INTERNATIONAL JOURNAL OF NEUROPSYCHOTHERAPY

IJNPT

VOLUME 6, ISSUE 1 (2018) ISSN 2202-7653

INTERNATIONAL JOURNAL OF NEUROPSYCHOTHERAPY
VOLUME 6 ISSUE 1 - 2018

VOLUME 6 - 2018

Aims & Scope

The International Journal of Neuropsychotherapy (IJNPT) is an open access, online journal that considers manuscripts on all aspects of integrative, biopsychosocial issues related to psychotherapy. IJNPT aims to explore the neurological or other biological underpinnings of mental states and disorders to advance the therapeutic practice of psychotherapy.

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Editor's Note

This issue of the *International Journal of Neuropsychotherapy* (IJNPT) marks another significant shift for the journal. In its inception the IJNPT was a product of *The Neuropsychotherapist* as a peer reviewed and open access journal for those writing and researching the nascent field. The journal was then handed over to the International Association of Neuropsychotherapy (IACN) for Volume 5 in 2017 and was administrated by Pieter Rossouw's company Mediros. Now, after the loss of Pieter Rossouw in early 2018, the journal is fully administrated and published by the IACN.

To begin this new season of administration the IACN is pleased to present a special issue of the IJNPT featuring a comprehensive article by Bruce Ecker on the clinical application of memory reconsolidation. I consider the transforming effects of memory reconsolidation to be one of the most important discoveries in psychotherapy. Ever since Freud's topographical model of the unconscious mind, memory, operating between the conscious and subconscious mind, has been integral to our self perception. I hold this view, not from academic acknowledgment of sound science but from an experiencing the powerful effects of memory reconsolidation both through demonstration by masterful clinicians and in my own therapy room. So it is with much personal bias that I recommend what Bruce is proposing in the following pages.

It is my hope that as the IACN progresses the IJNPT will become a significant open access resource of knowledge for everyone in the fields of mental health. And I do hope you find significant clinical advantage from this powerful understanding Bruce Ecker brings us in this issue.

Matthew Dahlitz

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Clinical Translation of Memory Reconsolidation Research:

Therapeutic Methodology for Transformational Change by Erasing Implicit Emotional Learnings Driving Symptom Production

Bruce Ecker

Abstract

After 20 years of laboratory study of memory reconsolidation, the translation of research findings into clinical application has recently been the topic of a rapidly growing number of review articles. The present article identifies previously unrecognized possibilities for effective clinical translation by examining research findings from the experience-oriented viewpoint of the clinician. It is well established that destabilization of a target learning and its erasure (robust functional disappearance) by behavioral updating are experience-driven processes. By interpreting the research in terms of internal experiences required by the brain, rather than in terms of external laboratory procedures, a clinical methodology of updating and erasure unambiguously emerges, with promising properties: It is applicable for any symptom generated by emotional learning and memory, it is readily adapted to the unique target material of each therapy client, and it has extensive corroboration in existing clinical literature, including cessation of a wide range of symptoms and verification of erasure using the same markers relied upon by laboratory researchers. Two case vignettes illustrate clinical implementation and show erasure of lifelong, complex, intense emotional learnings and full, lasting cessation of major long-term symptoms. The experience-oriented framework also provides a new interpretation of the laboratory erasure procedure known as post-retrieval extinction, indicating limited clinical applicability and explaining for the first time why, even with reversal of the protocol (post-extinction retrieval), reconsolidation and erasure still occur. Also discussed are significant ramifications for the clinical field's "corrective experiences" paradigm, for psychotherapy integration, and for establishing that specific factors can produce extreme therapeutic effectiveness.

KEYWORDS: Memory reconsolidation, clinical translation, destabilization, psychotherapy, memory erasure, behavioral updating, memory interference, emotional schema, transformational change, unlearning, specific factors, reactivation-extinction, retrieval-extinction, corrective experiences

Submitted: January 19, 2018. Accepted for publication: January 29, 2018 Published online: June 7, 2018

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Cite as: Ecker, B. (2018). Clinical translation of memory reconsolidation research: Therapeutic methodology for transformational change by erasing implicit emotional learnings driving symptom production. *International Journal of Neuropsychotherapy, 6*(1), 1–92. doi: 10.12744/ijnpt.2018.0001-0092

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1. Introduction

A primary dilemma in clinical psychology has been described by one of that field's leading voices in this way: "After decades of psychotherapy research, we cannot provide an evidence-based explanation for how or why even our most well studied interventions produce change, that is, the mechanism(s) through which treatments operate" (Kazdin, 2007, p. 1). The present article proposes that a fundamental breakthrough in that dilemma may be developing through the translation of memory reconsolidation neuroscience into clinical application.

Memory research has identified an innate type of neuroplasticity in the brain, known as memory reconsolidation, that can destabilize the neural encoding of learnings of many types, including emotional learnings. Destabilization in turn allows the target learning to be nullified either endogenously, by behavioral counter-learning, or exogenously, by pharmacological blockade that disrupts the natural molecular and cellular process of restabilization, or reconsolidation, that normally would occur after several hours (Duvarci and Nader, 2004; Pedreira et al., 2002; Pedreira and Maldonado, 2003; Walker et al., 2003). Thus nullified, the subsequent durable, robust disappearance of all expressions of the target learning has been termed its erasure by many researchers (e.g., Kindt et al., 2009; for reviews see, e.g., Agren, 2014; Nader, 2015; Reichelt and Lee, 2013; Schwabe et al., 2014; for a review of early, anomalous observations of erasure prior to discovery of reconsolidation, see Riccio et al., 2006). By putting the transformational change of memory on empirical solid ground, research on memory reconsolidation has paved the way for new common ground between neuroscientists and clinicians, who have filed fine-grained anecdotal reports of such transformational change for decades (e.g., Ecker and Hulley, 1996, 2000a, 2008; Fosha, 2000; Greenberg et al., 1993; Shapiro, 2001).

Memory reconsolidation is a neurological process that is experience-driven: behavioral and perceptual events trigger it into occurring and can govern the resulting effects on the target learning. The relevance of reconsolidation research findings to psychotherapy is potentially very great because clinical symptoms are maintained by emotional learnings held in implicit memory, outside of conscious, explicit awareness, in a wide range of cases, including most instances of insecure attachment, post-traumatic symptomology, compulsive behavior, addiction, depression, anxiety, low self-esteem, and perfectionism, among many other symptoms (e.g., Greenberg 2012; Schore, 2003; Toomey and Ecker, 2007; Van der Kolk, 1994). A versatile, reconsolidation-based clinical methodology that targets and reliably nullifies the specific emotional learnings maintaining such symptoms would revolutionize the field of psychotherapy. Envisioning that new landscape, neuroscientists Clem and Schiller (2016, p. 340) wrote, "To achieve greatest efficacy, therapies… should preclude the re-emergence of emotional responses." Defining complete elimination of unwanted emotional responses as the goal of psychotherapy is a statement that no neuroscientist would have ventured to make prior to 2000, before the discovery of memory reconsolidation. It is a goal now recognized as a possibility grounded in empirical research. That goal is the operational definition of erasure in this article: lasting, effortless, complete cessation, under all circumstances, of an unwanted behavior, state of mind, and/or somatic disturbance that had occurred either continuously or in response to certain contexts or cues.

Currently, at the end of the second decade of laboratory research into reconsolidation, researchers' attention is extending to considerations of clinical translation at a rapidly accelerating pace (e.g., Beckers and Kindt, 2017; Dunbar and Taylor, 2016; Elsey and Kindt, 2017a; Krawczyk et al., 2017; Kroes et al., 2015; Lee et al., 2017; Nader et al., 2014; Treanor et al., 2017). Those authors have consistently called for a two-way flow of knowledge between researchers and clinicians in order to achieve the fullest clinical utilization of memory reconsolidation. Nader et al. (2014, p. 475) wrote:

> We feel that ongoing discourse between mental health clinicians and neuroscientists is beneficial both for scientific progress in neuroscience and mental health treatments. Neuroscientists may benefit from being educated about clinical models of mental disorders…. The reductionist approach intrinsic to scientific activity forces neuroscientists to simplify their models in the pursuit of scientific questions considered to be of a fundamental nature. Unavoidably, at times, this approach may ignore some aspects of mental disorders. A discourse with clinicians allows neuroscientists to realign their models to ensure that they represent processes thought to cause or maintain these disorders.

Similarly, researchers Elsey and Kindt (2017a) opined that "Dialogue between researchers and clinicians must be maintained" (p. 114) and, in concluding an extensive review of the prospects for effective clinical application of reconsolidation research findings, commented, "there are significant limitations to experimental research, and ultimately only attempts at treatment can reveal the utility of a reconsolidation-based approach" (p. 115).

Those comments serve to define the purpose of the present article, which is a report from the clinical trenches of observations made in the course of directly applying the empirically identified, endogenous process of memory erasure. This article describes what appear to be encouraging advances. The author, a psychotherapist and former research physicist, has since 2005 maintained close scrutiny of reconsolidation research while also closely observing the effects in therapy sessions of processes designed to translate memory reconsolidation research into clinical application.

Members of the clinical domain have been enthusiastically consuming and working to utilize the knowledge being generated by laboratory neuroscience researchers since the 1990s (e.g., Siegel, 1999; van der Kolk, 1994). There has been little to indicate a flow of knowledge in the other direction, however. Undoubtedly there is more than one reason for that asymmetry, which is particularly acute at present as regards reconsolidation. There is now a substantial clinical literature that documents observations ascribed to reconsolidation and that delineates clinical methodologies demonstrating translation of reconsolidation research (e.g., Ecker, 2008, 2010, 2015a,b, 2016; Ecker and Hulley, 2008, 2017; Ecker and Toomey, 2008; Ecker et al., 2012, 2013a,b; Högberg et al., 2011; Lasser and Greenwald, 2015; Sibson and Ticic, 2014; Soeter and Kindt, 2015a; Ticic and Kushner, 2015). Rarely, however, is such literature cited in the writings of laboratory researchers, who regularly express anticipation of and need for advances already made by clinicians. Examples of that are myriad; the two most recent instances encountered by the author are these: Krawczyk et al. (2017, p. 16) commented that "outside the laboratory settings such as in clinical ones, it is unclear how the reconsolidation process might work." Elsey and Kindt (2017a, p. 114) commented that laboratory research has focused largely on fear learnings and that "experiences of other emotions, such as disgust…or of more complex feelings such as guilt and shame after reconsolidation-based procedures are essentially untapped." In fact, numerous clinicians' reports have documented in a fine-grained manner how a wide range of complex emotions and emotional learnings have been subjected to the empirically confirmed reconsolidation process of behavioral erasure (see citations above in this paragraph; for online listings of relevant clinical reports, see http://bit.ly/2tKXdyX and http://bit.ly/15Z00HQ). Section 7 of this article provides samples of such clinical work and its documentation.

The rigor of the clinical observations reported here is of a different type from that of the quantitative measurements made in laboratory controlled studies by neuroscientists. Here the aim is phenomenological rigor that capitalizes on the unique ability of human subjects (therapy clients) to direct attention to their own mental and emotional states and to describe the moment-to-moment effects as the steps of the destabilization and erasure process are carried out. Neuroscientists have barely begun to utilize such articulation of subjective experience for gaining access to the memory reconsolidation process, but even their first forays in that direction were very fruitful (Sevenster et al., 2013, 2014). The clinical case studies documented in this article are intended to show that examining the raw data of therapy clients' real-time phenomenological reports can significantly help advance the clinical translation of memory reconsolidation research (see also Heatherington et al., 2012).

The clinical work reported here is intended to demonstrate the application of reconsolidation research, so an examination of relevant research and its translational implications precedes the clinical material detailed in Section 7. As noted, reconsolidation has been demonstrated and studied for many different types of memory, but the research covered here is limited to how the process applies to emotional learning and emotional memory, as they play by far the principal role in psychotherapy. (See reviews cited above for the full range of research.) The cellular and molecular levels of reconsolidation research are also not covered here. Clinicians need not attend to the highly complex neurophysiological and neurochemical substrates of destabilization and erasure (for a review of which, see Clem and Schiller, 2016). However, clinicians should understand that robust functional erasure does not

necessarily correspond to total loss or ablation of the entire neural encoding of the erased responses and learnings, according to recent findings (Ryan et al., 2015), and any simplistic image of what happens to neural circuits when erasure is achieved is almost certain to be significantly incorrect.

Lastly, regarding this article's usage of an emotional "learning": A terminology bridge between neuroscientists and clinicians is much needed. Memory researchers as a rule refer to a learned item of any type as a "memory," not as a "learning"; they refer to the "target memory" rather than the "target learning." If the learned item in question is, for example, implicit knowledge that would be verbalized as "If I express myself I'll be criticized and rejected," researchers would refer to that as the "memory" under study. That usage of "memory," while perfectly clear to memory researchers, is likely (in the author's experience) to create considerable confusion for clinicians, who would tend to understand "memory" as referring to the person's episodic memory and/or declarative memory of the original childhood events involving rejection, rather than the semantic memory consisting of a generalized model and expectation of people being active rejecters. In order to avoid that confusion for clinician readers (this article being intended for both memory researchers and clinicians), the text here refers to an "emotional learning." That syntax is identical to how "understanding" may be used as in "it resulted in the understanding that…."

2. Emotional learnings and memories underlying clinical symptoms

For exploring the clinical application of memory reconsolidation research, a realistic view of the emotional learnings typically encountered in psychotherapy is necessary. The characteristics delineated in this section figure extensively in subsequent sections of this article.

Understanding symptom production as an effect of learning and memory is a well established perspective within clinical psychology and cognitive neuroscience (e.g., Bouton et al, 2001; Eysenck, 1976; Mineka & Zinbarg, 2006). At the outset of psychotherapy, the implicit emotional memories and learnings underlying and maintaining a therapy client's specific symptom(s) are largely or completely outside of awareness, as a rule. However, in nearly all cases they can be brought into direct, conscious experience and accurate verbal representation not through analytical insight, but using experiential methods developed for that purpose (e.g., Badenoch, 2011; Ecker and Hulley, 1996; Ecker et al., 2012; Ecker and Toomey, 2008; Greenberg et al., 1993; Lipton and Fosha, 2011; Shapiro, 2001). Though initially implicit and nonverbal, the symptom-generating learnings prove to be well-defined and sufficiently retrievable and accessible for further therapeutic processing to proceed. They also prove to be held in two different types of memory: episodic memory of the personal, subjective experience of particular experiences and events (not to be confused with declarative, factual memory of the same events) (Tulving, 2002, 2005), and semantic memory of generalized patterns, rules, mental models, expectations and meanings (Markus and Wurf, 1987; Reber, 1989). (For a review of those two memory systems and their linkage, see Ryan et al., 2008.)

Episodic memories that generate clinical symptoms are those that contain unresolved distress of an intensity that the individual is unable or unwilling to fully forget, resolve, or contain, and those that contain reward or pleasure so potent as to generate obsessive craving and compulsive behavioral repetition. The most extreme forms of those situations may be, respectively, the post-traumatic condition in which highly distressing episodic memory intrudes into awareness, commonly known as flashbacks, and addiction. Other problematic expressions of episodic memory are very common. For example, many people carry a large number of episodic memories of specific mistreatments inflicted by a sibling, and the cumulative hurt or anger of this set of memories has strong effects on mood and behavior during family gatherings and possibly in non-family situations as well; the individual might strive unconsciously to get relief from the feeling of being at the bottom of the pecking order inside the family by dominating others outside the family, resulting in interpersonal problems or job firings.

The mind and brain actively extract generalized patterns, abstractions and meanings from particular experiences, in order to be ready and oriented for novel situations in which similar features appear. Such generalized or schematic knowledge constitutes implicit semantic memory (Dunsmoor et al., 2009; Frith and Frith, 2012; Seger and Miller, 2010). Operating entire-

ly outside of awareness, this form of memory generates a vast range of clinical symptoms. For example, consider the emotional learnings brought into awareness in therapy by a married, middle-aged man who sought relief from chronic depression, anxiety, bouts of shame, and compulsive viewing of pornography. Throughout his childhood, his expressions of distress or needs were regularly met with his parents' frightening anger or cold dismissal, responses that inflicted an even more intense suffering than he was initially feeling. That often-repeated experience set up this cluster of semantic emotional learnings that had no verbal or conceptual representation:

- the knowledge that his very being is disgusting and unacceptable (which generates shame; a young child's intense feelings of needs and distress seem to be his very being)
- the generalized expectation of receiving the same responses from anyone, were he to express any distress or need (which generates his anxiety)
- the expectation that his entire lifetime will be desolately devoid of caring understanding, warmth, help or comfort from others (which is both frightening, adding to his anxiety, and also generates despair felt as his mood of depression)
- the urgent necessity of avoiding the expected responses of anger or indifference by never expressing or even feeling his own distress or needs (which requires dissociation of feelings and avoidance of intimacy, and maintains perpetual aloneness, which is another source of despondency felt as depression)
- the urgent, ongoing need to blot out and escape the engulfing desolation, despair, aloneness, and fear (by frequently filling his consciousness with the intensely pleasurable stimulation of pornography and accompanying fantasies)

Those adaptive yet symptom-generating emotional learnings are specific, well-defined and coherent constructs, yet prior to being retrieved, felt, and verbalized in therapy, they existed only in implicit memory and operated outside the explicit domain of words, concepts and conscious awareness. They are examples of semantic memory, as distinct from episodic or autobiographical memory of particular experiences and events; they are generalized, schematic patterns abstracted from the concrete instances experienced by the individual. They are emotionally compelling models of reality, and the symptoms they generate are coherently necessary according to each construct's model of reality. The importance of addressing generalized emotional learnings in psychotherapy is widely recognized (e.g., Beckers and Kindt, 2017; Dunsmoor et al., 2015; Lane et al., 2015).

The mind's organization of acquired implicit knowledge into schemas has long been an important feature of cognitive science and its clinical applications (e.g., Eichenbaum, 2004; Foa and Kozak, 1986; Rumelhart and McClelland, 1986; Toomey and Ecker, 2007; for a review see Ghosh and Gilboa, 2014). A schema is a coherent, composite mental model consisting of linked, related elements; for example, the five items-of-learning listed above are linked components of one schema in semantic memory. Emotional schemas "carry our emotional learning and memories and are responsible for the provision of the majority of our emotional experience….These affective / cognitive / motivational / behavioral emotion schemes are thus a crucial focus of therapeutic attention and...are important targets of therapeutic change" (Greenberg, 2012, pp. 698–699).

Semantic and episodic memory are not completely dissociated systems, but their linkages are a complex and subtle matter (reviewed by Ryan et al., 2008). A frequent observation in clinical practice is that consciously accessing either one does not necessarily also consciously access the other. Likewise, attending to an emotional response or behavior generated by either one does not automatically consciously access the underlying episodic or semantic material. There have been many cases in the author's clinical experience of a therapy client retrieving an emotionally potent schema into lucid awareness from semantic memory without this bringing any corresponding episodic memory of the experiences in which the schema was learned. In some of those cases, the client was then able to retrieve episodic memory through deliberate internal searching, but in other cases was not able to do so and remained mystified by how the retrieved schema had been learned. Though awareness of episodic memory is helpful to therapy, and can itself serve as a portal for accessing semantic memory, absence of episodic

memory is found clinically not to be an obstacle to unlearning and nullifying a retrieved emotional schema through the memory reconsolidation process.

As can be seen in the examples listed above, the schema is the root cause of any symptom that it generates, so any symptom based in emotional schemas ceases to occur as soon as all of that symptom's underlying schemas have been unlearned and erased. Therefore, for any symptom produced by an emotional schema, the schema is the optimal target of change, rather than the symptom (Ecker and Toomey, 2008). Attempting to prevent or reduce a symptom with counteractive methods that leave the underlying memory material intact positions a therapy client to be prone to relapses (Ecker et al., 2012).

There is a class of symptoms that serve the function of suppressing all awareness of distressing episodic memory or distressing knowledge that conscious episodic memory would create. Examples of episodic-memory-suppressing symptoms are disconnection from affect, compulsive eating, continual self-distraction via compulsive focus on work, video games, pornography or any other form of intense excitement such as gambling, and avoidant behaviors that prevent encounters with specific reminders of episodic memory. Such symptoms of episodic memory avoidance are not produced directly by the episodic memory that is being avoided. They are produced, rather, by the implicit (non-conscious) expectation that experiencing the avoided episodic memory would be unsurvivably overwhelming, damagingly devastating, or cause insanity. That expectation and the rule of avoidance that it necessitates are semantic memory formations. Thus, this episodic-memory-avoiding class of symptoms is produced by semantic memory, and the optimal target of change is the expectation of devastation.

Clinical experience reveals yet another subtlety of the interplay of episodic and semantic memory: In any subjective experience recalled in episodic memory, the particular emotional qualities and felt meanings of the experience are produced on the basis of the semantic knowledge that was already operating at the time (mental models, attributed meanings, rules, roles, expected patterns and sequences, etc.). Formation of semantic knowledge through implicit learning has been detected at quite early developmental stages (e.g., DeCasper and Carstens, 1981; Olineck and Poulin-Dubois, 2005; Repacholi and Gopnik, 1997), and its involvement in episodic memory has been noted by Tulving (2002, p. 5): "It [episodic memory] makes possible mental time travel through subjective time, from the present to the past, thus allowing one to re-experience, through autonoetic awareness, one's own previous experiences. Its operations require, but go beyond, the semantic memory system."

An illustration of how semantic knowledge generates the emotional quality of a given experience, both in its original occurrence and in episodic memory recall, is provided by one of the author's clinical cases (Ecker et al., 2012, pp. 86–91). The client is a woman in her 30s who, during a therapy session, unexpectedly began experiencing, for the first time, intrusive episodic memory of a traumatic experience at age 8, when she was in the rear seat of the family car as her drunk father drove her, her mother and sister on a careening, lurching trajectory at high speed toward a bridge visible in the distance. By the very nature of the flashback, she was not merely remembering the incident; rather, she was re-inhabiting the scene and the experience, and describing it from the vantage point of being there in the car as the living memory re-played itself in the present. She felt the car graze the railing at the side of the road and knew she was going to die. Feeling helpless in hurtling toward her death, her body was frozen and stiff in panic. Yet her traumatizing feelings of helpless vulnerability and panic were not actually caused by the external physical situation. Rather, they were caused by her particular semantic knowledge. The plausibility of that assertion becomes apparent through the thought experiment of imagining a different 8-year-old in her place in that car: a boy who had recently moved in next door with his family, who needed a ride, who had spent most of his eight years out on the streets among violent youth, and who had remained alive by being as assertive and aggressive as necessary for doing so. This boy's semantic knowledge of the rules dictating his possible responses is quite different. As soon as he sees the degree of danger developing in the car, he lunges forward, grabs the driver's hair with one hand and throat with the other and screams in his ear, "If you want another breath, motherf****r, you hit those brakes and pull over right now!" The driver does exactly that in seconds, and the boy gets out. He knew he would take command of the situation, never felt helpless, and therefore experienced the incident not as a trauma, but as only another momentary set of choppy waves in a much bigger choppy

ocean of life.

The therapist guided the woman through an imaginal empowered re-enactment experience of screaming at her father, commanding him to stop the car. When he did not do so immediately, she opened the car door anyway, and then he applied the brakes and pulled over, and she exited from the car to safety, calling to other drivers for help and police assistance. That imaginal experience felt vividly and almost physically real to her. Such assertive behavior not only violated her semantic rules, but also disconfirmed what had seemed to be their inviolability and absoluteness. Thereafter, the episodic memory no longer contained helplessness, and the somatic frozen state, which was a frequent and, until this session, a mystifying symptom in various social situations in her adult present, ceased to occur. She retained declarative (factual, cognitive) memory of having suffered terror in that car incident, but recalling the incident no longer re-aroused that feeling of terror as part of the episodic memory. (See Kindt et al., 2009, and Soeter and Kindt, 2012, for laboratory studies that demonstrated such retention of declarative memory after erasure of fear.) The semantic and emotional components of an episodic memory prove to be mutable independently of the perceptual components of the memory.

Clinical observations such as that one seem to indicate a phenomenology that operates in this manner: When an episodic memory is retrieved, addressed in therapy and successfully updated, transforming the emotion inherent in the memory, what has been updated is the semantic knowledge that was operating at the time of the original experience and has been an implicit component of the episodic memory. That update fundamentally and retroactively changes the encoded personal meaning of the experience, which in turn changes the emotion generated by the incident as it now exists in episodic memory. Declarative, factual memory of the concrete happenings of course remains unchanged; it is the (semantic) personal significance and expected contingencies of those happenings that have been transformed. A common clinical instance involves erasure of what is referred to in Coherence Therapy as parents' *terms of attachment* (Ecker et al., 2012, pp. 102–114), consisting of rules that define how connection, acceptance, and punishment work, and that are installed in the child's implicit semantic knowledge, such as the rule that "I must obey my parents' rules requiring my compliance and non-assertiveness." Nullification of that rule in a replay of a distress-laden episodic memory can transform an experience of helplessness, defenselessness and passive victimization into one of agentive self-assertion and self-protection. That shift in turn transforms the emotional quality of the memory from traumatizing endangerment and terror into a far reduced degree of dysphoric feelings and meanings, such as troubled recognition of parents' self-absorption and incapacity to give emotional understanding. Such lesser distresses are directly amenable to therapeutic processing, such as by the emergence of feelings of anger and/or grief that have until now been blocked.

Thus, semantic memory appears to be always the critical target of the updating and erasure process, even when the working target memory is an experience in episodic memory. The episodic memory serves as a portal to the semantic knowledge governing the emotional quality of the experience. (The same principle is central to Coherence Therapy and is formulated as, "How a person experiences and responds to a situation is caused not by circumstances, but by viewing circumstances through the lens of unconscious personal constructs…" (Ecker and Hulley, 2017a, p. 1)). The same phenomenology is illustrated in clinical case examples in Sections 7.1 and 7.5.4.

Schemas in semantic memory, being derived from particular experiences, have linkages to episodic memory (Ryan et al., 2008), as well as to schema-driven emotional states and implicit procedural knowledge of adaptive (if consciously unwanted) responses. Any or all of those components of memory may have to be navigated in the unavoidably complex course of therapy with a particular client, and any of those components may serve as a portal or pathway for accessing the others, though the linkages may be faint or barricaded and require inner work to utilize. The full constellation of linked components has been delineated by Ecker and Hulley (1996, 2017) and by Ecker et al. (2012, pp. 53–54), who define this orienting map for clinicians:

- *[Episodic memory] Perceptual, emotional, and somatic memory of original experiences:*
  This is the "raw data"; matching features in current situations are triggers of activation of either episodic or semantic memory and the internal

and/or behavioral responses they generate.

- *[Semantic memory] A mental model or set of linked, learned constructs operating as living knowledge of a problem and a solution:*
  - *The problem: knowledge of a vulnerability to a specific suffering*
    This is an ontological model of how the world works in some area (self, others and/or the nature of the world itself), and current situations that appear relevant to this model are triggers of the whole schema.
  - *The solution: knowledge of an urgent strategy and specific tactics (internal and/or behavioral) for avoiding that suffering; or*
  - *Knowledge of lacking any solution to the problem*
    This drives emotional states of helpless fear/anxiety and/or helpless despair/depression, plus behavioral expressions of those emotional states (such as insomnia, inaction or substance abuse)

Thus there are several pathways of access by which a therapy client can arrive at direct, affective awareness and verbalization of the schema(s) and/or memories generating a given symptom. Each pathway may be characterized by its starting point:

- The client's *behavior* in the problematic state or situation
- The client's *mood, emotion or emotional reactivity* in the problematic state or situation
- *Somatic disturbances* in the problematic state or situation
- An *image* that arises in considering the problematic state or situation
- Identification of *feature(s) common to all instances* in which the symptom has occurred
- *Episodic memory* of formative events and experiences earlier in life, whether coherent or fragmentary
- The *contents of a dream*, particularly if the dream is recurring or if the dream occurred on the night before or after a therapy session

Ecker and Hulley (2017a, p. 8), emphasizing that a clinician's awareness of all avenues for retrieval of schemas is important for optimally efficient and effective therapy, state:

> The discovery work could, for a particular client, most readily open up through focusing on an image that has arisen, or on a kinesthetic sensation, for example, rather than through an initial focus on an emotion or mood. Then, as discovery and accessing proceed, all other components of the full…schema come into being experienced and processed, including the affective dimension. In short, to regard affect as the necessary point of access to the deeper material greatly limits the many ways and many opportunities through which the therapist can usher the client into the material. The core material, too, may or may not be experienced as predominantly emotional. It is experienced by some clients more intensely as a felt meaning than as emotion.

The above multi-component view of memory in relation to symptom production and psychotherapy has been reiterated by Lane et al. (2015), though with some notable differences, among them the present article's emphasis on semantic memory as being the primary target for updating and erasure through memory reconsolidation. Section 9.3 provides a more extended discussion of those authors' approach to reconsolidation-oriented psychotherapy.

Psychotherapists observe in daily practice the tenacious, long-term persistence and retriggering of implicit emotional learnings formed decades earlier. That durability has also been well established by researchers, who went so far as to characterize emotional learnings as "indelible" (LeDoux et al., 1989) prior to the discovery of memory reconsolidation. Pine et al. (2014, p. 1) observed that "A unique feature of preferences [acquired, emotionally compelling 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." Selection pressures in the course of evolution favored the unfading retention of emotional learnings: any learning accompanied by strong emotion is made exceptionally durable, due in large part to the effects of emotion-related hormones on the memory encoding

process (McGaugh, 1989; McGaugh and Roozendaal, 2002; Roozendaal et al., 2009).

While implicit emotional learnings are resistant to extinction procedures, they are susceptible to the memory reconsolidation process, as many researchers have demonstrated (e.g., Pine et al., 2014; Reichelt and Lee, 2013; Schiller et al., 2010). It is a consistent clinical observation, described in detail in Section 7, that as soon as a particular emotional learning is verifiably unlearned, nullified and erased through the process identified in memory reconsolidation research, the symptoms it has been maintaining cease to occur (Ecker, 2015a; Ecker and Toomey, 2008; Ecker et al., 2012, 2013a). That observation lends support to the view that discrete modules or schemas of emotional learning are the root cause of the symptoms they maintain.

Viewing symptom production as internally driven by schemas and memories is not to deny or neglect the role of systemic and social processes in maintaining symptoms. Rather, the individual's implicit emotional learnings are formed in response to the entire experiential ecology in which she or he is immersed, including all received systemic and social meanings, messages, contexts and contingencies, and are the very means of their influence (Bateson, 1979; Hermans and Dimaggio, 2007; Siegel, 2015). Systemic and social aspects are often of primary importance in the emotional learnings accessed in therapy.[1]

Another relevant clinical observation is the uniqueness of the underlying emotional learnings brought into awareness by different therapy clients who have presented the same type of symptom, such as panic attacks or dysthymic depression. For example, one woman's full-strength, physiological panic attacks were found to arise from her expectation of absolute rejection by her father were she to fail to be "head and shoulders above" (superior to) all others at all times (Ecker, 2015a: NPT article); whereas another woman's physiologically similar panic attacks were driven by the urgent necessity of fulfilling the weekly quota of suffering that the universe requires of each family, a quota which the universe is ready to fulfill at any time by inflicting catastrophe, if it is not already fulfilled adequately in the ordinary course of things (Ecker and Hulley, 2000a). One middle-aged woman's depression was actually her mood-state of despair and hopelessness following her "illegitimate" pregnancy which, at the age of 18 in a conservative small town, had plunged her into certainty of lifelong stigma, rejection and ruin, an expectation that had never been updated by the subsequent, actual course of events later in her life (Ecker, 2015a); whereas another woman's depression arose from feeling devoid of interests and motivation and expecting her entire future life to feel the same, without awareness that this state of blankness was a deliberate, self-protective tactic that she had resorted to in desperation as a child, in order to prevent her severely self-absorbed mother from continuing to take over, take away and take credit for everything and anything the daughter ever did or enjoyed (Ecker and Hulley, 2002).

As can be seen in the foregoing examples of implicit emotional learnings that maintain symptoms, they are adaptive in that they consist of living knowledge of a particular suffering plus either how to avoid it or the dire dilemma of having no way to avoid it. (For a more detailed mapping of the content and structure of symptom-generating emotional schemas, see Ecker et al., 2012, pp. 53–55; Ecker and Hulley, 2017a.) They are also coherent, in the sense that they consist of a sensible, well-knit account of how suffering and safety operate, a mental model that is faithfully based on what was personally and subjectively experienced earlier in life (Ecker and Toomey, 2008; Toomey and Ecker, 2007).

The recognition that implicit emotional learnings are inherently adaptive, coherent, and neurologically built to persist for a lifetime amounts to a non-pathologizing view of symptom production that contradicts the widespread characterization of "pathogenic," "maladaptive" beliefs driving symptom production. The emotional learning and memory systems of each of the five therapy clients described above certainly were generating unwelcome behaviors and states of mind and body, yet were functioning properly in doing so, not malfunctioning or dysfunctioning, similar to how the unwelcome swelling, painful tenderness and redness around a recent wound express the proper functioning of healing and immunity systems. In that sense, many conditions often termed "disorders of emotional memory" are not actually disorders at all. To describe a therapy client's core beliefs or schemas as incorrect,

1 Of course, there are many symptoms that are not caused by emotional learning and therefore cannot be dispelled by memory reconsolidation, including physiologically based conditions such as hypothyroidism-induced depression, neurologically based conditions such as dyslexia and autism, and emotional styles based in genetically determined temperament.

maladaptive or pathogenic is actually to accuse the process of natural selection of having those attributes, because a person's persisting beliefs and schemas exist due to the proper functioning, not the malfunctioning, of the emotional brain.

The task of psychotherapy, in this view, is to facilitate the thorough unlearning, via memory reconsolidation, of the compelling expectations, meanings, models, roles, rules and tactics that were learned earlier in life, and are now maintaining unwanted effects, and can now be updated and replaced by more effectively adaptive constructs. That is an unlearning of semantic memory, an unlearning of how events and experiences were construed and have continued to be construed by the implicit knowledge system (Ecker and Toomey, 2008; Ecker et al., 2012). In the subjective experience of such unlearning, some aspect of the world that has felt compellingly real and inescapably life-constraining is recognized as a mirage that has no reality at all. That nullification, which is clearly apparent in the clinical case examples in Section 7, leaves intact one's episodic, autobiographical memory of events and experiences in one's life. (For a laboratory demonstration that event memory is unaffected by erasure of emotional learning, see Kindt et al., 2009.)

