted by MK-801 only if the reinforcement schedule during reactivation was changed from fixed to variable ratio, creating prediction error.
2014	Sevenster et al.	Human	Associative fear memory (classical conditioning)	Reactivated fear memory is destabilized, allowing disruption by propranolol, only if prediction-error-driven relearning is also experienced, and termination of prediction error terminates destabilzation.

for destabilisation and subsequent reconsolidation of a memory" (p. 375). That is the research finding that translates into major advances for the psychotherapy field (Ecker, 2011; Ecker et al., 2012, 2013a,b).

For those advances to materialize, it is necessary for clinicians to understand well what the brain regards as an experience of mismatch or prediction error. Misconceptions abound on this point as well. The following example shows the meaning of mismatch at the basic level of classical conditioning in the laboratory, as demonstrated by Pedreira et al. (2004) and other studies listed in Table 1. Clinically relevant learnings are often far more complex, and the guiding of mismatch experiences in psychotherapy looks very different, as a rule, from the laboratory instances described in this article, but the principles of mismatch are usefully clarified at this basic level.

Consider a target learning that was created by several repetitions of turning on a blue light and delivering a mild electric shock several seconds later, during the last half-second of the light being on. Subsequently, if the blue light is turned on again, the learned expectation of the shock is reactivated immediately, along with fear and the physiological expressions of fear, such as a mouse's freezing or a human's change of skin conductance. However, this reactivation does not deconsolidate and destabilize the memory circuits of this learned association of light and shock, because no mismatch experience has occurred as yet. While the blue light stays on without any shock being delivered, a mismatch or prediction error has not occurred because the shock might still occur. The target learning is in a state of expectancy of the shock. Mismatch occurs when the blue light is turned off with no shock having been experienced. Only then are perceptions discrepant with what the target learning "knows." Now the synapses encoding the target learning unlock into a modifiable state, because now it is definite that no shock occurred as expected while the blue light was on.

Understanding the mismatch requirement allows us to interpret correctly the results of various studies that were misinterpreted by the researchers because they analyzed their studies without reference to the mismatch requirement. The simple logic of the situation, as stated by Agren (2014), is that "the studies that have shown effects of reconsolidation . . . must somehow have induced a prediction error" (p. 80). Ecker et al. (2012) articulated the same principle: "Whenever the markers of erasure of a learning are observed, both reactivation and a mismatch of that learning must have taken place, unlocking its synapses, or erasure could not have resulted. This logic can serve as a useful guide for identifying the critical steps of process in both the experiments of researchers and the sessions of psychotherapists" (p. 23).

Therefore, identifying the presence or absence of mismatch in each of the many published studies of reconsolidation that lacks consideration of the mismatch requirement is an exercise necessary for bringing the field of reconsolidation research to maturity from its present fragmented condition. The remainder of this section begins that unifying exercise by describing several key studies, analyzing the presence or absence of mismatch in them, and reinterpreting their results accordingly. This analysis of mismatch in

published studies yields instructive insights into how mismatch may function.

The study by Nader, Schafe, and LeDoux (2000), which repeated the basic design of some other early studies (Przybyslawski et al., 1997, 1999; Roullet et al., 1998), is often regarded as the one that brought the initial research to a tipping point of establishing the reconsolidation phenomenon conclusively. Nader et al. used the same classical conditioning procedure as described in the example just above, but with an audible tone rather than a blue light. They taught rats to expect a shock during the last half-second of a 30-s tone. Later, their procedure accomplished memory reactivation with the *onset* of the 30-s tone, and it accomplished memory mismatch with the *offset* of the tone with no shock occurring, triggering destabilization of the target learning and launching the reconsolidation process. However, the researchers were unaware of the mismatch requirement (which was discovered four years later by Pedreira et al., 2004) or of the crucial role of this mismatch in triggering deconsolidation of the target learning. It was by chance that their procedure happened to include the needed mismatch. Memory erasure resulted from anisomycin administered soon after that mismatch experience (but not when administered 6 hr later, when the reconsolidation window was no longer open), confirming that memory destabilization (deconsolidation) had occurred, because anisomycin destroys only nonconsolidated synapses.

Understandably but erroneously, Nader et al. concluded that memory reactivation was sufficient for triggering destabilization. If their design had included reactivation by the tone together with the expected shock, eliminating the mismatch of expectations, no deconsolidation or erasure would have occurred. Such failure to achieve destabilization of a reactivated target learning has been reported in many studies (e.g., Bos, Becker, & Kindt, 2014; Cammarota, Bevilaqua, Medina, & Izquierdo, 2004; Hernandez & Kelley, 2004; Mileusnic, Lancashire, & Rose, 2005; Wood et al., 2015), and we can now recognize that this failure was due to an absence of mismatch or prediction error in the procedure used. (For example, as reported by Hernandez and Kelley in 2004, a rat's memory that pressing a certain lever brings a sugar reward was indeed reactivated when the rat was once again placed in the chamber with the lever, pressed it, and received a sugar pellet, but this reactivation provided the expected reinforcement and entailed no experience of prediction error, so memory destabilization did not occur.) In these studies, too, the researchers made no mention of a mismatch or prediction error requirement in their interpretation of results. Instead, they concluded incorrectly that the particular type of memory under study was not subject to reconsolidation. Subsequently, other studies successfully demonstrated reconsolidation for those types of memory (see, e.g., Wang, Ostlund, Nader, & Balleine, 2005).

All 23 studies listed in Table 1 have shown that reactivation alone does not launch the reconsolidation process, but reactivation plus mismatch does. This point was particularly emphasized by Forcato, Argibay, Pedreira, and Maldonado (2009) in titling their article, "Human Reconsolidation Does Not Always Occur When a Memory Is Retrieved," and by Sevenster, Beckers, and Kindt (2012), who titled theirs "Retrieval Per Se Is Not Sufficient to Trigger Reconsolidation of Human Fear Memory." The latter authors characterized their next published study by stating, "we show in humans that prediction error is (i) a necessary condition for reconsolidation of associative fear memory and (ii) determined by the interaction between original learning and retrieval" (Sevenster, Beckers, & Kindt, 2013, p. 830).

Reconsolidation can also be triggered by a mismatch of *when* events are expected to occur, with no change in *what* occurs, as demonstrated by Díaz-Mataix, Ruiz Martinez, Schafe, LeDoux, and Doyère (2013). On Day 1 in their study, rats heard a 60-s tone and received a momentary electrical shock at the 30-s point, midway through the tone. For each rat this was repeated 10 times to create a reliable conditioned response of fear to the tone. On Day 2, each rat heard the tone and received the shock again just once, reactivating the learned association of tone and shock. The shock occurred at the same 30-s point for some rats, but for others it occurred at the 10-s point. Immediately after this reactivation experience, researchers administered a chemical agent (anisomycin) that disrupts nonconsolidated memory circuits. On Day 3, the tone was played again for each of the rats five times with no accompanying shock, and the strength of fear responses was measured. Rats that had unchanged shock timing on Day 2 reacted with fear on Day 3 fully as strongly as they had done on Day 2, indicating that anisomycin had no effect and, therefore, that the reactivation without mismatch on Day 2 had not destabilized the target learning. In contrast, rats whose shock timing had been changed on Day 2 reacted on Day 3 with only half as many fear responses as on Day 2, indicating that anisomycin had significantly impaired the target learning and, therefore, that the reactivation with timing mismatch on Day 2 had indeed destabilized the target learning.

This important finding that temporal mismatches trigger reconsolidation will figure significantly in other discussions later in this article. Díaz-Mataix et al. did identify the prediction error that played a critical role in their procedure, and they concluded from their observations that new information must accompany reactivation in order to destabilize the target learning. That conclusion corroborates what was demonstrated in at least sixteen prior studies listed in Table 1, so it unclear why Díaz-Mataix et al. describe their finding as though it is a new discovery and cite only one of prior studies (Sevenster et al., 2012).

A target learning that has been destabilized by mismatch can be erased not only by chemical agents, but also by a counterlearning experience with no use of chemical agents. It is this endogenous approach that is most desirable for psychotherapeutic use and which has been applied extensively in that context (Ecker et al., 2012). In laboratory studies, endogenous erasure or modification of a target learning has been demonstrated with both animal and human subjects (e.g., Galluccio, 2005; Liu et al., 2014; Monfils, Cowansage, Klann, & LeDoux, 2009; Schiller et al. 2010; Steinfurth et al., 2014; Walker et al., 2003; Xue et al., 2012).

Monfils et al. (2009) used three pairings of a 20-s audio tone (the conditioned stimulus, CS) and half-second footshock (unconditioned stimulus, US), with 3 min between pairings, to train rats to respond to the tone with fear. One day later, the target learning was reactivated by the CS/tone, but there was no accompanying US/shock, which is a mismatch of the expectation of the US. So far, the procedure is basically the same as that of Nader et al. (2000), described above, but rather than disrupt the target learning chemically at this point, Monfils et al. continued to present the CS without US repeatedly. CS2, the second tone, was presented 10 min or 1 hr after the first one, but then additional CS tones came at 3-min intervals, for a total of 19 CSs. That procedure successfully and robustly erased the rats' learned fear of the tone.

Note that if the initial 10-min or 1-hr interval had been a 3-min period like all of the ensuing intervals, the repetitive CS counterlearning procedure would have been a standard multitrial extinction training, which is well known not to bring about erasure. Thus the longer interval between CS1 and CS2 was critically important for achieving erasure through reconsolidation rather than suppression through extinction. The fact that erasure occurred implies that the target learning was destabilized and erasable during the series of CSs, which in turn implies that the longer interval from CS1 to CS2 resulted in a mismatch of expected and actual timing. (The discussion of results provided by Monfils et al. does not refer to the concept of mismatch or prediction error, however.)

The key role of a temporal mismatch in inducing destabilization in both Monfils et al. (2009) and Díaz-Mataix et al. (2013) makes it clear that the brain learns the temporal features of new emotional experiences no less than it learns other characteristics, and that mismatches of timing can be highly effective for inducing reconsolidation in cases where the target learning has distinct temporal structure. In other recent research, networks of dedicated "time cells" in the hippocampus have been found to measure and remember time intervals (Jacobs, Allen, Nguyen, & Fortin, 2013; MacDonald, Lepage, Eden, & Eichenbaum, 2011; Naya & Suzuki, 2011; Paz et al., 2010).

The important observations made by Monfils et al. (2009) will be revisited and utilized later in this article to address fundamental questions of what governs whether reconsolidation or extinction occurs and why extinction fails to produce erasure. It is noteworthy too that the erasure procedure used by Monfils et al. was subsequently adapted for use with human subjects by Schiller et al. (2010), who demonstrated the first endogenous erasure of a fear learning in humans in a controlled study. The Appendix to this article provides a detailed examination of the mismatches involved in their procedure.

Fulfillment of the mismatch requirement is evident in the successful inducing of reconsolidation in a wide range of experimental procedures. For example, an associative fear learning can be triggered into reconsolidation by a reexperiencing of only the unconditioned stimulus (US) without the conditioned stimulus (CS) (Díaz-Mataix et al., 2011; Liu et al., 2014). The target learning, consisting of experiencing first the CS followed by the US, is mismatched if the US occurs without the CS. That mismatch consists of both the absence of the expected CS and also, importantly, a change in the expected temporal sequence of events, because the target learning expects the US to occur after the CS, not without the prior occurrence of the CS. Another example is the case of having two different co-occurring CSs, both of which have been paired with the same US. Debiec, Díaz-Mataix, Bush, Doyère, and LeDoux (2013) showed that reexposure to either one of the CSs can trigger the reconsolidation of the memory of the other. Here the expected co-occurrence of both CSs is mismatched when only one CS is presented. None of the authors referenced in this paragraph explained their results in terms of the mismatch requirement, however. They

discussed their results as though the triggering of reconsolidation can be attributed to reactivation alone.

Even researchers who are well aware of the mismatch/prediction error requirement can overlook the occurrence of mismatch in their own procedures. For example, Pine et al. (2014) provided an ingenious and intricate demonstration that reconsolidation occurs for complex, unconscious emotional learnings in humans—and in doing so they have supplied the strongest empirical support to date for the anecdotal clinical observations reported by Ecker et al. (2012, 2013a,b)—but they commented, "Our results seem to counter a recent theory that new learning (or the generation of a prediction error) is required during reactivation in order to trigger reconsolidation. . . . Here, no new learning took place during the reminder" (p. 11). However, the "reminder" (reactivation) that they used for triggering reconsolidation on Day 2 of their procedure contained three distinct temporal mismatches relative to the original learning on Day 1: a reversal of overall sequence, an overall duration of the series of trials that was one-fourth as long, and the introduction of a 10-min delay within the overall sequence. Thus, due to these temporal mismatches of the original learning, its reactivation was actually accompanied by an abundance of new learning, triggering destabilization and reconsolidation. As noted above, we know from Díaz-Mataix et al. (2013) that even a single temporal mismatch can be an effective destabilizer.

As this section's final example of how the mismatch requirement can account for diverse reconsolidation phenomena, there have been several studies of how the age or strength of a target learning effects the triggering of memory destabilization (Boccia, Blake, Acosta, & Baratti, 2006; Clem & Huganir, 2010; Debiec, LeDoux, & Nader, 2002; Eisenberg & Dudai, 2004; Frankland et al., 2006; Inda et al., 2011; Milekic & Alberini, 2002; Steinfurth et al., 2014; Suzuki et al., 2004; Winters, Tucci, & DaCosta-Furtado, 2009). Reviewing all results of these studies is beyond the scope of the present article, other than to summarize that, as a rule, stronger reactivation is required in order to destabilize stronger or older target learnings. Some of the studies in this area successfully destabilized both young and older target learnings (up to 8 weeks after acquisition), but others failed to destabilize older memories. Lee (2009) commented, "it is also possible that all memories undergo reconsolidation regardless of their age, but that previous studies have failed to use sufficiently intense memory reactivation conditions for older memories" (p. 416).

The results found by Suzuki et al. (2004) can serve to illustrate the further research possibilities that become apparent as a result of asking the question: If the mismatch requirement is responsible for experimental observations, what are those observations showing about how mismatch functions under various circumstances? Suzuki et al. taught rats to fear a test chamber (context/CS) by placing each rat in the chamber for 2.5 min and then administering a 2-s footshock (US). Rats in one group received just one shock; those in another group received three shocks separated by 30 s. All rats were removed from the context/CS 30 s after their final footshock. Then, either 1 day, 1 week, 3 weeks, or 8 weeks later, immediately after administration of anisomycin, rats were placed in the context/CS for various amounts of time and then removed with no shock, in order to reactivate the fear learning and disrupt it if it had been destabilized by the reactivation. One day later their fear level was measured during a 3-min reexposure back in the context/CS. This procedure resulted in the following findings:

For fear memory created by a single context-shock pairing, a 1-min shock-free reexposure to the context did not destabilize the fear learning, but a 3-min reexposure did destabilize it if memory age was 1 day, 1 week, or 3 weeks. The implication is that a 1-min shock-free reexposure did not create a mismatch experience, but a 3-min reexposure did create a mismatch. This is suggestive of a temporal structure in the target learning. The 2.5-min period of initial exposure to the context/CS fits that possibility well, because relative to that learned 2.5-min period, the 1-min reexposure could have been too short to be experienced as a nonreinforcement, so it would not create a mismatch experience, but the 3-min reexposure would.

If memory age was 8 weeks, a 3-min reexposure no longer caused destabilization, but a 10-min reexposure did destabilize. This possibly implies that memory of the 2.5-min period lost definiteness over time and therefore required a longer reexposure for decisive nonreinforcement and mismatch to be experienced.

For fear memory created by three context-shock pairings instead of one, a 3-min reexposure no longer caused destabilization, but a 10-min reexposure did destabilize. Here the challenge is to understand how a stronger fear training would alter the timing memory. Three 2-s shocks coming every 30 s is a grueling minute that might feel to a rat much longer than a minute spent sniffing around curiously in a harmless place, just as a human also experiences time periods very differently depending upon the presence or absence of pain. The prior 2.5-min duration may have been

distorted or blurred retroactively by this long, traumatic minute, such that the longer 10-min reexposure was necessary for decisive nonreinforcement and mismatch to be experienced.

The interpretations sketched above are not the only possible ways in which the mismatch requirement could have resulted in the observations made by Suzuki et al. (2004). They are offered here heuristically, by way of showing how the mismatch requirement can be logically applied to illuminate how experimental procedures interact with the inherent properties of the brain's memory systems.

The experimental procedures discussed in this section in relation to the mismatch requirement illustrate a principle that is critical for understanding reconsolidation phenomena: *What does, or does not, constitute a mismatch experience depends entirely on the specific makeup of the target learning at the time of mismatch*. That is a principle that I will refer to henceforth as *mismatch relativity*. It is essential for understanding the effects of reconsolidation procedures used in both laboratory studies and therapy sessions. In the small minority of reconsolidation research articles that do address the mismatch requirement, I have never seen mismatch relativity articulated explicitly; rather it is either tacitly assumed or asserted in an abstract manner (as in Bos et al., 2014, and Sevenster et al., 2013, 2014; for example, Bos et al. state, "The experience of a prediction error upon reactivation critically depends on the interaction between the original learning of the fear association and the memory retrieval" [p. 6]). Mindfulness of mismatch relativity is critical for consistent outcomes in utilizing reconsolidation in psychotherapy to bring about transformational change. Only by attending closely to the specific elements of a symptom-generating emotional learning can a psychotherapist reliably guide mismatch experiences that disconfirm those specific elements, as is necessary for their nullification and dissolution.

A question often asked by clinicians learning about reconsolidation is: When my panicky therapy client drives on the highway and the feared terrible fiery crash doesn't happen, that seems to be a mismatch experience, as needed to launch reconsolidation, yet it doesn't unlock or erase the learned fear. Doesn't this show that the model is incorrect? To clarify this, we need to apply the mismatch relativity principle and examine whether or not a mismatch experience actually took place. That begins with examining the detailed makeup of the target learning in question. In this case, the target learning is not that a car crash happens on every drive; rather it is that a crash *might* happen unpredictably on *any* drive. That learning is not mismatched or disconfirmed by an accident not happening on any one drive or on any number of drives. A safe, uneventful drive creates no prediction error and therefore does not induce deconsolidation, so the target learning is not revised and the model has not failed to apply.

This example naturally raises the question: For that target learning, what *would* be a mismatch experience? The knowledge that a crash might happen unpredictably on any drive is true as a recognition of existential reality, so no mismatch or disconfirmation of that knowledge is possible. However, that knowledge is not the entire learning maintaining the panicky dread of a fiery car crash. Some other learning is responsible for that emotional intensity, and it is for elements of that learning that mismatches *can* be created. The most common form of this other learning, though not the only possibility (see Ecker, 2003, or Ecker & Hulley, 2000, for an account of diverse learnings underlying anxiety and panic symptoms), is suppressed traumatic memory of the same or a similar kind, such as a car crash, a fiery explosion, the death of high school classmates in a head-on collision, a terrible scare from skidding on ice on a mountain road or from being pulled along very fast at 3 years old in a little wagon tied to the bicycle of an older sibling, and so forth. The suppressed state of the traumatic memory preserves its emotionally raw, unprocessed quality, including desperate fear and helplessness. De-suppression of the memory (in small enough steps to be tolerable) reveals a set of specific elements, each of which is a particular learning. It is these component learnings that can now be subjected to a mismatch experience. For example, the helplessness felt and learned in the original situation can in many cases encounter a mismatch experience through the technique of empowered reenactment, which is widely used in trauma therapy to create a vivid experience of potent self-protection in the original scene. For a detailed clinical example of that kind, see Ecker et al. (2012, pp. 86–91).

In summary of this section, the research findings on memory reconsolidation represent a nontheoretical set of instructions for bringing about transformational change in a target learning. These instructions specify that in order for a target learning to become destabilized and susceptible to being unlearned and nullified, it must be both reactivated and subjected to a mismatch or prediction error experience. The mismatch relativity principle has been introduced here, within the exercise of analyzing the occurrence of mismatch in published studies, to emphasize that

what is, and what is not, a mismatch experience is always defined in relation to the specific elements of the target learning and what the target learning "knows" or expects. This exercise of examining the role of mismatch in published studies will continue in each of the next two sections. (For numerous examples of creating mismatch experiences in psychotherapy, see Ecker et al., 2012, Chapters 3–6.)

Misconception 2: The Disruption of Reconsolidation Is What Erases a Target Learning

As soon as a reactivated target learning encounters a single brief but vivid mismatch experience, the target learning is deconsolidated and for about five hours is open to being changed or erased at the level of its synaptic encoding. Erasure is the focus of this article, because it is erasure that is experienced clinically as liberating, transformational change, that is, complete and permanent disappearance of an unwanted behavior or state of mind.

As noted above, erasure of a deconsolidated target learning has been accomplished by researchers either by guiding new learning that nullifies the target learning or by applying chemical agents. Those two processes of erasure are fundamentally different.

Chemical agents used for this purpose are those that block some step in the complex cellular and molecular process by which a memory circuit restabilizes into a consolidated state (for a review, see Reichelt & Lee, 2013). Administered just before or after a target learning is destabilized, these chemical agents selectively act upon only deconsolidated, destabilized memory circuits without affecting consolidated ones. This blockage of the reconsolidation of the target memory circuits impairs and destroys these circuits, erasing the target learning by disrupting the very process of reconsolidation. This disruption takes effect not immediately upon administration, but when restabilization would normally happen, about five hours after initial destabilization.

In contrast, erasure by new learning is understood by researchers as de-encoding and/or reencoding the target learning's synapses, unlearning and nullifying the prior content of that learning, but leaving the neurons and synapses operating normally and allowing the natural restabilization/reconsolidation of the circuits to occur. This results in memory circuits that no longer contain the target learning. Erasure by new learning occurs through the *utilization* of the reconsolidation process rather than through its disruption. One might characterize this type of erasure as disrupting the *content* of the target learning, but the reconsolidation process itself is not disrupted. Thus, referring to this type of erasure as a "disruption of reconsolidation" is a misconception and a misrepresentation of the actual process.

Most of the chemical agents successfully used in animal studies to disrupt reconsolidation are unsuitable for use with humans due to toxicity, side effects, or slowness of action (Schiller & Phelps, 2011). However, with human subjects the beta-adrenergic blocker propranolol is safe and has been tested in numerous studies ranging from Pavlovian (associative) fear conditioning to genuine PTSD conditions in clinical trials, as reviewed by Agren (2014). Results have varied widely for both associative fear conditioning and genuine PTSD. For associative conditioning, full erasure by propranolol was demonstrated by Kindt et al. (2009) and Soeter and Kindt (2011), but Bos et al. (2014) measured no reduction of fear at all. Bos et al. acknowledged, "The current findings clearly indicate that we did not trigger reconsolidation during memory reactivation" (p. 6). They offered the speculation that the cause of the negative result appeared to be a failure of their reactivation procedure to generate the required memory mismatch/prediction error experience, and they drew the lesson that "Future studies may benefit from protocols that are explicitly designed to assess and manipulate prediction error during memory retrieval" (p. 7). For PTSD, Brunet et al. (2011) measured a significant reduction of symptoms due to propranolol, but Wood et al. (2015) reported no reduction of symptoms using either propranolol or mifepristone, a glucocorticoid blocker that interferes with the neural (and other) effects of the stress hormone cortisol. Wood et al., in discussing various possible causes of their negative results, gave no consideration or mention of the requirement for memory mismatch. It seems probable that in chemical disruption/PTSD studies that did achieve symptom reduction, the procedure included mismatch unwittingly. For example, Brunet et al., unlike Woods et al., had subjects speak out their account of a traumatic experience to an interviewer, thus creating what trauma therapists term *dual focus*, an experiential state in which attention is simultaneously directed to a safe external environment and an internal traumatic memory. Dual focus maintains a dissociation and subjective distance between conscious attention and the attended contents of traumatic memory, and appears to be a critical ingredient in some trauma treatment procedures that achieve rapid, lasting depotentiation of traumatic memory and cessation of PTSD symptoms (see, e.g., Lee, Taylor, & Drummond, 2006). Dual fo-

cus creates memory mismatch in the form of a strong perception of safety concurrent with traumatic memory reactivation, as well as through facilitating internal accessing of existing personal knowledge that contradicts the contents of the traumatic memory schema (Ecker, 2015).

For clinical purposes, a natural process of erasure through unlearning rather than through chemical agents is of course greatly preferable, as a rule. The clinical feasibility and effectiveness of erasure through new learning have been demonstrated for symptoms and target learnings of many kinds, including but not limited to anxiety and posttraumatic symptoms (Ecker et al., 2012; Gray & Liotta, 2012; Xue et al., 2012). Thus, in the endogenous clinical context in particular it is a misconception to describe erasure as occurring through the disruption of reconsolidation, though the chemical approach is exactly that.

At the opposite end of the terminology spectrum, researchers sometimes use the phrase, "the enhancement of reconsolidation." This phrase denotes not a strengthening of the reconsolidation process itself, but a strengthening of the behavioral expression of a target learning that results, after its reconsolidation, from various procedures applied during the period of destabilization (for reviews, see Delorenzi et al., 2014; Forcato et al., 2014). The phrase therefore is essentially synonymous with "reconsolidation-induced enhancement of memory expression." Here we have yet another way in which the word reconsolidation is used by researchers, and again we see that for accurate understanding, readers of reconsolidation literature must consider carefully what an author's phrasing actually is intended to mean.

Misconception 3: Erasure Is Brought About During the Reconsolidation Window by a Process of Extinction: Reconsolidation Is an Enhancement of Extinction

Reconsolidation and extinction are different phenomena, with distinctly different effects, but misconceptions have developed for reasons described in this section.

In the process that has been known for a century as extinction, the target learning is not revised or erased, but only suppressed temporarily by new counterlearning, and the new learning is encoded in its own memory circuitry that is anatomically separate from, and in competition with, the circuits of the target learning. Later, however, the target learning wins that competition and reemerges into full expression (Bouton, 2004; Foa & McNally, 1996; Milner et al., 1998). In contrast, during the reconsolidation process, a target learning is destabilized and rendered susceptible to being revised fundamentally by new learning, which can either weaken it, strengthen it, alter its details, or fully nullify and erase it, and these changes are lasting, as described earlier.

Researchers have determined that reconsolidation and extinction are distinct and even possibly mutually exclusive processes at the behavioral, neural, and molecular levels (Duvarci & Nader, 2004; Duvarci, Mamou, & Nader, 2006; Merlo, Milton, Goozée, Theobald, & Everitt, 2014). "Reconsolidation cannot be reduced down to facilitated extinction" was the conclusion of Duvarci and Nader (p. 9269).

Despite those signature differences in process and effects produced, confusion about the relationship between reconsolidation and extinction nevertheless arises because to a degree they share certain operational and procedural patterns:

First, while the nullification learning that contradicts and erases a destabilized target learning can have any convenient procedural design, in many studies it has had the same design as a conventional extinction training—a series of numerous identical counterlearning/unreinforced trials—so it can be confused with and mislabeled as an extinction training, even though extinction is not actually involved.

Second, extinction, like reconsolidation, begins with the two-step sequence of reactivation and nonreinforcement (that is, a recueing of the target learning followed by nonoccurrence of what the target learning expects to happen, such as playing an audio tone without also delivering the mild electric shock that had previously been paired with the tone). It can be confusing and difficult to see how reconsolidation and extinction are two separate phenomena if they share the same initiating sequence of reactivation and nonreinforcement.

The main aim of this section is to dispel those two confusions, as well as to review various research findings that clarify the nature and relationship of reconsolidation and extinction and their differential triggering. In addition, the discussion will explore these questions: Are the empirical findings on reconsolidation and extinction understandable entirely, or only partially, in terms of the mismatch requirement and mismatch relativity (MRMR)? Does it prove instructive to consider how the findings would have to be understood in order for them to be entirely consistent with MRMR? The heuristic exploration of those ques-

tions in this section extends significantly the degree to which the mismatch/prediction error requirement has been applied, to date, to the interpretation of experimental findings.

Extinction-like procedure used for nullification learning. As noted earlier, for inducing erasure, some reconsolidation researchers have used a format of nullification learning during the reconsolidation window that has the same procedural structure as classical extinction training: a series of numerous identical counterlearning (nonreinforcement) experiences. The result of this procedure is not extinction (temporary suppression of the target learning), but rather the permanent erasure of the target learning, such that even strong recueing (reinstatement) cannot reevoke the target learning into expression. Nevertheless, these researchers have unfortunately labeled this procedure as "extinction" by naming it with such phrases as the "memory retrieval-extinction procedure," "extinction-induced erasure," "extinction during reconsolidation," or other phrases containing "extinction" (e.g., Baker, McNally, & Richardson, 2013; Clem & Huganir, 2010; Liu et al., 2014; Monfils et al., 2009; Quirk et al., 2010; Schiller et al., 2010; Steinfurth et al., 2014; Xue et al., 2012). Such labeling is a source of much misunderstanding of reconsolidation and extinction.

We are faced with these empirical facts: When a repetitive counterlearning procedure is applied to a target learning that is in a stable state when the procedure begins, the result is extinction—the target learning is suppressed but is intact and later reemerges into expression. However, when the same repetitive counterlearning procedure is applied to a target learning that is already in a destabilized/deconsolidated state, the result is erasure—the target learning's encoding is rewritten according to this new counterlearning, permanently nullifying the content of the target learning (Monfils et al., 2009; Schiller et al., 2010). Thus a particular learning procedure (repetitive counterlearning) can have extremely different neurological and behavioral effects depending on whether or not it is carried out during the reconsolidation window. So, any label for the erasure procedure that includes the term "extinction" is a misnomer that invites the misconception that reconsolidation utilizes and enhances the process of extinction.

The use of repetitive counterlearning during the reconsolidation window could more appropriately be labeled "nullification learning," "update learning," or "erasure learning," rather than "extinction training," to avoid conceptual errors and confusion. However, the "extinction" labeling has already become standard among researchers who use this particular procedure and is probably here to stay.

The great significance and usefulness of the reconsolidation window lies in the fact that, during that window, to unlearn is to erase, regardless of the specific form of the unlearning or nullification experience. The repetitive counterlearning procedure is a convenient protocol under the highly simplified conditions of laboratory studies but is not suitable in general for nullification of the far more complex emotional learnings encountered in real-life psychotherapy. There is a potentially unlimited number of formats in which nullification learning can occur in psychotherapy (for many examples of which, see Ecker et al., 2012).

The triggering of reconsolidation versus extinction. As already described, both reconsolidation and extinction begin with the two-step sequence of reactivation and nonreinforcement, that is, a recueing of the target learning followed by nonoccurrence of what the target learning expects to happen. What, then, determines whether reconsolidation or extinction is the result?