If, as noted above, emotional learnings are the root cause of the symptoms they drive, then their observed heterogeneity for the same symptom implies that psychotherapeutic treatment, if it is to dispel the symptom at its roots, must be uniquely tailored to each client. As described in Sections 4 and 5, memory reconsolidation research has come to the same conclusion, namely that the specific, unique features of a target emotional learning dictate the design of the experiences needed to induce the destabilizing, unlearning, and nullifying of it. Section 6 shows that research has identified a well-defined, endogenous process that is readily tunable to the unique contours of the target learnings discovered in each clinical case.

3. Verification of memory reconsolidation in psychotherapy

Laboratory researchers conduct a variety of direct neurological tests on animal subjects to ascertain with certainty whether or not reconsolidation has occurred. The definitive tests are either toxic or require euthanizing, ruling out use with human subjects. Consequently, "As we do not have any incontrovertible neural measure of whether reconsolidation has taken place in humans, we can only indirectly infer its presence…." (Elsey and Kindt, 2017a, p. 114). This situation begs the question: Are the indirect markers of reconsolidation sufficiently clear and substantial to verify reliably its successful induction in psychotherapy? Therapists having reliable markers for verifying their use of reconsolidation is obviously a necessity.

The verification markers that reconsolidation researchers themselves use in human studies are presumably the best choice. For verifying reconsolidation in human studies, researchers rely upon the behavioral markers of erasure observed in animal studies where the decisive tests were conducted, confirming that reconsolidation had occurred. The prototype of that is the landmark human study by Schiller et al. (2010), the first in which a fear memory was erased. Schiller et al. concluded that reconsolidation had occurred by observing the same behavioral markers of erasure as were observed in rats by Monfils et al. (2009), who used largely the same procedure and also confirmed reconsolidation decisively. The logic of regarding erasure as confirmation of reconsolidation also takes into account the fact that reconsolidation is the brain's only known neuroplastic process that can produce those markers.

There are three well-defined behavioral markers of erasure:

1. *Non-reactivation:* An acquired physiological and/or affective response that formerly occurred immediately upon perceiving a certain cue or context no longer occurs. (For example, the rate of a mouse's heartbeat no longer increases upon seeing the red light that formerly became associated with soon receiving a foot shock.)
2. *Non-expression:* The overtly manifested behavioral expression of that physiological and/or affective response no longer occurs. (The mouse no longer freezes in response to the red light.)
3. *Effortless permanence:* The above two changes persist without relapse under all conditions and without any further training or special conditions implemented to maintain them.

Erasure in the clinical context, as noted in Section 1,

means lasting, effortless, complete cessation under all circumstances of an unwanted behavior and/or state of mind and/or somatic disturbance that had been a persistent occurrence, particularly in certain contexts or in response to certain cues. In other words, the emotional learning in question can no longer be reactivated into being felt affectively or somatically, or into being expressed behaviorally or physiologically. The therapy client's experience and behavior are now enduringly as though the target learning no longer exists (though, as noted in Section 1, there is evidence that portions of the target learning's engram or physical encoding may still exist (Ryan et al., 2015), as reviewed by Clem and Schiller (2016)).

The definition of erasure in the previous paragraph is specifically what the phrase *transformational change* denotes in this article. In terms of achieving relief from suffering, such transformational change, resulting from erasure, is the most effective therapeutic outcome.

Erasure is technically not the only behavioral marker of reconsolidation. A statistically significant, permanent change of any kind in an acquired response or memory is also a reconsolidation marker that has been used in some studies, for example those of Hupbach et al. (2007, 2009) and Forcato et al. (2010), in which the destabilized target learning incorporated new learning that either modified or partially interfered with expression of, but did not fully erase, responses driven by the original learning. The carefully controlled conditions, quantitative measurements and statistical power of laboratory studies render such partial interference effects conclusive, but in the uncontrolled complexity of clinical work, partial diminishment of symptoms in individual cases would not be a decisive verification that reconsolidation had occurred. Erasure is total 100 percent interference. Its markers, defined above, are unambiguous in individual therapy cases and therefore easy to confirm, as the author has personally observed in many hundreds of cases. Erasure also rules out all mechanisms of change other than reconsolidation, according to present scientific knowledge, which partial symptom relief does not do. For those reasons, erasure is the only reliable form of verification of reconsolidation in therapy. Achieving transformational change in therapy is unmistakable and, of course, is a therapeutic breakthrough that makes both clients and therapists very happy.

However, the emotional learnings addressed in psychotherapy are in most cases considerably more complex than the elementary emotional learnings created in laboratories for controlled study of destabilization and erasure. Do the above markers of erasure verify reconsolidation in psychotherapy as reliably as in laboratory studies? In other words, when the markers of erasure defined above are observed to appear in immediate response to some steps of psychotherapeutic treatment, is it valid to infer that the therapeutic process has successfully induced memory reconsolidation and erasure? Would it be scientifically valid for therapists to use the markers of erasure as a reliable means of confirming recruitment of the memory reconsolidation process in therapy sessions? If so, close study of such sessions could help reveal and define therapist actions that are effective for facilitating the destabilization and unlearning that nullify and erase clients' emotional learnings.

Given the difference between laboratory and clinical contexts as regards the complexity of target learnings, it seems prudent to allow for the possibility that the markers of erasure observed in therapy might conceivably be the result of some process other than memory reconsolidation. Beyond such *a priori* conservative prudence, however, there is nothing in the existing body of memory research that is recognized as indicating either that some other mechanism of erasure is involved or that acquired emotional schemas of clinical relevance are not susceptible to destabilization and nullification. Rather, one is faced with the fact that reconsolidation has been found to occur for all of the many types of memory that have been tested. Noting that fact, Schiller and Phelps (2011, p. 6) summarized, "These findings suggest that reconsolidation is a general property of memory and is common to different memory systems." It therefore seems not imprudent to apply the same logic to therapy sessions as to laboratory studies, while still respecting the possibility that some qualitatively different mechanism of erasure could eventually be discovered. Thus it appears valid to proceed according to the working hypothesis that the markers of erasure observed in therapy signal the preceding occurrence of memory destabilization and nullification.

The ecological validity and internal consistency of that approach are supported by clinical observations made prior to the discovery of memory reconsolida-

tion. In a systematic search for the therapeutic specific factors directly responsible for sudden, lasting, transformational changes occasionally observed in their therapy sessions, Ecker and Hulley (1996) selected and closely examined sessions that unambiguously produced the same three markers (cessation of triggerability of a specific, affective, problematic emotional schema and ego-state, cessation of long-term symptoms of behavior, cognition, mood and somatics produced during activation of that schema and ego-state, and effortless persistence of those changes). Their scrutiny and reverse engineering of therapeutic process were encouraged by the local clinical scientist paradigm (Stricker, 2006; Stricker and Trierweiler, 1995), and were likewise recently employed by clinical researchers who proceeded "by pinpointing precisely where in the therapeutic discourse the client's self-narrative shifts and then working backward" (Friedlander et al., 2016). Ecker and Hulley in that way identified a specific sequence of experiences that was always present as the immediate precursor of the markers' appearance. The focused, deliberate facilitation of that sequence was then pursued with a wide range of clients and symptoms, resulting in observations of transformational change (the three markers) with unprecedented frequency in day-to-day clinical practice, for a major increase of therapeutic effectiveness.

The same critical sequence of experiences as identified by Ecker and Hulley (1996) was subsequently identified in memory reconsolidation research as the experiences required by the brain for destabilizing a target emotional learning's neural encoding (Pedreira et al., 2004, plus numerous confirming studies, as discussed in Section 4) and then behaviorally updating the destabilized target learning with nullifying counter-learning (first demonstrated by Monfils et al. (2009) and Schiller et al. (2010), as discussed in Section 5). That confirmation of the clinically identified sequence of experiences by rigorous empirical studies using radically different methodology indicates the robustness with which the markers of erasure serve to reveal in psychotherapy that the sequence of experiences necessary to induce destabilization and behavioral nullification has occurred. Ecker and Hulley (1996, 2017) developed a system of psychotherapy designed for maximally efficient facilitation of the critical sequence (initially named Depth-Oriented Brief Therapy, later renamed Coherence Therapy).

The hypothesis that reconsolidation has been induced in therapy sessions that produce the three markers of erasure, advanced by Ecker (2006, 2015b) and Ecker et al. (2012, pp. 126–130), has received support from fine-grained examination of nine clinical cases yielding transformational change, each from a different psychotherapy system (for a listing of which, with citations, see http://bit.ly/15Z00HQ). All nine cases were found to contain the same sequence of experiences identified in reconsolidation research as being necessary for inducing destabilization and erasure, embedded but unambiguously recognizable in the therapeutic process, where they were immediate precursors of transformational change, that is, the appearance of the markers of erasure. The hypothesis that reconsolidation is a deep structure universally responsible for transformational change of acquired states and responses, advanced by Ecker (2011) and Ecker et al. (2013b), has significant implications for psychotherapy integration, which is discussed further in Section 8.2.

Elsey and Kindt (2017a), in discussing therapeutic use of the pharmacological blockade, have suggested verification of reconsolidation via observations of effects that result only from pharmacological blockade of a Pavlovian-type target learning, that is, cue-induced expectation of suffering or pleasure. With one exception, their verification criteria are not relevant to the behavioral updating and erasure process that is the principal focus of this article. The exception is "memory specificity (the manipulation should not indiscriminately affect memory, but only the reactivated memory trace)" (p. 114). That criterion could be fulfilled by a wide range of processes other than reconsolidation, unlike the markers of erasure, which are unique to reconsolidation and appear to be its most reliable verification until such time as neuroscientists devise a method of direct detection of engram nullification that is safe and practical.

4. How destabilization of memory occurs

The destabilization of a target learning's neural encoding opens the so-called reconsolidation window, a period of several hours of lability that allows memory content and/or strength to be altered fundamentally, and that ends naturally with an automatic restabilization (reconsolidation). The experiences that induce destabilization were first identified by Pedreira et al.

(2004), who used animal subjects and created a conditioned fear learning in a standard manner, by pairing an aversive unconditioned stimulus (US, in this study the visual image of a predator) with a conditioned stimulus (CS, in this study a unique context, i.e., a particular chamber). To reveal whether destabilization of that target emotional learning had occurred under various conditions, they used the pharmacological blockade technique: only if the target learning has been destabilized does the administered chemical agent erase the conditioned fear behavior in response to CS presentation. (A newly destabilized target learning continues to function as before, is not degraded by being destabilized, and gives no indication of being destabilized, hence the need for an additional process that reveals destabilization decisively.)

Previous researchers had concluded that destabilization results from memory reactivation and that a memory destabilizes every time it is reactivated (e.g., Nader et al., 2000; Eisenberg et al., 2003). Pedreira et al. (2004) found rather that destabilization did not occur after the target learning had been reactivated by the CS (exposure to the context), but did occur after reactivation was followed by an experience of "memory mismatch" in which the subject's perceptions of the present situation differed from what the reactivated learning expected (in this case, non-appearance of the expected US after being placed in the CS context).

The conclusion reached by Pedreira et al. (2004) was that destabilization requires a sequence of two experiences, memory reactivation plus memory mismatch. This finding subsequently has received independent confirmation by at least twenty-five studies, listed in Table 1. Many other studies have also reported corroborative results, such as that of Piñeyro et al. (2014), whose study focused particularly on determining the role of memory destabilization and who arrived at the view that "equating mere reactivation to memory destabilization could lead to erroneous conclusions" (p. 52).

The critical memory mismatch experience is also referred to as a *prediction error* experience in many of these studies. The two phrases are synonymous. The critical role of mismatch/prediction error for triggering destabilization has been recognized in numerous research review articles. For example, Delorenzi et al. (2014) observed, "strong evidence supports the view that reconsolidation depends on detecting mismatches between actual and expected experiences" (p. 309). Agren (2014), in reviewing research on reconsolidation of emotional learnings in humans, commented, "it would appear that prediction error is vital for a reactivation of memory to trigger a reconsolidation process" (p. 73) and "the studies that have shown effects of reconsolidation… must somehow have induced a prediction error" (p. 80).

Table 1. Studies demonstrating that memory destabilization requires a memory mismatch or prediction error experience in addition to memory reactivation.

Year, Authors	Species, Memory Type	Design and Findings
2004, Pedreira et al.	Crab: Contextual fear memory	Learned fear response can be erased by chemical blockade (bicuculline and cycloheximide) only after memory reactivation is accompanied by memory mismatch experience (prediction error).
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	Reactivated memory is erased by new learning only if a novel contingency is also experienced.
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 destabilized and lost only if, after reactivation, the expected opportunity to match syllables does not occur, creating mismatch.
2009, Perez-Cuesta & Maldonado	Crab: Contextual fear memory	Reactivated learned expectation of visual threat must be sharply disconfirmed for memory to be erased by cycloheximide.
2009, Winters et al.	Rat: Object recognition memory	Memory is erased 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.
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 erased 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 and erased 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 disrupted and erased by propranolol only if prediction-error-driven relearning is also experienced.

2015, Alfei et al.	Rat: Contextual fear memory	Reactivated fear memory is disrupted and erased by midazolam only if reactivation conditions involve prediction error (a temporal prediction error in this study).
2015, Jarome et al.	Rat: Contextual fear memory	Reactivated fear memory is disrupted and erased by a protein synthesis blocker only if reactivation conditions include a novel contextual feature (mismatch/prediction error).
2016, Forcato et al.	Human: Declarative memory	Recall of memorized items is impaired, revealing destabilization, only when a memory mismatch (prediction error) accompanies reactivation.
2016, López et al.	Crab: Contextual fear memory	Learned fear response can be erased by chemical blockade (bicuculline and cycloheximide) only after memory reactivation is accompanied by prediction error.

Exton-McGuinness et al. (2015) reviewed the role of prediction errors in reconsolidation studies and summarized their position by stating, "We propose that a prediction error signal…is necessary for destabilisation and subsequent reconsolidation of a memory" (p. 375). Krawczyk et al. (2017), reviewing the functional role of prediction error in the neurobiology of learning and memory, stated, "Prediction error induces updating of consolidated memories in strength or content by memory reconsolidation" (p. 13) and "When our predictions or understandings of the world do not fit with the current experience, the detection of this incongruence triggers the destabilization-reconsolidation process, which allows us to adjust our internal models." (p. 15).

The experience-driven nature of memory reconsolidation is strongly apparent in the mismatch requirement. An experience of mismatch, or prediction error, inherently involves the perceived presence of a novelty or discrepancy relative to expectancy in a previously learned milieu, creating a subjective element of surprise (see, for example, Fernández et al., 2016c; Lee, 2009; Sevenster et al. 2014). What matters to the subject's brain is not the concrete procedure used by experimenters to set up mismatch in a particular study, but rather the subjective experience created by the procedure, an experience in which the world is in some way not as believed and expected. Behavioral protocols for creating mismatch vary greatly across studies. For example, Pedreira et al. (2004) used a procedure in which mismatch consisted of non-reinforcement that followed reactivation by a variable time delay, making it clear that the two are distinct experiences, whereas Rossato et al. (2007) used the presence of a novel object to create a mismatch that was copresent with reactivation.

Neuroscientists regard reconsolidation as being the brain's innate process for updating memories because it launches only if an experience of discrepancy and surprise accompanies reactivation of an existing learning or schema. Various studies have contributed to a growing understanding of the boundary conditions of memory destabilization, i.e., the types and degrees of mismatch that do or do not trigger memory destabilization for memories of various types, ages, or strengths (e.g., Gallucio, 2005; Suzuki et al, 2004; Sevenster et al., 2013, 2014; Schroyens et al., 2017). Importantly for both research and clinical application, reliable design of mismatch experiences depends heavily upon knowledge of the detailed content and structure of the target learning, because that content and structure determine which experiences register as mismatch/prediction error and with what strength. That fundamental principle has been revealed by several studies in which target learnings containing knowledge of various reinforcement schedules or timing patterns were tested for destabilization by candidate mismatch experiences of various designs (e.g., Alfei et al., 2015; Jarome et al., 2012; López et al., 2016; Merlo et al., 2014; Sevenster et al., 2013, 2014; Schroyens et al., 2017). The same studies have also shown that if an intended mismatch experience differs too greatly from the target learning's expectations, it does not induce destabilization, presumably because a too-different experience registers not as a mismatch, but as being in a qualitatively different context or category of experience from that of the target learning, rather than a needed correction to the target learning. The threshold of excessive mismatch itself depends on memory strength and age, because stronger degrees of reactivation and mismatch are required to destabilize stronger and older memories (Eisenberg and Dudai, 2004; Frankland et al., 2006; Su-

zuki et al., 2004; Winters et al. 2009; Wang et al., 2009).

Several studies have shown additionally that by adding a small continuation (such as two more CS-only presentations) at the end of a post-reactivation protocol previously shown to leave the target learning in a destabilized condition, the target learning is then left in a stable condition by the extended protocol (Jarome et al., 2012; Merlo et al., 2014; Sevenster et al., 2013, 2014). This finding implies that a target learning is dynamically switched in real time from stable to unstable or vice versa as the component steps of a post-reactivation protocol are progressively implemented. Ecker (2015a, pp. 19–23) has proposed that such switching occurs because each part of the post-reactivation protocol promptly creates new learning that influences the level of prediction error created by the next part of the protocol, and in that sense functions as (and possibly may actually be) a modification of the target learning. In Ecker's hypothesis, if at any point the new learning created by the unfolding protocol causes the next part of the protocol to be experienced with little or no prediction error, the target learning is switched from destabilized to stable condition, abruptly closing the reconsolidation window and preventing the remainder of the protocol from having any updating effect (unless it is structured so as to mismatch the now-revised target learning, which would again destabilize the target learning). Ecker (2015a) applied that hypothesized phenomenology to generate for the first time an analysis of the time-resolved effects of the protocols used by Monfils et al (2009) and Schiller et al. (2010), as well as a time-resolved analysis of how the standard extinction protocol operates (that is, an analysis of the evolving state of the target learning and the effect of each successive non-reinforcement trial).

Reviewing experimental findings noted in the previous two paragraphs, Ecker (2015a, p. 13) defined a formal principle of *mismatch relativity*, a clarified formulation of which is: Whether a particular component of a post-reactivation procedure creates a destabilizing mismatch experience depends entirely on the model of reality at that point in time in the target learning, including modifications or supplementations by any prior components of the post-reactivation procedure.

Thus, while the various specialized protocols described above were designed for the highly simplified conditions of controlled studies and therefore may have limited clinical applicability, collectively they serve to identify a general principle that is critically important for clinical application: An experience that is intended to destabilize a particular target learning must be accurately tailored to the specific content of that target learning.

For example, for a target learning acquired through an intermittent reinforcement training, a single experience of nonreinforcement does not create an experience of mismatch or counter-learning, because the expectation maintained by the target learning includes the occurrence of nonreinforcements (e.g., Sevenster et al. 2013, 2014). Even two or more nonreinforcement experiences would fail to create a mismatch if the original, learned pattern of nonreinforcement was similarly sparse. That example is a rather obvious case, but in some studies the target learnings created by researchers had subtler features that are not addressed or accounted for in researchers' interpretation of observations. For example, Pine et al. (2014) achieved destabilization but regarded their experimental procedure as creating no prediction error, so they viewed their results as indicating that prediction error is not necessary for achieving destabilization, when actually their procedure generated prediction error in three different ways (for details of which, see Ecker, 2015a, p. 12).

Cognizance of the mismatch requirement did not spread efficiently among reconsolidation researchers after its discovery by Pedreira et al. (2004). The prior, incorrect notion that each reactivation by itself is destabilizing continued to be asserted in journal articles by many reconsolidation researchers, as well as in science journalism. This lack of recognition of the mismatch requirement has caused the authors of numerous studies to misinterpret their results, particularly in studies that reported a failure to achieve destabilization and erasure. The negative result is attributable, as a rule, to an absence of any mismatch experience in the experimental procedure (see Ecker, 2015a, for discussion of such cases). In most instances, these studies' authors made no mention of the mismatch requirement and appeared to be unaware of it (e.g., Cammarota et al., 2004; Hernandez and Kelley, 2004; Mileusnic et al., 2005; Wood et al., 2015). An exception is the discussion by Bos et al. (2014), who surmised that their negative result was due to the absence of a mismatch/prediction error experience and commented, "Future studies may benefit from protocols that are explicitly designed to assess and manipulate prediction

error during memory retrieval" (p. 7).

Given that clinical symptoms are frequently maintained by generalized, semantic emotional learnings or schemas, as discussed in Section 2, a critical question is whether a schema can be reactivated by generalized, abstract cues, with no reference to any specific experience that contributed to the formation of the schema, and then be destabilized and erased. Soeter and Kindt (2015b) demonstrated that after the images of two distinctive spiders were separately paired with electric shocks and became fear-inducing, seeing the name of the category of the feared items, "spider," was effective for reactivating the subcortical fear memory, allowing destabilization and erasure to follow. This finding begins to provide an empirical basis for the frequent clinical observation that a client's emotional schema is readily reactivated by verbally naming its constituent categories, without any reference to the original perceptions or experiences that led to the formation of the schema.

In another common clinical situation, the expectation of a particular form of suffering (US) is triggered by numerous different occurrences or perceptions (CSs), such as a feeling and expectation of social rejection (the US) being evoked by holidays, parties, dancing, restaurants and weekends (the CSs). The clinical ideal would be a single process that dissolves all such CS linkages, rather than addressing each separately. Several laboratory studies have simulated this situation by pairing a US with two or more CSs (Debiec et al., 2010; Díaz-Mataix et al., 2011; Doyére et al., 2007; Liu et al., 2014; Schiller et al., 2010; Soeter and Kindt, 2011). All of those studies found that when reactivation was induced by the presentation of one of the CSs alone (without the expected US occurring, creating mismatch), followed by post-reactivation pharmacological blockade or behavioral counter-learning, the conditioned response to only that one CS was then erased, and all other CSs continued to trigger the state of expecting the US. In contrast, when reactivation was induced by re-experiencing only the US (without any of the expected prior CS presentations, creating multiple mismatches), the conditioned responses to all CSs were then erased or impaired (Debiec et al., 2010; Díaz-Mataix et al., 2011; Liu et al., 2014; Luo et al., 2015). Further, one of those CSs, which had also been paired with a different US, continued to evoke the distinct conditioned response created by that pairing (Liu et al., 2014), showing the specificity of the destabilizations induced via US reactivation. Summarizing a review of those findings, Dunbar and Taylor (2017, p. 168) comment, "Whereas the conventional CS-reactivation procedure may recruit the neural correlates of only a single CS-US memory, US reactivation may recruit neural correlates of all CSs associated with the reactivated US, thus allowing for destabilization and, thus, disruption of all CS-US associations for the reactivated US." This indicates that in optimal clinical translation, reactivation of the target learning would include a reminder of the US, that is, the specific suffering that the client experienced in the past and learned to anticipate and strive to avoid. Incorporation of that US-reminder strategy into clinical methodology is described in Section 6.3 below.

5. How erasure of memory occurs

When a target learning is in the destabilized, labile condition, its nullification, resulting in the markers of erasure, can be accomplished in two fundamentally different ways, one of which is endogenous and the other, exogenous (for reviews, see Agren, 2014; Lee et al., 2017; Reichelt and Lee, 2013). The endogenous method is often labeled behavioral memory updating or behavioral interference. The exogenous method is known as pharmacological blockade, pharmacological interference, or disruption of reconsolidation.

In behavioral updating, first demonstrated in animal studies by Sekiguchi et al. (1997) and in human studies by Walker et al. (2003) and Galluccio (2005), a destabilized learning can be revised in its strength and/or content by suitably designed new learning, according to how the new learning differs from the target learning (without differing so much that, due to lack of relevance to the target learning, the new learning forms separately rather than updating the target learning, leaving the target learning unchanged). A target learning can thereby be strengthened, weakened, modified in its particulars, or erased by suitably designed new learning during the reconsolidation window (for reviews see Agren, 2014; Beckers and Kindt, 2017; Reichfelt and Lee, 2013; Schiller and Phelps, 2011). The updating/erasure effect occurs through different molecular and cellular processes from the destabilization/restabilization process (Jarome et al., 2012; Lee et al., 2008)

Because this article's primary topic is the psychotherapeutic use of memory reconsolidation, the focus here is mainly on the erasure of target learnings, as it is erasure that is experienced by therapy clients as decisive, transformational change, that is, complete and permanent disappearance of an unwanted behavior and/or state of mind and/or somatic disturbance. Erasure via behavioral memory updating results from following the destabilization of the target learning with counter-learning consisting of experiences that contradict and disconfirm the target learning's specific model and expectation of the behavior and qualities of self, others and/or the world. The markers of erasure are then observed, which is the basis for researchers concluding that counter-learning during the reconsolidation window drives unlearning that nullifies and replaces the labile target learning (e.g., Clem and Schiller, 2016). Erasure produced in this way can be regarded as behavioral memory interference (Bjork, 1992; Robertson, 2012) at the maximum possible degree of effectiveness.

The historic first demonstrations of counter-learning erasing an emotional learning (conditioned fear) were the animal study by Monfils et al. (2009) and the human study by Schiller et al. (2010). In those studies, fear was acquired through Pavlovian associative conditioning, in which an initially emotionally neutral CS was repeatedly paired with an electric shock US. Thereafter, the CS by itself triggered a fear response due to the learned expectation of the US occurring next. The procedure employed by Monfils et al. and Schiller et al. for erasing that acquired fear response behaviorally with counter-learning was a protocol that has come to be known as post-retrieval extinction, retrieval-extinction, or reactivation-extinction. Erasure by this protocol has been confirmed in several animal studies (e.g., Clem and Huganir, 2010; Flavell et al., 2011; Piñeyro et al., 2014; Rao-Ruiz et al., 2011) and human studies (e.g., Agren et al., 2012; Björkstrand et al., 2015; Oyarzún et al., 2012; Steinfurth et al., 2014). Negative results from the post-retrieval extinction protocol were obtained in several other studies (with animal subjects: Chan et al., 2010; Costanzi et al., 2011; Flavell et al., 2011; Ishii et al., 2012, 2015; Stafford et al., 2013; and with human subjects: Golkar et al., 2012; Kindt and Soeter, 2013; Meir Drexler et al., 2014; Soeter and Kindt, 2011). These negative studies have been interpreted by Auber et al. (2013) and Piñeyro et al. (2014) as indicating that some of the protocol's several adjustable parameters were set outside the brain's reconsolidation boundary conditions, the range of parameters of a given protocol that induce destabilization or counter-learning. That explanation appears to have been confirmed by a systematic varying of parameters by Ferrer Monti et al. (2017). However, the matter is not yet fully resolved, because Luyten and Beckers (2017) attempted an exact replication of the study by Monfils et al. (2009) and did not observe erasure.

The post-retrieval extinction protocol begins with reactivation of the target learning by a single unreinforced CS presentation, followed by a 10-min time interval, which is followed by a series of unreinforced CS presentations. That series of CS-noUS trials is identical to the conventional protocol used for studying extinction for a century, so researchers refer to that part of the protocol as an extinction training (which is arguably a misnomer, as discussed further in Section 6.2 below). Thus in its entirety (retrieval plus extinction), the post-retrieval extinction protocol can be understood as being a standard extinction training with a single modification that may seem minor but has major effects: an increased time interval of 10 min between the first two CS presentations. Monfils et al. and Schiller et al. compared the results obtained with and without the increased time interval: Without it, the protocol becomes that of standard extinction training, and the result was the familiar one known from a century of extinction studies (Bouton, 2004), namely, initially the fear response was largely suppressed but then could be reinstated. In extinction, repetitive counter-learning applied to a stable target learning creates a separate contradictory learning that competes against the target learning and temporarily suppresses it. (See Ecker 2015a for a detailed analysis of why standard extinction training does not destabilize and erase the target learning. Strong evidence that reconsolidation and extinction are distinct and mutually exclusive phenomena has been produced by Duvarci and Nader, 2004; Duvarci et al., 2006; Merlo et al., 2014.) Suppression induced by extinction training is prone to relapse because the target learning still exists and again becomes reactivated and dominant under various circumstances (Neumann and Kitlertsirivatana, 2010; Vervliet et al., 2013).

In sharp contrast, with the first time interval increased to 10 min, the fear response was erased, that is, it disappeared and could not be reinstated, and the du-

rability of non-reinstatement was confirmed after 1 yr by Schiller et al. (2010) and after 1.5 yr by Björkstrand et al. (2015). Evidently the increase of the first time interval to 10 min created a mismatch experience that destabilized the target learning, allowing the following series of non-reinforced CS presentations to serve not as a conventional extinction training, but rather as counter-learning that accomplished erasure.

Understanding how and why the increased time interval served to create a memory mismatch (prediction error) experience is vitally important for reliable, successful future utilization of this procedure and its variants in both research and psychotherapy. However, neither Monfils et al. (2009) nor Schiller et al. (2010) made reference to the mismatch requirement in their discussions of results. Ecker (2015a) has proposed a detailed analysis of the mismatch characteristics of both studies, and López et al. (2016) have replicated the experimental result and interpreted it in terms of prediction error. Both of those accounts join with those of Fernández et al. (2016a) and Hupbach (2011) in emphasizing a central principle: In laboratory studies, the effect of new learning on a target learning is accurately understood only by examining in detail the relationship between the acquisition experiences that originally were encoded into the target learning and the structure and content of the new post-reactivation learning.

That principle applies as follows to the studies by Monfils et al. (2009) and Schiller et al. (2010): In acquiring the target learning, subjects learned not only the CS-US association but also the time interval between CS-US pairings: 3 minutes in Monfils et al. and 15 seconds in Schiller et al. Therefore, upon perceiving the first CS of the post-retrieval extinction procedure, subjects in the two studies expected the next CS to appear after those time intervals, respectively. That expectation was then mismatched by the passage of 10 minutes before the next CS appeared, instead of the expected 3 minutes or 15 seconds. (Studies have since demonstrated the use of a purely temporal mismatch to induce memory destabilization, e.g., Alfei et al. 2015; Díaz-Mataix et al., 2013.) Schiller et al. in addition had subjects view a TV show during the 10-minute interval, which was a qualitatively different type of novelty relative to the target learning's expectation of a blank screen, possibly creating a second mismatch concurrent with the temporal one.

The increased, 10-minute interval is clearly the distinctive feature responsible for the post-retrieval extinction procedure's effectiveness in the Monfils et al. study, so it has been copied in nearly all subsequent replications and variants. Yet the analysis in the preceding paragraph implies that the increased time interval was effective only because it happened to mismatch the time structure in the acquisition training, not because of any intrinsic role of delays or timing in the destabilization process. In other words, with a target learning that has no timing structure, the extended 10-min time interval of the post-retrieval extinction procedure would not create mismatch or contribute to destabilization. There is no timing structure in any of the emotional learnings of therapy clients that were detailed earlier in Section 2, such as the middle-aged man's learned, generalized expectation that anyone would respond to him with anger or cold indifference were he to express some need or distress. With target learnings such as those, which are representative of most of the emotional learnings that emerge in therapy, the timing structure of the post-retrieval extinction procedure could not contribute to creation of mismatch/prediction error and therefore could not induce destabilization.

Thus the analysis above suggests that the post-retrieval extinction procedure of Monfils et al. (2009) and Schiller et al. (2010) would have limited clinical applicability. It will be argued in Section 6.1 that the most versatile and optimal procedural format for clinical application consists of using each therapy client's unique target learning as the absolute basis for tailoring effective experiences of reactivation, mismatch, and counter-learning, unconstrained by any preconceived procedural format and utilizing any known therapeutic techniques.

The foregoing analysis serves to illustrate why the superordinate principle put forward in this article is that an experience-oriented interpretation of reconsolidation research rather than a procedure-oriented interpretation is necessary for arriving at accurate understanding and reliable, fine-grained control of memory reconsolidation in clinical use. Adherence to the experience-oriented interpretation consists of analyzing occurrences and non-occurrences of memory reconsolidation in terms of the experiences required by the brain for inducing destabilization and revision of a target learning. The experience-oriented interpreta-

tion therefore illuminates why, and when, a particular procedure is or is not effective. The procedure-oriented interpretation in itself cannot do that because, in effect, it equates memory reconsolidation with certain procedures rather than with the critical experiences that actually govern the process.

For example, the experience-oriented interpretation may provide a simple explanation for what is currently perhaps the most controversial finding in reconsolidation research. Both Baker et al. (2013) and Millan et al. (2013) carried out the post-retrieval extinction protocol (targeting conditioned fear memory in adolescent rats and alcoholic beer seeking memory in adult rats, respectively) and observed significantly diminished expression of the target learning. They also found that the same diminishment of memory expression resulted from reversing the protocol, with the single CS-only "retrieval" component *following* the extinction training by 10 min rather than preceding it. That has widely been viewed as a "remarkable" finding indicating potentially that the post-retrieval extinction protocol does not engage reconsolidation mechanisms after all, because "Reconsolidation theory would posit that retrieval must come before extinction for the procedure to impair reinstatement" (Dunbar and Taylor, 2017, p. 163) and "it is difficult to reconcile how reconsolidation can be initiated when the reminder trial follows, rather than precedes, extinction learning" (Treanor et al., 2017, p. 293) (see also Hutton-Bedbrook and McNally, 2013).

Those are procedure-oriented analyses. The experience-oriented interpretation provides an alternative analysis that is consistent with the reconsolidation process, as follows. In this view, the critical condition that accomplishes behavioral erasure via reconsolidation is the concurrent experiencing of a reactivated target learning that is destabilized, and a counter-learning that contradicts the target learning's model of how the world works. Importantly, it does not matter which of those two experiences is induced first, because what the brain requires for updating, in this view, is the juxtaposition of the two. As soon as both are being experienced concurrently, erasure is a likely result. The idea that counter-learning could precede destabilization and still be effective for erasure ceases to seem counter-intuitive with recognition that for updating, the brain requires only a juxtaposition (concurrence, simultaneity) of the two experiences, regardless of which of the two is activated first. Each of those two experiences has an extended though limited duration of activation, which is why their juxtaposition is possible.

For example, such juxtaposition could have occurred as follows in the study by Baker et al. (2013). Acquisition training consisted of three pairings of a 10-sec white noise sound (CS) with paw shock (US) on day 1. The procedure on day 2 (in Experiment 4) began with an extinction training in which 30 CS-only presentations were separated by an interval of 10 sec. That constitutes strong learning of the expectation that a CS is followed by another CS in 10 sec. That expectation was mismatched when, 10 min after the extinction training ended, only the single "retrieval" CS presentation occurred, with no other CSs following it. That mismatch of CS-driven expectation destabilized the target learning. At that point, the destabilized target learning and the just-created counter-learning experience of safe CS were simultaneously present, so behavioral updating of the target learning occurred and a significant decrease of fear response was observed. Also tested was a period of 6 hr instead of 10 min after extinction until the single retrieval trial. In this case, the enhanced impairment effect on the fear response was lost. Since the target learning was destabilized by the single CS-only presentation no differently after 6 hr as compared to after 10 min, it must be the other ingredient needed for erasure that was absent: After 6 hr, the extinction training experience of safe CS was no longer a currently activated experience, so there was no juxtaposition of the destabilized target learning with a counter-learning experience and therefore no updating. A separate finding that strongly supports this interpretation is the recent demonstration that the neural ensemble encoding a new learning interacts with and participates in a subsequent new learning for 5 hr but does not do so after 6 hr (Cai et al., 2016; Rashid et al., 2016). Thus in the 6-hr case, the 30 CS-only trials functioned as a standard extinction training, not as counter-learning for memory updating, so the enhanced impairment of the fear response was lost.