We know that the experience of mismatch (prediction error) is what triggers destabilization and reconsolidation, as discussed earlier. This seems to imply that when memory reactivation plus nonreinforcement create a mismatch experience, reconsolidation is triggered, whereas when reactivation plus nonreinforcement occur without creating a mismatch experience, the extinction process begins. Therefore, in order to understand what causes the triggering of reconsolidation versus extinction, it may be necessary to understand why reactivation with nonreinforcement creates mismatch in some circumstances but not in others. With that question in mind, it is instructive to examine a range of instances where reconsolidation or extinction was induced.

Many observations of the triggering of reconsolidation versus extinction have been made in animal studies through reactivating a CS-US target learning by presenting the unreinforced CS only, and then promptly applying a chemical agent that disrupts nonconsolidated or deconsolidated learnings but has no effect on stable, consolidated memory circuits. The findings of many such studies can be summarized in terms of how the effect of reactivating a target learning with CS-only presentations depends on their time structure. In the studies summarized here, the target learning was formed by two or more CS-US pairings with 100% reinforcement.

Reconsolidation is triggered. After a single brief

CS presentation, there is no extinction learning and the target learning is still at full strength (Nader et al., 2000; Eisenberg, Kobilo, Berman, & Dudai, 2003; Jarome et al., 2012; Merlo et al., 2014; Pedreira et al., 2004). In this case, prompt application of a chemical agent that blocks consolidation (and reconsolidation) disrupts the target learning, which is found to be significantly weakened or completely erased 24 hr later, indicating that the target learning was destabilized (deconsolidated) by the CS presentation, triggering the reconsolidation process. This implies that unreinforced reactivation by a single CS presentation does create a mismatch experience.

Extinction is triggered. After a single prolonged CS or a series of many short CS presentations, an influential extinction learning exists and largely suppresses the target learning, so the behavioral expression of the target learning is significantly diminished. In this case, prompt application of a consolidation-blocking chemical agent disrupts the newly formed, not yet consolidated extinction learning, and this restores the target learning to full strength (see, e.g., Eisenberg et al., 2003). The return of the target learning to full strength implies that the target learning was unaffected by the chemical agent and therefore was not in a destabilized state when the chemical agent was administered after the single prolonged CS or the series of many short CSs. This in turn implies that although the first of the many short CS presentations must have created a mismatch experience (as in the single brief CS situation), mismatch must have been terminated promptly by the ensuing CSs, restabilizing the target learning, despite the fact that each CS in itself would seem to be a nonreinforced reactivation that should maintain mismatch. These logical inferences have been corroborated by several studies, described below.

Neither reconsolidation nor extinction is triggered. For an intermediate number of unreinforced CS presentations, the target learning remains at full strength and a variety of chemical interventions that either disrupt or enhance reconsolidation or extinction have no effect on subsequent target learning expression/ This is understood to mean that neither reconsolidation nor extinction is underway (Flavell & Lee, 2013; Merlo et al., 2014; Sevenster, Beckers, & Kindt, 2014). Merlo et al. (2014) commented, "In the continuum of possible retrieval conditions, reconsolidation and extinction processes are mutually exclusive, separated by an insensitive phase where the amount of CS exposure terminates the labilization of the original memory, but is insufficient to trigger the formation of the extinction memory" (p. 2429).

Of the many studies that have reported the kinds of findings summarized above, very few also addressed the question of *why* one, or a few, or many nonreinforced CS reactivations have the observed effects of triggering or not triggering the destabilization and reconsolidation of a target learning. Here the focus of discussion now turns to an examination of why reactivation with nonreinforcement creates mismatch and triggers destabilization in some circumstances and not in others.

MRMR model of triggering reconsolidation or extinction. The critical role of mismatch in triggering reconsolidation was first reported by Pedreira et al. (2004), as noted earlier. A mismatch exists when there is a significant discrepancy between what is expected and what is actually experienced. Thus reconsolidation and all of its complex cellular and molecular machinery is an experience-driven phenomenon.

A growing number of experimental observations require a view of mismatch as being a fluid, dynamical quality of experience that can vary on a moment-to-moment basis with the passage of time and with new experiences (see, e.g., Jarome et al., 2012; Merlo et al., 2014; Sevenster et al., 2014). The following paragraphs apply that dynamical view of mismatch and offer the proposal that the reconsolidation/extinction dichotomy may be largely or completely governed by the mismatch requirement and mismatch relativity (MRMR), as defined earlier.

To explore this proposal and show that MRMR potentially could be responsible for a wide range of reconsolidation and extinction phenomenology, what follows is a discussion of how several significant research findings can be understood as being entirely MRMR effects. The discussion shows specifically how reactivation with nonreinforcement creates mismatch, triggering destabilization, in some circumstances and not in others.

Single CS-only presentation. First consider the simplified case in which the sequence of reactivation and mismatch (nonreinforcement) occurs only once and is not repeated. For example, if a conditioned stimulus (CS, such as a blue light, an audio tone, or a particular physical environment) has previously been paired repeatedly with a mild electrical shock (unconditioned stimulus, US) just before the CS turns off, what happens subsequently if the CS turns on and then turns off unreinforced (no shock), just once?

The CS turning on immediately reactivates the target learning, generating the expectation of receiving a shock. Several studies have shown that the time

period from CS onset to CS offset (with no US) controls whether reconsolidation or extinction occurs, and that whichever process occurs is triggered by CS offset and does not begin before CS offset (Kirtley & Thomas, 2010; Lee, Milton, & Everitt, 2006; Mamiya et al., 2009; Pedreira & Maldonado, 2003; Pedreira et al., 2004; Pérez-Cuesta & Maldonado, 2009; Suzuki et al., 2004). In these studies, the CS onset-to-offset time that originally created the target learning (with CS-US pairing) was short, typically in the 1- to 5-min range. Subsequently, CS onset and offset with no shock induced reconsolidation if the CS onset-to-offset time was less than about one hour (destabilizing the target learning, making it revisable by new learning during the next five hours), but it induced extinction if the CS onset-to-offset time was more than about an hour (that is, the target learning remained stable and a separate counterlearning formed in competition with the target learning).

To my knowledge, researchers have not proposed or identified a mechanism that explains the observations that short versus long periods of CS onset-to-offset induce reconsolidation or extinction, respectively. If MRMR (mismatch requirement and mismatch relativity) are the cause, they would operate as follows.

Consider first the case where, after the target learning was formed by a series of CS-US pairings (100% reinforcement), there is a single unreinforced CS reexposure with short duration of onset-to-offset (about equal to the CS onset-to-offset time in the original CS-US training), triggering reconsolidation. As noted earlier in describing the study by Nader et al. (2000), the absence of the expected US creates a decisive US mismatch that destabilizes the target learning.

Next, consider the case where a single short CS-only presentation occurs after the target learning has been formed by a partial reinforcement schedule. Partial reinforcement results in the subject expecting not that the US will *always* occur following the CS, but only that it *might* occur. In this case MRMR predicts that a single short CS-only presentation would not constitute a decisive mismatch and would therefore not induce destabilization. The target learning would remain stable, as was found to be the case by Sevenster et al., (2014), who used a 50% reinforcement schedule to create a fear learning and showed that a single, short CS-only presentation did not induce destabilization. (Learnings created by partial reinforcement require significantly more extinction trials to suppress, as compared with learnings created by continuous reinforcement, because the initial trials are not experienced as a decisive mismatch or prediction error. This is the "partial reinforcement extinction effect", e.g., Pittenger & Pavlik, 1988.)

A special case of learnings formed by partial reinforcement is single-trial learning, which again results in the expectation that the US *might* occur following the CS, not that it will always occur. Here too, a subsequent single CS-only reexposure does not create a decisive US mismatch. This was the case in a study of conditioned taste aversion in rats reported by Eisenberg, Kobilo, Berman, and Dudai (2003). A single training trial produced lasting avoidance behavior, but a single CS-only reexposure did not destabilize the target learning (as evidenced by no disruption of the target learning from anisomycin administered immediately after the CS reexposure). When Eisenberg et al. used a series of two CS-US pairings to create the target learning instead of one pairing, creating strong US-expectancy due to the 100% reinforcement, the same single CS-only presentation now did trigger destabilization, allowing disruption by anisomycin, implying that now a mismatch was created.

The foregoing examples and those below illustrate that the principle of mismatch relativity emphasizes a detailed consideration of all features of the target learning, in order to predict accurately whether or not a given reactivation procedure creates a decisive mismatch/prediction error experience in relation to the target learning in question. Mismatch relativity also alerts us to understand that any given successful experimental destabilization procedure reveals not the inherent, fundamental properties of the brain's reconsolidation process, but only a way of creating mismatch relative to the particular features of the target learning created by the researchers.

Next we have to consider why, according to MRMR, a single long-duration, unreinforced CS causes extinction rather than reconsolidation. For example, Pedreira et al. (2004) found that a 2-hr CS presentation failed to destabilize the target learning into reconsolidation and instead produced an extinction learning. The original target learning began with a 5-min exposure to the CS (the training chamber), and then the US, a simulated predator, was presented every 3 min, 15 times. The fact that a 2-hr unreinforced CS reexposure did not trigger reconsolidation means, according to MRMR, that the 2-hr CS did not function as a mismatch of the target learning's 5-min exposure before the US began to appear. Why it did not function as a mismatch can be inferred from mismatch relativity: Relative to the original learning experience with its 5-min CS exposure, a 2-hr CS reexposure presumably was an experience that qualitatively differed subjec-

tively from the original learning to such a degree that the 2-hr reexposure experience registered as a contextually unrelated experience, not as a mismatch or even as a reminder of the 5-min experience encoded in the target learning. Thus the experience of mismatch, which would have occurred with CS offset for some time after the 5-min point, no longer occurred with CS offset at the 2-hr point. Due to the relativity of mismatch, an experience that is too greatly dissimilar to the original learning experience does not function as a reminder or mismatch of it, so the target learning does not destabilize, which causes the new learning driven by the unreinforced CS to form separately as an extinction learning.

This example suggests the possibility that the presence or absence of mismatch can change over time during CS presentation, which will figure importantly in the analysis of multitrial extinction below. The *general principle of mismatch relativity* is that experience B is a mismatch of expected experience A if B resembles A enough to register as a reminder and repetition of A, while also containing saliently discrepant or novel features relative to those of A.

Testable predictions arise from the MRMR interpretation above. For example, the original learning could be created by a 2-hr CS with the US occurring in the final minutes, with a repetition of that CS-US beginning 30 min later, and so on three or four times. Mismatch relativity predicts that now a 2-hr CS-only reexposure *would* serve as a reminder and mismatch and would achieve destabilization; and perhaps now a 5-min CS reexposure without US would fail to do so because the dissimilarity might be too great for the short reexposure to serve as a reminder of the extremely long duration in the target learning.

If the mismatch requirement and mismatch relativity govern whether reconsolidation or extinction occurs, then there is no absolute time duration of unreinforced CS reexposure that defines the boundary between the two phenomena. Rather, the time boundary (the largest and smallest unreinforced CS reexposure durations that function as a mismatch and trigger reconsolidation) would depend on the original learning's CS duration. That predicted dependency of the reconsolidation/extinction time boundary on the time structure of the original training serves as another test of the MRMR model and could be directly measured by extending existing studies to vary the reinforced CS duration in the original learning while measuring the maximum and minimum unreinforced CS reexposure durations that trigger reconsolidation.

Multiple CS-only trials. The foregoing paragraphs addressed reconsolidation and extinction having a shared initiating sequence in the case of single-trial, nonreinforced CS reexposure. Next, consider the case of a series of numerous identical counterlearning experiences of reactivation and nonreinforcement, that is, the classical extinction procedure. It is well known that the multitrial extinction procedure does not destabilize or erase the target learning, yet, as discussed above, a single short CS-only trial does do so (for a target learning created by multiple CS-US presentations). This raises the question: Given that the first CS-only presentation mismatches and destabilizes, how does the state of the target memory evolve with each successive CS-only presentation, such that there is no destabilization and no erasure resulting from the series?

It will be assumed in what follows that the target learning was formed originally by a series of CS-US pairings having the same time structure as in the subsequent extinction procedure. This assumption allows for an unambiguous delineation of the logic of MRMR in this instance, but it does not limit the relevance of MRMR to only these assumed conditions as a special case.

The question requiring an answer is this: Why does the standard extinction procedure fail to destabilize and then erase the target learning, given that the first CS-without-US in the series mismatches and destabilizes the target learning and the ensuing series of CS-without-US experiences could be expected to then function as a nullification learning that erases the target learning? MRMR implies that because the result of multiple-trial counterlearning *is* extinction rather than erasure, it must be the case that multiple-trial counterlearning does not sustain a mismatch experience long enough for erasure to occur. The question therefore becomes: Why does multiple-trial counterlearning not sustain a mismatch that keeps the target learning destabilized and allows erasure to occur, even though every unreinforced trial in the series seems to be a mismatch of the expected CS-US pairing? The answer to that question has emerged from several recent studies (Jarome et al., 2012; Merlo et al., 2014; Sevenster et al., 2014).

Jarome et al. (2012) paired sound and footshock to create a learned fear of the sound in rats, and then, 1 day later, applied anisomycin immediately following either a single unreinforced CS or two unreinforced CSs that were separated by 1 hr. (Longer periods were also tested.) On the next day, tests of fear in response to the CS showed that after single-CS reexposure, the fear learning had been largely disrupted and erased by

anisomycin, indicating destabilization had occurred, but after the two-CS exposure there was no reduction in fear due to anisomycin. This implies that the second CS rapidly changed the neurological condition of the target learning, either returning the target learning to stability (according to the standard interpretation of anisomycin's effect) or, alternatively and more conjecturally, launching the updating/erasure process and thereby altering the prevailing molecular mechanisms such that even though destabilization persisted, anisomycin no longer caused disruption (T. J. Jarome, personal communication, 24 November, 2014).

Sevenster et al. (2014) also demonstrated rapid changes in target memory condition caused by successive nonoccurrences of the US when it was expected according to the original training. A fear learning was created in human subjects by pairing an image with a wrist shock, and the effects of 0, 1, and 2 nonreinforcements by CS-only presentations were studied. Whether the target learning was destabilized was determined by administering propranolol, which disrupts destabilized CS-US fear learnings in humans (Kindt et al., 2009; Soeter & Kindt, 2011). This revealed that a single nonreinforcement functioned as a mismatch and destabilized the target learning, launching reconsolidation, but 0 and 2 nonreinforcements did not. This indicates again, as in Jarome et al. (2012), that a target learning destabilized by an initial unreinforced CS presentation is restabilized by the second unreinforced CS presentation. Here, however, the time interval from first to second CS was 40 s rather than 1 hr.

Importantly, in addition to measuring the level of fear in response to each unreinforced CS presentation, during each unreinforced CS presentation Sevenster et al. (2014) also measured subjects' subjective rating of their US-expectancy, that is, the felt level of anticipation that the shock would occur at the end of the current 7-s CS image presentation. US-expectancy was rated by subjects on a scale from –5 (certainty of not happening) to 0 (uncertain) to +5 (certainty of happening). This revealed that as the first nonreinforcement was about to happen, average US-expectancy was strong at +3.8, which created a mismatch experience when the US did not occur, but as the second nonreinforcement was about to happen, average US-expectancy had decreased sharply to 0.9, close to the "uncertain" level and presumably too low to create a mismatch experience when the US did not occur. The first US nonoccurrence had created new learning that reduced the US-expectancy created by the original training, and it was this reduced US-expectancy that then encountered the second US nonoccurrence. The direct implication is that immediately after the first nonoccurrence of the US when the US would be expected on the basis of the original learning, subjects were in the experience of mismatch, so the target learning was found to be destabilized, but immediately after the second nonoccurrence of the US when it would be expected according to the original learning, subjects were not in an experience of mismatch, so the target learning was found to be stable.

Thus the presence or absence of a mismatch experience evidently switches destabilization on or off, respectively, in real time. By comparing their measurements of fear and US-expectancy, Sevenster et al. also showed that the sharp drop in self-reported US-expectancy was not accompanied by a decrease in physiologically measured fear. This means that with accumulating unreinforced CS presentations, US-expectancy began to decrease, evidently returning the target learning to stability, before there had been enough counterlearning to initiate the formation of an extinction learning. This is consistent with other studies indicating that reconsolidation and extinction are mutually exclusive phenomena (e.g., Duvarci & Nader, 2004; Duvarci et al., 2006; Merlo et al., 2014). Thus after two US nonoccurrences, the target learning was stable and neither reconsolidation nor extinction was occurring.

Observations by Merlo et al. (2014) provide further corroboration that accumulating unreinforced CSs switch off reconsolidation before extinction is in effect. After 1, 4, 7, and 10 presentations of an unreinforced CS, Merlo et al. tested a conditioned fear learning in rats for susceptibility to alteration by various chemical agents applied locally in the basolateral amygdala (BLA). After the fourth CS presentation, the target learning was no longer chemically alterable, meaning that it was no longer in a destabilized state in the BLA. Furthermore, there were no behavioral or molecular markers of extinction, so neither reconsolidation nor extinction was occurring. Merlo et al. infer from these findings that the target learning's state (stable or unstable) may be reset on a moment-to-moment basis as CS-only presentations accumulate.

In light of the studies just reviewed, there is now growing evidence indicating why the multiple-trial counterlearning of conventional extinction training does not sustain mismatch or destabilization and does not erase the target learning: A target learning's state of destabilization and erasability evidently is maintained by the ongoing presence of the experience of mismatch or prediction error and can quickly terminate if the experience of mismatch or prediction error

terminates. Thus the mismatch requirement first identified by Pedreira et al. (2004) functions as a dynamic on/off switch. The destabilized state can be toggled on/off or off/on as mismatch is subjectively present/absent or absent/present, respectively. (Destabilization lasts for a time window of about five hours, as described earlier, if, once destabilized, the target learning is not further recued by additional experiences.)

In this picture of dynamic mismatch bipolarity, the principle of mismatch relativity governs how each successive unreinforced CS affects the target learning. In other words, the target learning consists of expectations that can be revised by an individual CS in the series if that CS deviates from the expectations extant just prior to that CS. The evolving expectational content of the target learning must be considered in detail in order to understand the effect of each successive CS.

In short, the studies by Jarome et al. (2012), Merlo et al. (2014) and Sevenster et al. (2014) indicate that MRMR principles determine the effects of the multiple-trial extinction procedure, as follows. With a target learning created by CS-US pairings with continuous (100%) reinforcement, the subject has the expectation that the US always accompanies the CS. The first CS-without-US presentation is therefore a decisive mismatch (that is, the nonoccurrence of the US creates strong surprise and a felt inability to anticipate accurately) because the learned expectation that the US always accompanies the CS has now encountered the mismatching current perception that the US does *not* always accompany the CS. This has two effects. First, this strong mismatch abruptly destabilizes the target learning. Second, the nonoccurrence of the expected US creates new learning that the US does not always accompany the CS. This new learning persists and results in a sharply reduced US-expectancy during the second unreinforced CS presentation. The second US nonoccurrence is therefore not experienced as a mismatch, because now there is no surprise or prediction error felt. Rather, there is now an experience that this US nonoccurrence is in accord with the expectation that the US might or might not happen. This termination of mismatch terminates destabilization, because destabilization is dynamically maintained in real time by the persisting experience or context of mismatch. The target learning shifts into a stable state. (Whether the new learning created by the first US nonoccurrence immediately updates the target learning's model of the CS-US association is not yet known, though molecular findings by Monfils et al., 2009, and Jarome et al., 2012, seem to imply that the destabilization event does not also launch updating. Possibly, updating is launched only if mismatch saliently persists after destabilization occurs.)

In that way, the multitrial extinction procedure destabilizes and then quickly restabilizes the target learning before erasure can occur. With the third unreinforced CS, presumably there would no longer be any surprise or mismatch whatsoever. With the target memory in a stable state as CS repetitions continue, the target learning remains intact and the new learning created by the ongoing series of harmless CS presentations forms separately. That is the MRMR account of standard multitrial extinction.

Standard multitrial extinction training was converted into an effective erasure procedure in studies by Monfils et al. (2009) and Schiller et al. (2010), as described in a previous section, simply by increasing the time interval between the first and second CS-only presentations. Why that seemingly minor alteration of extra time in the first interval could make such a qualitative and drastic difference in outcome becomes apparent by applying the MRMR model and examining the timing difference in terms of its mismatch effects. That exercise is carried out here next for the Monfils et al. study, as this section's final and most intricate example of applying the MRMR model. The Schiller et al. study, which had human subjects, is described in the Appendix of this article.

In the procedure that Monfils et al. (2009) used with rats, the original fear acquisition consisted of three CS-US (tone-shock) pairings every 3 min, with CS duration of 20 s, ending with a half-second shock. On the next day, the interval between the 19 CS-only presentations was also 3 min, except for a longer initial interval between CS1 and CS2 of 10 min or 1 hr, both of which resulted in long-term erasure of the learned fear, which could not be reevoked later by either the CS or the US. The control group did not have the longer initial interval, making the procedure a conventional extinction training, and for these rats the learned fear was later reevoked.

The functioning of the erasure procedure is understood as follows according to the MRMR model. It can be reliably assumed, based on many other studies as described earlier, that CS1 created a US mismatch that quickly destabilized the target learning. Therefore, after CS1 the target learning was open to being updated by any variations in the procedure relative to the original training. An immediate and salient variation was the appearance of CS2 defining a 10-min or 1-hr interval since CS1, far longer than the 3-min interval expected based on the original acquisition training. The already destabilized target learning was updated according to that longer interval, so the timing expec-

tation going forward was now that after each colored square there would be *either* 3 min or the longer time (10 min or 1 hr). The longer interval defined by CS2 also was a mismatch of timing expectations, and that second mismatch experience, coming while the target learning was already destabilized, would only have made the destabilized state more robust.

However, as discussed earlier, CS2 would not create a US mismatch as CS1 had done. Thus CS2 ended the US mismatch while creating a timing mismatch. Did the target learning restabilize due to the termination of US mismatch, or did it remain destabilized due to the timing mismatch?

One indication comes from Jarome et al. (2012), who largely replicated this situation with two CSs 1 hr apart, as described earlier. Anisomycin applied immediately after the second CS did not reduce fear in response to another CS 1 day later. That is usually understood as meaning that the target learning was stable, because anisomycin disrupts a destabilized memory. However, while anisomycin disrupts a memory that is newly destabilized but not undergoing updating, its effect on a memory during the updating process is not known. On the cellular and molecular level, the process that destabilizes the target learning and the process that updates/erases it appear to be two distinct though coupled processes (Jarome et al., 2012; Lee et al., 2008). Updating occurs through a molecular mechanism that potentially alters the molecular processes involved in the memory's dynamical progression. Anisomycin is a protein synthesis blocker. If the updating/erasure mode eliminates the protein synthesis that a nonupdating memory requires for restabilization, then anisomycin would not have a disruptive effect on a destabilized memory that is undergoing updating, as Timothy J. Jarome (personal communication, 24 November, 2014) has pointed out.

Only further research can settle the question of whether the target learning in Monfils et al. (2009) was stable or unstable after CS2, so here the MRMR account must branch to follow both possibilities.

If CS2 caused restabilization due to elimination of US mismatch, the fact that erasure then resulted from CS3 to CS19 implies that CS3 must have destabilized the target learning yet again. That in turn implies that a new mismatch experience was created by CS3, which in turn directs us to identify the procedural elements that created that mismatch. CS3 occurred 3 min after CS2, which created another timing mismatch because an interval of 10 min or 1 hr was expected after the updating driven by the longer interval from CS1 to CS2. This timing mismatch created by CS3 onset would have redestabilized the target learning. (This is a prediction of MRMR that could be tested by extending the Jarome et al. study to include a CS3 that occurs 3 min after CS2, and conducting molecular tests for destabilization promptly after CS3.) Having been destabilized by CS3, the target learning would then be updated by the 3-min interval from CS3 to CS4, as well as by CS4 itself as an experience that the CS is harmless. The condition required for erasure to be occurring is having the target learning in a destabilized state concurrent with a fresh or freshly remembered experience that contradicts and disconfirms the target learning's expectations or model of how the world functions. Erasure of the CS-US association may have been underway following CS3, and more so when the destabilized target learning encountered CS4. The next 3-min interval from CS4 to CS5 would have been as expected, ending the experience of mismatch, which may have terminated the destabilized state and, with it, the erasure process also. That would imply that erasure was fully accomplished by CS1 through CS4, and that CS5 through CS19 were not needed, which could be tested by repeating the Monfils et al. (2009) experiment without CS5 through CS19 and seeing whether or not the results are unchanged.

If, on the other hand, CS2 maintained prior destabilization by creating a timing mismatch, it is probable that CS2 began the erasure process. Then, after CS2, the effects of the procedure's time intervals and CSs would be the same as described in the previous paragraph (with the exception that CS3 would now maintain rather than reinitiate destabilization). Thus the question of whether or not CS2 restabilizes the target learning does not influence the outcome, according to the MRMR model.

There is an additional possibility for how the updating process could affect the unfolding dynamics of the target learning. Engagement of the updating/erasure process possibly could maintain the destabilized state even without an ongoing experience of mismatch. In order for the adaptive process of updating to proceed, destabilization must be in effect during the new learning that is driving the updating (otherwise what occurs is not updating but a separate encoding of new learning, as in extinction). Therefore, because the adaptive success of updating depends on destabilization, it is likely that whenever new learning during destabilization is driving updating, the destabilized state is maintained directly by molecular signals from the updating/erasure process and is no longer dependent on an ongoing experience of mismatch, so that updating will not be prematurely terminated by an absence of mismatch causing a return to stability. This could

be termed *maintenance of destabilization by updating*, or MDU. Presumably, at the point where no further encoding, reencoding, or de-encoding is occurring for updating, the molecular signals driving MDU cease, and the updated target learning then returns to stability promptly. It is well established that a target learning returns to stability after about five hours if there has been destabilization but no updating (such as by a single short CS-only presentation; Duvarci & Nader, 2004; Pedreira et al., 2002; Pedreira & Maldonado, 2003; Walker et al., 2003), but if updating has also occurred, it is possible that restabilization occurs through a different molecular process with a different temporal characteristic.

If MDU is included in the MRMR framework, the picture becomes one of memory mismatch initiating and maintaining destabilization until memory updating is occurring, from which point destabilization is maintained directly by the updating process and continues until updating terminates either due to saturation of encoding or cessation of new learning input. The MRMR account of the erasure procedure used by Schiller et al. (2010; see Appendix) more strongly requires and implies MDU. Obviously, further studies are needed to test these possibilities and clarify how the stability status of the target learning evolves with each successive CS presentation in various procedural configurations.

The above analysis of results of Monfils et al. (2009) illustrates how assuming the results of experimental procedures to be governed by MRMR principles can illuminate previously unrecognized dynamics and resolve dilemmas of interpretation and apparent inconsistencies between studies. The foregoing MRMR accounts are offered heuristically, to indicate the kinds of phenomenology that are brought into consideration by the MRMR framework.

The MRMR model has the systemic implication that the neural and molecular processes of reconsolidation or extinction are under the direct control of brain regions and circuits that assess, detect, and signal mismatch (prediction error) occurring between learned expectations and currently experienced temporal, spatial, and/or somatosensory perceptions (as well as, in the human clinical context, attributed meanings). A direct indication of that supervening role of mismatch detection can be seen in the findings of Reichelt, Exton-McGuinness, and Lee (2013) and Sevenster et al. (2014). The latter showed, as described above, that the switching off of reconsolidation during a series of unreinforced CSs (reminders) can be directly attributed to a sharp decline in US-expectancy and corresponding termination of the experience of mismatch. Reichelt et al. demonstrated that a successful mismatch procedure for destabilizing goal-tracking memory in rats, allowing chemical disruption, became ineffective as a result of impairment in the ventral tegmental area, a brain region that is believed to be critical for generating prediction error signals but is not a site of memories undergoing reconsolidation. Understanding how mismatch signals are generated and how they supervene upon the machinery of reconsolidation and extinction may prove to be particularly fruitful for arriving at dexterous control of these phenomena. For a discussion of prediction error signal generation and ideas for future research, see Exton-McGuinness et al. (2015).

In summary, from the MRMR perspective, the triggering of reconsolidation versus extinction by any particular reactivation procedure is to be understood in terms of the presence or absence of a mismatch (prediction error) experience at each point of the procedure. In addition to identifying what may control the reconsolidation/extinction dichotomy, the MRMR model provides a new, fundamental understanding of classical extinction by identifying why repetitive counterlearning creates a separate learning in competition with the target learning, rather than erasing the target learning. The MRMR account potentially unifies a broad range of reconsolidation and extinction phenomena.

Misconception 4: Anxiety, Phobias, and PTSD Are the Symptoms That Memory Reconsolidation Could Help to Dispel in Psychotherapy, but More Research Must Be Done Before It Is Clear How Reconsolidation Can Be Utilized Clinically

This section really comprises a blend of two misconceptions. First is the view that for clinical use, reconsolidation could be suitable for helping to dispel learned *fears* of various kinds, with symptomology such as PTSD, phobias, panic attacks and anxiety. This impression probably stems from the consistent tendency of researchers to comment in their research articles that reconsolidation has significant potential for treatment of PTSD and anxiety disorders. Researchers have to be ultraconservative in what they write so that everything they propose is firmly based on what is known according to the current state of research. Reconsolidation is relevant as a candidate treatment only for conditions that are maintained by memory, and for a brain researcher there is no risk that PTSD could be unrelated to memory and therefore no risk of a departure from the required empiri-

cism. Furthermore, fear is the most reliably detectable and measurable type of negative emotional response, so that researchers preferentially envision applications of the reconsolidation process to fear symptomology. Clinicians, however, regularly observe phenomenology showing that an extremely wide range of other conditions also are rooted in and driven by implicit memory (Ecker et al., 2012; Ecker & Toomey, 2008; Toomey & Ecker, 2007; Schore, 2003; Siegel, 2006). Nevertheless, it is not conventional practice for neuroscience researchers to reference that body of knowledge.

In fact, reconsolidation research has already demonstrated that the process applies to many types of learning other than fear learnings—for example, appetitive (pleasure) learnings (Stollhoff et al., 2005), operant (instrumental) learnings (Exton-McGuinness, Patton, Sacco, & Lee, 2014; Gallucio, 2005), spatial learnings (Rossato et al., 2006), object recognition learnings (Rossato et al., 2007), motor task learnings (Walker et al., 2003), taste recognition learnings (Rodriguez-Ortiz, De la Cruz, Gutierrez, & Bermidez-Rattoni, 2005), human declarative learnings (Forcato et al., 2007), human episodic learnings (Hupbach, Gomez, Hardt, & Nadel, 2007), and emotionally compelling human preferences (Pine, et al., 2014), among others. In fact, to my knowledge, as of this writing, all tested types of learning and memory have been found to submit to the process of reconsolidation.