The observations of Baker et al. (2013) and Millan et al. (2013) can therefore be seen as supporting the experience-oriented interpretation of reconsolidation phenomena rather than as indicating non-recruitment of reconsolidation by the post-retrieval extinction

protocol. In clinical work, likewise we find that the required juxtaposition can be assembled either way, by reactivating the target learning first (for example, "The only way to get attention is to do something bad") or by reactivating the contrary knowing first (for example, "My boss readily met with me to discuss my concerns, and my friends all showed up for my birthday party"). Both sequences are equally effective for setting up the juxtaposition that results in erasure.

Erasure by counter-learning during the reconsolidation window is the fully endogenous utilization of memory reconsolidation. Alternatively, administration of a chemical agent during memory destabilization can produce exogenous erasure with no counter-learning. In that methodology, the agent blocks the neural protein synthesis necessary for return of labile memory to the stable consolidated state, but has no effect on stable memory circuits. When fully effective, this pharmacological blockade prevents restabilization (reconsolidation) of the destabilized neural circuitry of the target learning from ever taking place. The target learning's neural encoding becomes nonfunctional and the markers of erasure are observed. The pharmacological agent is administered soon before or soon after the target memory is destabilized by reactivation with mismatch, and the blockade takes effect when restabilization would normally occur, about five hours after destabilization was induced. The pharmacological blockade procedure is therefore also referred to by researchers as disruption of reconsolidation. (For a review see, e.g., Taylor and Torregrossa, 2015.) Erasure via pharmacological blockade was first demonstrated for learned fear by Nader et al. (2000) and Przybyslawski et al. (1999) in animals and by Kindt et al. (2009) in humans.

Human studies and clinical applications of pharmacological blockade have relied on the use of propranolol, as all other blockade agents used in animal studies have toxicity that precludes them from use with people. Both positive and negative results have been reported in numerous studies; for reviews see Beckers and Kindt (2017) and Steenen et al. (2017). The latter concluded:

> These meta-analyses found no statistically significant differences between the efficacy of propranolol and benzodiazepines regarding the short-term treatment of panic disorder with or without agoraphobia. Also, no evidence was found for effects of propranolol on PTSD symptom severity through inhibition of memory reconsolidation. In conclusion, the quality of evidence for the efficacy of propranolol at present is insufficient to support the routine use of propranolol in the treatment of any of the anxiety disorders.

However, an examination by Ecker (2015, pp. 14–15) of some pharmacological blockade studies has revealed a significant procedural flaw: the absence of any mismatch experience following reactivation of the target memory. The lag in recognition of the mismatch requirement by many researchers was noted earlier, and in a review of propranolol studies in humans, Beckers and Kindt (2017, p. 111) commented, "The notion that memory destabilization will occur only when there is an expectancy violation or an experienced mismatch at the time of memory retrieval…has not been taken into account in the majority of clinical trials that have been conducted so far." In studies with no mismatch experience, there would be no destabilization of the target memory and therefore no pharmacological blockade effect, so the target memory would be unaffected and remain in operation. That would be the reason for the observed failure of the chemical agent to produce erasure in these studies (which in their own way add usefully to the evidence supporting the necessity of mismatch for inducing destabilization). These flawed studies therefore have no significance regarding the inherent effectiveness of the exogenous/pharmacological approach, and should not be included in evaluations of that approach. The inclusion of such methodologically flawed studies in the meta-analysis by Steenen et al. (2017) could be responsible for arriving at a negative conclusion regarding the clinical effectiveness of propranolol for disrupting the reconsolidation of humans' fear learnings. The true clinical value of propranolol treatment therefore remains ambiguous, and it is possible that by using methodology that reliably fulfills the brain's requirement for memory destabilization via reactivation and suitable mismatch, the effectiveness of propranolol treatment might be established. If Steenen et al. were to repeat their calculations after screening out the methodologically flawed studies, the new results could be a significantly more reliable indicator of propranolol's clinical effectiveness if sufficient statistical power remains.

In contrast to erasure via pharmacological blockade, the fully endogenous process of erasure through counter-learning (i.e., behavioral memory updating) allows completion of the reconsolidation of the target learning's neural encoding in its re-encoded, content-revised form. Behavioral erasure is a disruption of the *content* of the target learning, accomplished *through* the reconsolidation process, not the neurophysiological disruption of the reconsolidation *process* itself.

For psychotherapeutic use, the endogenous (fully psychological) and exogenous (pharmacological blockade) approaches have their respective advantages and disadvantages. It is widely recognized, as Soeter and Kindt (2011, p. 358) stated, that "Obviously, a behavioral procedure will be preferred over pharmacological manipulations provided that similar effects can be obtained." Behavioral updating in fact appears to have greater effectiveness, according to both laboratory studies described in this paragraph and clinical observations described in Section 7. In humans, behavioral erasure of fear memory by the post-retrieval extinction protocol has been shown to occur in the subcortical emotional memory system of the amygdala (through fMRI brain imaging by Agren et al, 2012; Björkstrand et al., 2015; Schiller et al., 2013) and also in the declarative, contingency-learning memory system of the neocortex (through skin conductance measurements by, e.g., Oyarzún et al., 2012; Schiller et al., 2010). Pharmacological blockade of fear memory by propranolol, in contrast, erases subcortical fear memory but leaves intact the fear generated by declarative memory of the CS-US contingency (as detected in the form of undiminished skin conductance and US-expectancy measurements by Kindt and Soeter, 2013; Soeter and Kindt, 2011; reviewed by Beckers and Kindt, 2017). Further, it has been shown that after pharmacological blockade and erasure of a newly encoded memory, some components of the engram (physical encoding) continue to exist (Ryan et al., 2015), allowing optogenetic (artificial) activation of participating neurons to drive re-expression of the erased behavioral response. The same test of engram persistence for a behaviorally erased memory has not yet been reported, to the author's knowledge. Neurochemical evidence suggests that the neural encoding of the target learning is reconstituted when behaviorally updated by new learning (Clem and Huganir, 2010; Debiec et al., 2010; Díaz-Mataix et al., 2011; Jarome et al., 2012, 2015), but whether the entire engram is thus reconstituted is not yet known. Implications of post-erasure engram persistence for the durability of changes produced by erasure in psychotherapy are discussed below in Section 7.5.3.

Table 2. Comparison of features of the endogenous and exogenous clinical use of memory reconsolidation, based on the totality of controlled studies and clinical observations to date.

Feature	Endogenous/psychological	Exogenous/pharmacological
Range of symptoms dispelled to date	Wide[1]	Narrow[2]
Effectiveness in clinical practice to date	High[3]	Uneven
Memory systems affected in lab studies	Subcortical + cortical	Subcortical
Resolves varied and complex emotional issues	Yes[4]	Not demonstrated
Duration of treatment	Unpredictable; often brief[5]	Brief
Level of clinical training required	Advanced	Intermediate

[1] See Table 4 of this article.

[2] Fear-based symptoms, specifically phobias, panic and symptoms of post-traumatic stress disorder from single-incident, acute trauma.

[3] In twenty years of clinical use, advanced practitioners observe erasure rates of up to 95% of clients.

[4] For online listing of published case studies indexed by symptom, see http://bit.ly/2tKXdyX, and for numerous additional case studies see http://bit.ly/15Z00HQ .

[5] For advanced practitioners in general practice, the number of therapy sessions needed to dispel a given symptom is usually in the range of 6 to 20, though as few as 2 sessions are sometimes sufficient and, in cases of severe complex attachment trauma, on the order of 100 sessions may be required to dispel numerous symptoms and their numerous underlying emotional schemas.

Table 2 compares the currently known attributes of the endogenous/behavioral and exogenous/pharmacological methods of erasure. The endogenous approach is the focus of the remainder of this article. Sections 6 and 7 below describe a versatile clinical methodology developed and manualized by Ecker and Hulley (2017a) (see also Ecker et al., 2012, 2013a,b) for efficiently and directly implementing the empirically confirmed process of behavioral erasure. That methodology has been utilized by clinicians worldwide, many of whom have reported their own observations of the markers of erasure (see Ecker et al., 2012, pp. 157–200 for four such detailed accounts; also Sibson and Ticic, 2014). Furthermore, as noted in Section 8 below, the methodology has potential for contributing a number of significant conceptual advances to the clinical field, apart from enhancing the effectiveness of individual practitioners.

6. From research findings to clinical methodology

On the basis of the foregoing examination of research, the primary subject of this article can now be addressed: the translation of reconsolidation research into clinical application. The main purpose of this section is to ask and answer this question: *What is the most general clinical methodology of behavioral updating that is directly and entirely dictated and defined by reconsolidation research?* Section 7 then illustrates and examines the actual implementation of that proposed most general clinical methodology; Section 8 identifies the potentially major ramifications of this methodology for several fundamental theoretical issues in the psychotherapy field; and Section 9 uses this methodology as a frame of reference for analyzing several other approaches for clinically recruiting reconsolidation.

6.1. The empirically confirmed process of behavioral erasure

The extensive memory reconsolidation research examined in Sections 3, 4 and 5 may be summarized in essence in this manner: The behavioral erasure of a target emotional learning is an experience-driven process, with the requisite experiences being reactivation, mismatch, and counter-learning. Reactivation and mismatch experiences destabilize the target learning, and then counter-learning experiences disconfirm and nullify the target learning and reconstitute its neural encoding. Verification of erasure then consists of observing the three markers defined and discussed in Section 3: nonreactivation, symptom cessation, and effortless permanence.

The tripartite sequence of reactivation, mismatch, and counter-learning experiences will be referred to henceforth as the *empirically confirmed process of erasure* (ECPE). The ECPE is proposed here as a completely non-theoretical, empirically identified core methodology for directly applying in psychotherapy the research on endogenous memory reconsolidation.

As shown in clinical case examples in Section 7, a therapy client's experience of behavioral erasure is not a merely mechanistic elimination of symptoms. Erasure occurs through a counter-learning experience in which the individual decisively unlearns and thereby profoundly resolves a specific schema or mental model maintaining emotional distress. Such disconfirmation of what had seemed reality frequently occurs in quite noticeable, identifiable moments during the facilitation of a counter-learning experience. Conventional notions of the time needed for major therapeutic effects to develop are challenged by this process of transformational change through the ECPE. This is a fundamentally different process of change from the Hebbian process of building up preferred, competing responses through their extensive repetition over a prolonged period to create and strengthen the alternative, competing neural circuits.

If, as the current state of empirical knowledge suggests, the markers of erasure can appear endogenously only as a result of the ECPE's component experiences, then the occurrence of the three critical experiences usually ought to be detectable in hindsight in any therapy sessions or clinical study in which the unambiguous markers of erasure were observed, regardless of whether the therapy or study was conducted with cognizance of memory reconsolidation or research findings. As previously noted, such ECPE detection was reported by Ecker and Hulley (1996) prior to the discovery of memory reconsolidation, and recently ECPE detection has been demonstrated in the same way for eight different systems of psychotherapy by studying sessions that produced the markers of erasure (Ecker, 2015c; Ecker et al. 2012, pp. 126–155; Feinstein, 2015; Lasser and Greenwald, 2015; Ticic and Kushner, 2015; for a list of the individual therapy systems, see http://bit.ly/15Z00HQ).

6.2. Experiences versus the procedures that induce them

The ECPE was defined above as a sequence of three experiences without reference to any concrete behavioral procedures for creating those constituent experiences. Deliberate clinical implementation of the ECPE of course requires use of concrete behavioral procedures (often termed *interventions* in the standard parlance of psychotherapy). This matter of behavioral procedures is important not only with regard to equipping clinicians for effective implementation of the ECPE, but also with regard to eventual designation of an ECPE-centered clinical methodology as an Empirically Supported Treatment (EST) or Evidence Based Treatment (EBT), which requires a manualized procedure to be tested for efficacy and/or effectiveness in controlled studies.

A broad array of behavioral procedures for carrying out the ECPE in therapy has been developed and is in use by clinicians (Ecker and Hulley, 1996, 2017; Ecker et al., 2012). However, most relevant to the present article is a discussion not of those various concrete procedures, but of the relationship between the ECPE and any concrete procedures for carrying it out.

The concrete behavioral procedures used by reconsolidation researchers for inducing reactivation and mismatch are myriad. The concrete procedures in each study were necessarily tailored to (a) the type of memory under consideration (as noted, many different types of memory have been studied, only a subset of which is listed in Table 1) and (b) the detailed content and structure of the target learning used in the study.

Neurological destabilization of a target learning is triggered by the brain registering subjective experiences of reactivation and mismatch, not by the external concrete arrangements and procedures used to induce those experiences. Likewise, profound unlearning and erasure are the result of an experience of counter-learning, which may be arranged in any concrete manner that is suitable for disconfirming the target learning, and is not restricted to any particular concrete procedure or protocol.

Thus, the empirically confirmed process of erasure does not dictate any particular behavioral procedures for creating the required, subjective experiences of reactivation, mismatch and counter-learning that result in erasure. The distinction between, on the one hand, the subjective experiences required by the brain for destabilization and erasure to occur, and, on the other hand, the concrete procedures used for inducing those experiences, is of fundamental importance in order for memory reconsolidation to be utilized clinically to its fullest potential. For effectively and responsibly utilizing memory reconsolidation in an empirically supported manner, clinicians must facilitate the required sequence of experiences and then verify erasure by testing for and observing its markers. For doing so, however, clinicians need not, and in fact must not, limit themselves to concrete procedures used in laboratory studies, because those procedures were designed to be effective only for the particular design of target learning created in the respective experimental study. In short, the ecological validity of most experimental protocols is too limited for general clinical application.

Nevertheless, there has been much reliance on laboratory protocols for attempting clinical translation, most notably the post-retrieval extinction or reactivation–extinction protocol used by Monfils et al. (2009) with rats and by Schiller et al. (2010) with humans for the first demonstrations of endogenous erasure of a learned fear, as discussed in Section 5. Successful erasure of a symptom having very high clinical relevance motivated numerous subsequent laboratory and clinically oriented studies of this protocol and of variants based upon it (reviewed by Auber et al., 2013; Kredlow et al., 2016; Lee et al., 2017).

The original post-retrieval extinction protocol consists of a series of unreinforced presentations of a conditioned stimulus with an increased time interval of 10 minutes between the first two CSs. With that structure, the procedure is suitable to induce reactivation, mismatch and counter-learning only for a target learning having a special structure, namely a Pavlovian target learning consisting of (a) a CS-cued expectation of a feared or desired event, plus (b) the expectation that CS-US pairings will repeat with the same timing as originally experienced in the acquisition training (a time interval of significantly less than 10 minutes). Most target learnings encountered in real-life clinical cases have a very different composition, such as the five clients' emotional learnings noted in Section 2, and as shown in the case vignettes in Section 7. Therefore the original post-reactivation extinction protocol cannot reasonably be expected to be effective in that large majority of clinical cases.

To some degree, variants of the original post-retrieval extinction protocol can have a wider range of clinical applicability by replacing the conventional extinction protocol with forms of counter-learning that are more suitable for the target learning being addressed. Many such studies have been made, with both positive and negative results, as reviewed by Auber et al. (2013), Kredlow et al. (2016), and Lee et al. (2017). (See Section 9.1 for further analysis of this approach to clinical translation.) Of relevance here is to note the procedure-minded use of the term "extinction" to label counter-learning after target learning destabilization. To term post-destabilization counter-learning "extinction" is a misnomer that creates misconceptions, in the author's opinion, because it produces none of the effects that have been identified with the term "extinction" for a century. Even when post-destabilization counter-learning has the identical procedural form as in conventional extinction training, its learning function (namely, disconfirmation and unlearning) and its neurological effect (namely, re-encoding of target learning) are qualitatively different from those of extinction. It is well established that reconsolidation and extinction are distinct, mutually exclusive phenomena (Duvarci and Nader, 2004; Duvarci et al., 2006; Merlo et al., 2014). The operative principle here is this: A particular learning procedure (such as the repetitive non-reinforcement of conventional extinction) can have entirely different neurological and behavioral effects depending on whether or not it is carried out during the reconsolidation window.

In relation to the target learnings encountered in psychotherapy, the possible designs of experiences of counter-learning are virtually unlimited (Ecker, 2016; Ecker and Hulley, 2017a; Ecker et al., 2012). Monfils et al. (2009) and Schiller et al. (2010) have shown that, for the specialized case of a Pavlovian target learning that has been destabilized, counter-learning in the form of the standard extinction training protocol can accomplish erasure. However, that is a specialized format of counter-learning for that case.

Thus it is apparent that the post-retrieval extinction protocol is a highly specialized instance of the much more broadly defined ECPE. The procedures used in psychotherapy for implementing the ECPE with a given client must necessarily be designed according to the structure and content of the target learning(s) revealed by that client, in order to successfully induce reactivation, mismatch and counter-learning experiences. It is always the specific content and structure of the target learning that determines which experiences will, or will not, register as reactivation, mismatch and counter-learning. The clinical case examples later in Section 7 illustrate this critical point.

In psychotherapy, each revealed, symptom-generating target learning is found to be a unique, idiosyncratic, multi-component formation that was shaped by the unique life experiences of that individual; and, as noted earlier, idiosyncrasy of underlying emotional learning is the case even among therapy clients whose symptoms are in the same formal diagnostic category, such as panic disorder or dysthymic depression. Therefore, effective ECPE implementation with each client necessarily requires clinicians to have a free hand in choosing concrete behavioral measures (interventions) that will adequately custom-tailor the critical sequence of experiences for the unique emotional learnings of each client. Essentially the same conclusion was reached by Elsey and Kindt (2017a, p. 113), who reviewed many factors that influence whether a particular memory reactivation procedure will create a mismatch that achieves destabilization, and in summary commented, "Taking into account these different factors, it begins to look unlikely that any single reactivation procedure will prove effective for all who undergo it, potentially undermining the use of very standardized reactivation procedures that may be pursued in clinical trials."

It may seem paradoxical that clinicians' adherence to the ECPE's components of reactivation, mismatch and counter-learning actually requires fluidity on the concrete level of treatment, rather than some pre-defined concrete protocol that was previously used successfully in laboratory studies or clinical trials. Clinical use of a fixed concrete intervention protocol is contra-indicated not only by the totality of the memory reconsolidation research, as described above, but also by the thorough idiosyncrasy inherent in human emotional learning histories. To view any particular behavioral procedure or protocol as necessary or inherent for utilizing memory reconsolidation in psychotherapy is a misconception that would limit the range of use and effectiveness to the particular type of emotional learnings for which the favored protocol happens to be suitable. Whereas, by allowing the clinical use of the empirically confirmed process to be open-ended and eclectic on the level of concrete

behavioral procedure, the range of applicability and effectiveness encompasses the entire universe of symptoms generated by implicit emotional learning.

The necessary eclecticism regarding behavioral procedures runs counter to the conventional assumption that adherence to a proven, well-defined concrete treatment protocol is essential for establishing scientific validity, and has important implications for how the status of Empirically Supported Treatment (EST) or Evidence Based Treatment (EBT) is to be achieved for ECPE-centered psychotherapy. Procedural eclecticism of course has implications also for clinical training in the endogenous use of memory reconsolidation, but that topic is beyond the scope of this article.

6.3. A proposed universal clinical methodology of memory reconsolidation

Having defined the *empirically confirmed process of erasure* (ECPE), the quest for clinical translation takes the form of the pragmatic question: How can psychotherapists best facilitate the ECPE for nullifying symptom-generating emotional learnings? The requisite experiences identified by extensive research are clear, as summarized in Section 6.1, namely experiences of target learning reactivation, mismatch and disconfirmation by counter-learning. What has to happen in therapy sessions for those experiences to occur? What general methodology of psychotherapy is implied or even necessitated?

A general clinical methodology for ECPE facilitation, proposed by Ecker et al. (2012, 2013a, 2013b), is based on one of the fundamental findings of reconsolidation research, the necessity of tailoring the requisite ECPE experiences to the specific composition of the target learning or schema. Whereas laboratory researchers have detailed knowledge of the target learning because they create the target learning in the first place, in contrast a psychotherapist is completely unaware of the emotional learnings maintaining a new client's presenting symptoms. Even the symptoms (unwanted behaviors, states of mind, and/or somatic disturbances) are unknown to the therapist at the start. Furthermore, even when symptoms have been well identified, their underlying emotional learnings are not thereby inferable because, as noted in Section 2, the emotional learning history of each person is unique, and different individuals have different schemas or memories manifesting the same diagnostic category of symptom.

Therefore Ecker et al. (2012, 2013a,b) maintain that a therapist, in order to carry out the ECPE with reliable consistency across clients presenting diverse symptoms, must first (A) elicit specific descriptions of the symptom(s) to be dispelled and then (B) elicit fine-grained descriptions of the emotional learnings that necessitate and generate those symptom(s). Then, guided by familiarity with the details of a particular target emotional schema, the therapist can now (C) find how to guide a counter-learning experience that will be used for mismatching and then disconfirming and nullifying that schema.

As soon as the three preparation steps A, B and C are completed, the therapist is now equipped to facilitate the ECPE's three experiences of reactivation, mismatch, and counter-learning. Lastly, after completing the ECPE, the therapist must obtain verification of erasure in the form of observations of the markers of erasure delineated in Section 3.

Table 3 lists that seven-step clinical process defined by Ecker et al. (2012, 2013a). Those authors designate this methodology as the *therapeutic reconsolidation process*, or TRP, and they propose it as being a universal map of therapeutic process for utilizing memory reconsolidation to produce transformational change. The universality of this methodology is posited on the basis of its applicability for all unwanted behaviors, states of mind, and somatic disturbances maintained by implicit knowledge acquired through emotional learning, as well as its open access to all clinicians without favoring or requiring any particular clinical methods or theoretical orientation.

The TRP is a methodology of experiences, not behavioral procedures, a distinction discussed in Section 6.2. Clinicians are free to fulfill the steps of the TRP using the concrete methods and techniques in which they have training and which they deem most suitable for a particular client. Ecker et al. view the TRP as being a meta-methodology that is determined entirely by the brain's innate functioning, which, if true, would have two implications: All other methodologies designed to utilize memory reconsolidation in therapy would prove to utilize a subset of the instructions provided by the TRP (see Section 9 for an examination of several other such methodologies in relation to the ECPE and TRP); and if some systems of psychotherapy

were to prove less suitable for implementing the TRP than others, that would not be due to any bias in how the TRP was conceived or structured.

Table 3. The Therapeutic Reconsolidation Process, proposed as a universal template derived from reconsolidation research for utilizing memory reconsolidation in clinical practice.

Therapeutic Reconsolidation Process	
Preparation phase	A. Symptom identification
	B. Retrieval of target schema
	C. Identification of disconfirming knowledge
Erasure sequence (ECPE)	1. Reactivation of target schema
	2. Destabilization of target schema: Activation of contrary knowledge mismatches target schema (first juxtaposition)
	3. Nullification of target schema: Several repetitions of juxta-position for counter-learning during remainder of session
Verification phase	V. Verification of target schema erasure:
	• Symptom cessation
	• Non-reactivation of target schema
	• Effortless permanence

The remainder of this section provides a degree of expanded description of the seven steps, all of which are demonstrated in two case examples of TRP facilitation in Sections 7.1 and 7.2 below. (For more extensive and intensive accounts, including an array of specific therapeutic techniques useful for each step, see Ecker and Hulley, 2017a; Ecker et al., 2012; Ecker, 2015c.)

TRP Step A, *symptom identification*, consists of actively engaging the client in recognizing and labeling the specific behaviors, somatics, emotions, and/or thoughts that the client wants to eliminate, as well as identifying when these unwanted experiences happen, that is, the situations and perceptions that evoke or intensify them. This information is essential for carrying out Step B effectively. In many cases, symptom identification can be fully accomplished within the first session, but it can require several sessions with some clients. As basic as this step may seem, the author has found in over two decades of conducting clinical trainings that many experienced therapists are unfamiliar and unskilled with obtaining a well-defined symptom picture efficiently at the outset of therapy.

TRP Step B, *retrieval of target learning*, is an experiential process of eliciting into explicit awareness the emotional learning and memory maintaining a symptom, guiding the client to verbalize the emergent material while feeling it, and then integrating the newly conscious knowings and feelings into routine daily awareness as a personal emotional truth (such as the sample emotional learnings provided in Section 2). The subjective, affective quality of this retrieval is critically important, as has been shown in both clinical and laboratory studies (e.g., Greenberg, 2012; Yacoby et al., 2015). The retrieved, symptom-generating material may consist of episodic memory (specific experiences, including affective and somatic elements and construed meanings), semantic memory (schema-structured, generalized knowings regarding certain types of situation, meanings, self's vulnerability to a specific form of suffering, the expected behavior of others/self/world, self-protective tactics necessitated, etc.), or both. As described in Section 2, symptom-generating schemas are multi-component, layered formations with a recognizable structure that enables the therapist to know when retrieval of a schema is complete. One of the components, vivid personal knowledge of a specific suffering that is urgent to avoid, corresponds to the US (unconditioned stimulus) in laboratory studies, and, as suggested by research noted at the end of Section 4 above, retrieval and reactivation of this component (which is mandatory in TRP Step B) may allow efficient erasure of all of its linkages to associated cues and contexts (CSs). Typically, one or two of a schema's component constructs are the most effective targets for disconfirmation (Ecker and Hulley, 2017a; Ecker et al., 2012, pp. 68–70). Retrieval of a symptom-generating emotional schema may entail the client feeling significant vulnerability and dysphoric emotion, and therefore requires much skill on the part of the therapist, who must pace the process workably for the client's tolerances and provide empathic accompaniment necessary for the client's sense of safety and trust. While an advanced practitioner can often complete retrieval of a schema in one or two sessions, more generally a few sessions are needed, and the number of sessions increases commensurate with the complexity and emotional intensity of the material. Material revealed in Step B is the target of change; symptoms identified in

Step A are not themselves direct targets of change, but cease when their underlying emotional learnings and memories are unlearned and nullified. Close familiarity with the learnings and memories revealed in Step B is essential in order for the therapist to be able to embark upon TRP Step C, identification of disconfirming knowledge.

TRP Step C, *identification of disconfirming knowledge*, consists of finding past or present experience(s) in which the client has direct, living knowledge that is fundamentally contrary to the target learnings and memories retrieved in Step B, such that both cannot be true. Specificity of disconfirmation is critically important for consistently achieving successful nullification of the underlying target material with client after client. The needed contrary knowing can either be found in the client's already-existing knowledge from past experiences or it can be created by a new experience that occurs during or between therapy sessions. Each of those main sources has several subtypes, and all can be accessed through a wide variety of techniques (Ecker, 2016; Ecker and Hulley, 2017a). Step C consists only of finding where and how such disconfirming knowledge can be readily accessed for carrying out TRP Steps 2 and 3, described below.

TRP Step 1, *reactivation of target learning*, is readily accomplished using basic experiential methods of guiding the client's attention to key features of the target schema or memory and inviting the client to allow and attend to the affective and somatic aspects of what arises. The client feels empathically accompanied by the therapist while opening to and entering into the altered state inside the schema or memory. Reactivation is adequate when the client's consciousness, while maintaining relational connection and communication with the therapist, is significantly sampling and inhabiting the subjective, affective reality and self-state generated by the target material, with particular, mindful recognition of that material's specific feature that is going to be mismatched and disconfirmed in the next step.

TRP Step 2, *activation of disconfirming knowledge, mismatching target learning*, consists of guiding an initial experience of the contradictory knowledge that was found in Step C, while the target schema or memory remains reactivated from Step 1. This first instance of the client experiencing a juxtaposition of the target material and the contradictory knowledge is a mismatch or prediction error experience, which is needed for destabilizing the neural ensemble encoding the target learning, as reviewed in Section 4. In therapy, using the counter-learning experience designed for the next step, Step 3, to first create the mismatch here in Step 2 is simply efficient; there is no need for what would be the extra step of creating some other type of experience for the mismatch. Contradiction goes by the name of non-reinforcement in laboratory studies that use a Pavlovian target learning. (In some clinical cases, the reactivated target schema or memory is mismatched and destabilized presumably by the presence of the therapist, the therapist's office and the safety of the environment. That is most likely in the subset of cases where the target material allows the client's experience of the therapist to be the contrary knowledge used as counter-learning. In many other cases, the therapist and therapy session environment are not directly relevant to the target schema (discussed at length by Ecker et al., 2012, pp. 93–100) and are merely novelties in relation to it. Novelty too can serve to create mismatch, as noted in Section 4, if the novel item also has subjective relevance to the target schema and therefore requires an update of the schema; novelty without relevance requires no updating and therefore does not register as a mismatch. For that reason, Step 2 requires something more reliable than the therapeutic setting to serve as the mismatch. Activation of the contrary, disconfirming knowledge identified in Step C is always a definite and reliable mismatch because contradiction always has strong relevance to the target learning and always requires updating.)

TRP Step 3, *counter-learning by repetitions of the disconfirming juxtaposition*, consists of guiding the client a few more times to attend to and affectively feel both experiences, the target material and the contrary knowledge. This accomplishes the disconfirmation, unlearning and nullification of the target schema or memory. (In some cases, complications arise, requiring extra steps, as discussed below.) In that juxtaposition of two mutually contradictory knowings, it is the target learning that is disconfirmed and unlearned because the target learning consists of a less complete, less inclusive model of reality (having been formed at a young age and/or while in distress under extreme, special circumstances), and its falseness or too-incomplete account of reality becomes vividly apparent and viscerally felt in being juxtaposed with a more complete and inclusive model of reality. A reactivated target schema

consists of certain definite knowings and expectations of how the world is, yet now there is a concurrent experience with direct perception that that is definitely not how the world is. This subjectively felt *juxtaposition experience,* consisting of the two mutually contradictory knowings, is regarded as fulfilling the brain's requirements for behavioral erasure as identified in reconsolidation research, and therefore as being the critical experience required for schema nullification and transformational change in psychotherapy (Ecker, 2006, 2008, 2010, 2015/2006; Ecker and Hulley, 1996, 2000b, 2002, 2008; Ecker and Toomey, 2008; Ecker et al., 2012, 2013a, 2013b). Initially both sides of the juxtaposition experience feel real and true to the therapy client, yet they cannot both be true. The both-at-once experience inherently entails a peculiar tension or edginess, similar to the experience of cognitive dissonance (Festinger, 1957), but here it is fully experiential and visceral rather than only conceptual. With counter-learning via a juxtaposition experience repeated a few times during the remainder of the therapy session, the compelling realness and urgency of the target learning's version of reality immediately wither and lose all feeling of realness or urgency, and cease driving symptom production. That is demonstrated in the case examples in Sections 7.1 and 7.2. Carrying out the ECPE in psychotherapy amounts to guiding a juxtaposition experience a few times. The entire TRP exists to bring about that set of juxtaposition experiences.

TRP Step V, *verification of erasure of target memory material*, consists of observing and documenting the markers of erasure discussed in Section 3, namely unambiguous reports from the client that (a) the client's initially identified symptomatic behavior and/or state of mind and/or somatic disturbance has ceased to occur in all situations where it had been occurring, (b) the affective self-state or compelling emotional "spell" created by the reactivated target schema no long occurs in response to any cues or contexts that previously evoked it, and (c) changes (a) and (b) persist under all circumstances, without relapse and without any effort or measures taken to maintain them. In most cases, marker (b) becomes apparent in the same session following facilitation of the ECPE, in the form of the client giving clear verbal and nonverbal messages that the reactivated self-state of the target memory material has disappeared and that it is not re-evoked when the therapist reapplies imaginal cues that had potently evoked it previously. Some clients state that the specific content of the target schema or memory, which had always felt compellingly real and intensely dysphoric, now feels "absurd" or "ridiculous" to believe. However, verification is made conclusive only by the persistence of the markers over many months and in all real-life situations that formerly triggered symptom production. The therapy work addressing a given symptom and its underlying emotional learning(s) can be regarded as complete only when the markers of erasure are firmly established.

The TRP, consisting of the seven steps A–B–C–1–2–3–V described above, is fairly simple in its conceptual essence, but it is complex and subtle in its overall clinical implementation across therapy clients who differ widely in personality, tolerance for emotional experience, extent and depth of suppressed emotional distress, readiness to trust the therapist, and other variables. The clinical methodology now known as the TRP has been in use by the author and colleagues plus numerous clinicians worldwide for 25 years (because, as noted in Section 3, the methodology was developed based on clinical observations prior to the laboratory discovery of memory reconsolidation). We have gained much understanding of how to attune the process to the unique individual therapy client (Ecker et al., 2012) and have observed the markers of erasure ending a wide range of clinical symptoms, as summarized in Table 4. Such broad versatility of application significantly extends the clinical translation efforts previously covered in review articles. Nearly all of those efforts have addressed only two symptoms: the intense, fearful aversion that is a chief characteristic of post-traumatic symptoms, and the intense craving that is a chief characteristic of addiction. In both cases, several successful clinical or pre-clinical utilizations of reconsolidation have been reported; for reviews see Dunbar and Taylor (2017), Lee et al. (2017), Schwabe et al. (2014), and Treanor et al. (2017). It is well established, however, that all of the many types of memory that have been tested undergo reconsolidation, as noted in Section 3. The TRP is proposed as a framework that positions clinicians for addressing the entire range of memory-based symptomology. Section 7.1 below demonstrates TRP implementation for a symptom of lifelong reactive anger. Section 7.2 does likewise for an adult's symptoms of formless terror accompanied by severe kinesthetic disturbances, which were found to be based in childhood attachment trauma.

Table 4. Clinical symptoms observed to be dispelled by the therapeutic reconsolidation process as carried out in Coherence Therapy*

Symptoms Dispelled	
Aggressive behavior	Food/eating/weight problems
Agoraphobia	Grief and bereavement problems
Alcohol abuse	Guilt
Anger and rage	Hallucinations
Anxiety	Indecision
Attachment-pattern-based behaviors and distress	Low self-worth, self-devaluing
Attention deficit problems	Panic attacks
Codependency	Perfectionism
Complex trauma symptomology	Post-traumatic symptoms
Compulsive behaviors of many kinds	Procrastination / Inaction
Couples' problems of conflict / communication / closeness	Psychogenic / psychosomatic pain
Depression	PTSD symptoms
Family and child problems	Sexual problems
Fidgeting	Shame
	Underachieving
	Voice / speaking / singing problems

*An online bibliography of published case examples indexed by symptom is available at http://bit.ly/2tKXdyX

Brief comments follow on a few aspects of TRP implementation particularly relevant to the current article. Facilitating the TRP can be complicated by what therapists typically term client resistance, meaning that the client does not comply with or allow the process that the therapist is attempting to facilitate. Resistance is self-protective, is done without conscious awareness in many instances, and can develop at any step of the TRP. Resistance occurring in TRP Step 3, counter-learning by repetitions of the disconfirming juxtaposition, is notable as being unique to this methodology. Clinical experience has shown that a target schema will not be disconfirmed and nullified by the juxtaposition experiences in Step 3 if the client (non-consciously) anticipates consequences of nullification that feel too distressing or costly in any way. (For a detailed case of this effect, see Ecker et al., 2012, pp. 77–86.) The brain's implicit predictive capability proves to be remarkably astute regarding unacceptable adjustments entailed by a particular schema losing realness and being decommissioned. The schema simply remains in force (continues to feel compellingly real and potent) despite well-crafted juxtaposition experiences being guided. That persistence of the schema is the therapist's indicator that there is some blocking contingency that now must be sensitively brought into awareness, recognized and addressed. When the client arrives at feeling that the (now consciously) anticipated difficulty is workable, the therapist repeats the juxtaposition experiences of Step 3, and schema nullification now is allowed to occur.

A related phenomenon is the persistence of emotional learnings despite numerous personal experiences in which the schema's expectations clearly do not occur. A schema can remain immune to disconfirmation by such experiences in a number of different ways (for discussion of which, see Clarke, 1999; Ecker, 2015a, p. 13; Ecker and Toomey, 2008, pp. 115–116; Fernández et al., 2017; Proulx et al., 2012), some of which can also block the disconfirmation effect sought in TRP Steps 2 and 3. As noted in the previous paragraph, the TRP equips clinicians to reveal and work directly with a given therapy client's particular dynamics maintaining such immunity to disconfirmation.

The TRP's preparation Steps A–B–C make it possible for clinicians to carry out the erasure sequence, Steps 1–2–3, systematically, consistently and effectively in day-to-day practice. In other words, Steps A–B–C are pragmatically necessary in order to implement Steps 1–2–3 as a deliberate methodology. However, because Steps 1–2–3 are defined in terms of experiences to be created, not procedures, they can also occur serendipitously and implicitly, unbeknownst to the therapist, without Steps A–B–C being carried out. That is not only possible, but also common. Probably a majority of clinicians at least occasionally observe a transformational change (the markers of erasure defined in Section 3) resulting from methods that may have little or no obvious similarity to the TRP and without conceptualizing therapy in terms of memory reconsolidation. As discussed in Section 3, the markers of erasure imply the occurrence of the experiences that Steps 1–2–3 (the ECPE) are designed to create. The likelihood of facilitating those critical experiences without awareness of the TRP or knowledge of memory reconsolidation presumably varies from one system of psychotherapy to another (even assuming perfect adherence to and implementation of each system's methodology). Also presumably, *having* knowledge of

and training in the TRP should significantly increase any clinician's likelihood and frequency of facilitating those critical experiences and producing transformational change in daily clinical practice.

In addition to enhancing an individual clinician's effectiveness, the TRP also potentially has significant methodological and theoretical ramifications for the clinical field, some of which are discussed in Section 8 (and in the literature cited above in the paragraph on TRP Step 3.)

7. Clinical observations of the therapeutic reconsolidation process

The previous section drew upon reconsolidation research findings to assemble a maximally general clinical methodology of behavioral updating that is directly and entirely dictated and defined by the research, yet is not restricted to any particular laboratory procedures. The methodology that emerges from the research in that manner is mapped out in Table 3. Its core is the empirically confirmed process of erasure (ECPE), a sequence of three experiences, the consistent facilitation of which requires the preceding three steps of preparation, in which relevant material is accessed and made ready. Following the steps of the ECPE, verification consists of observations of the markers of erasure discussed in Section 3. The entire methodology, designated the therapeutic reconsolidation process or TRP, is applicable to any symptom generated by emotional learning and memory and has been used by clinicians to eliminate the symptoms listed in Table 4.

The TRP is a clinician's map of process. Because it is a methodology of experiences, not behavioral procedures, the clinician has to choose the behavioral procedures to use for fulfilling the steps of process for a particular symptom of a particular therapy client.

This section begins by providing two demonstrations of TRP implementation. These are uncontrolled clinical case descriptions involving intense, life-long symptoms of reactive anger in Section 7.1 and terror accompanied by severe somatic disturbances in Section 7.2. Certainly these clinical accounts could be considered merely anecdotal. However, several accounts of uncontrolled clinical cases have been published in support of the clinical translation of reconsolidation research (e.g., Brunet et al., 2011; Högberg et al., 2011; Kindt and van Emmerik, 2016; Poundja et al., 2012). Each of the accounts below is a fine-grained phenomenological record, not merely a narrative gloss of what happened. It is proposed that each account provides unambiguous demonstration of the ECPE's sequence of distinctive experiences occurring within the overall methodology of the therapeutic reconsolidation process (TRP), followed promptly by observation of the markers of erasure, including complete, long-term disappearance of the presenting symptoms. As discussed in Section 3, a clinical outcome consisting of the markers of erasure is significant support for the hypothesis that erasure via reconsolidation has occurred. Such demonstrations are of value for developing clinical translation. Empirical knowledge of the sequence of experiences required for erasure allows the very moments of transformational change in therapy to be plainly apparent and makes the operative ingredients in those moments plainly apparent (Ecker et al., 2012), even amidst the complexities of the client-therapist interaction.

The case examples are followed in Section 7.3 by a consideration of whether the observed transformational changes can be ascribed plausibly to memory reconsolidation. Section 7.4 concludes this section by addressing reconsolidation researchers' anticipated obstacles to clinical application in light of actual, extensive clinical observations.

The case examples in Sections 7.1 and 7.2 are submitted here as a demonstration that the ideal therapeutic goal of a methodology that completely eliminates unwanted emotional responses, as put forth in Section 1, has become a clinical reality through facilitation of the process identified in reconsolidation research. The aim of the author (and colleagues) has been to conduct and document clinical cases with sufficient phenomenological detail and specificity to allow meaningful evaluation of the claim that reconsolidation and erasure have been demonstrated. To that end, the emphasis in such clinical accounts is on (a) showing the unambiguous implementation of the required experiences of reactivation, mismatch and counter-learning, (b) showing that even with the presence of all nonspecific factors widely known to be important for effective psychotherapy (e.g., Duncan et al., 2009; Wampold, 2001, 2015), it is not until the specific experiences required for destabilization and erasure occur that a transformational shift occurs, and (c) the transforma-

tional shift consists of the markers of erasure being manifested decisively by the client, including total elimination of emotional responses and associated behaviors for which the client sought therapy. That is the outcome defined by Clem and Schiller (2016, p. 340) in order for therapy "to achieve greatest efficacy."

Numerous case studies that fulfill those criteria have been published; a listing of them is available online at http://bit.ly/2tKXdyX. The system of psychotherapy used in those case studies is Coherence Therapy (Ecker and Hulley, 2017a), which has a methodology that explicitly consists of the TRP. The two case studies that follow were chosen for presentation here on the basis of the succinct instructional clarity they allow. Other than that, the results they present are not exceptional relative to the numerous other published case studies.

The following two clinical case vignettes are also intended to demonstrate several specific aspects of applying the empirically confirmed process of erasure in therapy:

- Use of methods developed for revealing the emotional schema maintaining a given symptom, defining the target learning
- Use of the revealed target learning's specific contents to guide the design of reactivation, mismatch and counter-learning experiences
- Markers of erasure verifying complete, long-term disappearance of targeted emotional responses
- Erasure of emotional learnings other than fear learnings and appetitive (addiction) responses, which reconsolidation researchers have so consistently specified as the potential target for application of their findings that clinicians might be led to assume that only such learnings can be updated and erased via reconsolidation
- Clients' accounts of the subjective experience of emotional memory erasure. This information is of interest to researchers because, as Elsey and Kindt (2017a, p. 113) have noted, "remarkably little research has considered what the subjective experience of these changes is."
- Favorable observations regarding researchers' anticipations of obstacles to applying experimental findings clinically

7.1. Clinical case example: erasure of chronic anger

The client is a woman in her early 50s, pseudonym Norina. The therapist is the author. After about 15 sessions focused largely on various difficult patterns in her marital relationship, Norina identified a feeling of angry resentment that had frequently gripped her and ruled her state of mind and behavior for hours, days or weeks, for as long as she could remember, since her childhood. Her simmering, angry mood had activated often toward her husband of 28 years and was a major factor in the chronic tension between them.

The therapist carried out the therapeutic reconsolidation process (TRP; see Table 3) to find and erase the emotional learning driving that anger, resulting in Norina reporting long-term cessation of this emotional reaction. The process unfolded as described below. This case is intended to demonstrate the applicability of the TRP to emotional learnings of any type (not only those maintaining fear or addiction, the two types of symptom that nearly all clinically oriented reconsolidation researchers have addressed to date, as reviewed by Dunbar and Taylor, 2017, Schwabe et al., 2014, and Treanor et al., 2017) and the uniqueness and specificity of the emotional learnings in each clinical case, requiring TRP Step B, retrieval, to be carried out diligently and thoroughly.

TRP Step A, symptom identification, was straightforward. Her reaction of anger and resentment was easy for her to name. However, that reaction had happened in so many different situations across the decades that she could not define the key features of situations that triggered it.

In pursuing TRP Step B, the retrieval into explicit awareness of an implicit schema generating the client's anger, the therapist's task was to elicit the emotional learning that was driving this angry resentment in so many different situations for a lifetime. In order to begin guiding her attention and awareness into that area of implicit learning, the therapist said to her, "Just see what comes to mind when I ask you this question: In your whole life, what is it that you resent the most?"

She answered that it was something she was already well aware of: her childhood ordeals of being sexually molested by her grandfather on a number of occasions, starting at 6 years of age. She explained that she had already had extensive therapy for that, long ago.

Hearing that, the therapist thought: It's natural that

a person would feel angry resentment about suffering such an ordeal of violation and betrayal. And yet, not every person who has suffered sexual molestation in childhood has a dominant mood of angry resentment for the next five decades, as Norina had. That indicates something unique and specific in her emotional learnings that has been generating such anger. How to elicit that specific material?

The therapist next asked gently, "What is it that you suffered in that ordeal that you resent more than anything else about it?" That question guided her attention into her implicit knowledge to a new degree. Her previous therapy had not led her to examine with this specificity what she had suffered. As Norina looked into that, her eyes were blinking, and then she said with a note of surprise, "Hmm, it doesn't feel like it's my grandfather that I resent the most." She pondered a bit more and then said, "It feels like, even more than resenting my grandfather, I resent the whole world, or life itself."

The therapist now asked, "What can you see about why you resent the whole world or life itself?" in order to continue the flow from implicit knowing into explicit knowing.

Norina examined this in silence for several seconds, and then said, "It's that the world is just too unfair, to make this happen to *me*, and to no one else."

That was the emergence of the emotional learning that was producing her anger. That construal of meaning, formed as a young girl, had never before entered her conscious thinking as an adult. It had remained a felt knowing in the implicit background, though it launched anger that came into the explicit foreground of awareness. The therapist immediately understood that that single, unique, implicit construct and attribution of meaning, that it had happened only to her, led her in turn to view the world as monstrously and unforgivably cruel and unfair, so she was indeed profoundly and unceasingly angry and resentful at life. After age 6, whenever Norina perceived anything else in life as being an unfair and arbitrary treatment, it retriggered that same smoldering anger and resentment, but without awareness of the source of that anger. Arbitrary, unfair things, small and large, happen fairly often in day-to-day life, and in a marriage, so she was often swept up into that resentful anger throughout her life.

She had voiced the words with an indignant intonation and a facial expression of consternation, which gave two important indications to the therapist: First, she was affectively experiencing this schema in the moment, not merely intellectualizing about it, which is the needed quality of accessing here in TRP Step B. For schemas to become consistently available for disconfirmation and erasure, therapists must guide clients to inhabit the material subjectively and articulate it from inside the felt realness of it. Second, the schema felt as true as ever to her, even though it was now in her explicit awareness. This continuing potency of the retrieved schema is found to be the norm in carrying out Step B. Although in full awareness, the schema remains in full force and continues to generate symptoms despite the high-quality presence of the nonspecific common factors (as discussed in Section 8.3). Nullification of the felt realness of an emotional schema is observed to occur not by bringing it into awareness, but by subjecting it to disconfirmation and unlearning in TRP Steps 1–2–3, the ECPE, which was yet to come.

The target schema's full verbal representation can be formulated as, "The world allowed that horrible ordeal to happen only to me, and to no one else, and that means the world has been unforgivably unfair and cruel to me. I protest by feeling furious resentment at the world for being so arbitrarily cruel and unfair to me, and every time I see or feel any more of that arbitrariness or unfairness, I protest again with my angry resentment."

TRP Step B was now accomplished, but before describing how the therapist undertook Step C, there are two noteworthy points illustrated by the revealed schema. Knowing the detailed content of this target schema makes it apparent that:

- The schema could not possibly be disconfirmed by the client's experience of the therapist. The therapeutic strategy of using the client-therapist relationship for a corrective emotional experience could not be effective for this schema, because the client's positive experience of the therapist is irrelevant to the specific content of the schema. TRP Step B has the built-in value of revealing whether or not this widely pursued clinical strategy is appropriate.
- The symptom of reactive anger was arising from semantic memory, not from episodic memory.

> The sexual molestations by her grandfather were traumatic, obviously. They were both acute incident trauma and complex attachment trauma. TRP Step B had revealed that Norina's anger was a post-traumatic symptom. Yet the aspect of the trauma that had remained highly reactive in memory and generated life-long, life-shaping symptoms was neither the physical perceptions, nor the somatic sensations, nor the emotional experience with its various intense aspects of violation, helplessness, fear, entrapment, and betrayal. It was the client's semantic construal of the meaning of the ordeals that was the symptom-generating memory. Post-traumatic symptoms are most often conceptualized by clinicians as arising from episodic memory, but TRP Step B most often reveals semantic memory at the root of symptom production, and that phenomenological observation is confirmed when nullification of the semantic structure (the retrieved schema) is followed immediately by permanent cessation of the symptom. (See Section 2 for discussion of the full constellation of interlinked semantic memory, episodic memory and affective/somatic activation.)

Detailed familiarity with the contents of the target schema from Step B thoroughly guides TRP Step C, which is finding a decisive, experiential, highly specific disconfirmation of that schema. Step C requires the therapist find how to guide the client into experiencing personal knowledge that feels undeniably true and that sharply contradicts what the client knows in the target schema. This contradictory knowledge is to be found either in the client's existing knowledge from past experiences or in new experiences to be created in the present.

Numerous methods for carrying out Step C are mapped out by Coherence Therapy, as noted in Section 6.3. A particular one of them is best for beginning Step C, as a rule, because it is the most likely to succeed. That method consists of submitting the discovered schema to the brain's always-active mismatch detection system, by simply having the client declaratively voice the schema out loud (Ecker et al., 2012; Ecker and Hulley, 2017a). This method takes advantage of the fact that in at least half of all cases, the client is found to already possess vivid contradictory knowledge, but it has not disconfirmed the target schema because the two knowings are held in different memory systems that have little if any direct communication between them. The target schema has existed (prior to Step B) only in the implicit and procedural knowledge networks of the subcortical brain, and the contradictory knowledge typically exists in the declarative and social knowledge networks of the cortical and neocortical brain. Voicing the schema out loud can create a mismatch with any existing contradictory knowledge, activating the latter and drawing it into focal awareness. In the present case, a disconfirmation and nullification of "It happened only to me" would eliminate the very basis of both viewing the world as cruelly unfair and the angry resentment in protest of that unfairness.

The therapist, figuring it was likely that Norina's conscious, adult understandings included the certain knowledge that it most definitely *did not* happen only to her, decided to use the mismatch detection method to access the needed contradictory knowledge. That decision completed TRP Step C.

In going forward next to actually carry out that mismatch detection process, the therapist was embarking upon TRP Steps 1–2–3. He said simply, "Please say it to me again: 'It happened only to *me*.'"

Norina said out loud, "It happened only to me." Norina had already been experiencing the affective realness of those words, and now they again simply felt true to her. This fulfilled the experience defined by TRP Step 1, reactivation of the target learning; but the reactivated material was not yet circulating into other memory systems containing contradictory knowledge. Mismatch detection often requires two or three cueings, so the therapist asked her to please say it again.

When Norina said it this time, immediately her facial expression changed into a look of puzzlement, which is a familiar marker of the first conscious sensation of mismatch, just prior to having cognitive clarity. The target knowledge had begun to register in her present-day, declarative knowledge networks. The therapist now invited her to say yet another repetition of "It happened only to me."

After saying it this time, Norina's eyes began darting around as cognitive clarity formed. This is the moment of the *juxtaposition experience* defined by TRP Step 2, in which both the target knowledge and the

contradictory knowledge are present concurrently. The client's subjective feeling is as if to say, "Wait a minute. Hold everything. What I was taking to be reality *isn't*."

On Norina's face now was a look of amazed surprise, and what she said after a few seconds made it clear that Step 2 had occurred. She softly said, "Oh my God, I really thought it happened just to me. But it happens *everywhere*. It's a part of life everywhere. It's an *ugly* part of life, but it keeps happening to girls and boys too all the time, everywhere. I *wasn't singled out*."

Use of the brain's mismatch detector had worked and the mismatch had occurred, experienced as a strong disconfirmation of the target schema. The neural encoding of the target learning was now rapidly destabilizing, according to reconsolidation research. It was natural for client and therapist to remain focused on what had just happened. Norina kept giving amazed attention to this fresh realization, which repeated the juxtaposition each time, carrying out TRP Step 3, the counter-learning that nullifies the target learning and re-encodes it accordingly. In addition, with empathy the therapist explicitly reviewed both her old belief and her new realization, in order to repeat the juxtaposition experience a couple of times more by empathically reviewing it.

If the process was successful, in those few minutes Norina's childhood learning that she had been cruelly and unfairly singled out by life had been unlearned, dissolved and erased. Erasure means that "It happened only to me" would no longer feel real or true in any part of herself, or in any memory network. Without that construct that life had singled her out for such suffering, there would be no view of life as being hideously unfair to her and, in turn, no more generating of angry resentment over that. The dissolution would ripple through that whole linkage.

In the next therapy session one month later, the therapist began by asking whether she had noticed any subsequent effects of the previous session's work. This was now in pursuit of TRP Step V, verification of the markers of erasure. In response, Norina's exact words were, "I've been angry and resentful my whole life. It's like something has just turned to dust. It's not *alive* any more. Before, something felt like cords and cables strangling me. I feel *so freed up*."

Over the next couple of months she described a new ease, friendliness and warmth in the marital relationship. Eight months later, she and her husband had a particularly stressful month of not feeling emotionally in sync with each other during struggles with the extended family. Norina said, "It was rough, but I haven't felt any resentment toward him."

The therapist took this opportunity to elicit more follow-up for TRP Step V and asked her, "Can I check with you about the work we did on that core belief, 'It happened only to me'? I'm wondering whether or not the shift that you initially described has held."

She replied (and again these are her exact words), "My resentment had been relentless. Even with all these troubles, that anger is not taking over…. Most of the time I'm in a wonderful, energized, peaceful state. That's the way I would describe it…even with all these troubles."

Thus the markers of erasure and transformational change appeared to be well established: The unwanted emotional reaction and the behaviors it had produced had disappeared, and were no longer evoked by situations that formerly evoked them strongly, and this shift was persisting long-term and effortlessly.

Norina explained that when the molestation ordeal began as a little girl, she regarded it as something that was happening only to her, and not to anyone else in the world, simply because such a thing seemed not to exist anywhere else. She had never heard of it, and no one ever spoke of it. Yet it was happening to her, and its existence overshadowed her entire world. How she construed it, in all innocence, would have dominant effects on her life for almost fifty years.

The internal process of change in this case consisted of retrieving into awareness and then re-evaluating the meaning attributed to a childhood ordeal. That process could be conceptualized as a cognitive reappraisal (e.g., Ochsner and Gross, 2005; Ray et al., 2008), but certain distinctions are essential to make if misconceptions are to be avoided. Such reappraisal occurs in ECPE/TRP methodology (as noted in defining TRP Steps 2 and 3 in Section 6.3) in a juxtaposition of the original and new meanings that is fully experiential, as is apparent in the above case vignette, not a merely intellectual, cognitive consideration of the two meanings. The new meaning must register *in the client's emotional learning system* as being unmistakably real and true, because that is the memory network containing the target meaning, and that requires the new meaning to

be *subjectively felt* by the client as definitie personal knowledge, not merely a dry fact that is recognized intellectually to be true. Therefore the therapist's task is to guide the client into having her or his own living experience of the new meaning (and, more generally, of the contradictory knowledge found in TRP Step C).

The necessity of such fully experiential process in forming the reappraisal juxtaposition is not always recognized by researchers, with major effects on experimental results and conclusions. For example, Thome et al. (2016) tested for behavioral memory updating via cognitive reappraisal in the following manner: Human subjects learned to fear an image of a spider or a snake by experiencing a series of eight presentations of the image paired with an electric shock. One day later, that fear learning was destabilized by a mismatch experience consisting of a single presentation of the image without the shock. This was followed by the intended cognitive reappraisal experience (which was one of four experiments in this study), consisting of listening to a 15-min. neutral, informational narrative about spiders or snakes, heard via headphones binaurally. One day later, physiological measurements of subjects' fear in response to a single presentation of the image showed no reduction in fear, and on that basis (plus other findings) the authors state, "In conclusion, our findings do not support a beneficial effect in using reconsolidation processes to enhance effects of psychotherapeutic interventions" (p.1). That weighty conclusion in relation to the reappraisal experiment is premature and unwarranted, given the weakness of the tested reappraisal content. According to the criteria defined above, recorded neutral information for inducing reappraisal of images subcortically linked to a fearful expectation is predictably ineffective, because hearing dry facts does not generate one's own direct, real-feeling personal experience of how the world is. As such, it does not create a new and different knowing in the memory systems that contain the target learning, namely the emotional and sensory memory systems. Here again, the importance of understanding behavioral memory updating in terms of experiences rather than procedures is apparent.

7.2. Clinical case example: erasure of complex attachment trauma

A pervasive, lifelong feeling of "terror" was the symptom identified by a female client, age 66, in her fourth session. She had no awareness of the cause or content of her terror and could only label the feeling. Accompanying the session transcripts below are supplemental online session videos in which the subjective, experiential aspects of the developing process are more apparent. (Links to the videos are provided in the text below, but are password-protected. The videos are available for viewing, with the client's permission, only by mental health professionals, researchers, graduate program teachers, and graduate students. To obtain the password, send an email to articlevideo@coherenceinstitute.org and provide a credible form of documentation that you are in one of those categories.)

The client's three preceding sessions had addressed and dispelled a puzzling compulsive behavior of avoiding (procrastinating) important writing tasks in her professional work. Those sessions revealed a childhood in a family system that kept her perpetually in high anxiety and intense insecurity, and regularly inflicted emotional trauma of various kinds, creating the condition designated in the clinical literature by the various labels of complex attachment trauma, relational trauma and developmental trauma (e.g., Courtois, 2004; Sar, 2011).

As the oldest of six children, she was ordered in no uncertain terms by intensely self-absorbed, self-important, authoritarian, recklessly harsh parents to keep the other children safe, and her explosively rageful father threatened to kick her out of the family if she failed to do so. She therefore felt desperate to maintain continuous hypervigilant monitoring of her younger siblings. Her emotional learning that it was her job to keep the children safe and that she would be kicked out of the family for failing to do that job was a knowledge module or schema that was found to be still operating in the present, outside of awareness. That schema and the fear and urgency that it generated were compelling her to avoid doing tasks that would strongly absorb her full attention and allow an accident to happen to some sibling while she was wasn't closely watching over them—even though her siblings were now middle-aged adults living in distant cities.

In her fourth session, she reported that she had become able to do the tasks that she had been compulsively procrastinating. She then identified the lifelong feeling of terror as the next focus of therapy. It soon emerged that this terror was accompanied by intense

somatic and kinesthetic symptoms, as described below.

The following transcript and videos document how the therapist (the author of this article) carried out the therapeutic reconsolidation process (TRP), eliminating these symptoms in two sessions, followed by 2 years of verification of all markers of erasure. (C is the client, T is the therapist.) Comments inserted in the transcript are limited to pointing out implementation of the TRP steps; for more extensive clinical commentaries on this case, see Ecker and Hulley (2017b). Deleted portions of the session, indicated by an ellipsis (…), have been made according to the criterion that the deleted segment contains nothing that was necessary for the therapist to reach the next included segment or to make progress in carrying out the TRP.

VIDEO SEGMENT 1: http://www.coherencetherapy.org/videos-pwd/articlevideo/1.htm

C: … I feel like what's still there is terror.

…

T: Are there specific situations where that fear is stronger, noticeably stronger, where it's triggered?

The client's specification of "terror" begins to fulfill TRP Step A, symptom identification. The therapist's inquiry about when the terror intensifies would further define the symptom and could also elicit information that begins TRP Step B, retrieval of an emotional schema (semantic knowledge) that generates the terror.

C: [Referring to her boyfriend:] It's stronger if he's angry with me—if he gets angry. He even asked me the other day—I said, "I really get terrified." And he goes, "I don't really understand it." I said, "I don't really understand it either."

…

T: This terror of anybody being angry or upset with you—angry-ish, irritated, annoyed—

C: Oh yeah. Or criticized. If I'm criticized, that's the other one. Displeased with me, almost like in any way, that kind of displeasure, whether it's angry or disappointed or critical.

…

T: If someone becomes angry, or critical, or displeased, either what might happen that's really terrifying for you, or what does it *mean* that's really terrifying, or both?

The therapist's question comes from assuming that her terror arises directly from an implicit knowledge that anger or criticism directed at her could have an extremely dire result. The question is intended to prompt her to imaginally sample the experience of someone responding to her in that way and to attend to her implicit expectation of the terrifying results. This is more schema retrieval, TRP Step B.

C: Oh, man. [Silence as she searches internally for what is terrifying.]

T: Mm-hm. Yeah. And you know I'm not asking you to figure it out. I just want you to imagine or just admit to yourself and me—imagine somebody becoming—maybe Burt [boyfriend, pseudonym], just Burt becoming irritated. What might happen? Or what does it mean?

C: I mean like, I can go now, like I can sometimes say when he does that, internally, "I hate him," how he's such a jerk. I can carry on like that, but what's really terrifying is it's almost as if like I'll go into this, like, really dark place.

T: Mm. What's terrifying is that *you* go into a really dark place.

C: A dark place, yeah.

Her facial expression and tone of voice now indicate significant affect accompanying explicit recognition of the "really dark place," which is an experience that has been plaguing her for a lifetime yet is new for her to examine and describe.

T: Ok, let's—and what's that dark place like? What's that experience made of?

C: It's like a dark place I'll never be able to get out of.

T: That sounds terrifying.

C: Yeah, yeah. Not sure. Man.

T: Just sit with it. It'll show up. You've already found it. Let's just sit with that much. That's good. Mm-hm. Yeah, it's that you'll go into a really dark place and you might not ever get out of it. Yeah. [Silence.] Do you feel that in your body in any area? What is it you're feeling in your body?

C: It feels—and there's like a—well it's like, it's almost like I've become frozen? You know, like—um—

T: Frozen in the terror of being caught forever in that dark place?

C: Yeah, yeah.

Her description of the frozen terror that is triggered in response to receiving anger or criticism is a strong indication that it is a reactivation of traumatic memory.

T: Yeah. You're feeling some of that frozenness now?

C: Yeah, exactly, I'm trying to like stay in this like, this dark, you know— [Silence.] It's interesting. I just wanted to say something: this is the same thing, you know, this dark place I'm talking about—um, 'cause it feels like when I'm in this—the edges of this—or the dark place, it's almost as if, like, *I'm* not there and nobody else is there either. It's a weird—I mean—but if somebody forgets something I told them, I get so upset by that and literally the floor starts to feel like it's falling under me and I'm descending into this dark place. It's the same thing.

T: I see.

Somatic and kinesthetic symptoms are now also being attended to and labeled. Traumatic experience creates a schema in semantic memory that is retriggered by current perceptions that in some way match the original experience. Therefore the specific perceptions that evoke post-traumatic symptoms are indications of a trauma-based schema's contents. The client has for the first time recognized that the same plunge into dark, stark aloneness happens both when she receives a sharply negative response and when someone has forgotten something from their previous conversation. Those two types of trigger, taken together, indicate that the target schema contains an expectation of interpersonal attunement rupture that is so severe as to feel annihilative—a traumatic plunge into absolute disconnection and aloneness. The pursuit of TRP Step B is making progress.

C: And then I'll like stamp my foot and go, "I *told* you." Like that, I'll be really mad. But the stamping my foot stops me from falling into that dark place. I've always been wondering, what is this dark place? And why do I have such a strong reaction to somebody forgetting something I told them, you know? It's almost as if like I go into, like, I don't *exist* or something.

T: Yeah, yeah.

C: It's very like—very destabilizing.

T: Yeah, very, yeah.

C: I mean, I have all these like body tools to ground myself and do all those things—plant myself to feel my—stay in touch with my body. But there's—I've never been able to *get* to this thing.

T: Yeah, let's get to it. Mm-hm. Yeah, the same dark place.

C: Because, also, then what has happened is like, sometimes then I think I'll just, like, shut down. My body goes into a shutdown, kind of a numb.

T: Yes, because what you're starting—that dark place is so terrifying.

C: Yeah, exactly.

T: Nobody else is there. *You're* not even there.

C: Right.

T: And it's like you don't exist and you could be stuck there forever.

C: Yeah yeah yeah.

T: So, that's so terrifying that of course you go numb and just shut yourself down to not be having that experience. Yeah.

C: Yes, it's like being—like being—sometimes, I think I used to talk about it, or it's like being in this, like, um—like a solitary confinement. And also like, it's as if I was floating in outer space without a tether. Like, soundless, lifeless, um—no presence.

…

C: And no way out.

T: No way out, yes, yeah, like, yes, stuck forever.

C: Yeah, stuck forever.

T: So scary. And, when—let's see. When someone doesn't remember what you said, that can open—you can be falling into that dark place?

C: Yeah, literally feel myself starting to, like, it's as if you like, like you were suddenly like an elevator dropped really fast. You know, koonnnng.

T: Sinking feeling. Physical.

C: Like a dropping feeling, even literally, like it's as if the floor just came out from under me and I'm just dropping, I'm in a free-fall, or free-drop. That's what

it feels like.

…

C: And it's like, *terror* with it.

T: Yes, yes. What else would you want to say in that "but I told you!" if you were to really speak from the terror of what's *really* going on and feeling like you don't exist? You're starting to not exist, it feels like to you, when someone doesn't remember what you told them. So what would you say to the person explicitly about, "Hey, don't I exist?" Or how would you say something about that?

The therapist is guiding her experientially to revisit the first jolting moments of perceiving an interpersonal rupture with someone, in order to continue retrieving into explicit awareness the cognitive and emotional components of her emotional learning. Her next response shows that this focused attending to her generic (semantic) knowledge of the moment of rupture has now evoked episodic memory of particular childhood experiences of such traumatic ruptures.

C: I remember I used to have fights with my mother about this all the time. "I told you." And then she'd say, "No, you didn't." Or, "You *told* me this." "No I didn't." I'd go, "Yeah you did, I remember!"

T: Yeah, "I *did* tell you."

C: "I *did* tell you." "No I don't—" "Yeah, you did, you said it, I know you did!" And I was just like—

T: Fighting to exist.

C: And I'd just be so *terrified*, like, you know, like—

T: This memory of it happening with your mother—

C: Again and again and again, like she would deny that she said things or that I told her things. [Through tears and with anguish:] It was like, yeah, "You don't even remember me, I don't even *exist* for you! You can just forget me so quickly."

T: That's it: "I don't exist to you."

C: Yeah, I don't exist for you—

T: Say it again: "Mom, I don't exist for you."

C: [Through tears:] It's just like you don't even have—it's kind of like something like—you don't even have—I don't even know what's *real!* There's that sense of I don't even know what's real here because I can't even—you don't even remember what you *said* to me! And you tell me you didn't say it and I *remember* you said it. Kind of like I'm *depending* on you to remember what I tell you and what you tell me. I'm depending on you to.

T: Yes. Stay in shared reality.

C: Yeah, have a shared reality! Yeah, like something here that, this did happen, this did occur. Instead of you constantly saying, "No, it didn't occur, no it didn't. This didn't happen." I remember I would sometimes feel, "Am I going crazy?" And you know of course, I was so young.

For a young child, having a consistent, shared reality with caregivers stabilizes the child's knowledge of both herself and the world, and sharp, sudden losses of shared reality destabilize both areas frighteningly.

…

T: I wonder if that's the sinking feeling, that the reality is just dissolving.

C: Right!

T: And you're in the black void.

C: That's exactly it.

T: That's it.

C: That's it.

T: And it happened again and again and again with her, with mom.

C: Again and again.

T: I hear how *desperately* you would try to get her to hold up her side of reality.

C: Exactly, what—

T: And she wouldn't do it.

C: And she wouldn't do it.

T: And it would dissolve.

C: Yeah! But I remember just feeling so—

T: What's real? I don't know what's real.

C: Right!

T: I don't know if I'm insane. Am I crazy?

C: Yeah!

T: Yeah. So intense.

C: Yeah, and then I would really try to like track everything, keep track of *everything*. Just make sure that

I didn't forget a thing. Really trying to hold onto, like—

T: Oh, desperately. Because *you're* the one who has to hold it all together. You can't trust key other people to hold it together with you.

As she continued retrieving episodic memory of the maternal attachment trauma that she suffered myriad times, her attention shifted from her problem of mother's sudden, massive misattunements that feel annihilative, to her solution of trying to proactively prevent such ruptures by tracking "everything" herself, hypervigilantly. This is a desperate attempt to be ready to offset the predictable failure of her mother to maintain shared reality or show any caring concern for her feelings and needs. However, that solution is not a reliable preventative, so the client feels continual vulnerability and terror. Clinical experience in carrying out TRP Step B using Coherence Therapy shows that as a rule, a symptom-generating emotional learning or schema has two main sections: knowledge of a problem—namely one's vulnerability to a specific suffering, in this case the retriggered trauma of shared reality suddenly vanishing—and knowledge of a solution that is the urgently needed way of trying to avoid that suffering—in this case, hypervigilantly keeping track of everything needed to maintain shared reality.