That is extremely good news for psychotherapy, as the learnings that underlie and drive individuals' problems and symptoms are of many different kinds and not necessarily fear-based. Examples from my own practice of non-fear-based implicit emotional learnings brought into direct awareness include: the expectation to be allowed no autonomy, with reliance on secrecy and lying to maintain personal power; the heartbreak-laden memory of father abandoning the family when the client was 4 years old and the ensuing conviction that the cause was her own deficiency; and the expectation of severe devaluing and derision from others for any mistake or misstep, generating paralyzing states of shame and inhibition.

The second misconception in this category is this: In reconsolidation research articles, the authors typically comment that much more research must be done before it is clear how reconsolidation can be utilized in psychotherapy. This is hardly the case. In reality, for over a decade before neuroscientists' discovery in 2004 of the sequence of experiences that triggers reconsolidation (Pedreira et al., 2004), psychotherapists had been knowingly guiding clients through that sequence, having recognized from clinical observations that it was responsible for transformational therapeutic change (as described below). Furthermore, since 2006, psychotherapists have been translating reconsolidation research findings into successful therapeutic methodology. In 2006 I gave a keynote address to a conference of psychologists and psychotherapists (Ecker, 2006), describing the critical sequence of experiences that is required, according to reconsolidation research, for erasing a target emotional learning. In that talk, a clinical case example from my practice illustrated the guiding of that sequence and the resulting permanent disappearance of a longstanding, intense emotional reaction. In subsequent years, many articles and conference talks have presented the critical sequence in many clinical case examples of using it to decisively dispel a wide range of symptoms and problems (e.g., Ecker, 2008, 2010, 2013; Ecker, Ticic, & Hulley, 2012, 2013a,b; Ecker & Toomey, 2008; Sibson & Ticic, 2014).

Note that according to current neuroscience, memory reconsolidation is the only known process and type of neuroplasticity that can produce what we have been observing clinically: the abrupt, permanent disappearance of a strong, longstanding, involuntary emotional and/or behavioral response, with no further counteractive measures required. So, in psychotherapy we have been guiding the same well-defined sequence of experiences and observing the same distinctive signs of erasure as reconsolidation researchers have. We have applied the process successfully to the real-life, highly complex emotional learnings that underlie and maintain symptoms of many different types (see citations in the previous paragraph). Also, successful clinical use of protocols designed to induce reconsolidation and erasure have been reported by Högberg et al. (2011) and Xue et al. (2012). The latter demonstrated, in a controlled study, a strong degree of elimination of heroin addicts' cue-induced craving for heroin.

Thus the new era of the psychotherapy of memory reconsolidation is well underway. It had a curious birth: From 1986 to 1993, my clinical colleague Laurel Hulley and I closely scrutinized the occasional therapy sessions in our practices in which abrupt, liberating change had somehow occurred—the lasting cessation of a problematic pattern of emotion, behavior, cognition and/or somatics. Finally we identified a sequence of experiences that was always present, across a wide range of clients and symptoms, whenever such transformational change occurred. We developed a system of therapy focused on facilitating that key sequence of experiences right from the first session of therapy,

and found that working in this way made our sessions far more consistent in producing transformational therapeutic breakthroughs. We began teaching this methodology in 1993 at a workshop in Tucson, Arizona, followed by our first published account of it in the volume *Depth Oriented Brief Therapy* (Ecker & Hulley, 1996). Subsequently the same sequence of experiences emerged in reconsolidation research, providing corroboration of our clinical observations by empirical, rigorous studies. It seemed remarkable that the same process for erasing emotional learnings had been discovered independently in the therapeutic domain of subjective, experiential phenomenology and in the laboratory domain of research on animal memory circuits. In hindsight that convergence now seems most natural, because any process of lasting change that is truly innate to the brain would inevitably be apparent in both domains.

Our psychotherapy system, now known as *coherence therapy*, guides the series of experiences required by the brain for reconsolidation and erasure to occur, creating transformational change (Ecker & Hulley, 2011). It is the only system of psychotherapy that explicitly calls for and maps directly onto the process identified in reconsolidation research, but there are many other systems of therapy in which the same process also takes place, albeit embedded within methodologies conceptualized quite differently. It is clear that no single school of psychotherapy "owns" the process that induces memory reconsolidation, because it is a universal process, inherent in the brain. In any therapy sessions, the occurrence of transformational change can now be presumed to mean that reconsolidation and erasure of the target response have occurred, whether or not the therapist was knowingly guiding that process. Toward confirming that universality, we began an ongoing project of explicitly identifying the embedded steps of the reconsolidation and erasure process in published case examples of various forms of psychotherapy (Ecker et al., 2012; see Chapter 6).

Thus, knowledge of memory reconsolidation can enhance the effectiveness of individual psychotherapists, but more importantly, it also translates into a unifying framework of psychotherapy integration in which the many different systems of therapy form a huge repertoire of ways to guide the brain's core process of transformational change. This framework gives practitioners of different therapies a shared understanding of their action and a shared vocabulary for their action. Of course, not all systems of psychotherapy are equally consistent and reliable in fulfilling the sequence required by the brain for erasure of a target learning, and this too becomes apparent through this unified framework.

Misconception 5: Emotional Arousal Is Inherently Necessary for Inducing the Reconsolidation Process

Quite a few psychotherapies of focused, transformational change have emerged since the 1980s, and one of the tenets they have in common is that the client's engagement in therapy needs to be emotional for deep, lasting change to take place. Perhaps this important clinical tenet contributes to the view maintained by some clinical psychologists that for inducing memory reconsolidation, emotional arousal is necessary (see, e.g., Lane, Ryan, Nadel, & Greenberg, in press). However, the research shows that the reconsolidation process does not inherently involve emotional arousal. As noted earlier, successful deconsolidation and erasure have been demonstrated for learnings of many types, some of which have no emotional content per se, such as neutral declarative learnings (set of syllable pairings: Forcato et al., 2009), object recognition learnings (Rossato et al., 2007) and motor task learnings (Walker et al., 2003). In such cases no emotional arousal is involved either in the reactivation and mismatch of the target learning, triggering the reconsolidation process, or in the new learning that then revises the target learning. The brain clearly does not require emotional arousal per se for destabilizing and erasing the existing learning. That is a fundamental point.

If the target learning happens to have emotional components, then its reactivation (the first of the two steps required for deconsolidation) of course entails an experience of that emotion. Naturally, target learnings or schemas in psychotherapy usually are emotional, so observable emotion accompanies reactivation and is a key marker of adequate reactivation. However, this emotional arousal is not inherent in the reconsolidation process, and is present only because the target learning happens to involve emotional material. Clinical psychologists and psychotherapists sometimes conflate the emotional nature of target learnings in therapy with the inherent phenomenology of the reconsolidation process, as Lane et al. (in press) appear to do. For an accurate understanding of memory reconsolidation this distinction is important, though from a pragmatic clinical perspective it may seem to be hair-splitting.

Emotional arousal is not inherently required in any of the steps that erase a target learning. When researchers create a new learning to nullify and erase

a target learning, this new learning necessarily consists of experiences that sharply contradict the target learning's expectations and model of the world. Prior to erasure, the target learning is deconsolidated by a mismatch experience that typically consists of either an initial, brief instance of that same contradictory experience or some salient novelty not predicted by the target learning. For example, the target learning in the human study by Schiller et al. (2010) was a learned fear, specifically the classically conditioned expectation that the appearance of a yellow square on a computer screen would be accompanied in a few seconds by an electric shock to the wrist. For nullification of that learned fear after it had been mismatched and destabilized by a novelty (see Appendix for details), subjects were repeatedly given the contradictory experience of seeing a yellow square appear and disappear with no shock occurring. The simple experience of seeing each yellow square disappear was not an emotionally arousing experience, yet precisely for that reason it erased the fearful expectation of the shock.

Likewise, in psychotherapy we observe that erasure results from a contradictory experience that sharply disconfirms the target learning, and we observe that in some cases the contradictory experience is not in itself emotionally arousing, even though the target learning is strongly emotional. This is possible because the target of unlearning and nullification is the target learning's schema or model of reality (the semantic knowledge in the target learning), not the emotions generated by that model. This important point is illustrated by the following case vignette from my psychotherapy practice, which shows successful nullification and erasure of an emotional target learning resulting from a contradictory knowledge that is not emotional.

The client, a married woman, aged 50 and the mother of one child, sought therapy to dispel her aversion to sexuality with her husband, her depression, and her panic attacks, all of which had been afflicting her for at least a decade. I was using coherence therapy, in which the nonconscious, implicit emotional learnings that underlie and drive a given symptom are first brought into direct, explicit awareness, and then subjected to the process of memory reconsolidation and erasure, creating transformational change.

Session by session, into explicit awareness was emerging a complex array of underlying, implicit emotional learnings, some of which involved traumatic memories from various developmental stages of her life. In her first session I found that she would dissociate and become glazed and wooden in response to even a small step of interior exploration. She had a total of 45 sessions and was symptom-free at the end. This vignette focuses only on the particular emotional learning that emerged in her ninth session. This learning had formed when she was 18 years of age and had become pregnant by her boyfriend while living with her parents in a conservative town. She was living in shame and "desperate loneliness," did not want the baby or the boyfriend, and was struggling to decide about having an abortion when she had a miscarriage.

Wanting to find the emotional learnings she had formed in this ordeal, I gently guided her into experientially revisiting and reinhabiting that situation imaginally, and voicing her thoughts and feelings in present tense. This technique is often useful for bringing the implicit meanings of the original experience into explicit awareness. She seemed absorbed in the subjective reality of this material, and her voice was soft but somber as she said, "In this town, a girl who's been pregnant outside of marriage is just ruined, completely ruined."

In order to elicit fully and explicitly the learning she had formed, I asked softly, "What does 'ruined' really mean? What's going to happen to you now?"

After a silence, in an even quieter voice she said, "The rest of my life as a woman is ruined. I'll never marry, and I'll never have children." There it was, the specific learning she had formed. According to this learning, which had been implicit and outside of awareness for decades, having sex had results that had ruined the rest of her life. Immediately I understood that this dire model of her future was a potent source of both her depression and her sexual aversion.

With this clarity about the makeup of this target learning, I saw a possible way to create a contradictory experience: use of the brain's automatic detection of mismatches, a background process that is always scanning current conscious experience. So in reply to her words, I said, "Please say that again."

Somberly, and clearly feeling the emotional reality of the words, she said again, "The rest of my life as a woman is ruined. I'll never marry, and I'll never have children." As soon as she spoke the words this time, her wider conscious knowledge networks registered this information, which was new to her conscious networks though it was old in her implicit memory system. Her head made an abrupt movement, and in a sharper, louder voice she said with obvious surprise, "Wait—that's not true! I *did* marry! I *did* have a child!"

This first encounter between the target learning and vivid contradictory knowledge was the mismatch experience or prediction error needed for deconsol-

idating the target learning. This both-at-once experience of the target learning and vivid contradictory knowledge is termed a *juxtaposition experience* in coherence therapy to emphasize the simultaneous activation of the two as copresent conscious experiences.

Note that in this instance, the mismatching knowledge—"I *did* marry! I *did* have a child!"—was familiar, ordinary knowledge that was very real to her experientially, as real and certain as her own existence, but it was not inherently emotional in quality. It would not normally induce emotional arousal by itself. For successful mismatch, the knowledge or experience utilized must feel decisively real to the person on the basis of his or her own living experience, but that does not require this mismatching knowledge to be emotionally arousing in itself, even though the target learning is strongly emotional. (As noted above, the emotional quality of the target learning is extraneous and incidental to the reconsolidation process, not inherent in it.)

Presumably the neural encoding maintaining "My life as a woman is ruined, I'll never marry, I'll never have children" was now rapidly destabilizing, opening that set of learned meanings to being rewritten and erased by the knowledge, "I *did* marry! I *did* have a child! My life *isn't* ruined!"

She said in almost a whisper, "That just feels *huge*." Then her head tipped back against the top of her chair, and she gazed at the ceiling with blinking eyes. Then her eyes closed, and after about ten seconds she said, "I feel tingling and buzzing all over my body. It's weird—I can feel the skin between my toes. It's huge, it's huge." Internally she was repeatedly beholding and marveling at the new realization, which served as the several repetitions of it needed for rewriting the now deconsolidated target learning. For good measure, I soon created an explicit, out-loud repetition by jokingly saying, "I'm seeing an image of you running down the street waving your arms and shouting, 'I *did* get married! I *did* have a child! My life *wasn't* ruined!'" She laughed heartily at that, but even before I said it, her mood had shifted into a happy lilt that I had never seen in her before. Her contradictory knowledge was not emotional in itself, but the liberating effect of its use in the reconsolidation process certainly was.

I then reminded her that in our previous session she had raised a major question: "Why did I start feeling unbearable sadness and depression when I became pregnant with my son 13 years ago?" I asked her, "Does today's session help you see why?" Her eyes widened with this further powerful realization that the later pregnancy had reevoked her emotional memory of the much earlier one, reimmersing her in the complex emotional miseries that accompanied that pregnancy and the miscarriage. She said, "Ohhh—that's an amazing insight."

After that session, her longstanding depressed mood was gone and did not return. This confirmed that the targeted learning had been producing that mood, and that erasure or dissolution of that learning had been accomplished—meaning that "I'll never marry, I'll never have children" no longer felt real or true in any memory network. Her depressed mood had been the conscious surface of the unconscious despair and grief generated by the target learning.

That session was also the beginning of the end of her sexual aversion, which was dispelled after several more sessions that revealed a number of other episodes in her life where great suffering had resulted from or accompanied sex. Finally she no longer felt any urge to avoid her husband's overtures, though she did feel vulnerable and cautious about entering into a new level of sexuality with him. Those of course were natural, appropriate feelings, and I coached her on expressing to him her need for him to sensitively honor her pace and her cues.

Her panics attacks proved to be based in yet other emotional learnings. They ceased after the discovery and dissolution of those other learnings through juxtaposition experiences tailored to them.

The main purpose of the case vignette above is to show that the disconfirming knowledge or experience used for creating a mismatch experience and then nullifying the target learning does not necessarily have to be emotional in itself. The vignette also illustrates the lifelong durability of original emotional implicit learnings or schemas, as well as their dissociated, encapsulated state, which keeps them insulated from and immune to new experiences and new knowledge formed later in life. By being retrieved into conscious, explicit awareness, emotional implicit learnings become fully available for contact with other, disconfirming knowledge that can induce transformational change through juxtaposition (mismatch) experiences.

Thus, for consistently guiding decisive change through the reconsolidation process in therapy, the required reactivation of a target learning has to be its reactivation *as a conscious, explicit experience of the retrieved, specific elements of the target model* (such as "I'll never marry or have children, so my life as a woman is ruined"), not merely the retriggering of a still nonconscious, unretrieved implicit schema. Such implicit learnings are often retriggered in day-to-day

life *without* conscious awareness, and often life also provides strong disconfirmations, but because the schema remains outside of awareness, there is no juxtaposition experience—no conscious coexperiencing of both the old and new knowledge of what's real—and therefore no change takes place.

In psychotherapy, when an implicit schema maintaining symptom production becomes a conscious, explicit experience, the schema continues to feel subjectively completely real and compelling, and it persists in driving symptom production. This continues until the schema encounters a mismatch and disconfirmation experience, creating the conscious juxtaposition described above. Then abruptly the schema can lose its feeling of emotional realness and its power to control behavior or state of mind, and symptoms cease. Now the schema is not retriggered by situations and cues that formerly triggered it. I and other practitioners of coherence therapy have observed this clinical phenomenology many, many times (Ecker & Hulley, 1996; Ecker & Toomey, 2008; Ecker et al., 2012). Thus, conscious, subjective awareness and attention appear to function as the arena where separate, differing schemas (learnings, knowings, representations of reality) can come into mutual contact and undergo a combined semantic evaluation that allows for a revision of one schema by the other through the reconsolidation process.