C: That's right. I couldn't trust my mom to do it. I really couldn't. I mean she was just so, like, where she'd promise to do something, she'd be in the middle of doing it, and then she'd just get [up and say,] "Okay, I'm done," and just walk away!

T: And the shared reality dissolves right there.

C: Yeah. Then I'd say, "But you said you'd do this for me!" "Well, I'm done." And I would just be so crestfallen and cry and just, like, and be mad at her, but it didn't matter. It's always feeling like the ground is almost like quicksand. It's like it wasn't solid. I couldn't count on her to—

T: To a degree that reality disappears.

C: Right, exactly, yeah.

T: The whole framework just—and you're just floating.

C: Exactly.

T: Ungrounded. You're like that astronaut in the black void.

C: Yeah, exactly. Yeah, that's exactly—

T: And disconnected!

C: Disconnected. Yes, right. That's it. That's exactly it. Yeah.

T: Wow. Very intense.

C: And I think when somebody gets angry or criticizes, it sort of like it's kind of like it comes as like this, sort of like a jolt or a shock.

T: Is that also a reality disconnect?

C: Yes, yeah, because it catches me by surprise. You know, like the surprise element of it whenever like I'm caught by, like, surprise. I'm not expecting it. There's a sense of being blindsided. Then it's like that jolt, and then it's back into that, like, I'm terrified of that darkness, of that emptiness.

…

T: Yeah, I feel I get it. Um, let's see. Yeah, we started on this—you were wondering, did it—we were facing the mystery of, why am I always terrified? What's always endangering me? Is this it?

C: Yeah, this is it.

T: That with every person—especially important people, but maybe with *any* people—you've learned in life that it happens without seeing it coming. It's unpredictable how suddenly the fabric of reality is ripped, and there's a gap that's black infinite void, and you're goin' in.

C: That's right.

T: And it can happen with anybody, because human communication is like that.

C: That's right, yeah.

The therapist has been confirming that what has been retrieved and verbalized is actually felt by the client herself as being the source and the emotional truth of her chronic terror. This explicit confirming is important for two reasons: First, the therapist must have an accurate understanding of the client's symptom-generating schema in order to find next how it can be disconfirmed, unlearned and nullified. Only by attending closely to the specific elements of a client's symptom-generating emotional learning can a therapist reliably facilitate the empirically established process of reactivation, mismatch and counter-learning that yield erasure and transformational change. Second, engaging the client in a close review of her

newly retrieved schema promotes integration of that material. That is needed because a newly conscious emotional schema is not yet firmly anchored into conscious awareness and can easily be lost. Integration makes the schema maximally available for the disconfirmation and erasure process. Coherence Therapy follows discovery work with an active cultivation of integration of the newly conscious material through repeated mindful experiences of it.

…

T: What's coming back to me right now here at the end of the session is the moments when you were in it back with Mom, here—that little girl, desperate: "But you said it, don't you remember?" Just *struggling* to stay out of falling off the edge of that cliff.

…

C: That's right.

T: And having reality dissolve out from *under* you! That plunging feeling.

C: Right, yeah, that's right, yeah. Like in a free-fall, or just suddenly like in a free float.

T: Yes. And it's terrifying.

C: Yeah!

T: You're helpless and so alone.

C: That's right.

T: And it feels like it'll last forever. There's *no way out*.

C: Yeah, exactly, yeah.

T: And the terror of that is what is the danger you feel in daily life now.

C: Yeah, that's right. Yeah, I'm always scared of going into that.

As is often the case with traumatic memory, there are two distinct terrors plaguing this client: There is the primary terror that she feels when her traumatic memory is actually retriggered by someone losing track of shared reality and plunging her into the dark, no-exit void; and there is the secondary terror that she feels in daily life in anticipation of that terrifying plunge happening again at any time. That secondary terror was learned from having numerous experiences of the primary terror, and is often termed fear of fear. That anticipatory fear of fear generates hypervigilance. She is describing that secondary terror when she says, "Yeah, I'm always scared of going into that." The session has been almost entirely devoted to TRP Step B and has made significant progress retrieving the terror-generating emotional learning into awareness and verbalization.

T: Yes. Yes, okay, good, we've found what that terror, what that danger is made of.

C: Wow. This is so—I've been wondering about this my whole life, for a lot of years.

…

T: Good, good. All right, so how about if we set you up for being between sessions with the awareness of this? Let's create a card. …

The card, which is reproduced below, is a device used in Coherence Therapy to sustain the in-session process between sessions. In this instance, the card consists of the key verbalizations from the session, capturing the schema as revealed so far. The purpose of this card is to keep promoting integration of the retrieved material simply by returning the client to mindful, affective experience and awareness of the material daily. The card is written collaboratively by therapist and client, with the therapist relying on the client's felt sense of the wording that most accurately captures her emotional truth. The card was emailed in this case, but some clients prefer a physical index card.

BETWEEN-SESSION CARD

There is always the danger that Burt or anyone could suddenly be angry or critical or displeased with me, or forget what one of us has said to the other—and that would mean that our shared reality is disintegrating and falling apart before my very eyes, just like it did again and again with Mom, and then I'll be plunged again into that terrifying darkness, where I don't exist for them and I don't exist for me, and I'm totally alone and stuck there forever with no way out. So it's urgent for me to stay totally alert to everything that's going on, so that I can keep my grip on the shared fabric of reality, keep it from ripping apart and not get plunged into that terror.

NEXT SESSION, ONE WEEK LATER

VIDEO SEGMENT 2: http://www.coherencetherapy.org/videos-pwd/articlevideo/2.htm

T: I am really interested in hearing about how the card was for you, but we can start wherever you actually—

C: No, no, I want to do that, because I've been reading it a bunch during the week, and it's been really—I mean, this is, this is, ah—it so fits. What's interesting though is, just as I was coming in, I just had this realization that this whole piece about being in danger isn't just about my mom, it's also about my dad. It's almost like [it] started with this like sense of this shared reality piece of that being—you know, becoming, feeling so like fragile like with him, too. It was somewhere between two and three, because—I'll share this and I'll just come back to this. I remember being a little girl and getting up in the morning, and I think taking off my clothes, not knowing how to change myself or anything, [and] climbing in bed with my parents, to snuggle. And one morning I went in, just felt like this is my thing to do, like I get to go in and snuggle with them for a little bit. And I walked into the bedroom, and my dad just leapt up and said, "Get the hell out of here, and don't ever come back. You wait outside and be quiet till I come and get you."

T: Don't ever come back?

C: Yeah.

T: How did that feel?

C: Just like heartbroken.

…

C: It was such a shocker, you know, like here I thought—

T: This was yours to have. It's like a right—so unquestioned.

C: Unquestioned.

T: And this rejection: "Don't ever come back."

C: "Don't ever come back. You sit out there and wait. You be quiet and you wait until I come and get you, and don't come in here ever again."

…

T: So that's a heartbreak blow.

C: Yeah. I think that's part of that darkness, that slipping into that, what I kept calling it over the week, the dark hole of nonexistence, the *black* hole of nonexistence.

T: That's what you've been calling it?

C: [With emotion.] Yeah, yeah, yeah, that's what came to me—this terrifying darkness and it just kept coming up: black hole of nonexistence. And that just kept coming up. So it's like I lost it with my dad, and then I lost it with my mom too! Or I'd think I would have it with her, and then I'd lose it; I'd think I would have it with her, and then I'd lose it.

TRP Step B has continued with the retrieval of episodic memory of her father suddenly shattering her emotional and physical connection with both parents by ragefully casting her out of the parental bed into what she calls "this terrifying darkness" and "the black hole of nonexistence." Feeling extreme unsafety and fragility of attachment with both parents was a continuous state of terrifying vulnerability throughout her childhood. Her attachment to both parents is of the insecure/disorganized type: the parents are needed for security but are experienced by the child as a dangerous source of arbitrary aggression because they behave in unpredictable, extreme and punitive ways, arousing severe distress in the child but not providing any comfort or help. The child is helpless and defenseless in the face of repetitive abuse and harm, and therefore chronically feels disoriented, frozen or terrorized, as the client here has described in this and the previous session. Both her episodic and semantic memory carry vivid knowledge of abruptly being cut off from all sense of intersubjective connection and plunged into a black hole of nonexistence, a state that she re-experiences in response to any current interpersonal misattunement or negativity. Knowing she is ever vulnerable to that plunge is what maintains the terror she identified as the problem.

T: Yes, yes. And this one you're telling me about now—the black hole of nonexistence—is *so* powerful. You thought you had the shared reality of that cuddly coziness together in the bed!

C: Yeah, yeah I did!

T: And that was annihilated so suddenly.

C: Yeah, exactly.

T: So, I see why there's the black hole opening up.

C: That's right.

T: What you thought was shared reality suddenly is gone, annihilated; isn't—

C: Yeah, it's just like, it's just totally taken from me, [snaps fingers] just like that! You know, what I thought was so unreal [*sic*], isn't. [Tearful:] And then Mom would do that, you know *she* did that, all these—

T: Yes, in terms of things she said, for example.

C: Things she said or things that she promised she would do and then deny that she promised it.

T: Promises!

C: Yeah, she would say, "I'll do this for you," and she'd go, "No, I didn't. I never said that." "No, no, you said you would do that for me!" So, there was sense of like she's there and then no, she's *not* there.

T: Yes, and the sense that she sees you, and remembers and cares about what matters to you, and then, oh no, she doesn't.

C: "No, I don't. I never said it. I don't remember that."

…

C: [Through tears:] So much of my life has been organized around this, Bruce. So, many choices—*bad* choices, you know. Just so *much* about—just like all that being deferential, like try to care—create a shared reality with someone.

T: That's been priority one, two, and three, instead of what's really right for you.

C: Right, exactly. And being able to like—so afraid to like, find out—you know, what if I find out that your reality doesn't match mine? Like, scared to do that.

…

C: It would shatter at a moment's notice!

T: Yes, so, naturally, you're anticipating that, and vigilant for that. Is it like bracing yourself for it?

C: A bit that too, yeah. You know, there's vigilance, there's checking, but there's a bracing too, yeah. I can *feel* all the tension in my body from it. Like almost—all through here, in my shoulders, everything. It's almost like in this kind of like, you know, a vigilant threat response, like walking around like that. I can literally *feel* it. ...

To foster integration of the emerging explicit knowledge of what she suffered, the therapist has been prompting her into repeated verbalizations of it as she is affectively feeling it. Additional somatic aspects have now also been felt and identified, making the accessing of her implicit knowledge even more thorough. To the therapist, the Step B retrieval work now seems adequately accomplished because the emotional learning underlying the symptom of terror is explicit in some detail and there remains no mystery as to why terror arises when it does. The retrieved schema consists of specific knowings or constructs that may be verbalized as follows:

TARGET EMOTIONAL SCHEMA

[The learned problem:] No one maintains shared reality. When anyone loses track of the understandings we shared, it means:

- Shared reality is irretrievably gone.
- I don't matter and don't exist to him or her.
- No shared reality exists with anyone.
- I'm plunged into a terrifying vast darkness of being utterly cut off and alone in the universe, and there is no way out.

  That can and will happen again at any time, and that is perpetually terrifying!

  [The learned solution:] I've got to stay totally alert to everything that's happening and remember everything so that I can keep shared reality stitched together when anyone would let it rip apart. I've got to keep my body tight and braced for the next time anyone rips apart shared reality and I plunge into the black hole of nonexistence.

All of that material consists of semantic memory, that is, general patterns and rules that were formed on the basis of concrete experiences but do not refer to those formative experiences in their operation. Here again, as in the previous case example in Section 7.1, post-traumatic symptoms are found in TRP Step B to be arising from semantic memory rather than episodic memory. The above detailed contents of the schema are the therapist's crucial guide in the next step, TRP Step C, the search for specific contrary knowledge that can then be used in TRP Steps 1–2–3 (the ECPE) to disconfirm, nullify and erase some key part of the schema. In Step C, finding contrary knowledge requires knowing exactly what is to be contradicted from Step B: the specific constructs in the target schema. For Step C, Coherence Therapy instructs the therapist to search for disconfirmation (contrary knowledge) in the client's past experience (that is, in knowledge that she already possesses, without

realizing it), or in some experience that has occurred in daily life since the target schema came into awareness, or in a new experience that the therapist deliberately creates. The therapist's attention and thinking are now engaged in that search.

SESSION CONTINUES

VIDEO SEGMENT 3: http://www.coherencetherapy.org/videos-pwd/articlevideo/3.htm

…

C: It came up this morning with my boyfriend. …I said, "Wait a minute! We talked for an *hour*. And we got into some pretty personal stuff in that hour! I was sharing about my work with Bruce and you were sharing about something that you were realizing about you. What about *that*?" Now there's that, "Wait a minute, where is this reality that we just shared?"

T: Very good!

C: But I started crying, you know, like I was that piece about—but I stayed—I said, "What *about* this? You know, it's like, you just *erase* that?" … You know, here it is again. I'm crying and feeling like once again, I don't exist. …

Her account of the morning's incident with her boyfriend shows that the target schema has remained in force even though it was retrieved into awareness in the previous session. The therapist has noticed that her account has stopped at her collapse into non-existence, and he is now wondering about what had actually happened next. Did her boyfriend indeed fulfill her schema's expectations that shared reality was irretrievably lost and that he would be deaf to her protestations? Or did his ensuing behavior violate those expectations, which might serve as a disconfirmation? To probe that possibility, the therapist next asked her the following question:

T: How did it go with him?

C: Well, he said, "Oh yeah, you're right." He said we—"I guess I dismissed that."
I said, "Yeah!" I said, "What *about* that?" I said, "I'm really hurt that you just *erased* that." And then—

T: Perfect, great!

C: —he goes, "Okay, I forgot it. You're right, okay."

It is now clear that her boyfriend's behaviors differed fundamentally from her schema's expectations. This completes TRP Step C, the task of finding an experience that can be used as a contradiction and disconfirmation of the target learning. The client is not yet recognizing her boyfriend's response as a disconfirmation of the expectations of her traumatic memory, even though she is describing his behavior in detail. The therapist must now guide her to register her boyfriend's cooperative, respectful response to her protests as being a direct contradiction of her schema's expectations. Holding both the schema's expectations and the contradictory experience in awareness concurrently, in order for the disconfirmation to register viscerally, is the juxtaposition experience (a Coherence Therapy term) that implements TRP Steps 1 and 2, and a few repetitions of the juxtaposition fulfill Step 3, completing the ECPE. The therapist is now aiming to facilitate that sequence.

T: All right. So, right now, I want you to, as you revisit it—how that went, and what he said, and your *success* in calling his attention to what he had lost. Yes, he *did* lose track of shared reality, but this time, you prompted him to retrieve it, and restore it, and even acknowledge to you, "Yes, I lost track of something important between us and I've got it back."

C: He said, "Oh yeah, I guess I did. I forgot." I minimized that.

T: See if you can let those words be *nectar* in your life. Those words from him could be gold, could be nectar. It's the other person holding up their end of reassembling shared reality when they fumble it. What's more important to you than *that*?

C: Right. He's done that *many* times with me.

T: And maybe it has flown by. …

C: Yeah, yeah. I think because also I've been so troubled about how intense my reaction was *to* his forgetting, you know, or like—and how, like—

T: You were focused on that.

C: That, and just, like, the *hurt* and like, the *heartbreak*, you know. Like, how could you *forget* about me? …

T: There's a lot of intense components that were in play for you when this was happening. And so, your attention naturally was on those things. So, right now, though, selectively I'm prompting you, inviting you, to—here's an opportunity to focus on

this *non-helplessness* you experienced. Your success, your actual ability to get him to retrieve the piece of shared reality that he had lost track of, and put it back in place and have it be shared again, and in addition, acknowledge to you and even *apologize* for losing track of it. See if you can let that—let's see, on the one side you're holding that. On the other side is your old lifelong expectation that that will not happen—you'll be all alone, stranded and helplessly stuck in that black hole once the shared reality is dropped.

C: Right.

T: So, see if you can, you know, feel *both* of those.

C: I guess what I'm doing right now is just going—thinking about all the other times he's done this too with me. I mean, he's done this a lot.

The therapist has just now explicitly cued the juxtaposition experience, but the client has not yet directed her attention to the both-at-once experience.

…

T: Yeah, let that really sink in now—all those past ones, as well as this fresh one—how it's not like it was back then [with her parents]. You're with someone who doesn't irreversibly drop out of shared reality. You can express it and he'll retrieve it and reassemble it together and even acknowledge that, and you're not helplessly stranded when it does happen.

C: Ok, yeah, that's the part, about not being helplessly stranded.

T: …Wow, all along I *have* been helplessly stranded, and I've been expecting to be again and that's why it's terrifying. But, wait a minute. With him I'm *not* helplessly stranded.

C: No, I'm not. No, I'm not! I'm not helplessly stranded. Yeah, that's a key piece. I'm not helplessly stranded. And, he will do this *with* me.

T: Yes.

The client is now having her first juxtaposition experience, in which the emotional schema that generates her terror ("I'm helplessly stranded in total disconnection") is present in awareness concurrently with strongly contradictory, disconfirming knowledge ("I'm able to get him to reconnect with me, so I'm not helplessly stranded"). The tonality of her voice in her latest utterances is different than at any point in this or the previous session, and expresses both her wonderment at this surprising realization of not being helplessly stranded and the depth of meaning this has for her. Feeling helpless is what makes an encounter with danger so frightening as to create traumatic memory, as a rule. The memory then includes that helplessness as an expectation: "Whenever situation X occurs, I'm helplessly endangered!" That expectation in turn produces panic and/or dissociated freezing. Disconfirmation of the expected helplessness is therefore usually the most effective juxtaposition experience for unlearning and nullifying traumatic memory and post-traumatic symptoms.

C: I mean— [Silence.]

T: Yes. [Long silence.]

C: Wow.

T: What's that?

C: Well, just that piece, because that's definitely been in there about just feeling helplessly stranded. Yeah. Like there was nothing I could *do* about it. Back then, I *couldn't*.

T: Right.

C: Even with all my efforts, it didn't make any difference.

T: You *were* helplessly stranded back then.

C: I *was* helplessly stranded back then.

T: You felt the floor falling away. I mean, your *body* felt the falling away of shared reality.

C: Yeah, and there was no way to get it back.

T: No way. There was no way.

C: There was just—my mother wouldn't do it—

T: It takes two. It takes two to get it back.

C: It takes two, and my mom wouldn't do it with me and my dad wouldn't do it with me.

T: They didn't have those skills.

C: Yeah, they didn't.

T: But he does.

C: Yeah. But Burt does, yeah. It's interesting how many people I've been with who have not had that skill.

T: Mm-hm. And for you, that's just huge—

C: It's been so critical.

T: —huge, huge. Yes.

C: Didn't have it, or didn't want to do it, or whatever.

T: Yeah, whatever. So, yeah, you're letting it sink in, in this new way. There was an "oh wow" there.

C: Yeah. Wow!

T: Yes, yes.

The therapist has continued prompting her to recognize how "helplessly stranded" in shattered rapport she truly was again and again throughout her childhood, and in later relationships as well. The purpose of this is to then have her freshly re-encounter the new disconfirmation of that expectation of helplessness, creating repetitions of the juxtaposition experience for TRP Step 3. The therapist guided her back into the juxtaposition by saying, "But he [Burt] does." There will be several more repetitions of the juxtaposition in the rest of this session. In response to the client's various indications that the disconfirmation has been registering strongly, the therapist will now begin TRP Step V, making enquiries that probe for markers of erasure.

T: How does it feel in your body?

C: It feels like there's a— I just feel really softer, kind of like I'm aware of my breathing and just letting go tension, or something like a little spacy. There's sort of a melting quality, and there's, like, "ahhh, ahhh."

T: Softening.

C: Softening, yeah, this softening. Wow. Wow. And I really *was* helpless back then, and they *didn't* have the skill to retrieve it. But Burt does, and he's done it repeatedly with me. And *I'm not helpless*. Those are the key pieces. You know, that there are people in the world that *do* have the skill to do that with me.

T: Yes, that's it.

C: That's a piece. Then that there *are* people in the world that have the skill to re-establish shared reality—I guess that's a way of saying it. And, *I'm not helpless*.

T: Yeah, yeah, let that sink in.

C: I'm not helpless, yeah.

The client's description of somatic softening and opening, in contrast to the tightness and bracing described earlier, possibly could be early indications that the schema has been unlearned and nullified in the last few minutes, changing her model of the world. Also, an additional contradictory knowing emerged: In revisiting her amazed realization about her boyfriend, she realized further that he is not a unique anomaly, but is representative of a class of people who strive to maintain shared reality. This recognition is the most potent schema-disconfirming new knowing yet experienced, so the therapist will actively keep incorporating this new knowing into the juxtaposition as the session continues.

T: Mm-hm. When you're with a person who does have the skills and the willingness or cooperativeness to do that, you are not helpless about the black hole. It doesn't even have to develop into a *black* hole.

C: That's right. …There are people in the world who will restore that with me and I can do that with them, and I have the *capacity* to do that and *will* do that, so I'm not helpless. And if I encounter people that either don't have the capacity or don't want to do that, I'm not helpless with them either, because I'm not stuck. I don't have to stay in that. I can walk away. I can disengage and go, "Thank you very much. Have a merry life, but I ain't dancin' with you," or whatever. [Chuckles.]

T: I'm writing down your exact words: "I'm not helpless with them either, I'm *not* stuck."

C: I'm not stuck.

T: "I don't have to stay."

C: And I *was* stuck as a little girl. I was *so* stuck; subjected to that.

T: That's right. Couldn't go away.

C: Didn't have a choice. Wow. I mean, it's so obvious, but I'm just putting it—

T: It's obvious to one part, but there are other parts that needed to know this.

C: And just to be acknowledged: yeah, really, I *was* helpless as that little girl, and there *wasn't* any real way out of it. I would come out of that experience, but it was always there to go into again. I had no control over that, because I was a kid dependent on my parents.

T: Yeah, totally. Yeah.

C: Wow, this is so big. [Chuckles.] This is so big.

T: Good. Feel it, feel it. Let it course through you. Yes.

C: Wow. I just feel my whole body going, "Yes, yes, yes." I mean I'm aware of this spontaneous nodding that I'm doing.

T: Good. [Silence.]

C: [Big exhale.] Actually, I'm really feeling my feet, too. Because this week when I was at one point really experiencing the terror, I was very aware of, "Oh my God, I don't even feel my feet." It's almost like I don't even have—I couldn't even stand, it was that kind of quality to it. And I was just very aware of, oh, I can actually feel my feet.

T: I can see you're wiggling them and rocking them.

C: Feel my legs, my—kind of like my capacity, my adultness.

T: Well, you're *in* your *body*.

C: I'm in my body.

T: You're not *fleeing* your body—

C: Yeah, right.

T: —in terror—

C: Exactly.

T: —because it's plunging through black space!

C: Right. [Laughs.] Yes, right. Yeah, yeah, and also this kind of sense of like I'm not helpless, like, I can stand up for myself.

T: Yes, you've got your feet under you.

C: Yeah, I've got my feet under me. I've got my ground. I literally can *stand* for myself, *and* I can walk away, you know.

T: Yes, both.

C: I can move, move. Yeah.

She has re-encountered the juxtaposition several more times and her revised experience of the world is persisting. More somatic markers of fundamental change have emerged, all bodily expressions of feeling empowered rather than helpless in response to the situation of someone failing to maintain attunement with her. She feels freed from the grip of a severely traumatized state of mind. A terrifying, ever-present danger in her world seems to have disappeared. Someone dropping shared reality now means, "Oh, this person doesn't see my existence, but there are other people who do and who will continue to see my existence." Consequently there is no black hole to fall into, so her anticipatory terror of the black hole disappears. Her response to a lapse of attunement would be fundamentally different because her model of the world of people has fundamentally changed. If these shifts were to persist effortlessly even when Burt and others fail to maintain shared reality with her, TRP Step V would be fulfilled and erasure would be verified.

C: …I want to be with those people that *have* this capacity to re-establish shared reality.

T: *Those* are your people.

C: Those are my people, yeah. Yeah, those are my people. [Silence.] Yeah. [Silence.]

…

T: The people of the shared reality.

C: The people of the shared reality. [Laughs.] Yeah, that I can fall back on in that circumstance like a *safety net* of sorts, you know? That's what it feels like, rather than just being all alone in the world and all I have is *this*. And so if I turn away from *this* I have *nothing*. Yeah, you're right, I don't have that now. I don't—it isn't that I have nothing. I have the people of the shared reality to turn to. I'm not alone! Yeah, that is so—yeah! I'm really glad you mentioned that, 'cause that's a piece of this too. A *really* big piece. …

To keep making the disconfirming juxtaposition as potent as possible, the therapist has continued to cultivate her new knowing that a subset of human beings actually cares about maintaining shared reality and has the skills to restore shared reality when it has been lost. In commenting, "Those are your people" and in designating them as "The people of the shared reality," the therapist has been further developing her basic recognition that such people exist, by inviting her into regarding those people as her own sacred community in which she intrinsically is a full, permanent member. In relation to those people, she always exists and is always in the web of connection, because they maintain shared reality. Her immediate response to that was a felt sense of having what she termed a "safety net." That phrase expresses her bodily feeling of now being protected from falling into the black hole. That feeling and that phrase will grow into a bedrock of security as the session continues.

Knowing she has the people of the shared reality as her safety net is the fully contradictory knowing that disconfirms, unlearns and nullifies the schema that had been generating her terror.

C: It feels like that—not helplessness, not stranded, not desolate, stuck, all that. That's the key piece—and then this thing that there are people in the world. You know that literally feels like a safety net. It really does! I never *had* one!

T: Never!

C: Ever!

C: So I would always feel like—

T: There *was no safety net*!

C: There was no safety net.

T: You were only as safe as whether the person you were interacting with is going to stay in shared reality.

C: That's right.

T: You were *so vulnerable*—

C: I was.

T: —*all the time*, as I understand it.

C: Yeah, all the time.

T: All the time!

C: Just like how— This is like— In the sense of, like, relief about: oh my God, it's over. It's really over.

T: Yes. Ah.

C: It's really, really over. Wow. [Chuckles through her tears.]

T: It's almost—it's like mythic.

C: It is!

T: How big it is.

C: It is. It is, that's exactly it.

T: It's like the darkness is off of the land.

C: Yes, right.

T: It's over.

C: For the first time in my life, having the sense of feeling like I have a safety net!
I mean that's *huge*.

T: It's hard to find words big enough to capture how huge it is.

C: Yeah, yeah. And I don't have to be—it's not all up to *me* to be the safety net.
I have my capacity and I'm not helpless, but there's a bigger holding for me.

T: Yes, yes. The people of shared reality are your safety net. And if a person isn't one of them, okay.

C: Yeah, that's okay. But there are people who are, that's my safety net. They're my safety net.

T: Yes.

C: Yeah.

T: Yes.

Next, therapist and client collaboratively wrote a card that would keep her in touch with these crucial felt knowings between sessions. The card, shown below, uses her own phrases to capture and maintain the juxtaposition experience created in the session. The first paragraph expresses her life-long, terror-generating emotional learning that she is totally and forever alone because no one will stay in shared reality. The second paragraph juxtaposes and disconfirms that with her own living knowledge that there are people who do maintain shared reality, enabling her to walk away securely from people who do not maintain it.

BETWEEN-SESSION CARD

All along I've felt helpless and powerless and totally alone as soon as anyone drops out of shared reality, stranding me in the black hole of nonexistence.

What a surprise that I now have a safety net and I'm *not* helpless or totally alone: I can *walk away* from people who don't stay in shared reality. I can walk away because there are people who *do* value shared reality and restore it when gaps occur. So I'm not stuck like I was as a little girl with no way out. People who care about tracking and maintaining shared reality are my people. With them I can exist and stand for myself and get them to restore shared reality, like Burt does.

All my life I've been tiptoeing around on thin ice above a black hole.
It's finally over.

SUBSEQUENT SESSIONS 0.5, 3.5, AND 11 MONTHS LATER

VIDEO SEGMENT 4: http://www.coherencetherapy.org/videos-pwd/articlevideo/4.htm

In the next session two weeks later, TRP Step V, verification of markers of erasure, was fulfilled to a significant degree when the client reported complete absence of both her hypervigilant terror in general and the somatic downward plunge sensation in response to recent sudden ruptures of shared reality with her boyfriend. (See video for that report from client; it is not included in transcript here.) Other symptoms of complex attachment trauma now became the focus of therapy. In a session three months later (see video), the therapist followed up on Step V for the terror and somatic plunge. The client cited recent sharp conflicts with her boyfriend, which previously would certainly have triggered the plunge into the black hole state, but now did not do so. About those conflicts she said:

C: And it bugs me. It's a little disconcerting, disorienting in a certain sense, but nothing that's debilitating, or that I'm scared that the floor is falling out from under me, or like I'm dropping into that elevator like before, or like the elevator's just going *kthoom*.

T: Yeah, the plunge.

C: Yeah, that plunge, like, quick drop, *kthoom*.

T: So is that gone?

C: Yeah, that feels *gone*. That really does.

T: All right. It sounds like you're feeling the sensible experience of a loss of connection, but it's not at all in that zone anymore.

C: Right, yeah, right.

T: So is that a zero? It's a zero plunge, zero black hole.

C: Yeah, I would say a zero. Yeah, I would say a zero.

In a session 7.5 months later (11 months after the ECPE session of juxtaposition experiences), the therapist again pursued Step V for the anticipatory terror and the plunge into the black hole, and again the client reported the effortless and complete absence of those symptoms under all circumstances:

T: I got your email…and you mentioned—well, there's a lot of important pieces here. One of them I'll just mention now is how you said, even though things were rocky and shaky between you [and your boyfriend], you said "although I feel some anxiety and fear and confusion about what happened this morning, I am not feeling like I am in danger of going into stark/dark aloneness or the sense that I have no shared reality."

C: Right. Yeah. So that's like—

T: That's holding.

C: Yeah, that's really holding; because—that's so big for me. You know, it's like…the floor was going to come out from under me, that kind of—and that dropping sensation that would come with that. And I don't—that doesn't—that's not coming up. I'm mean, I'm still kind of like troubled or preoccupied, but it isn't, like, umm, this, you know, *terror* that I would feel before.

T: Wonderful.

C: Yeah, yeah.

Two years after the ECPE session, the client again reported absence of the anticipatory terror and the plunge into the black hole in a very recent situation which, she said, definitely would have triggered them previously.

The two sessions of TRP facilitation described here were a small subunit within a far more extensive course of therapy addressing the client's complex attachment trauma, which comprised numerous other symptoms and their underlying emotional learnings. For each symptom identified by the client, the therapist carried out the TRP, in that way progressively erasing schema after schema, resolving emotional issues and ending symptoms.

7.3. Are the observed transformational changes due to reconsolidation?

The proposal that reconsolidation and erasure have been demonstrated in the foregoing two case examples (and in many other published case studies, such as those listed online at http://bit.ly/2tKXdyX and http://bit.ly/15Z00HQ) is supported by the following considerations:

First, as noted in Section 3, the observed effortless permanence of non-reactivation of schema and non-expression of symptoms are the same markers used by neuroscientists to confirm erasure via recon-

solidation in human studies (e.g., Schiller et al., 2010), based upon animal studies that linked the same behavioral markers to rigorous neurobiological proof of memory destabilization via the pharmacological blockade test (e.g., Monfils et al., 2009).

Second, the markers do not occur in therapy until the requisite sequence of experiences (the ECPE) occurs, and then are manifested by the client immediately.

Third, the changes observed clinically cannot be explained by other known types of neuroplasticity, that is, by a type of learning different from behavioral updating via reconsolidation. Regulation (inhibition) of an existing, acquired emotional response can be developed through various other types of learning, all of which are competitive in nature (e.g., Ochsner and Gross, 2005; Toomey and Ecker, 2009). However, none of those processes of inhibition by competitive learning have been shown capable of the decisive, lasting changes described in the examples detailed in Sections 7.1 and 7.2. In those examples, the first juxtaposition experience, TRP Step 2, was followed in a matter of seconds by the client experiencing and describing complete loss of the compelling subjective realness and motivating power of an emotional schema that had dominated life for decades, and neither the schema nor the symptoms it had been driving were ever again reactivated under any circumstances. The case in Section 7.2 involved post-traumatic, hypervigilant terror, a symptom that is deeply rooted in the subcortical brain and is widely regarded by clinicians as being tenaciously treatment resistant. To the author's knowledge, there is no process or mechanism of competitive learning known to science that is capable of producing those effects, including recent research on enhanced extinction protocols (Dunsmoor et al., 2015). That is strong support for the hypothesis that the observed changes were not due to competitive learning causing inhibition of target schema reactivation. The only viable explanation appears to be erasure via reconsolidation.

The circumstantial evidence delineated above is substantial, and, given that there currently exist no direct, neurological detection and proof of reconsolidation in humans, it is the highest standard of verification currently available.

7.4. Clinical feedback on researchers' anticipated translation difficulties

Reconsolidation research has seen explosive growth since its launch 20 years ago. Quite recently, laboratory researchers' review articles have focused to an unprecedented degree on clinical application (e.g., Beckers and Kindt, 2017; Dunbar and Taylor, 2017; Elsey and Kindt, 2017a; Krawczyk et al., 2017; Kroes et al., 2015; Nader et al., 2014; Treanor et al., 2017). One of the topics addressed in these reviews is the possibility of inherent obstacles to the translation of research findings into clinical application. This section responds to the main concerns identified by laboratory researchers by reporting clinical observations regarding whether those concerns materialize, in hope of providing researchers with useful feedback that helps to clarify and expedite the translation process.

Here it may be worth repeating the opinion of Elsey and Kindt (2017a, p. 115) that "there are significant limitations to experimental research, and ultimately only attempts at treatment can reveal the utility of a reconsolidation-based approach." One of main purposes of this article is to report back from the clinical trenches about more than 25 years of "attempts at treatment" in which the ECPE/TRP methodology was deployed in several thousand cases by the author and colleagues plus numerous experienced clinicians worldwide (the methodology having been developed, based on clinical observations, almost a decade prior to the laboratory discovery of memory reconsolidation, as noted in Section 3).

The observations summarized below reference the case examples in Sections 7.1 and 7.2 for illustration, but it should be borne in mind that, as noted earlier, those two cases are representative of a large array of well documented clinical cases of ECPE/TRP methodology listed online at http://bit.ly/2tKXdyX and http://bit.ly/15Z00HQ.

7.4.1. Will mismatch experiences be too exacting for clinicians to create reliably?

As discussed in Section 4, for the reconsolidation process to be triggered, the post-reactivation experience has to be similar to the original learning, but not identical to it. For a target learning acquired through Pavlovian-type associative conditioning, small varia-

tions in laboratory post-reactivation procedures can have the large effect of determining whether or not the target learning is left in a destabilized condition (Alfei et al., 2015; López et al., 2016; Jarome et al., 2012; Merlo et al., 2014; Sevenster et al., 2013, 2014; Schroyens et al., 2017). The failure to destabilize has been found to be due to the post-reactivation procedure creating either too little or no prediction error or too great a dissimilarity from the target learning. For a given acquisition training, a given type of post-reactivation procedure has to satisfy boundary conditions, a limited range of parameters that produce destabilization.