Our clinical observations suggest that the brain and mind appear to operate according to a metarule that allows dissociated schemas to differ but requires consistency between schemas that are experienced concurrently in the same field of awareness (Ecker & Hulley, 2011). Guiding a juxtaposition experience cooperates with this metarule in order to transform a symptom-generating schema. When two mutually contradictory schemas are juxtaposed consciously, the schema that more comprehensively or credibly models reality, and therefore more usefully predicts how the world will behave, reveals the other schema to be false, and the falsified one is immediately transformed accordingly. Maximizing predictive power is well known to be a primary function and organizing principle of the brain (Clark, 2013; de-Wit, Machilsen, & Putzeys, 2010; Friston, 2010).

The previous paragraph emphasizes that a conscious, vividly experienced juxtaposition is found to be critically important in the psychotherapeutic utilization of memory reconsolidation. That emphasis could appear to be at odds with recent research and recent reviews of research: Delorenzi et al. (2014), Pine et al. (2014), and Santoyo-Zedillo, Rodriguez-Ortiz, Chavez-Marchetta, Bermudez-Rattoni, & Balderas (2014) have demonstrated that a target learning can be reactivated *without* coming into conscious awareness or behavioral expression, and that even under such conditions a mismatch/prediction error can then take place and destabilize the target learning, allowing it to be updated by new learning or disrupted chemically. How can these findings be reconciled with the clinical picture, in which the unconscious learnings maintaining symptoms are not dissolved until they come into conscious juxtaposition with contradictory knowledge?

There are several dynamics that might provide answers to that question (Ecker & Toomey, 2008). First and foremost, researchers have shown that the stronger and/or older the target learning is, the stronger must be the reactivation in order for destabilization to occur (Frankland et al., 2006; Suzuki et al., 2004). The target learnings in psychotherapy typically are both very old and also very strong, as they involve (and were formed in the presence of) intense emotion and urgent contingencies. Reactivation that produces conscious, bodily felt emotion, expectations, and meanings (as facilitated in therapy) is much stronger than reactivation that remains outside of awareness, which may be why conscious juxtaposition is observed to be necessary for achieving transformational change in therapy.

Another relevant dynamic is the active suppression and dissociation of emotional learnings that have strongly distressing content, which is the case for many of the target learnings that figure significantly in psychotherapy. Such active, self-protective suppression and dissociation could insulate these nonconscious, implicit learnings from direct juxtaposition with contradictory experiences in everyday life, preventing them from being updated. That insulation is removed in therapy by gently and gradually bringing these learnings into conscious awareness, allowing juxtapositions and transformational change to occur.

There is yet another reason why the versatile clinical utilization of memory reconsolidation requires first bringing a target learning into conscious experience. Researchers know the detailed makeup of the target learning, having created it themselves. This knowledge allows them accurately to reactivate and mismatch the target learning, destabilizing it, and also to then conduct new learning designed precisely to nullify and erase it. In sharp contrast, clinicians are in the dark at the start of therapy, with no knowledge of the makeup of the target learning driving symptom production. Therefore it is only by bringing the target

learning into explicit awareness and verbalization that its makeup can be known, and only then and not before can the therapist design and guide experiences of mismatch and nullification learning.

In summary, this section began by explaining that the reconsolidation and erasure process does not inherently involve or require emotion in either the target learning or the new learning that is juxtaposed with and nullifies it. In psychotherapy, however, the target learnings usually are richly emotional, so emotional arousal accompanies the therapeutic reconsolidation process as a rule, but this presence of emotion should not be conflated with the intrinsic nature of the reconsolidation process. The examination of juxtaposition phenomenology then continued into a clarification of why therapeutically effective juxtapositions have to be conscious experiences, even though reconsolidation research has shown that under special laboratory conditions, the process can take place outside of awareness.

Misconception 6: What Is Erased in Therapy Is the Negative Emotion That Became Associated With Certain Event Memories, and This Negative Emotion Is Erased by Inducing Positive or Neutral Emotional Responses to Replace It

As the clinical example in the previous section shows, what is erased through the reconsolidation process is a specific, learned schema or model or template of reality, verbalized in the example as "I'll never marry or have children, so my life as a woman is ruined." That schema was the target for erasure, and the mismatch that deconsolidated and then nullified it consisted of experiencing a sharp disconfirmation of that specific schema. With dissolution of the schema, the negative emotions that it was generating (despair, grief, and depression) disappeared, though those emotions were not themselves the target for mismatch or erasure, and the mismatch did not consist of creating a positive or neutral emotion instead of despair and depression.

Notice also that the client's negative emotion was arising directly from her existing model of the rest of her life, not from episodic memory (event memory) of the traumatic pregnancy and miscarriage. In other words, the traumatic experience resulted in her model (which is semantic memory), and that model in turn generated and maintained her emotional symptoms. Erasure of that model caused no loss of autobiographical memory.

Therapy clients' unwanted symptoms and problems are of course not limited to negative emotions, but can also be behaviors, thoughts, dissociated states, somatic sensations or conditions, or any combination of these. In any case, the target for erasure is not the manifested symptom or problem. The target is the learned implicit schema or semantic structure that underlies and drives production of the symptom. Erasure occurs when the target schema is activated as a conscious, explicit experience and is directly disconfirmed by a concurrent, vivid experience of contradictory knowledge. In other words, erasure does not occur simply through evoking a nonsymptomatic state when normally the symptom would be occurring (with one important exception, discussed at the end of this section). The occurrence of a symptom does not in itself bring the underlying, symptom-generating schema into conscious, foreground awareness, as is necessary for guiding the erasure process in therapy, so methods for evoking a nonsymptomatic state are not likely to disconfirm the underlying schema. The woman in our example might arrive at a session in a depressed mood, and there are techniques of somatic therapy, positive psychology, or mindfulness practice that could be used to shift her into a depression-free sense of well-being. However, that would not disconfirm and dissolve the underlying implicit schema maintaining her depression, "I'll never marry or have children, so my life as a woman is ruined." Her depression would therefore recur.

An example of the misconception that negative emotion is erased by inducing positive or neutral emotion is the view of Lane et al. (in press) that "changing emotion with emotion" characterizes how the system of psychotherapy known as emotion-focused therapy carries out reconsolidation and erasure. Rather, "changing old model with new model" is the core phenomenology of erasure through reconsolidation in any system of therapy. Emotions then change *as a derivative effect* of change in semantic structures (models, rules, and attributed meanings), just as in our example the client's depression disappeared as a direct result of dissolution of her target schema. In therapy, mismatch consists of, and erasure results from, a direct, unmistakable perception that reality is fundamentally different from what one currently knows and expects reality to be.

There is one important exception to the rule that lasting change does not result from evoking a nonsymptomatic state when normally the symptom would be occurring. The exception is target learnings that consist of a learned expectation of having a strongly problematic response in a particular kind of situation. Perhaps the most common instance of this

is the "fear of fear" that typically accompanies or even largely maintains phobias. In such cases there is a primary learned fear, such as a terror of bees stemming from a traumatic experience of being attacked by a swarm of bees in childhood, as well as a secondary learned, fearful expectation of suffering intense fear if a bee appears. The primary learning is the fearful expectation of being painfully stung by bees; the secondary learning is the fearful expectation of feeling terrorized by any bee. That secondary fear of fear is often the major force maintaining a phobia.

The expectation of feeling intense fear if a bee appears can be mismatched, disconfirmed, nullified and erased by using techniques that allow the person to encounter a bee in photos or imagination without feeling any fear. The absence of the expected terror is the mismatch experience. Clinically such techniques are found to dispel longstanding phobias abruptly and permanently (see, e.g., Gray & Liotta, 2012). However, guiding a therapy client into a neutral or positive emotion instead of the usual problematic emotion brings about lasting change only when the problematic emotion arises from a learned expectation of experiencing the problematic emotion, as in fear of fear. This is a special case that does not apply in the great majority of clinical cases.

Misconception 7: The Much Older Concept of Corrective Emotional Experience Already Covers Everything Now Being Described as Reconsolidation and Erasure

The familiar concept of the corrective emotional experience, introduced by Alexander & French (1946), denotes a therapy client's experiencing of something that was needed in earlier stages of development for well-being or healthy development but was missing: some new experience that could significantly undo and repair the effects produced by harmful experiences in the past. Most often this concept is applied in attachment-focused therapies, where it is typically understood as implying that the therapist's empathy and nonjudgmental acceptance can create corrective emotional experiences of interpersonal relationship that repair early interpersonal traumas and the patterns of insecure attachment learned in those ordeals.

What, then, is the relationship between the concept of the corrective emotional experience as it is widely understood, and the process of profound unlearning through memory reconsolidation? Are they the same, or are there significant differences? Is the reconsolidation framework just old wine in a new bottle?

To answer that question, we have to translate it into more specific terms: Does the guiding of a corrective emotional experience automatically and inherently include the creation of the juxtaposition (mismatch) experience that is required for erasure through memory reconsolidation?

The answer to that question is no: corrective emotional experiences do not necessarily include juxtaposition experiences. In a juxtaposition experience, the client lucidly experiences *both* the problematic original learning or schema *and* a contradictory, disconfirming new learning in the same field of awareness—not just the desired new experience by itself. In widespread clinical practice, corrective emotional experiences often consist of the desired new experience by itself.

Both therapists and clients are prone to what I have described as a counteractive tendency or reflex (Ecker, 2006, 2008; Ecker et al., 2012), an urge to avoid and suppress unwanted states of mind by building up preferred states of mind. Corrective emotional experiences are all too easily shaped by the counteractive tendency: the client's attention is fully engaged in the desirable new experience and disengaged or dissociated from the unwanted reaction or ego-state and its core schema. This disconnection from the problematic target schema during a corrective emotional experience is the very opposite of the explicit, foreground, experiential awareness of that schema that is needed for reliably guiding juxtaposition and transformational change. Corrective emotional experiences structured in that counteractive, one-sided manner can feel deeply meaningful and satisfying in the moment, but they cannot result in lasting change if the core schema underlying the problem remains intact, as it does if it is not being subjected to a juxtaposition that dissolves it. In short, as widely carried out by clinicians, a corrective emotional experience might supply the material for one side of a potential juxtaposition experience but does not inherently access and reactivate the other side—the emotional learning underlying the problem—to actually create the juxtaposition.

On the other hand, if we regard juxtaposition experiences to be the true corrective emotional experiences, then we have a definition that does inherently call for all of the ingredients needed for inducing memory reconsolidation and a lasting transformation of the emotional learning maintaining unwanted emotions, behaviors, thoughts, and somatics. A therapist who understands that reconsolidation and transformational change require juxtaposition guides a one-sided corrective emotional experience into be-

coming a two-sided juxtaposition experience by eliciting concurrent, direct awareness of the problematic learning that is thereby disconfirmed by the desired new experience.

For example, a client accidentally knocks over a small clock in the therapist's office and apologizes anxiously and profusely. The therapist says with a relaxed, warm smile, "It's really OK. To me that's a very small thing and not a problem at all. Little accidents like that happen for all of us, including me. Can you see that I'm not at all upset?" The client takes this in and feels much relieved to recognize that with the therapist he is safe from negative judgments, anger, humiliation, or rejection over such things. Probably most therapists would regard that as a corrective emotional experience for this person. However, if the insecure attachment learnings underlying the client's fearful apology have not yet been made conscious and explicit, this new experience is not being juxtaposed with those learnings, so transformational change is not occurring. In order for that positive new experience to help bring about transformational change, the therapist has to guide the client into experiential, embodied awareness and verbalization of the underlying target learning, such as, "Mom's rage and disgust at me for any accident or mistake mean I'm worthless if do anything wrong, and I expect anyone else to react to me that way too." Then the therapist guides a juxtaposition experience, for example by saying empathically, "All along you're expecting that anyone would go into rage and disgust at you for any little thing you do wrong, just as Mom did so many times, and yet here you're having an experience of me feeling it's really no big deal at all that you accidentally knocked over this little clock. Can you hold both of those at once, and see what that feels like?" That explicit, experiential juxtaposition gives the new experience its maximum influence toward actual unlearning and dissolution of the target learning.

New experiences that can disconfirm and dissolve existing problematic schemas arise not only in the form of the therapist's responses, but also in the course of the client's daily life, and these are fully as useful for juxtaposition as the client's experience of the therapist. (For a detailed case example, see Ecker et al., 2012, pp. 43–61.)

As a final comment on this topic of how the reconsolidation framework illuminates the concept of corrective emotional experiences, the drawbacks of the term "corrective" are worth noting. The term implies that the client's existing learnings and responses, formed in earlier life experiences, are "incorrect." However, when we bring these existing learnings into awareness and verbalization in therapy, making their content explicit, it always becomes apparent that the client's implicit emotional learning system did its job faithfully and properly in (a) forming those learnings adaptively in response to what was subjectively experienced, and (b) maintaining and utilizing those learnings ever since they were formed until the present day, however many decades that may be. Emotional implicit learnings are specially formed so as not to fade out for the life of the individual, as noted earlier. To describe a therapy client's core beliefs or schemas as incorrect, maladaptive or pathogenic is actually to accuse the process of natural selection of having those attributes, because a person's persistent beliefs and schemas exist due to the proper functioning—not the malfunctioning—of the emotional brain.

Misconception 8: To Induce Memory Reconsolidation and Erasure, Therapists Must Follow a Set Protocol Derived From Laboratory Studies

Memory reconsolidation research tells us that a well-defined sequence of experiences is required by the brain in order to destabilize a target learning and then unlearn and eliminate it: the target learning must first be reactivated into conscious awareness, then destabilized by a mismatching experience, then updated and reencoded by new learning that nullifies it. That is a sequence of three experiences, but each is defined without reference to any particular procedure for bringing it about. Researchers and clinicians are free to devise any suitable means for creating those experiences, and the creative possibilities are unlimited. The brain does not care what concrete conditions or procedures induce those experiences. Hundreds of studies of reconsolidation have been published by neuroscientists as of this writing, and across them there is a great diversity of concrete procedures used.

Likewise, many clinical methods for guiding the critical sequence of experiences have been described by Ecker et al. (2012), who propose that the 3-step sequence is the core process shared by many different-seeming therapy systems that produce transformational change. Thus, as noted earlier, memory reconsolidation serves as a new framework of psychotherapy integration, and within that framework, the many therapies of transformational change are seen as a broad range of methods for guiding the one core process, giving clinicians great versatility in how they do so. Current neuroscience is consistent with that picture, in the sense that reconsolidation is the brain's only known process for eliminating (not merely sup-

pressing) an established learned emotional response. Thus the view that a set protocol is dictated by the memory reconsolidation process could not be further from the reality.

Misconception 9: A Long-Standing Emotional Reaction or Behavior Sometimes Ceases Permanently in Psychotherapy Without Guiding the Steps That Bring About Erasure Through Reconsolidation, and This Shows That Reconsolidation Is Not the Only Process of Transformational Change

As implied in the previous section, various therapy systems involve concepts and methodology that make no reference to memory reconsolidation or the sequence of experiences required by the brain to induce it, yet their methodologies do result in that sequence of experiences occurring with some degree of consistency, resulting in transformational change. A close examination of the moment-to-moment process in published case studies makes the occurrence of the required steps apparent (Ecker et al., 2012, see pp. 126–155). Practitioners of such therapy systems might maintain that they have not guided those experiences when in fact they have done so. It is a well-known meme in the clinical field that how therapists conceptualize what they do, and what they actually do, are not necessarily the same.