Researchers have therefore concluded it is a delicate matter to design a post-reactivation procedure that creates the degree of prediction error that serves as a memory mismatch experience and destabilizes the target learning. That conclusion in turn implies that clinical translation of research into effective clinical application may be elusive (reviewed by, e.g., Beckers and Kindt (2017); Treanor et al, 2017). For example, Sevenster et al. (2014, p. 583) concluded, "Even though the fear reducing effects are very robust and promising for the development of reconsolidation-based treatments, the success of the manipulation depends on subtle differences in the reactivation procedure. This poses a real challenge for clinical practice, which can be resolved by careful selection of the reactivation parameters." Likewise, Beckers and Kindt (2017, p. 111) observed, "The transition from insufficient prediction error to sufficient prediction error to excessive prediction error for inducing memory destabilization may be quite subtle…. That only a narrow degree of expectancy violation allows for the induction of amnesia upon memory retrieval greatly limits the translation into effective clinical interventions. It remains to be seen whether it will be feasible to establish, in a principled way, how to elicit an optimal level of expectancy violation during memory retrieval in the individualized context of clinical treatment."

Those somewhat pessimistic anticipations are fully warranted on the basis of laboratory results. However, in the two illustrative clinical cases detailed in Sections 7.1 and 7.2, there is no sign of difficulties achieving a destabilizing mismatch with the first juxtaposition experience (TRP Steps 1 and 2), as indicated by prompt and lasting appearance of decisive markers of erasure of the target schema after a few repetitions of that disconfirming juxtaposition (TRP Step 3) for the counter-learning that nullifies the schema. Presumably, the markers of erasure could not appear if the target schema had not been destabilized. Those two illustrative cases are representative of general experience in using the ECPE/TRP methodology in therapy: The foreseen delicacy of creating just the right degree of mismatch that destabilizes, allowing erasure to ensue, is not observed. Emotional learnings retrieved in therapy are found to be wide targets for a destabilizing mismatch, not narrow targets, via ECPE/TRP methodology. Two factors, taken together, may explain that clinical good news:

First, the delicacy of mismatch observed in laboratory studies is a direct effect of the artificially precise structure of the target learnings created by researchers, not an inherent feature of the reconsolidation process that would necessarily carry over into the clinical setting. As explained in Section 4, the detailed structure and content of the target learning determine the post-reactivation experiences that will, or will not, mismatch and destabilize the target learning. Thus the finding that Pavlovian target learnings are mismatched and destabilized by post-reactivation procedures that may vary within only a narrow range of parameters is entirely due to the precisely ordered structure of Pavlovian target learnings designed by researchers. In other words, laboratory studies have had to create mismatch of subjects' episodic memory of the acquisition training, which involves all details of that training in the mismatch. In contrast, clinical experience has shown that as a rule, generalized semantic schemas, not episodic memories, emerge as the emotional learning driving symptom production, as illustrated by the case examples in Sections 7.1 and 7.2. Even when the focus of therapy is on episodic memory, what emerges are the semantic learnings that were operative at the time. Reactivation of such schemas in therapy does not require any reference to episodic memory (also observed in a laboratory study by Soeter and Kindt, 2015b), and mismatching such schemas is a qualitative matter, not a quantitative matter, which eliminates the need for parameter precision in forming mismatches. For example, no careful adjustment is needed on duration of reactivation of a schema that is being mismatched by a contradictory knowing, whereas for mismatching a Pavlovian conditioning memory, duration of reactivation is a critically sensitive parameter (e.g., Alfei et al., 2015; reviewed by Treanor et al., 2017).

Second, while the TRP does not dictate any particular behavioral procedure, it does dictate creation of a type of mismatch that has been shown in numerous laboratory studies to be effective for destabilizing target learnings. This mismatch consists of an absolute or ontological contradiction and disconfirmation of the target learning. In laboratory studies, absolute contradiction of a 100% reinforcement, asymptotic, Pavlovian target learning is created by a single non-reinforcement trial (CS–noUS) (e.g., Pedreira et al., 2004; Alfei et al., 2015), which is well established as a destabilizing mismatch. As discussed in Section 6.2, what matters to the subject's brain is the *experience* of absolute contradiction of expectation, not the CS–noUS procedure that created that experience. The TRP creates mismatch consisting of absolute contradiction by requiring the therapist to elicit the detailed contents of the symptom-generating schema (in TRP Step B) and then find how the client can have an experience that specifically contradicts that schema's model of reality (in TRP Step C). That is followed in TRP Steps 1 and 2 by pairing those two experiences, creating the mismatch, or juxtaposition experience, in which both knowings or models of reality are simultaneously present but both cannot be true. Destabilizing mismatch is in that way reliably built into the TRP. Use of an ontologically contradictory experience for mismatch appears to solve the problem of boundary conditions in clinical work. Absolute contradiction has direct, strong relevance to the target learning and clearly requires an updating of the target learning, so destabilization is triggered. Whether implementation is successful depends on the skill of the individual clinician, not on the inherent properties of the methodology or the reconsolidation process.

7.4.2. Do the age and strength of target learnings in therapy cause mismatch difficulties?

In clinical practice, the memories encountered (both episodic and semantic) are usually much older and stronger than the memories created for laboratory studies of reconsolidation. Various laboratory studies have demonstrated that both recent and old memories can be destabilized and undergo reconsolidation in animals (e.g., Debiec et al., 2002; Debiec and LeDoux, 2004) and humans (Steinfurth et al., 2014). As noted in Section 4, several studies have found that older and stronger target learnings have lower susceptibility to destabilization and require stronger reactivation and mismatch for destabilization to occur (e.g., Robinson and Franklin, 2010; Schroyens et al., 2017; Suzuki et al, 2004; Wang et al., 2009; Winters et al. 2009; for a review see Elsey and Kindt, 2017b). Regarding some studies in which older memories did not destabilize, Lee (2009, p. 419) 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." Likewise, Elsey and Kindt (2017b) concluded from their research review that greater age and strength of memories do not inherently prevent reconsolidation, and they demonstrate that point with a single-case study in which a woman's intense, 30-year phobia of mice lastingly disappeared after reactivation via exposure to a live mouse followed by propranolol ingestion.

Ecker (2015a, pp. 12–13) has proposed that the observed effects of memory age and strength on susceptibility to destabilization are actually more fundamentally the effects of mismatch relativity for the highly structured acquisition trainings used in laboratory studies. In other words, the memory age and strength findings would be accurately interpreted only by closely examining how reactivation conditions interacted with the detailed structure of the acquisition trainings that formed the target learning in each case. This interpretation and the detailed analyses that it yields are qualitatively different from those made by laboratory researchers to date.

For example, in the study by Suzuki et al. (2004), each rat was placed in a context/CS for 2.5 min before receiving a 2-s foot shock either once or three times at 30-s intervals, and then were removed from the context/CS 30 s after the final shock. Researchers then waited 1 day, 1 week, 3 weeks or 8 weeks and tested for memory destabilization after memory reactivation by shock-free re-exposure to the context/CS of duration 1 min, 3 min or 10 min. In that way, the duration of reactivation needed to achieve destabilization was determined for target memories of varying strength and age, for a total of 24 different permutations of the parameters. Discussing only two of those permutations here will suffice for present purposes.

In the case of 1 shock, a 1-min reactivation did not destabilize, but a 3-min reactivation did destabilize for memory age of 1 day, 1 week, or 3 weeks. That

observation, as analyzed by Ecker (2015a, pp. 12–13) according to mismatch relativity, was caused by the 2.5-min pre-shock interval in the acquisition training: Each animal learned to expect that no shock occurs for 2.5 min, and in relation to that expectation, a 1-min shock-free re-exposure did not create a mismatch, but a 3-min shock-free re-exposure did mismatch that expectation, triggering destabilization. (See Section 4 for review of research on temporal mismatches.) Thus the longer 3-min re-exposure was effective not for the reason that it was a *stronger* reactivation (which is the interpretation of the study's authors), but simply because it was actually a memory mismatch.

In the case of 3 shocks, which certainly is a stronger emotional learning (being a more severe and prolonged experience of suffering), the 3-min re-exposure no longer destabilized at 1 day, 1 week, or 3 weeks, but a 10-min re-exposure did destabilize. In the mismatch relativity analysis, the fact that the stronger memory required a longer re-exposure for mismatch implies that the memory of the duration of the 2.5-min pre-shock period was altered by being followed by a full minute of repeated shocks instead of a single shock. Ecker (2015a, pp. 12–13) points out that three 2-s shocks coming every 30 s is a grueling minute that could presumably feel to a rat much longer than one minute of curiously exploring a harmless place, just as humans too experience the subjective flow of time very differently depending upon the presence or absence of pain. The 2.5-min period could have been lengthened or blurred in memory retroactively by this long, traumatic minute of shocks, such that a 3-min shock-free re-exposure did not register as a definite mismatch of expectation, but a much longer 10-min re-exposure did create a clear mismatch, achieving destabilization.

Here again, as noted throughout this article, the potential importance of considering qualities of subjective experience, as distinct from external procedures, is apparent. Attunement to how the qualities of subjective experience drive psychological dynamics is a critically important skill set for most clinicians. More broadly, however, the devaluing and disregard of subjectivity has been a pervasive influence in Western civilization, particularly in the sciences, as documented, for example, by Wallace (2000).

If the foregoing considerations based on the principle of mismatch relativity are valid, they would imply that there is no extra difficulty in creating memory mismatch due to memory strength and age, because in every case all that matters is designing the mismatch according to the unique particulars of the target learning being addressed. In that view, what appeared in laboratory research to be mismatch challenges due to greater age and strength of memories were actually mismatch relativity effects caused by the highly structured features of laboratory acquisition trainings.

That conclusion appears to be supported by the full range of clinical experience in applying the ECPE/TRP methodology, as represented by the two case studies in Sections 7.1 and 7.2. Mismatches created by that methodology, consisting of highly specific, ontological contradiction of decades-old, emotionally intense, symptom-generating semantic memory, are found to result consistently in the markers of erasure, indicating successful destabilization. The case detailed in Section 7.2 involves possibly the strongest clinical challenge posed by memory age and strength, namely, long-term, severe symptoms arising from memory formed by multiple, cumulative traumatic experiences that occurred throughout all developmental stages. In such a case, the lasting disappearance of both emotional reactivation and symptoms, immediately following ECPE implementation, would seem to be a maximally encouraging indication of the effectiveness of reconsolidation-based behavioral updating in psychotherapy.

7.4.3. Will therapeutic changes made via reconsolidation be durable?

The value of memory reconsolidation for psychotherapy would be maximal if changes induced through reconsolidation were permanent. In laboratory studies, observations of the markers of erasure have been made for at most several weeks in nearly all cases. A notable exception is the one-year confirmation obtained by Schiller et al. (2010) for the behavioral erasure of Pavlovian fear in human subjects. Those findings regarding durability are promising, but not proof of permanence. Whether erasure is permanent therefore remains an important open question from the viewpoint of researchers (e.g., Elsey and Kindt, 2017a).

The possibility of non-permanence of erasure seems indicated by a study (Ryan et al., 2015) that used pharmacological blockade to erase a fear response in mice and then reactivated the erased response by

optogenetically stimulating individual nerve cells that had participated in the encoding of the fear memory. Encoding neurons were also shown to have more connectivity with each other after erasure than with non-encoding neurons, even though their levels of synaptic potentiation and dendritic spine density had now decreased to the levels measured prior to memory acquisition. The fact that an artificial stimulation of nerve cells was required to produce relapse may mean that relapse would never happen otherwise. Mice that had the erased fear response optogenetically restimulated did not display the fear response subsequently when re-exposed to the fear context under normal (non-stimulated) conditions. However, the observations imply that robust functional erasure induced by pharmacological blockade does not necessarily eliminate the target learning's encoding entirely. This discovery of persistence of encoding after erasure raises doubts about the permanence of erasure, because if the target memory's engram (physical encoding) continues to exist to any degree after erasure, relapse is possible, at least in principle.

Behavioral erasure has not been similarly tested as yet, to the author's knowledge. If it were found that behavioral erasure fully eliminates encoding, permanence of therapeutic effects would be a possibility recognized by laboratory researchers if clinical translation were successful.

The claim made in the present article is that clinical translation actually is quite far along and already is successful, with abundant demonstrations that such is the case, some with verification of long-term erasure. Examples include the case of complex attachment trauma in Section 7.2, with erasure verified for 2 years as of this writing; a case of long-term depression also with 2 years of verification of erasure (Ecker and Hulley, 2002); and a case of long-term compulsive behavior with 6 years of verified erasure (Ecker, 2008). The erasure of lifelong anger detailed in Section 7.1 was verified for 8 months, and Högberg et al. (2011) verified for 22 months the cessation of severe PTSD symptoms in adolescents after use of a treatment protocol that is analyzed below in Section 9.2.2. In all of those cases, erasure persisted even as clients' life circumstances produced many potent re-cueings of schemas and symptoms under novel, stressful conditions, which satisfies researchers' most stringent tests for erasure.

Thus the possibility that behaviorally induced erasure is permanent currently appears to have more support from clinical observations than from laboratory studies. It is worth noting here that, as discussed in Section 3 regarding the markers of erasure, the criteria used by the author and colleagues for verifying reconsolidation and erasure in psychotherapy (in the final step of the therapeutic reconsolidation process, TRP Step V) are the same as those used in human studies of behavioral erasure by laboratory neuroscientists (Schiller et al., 2010; Oyarzún et al., 2012).

7.4.4. Is episodic memory resistant to destabilization and updating?

Perhaps the largest potential obstacle to clinical translation is the fact that the highly specialized emotional learnings that laboratory researchers create for studying the fundamental properties of reconsolidation are not representative of the real-life, symptom-generating emotional memory contents of therapy clients. The sizable gap between bench and clinic means that procedures developed in laboratory studies may not be effective in clinical practice for many possible reasons. In full agreement with that sober outlook, in Section 6.2 of this article, the author has argued for abandonment of the procedure-oriented perspective, in favor of an experience-oriented perspective, for the translation of reconsolidation research into clinical practice.

Some researchers have described interpretations of research that would imply that episodic memory may have relatively low susceptibility to destabilization and/or updating, and therefore might be resistant to change through reconsolidation-based methods in clinical work: Beckers and Kindt (2017) have conjectured about two ways in which the post-traumatic symptom of intrusive episodic memory (flashback) might not readily succumb to reconsolidation-based therapy. Schiller and Phelps (2011) and Kroes et al. (2015) have hypothesized that memories that are inherently encoded in an anatomically distributed set of brain regions, such as episodic memory, rather than in a single highly localized region, such as conditioned fear or motor procedural memory, are less susceptible to erasure by behavioral updating. As indicated in the following paragraphs, clinical observations of the effects of ECPE/TRP methodology on episodic memory suggest that those concerns may be unwarranted.

Beckers and Kindt (2017, p. 112) point out that laboratory studies have addressed memories consisting of "the anticipated repetition of a remembered event" (as created by Pavlovian associative conditioning) in contrast to "intrusive memories of a trauma where memory retrieval is not accompanied by the expectation of the actual traumatic event repeating itself; such memories may not be violated [mismatch and destabilized] as easily, because they do not imply an expectation of the traumatic event happening."

Indeed, an intrusive episodic memory episode or flashback is a re-immersion in and re-living of the original experience and not an expectation of the event repeating, so it is not possible during such a flashback to create a mismatch consisting of the non-reinforcement type (CS–noUS), that is, a perception that the event is not repeating, because subjectively the event *is* repeating. However, there are other types of mismatch to which the episodic memory is susceptible. These are described below, after noting first that complicating the symptomology in such cases is a secondary but clinically equally severe symptom often termed fear-of-fear: The personal history of having flashbacks *does* maintain a fearful expectation, between flashbacks, of the traumatic event repeating itself in the next flashback. (The case example in Section 7.2 above is of this type, a primary traumatic memory that has been retriggered numerous times and is therefore accompanied by continuous anticipatory "terror" of the next retriggering.) As long as the primary traumatic memory remains intact, maintaining the client's vulnerability to another retriggering or flashback, the fear-of-fear expectation remains intensely real-feeling to the client and is not feasible to mismatch, as a rule. The fruitful focus of memory erasure is therefore the primary traumatic memory, not the fear-of-fear memory.

The primary traumatic episodic memory can be mismatched within ECPE/TRP methodology in ways other than non-reinforcement of an expected event, in many cases. One approach involves facilitating in imagination a surprisingly different emotional experience of the same event. If the intrusive memory is only a perceptual fragment, a somatic sensation or an affective state, it is necessary first to guide de-suppression and retrieval of the more complete, unified episodic memory of the incident. Such retrieval is usually a delicate therapeutic task that requires a number of clinical skills and processes, both relational and technical, and is beyond the scope of this article (see, for example, Ogden and Minton, 2000; Payne et al., 2015). The altered emotional experience of the event can then be created in various ways, limited only by clinicians' creativity. Two methods used many times by the author, and described below, consist of replaying the memory as a differentiated witness (viewing the original incident from a different ego-state from the traumatized ego-state that the memory normally induces) and empowered re-enactment with disconfirmation of helplessness.

Witnessed memory replay has many variations of technique. One of the simplest and most natural is applicable if the trauma occurred in childhood and the client is now an adult. The emotional intensity of the memory can be drastically reduced by shifting the client's viewpoint in replaying the incident from the child's original viewpoint to the adult's viewpoint. For example, being left alone at home all night at 4 years of age was horrifying, and that unbearable horror of the child is thereafter a dominant feature of the episodic memory and necessitates its suppression. When the memory is de-suppressed in therapy in adulthood, after the child's horrified viewpoint is first experienced, in a natural manner the adult's view of the experience comes to the fore or can be invited to do so. The incident as witnessed in imagination by the adult is not horrifying. The differentiated adult's experience is verbalized typically as, "Oh, what happened doesn't look or feel so intense to me now as an adult, but I can see how intense it was for me as a child." That experience, sustained and dwelt upon for several minutes, is a mismatch, disconfirmation and counter-learning of the expected emotional intensity of the memory, so it carries out TRP Steps 1–2–3 (the ECPE) and permanently adjusts (updates) the memory's emotional valence to a much reduced, sub-traumatic level. This brings immediate cessation of memory intrusion as well as fear-of-fear symptoms and various symptoms produced for memory suppression (enumerated in Section 2), because memory suppression is no longer necessary.

Numerous other techniques of witnessed memory replay are in clinical use for treating traumatic episodic memory, such as EMDR (e.g., Shapiro, 2001; Solomon and Shapiro, 2008), Neurolinguistic Programming (e.g., Ecker, 2015c; Gray and Liotta, 2012; Gray and Liotta, 2012; Gray and Bourke, 2015; Gray and Teall, 2016), Traumatic Incident Reduction (e.g., Volkman,

2008), and Progressive Counting (e.g., Lasser and Greenwald, 2015). All of these techniques arrive at a mismatch and disconfirmation of the expected experience of the memory and also, in many cases, of generalized learnings (schemas) based on the original experience. Published case examples of these techniques typically contain observations of the markers of erasure, which, as discussed in Section 3, serves to verify that reconsolidation and behavioral memory updating have occurred (though no such conceptualization may be indicated). Analyzing such accounts for how experiences of mismatch and disconfirmation were created (because they must have been) is an effective way to build an extensive repertoire of techniques that carry out the steps of the TRP for traumatic memory. (See Section 9.2.2 below for such an analysis.) Clinical experience with this class of techniques suggests that viewing episodic memory contents from a vantage point outside of the ego-state inherent in the memory allows mismatches and disconfirmations to be found and brought into juxtaposition by the brain's always-operating mismatch detectors (for further discussion of which, see Ecker, 2015c).

Another way of erasing much of the emotional charge in a traumatic memory is by disconfirmation of the helplessness component of the episodic memory through empowered re-enactment (e.g., Ogden et al., 2006). De-suppression of the memory produces familiarity with the details of the traumatic incident, which allows the therapist to call attention to the specific conditions in response to which the client felt helpless or unbearably vulnerable in the face of danger. As noted in Section 7.2, the encounter with serious danger or harm is made traumatic due to feeling helpless and defenseless. In many cases, the helplessness component of an episodic traumatic memory is amenable to being disconfirmed in therapy through an imaginal empowered re-enactment experience. In the author's experience, nullification of the helplessness component of the original episodic memory immediately reduces the memory's accompanying emotional distress dramatically and de-traumatizes the memory. (Note that this process is different from a disconfirmation and nullification of helplessness expected in future instances of the same type. The latter is an erasure of semantic memory and is illustrated by the case example in Section 7.2.)

In empowered re-enactment, the client is guided to imaginally reinhabit the traumatic incident and respond to the situation in a vigorously self-protective manner, either by giving full expression to a self-protective action impulse that was blocked in the original experience or by being coached by the therapist to consider and select from various self-protective possibilities. The author has found it effective in some cases to suggest that the client, as a child in the scene, imagine her or his adult-self arriving and carrying out the protective action; or the therapist can join the action and protectively confront and fend off a perpetrator and give comfort and understanding to the child.

The examples of episodic memory transformation in the preceding paragraphs serve also to illustrate the point, discussed in Section 2, that the target of updating and erasure is semantic knowledge even when an episodic memory is the focus of attention in therapy. The helplessness experienced in the original incident was generated by the individual's implicit semantic learnings that were operative at the time, in many (but not all) cases. That semantic knowledge, which is embedded in the episodic memory, is fundamentally revised by the empowered re-enactment process (updating via ECPE), which correspondingly produces a profound change in the episodic memory's emotional quality of the event. The original semantic knowledge that is accessed through the episodic memory and transformed pertains to identity and developmental stage as well as interpersonal rules that dictate and restrain personal choices and responses (Frith and Frith, 2012; Markus and Wurf, 1987).

The emotional realness of the imaginal experiences described above is what gives them disconfirming potency; research has shown that the emotional learning and memory system responds to imaginal experiences almost indistinguishably from *in vivo* experiences (Agren et al., 2017; Kreiman et al., 2000). Therefore use of imaginal processes that replay the original scene with novel features and/or novel subjective viewpoints have much promise for dispelling traumatic memory through ECPE/TRP methodology. (A therapeutic system with a particularly rich repertoire of techniques of this kind is Neurolinguistic Programming (e.g., Gray and Bourke, 2015; Gray and Liotta, 2012; Gray and Teall, 2016). For detailed case examples of such techniques implemented within the TRP, see Ecker, 2015c; Ecker et al., 2012, pp. 86–91.) It remains to be seen whether clinical inventiveness eventually can subject

all types of traumatic memory to transformational change through ECPE/TRP methodology.

The second conjecture about how intrusive traumatic memory might elude reconsolidation-based therapy, according to Beckers and Kindt (2017), is based on various laboratory studies showing that an associative (Pavlovian, CS–US) memory becomes less susceptible to destabilization if elevated stress is present either at original acquisition or at subsequent reactivation. Both instances of elevated stress are characteristic of traumatic memory, so both adverse effects might apply, blocking memory destabilization in therapy for post-traumatic symptoms.

For emotional learnings and memories addressed in therapy, elevated stress at original acquisition and at reactivation during recall in therapy is the rule, not the exception. Certainly those two stresses are higher still in cases of more severe trauma. Clinical observation of successful erasure in many such cases (as discussed above and exemplified by the case example in Section 7.2) seems to indicate that the conjectured stress-related obstacle does not in fact block the effective use of reconsolidation in therapy for post-traumatic symptoms.

Likewise, the concern raised by Schiller and Phelps (2011) and Kroes et al. (2015), that the anatomically distributed encoding of episodic memory inherently reduces susceptibility to nullification and erasure of memory features, seems mitigated by the observed clinical successes of transforming the emotional quality and/or the meaning of original experiences in episodic memory (the case study in Section 7.1 being an example of erasure of meaning attributed to traumatic incidents). The clinical observations appear to support instead the analysis given by Hupbach (2011) in reply to Schiller and Phelps (2011), which proposes that susceptibility to erasure is governed not by anatomical extent of encoding, but by whether the content of the target memory is amenable to disconfirmation. Episodic memories are multi-component, multi-dimensional formations, within which are effective therapeutic targets for disconfirmation, unlearning and erasure, as described above. For example, certainly the fact of being left alone all night at age 4 cannot be disconfirmed, but the emotional significance of that situation *can* be disconfirmed by viewing the incident from the subjective standpoint of the client's progressed developmental stage.

7.4.5. Can clinicians navigate complex memory structure?

The heterogeneity of symptom-producing memory is a potential problem for translation of reconsolidation research in that clinicians face the challenge of targeting the specific components of memory that fully govern production of a given symptom. In that regard, researchers have identified two particular aspects of memory heterogeneity that could prove problematic for translation: a given symptom may be generated by episodic and/or semantic memory (that is, by event memory and/or generalization/schema memory) (Beckers and Kindt, 2017); and a given symptom may be generated by a learning that was formed on the basis of a more primary learning while also by the primary learning itself (Elsey and Kindt, 2017a), a configuration whose reconsolidation behavior has been studied using the simplifying experimental paradigm of second-order conditioning (Debiec et al., 2006). In addition, not mentioned by researchers (to the author's knowledge) are cases in which more than one separate and distinct emotional learning drives production of the same symptom (discussed with clinical illustration by Ecker et al., 2012). Will clinicians be able to navigate those complexities astutely, and thereby consistently identify the correct symptom-governing memory or memories as target for the empirically confirmed process of erasure? Discussion of each of those clinical challenges follows.

Learning and memory researchers have given much attention to the phenomenon of generalization of memory (e.g., Dymond et al., 2015), in which patterns, rules, abstractions, expectations, adaptive tactics and emotional responses are extracted from episodic memory of particular experiences and are encoded as categories and schemas in semantic memory (e.g., Eichenbaum, 2004). As discussed in Section 2, semantic/schematic memory of generalizations operates as the basis of adaptive responses largely autonomously from its episodic origins (though linkages may persist between the two memory systems and may be utilized in therapy). In that way, the individual is set to respond to novel situations that share salient features with original experiences.

The fact that a given symptom of a therapy client is produced by episodic memory of events, semantic memory of generalizations, or both, poses potential problems for clinicians aiming to utilize reconsoli-

dation for behavioral updating and erasure (Beckers and Kindt, 2017). One problem would be clinicians selecting event memory as the target learning when actually the symptom is being driven by generalization memory. Another problem is the reverse: clinicians might target generalization memory to treat a symptom driven by event memory.

Avoiding those two potential pitfalls in translation will obviously require clinician education and training that emphasizes cognizance of how both types of memory can drive symptom production (as discussed in Section 2). Assuming such cognizance on the part of the therapist, it is proposed that ECPE/TRP methodology addresses and dispatches those two problematic scenarios as follows.

The potential clinical error of targeting event memory for symptoms based in generalization (semantic) memory seems related to what the author perceives as a widespread tendency among clinicians, noted earlier, to assume that symptoms arise from episodic memory and to be relatively unfamiliar with semantic memory and implicit emotional schemas. TRP Step B by definition requires clinicians to carry out retrieval of all contents in memory, whether episodic or semantic, that drive production of the symptom identified in Step A. That requirement is based on this simple principle: *The symptom will cease to occur only when every one of the underlying, symptom-generating episodic and semantic memory formations have been subjected to the ECPE and transformed (updated) so that nothing remains in memory that necessitates the symptom.* The client's generalizations are in that way kept squarely on the clinical radar in ECPE/TRP methodology (and in Coherence Therapy, which is based on that principle of symptom cessation, and which supplies an array of techniques for TRP implementation (Ecker and Hulley, 1996, 2017); as noted in Section 6.3, ECPE/TRP methodology is a meta-map that does not define concrete methods or techniques for implementation).

The reverse potential clinical pitfall, addressing a client's generalizations for a symptom that is maintained by event memory, obviously also is addressed by the approach just described.

Clinicians face another complexity of memory when a given symptom is produced not only by some primary emotional learning, but also by a secondary learning that formed on the basis of the primary one (Elsey and Kindt, 2017a). Researchers have used the simplifying experimental paradigm of second-order fear conditioning (SOFC) to begin to study the reconsolidation behavior of that configuration (Debiec et al., 2006). In SOFC, animals are trained first by a standard fear-conditioning procedure, the pairing of a tone (CS1) with a shock (US). Then a second conditioned stimulus (CS2) is paired repeatedly with CS1, and the animal also develops a fear response to CS2. Debiec et al. found that after presenting CS1–noUS for reactivation and destabilization, followed by administration of a pharmacological blockade, fear responses to both CS1 and CS2 were dramatically reduced. When CS2–noCS1 was used for reactivation and destabilization, pharmacological blockade then impaired responses to CS2, but CS1 responses were unchanged.

The implication for clinicians is that targeting a derivative emotional learning will not necessarily also access the primary one. For example, an adult therapy client's primary emotional learning involved anger at his father for repeated physical violence inflicted on his mother throughout his childhood. His secondary emotional learning formed from a single incident at age 8 in which a police officer arrived, made excuses for the father's behavior and failed to protect the mother, resulting in the client forming a generalized anger at police officers and other male authority figures, which was the secondary learning that was based on the primary one. In therapy at age 45, the man presented his anger at male authorities as the problem, with no mention or awareness of the original incidents. Initially the TRP was used in an attempt to disconfirm and nullify the client's felt knowing that police officers do bad things and deserve anger and distrust, but no shift ensued. Then episodic memory of the incident at age 8 emerged into awareness, and the therapist recognized that the primary emotional learnings pertained to the father. Focusing the TRP now on that set of learnings, both episodic and semantic, resolved and eliminated anger and other distresses in relation to father, and the client then found that his anger and distrust toward police officers had disappeared with no further work in that area.

As that example suggests, in practice the pragmatics of systematically applying the TRP solve the problem of memory complexity described by Elsey and Kindt (2017a): If, after carrying out the ECPE (TRP Steps 1–2–3), the current target learning remains in force and seems immune to disconfirmation, that is an

indication that either the target learning is secondary to some other one that is primary and must now be found, or that disconfirmation is being blocked to prevent some intolerable consequence of disconfirmation, which must now be retrieved into awareness and addressed, as discussed in Section 6.3.

Lastly, the memory complexity of two or more separate, primary emotional learnings generating the same symptom is routinely navigated by clinicians using the TRP in much the same systematic manner as described just above (Ecker et al. 2012). If the client reports that the distinctive affective experience of the current target learning is now never felt in any of the situations that formerly triggered it, indicating successful unlearning and erasure, yet the symptom that that target learning had been generating continues to occur in those or other situations, the therapist understands this to indicate the existence of another symptom-generating emotional learning that must now be found. The therapist then carries out the TRP anew, beginning with identifying the situations in which the symptom now still occurs (TRP Step A), and then works to retrieve the underlying learning that is responding in those situations and producing the symptom (TRP Step B). In that way any number of symptom-generating schemas and/or episodic memories are progressively retrieved and nullified until full cessation of the symptom under all circumstances indicates that the unlearning and erasure process is complete for that symptom.

8. Ramifications for fundamental issues in psychotherapy

Sections 3, 6, and 7 present the manifold indications that transformational change of a person's established patterns (including, but not limited to, the unwanted patterns presented in psychotherapy) is always the result of certain internal, concurrent experiences, which have been identified independently in clinical observations and in reconsolidation research, and which in this article are labeled the empirically confirmed process of erasure, or ECPE. If it is indeed true that only this combination of experiences produces transformational change of acquired (implicitly learned) states of mind, body, and behavior, this would have significant ramifications not only for case conceptualization in psychotherapy, but also for several of the psychotherapy field's important conceptual frameworks, including corrective experiences, psychotherapy integration, and the roles of nonspecific versus specific factors. This section sketches how our knowledge of reconsolidation and the ECPE in particular would drive the further evolution of understanding in those three areas. Reference is also made below to the therapeutic reconsolidation process or TRP, defined in Section 6.3 as being the general form of clinical implementation of the ECPE.

8.1. Reconsolidation clarifies the "corrective experience"

Beginning with the *corrective emotional experience* (CEE) defined by Alexander and French (1946), an important strand running through the development of psychotherapy has been the various efforts to identify, based on clinical experience and observations, how transformational change occurs. If one regards the empirically confirmed process of erasure (ECPE) that has emerged from laboratory reconsolidation research to be a fundamental and decisive breakthrough in understanding how transformational change occurs, it then sheds new light on those prior clinical accounts and authoritatively clarifies their strengths and limitations.

A thorough survey of relevant clinical methodologies of transformational change and how they compare to the findings of reconsolidation research is beyond the scope of this article. (For analyses of ECPE/TRP fulfillment by eight widely used psychotherapy systems, see Ecker et al., 2012, pp. 126–155, and *The Neuropsychotherapist*, issue 10, January 2015.) Here, a few sample points are offered regarding the CEE and its subsequent evolution, as regards fulfillment of the ECPE/TRP.

The description of CEEs provided by Alexander and French (1946) has recently been closely examined and itemized in detail by Sharpless and Barber (2012, p. 34), who listed twelve significant components as follows:

1. The client must have experienced traumatic events (construed fairly broadly) or events that caused a traumatic influence which were not successfully or adaptively dealt with in the past. (p. 66)
2. The client must be reexposed to these emotional

situations that were not successfully/adaptively dealt with. (p. 66)

3. This reexposure must occur in more favorable circumstances than the original situation allowed. (p. 66)
4. The client must be able and willing to face the reexposure (implied in definition).
5. This reexposure does not necessarily need to take place with the therapist or within typical session confines. (p. 66)
6. The therapist (or another person in the client's life) must assume or express an attitude different from that of the individual or individuals involved in the original traumatic event. (p. 66)
7. Building on Item 6, with CEEs specifically involving the therapist, the therapist may or may not self-consciously assume a particular role or attitude (or, similar to Kierkegaard [1884/1980], facilitate a particular emotional atmosphere) to elicit the emotional situation (i.e., manipulation may be present, but not necessarily; Alexander, 1961; Alexander & Selesnick, 1966). (p. 66–67)
8. The client must handle or react to this novel situation (Item 6) in a manner different from before. (p. 67)
9. Such a result often takes repetition of the conflicts before a new ending occurs (i.e., it seems unlikely that CEEs occur with a single reexposure). (p. 67)
10. Patient insight into these patterns may accompany a CEE but is neither necessary nor sufficient to cause the CEE, and the experiential component holds predominance. (p. 67)
11. As a result of the above, the trauma becomes "repaired" in some way. (p. 66)
12. The results of the CEE should generalize to other situations and experiences (implied).

From that fine-grain itemization by Sharpless and Barber (2012), it is apparent that the CEE of Alexander and French (1946) significantly fulfills the ECPE/TRP: First and foremost, the CEE calls for the first two steps of the ECPE, namely, a reactivation of the problematic response (via reexposure to its particular cues) concurrent with an experience that contradicts the expectations inherent in the problematic response (items 2, 3, and 6 in the list above). Those two steps form the juxtaposition that creates the all-important memory mismatch, which destabilizes the neural encoding of the problematic response, launching the reconsolidation process. The ECPE's third step, a few repetitions of the juxtaposition experience during the remainder of the session, driving unlearning and erasure, is not explicitly called for in the CEE definition, but once the juxtaposition experience is created, including repetitions of it is a simple matter in practice.

In addition, the CEE is also in accord with the ECPE/TRP in two other important ways: It emphasizes the experiential nature of the process, with cognitive insight in a secondary and variable role (item 10); and it recognizes that the crucial violation of expectations is not necessarily produced only by the client's experience of the therapist, and therefore allows for the juxtaposition to occur either within or outside of a therapy session (item 5).

Certain limitations of the CEE as originally defined also become apparent. Juxtaposition is seen as occurring only in interpersonal interactions, that is, from the behavior of people. However, both laboratory studies (e.g., Galluccio, 2005; Schiller et al., 2010) and clinical observations (e.g., Ecker et al., 2012, pp. 93–97; Högberg et al., 2011) have shown that other types and domains of experience can drive unlearning and erasure of acquired emotional responses. As noted in Sections 5 and 6, disconfirmation of a target emotional learning must be highly specific to the content of the target learning. In therapy it is found that clients sometimes have problematic emotional learnings that were not formed in response to other people and therefore cannot be disconfirmed by experiences of people. An example is the man who had no initiative to escape an abusive situation at work. The underlying basis of his passivity was found: At the end of high school, he was diagnosed with a serious osteopathic illness, which suddenly ruled out the brilliant athletic career he was eagerly expecting to enjoy in college. In response to this crushing loss, he implicitly learned a self-protective strategy that became verbalized in therapy as, "If I try for what I really want, the world will crush it, so I better *not* try for, or even feel, what I really want." That learning of self-protective passivity was not produced by, and therefore was unlikely to be disconfirmed and

unlearned by, having experiences of other people.

The original CEE definition also falls short in two other ways. First, it leads clinicians not to expect or strive for decisive, transformational change in a single session of disconfirming reexposure (item 9). However, such rapid, decisive change does indeed occur, as illustrated by the case examples in Sections 7.1 and 7.2, and is often observed by experienced practitioners of the ECPE/TRP. Second, while the CEE specifies *what* conditions bring about change, it does not identify *how* change occurs, i.e., the mechanism of change, invoking only a general notion of "repair" (item 11). The ECPE/TRP framework provides a full and clear account of both the mechanism of symptom production (via adaptive emotional implicit learning) and the mechanism of symptom cessation (via unlearning and erasure of implicit knowledge through the memory reconsolidation process).

Alexander and French (1946) had introduced the CEE within the context of psychoanalytic psychotherapy (where it encountered fierce opposition, as reviewed by Sharpless and Barber (2012)). Goldfried (1980), aiming to make the CEE phrase and concept transtheoretical, so as to be broadly relevant to pivotal experiences observed in various types of psychotherapy, used instead the phrase *corrective experience* (CE) in his seminal article, "Toward the Delineation of Therapeutic Change Principles." Goldfried proposed that CEs are a common factor found in many different forms of psychotherapy, and his article launched the study of CEs as a way to identify therapeutic change principles. More recently, the CE construct was addressed by a group of twenty-seven psychotherapy scholars representing diverse theoretical orientations, who convened to discuss and pool their latest conceptualizations, clinical observations, and research on transformational change events in psychotherapy (Castonguay & Hill, 2012).

The group arrived at this consensus definition of a CE: "Currently, we understand CEs in psychotherapy to involve a disconfirmation of a client's conscious or unconscious expectations…as well as an emotional, interpersonal, cognitive, and/or behavioral shift. In CEs, clients typically reencounter previously unresolved conflicts…or previously feared situations (whether internal or external) but reach a new outcome in terms of their own responses, the reactions of others, or new ways of interacting with others" (Hill et al., 2012, pp. 355–356).

In that definition, the primary emphasis on "disconfirmation of a client's conscious or unconscious expectations" is in exact agreement with the reconsolidation research findings reviewed above in Sections 4 and 5. Also matching the neuroscience research is the breadth of that definition: The experiences that disconfirm existing emotional learnings and induce transformational shifts are diverse in nature and are not necessarily themselves "emotional" (as can be seen in the case example in Section 7.1; on this point see also Ecker et al., 2015).

Numerous aspects of CEs were considered by group participants, including CEs induced by a new inner experience versus those induced by trying a new behavior in the problematic situation; CEs that occur fully in one decisive, discrete event versus those involving a series of events that reach a tipping point; the variable role of cognitive insight in CEs; client factors that contribute to CEs; the range of therapist behaviors that can precipitate a CE; and the consequences of CEs.

Section 1 of this article addressed the current absence of reciprocal cognizance between reconsolidation neuroscientists and clinical psychologists. That mutual unawareness is well illustrated by the absence of any reference to memory reconsolidation in the seventeen chapters of the group members' published contributions (Castonguay & Hill, 2012), even though reconsolidation research has achieved a major breakthrough of empirical clarification of transformational change events, the phenomenon addressed by the group.

In fact, as described below, reconsolidation research findings have illuminated important features of transformational change events/CEs that are not identified in the CE accounts produced by the group (Castonguay & Hill, 2012). Three such features are described here next: 1. Transformational change events consist of a well-defined process of profound *unlearning* of implicit knowledge that was adaptively learned by the subcortical brain earlier in life. 2. Designating transformational change events as "corrective" perpetuates pathologizing views of symptom production that are inconsistent with the neuroscience of implicit learning and unlearning. 3. Disconfirmation, the crucial ingredient for transformational change, requires specific conditions for clinical implementation that are mentioned minimally, if mentioned at all, in CE litera-

ture. Elaboration of those three features follows:

1. Transformational change events are the profound unlearning of implicit knowledge that was adaptively learned by the subcortical brain earlier in life. This is illustrated by the case examples in Sections 7.1 and 7.2. As described throughout this article, there is much usefulness for clinical work in understanding transformational change within a broader understanding of emotional learning and unlearning (Ecker et al., 2012). Reconsolidation is the brain's innate process for updating existing learnings, and one form of such updating is profound unlearning that erases previously acquired semantic knowledge (expectations, meanings, schemas, rules, roles, mental models). This meta-level perspective on the process and mechanism of change is not yet supplied by the CE framework (Castonguay & Hill, 2012).

2. Designating transformational change events as "corrective" perpetuates pathologizing views of symptom production that are inconsistent with the neuroscience of implicit learning and unlearning. "Corrective" implies that the client's prior emotional learnings are *in*correct at least in the milieu of the present, even if they were correct in the original learning context. However, as noted in Section 2, brain science contradicts the conventional notion that the acquired beliefs or schemas underlying symptoms are incorrect, irrational, maladaptive or pathogenic. The brain evolved in such a way that implicit emotional learnings are neurologically built to persist and reactivate for a lifetime (see, e.g., LeDoux et al., 1989) unless erased via reconsolidation, which requires special conditions that rarely occur in the ordinary course of life. Thus the persistence for decades of implicit beliefs, expectations and schemas adaptively formed in childhood represents the proper functioning, not the malfunctioning, of the subcortical emotional learning and memory system, and should not be described in pathologizing terms. "Corrective experiences" could be replaced by "unlearning experiences," "erasure experiences," or "transformational experiences," for example.

3. Disconfirmation, the crucial ingredient for transformational change, requires specific conditions which, as a rule, are barely mentioned, if mentioned at all, in CEE and CE literature. Disconfirmation is usually depicted as consisting of the client having the new, unexpected experience. However, therapists are all too familiar with guiding a client into having such experiences without any lasting shift resulting. Thus a standing question of much importance for the clinical field is: Why do such experiences induce sudden, transformational change in some instances but not in many others?

An empirically based answer to that key question is provided by the memory reconsolidation research reviewed in Sections 4 and 5: In order for any experience to produce disconfirmation and robust, enduring cessation of an unwanted behavior or state of mind, certain specific factors are required by the brain, namely, the new experience must occur concurrently with reactivation of the problematic emotional learning (negative expectation) maintaining the unwanted behavior or state of mind, and must contradict that emotional learning with high specificity. Disconfirmation consists of the client having the new, unexpected experience *while also experiencing the old expectation or attribution of meaning*, as delineated in Section 6.3 and exemplified in Sections 7.1 and 7.2 (and as specified by Alexander and French (1946), as noted above). It is the subjective juxtaposition and felt dissonance of the two that creates the memory mismatch (prediction error) that destabilizes the neural encoding of the old expectation, launching the reconsolidation process and allowing unlearning and erasure to occur.

In short, the new, unexpected experience must occur as part of a juxtaposition experience, but it is by no means automatic that it would do so. It is entirely possible and even commonplace for a therapy client to have a new, unexpected experience while the old, negative expectation is dissociated and suppressed, outside of awareness, such that no juxtaposition occurs. In that case, no transformational change occurs, puzzling or frustrating the therapist and bringing disappointment and perhaps also a sense of personal failure for the client. Attempted CEEs and CEs often consist of only the desired new experience, without the full juxtaposition. Both therapists and clients are highly prone to what has been described as a counteractive reflex (e.g., Ecker, 2006, 2008, 2015; Ecker et al., 2012), an urge to avoid and suppress unwanted behaviors and states of mind while building up and attending to preferred behaviors and states of mind. Attempted CEEs and CEs are all too easily shaped by that counteractive tendency: The therapist guides the client's attention to be fully engaged in the desired new experience and disengaged and dissociated from the unwanted reac-

tion or ego state and its core schema. That configuration is the opposite of the explicit, foreground, experiential awareness of the target schema that is needed for reliably creating the juxtaposition that destabilizes the target schema. New experiences structured in that one-sided manner in therapy can feel deep, meaningful and freeing in the moment, but they nevertheless cannot result in lasting change if they create only competitive new learning, with the core schema underlying the problem remaining intact, as it does if it is not subjected to a juxtaposition that disconfirms and dissolves it.

The requirement of the two-sided (i.e., juxtaposition) experience for producing transformational change was articulated, based on clinical observations, by Ecker and Hulley (1996, 2000b, 2002). For example:

> "The new construct or view of reality must be clearly and compellingly inconsistent with the view of reality in the client's pro-symptom position [symptom-necessitating schema]…. The task of the therapist is to arrange for the client to take in this new construct *while inhabiting and vividly experiencing the pro-symptom position*, so that both the new and the old constructs are vivified and experientially real to the client at the same time." (Ecker & Hulley, 1996, p. 239)

> "If the new constructs are created while the old position is not activated, the client does not actually experience an inconsistency or disconfirmation. *The simultaneous vivifying of the old and the new constructs in the same field of awareness is the essential condition for the transformation of position to occur.*" (Ecker & Hulley, 1996, p. 237)

> "[T]he strategy consists of having the client experience subjectively both the pro-symptom emotional reality and some other, incompatible construction of reality simultaneously, in the same field of awareness. The experiential quality of the disconfirmation is essential, and is not achieved by merely attempting to refute, convince, or "correct" the client by contrasting "irrational beliefs" with "rational" ones…" (Ecker & Hulley, 2000b, p. 169)

Subsequently the requirement of a juxtaposition experience was shown to be a primary clinical implication of reconsolidation research (Ecker, 2008, 2010, 2015; Ecker et al., 2012; Ecker & Toomey, 2008).

Thus the clear message of reconsolidation research to clinicians is: An experience that *could* effectively serve to create disconfirmation and transformational change fails to do so if the target learning is not concurrently reactivated. That point was emphasized in comments to the author from neuroscientist Javiera Oyarzún (as noted in Section 1, neuroscientists use the term *memory* to refer to all types of memory contents and memory systems, including episodic and semantic memory): "If we don't remember many details of such memory (a very old one), we won't be able to generate a reliable prediction error and thus we won't have much of a memory transformation. The better we retrieve our old memories, the better we will be able to generate this PE." (Javiera Oyarzún, private communication, 20 June 2016)

In light of the foregoing considerations, it is proposed here that the *juxtaposition experience* should be regarded as an update of the CEE and CE concepts according to memory reconsolidation research, with the updated therapeutic change principle perhaps stated as follows:

> In psychotherapy, cessation of an unwanted, acquired (learning-based) behavior, state of mind, or somatic disturbance is accomplished most consistently by facilitating a few repetitions of a juxtaposition experience in which the individual lucidly experiences both the underlying emotional learning or schema and, concurrently, any personal, direct knowing that specifically and inescapably contradicts and disconfirms that emotional learning.

That definition embodies the empirically confirmed process of erasure, and its implementation is illustrated by the case examples in Sections 7.1 and 7.2. A therapist who regards a juxtaposition experience as necessary for reconsolidation and transformational change does not stop with creating the missing, needed new experience but also works to create a concurrent experience of the problematic learning underlying the problem. An instance of such mindful facilitation of juxtaposition has been given by Ecker (2015a, p. 31):

> [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 juxtaposing 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 I do anything wrong, and I expect everyone 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.

The CEE and CE memes and accounts of transformational change are venerable in the psychotherapy field. At the same time, in light of progress in reconsolidation research and its clinical translation (labeled ECPE/TRP methodology in this article), it may now be appropriate to regard the reconsolidation framework as superceding the historical CEE and CE accounts of transformational change.

8.2. Reconsolidation provides a unifying framework of psychotherapy integration

Section 3 addressed the hypothesis that transformational change produced by psycho-therapy is always the result of memory reconsolidation, and Sections 4, 5 and 6.1 refined that idea into the hypothesis that transformational change produced by psychotherapy is always the result of the specific experiences in the empirically confirmed process of erasure (the ECPE, which is TRP Steps 1–2–3).

That hypothesis has potential ramifications for the field of psychotherapy integration, as described by Ecker (2011) and Ecker et al. (2012, pp. 126–155; 2013c), who proposed that the TRP is a deep structure shared by all systems of psychotherapy that demonstrably produce transformational change (a continuation of Goldfried's (1980) quest for delineating common factors that produce transformational change across therapy systems). If the TRP actually is universal in that sense, then TRP Steps A-B-C-1-2-3 should be detectable in hindsight in any sufficiently detailed account of any therapy sessions that produced the markers of erasure and transformational change in Step V (in much the same manner as Ecker and Hulley (1996) detected the TRP steps by looking back from the markers of erasure, as described in Section 3). Employing that logic, various authors have reported unambiguous detection of the TRP steps embedded in the therapeutic process in published case studies from ten systems of psychotherapy (listed online, with citations, at http://bit.ly/15Z00HQ). Both the ECPE's critically necessary Steps 1–2–3 and the preparation steps, Steps A–B–C, were detected in that way.

Thus the TRP is emerging as a common factor in therapies of transformational change. The steps of the TRP are *specific* common factors, in contrast to the *non-specific* common factors widely regarded as being responsible for therapeutic efficacy in randomized controlled trials (e.g., Wampold, 2001, 2015). The TRP could potentially serve as a unifying, empirically based framework for illuminating the operation of therapies of transformational change independently of theoretical constructs and biases. The TRP could provide practitioners of different therapy systems with a shared frame of reference and a shared, universal vocabulary, enabling them to discuss how their seemingly dissimilar methodologies facilitate the same empirically confirmed process of erasure, without challenging any

system's conceptualization of itself.

For individual clinicians, having the TRP as one's primary orienting framework reveals the TRP capability of every particular system of psychotherapy, and converts the panoply of psychotherapies from being a severely fragmented and confusing situation into a rich repertoire of options to choose from for best facilitating the brain's process of erasure for a particular client. (For a case vignette illustrating a clinician's use of that unifying framework, see Ecker, 2015c.)

Welling (2012) has proposed a psychotherapy integration scheme in which diverse therapy systems induce reconsolidation by creating experiences that connect "maladaptive" emotion with "adaptive" emotion. However, that account does not adequately or accurately represent reconsolidation research findings (as reviewed in sections 4 and 5; and see section 9.3 below for why that is the case). The psychotherapy integration scheme defined by Lane et al. (2015) is based on a comprehensive map of the different types of memory involved in symptom production and tags therapy systems according to which type of memory they target. That is an interesting and useful scheme, but it is not based on the memory reconsolidation and erasure process.

8.3. Reconsolidation explains psychotherapy RCT outcome research and refutes non-specific common factors theory

Seventy-five years of randomized controlled trials, or RCTs, have measured essentially the same efficacy for all tested systems of psychotherapy (at least fourteen of them; for reviews see, e.g., Duncan et al., 2009; Wampold, 2001, 2015). In the outcome research literature, a positive frame has often been put around this remarkable finding by referring to it as the Dodo bird verdict, meaning that all have won, all have done equally well, so "all must have prizes," as declared by the Dodo bird in *Alice in Wonderland*. Actually, the measured level of efficacy is quite modest, being little better than the efficacy of placebo therapy, leading Ecker (2006, 2015b) to suggest that perhaps all have lost. Be that as it may, the uniformity of efficacy has required explanation. The most predominant and now widely accepted hypothesis is nonspecific common factors theory, which interprets the uniformity of efficacy to mean that the *specific* processes and procedures of any given system of therapy contribute at most weakly (15%) to therapeutic change, and that efficacy must therefore be attributed mainly (85%) to *non-specific* features that are equally present in all therapies as statistically sampled in RCTs (e.g., Duncan et al., 2009; Wampold, 2001, 2015). The *nonspecific common factors* include the qualities of the client, the therapist, and the client-therapist relationship, such as qualities of trust, empathy, therapeutic alliance, and the therapist's belief in the treatment, among other aspects.

Nonspecific common factors theory faces two main challenges (reviewed by Ecker, 2015b; Ecker et al., 2012, pp. 153–155; Shean, 2014). First, the statistical nature of RCT data analysis inherently hides the existence of any potent specific factors utilized effectively in a small percentage of outlier sessions within the study. That fact makes it illogical and inappropriate to conclude from RCTs that specific factors are fundamentally weak. Second, numerous studies designed to detect potent specific factors have indeed done so, showing that the specific factor of inducing affective experiencing of previously suppressed emotion and emotional meaning has a much stronger correlation with therapeutic change than do the nonspecific common factors. For example, a meta-analysis by Weinberger (1995) found that the therapeutic alliance, which is one of the most widely emphasized non-specific factors, accounted for 11 percent of the variance in therapy outcomes, whereas the specific factor of guiding clients to face what they had been avoiding accounted for 40 percent of variance.

Memory reconsolidation research adds what appears to be decisive empirical evidence in favor of the existence of potent specific factors (Ecker, 2006, 2013, 2015b). The empirically confirmed process of erasure (or ECPE, defined in Section 6.3) consists of a sequence of specific experiences that produce the most effective therapeutic change possible, as detailed in this article. Application of the ECPE in clinical practice, producing such effectiveness, is a present reality, not an awaited future development, as shown by the clinical cases presented in Section 7 and in numerous other accounts (Ecker et al., 2012; http://bit.ly/2tKXdyX). For consistent clinical implementation, the ECPE inherently requires the three preparatory steps and subsequent verification step described in Section 6.3, forming all together the therapeutic reconsolidation process or TRP. One of the preparatory steps, Step B, the expe-

riential retrieval of a symptom-generating emotional schema, is inherently the effective specific factor described in the previous paragraph. Thus the TRP embodies not only the most effective specific factor previously identified by clinical researchers, but also the ECPE, the specific factor identified in reconsolidation research, which produces transformational change (permanent cessation of symptom and core emotional issue resolution). The TRP appears to fit naturally within an "empirically supported principles of change" system (Rosen and Davidson, 2003).

In light of the credibility now accruing to specific factors, Ecker (2013, p. 137) suggested that psychotherapy RCTs might be more accurately interpreted as showing that about 15% of therapists participating in RCTs across the decades of outcome research have applied effective specific factors knowingly or unknowingly. He concluded by noting that "the client-therapist relationship remains indispensably important for good psychotherapy. This is not an either/or situation. It now seems clear that in the most effective psychotherapy, an environment of good nonspecific common factors supports facilitation of the specific factors of emotional accessing and memory reconsolidation."

9. Other emerging clinical translation methodologies analyzed in relation to reconsolidation research

In defining the empirically confirmed process of erasure (ECPE) and its pragmatic extension into the therapeutic reconsolidation process (TRP), this article attempts to translate reconsolidation research into clinical methodology in the most general and broadly applicable manner possible, while maintaining rigorous fidelity to research findings to date. This article would be incomplete without a discussion of other efforts at translating reconsolidation research into clinical methodology, in comparison to the framework advanced here. This section covers a few approaches that have received considerable attention, but it is by no means exhaustive. The main aim in this section is to demonstrate how the action and effects of any given methodology or protocol can be specified in terms of research findings with clear, consistent, objective standards, by mapping the methodology in question onto the ECPE/TRP framework. The ECPE/TRP framework is claimed to be no more and no less than a distillation of all research on how a target learning is destabilized and then erased by behavioral updating.

As the importance of reconsolidation to psychotherapy has become increasingly apparent to clinicians over the past decade, more and more exponents of particular therapeutic systems have published accounts proposing that reconsolidation is the mechanism of change responsible for the observed effectiveness of the favored system's process of therapy (e.g., Badenoch, 2011; Coughlin, 2016; Gorman and Roose, 2011; Gray and Liotta, 2012; Greenberg, 2010; Solomon and Shapiro, 2008). Such claims may acquire plausibility by synchronizing with reconsolidation research in two main ways: by demonstrating observation of the markers of erasure in specific cases (because, as noted in Section 3, it is believed that only reconsolidation can produce those markers), and by showing in concrete and specific terms how the clinical methodology implements the empirically confirmed process of erasure. Those two types of plausibility are referred to below as *erasure evidence* and *methodology evidence*, respectively.

Erasure evidence in specific clinical cases seems necessary for making a strong claim that a particular system or protocol of psychotherapy actually recruits the reconsolidation process. Adding methodology evidence makes the case as strongly as is now possible, until neuroscientists create a method of direct detection of engram nullification that is safe and practical. The present author and/or clinical colleagues have documented both erasure evidence and methodology evidence for nine different systems of psychotherapy as of this writing; they are listed online, with citations, at http://bit.ly/15Z00HQ.

Unconvincing claims are those that lack erasure evidence. Some claims include erasure evidence but provide methodology evidence that is either too vague to establish ECPE fulfillment or is based on misconceptions of how reconsolidation operates and an inaccurate account of reconsolidation research findings. Adequate familiarity with and citation of relevant reconsolidation research findings is relatively rare in accounts of clinical application to date, in this author's experience. It is not uncommon for claims of methodology evidence to be manifestly far afield from the specific process (the ECPE) identified by research and delineated in this article. Lack of accountability to research findings and apparent lack of awareness

of research findings spurred the present author and colleagues to publish an article titled, "Minding the Findings: Let's Not Miss the Relevance of Reconsolidation Research for Psychotherapy" (Ecker et al., 2015).

Evaluating claims of methodology evidence by rigorously examining published clinical procedures in relation to reconsolidation research findings seems necessary and legitimate in support of the clinical field acquiring accurate, and thereby maximally effective, knowledge of the workings of reconsolidation. Carrying out such evaluation of claimed methodology evidence is also an instructive exercise.

9.1. Post-retrieval extinction

As the first behavioral procedure to demonstrate erasure of a learned fear (Monfils et al., 2009; Schiller et al., 2010), post-retrieval extinction has attracted much interest and use by both laboratory and clinical researchers (reviewed by Auber et al., 2013; Kredlow et al., 2016; Lee et al., 2017). In the discussions of post-retrieval extinction in Sections 5 and 6.2 above, it is apparent that, in the form originally implemented by Monfils et al. and Schiller et al., this protocol unambiguously carries out the ECPE (that is, it provides well-defined experiences of reactivation, mismatch and counter-learning as necessary for erasure to result). Both erasure evidence and methodology evidence are strong for this protocol in its original form.

Many subsequent studies have used variants of this protocol to target fear learnings or addiction learnings, while retaining the label of post-retrieval extinction or reactivation-extinction (see reviews cited above as well as those by Dunbar and Taylor, 2017; Schwabe et al., 2014; Treanor et al., 2017). In these studies, the constitution of the target learning, the manner of target learning reactivation, and the form of post-reactivation counter-learning have varied significantly from those in the studies by Monfils et al. (2009) and Schiller et al. (2010). As noted in Sections 4 and 5 of this article, numerous reconsolidation studies have shown that the brain requires a particular relationship between those three variables in order for destabilization and erasure to result: A memory mismatch experience must be created, consisting of a violation of the expectations or knowings of the reactivated target learning, and counter-learning must then disconfirm the specific content of the target learning. However, those findings have not guided the design of all studies, as noted in this article and by Ecker (2015a), and that also seems true of several attempts to use post-retrieval extinction protocol variants for clinical tranlation. Indeed, the reviews by Auber et al. (2013) and Kredlow et al. (2016) show many hits and many misses by procedures labeled post-retrieval extinction.

It was noted in Section 6.2 (and also by Ecker, 2015a) that labeling this protocol with the term "extinction" is a misnomer that invites misconceptions. However, the label has become standard terminology, so it is used here.

For understanding any given study, the principle of mismatch relativity is critically important: Whether the study's procedure can fulfill the ECPE is determined by the detailed structure of the protocol's post-reactivation experiences interacting with the detailed structure of the encoded target memory experiences (the original acquisition learning experiences). As shown in Section 5, the original post-reactivation extinction protocol has a specialized structure that is effective for a Pavlovian target learning acquired through multiple CS-US pairings evenly spaced in time. In particular, the protocol's most distinctive feature is a 10-minute time delay after a single CS presentation reactivates the target learning. In relation to a Pavlovian target learning created by an evenly repetitive time structure and an inter-training interval of much less than 10 minutes, the 10-minute delay creates a temporal mismatch that destabilizes the target learning (Alfei et al., 2015). However, most target learnings encountered in clinical practice were not acquired with such a time structure. The 10-minute delay in this protocol would create mismatch or destabilization only if the target learning happens to contain temporal expectations on a time scale significantly less than 10 minutes.

A second distinctive feature of this protocol is the unexpected viewing, during the 10-minute delay, of an entertaining TV show or cartoon. In clinical practice, whether this feature would create the needed mismatch experience is doubtful due to its lack of relevance to the target learning (except perhaps in a rare few serendipitous cases). As noted in Section 4, research has shown that a post-reactivation experience that is too dissimilar from the target learning fails to register as a mismatch requiring updating, and target memory destabilization does not occur.

Another distinctive feature of this protocol is the use of extinction training for counter-learning, as discussed in Sections 5 and 6.2. That format of counter-learning presumably succeeds only for a target learning consisting of cue-triggered expectation of a discrete event, whether appetitive or aversive. Some target learnings encountered in clinical practice do have that structure. However, as a rule it is not possible to have the real-life CS occur during therapy sessions in order to create non-reinforcement (CS-noUS) experiences. Imaginal experience can be highly effective in therapy for various purposes, but whether imaginal CS-noUS events could feel real enough to register as counter-learning is doubtful, in the author's opinion; controlled studies of imaginal CS-noUS experiences are needed to settle this question. Many target learnings encountered in therapy do not consist of cue-triggered expectation of a discrete event (such as in the case example of Section 7.1), and for such cases the post-reactivation extinction protocol presumably could not provide effective counter-learning.

Thus, according to research-based criteria for achieving erasure, the clinical applicability of the post-retrieval extinction protocol appears to be limited, and that conclusion appears to be supported by the reviews of Auber et al. (2013) and Kredlow et al. (2016). The above analysis again illustrates the discernments gained by understanding memory reconsolidation in terms of requisite subjective experiences rather than in terms of laboratory procedures that happened to be effective under specialized, simplified conditions, as discussed in Section 6.2. For purposes of clinical translation, interpreting reconsolidation research on the basis of procedures alone can result in the proverbial wild goose chase.

For example, Johnson and Casey (2015) elegantly demonstrated the effectiveness of the post-retrieval extinction protocol with adolescent human subjects, who acquired an aversive Pavlovian CS-US association on day 1 of the study and were completely free of that conditioned fear at the end of the study. However, Johnson and Casey interpreted their decisive results entirely in terms of procedures. Neither their text nor citations refer to the need for a mismatch/prediction error experience (which is consistently the case in studies of this protocol, beginning with its originators, Monfils et al. (2009) and Schiller et al. (2010)). Thus, they viewed the 10-minute delay as having an intrinsic destabilizing effect for any target learning, rather than recognizing it to be a specialized mismatch that can destabilize only a special, narrow class of target learnings, and they concluded,

> Our data highlight how modifying the timing of therapeutic sessions based on principles of memory reconsolidation could lead to more effective attenuation of conditioned fear in both adolescents and adults. A modified version of an exposure-based CBT protocol based on memory reconsolidation might involve reminding patients of why they are there when they first arrive at the clinician's office (i.e., reminder cue), then establishing a safe and positive rapport for approximately 10 minutes (i.e., waiting for reconsolidation window) before initiating desensitization with exposure therapy.

It is predictable, in light of the research-based considerations delineated above, that the procedure suggested by Johnson and Casey would not induce reconsolidation and would prove to be nothing more than, and no more effective than, exposure therapy as usual.

An important class of clinical emotional learnings consists of attributions of meaning that are nonverbal and outside of awareness, but can be verbalized after being attended to while felt, such as the first of the examples in Section 2: Getting Mom's and Dad's anger or cold disregard when I express my needs or distress means that my very self is disgusting and unacceptable. That learned construal of the parents' behavior would typically be one component of a more complex emotional schema found to be underlying and maintaining a therapy client's symptom. The full schema is found to be a multilayered formation of interconnected meanings, models, expectations, roles, rules and tactics (Ecker et al., 2012; Ecker and Toomey, 2008). With such target learnings, the post-retrieval extinction protocol is not suitable, as a rule, for inducing reactivation, mismatch and counter-learning, unless its definition is made to be so general as to designate any form of counter-learning following target learning reactivation. In that case, the term "extinction" is a clear misnomer, as noted above.

9.2. Episodic memory interference

This article focuses on behaviorally induced erasure of semantic memory because such erasure produces transformational change, which is the most effective therapeutic change in that symptoms completely cease and a potent theme of emotional distress is fully resolved. Behaviorally induced erasure is the most thorough degree of memory interference by new learning, and it requires new learning that is an absolute contradiction of the target learning, as described and illustrated in Sections 5, 6 and 7. When episodic rather than semantic memory is chosen as the target of change by behavioral updating, complete erasure is not possible because it is neither feasible nor ethical to create counter-learning experiences that contradict the very existence of experiences remembered by the individual's declarative/factual memory (Dunbar and Taylor, 2017).

However, as discussed above in Sections 2 and 7.5.4, erasure is possible (and therapeutic) for certain components of the episodic memory of an experience, specifically the semantic knowledge schemas that existed at the time of the experience and generated its emotional quality, and which still exist as implicit components of the episodic memory. The disconfirmation, unlearning and erasure of those schemas during episodic memory recall transforms the emotional quality of the episodic memory, eliminating present-day symptoms that had been generated by the episodic memory's original emotional quality.

As noted in Section 5, a target learning can be strengthened, weakened, modified in its particulars, or erased by suitably designed new learning that becomes incorporated into the target memory during the reconsolidation window. If, rather than decisively contradicting and disconfirming any component of the target learning or memory, the new learning rather is designed either to counteract, dilute or scramble the original material, the result can be a partial but significant diminishment of expression of the original learning or memory. Numerous laboratory studies have demonstrated such effects on episodic, declarative, and motor memory (e.g., Fernández et al., 2016b; Hupbach et al., 2007, 2009; Walker et al., 2003; for reviews see Schiller and Phelps, 2011; Scully et al., 2017).

Clinical studies of episodic memory interference have focused mainly on a particular subclass of episodic memory, the memory of a traumatic incident, and have sought reduction of post-traumatic symptoms. Two such studies are examined in this section, below, using the ECPE/TRP framework to analyze the action of their clinical procedures according to research findings. Section 9.2.1 reviews a study that employed solely episodic memory interference and produced mild effects, whereas strong effects (in fact, the markers of erasure) were observed in the study covered in Section 9.2.2, which will be shown to have gone beyond episodic memory interference by carrying out the ECPE, so the study's impressive results cannot be ascribed to episodic memory interference.

How effectively episodic memory interference alone can reduce post-traumatic symptoms is an open question. The assumption that post-traumatic symptoms arise from episodic memory is in many cases incorrect because semantic memory is often the source, as discussed in Section 2 and as the case examples in Section 7 demonstrate (though that assumption is made by probably a majority of clinicians, in the author's experience). Certainly the particular symptom of intrusive flashback of perceptual and sensory memory of a discrete traumatic event is due to episodic memory, as are symptoms driven (consciously or non-consciously) by the emotional distress component of non-traumatic episodic memories. Experienced clinicians are also familiar with various post-traumatic symptoms that are actually tactics needed for suppressing traumatic episodic memory out of awareness (such as disconnection from affect, compulsive eating, continual self-distraction via compulsive focus on work, video games or pornography, and avoidant behaviors that prevent encounters with specific reminders of episodic memory). Such symptoms of episodic memory avoidance are produced not directly by the episodic memory that is being avoided, but rather by the expectation of being unsurvivably overwhelmed, damagingly devastated or even driven insane were the episodic memory to be experienced.

That expectation and the rule of avoidance that it dictates, which are semantic memory formations, are the targets of change for dispelling that class of symptoms, and they are dispelled by the ECPE via experiential disconfirmation: The therapist uses empathic accompaniment, the safety of the client-therapist relationship, and sensitive pacing (what is termed "small enough steps" in Coherence Therapy) to guide

the client to bring attention to the episodic memory in progressively fuller degrees, until the client has the disconfirming experience of in fact being capable of experiencing the memory without being damaged or overwhelmed by it. The client's resulting recognition of his or her own mental and emotional sturdiness is a significant therapeutic gain that persists independently of the particular features of the traumatic incident. However, full, conscious familiarity with those particulars can then be used to identify generalizations (semantic schemas) that formed based on the incident and that generate other symptoms (if such schemas have not already been retrieved via pathways other than episodic memory). Those schemas are then targets of erasure using the ECPE/TRP.

9.2.1. Kredlow and Otto, 2015

Kredlow and Otto (2015) tested an interference protocol (hereafter termed the KO protocol) applied to post-traumatic symptoms by having individuals who had experienced the Boston Marathon bombing write an autobiographical account of the experience. With the event memory reactivated by the writing exercise, subjects then listened to either a negative, positive, or neutral story (all unrelated to the bombing incident), or no story (the control condition). One week later, subjects were given exactly the same instructions to write an account of the incident. Each subject's two accounts were analyzed for replication of specific elements, revealing a modest but statistically significant overall reduction in recall of episodic memory details for negative-story interference relative to the control condition, whereas the neutral- and positive-story interference had smaller and non-significant effects.