In my own psychotherapy practice I have occasionally seen transformational change result from sessions where I did not think the key sequence had occurred. In such cases I have made a point of then engaging my client in closely examining, in hindsight, the internal events that led to the shift or breakthrough. All such hindsight enquiries have revealed that a juxtaposition experience in fact occurred serendipitously, without being recognized or verbally labeled at the time. Thus my own clinical experience suggests and upholds the hypothesis that transformational change of an acquired response is always the result of a juxtaposition experience—that is, of the reconsolidation process—even when there has been no explicit guiding of the steps required for erasure.

A memorable example of such hindsight verification of juxtaposition emerged from a colleague's case consultation. Her therapy client was a woman, aged 32 and married for five years, who was struggling with her obsessive attachment to and compulsive pursuit of the man who had been her major love through her early twenties. This problem developed after she and this man happened to cross paths again two years earlier. There had been no physical intimacy in these two years, owing solely to the man's lack of responsiveness, but the woman's emotional infidelity was significant and was causing her much guilt.

The therapist had used a number of different types of therapy for many sessions, with little or no effect on the client's heavy preoccupation with her former boyfriend. Most recently there had been several sessions in which the therapist had an uncomfortable sense of flailing and being ineffectual. Then the client came into the next session and reported that a major shift had occurred. Her preoccupation and her pursuit of this man had stopped. This breakthrough was mysterious for both client and therapist. The client could offer nothing more than to speculate, "I think what you said sunk in somehow, that when an investment goes badly, sometimes it's best to cut your losses." This referred to an offhand, momentary comment made by the therapist in the previous session, a comment that seemed more like advice than therapy. It was counteractive in nature (an attempt to build up a cognitive understanding to override the emotionally driven symptom), was not dwelt upon, and the focus of the session had moved on. Yet the client indicated that the comment had somehow led to her liberating shift.

Soon after that, the therapist consulted with me and mentioned all of this. I suggested a way for her to guide her client to look more closely into the process that had occurred internally: She could ask, "If it was new for you to hear that ending it with him could be OK even though you had an emotional investment in it from long ago, what were you previously believing or expecting about how it would *not* be OK to end it?" This would be using the disconfirming knowledge to find the constructs or schema that had been disconfirmed, which is a reverse engineering of coherence therapy's usual process of first finding the client's symptom-generating schema and then, on the basis of the details of that schema, finding vivid contradictory knowledge to create a juxtaposition experience. But when a transformational shift occurs serendipitously, it is typically the disconfirming knowledge that becomes apparent first, while the disconfirmed schema is still unknown. Subsequently the disconfirming knowledge can be used to bring the now defunct schema into explicit awareness, as I guided the therapist to do in this case.

My colleague then briefed me on what emerged when she pursued, with some persistence, the enquiry I had suggested. The offhand comment happened to reach precisely into an unconscious schema that the client now put into words by saying, "I was struggling to keep my emotional investment in that relationship

from being lost because I'd really put my heart and soul into that relationship, and on some level I felt that if it ended, I'd be losing so much of myself that I would die or just be an empty shell or ghost forever. But when you said it's OK to get out of an investment even if you take a loss, all of a sudden that changed, because I saw, 'Oh—people do that all the time. It's *not* a disaster, it's just practical.' I saw that I *could* let go and lose that investment in him, and I wouldn't turn to dust."

That account points clearly to a juxtaposition experience that had formed in response to the therapist's offhand comment. The woman reported also that it was not a struggle to persist in not contacting the man, though she did feel "a quiet sadness" each time she would have contacted him but did not do so. The nonreactivation of the symptom-generating schema or ego-state and the effortlessness of remaining symptom-free are key markers of erasure and transformational change.

Thus, when the steps required for reconsolidation and erasure have not been overtly or deliberately guided in therapy and yet transformational change is observed to occur, this does not imply that a process other than reconsolidation is responsible for the change. Extensive clinical experience indicates rather that an unnoticed, nonverbalized juxtaposition experience is implicated and can probably be revealed by the type of inquiry illustrated in the example above.

Informational and psychoeducational comments made to a client in therapy tend to result in mere intellectual knowledge and therefore do not, as a rule, represent an effective method for setting up the disconfirming *experiential* knowledge required to create a juxtaposition experience. The example above shows that juxtaposition experiences can sometimes form, unbeknownst to the therapist, even in clinical situations where we would not imagine that they could do so, such as in response to an offhand, commonsense comment.

Misconception 10: Carrying Out the Steps Required for Reconsolidation and Erasure Sometimes Fails to Bring About a Transformational Change, Which Means That the Reconsolidation Process Is Not Effective for Some Emotional Learnings

In psychotherapy there are four distinct situations in which the reconsolidation process can appear to fail to produce decisive change when actually the process is not failing, but rather is not in fact taking place for some specific, identifiable reason:

1. Resistance to dissolution. In some cases, the therapist has indeed guided the sequence of experiences necessary for reconsolidation and erasure, but the target learning does not dissolve and remains in force (continues to retrigger, feel real, and produce symptoms). We will see below that in such cases, the shift is prevented by a blockage or resistance that can be cleared away, allowing dissolution to occur when the sequence is guided once again. The blockage is a separate, distinct phenomenon that does not imply a fundamental failure of the reconsolidation process.

2. Multiple symptom-generating schemas. In other cases, in response to the necessary sequence of experiences, the target learning does dissolve and no longer activates or feels real, but the symptom produced by that target learning continues to occur. This means that there is at least one other emotional learning or schema, distinct from the one that has been dissolved, that also produces the same symptom. It is common for therapy clients to present a symptom or problem that is driven by more than one emotional schema. A symptom ceases to occur only when all of its underlying emotional learnings have been nullified.

3. Nonimplementation. In other cases, the therapist believes he or she has guided the required sequence of experiences, but has not actually done so. As explained below, the necessary experiences have aspects that can be misperceived, particularly by clinicians who are relatively new to guiding this process.

4. Not based in learning. One other situation in which the reconsolidation process can erroneously appear to be failing is where the client's problem or symptom is not rooted in acquired, underlying emotional learning. This category includes autism spectrum and other conditions that have genetic causes, or purely physiological conditions such as depression caused by hypothyroidism. For dispelling or moderating such conditions, the memory reconsolidation process does not apply and should not be used, so it cannot correctly be said to fail in such cases. A very wide range of symptoms has been dispelled decisively in therapy by the reconsolidation process (Ecker et al., 2012, p. 42), which shows how pervasively emotional learnings are the underlying cause of presenting problems.

In the case of resistance to dissolution, the erasure sequence is well fulfilled by juxtaposition experiences, as required by the brain for dissolution of the target learning, and yet dissolution does not occur because it is blocked by another, distinct dynamic. The erasure of an emotional learning is the profound unlearning and dissolution of what has seemed to be a reality. For ex-

ample, after dissolution of an implicit emotional learning verbalized as "Dad never talking to me or playing with me means I'm unlovable and don't matter," the individual now either has *no* way of making sense of being neglected by Dad, or realizes emotionally that "I *was* lovable and *did* matter, and yet Dad never talked to me or played with me." Such alterations of personal reality entail difficult emotional adjustments, particularly when the target learning is a core element of a deeply vulnerable area, such as primary attachment relationships, identity, or sense of justice, for example. Even if the series of experiences required for dissolution has occurred as required, dissolution is blocked by the emotional brain if the emotional consequences of dissolution do not feel tolerable, whether or not those consequences are recognized consciously.

Thus the unlearning and dissolution process is not governed by mechanistic neurological processes. Higher-order, abstract meanings that are distressing can block it. For example, many times I have seen a therapy client hold back from a liberating shift because of an accompanying realization that if the shift were to occur, it would mean that decades of life were wasted by living according to unconscious, life-choking beliefs that have turned out to be completely false. That abstract meaning of "life wasted" tends to produce initially intolerable emotional pain of grief and injustice. If any consequences of dissolution feel unworkably distressing, the dissolution is blocked.

This unconscious blockage can be understood as a self-protective response to the expected consequences of the change. The therapist considers that such resistance may be occurring when he or she is reasonably confident that genuine juxtaposition experiences have occurred (with both the target learning and contradictory knowledge experienced concurrently and vividly), yet the target learning remains in effect (continues to feel real and to generate the client's symptoms). Then the therapist's task is to guide the client gently to bring awareness to the specific distress that is expected to result from dissolution (such as disorientation, loss, grief, pain, or fear), making dissolution too daunting to allow. The expected distress itself consists of meanings, models and ego states that now become the focus of transformational change. When, as a result of this work, there is no longer any intolerable emotional consequence to dissolution, the juxtaposition experience is repeated and dissolution readily occurs.

In other words, the dissolution of any one emotional schema necessarily takes places within the whole ecology or network of interconnected meanings and models that constitute the person's experiential world, and that world may first have to be prepared so as to make the emotional consequences of dissolution tolerable and acceptable. At that point, the required sequence of experiences (which is the creation of a juxtaposition experience repeated a few times) successfully dissolves the target learning maintaining the symptom. (For a case example illustrating this process, see Ecker et al., 2012, pp. 77–86.)

Nonimplementation of the required sequence of experiences is the other situation that needs to be examined here. Nonimplementation may be the actual situation though the therapist believes mistakenly that the sequence has been fulfilled. Such cases can involve misperceptions of various kinds. One mistake of this kind consists of assuming that a particular *procedure or technique* necessarily creates a particular subjective *experience* had by the client. The brain's requirement for deconsolidating and erasing a target learning is a certain sequence of internal experiences, not external procedures or techniques. In other words, there is an important distinction between the *procedure* that is carried out visibly in the room, and the *internal phenomenology* occurring in the therapy client's subjective experience. A particular procedure intended to create the necessary experience may or may not be successful at inducing that internal experience (be it reactivation of the target learning in explicit awareness, or a disconfirming mismatch of the target learning, or the juxtaposition of the two). If the therapist does not verify the quality of the client's inner experience, he or she might assume the experience was properly created when actually it was not created by the procedure used. In that case it will appear that memory reconsolidation has failed to be effective, when in fact it was not properly induced in the first place.

The first step of the erasure sequence is the reactivation in conscious awareness of the target schema that underlies and generates the client's problem or symptom. This requires the target schema to be not only retriggered by a suitable cue, but also *present in the foreground of conscious awareness*, so that the specific set of meanings and expectations that make up the schema are lucidly and explicitly in awareness. This explicit awareness is facilitated through specifically verbalizing this material while feeling it emotionally and somatically. Such conscious reactivation requires the implicit, nonverbal target schema to be integrated into conscious awareness. Typically, however, symptom-generating schemas are fully and deeply implicit and nonconscious, and in the course of decades they are retriggered hundreds or thousands of times without becoming conscious in the least. A therapist might guide a retriggering by guiding the

client to revisit imaginally a recent situation that did retrigger the schema and the symptom. The therapist might believe that this retriggering procedure has fulfilled the reactivation step, though it has not done so because the emotional reactivation of the schema is not accompanied by integrated, cognitive awareness of the specific contents of the schema. The inner experience of reactivation required for reconsolidation has not occurred, so transformational change will not result when the remaining steps are carried out. The therapist, believing all the steps to have been fulfilled, comes to the false conclusion that sometimes the reconsolidation process fails to work.

Similarly, a procedure that the therapist believes has created a disconfirming experience or vivid contradictory knowledge—the next step in the key sequence of experiences—might not have actually created the inner experience of juxtaposition (mismatch or prediction error) that the brain requires for unlocking synapses, deconsolidating the target learning. There are various ways in which a mistaken belief that a juxtaposition experience has occurred may arise. To begin with, both sides of the juxtaposition need to be richly experiential. That is, the client must be having her or his own lucid experience of the felt realness of both (a) the target schema and (b) some other personal knowledge that absolutely contradicts what the target schema "knows" or expects. Therapists may believe they are guiding a sufficiently experientially vivid state of mismatch, engaging the client's limbic system in the disconfirmation experience as is necessary, when actually the work is too cognitive and not sufficiently experiential to create a true juxtaposition experience. This too can give the impression that the process has been ineffective, when actually it has not been properly guided and the brain's requirements have not been fulfilled. The therapist, believing that the necessary conditions have been fulfilled, may conclude that the reconsolidation process has failed to work.

This was the case of a therapist who wrote to me that in his experience, he "can offer reframes, tell Ericksonian stories, etc.; [but] simply offering and juxtaposing a mismatch does not guarantee transformation." He was assuming that those techniques were creating juxtaposition experiences as required. He was defining juxtaposition by the procedure rather than by the quality of the client's inner experience. In reply I pointed out that the contradictory knowledge that creates the mismatch must be *the client's own living experience of contradictory knowledge*, not just something the client is hearing about informationally from the therapist. I mentioned also that a procedure that has successfully created an effective juxtaposition experience for one client may fail to do so for another.

Another aspect that can be misjudged by the therapist is the matter of *what* is being mismatched and disconfirmed. The target of disconfirmation needs to be a core symptom-necessitating construct, or symptom production will be unaffected by the disconfirmation. Identifying suitable target constructs requires doing a thorough job in the preparation steps of finding, making explicit, and guiding integrated awareness of the implicit learning or schema driving symptom production (the methodology for which is described in detail by Ecker et al., 2012, and Ecker & Hulley, 2011). Symptom-generating schemas often have several layers. Therapists sometimes do an incomplete job of retrieving this material into integrated awareness, and then target a relatively superficial or even tangential construct. A transformational shift will not result from a mis-targeted juxtaposition experience, but that is not a failure of the reconsolidation process to effect change. When all four of the situations described in this section are navigated skillfully, the therapeutic reconsolidation process is consistently effective in producing the distinct and verifiable markers of transformational change.

Conclusion

The profound unlearning and cessation of acquired behaviors and states of mind occurs through the process of memory reconsolidation, according to the best available scientific knowledge and as extensive clinical experience bears out. A sound understanding of memory reconsolidation is therefore a vital guide for facilitating lasting, liberating change in psychotherapy and counseling with maximum regularity. The study, practice, and effort required to arrive at a sound understanding and use of memory reconsolidation and avoid the various possible misconceptions are a price well worth paying for the clinical effectiveness gained. It is my hope that the accounts and clarifications provided in this article will help to communicate this invaluable body of knowledge to mental health practitioners everywhere.

Appendix

Understanding the Results of Schiller Et Al. (2010) in Terms of the Memory Mismatch (Prediction Error) Requirement

In neuroscience research on memory reconsolidation, the erasure of a learned fear in human subjects was first accomplished through an endogenous be-

havioral process by Schiller, Monfils, Raio, Johnson, LeDoux, and Phelps (2010). Previously there were at least six published studies reporting behavioral methods of memory erasure or modification in human subjects (Forcato et al., 2007, 2009; Galluccio, 2005; Hupbach et al., 2007, 2009; Walker et al., 2003). By doing the same for a learned fear—a human response of clinical importance—Schiller et al. made the relevance of memory reconsolidation to psychotherapy very clear to science journalists and the lay public, generating much interest.

Various aspects of the reconsolidation process elegantly demonstrated by Schiller et al. (2010) are of fundamental importance, as described below. However, the authors' discussion and interpretation of results did not take into account major findings that were already well documented by other researchers regarding the brain's requirement of a mismatch or prediction error experience for inducing the reconsolidation process (discussed above in the sections on Misconceptions 1 and 3). As a result, Schiller et al. discussed their successful procedure without identifying the causes of its effectiveness. In what follows, this procedure is examined and understood in terms of the broader research findings. The main purpose of this reinterpretation of the results of Schiller et al. is to promote an accurate understanding of how the reconsolidation process functions. The utilization of reconsolidation in psychotherapy can yield major advances of several different kinds (Ecker et al., 2012, 2013a), but the realization of these benefits depends on accurate understanding. The reinterpretation below also illustrates the application of the mismatch relativity principle discussed in this article's main text, as well as the necessity of "minding the findings" (Ecker, Hulley, & Ticic, in press) for understanding reconsolidation research procedures and the results of those procedures.

In the Schiller et al. (2010) study, the fear response to be erased was created by a training experience on Day 1 of the procedure. Each of the adult subjects viewed an electronic screen and saw a colored square appear for 4 s, about every 15 s, for a total of 26 times. The square was yellow 16 times, a random six of which were accompanied by a mild electric shock to the wrist. Thus the conditioned stimulus (CS) was a yellow square and the unconditioned stimulus (US) was a wrist shock. The shock occurred at the very end of the 4-s display of the square. The other 10 squares in the series were blue, were not accompanied by any shocks, and were randomly intermixed with the yellow squares. Subsequent responses to the blue squares served as the control condition in this study.

Through that training experience—a classical conditioning procedure—each subject learned, subcortically, the CS-US association of the yellow square and the unpleasant shock. As the 26 presentations progressed, by using standard electrical sensors of skin conductance the researchers detected the increasing development of an anticipatory fear response with each successive presentation of a yellow square. In this way a subcortical learned fear of yellow squares was established.

In addition to the CS-US association, the training experience contained other features that were also learned subcortically by the subjects, but were not discussed by Schiller et al. as learnings: A yellow square is not always accompanied by a shock; and whenever any colored square disappears, it is followed by a blank screen for 11 s, and then by another colored square, many times in succession.

All of those features made up the learned schema or expectation of what happened on the screen, so it was those three features that were predicted and expected by each subject's implicit emotional memory. Analyzing the results with awareness of all features of the implicit learning proves essential for understanding the erasure process and seeing the critical importance of the neglected research findings.

Each subject returned 24 hr later, on Day 2, and underwent one of three different procedures, creating three groups of subjects.

First group. For the main experimental test group, each subject viewed the electronic screen and experienced the following sequence:

1. a yellow square appearing just once, for 4 s as on Day 1, with no shock (unreinforced CS presentation); followed by
2. the images and sounds of a television show episode, lasting 10 min; then
3. a random sequence of yellow and blue squares appearing, with no shocks: 10 yellow and 11 blue squares in random order, each for 4 s every 15 s as on Day 1.

On Day 3 (24 hr later), in order to determine whether the CS (yellow square) still elicited a fear response, subjects again viewed a series of shock-free yellow and blue squares. For all subjects in this group, fear responses no longer occurred, as indicated by the skin conductance monitor. At a 1-year follow-up test, again there was no fear in response to the CS. This complete and lasting absence of responsiveness of the target learning is what is meant by saying that it had

been erased by the procedure on Day 2.

Without Experience 2, the procedure on Day 2 would have been a standard multitrial extinction procedure, and the result would have been only temporary suppression of the learned fear response, not its complete and long-lasting erasure. With Experience 2, the procedure instead induced reconsolidation and erasure, which is a qualitative difference. Schiller et al. attribute their procedure's successful erasure to the 10-min "break," as they term it, but they provide no analysis of how that time interval caused the qualitative difference. However, if the procedure is examined in terms of the mismatch requirement and mismatch relativity (MRMR) defined in the main text, the primary cause of reconsolidation and erasure that becomes apparent is not the 10-min break. The MRMR analysis is as follows.

Experience 1. Seeing the CS appear induced reactivation of the learned fear (the target learning) in the standard manner of presenting a conditioned stimulus to reactivate a conditioned response. The absence of the US was consistent with the target learning's expectation that a shock may or may not accompany a yellow square. Therefore, Experience 1 was not a mismatch (prediction error) experience, so it did not trigger destabilization and reconsolidation of the target learning (as was demonstrated by Sevenster et al., 2014, and discussed in the main text). Thus the target learning was still stable following Experience 1.

Experience 2. Seeing and hearing the TV show was immediately a mismatch experience, because the TV show was sharply discrepant with the learned expectation that what follows any square is a blank, silent screen for 11 s and then another colored square, and then more of the same in a long series. The initial training consisted of partial reinforcement of the US (shock), but it had continuous (100%) reinforcement of the blank and silent screen occurring between colored square presentations. The striking mismatch with those expected features in Experience 2 would have caused rapid destabilization (deconsolidation). This illustrates the use of a surprising novelty to create mismatch. In addition, the continuation of the TV show for 10 min would have driven updating of the destabilized target learning to expect a TV show after any subsequent CS presentation.

The TV show played for 10 min. If the screen had instead been left blank for 10 min, a timing mismatch (Díaz-Mataix et al., 2013) would have been created because the target learning expected only 11 s of blank screen before the next colored square appeared. (With the screen left blank for 10 min, the procedure would have been structurally very similar to that used with rats by Monfils et al., 2009, who reported successful erasure of a fear learning. MRMR analysis of that study is in the main text of this article.) However, the perception of TV show images and sounds would have created a mismatch experience immediately. A timing mismatch would not have developed until the next colored square appeared 10 min later, creating a recognizable interval significantly longer than the expected 3 min. This experience of a 10-min interval coming 10 min after destabilization would likely have updated the target learning to expect a 10-min interval between colored squares henceforward.

By the MRMR account, then, the mismatch experience that triggered destabilization and allowed erasure to ensue was that of the visual and audio content of the TV show, not its time duration of 10 min. MRMR predicts that a TV show duration of 11 s (no extra time) instead of 10 min would also have resulted in erasure. This MRMR analysis challenges the conclusion drawn by Schiller et al. (2010) that the time duration was responsible (p. 52): "The current results also suggest that timing may have a more important role in the control of fear than previously appreciated. . . . Our findings indicate that the timing of extinction relative to the reactivation of the memory can capitalize on reconsolidation mechanisms." (Here the labeling of Experience 3 as "extinction" seems a misnomer, since it did not produce extinction.) These considerations illustrate the utility of MRMR principles for identifying cause and effect in procedures that induce reconsolidation or extinction.

Experience 3. The series of shock-free squares, each followed by 11 s of blank screen, began with the target learning in a destabilized condition due to Experience 2. However, as discussed in the main text in examining standard multitrial extinction, initial destabilization at the start of a series of unreinforced CS presentations does not guarantee that destabilization will persist or that erasure will take place. Therefore it is necessary to examine specifically why Experience 3 did erase the learned fear in this case. The examination consists of tracing out the effects of every step of the procedure according to MRMR principles and building an account of the accumulating effects.

Here that account begins with the first unreinforced CS presentation (CS1) in Experience 1, in response to which the target learning became reactivated while remaining stable, as noted. Following this, Experience 2 destabilized the target learning by mismatching the expected blank, silent screen with a TV show. Experience 3 then began for some subjects

with an unreinforced CS (CS2, yellow square with no shock) or with a blue (non-CS) square for other subjects. CS2 by itself did not constitute a mismatch of US-expectancy and did not begin to erase the target learning, for the same reason as in Experience 1, namely that from the partial reinforcement schedule during initial acquisition of the target learning, subjects learned to expect that the US might or might not occur with any given CS.

Consequently, counterlearning and erasure of the target learning would require a series of unreinforced CSs that were unmistakably more numerous than the largest number of contiguous unreinforced CSs in the original training. In the latter, a random six of the 16 yellow squares were accompanied by shock, so the largest possible number of contiguous unreinforced CSs was five. (In this first group of eight subjects, statistically the likelihood of three, four, or five contiguous unreinforced CSs occurring among them was 66%, 40%, and 22%, respectively.) Therefore, since erasure did take place, the target learning must have been in a destabilized and erasable condition after significantly more than five unreinforced CSs had occurred. The MRMR model has to account for that if it is to be consistent with the results of this study.

The absence of a US mismatch in the experience of CS2 also occurs in the standard multitrial extinction procedure and there has the effect of terminating both the subject's ongoing experience of mismatch and the state of destabilization (as described in the main text). Here, however, such termination of mismatch and destabilization would not have occurred, for this reason: Whereas in standard extinction the target learning's initial destabilization is due to a mismatch of US-expectancy by CS1, in this case the initial destabilization was due not to a mismatch of US-expectancy, but rather to a mismatch of the blank, silent screen expected between CS presentations in Experience 2. That specific experience of mismatch was not canceled or terminated when CS2 brought no new US mismatch at the start of Experience 3, so the target learning remained destabilized. Furthermore, the appearance of CS2 created a timing mismatch, as noted above. These considerations are unchanged if it was a blue square that the subject saw first in Experience 3.

Following CS2 or non-CS blue square, the appearance of a blank, silent screen would have been a mismatch experience because the target learning had been updated in Experience 2 to expect a TV show. The target learning was updated by this to expect either a blank screen or a TV show between colored squares. This mismatch maintained the destabilized state of the target learning until the third appearance of a colored square after an interval of 3 min. That 3-min interval was a mismatch of the previously updated expectation of a 10-min interval, and this timing mismatch both updated the target learning and maintained its destabilization until the fourth colored square. After the fourth colored square, however, no more mismatches would have occurred, and there would be no lingering or fresh experience of mismatch for the rest of Experience 3. If destabilization were maintained solely by mismatch experiences, destabilization would have terminated too soon for a sufficient number of CS-without-US presentations to cause erasure to occur in this procedure.

This implies that destabilization was maintained in the other possible way in the MRMR model, namely MDU (maintenance of destabilization by updating), as hypothesized in the main text. MDU would occur through a molecular signaling pathway by which the updating process maintains destabilization independently of the mismatch requirement, so that updating has sufficient time to be accomplished once it begins. The MRMR account above identifies four distinct triggers of updating prior to the fourth colored square, and that updating would have triggered MDU and maintained destabilization presumably throughout Experience 3, allowing all 10 CSs to erase the target learning with the new learning that a yellow square is always harmless. The necessity of invoking MDU in order for the MRMR framework to account for erasure in this study puts a priority on testing the MDU hypothesis empirically. That would probably require detection of the separate molecular markers of destabilization and updating after each colored square throughout Experiences 1, 2, and 3.

Second group. For a second group of subjects, Schiller et al. (2010) carried out the same procedure with one difference: On Day 2, a 6-hr delay was added after the 10-min TV show, between Experiences 2 and 3. On Day 3 the researchers then found that for these subjects, the target learning's fear response was reevocable and had not been erased. The 6-hr delay was slightly longer than the approximately five-hour duration of the reconsolidation window (Pedreira, Pérez-Cuesta, & Maldonado, 2002; Pedreira & Maldonado, 2003; Walker et al., 2003). In other words, for this group of subjects, Experience 3 was conducted after the reconsolidation window had closed. The target learning, which was destabilized in Experience 2, had reconsolidated or restabilized and was no longer susceptible to being updated and erased by the series of no-shock squares in Experience 3, so the latter now functioned as a conventional extinction training, not

as an erasure learning, and created a separate learning that competed with the target learning.

Third group. For a third group of subjects, on Day 2 Experiences 1 and 2 were omitted and only Experience 3 was implemented, which was a conventional extinction training. Tests on Day 3 again showed that the target memory's fear response was reevocable and had not been erased, though it had been suppressed temporarily by extinction. This demonstrates once again the well-established fact that extinction does not yield erasure.

Experience 3 of the procedure had the familiar structure of repetitive counterlearning that has long been termed "extinction training," but when applied during the reconsolidation window for the first group of subjects, its behavioral and neurological effects differed qualitatively and radically from those of extinction, as described above. It is worth repeating here the conclusion of a study by Duvarci and Nader (2004), "Reconsolidation cannot be reduced down to facilitated extinction" (p. 9269). Yet Schiller et al. (2010) refer to Experience 3 of their procedure as an "extinction training" even for the first group of subjects, and they describe the entire 1–2–3 procedure as the "interference of reconsolidation using extinction" (p. 50), "extinction conducted during the reconsolidation window of an old fear memory" (p. 52), and "extinction training during reconsolidation" (p. 52). Using "extinction" terminology to refer to a learning experience created during the reconsolidation window to erase and replace a target learning invites much confusion and misunderstanding of the reconsolidation process. In light of the fundamental differences between reconsolidation and extinction (discussed in the section above on Misconception 3), and with a view to facilitating widespread, accurate understanding of that difference, it seems desirable to use terms that clarify rather than obscure the functional role of the learning experience in the case under consideration. Terms such as "nullification learning," "update learning" or "erasure learning" seem more appropriate for functionally labeling Experience 3 in the first group of subjects, whose learned fear was erased. (For the other two groups of subjects, the series of no-shock yellow squares in Experience 3 was true extinction training, as noted above, because it was conducted outside the reconsolidation window and was not part of a reconsolidation process. In these cases, therefore, Step 3 is appropriately termed an extinction training.)

However, the results of this study by Schiller et al. (2010) are so striking and significant that a growing number of other researchers have adopted their procedure and have retained the "extinction" misnomer (e.g., Baker et al., 2013; Clem & Huganir, 2010; Liu et al., 2014; Quirk et al., 2010; Steinfurth et al., 2014; Xue et al., 2012). With the persistence of that terminology, it is especially important to recognize that there is no inherent necessity for the erasure learning during the reconsolidation window to have the same procedural structure as conventional extinction (a series of many identical countertraining experiences). The form of erasure learnings is limited only by the creativity of researchers and clinicians (and many examples from the latter are detailed by Ecker et al., 2012; see also Högberg et al., 2011; Gray & Liotta, 2012; Xue et al., 2012).

Yet another important and elegant demonstration described by Schiller et al. (2010) concerns the memory specificity of reconsolidation and erasure. Any longstanding piece of emotional learning typically has linkages to many other learnings and memories. If the erasure process is to be clinically useful and safe in humans, it must affect only the target learning and not its network of linkages. Destabilization of a target learning without destabilizing closely associated learnings was first reported in an animal study by Debiec, Doyère, Nader, and LeDoux (2006). Schiller et al. then demonstrated with human subjects that reconsolidation can eliminate a specific implicit learning with surgical accuracy, leaving intact an adjacent learned fear that was formed in the same original experience.

This was done with a separate group of subjects who, on Day 1, underwent essentially the same initial training experience as described above except for the addition of squares of a third color I will call brown (Schiller et al. did not indicate the actual third color), 37.5% of which, as with the yellow squares, were accompanied by a shock. In that way, subjects learned to expect a shock in response to squares of two colors, yellow and brown.

On Day 2, Experience 1 of the procedure described above—memory reactivation by a single presentation of a square with no shock—was carried out with a yellow square but not with a brown square. Experience 2, the 10-min TV viewing, and Experience 3 were then implemented as described above, with Experience 3 now including no-shock presentations of squares of all three colors, for new learning that yellow and brown squares are harmless.

On Day 3, another series of no-shock presentations of squares of all three colors revealed that the fear response to yellow squares no longer occurred and had been erased, but the fear response to brown squares was reevoked and had not been erased. In oth-

er words, only the fear learning that had received a reactivation-and-mismatch prior to the new learning in Experience 3 had been erased by that new learning (though as noted, no mention of mismatch is made by Schiller et al.). The series of shock-free squares in Experience 3 served as erasure learning for the yellow squares and as an extinction training for the brown squares. The erasure of fearful expectation of a shock for squares of one color had not spread associatively to the other color, showing that the subcortical emotional memory system is capable of great selectivity and accuracy in destabilizing and revising learnings. Closely adjacent implicit memories are handled independently, as is necessary for safe clinical use of reconsolidation.

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