Viewed in relation to the ECPE, the KO protocol can be characterized as follows:

As used by Kredlow and Otto, the protocol does not contain an identified or well-defined mismatch experience. Their article's account contains no mention of mismatch or prediction error or the necessity of same for triggering reconsolidation. Their study therefore seems to have been conducted under the early, mistaken assumption that reactivation alone destabilizes the memory of the event. Nevertheless, some subjects may have experienced mismatch due to the novelty of attending to the traumatic memory while writing about it for researchers, or mismatch may have been created by the post-reactivation stories that were intended as memory interference, or both. As noted in Section 4, the occurrence or non-occurrence of mismatch is determined by how the details of the target learning interact with the details of reactivation and post-reactivation experiences. Each subject in this study had a unique target learning, so there may have been subjects for whom mismatch and reconsolidation did not occur, while for others it may have occurred to varying degrees, since the strength and quality of mismatch probably varied greatly across subjects, corresponding to differences in the extent and degree of memory destabilization. This a major confounding factor in interpreting results obtained with this protocol, and it constitutes a lack of methodology evidence for utilizing reconsolidation.

Lack of erasure evidence precludes verification of having induced reconsolidation. The observed effect of the KO protocol, a partial reduction of episodic memory expression, leaves much room for competing explanations other than reconsolidation-enhanced interference. For example, the negative story condition could have exacerbated subjects' non-conscious discomfort with the memory of the event, producing stronger non-conscious resistance to attending to the episodic memory, which would appear to be an attenuation of episodic memory. Therapists frequently witness such self-protective inattention in far more glaring forms. Memory interference protocols applied to episodic memory of distressing events may be fundamentally beset with confounding factors in such ways.

Targeting episodic memory for dispelling fear and other emotional reactions has inherently limited therapeutic effectiveness. The ease with which episodic memory can be accessed and tested makes it an appealing target of change for researchers, but for clinical application its limitations are intrinsic and large. As the case examples in Section 7 show, the semantic components of emotional learnings are often the most potently symptom-generating constituents, and as a rule the semantic components are not accessed by focusing attention on the episodic components, that is, the consciously noticed perceptions, sensations, emotions and thoughts experienced in the event. Kredlow and Otto (2015, p. 36) acknowledge that a reduction in episodic, declarative memory "may or may not be related to the emotional aspects of a trauma memory," and, significantly, they found no change in the net

emotional valence of words used in each subject's pair of written accounts. Thus episodic memory interference proposed as a therapeutic strategy raises the question of whether impairing access to memory of a distressing event is therapeutic.

In light of the lack of both erasure evidence and methodology evidence, it seems premature for Kredlow and Otto to have concluded (p. 36), "These findings indicate that reconsolidation interference effects can be achieved for trauma-related episodic memories…."

9.2.2. Högberg, Nardo, Hällström and Pagani (2011)

Högberg, Nardo, Hällström and Pagani (2011), referred to hereafter as HNHP, conducted a review of the brain's neuroanatomical correlates of post-traumatic memory, made a number of inferences based on that information, and designed a treatment protocol intended to utilize memory reconsolidation for enhanced interference of episodic memory of traumatic incidents. The protocol proved to be highly effective in eliminating the post-traumatic symptomology of suicidal adolescents in a small number of sessions. That is a challenging population, and such excellent effectiveness serves well to demonstrate the promise of memory reconsolidation for enhancing therapeutic effectiveness. The HNHP protocol is therefore important to understand rigorously in relation to reconsolidation research findings.

The account given by HNHP appears to provide erasure evidence in both a single case study and a series of 14 cases. In the single case, a 17-year-old male had symptoms of difficulty falling asleep, severe nightmares, frequent, inexplicable day-time crying spells, breathing difficulties, feeling depressed, fainting, and nausea. Medical examinations had detected no physiological causes. In his fourth session, client reported that now "He has no problem falling asleep, no anxiety spells, and no somatic complaints. His score in MFQ is 0 and in WHO-5 92…" (Högberg et al., p. 94). At follow-up 3 months later, he reported the same. In the case series (p. 94), "14 suicidal adolescents…presented a post-trauma reaction with suicidality, insomnia, bodily symptoms, and disturbed mood regulation. … Of the adolescents 13 out of 14 had lost their severe symptoms within 4 to 20 treatment sessions. …The significant change towards normality after treatment was still present at the 22-month follow-up."

The strength of the erasure evidence reported by HNHP (essentially complete and lasting cessation of symptoms) implies that the treatment protocol successfully induced both the reconsolidation process and counter-learning, which in turn implies successful implementation of the steps of the empirically confirmed process of erasure (ECPE). However, the methodology evidence as presented by HNHP does not, on the face of it, clearly show fulfillment of the ECPE. How the treatment protocol in fact does fulfill the ECPE is described below, but is not identified by HNHP, and they neither discuss nor cite the relevant research findings. Thus the presentation of methodology evidence is significantly compromised.

Lacking the full research picture, HNHP adhere to and reassert researchers' early and widespread misconception, discussed in Section 4, that every reactivation of a memory is destabilizing, despite extensive disproof of that view accumulating for over a decade, as documented in Table 1. Consequently, HNHP do not specify how their protocol creates the mismatch experience needed to trigger memory reconsolidation. They also assert that certain methodological choices are inherent necessities for utilizing reconsolidation clinically, but on close consideration those necessities are seen to derive not from reconsolidation research findings, but rather from the authors' choice of methodology and their particular conceptualization of their methodology.

Nevertheless, despite conceptualization problems, their procedure facilitates all necessary experiences in the ECPE. The HNHP protocol steps, listed next, are followed by a review of the conceptualization given by HNHP and then by a research-based analysis of their protocol according to the ECPE and TRP defined above in Section 6.3.

HNHP preparation step: Creation of positive self-state. *Client selects and assembles positive memories and resources to create a readily available fear-free state of positive emotional valence.* The authors explain, (p. 92), "This can be achieved by relaxation exercises: breathing and meditation, safe-place imagery, perhaps walking on a treadmill or bicycling on a spinning cycle. …Once activated, the positive emotion can be anchored in a scene or image with focus on all senses. This means that specific instruction can be given to note the sight, hearing, proprioception, gustatory

feeling, and total body sensation in a state of safety and control."

HNHP step A: Sensory review. *While based in the prepared positive experience of safety and control, client mentally revisits and moves through the traumatic incident as an inner movie, with focus on the sensory experience.* During this re-experiencing, the client receives cues to also maintain concurrent awareness of the positive emotional state, with its perceptions and feelings of safety and control.

HNHP step B: Somatic review. *While based in the prepared positive experience of safety and control, client mentally revisits and moves through the traumatic incident as an inner movie, with focus on the somatic experience.* This is the same as Step A, except now client is attending to how his or her body is responding and feeling as the incident unfolds. This is a focus on autonomic nervous system activity.

HNHP step C: Motor impulse review. *While based in the prepared positive experience of safety and control, client mentally revisits and moves through the traumatic incident as an inner movie, with focus on motor impulses.* This is the same as Steps A and B, except now client is attending to how his or her body wants to move and take action in the incident, but was blocked or disallowed from doing so. When that is felt and identified, client is guided to replay a new version of the scene in which client fully carries out the motor impulse.

HNHP step D: How it should have been. *Client creates and moves through a new version of the traumatic incident in which all happens in the ideal manner client wishes would have been the case.* For example, client's parents console and protect him when he arrives home badly bruised after being mugged, instead of denigrating and raging at him and canceling plans for him to acquire his first car because of how "irresponsible" this incident shows he is.

HNHP step E: Positive future expectation. *Client envisions life going forward as desired, without symptoms, in the coming time period, and then experiences imaginal inner scenes of that happening.*

HNHP are emphatic in regarding episodic memory (event memory) as the source of post-traumatic symptom production and the target of therapeutic treatment. They define episodic memory as including the somatic (autonomic) and motor impulse aspects of the original experience. They state, for example, "the key phenomenon of trauma-reaction pathology – the memory disturbance with reliving episodic memory…" (p. 91). However, their own case example begins by stating, "On the first visit the patient complains mainly about difficulties in falling asleep, severe nightmares, and frequent day-time crying spells without having any hunch as to possible cognitive content or any memory associated with the complaints." There was no reliving of episodic memory in that client's presenting symptomology. Episodic memory is by definition any aspect of memory of the event itself. Absence of event memory in the presenting symptoms seems to imply a less than central role of episodic memory in symptom production.

Further, HNHP state (p. 92), "The aim of the treatment in cases of dysfunctional fear-memory reaction is to change the valence and the intensity of the traumatic memory and to increase control of memory retrieval. The goal of the treatment is to change a reliving intruding memory into a more distant episodic memory." Regarding how the protocol causes change, the authors state (p. 93) that because their Steps A-B-C are "a process with a combination of negative and positive affects, the original event can finally be re-experienced without fear, and the final outcome will be an emotionally less disturbing memory." With Steps A-B-C completed, "there is space for a new memory of the original events, and this is done [in Step D] as imagination of how the original event should have been in a positive fantasy. This is a repair fantasy that will be added to the memories associated with the traumatic event." The net effect of Steps A-B-C-D is asserted as being that "the traumatic exposure…is linked less with negative associations but rather to positive alternatives…." The authors re-emphasize the point in their concluding remarks (p. 94): "the approach presented focuses on changing old emotionally negative episodic memories and on creating new positive memories."

All of those statements express the authors' view that the source of symptom production is episodic memory and that the memory reconsolidation process can embed or graft or at least strongly link emotionally positive states and feelings into the incident's fear-generating episodic memory, causing the emotional valence of the new composite memory to shift from extremely negative and fearful (and therefore intrusive) to moderately positive (and therefore non-intrusive).

That is a memory interference model of how their

protocol produces the observed cessation of symptoms, and it is a main aspect of the authors' conceptualization. According to HNHP, positive-valence states and feelings become memory additives in four ways in their protocol: by maintaining the initially prepared positive state while revisiting the negative incident memory in Steps A, B and C; by allowing expression of the blocked motor impulse in Step C; by experiencing a positive version of the incident in Step D; and by envisioning and sampling a symptom-free future in Step E.

Thus, in the conceptualization of HNHP, there is no consideration of semantic memory (the existence and involvement of implicit emotional schemas); and there is no mention of the *erasure* of anything in memory. The conceptualization relies rather on memory reconsolidation to bring about a strong fastening of new, positive episodic memories to the original negative episodic memory, making it impossible for the new composite memory to generate the original symptoms. Erasure is maximal, optimal memory interference produced by counter-learning in the ECPE, nullifying much (though evidently not all, as shown by Ryan et al., 2015) of the encoding of original learning. In contrast, memory interference as intended and conceptualized by HNHP loads the original negative learning with competing positive qualities, largely counteracting but not necessarily fundamentally disconfirming and nullifying the original learning. The HNHP version of memory interference has been demonstrated in numerous controlled studies, as reviewed by Scully et al. (2017).

Observation of the markers of erasure by HNHP suggests that that their protocol accomplished not counteractive interference of episodic memory but full nullification of semantic memory and/or critical elements of episodic memory. The analysis below shows that the protocol indeed fulfills the empirically confirmed process of erasure (ECPE) in more than one way.

HNHP emphasize that establishing the initial, positive state of well-being is a necessity before beginning to access the affectively negative memories in Steps A, B and C. Their account seems to suggest that the sequence of positive followed by negative states is inherently required by the memory reconsolidation process. However, that is not the case, as the structure of the ECPE, derived directly from research, makes apparent. Rather, it is the particular design of the HNHP protocol and the aim of inducing a counteractive memory interference effect that necessitates the sequence of positive followed by negative states.

HNHP's conceptualization also includes a second intended symptom-dispelling effect driven by Step C of their protocol, the blocked motor impulse review and replay with freely expressed motor impulse. Invoking the fact that certain pairs of brain networks are coupled so that only one or the other can be activated, HNHP reason as follows (pp. 91–92): "It is well known that a trauma in which the victim is immobilized is often related to severe post-trauma symptomatology. Could it be that immobilization leads to the passive expression of fear with a high level of anxiety, and that active coping motor activity during the traumatic event leads to subsequent less severe later manifestations of fear reactions?" In other words, they hypothesize that Step C switches the episodic memory from being coupled to the parasympathetic system's passive mode (creating high anxiety when partnered with a fear memory) to the sympathetic system's active mode (creating much lower anxiety during fear-memory reactivation). Presumably, they posit that the reconsolidation process allows this switching to be a decisive and permanent rewiring of neural connections.

By examining the HNHP protocol in relation to the ECPE and TRP methodologies defined in this article, additional, more potent memory-changing effects become apparent, namely erasure of a number of semantic, symptom-generating emotional learnings. The ECPE/TRP perspective calls for examining the HNHP protocol in terms of the emotional learnings that it subjects to the empirically confirmed process of erasure.

As noted, HNHP do not consider the existence and the symptom-generating action of emotional schemas in semantic memory, an important focus of ECPE/TRP methodology. The role of semantic memory is most apparent in therapy cases in which long-term post-traumatic symptoms are found to be generated entirely by semantic memory and not by episodic memory, as exemplified by the case studies detailed in Section 7. Studying the action of the HNHP protocol through ECPE/TRP lens reveals that episodic memory activation served as a portal for accessing other aspects of emotional learning and memory formed in response to the incident, including semantic (schema) memory,

to produce the observed symptom cessation.

How the HNHP protocol implements the ECPE becomes apparent by identifying the subjective experiences produced by the HNHP protocol. That analysis reveals how several different components of the HNHP protocol fulfill the ECPE and thereby transform symptom-generating memory for potent therapeutic effects. The ECPE/TRP conceptualization takes account of the subjectivity of emotional learning and unlearning, and in that way reveals dynamics of emotional learning and unlearning that are missed in purely reductive analyses based upon external procedures and the functions of brain regions.

The ECPE/TRP analysis, which follows, identifies these specific, symptom-generating semantic constructs that the client unlearns through the HNHP protocol: (1) the necessity of suppressing memory of the incident, based on the construed incapacity of the self to face and feel the intense suffering in the incident, (2) the state of helplessness originally felt and now expected in the class of situations defined by the incident's salient features, and (3) the necessity of avoiding facing and affectively experiencing the deprivation of what was needed but missing, resulting in the traumatizing incident.

Each of those emotional learnings is mismatched, disconfirmed, nullified and erased by the HNHP protocol, as understood from the ECPE/TRP perspective. From a clinician's viewpoint, that is a powerful set of therapeutic effects, and in many cases that set covers all of the main areas requiring resolution in order for an individual to get free of post-traumatic symptomology. To have designed a well-defined protocol capable of having those effects in a small number of sessions is a significant achievement. The range of applicability of the HNHP protocol is addressed at the end of this subsection. How the HNHP protocol acts on each of the above three target learnings is considered next.

Regarding target learning (1), urgent avoidance of feeling the suffering in the incident: In HNHP Steps A–B–C, the client has the experience of (a) being capable of revisiting, facing and feeling the full memory of the original incident without being destructively overwhelmed by doing so, and (b) being free to do so without costly social consequences of feeling emotional distress and vulnerability. That experience serves as a specific counter-learning that disconfirms what had been the implicit presuppositions of being incapable of, and/or disallowed from, facing and feeling the full memory. A client who non-consciously expects unsurvivable overwhelm or severe and unacceptable social costs does so on the basis of (semantic) emotional learnings to that effect. Different therapy clients have different schemas that urgently require avoidance of feeling emotion, with each schema centered on knowledge of a particular suffering that would result and must be avoided. HNHP Steps A–B–C guide a very thorough accessing of the experience of the original incident, thereby creating a decisive disconfirmation and erasure of the expectation of overwhelm. Some schemas of social prohibition could also be erased, but others would not be because the relationship with the therapist is not representative of social relationships for many clients, so the experience of visibly feeling emotional and somatic reactions in therapy would not necessarily disconfirm all such schemas.

The HNHP protocol does not call for explicit retrieval of the schemas involved in symptom production, so the therapist remains unaware of their specific contents. In contrast, the TRP does call for retrieving each target schema into affective experience with explicit verbalization, so that the therapist, equipped with detailed knowledge of schema contents, can guide new experiences that are accurately designed to disconfirm the unique content of each target schema, making achievement of erasure maximally reliable.

Regarding target learning (2), vulnerability to helplessness: In incidents where a natural, self-protective motor impulse arises but is blocked, preventing enactment of the impulse, the immediate result is a subjective construal and feeling of helplessness and defenselessness, which in turn maximizes the subjective construal and feeling of endangerment. Helplessness is the ingredient that makes the incident traumatizing, because (a) helplessness is both maximally terrifying in itself and (b) it actually converts a merely unpleasant situation into a highly endangering one due to the absence of a self-protective action that would avoid harm. That is presumably why, as HNHP note, immobilization during a frightening incident is positively correlated with post-traumatic symptoms. Enacting the self-protective motor response during a frightening incident (such as standing up and walking or running away) would in many cases even prevent the situation from being perceived as significantly dangerous or frightening in the first place. In other words, in many

(but not all) post-traumatic cases, the original incident reached traumatizing levels of helplessness and endangerment *because* of blocked motor impulses. Motor blockage during the incident can be caused in a number of different ways. It can be non-consciously compelled by a semantic emotional schema prohibiting assertive action, as in a freeze response; it can be enforced by a conscious assessment of the risk of expressing the impulse; or immobility can be enforced by physical constraints that make it impossible to express the impulse.

The effect of HNHP Step C, according to the ECPE/TRP perspective, is creation of the subjective experience of actually having the personal power and agency to keep oneself safe in the very circumstances where the original experience (and learning) was helplessness. That experience of unblocked empowerment is a counter-learning that mismatches and precisely contradicts and disconfirms the existing, semantic-memory expectation of being helpless in the class of situations having the original incident's salient features.

In that way, HNHP Step C in itself implements the complete ECPE and erases the learned, semantic-memory expectation of helplessness. Such nullification of the helplessness component of the original memory immediately de-traumatizes the entire memory in many (but not all) cases of post-traumatic symptoms, in the author's experience. This step alone can therefore result in the markers of erasure, ending post-traumatic symptoms, for many such clients. In addition, if it was an emotional schema that disallowed enactment of the motor impulse, fulfilling the motor impulse in Step C also subjects that specific schema to the ECPE, which can erase it.

This "empowered reenactment" technique (for an example of which see Ecker et al., 2012, pp. 86–91) is suitable and safe only for traumatic incidents in which there was perception of danger of imminent harm, making a self-protective response physically and humanly possible. Then an empowered reenactment can be subjectively credible to the client as a disconfirmation of helplessness. When that is not the case (for example, in experiencing an unforeseeable bomb explosion), a subjective replaying of the experience aiming for empowered reenactment is likely to be retraumatizing rather than therapeutic.

HNHP conclude in a very general manner that "the motor component in the emotional memory is central to pathology" (p.94). That statement would be true if it ended with three more words: "in many cases." In the general form given, that conclusion is not warranted, in light of the following considerations. HNHP addressed a specialized class of target learnings (those that maintain post-traumatic symptoms) and a specialized treatment protocol (one they designed based on making inferences from a review of the neurological correlates of post-traumatic symptoms). The authors arrived at the above broad conclusion with no apparent reference to research (discussed in Sections 4 and 5 above) showing that the outcome of behavioral updating via memory reconsolidation is determined by the interaction of the specific elements in the target learning with the specific elements of the post-mismatch counter-learning experience. Blocked motor impulses are involved in forming the target learning in many cases of post-traumatic symptoms, but not all such cases (for example, those detailed in Section 7 of this article, involving sexual molestation and annihilative attachment rupture). If the target learning was formed in experiences that did not involve blocked motor impulses, HNHP Step C would be irrelevant to the target memory and would therefore have no therapeutic effect, in great contrast to the transformational change it can produce when relevant. The details of the target learning completely determine what experiences will, or will not, have a disconfirming and nullifying effect, and the details of the target learning are idiosyncratic and must be carefully discerned, and not assumed, in every clinical case. Thus understanding the specific findings of memory reconsolidation research is indispensable for interpreting clinical results and for developing clinical methods for the full range of symptomologies based in emotional learning.

Regarding target learning (3), avoidance of feeling unmet needs and the pain of mistreatment: HNHP Step D targets this (though the authors do not comment on it) by guiding the client to identify, in contrast to the intensely negative, unkind treatment suffered at the hands of life in the incident, the kind treatment she or he wishes life would have provided instead.

As noted, HNHP regard Step D as generating further positive-valence memories that become linked to the target negative memory of the incident, strengthening the interference of the latter by the former. Step D does significantly more than that, however, as understood in terms of the ECPE/TRP framework. It carries

out the entire ECPE for this target learning.

Consider the example of Step D given above: the adolescent client imagines that his parents caringly console and protect him when he arrives home badly bruised after being mugged, instead of denigrating and raging at him and canceling plans for him to acquire his first car because of how "irresponsible" this incident shows he is. This imaginal experience would of course tend to generate positive feelings of receiving the desired and needed consoling and protecting. However, in probably a majority of cases it would also evoke long-suppressed, quite distressing feelings over having received not the needed, kind responses, but unkind responses in many instances over many years. Hurt, anger, unworthiness, emotional abandonment, neglect, betrayal, aloneness, being unseen, not mattering, desolation, despair and painful grief, among other feelings and reactions, can emerge strongly from a sustained focus on the caring responses that were needed but missing. This is familiar territory for almost any experienced practitioner of in-depth therapy, particularly those who address issues of insecure attachment and low self-esteem, which are among the most frequently encountered issues in therapy. Similarly as for target learning (1), most people are highly avoidant of allowing direct experience of such feelings and meanings, so they are chronically suppressed out of awareness. It therefore requires clinical skills of several kinds, both technical and relational, to facilitate de-suppression and affective experiencing of such emotions and meanings. For the client, the experience of allowing such emotions and meanings to emerge and be felt, and of having the capacity for that without being destroyed by it and actually valuing the deep connection-with-self it entails, disconfirms and erases the learned expectation of being damaged or destroyed by such feelings and the urgent necessity of suppressing them. The result is cessation of a wide range of avoidance behaviors, affect-phobic responses and generalized anxiety, all of which are concomitants of carrying significant levels of suppressed distress.

HNHP regard the initially prepared positive state, which is maintained and attended to throughout Steps A, B and C, as both a source of interfering positive input to the negative target memory and as an emotional stabilizer for the client while accessing distressing material. Once again, those effects are strongly plausible, but there are additional significant therapeutic effects that become apparent from the ECPE/TRP perspective.

Attending to perceptions and feelings of safety and control while also attending to reactivated negative memories is a technique used in several systems of trauma therapy (such as EMDR, NLP, progressive counting, and acupoint tapping) and is widely termed *dual focus* (e.g., Lee et al., 2006). By juxtaposing the target memory's "I'm in danger and don't have control over it" with "actually I'm safe and in control," the dual focus creates a mismatch experience, a violation of the target memory's model of reality. As explained in Section 4, a mismatch experience destabilizes the target memory's neural encoding, allowing it to be erased by counter-learning. Dual focus therefore carries out the first two of the three experiences in the ECPE (TRP Steps 1 and 2).

Dual focus also promotes the third and final required experience, that of counter-learning, by maintaining a self-state in which the client has maximal access to disconfirming knowledge. As noted in Sections 5 and 6.3, an extended experience of contrary, disconfirming knowledge, concurrent and juxtaposed with the experience of the reactivated target learning, is the needed counter-learning that completes the ECPE (TRP Step 3). Clinicians who utilize dual focus methods regularly observe the enhanced accessing of disconfirming knowledge that it promotes. Ecker (2015c) has proposed that the enhancement results from the client attending to the contents of the target memory from a subjective viewpoint that is outside of the affective self-state normally produced by that memory. Normally, attending to the target memory reactivates it, causing the client's consciousness to inhabit the memory and merge into the affective experience of the memory's percepts, somatics and semantic elaborations. In that subjective self-state, the contents of the memory completely fill the field of awareness and feel compellingly real, while the client's myriad other self-states and knowings disappear and are inaccessible for all practical purposes. In contrast, dual focus keeps the client's consciousness anchored and positioned in a safe context outside the memory while attending to the memory's contents. In this state of *unmerged attending* (Ecker, 2015c), the client's other self-states and knowings remain accessible, and existing knowledge that is contrary to the target memory can readily activate into foreground awareness due the brain's automatic

detection of mismatches, a background process always scanning current conscious experience (Ecker and Hulley, 2017a; Ecker et al., 2012). That activation of a contrary knowing in response to the target learning creates the juxtaposition, noted just above, that drives the counter-learning needed for unlearning, nullification and erasure to occur.

Regarding the range of applicability of the HNHP protocol, the foregoing discussion implies the following considerations. (a) The protocol is designed for treating post-traumatic symptomology and as such is organized completely around imaginal re-experiencing of a critical incident. Many therapy clients present symptoms rooted in distressing experiences that were repetitively part of the fabric of life in one or more contexts (family, social, work, or other), rather than acute incidents. How the protocol could be applied effectively in such cases is not readily apparent. (b) One of the protocol's main components, Step C for blocked motor impulses, to which HNHP give much discussion and emphasis, would be ineffective in cases where target learnings (whether episodic or semantic) involve no blocked motor impulses. Not all traumatizing incidents evoke motor impulses, but probably all do create the experience and expectation of helplessness. To the extent that Step C is the protocol's main way of erasing the learned expectation of helplessness in the target incident's class of situations, the protocol could be relatively ineffective in such cases. (c) The HNHP protocol focuses only on episodic memory, so it could fail to be effective in some cases where only implicit semantic memory drives symptom production (such as the lifelong reactive anger driven by the attributed meaning "it happened only to me" in the case example in Section 7.1). As noted earlier, accessing a specific, symptom-generating mental model or attributed meaning in implicit semantic memory is not prescribed by the protocol and is therefore by no means assured.

In summary, though the HNHP protocol has a limited range of applicability, the ECPE/TRP analysis of its operation reveals numerous strengths, beyond those described by HNHP, by identifying symptom-generating emotional learnings that are subjected to the empirically confirmed process of erasure. HNHP conceptualized their protocol as acting only upon episodic memory of a traumatic incident and as relying on counteractive memory interference effects. The ECPE/TRP analysis shows that the protocol uses episodic memory as a portal for accessing certain implicit, symptom-generating emotional schemas in semantic memory, and erases those schemas by subjecting them to the ECPE. Thus this study by HNHP is not actually a test of the strategy of counteractive interference of episodic memory, and the effectiveness of the protocol in eliminating post-traumatic symptoms is not a demonstration of the effectiveness of that strategy.

Even if pure counteractive interference of episodic memory (with no semantic memory effects) were to be implemented and shown to be effective at alleviating certain symptoms, its use in psychotherapy is contra-indicated, in the author's opinion, because making it more difficult to remember a distressing experience would be iatrogenic, not therapeutic. As any experienced practitioner of in-depth therapy knows, actual resolution and nullification of the effects of the worst experiences in one's life occur through deeply and fully revisiting one's distressing life experiences and consciously recognizing what was suffered and what coping tactics one put in place. Cutting off the episodic memory pathway to that material would be significantly disadvantageous and might perpetuate rather than eliminate distress.

9.3. Emotional arousal: Lane, Ryan, Nadel and Greenberg, 2015

Lane, Ryan, Nadel and Greenberg (2015), referred to hereafter as LRNG, state, "In this paper, we propose that change occurs by activating old memories and their associated emotions, and introducing new emotional experiences in therapy enabling new emotional elements to be incorporated into that memory trace via reconsolidation" (p. 3). Combining an emotional experience of the activated target memory with a new *emotional* experience is the condition required for producing change, according to LRNG. They reiterate that emphasis by later stating, "This model highlighting the importance of new emotional experiences…" (p. 16). The emphasis placed on emotional arousal is also apparent in the article's title, "Memory Reconsolidation, Emotional Arousal and the Process of Change in Psychotherapy."

LRNG show that assigning such importance to emotional arousal in therapy has much support from psychotherapy process research, which has consistently found a strong correlation between successful thera-

peutic outcome and experiences in therapy sessions of previously blocked emotion and/or emotional meaning. That correlation has also been cited by Ecker (2013) and Ecker et al. (2012, pp. 153–155) as constituting a significant refutation of nonspecific common factors theory, which is the prevailing model of therapeutic effectiveness and of why all therapy systems subjected to randomized controlled trials have scored essentially the same level of efficacy for over 70 years (Wampold, 2001, 2015).

While the importance of experiencing emotion in psychotherapy appears to be well established, what research has shown about the relationship between emotion and memory reconsolidation is a different matter. It seems inevitable that many of LRNG's readers would acquire the understanding that reconsolidation requires emotional arousal, but that would be a misunderstanding, because reconsolidation research shows that no such requirement exists, as is further explained below. Also, LRNG's treatment includes no consideration of, or reference to, any of the extensive research that identifies the specific experience-driven process of memory reconsolidation (namely, the requirements for a memory mismatch/prediction error experience for destabilizing a target learning, and for a disconfirmation or counter-learning experience for erasing a target learning, with both experiences designed strictly according to the specific content of the target learning being addressed, as reviewed above in Sections 4 and 5). Rather, the authors extensively refer to multiple trace theory and its assertion that every reactivation of a memory is destabilizing, which, as discussed in Section 4, was reconsolidation researchers' early misconception, disproven since 2004 by all of the studies listed in Table 1.

Similarly, the central premise of LRNG regarding the necessity of emotional arousal for recruiting reconsolidation is contradicted by extensive research (Ecker et al., 2015). This is one of several widespread misconceptions about memory reconsolidation (Ecker, 2015a). The independence of the reconsolidation process from emotion is apparent in the fact that memory mismatch has been shown to induce destabilization, launching the reconsolidation process, for numerous types of memory ranging from declarative, neocortical, factual learnings devoid of emotional content (e.g., target learning consisting of a set of meaningless syllable pairings; Forcato et al., 2009) to subcortical, intensely emotional learnings (e.g., target learning consisting of a safety platform position in animal studies; Morris et al. 2006). It is firmly established that emotional arousal per se is neither intrinsic to nor needed for inducing reconsolidation and erasure.

If the target learning happens to involve emotion, as is nearly always the case in psychotherapy, then its reactivation (the first step in the empirically confirmed process of erasure and TRP Step 1) of course entails an experience of that emotion. The visible presence of that emotion gives a therapist an important indication that an adequate degree of target learning reactivation has occurred. Those are clinical pragmatics having nothing to do with the inherent nature of memory reconsolidation.

Even with a reactivated, emotionally intense target learning, however, the disconfirmation experience needed next for mismatch, destabilization, nullification and erasure of the target learning (TRP Steps 2 and 3) is not required to be emotional in itself, as can be seen in both laboratory and clinical observations. This too is not in agreement with the methodology defined by LRNG, which calls for "new emotional experiences in therapy enabling new emotional elements to be incorporated…." The disconfirming new experience or knowledge must be experienced as being unmistakably true and real, but that is not necessarily an emotional experience. As an example of a laboratory observation of a non-emotional new experience inducing erasure of an emotional target learning, consider that after Schiller et al. (2010) reactivated their human subjects' Pavlovian fear response, the disconfirming experience that then destabilized and subsequently erased the fear was the completely neutral experience of nothing happening: No electric shock occurred.

As examples of corresponding clinical observations, each of the case studies in Section 7 shows an intensely emotional target learning being mismatched, disconfirmed and nullified by a juxtaposition of the target learning with a contrary knowing that was just a plain fact, that is, a knowing that was not in itself experienced as significantly emotional prior to the juxtaposition experience. The woman with lifelong anger rooted in the implicit construal, "Life allowed this to happen only to me," formed a juxtaposition of that learning with the contrary knowing that sexual molestation of children is widespread, which was a familiar fact already in her possession and which had no great

personal emotional significance before the juxtaposition occurred. Only when that familiar fact was newly perceived in relation to (i.e., in juxtaposition with) the target learning did it acquire intense emotional significance and charge, because in that context the familiar fact suddenly functioned as the liberating disconfirmation of a construal that had been distressing her profoundly for a lifetime. Her anger and her view of the world as arbitrary and unfair then disappeared. The woman with attachment trauma maintaining chronic terror and a kinesthetic plunge through the floor with every interpersonal misattunement, all rooted in the implicit construal, "There is no one who maintains shared reality," formed a juxtaposition of that learning with the contrary recognition of the plain fact that actually there is a large subset of people who do maintain shared reality and repair shared reality as needed, including her own boyfriend. Her symptoms then disappeared.

In the empirically confirmed process of erasure, the juxtaposition experience that unlearns, nullifies and erases an emotionally intense target learning does not require an emotionally intense contrary knowing or counter-learning for this reason: The essence of the target learning is its implicit model of reality (its semantic content), not the emotion that arises from that construal of reality. It is the model that is being disconfirmed and unlearned, not the emotion. (For further discussion of this point, see Ecker, 2015a, pp. 25–30.) A transformational change in the model immediately produces profound change in the emotion generated. That is clearly apparent in the two case examples noted in the previous paragraph.

In response to the critique above, the author would expect the G of LRNG to argue, in essence, that he regularly observes the markers of erasure as a result of carrying out the LRNG clinical methodology, so the critique must be incorrect. To which the author would reply: Various systems of therapy carry out reconsolidation and erasure with no conceptualization of doing so. LRNG's conceptualization has the problems indicated above regarding how the reconsolidation process operates, so it is not an accurate account of LRNG's methodology, which indeed works in practice. By combining emotional reactivation of the target learning with a very different emotional experience of the original situation, a disconfirmation of model occurs implicitly, even though the attention of therapist and client may be focused on derivative emotion. The methodology works, and yet the critique is correct and necessary. At stake is the degree to which mental health clinicians understand memory reconsolidation accurately and therefore become able to utilize its full potential for relieving suffering.

10. Conclusion

The empirically established existence of an erasure process now allows a new coordination between the observations made by memory researchers and those made by mental health clinicians, who have anecdotally reported the distinctive markers of such erasure for many decades. The present article maintains that, owing to a fortunate convergence of clinical observations and brain research, the translation of reconsolidation research findings into effective psychotherapeutic use appears to have advanced at an accelerated pace unexpected by and still largely unknown to neuroscience researchers.

It is widely assumed, as asserted by Elsey and Kindt (2017b, p. 477), that "uncontrolled trials and case studies alone are insufficient for demonstrating the therapeutic potential of reconsolidation-based procedures, as they cannot rule out factors such as placebo effects, or convincingly demonstrate that reconsolidation is the best explanation for the observed treatment effect." The present article challenges that *a priori* view by showing that clinicians facilitate the same distinct sequence of experiences that neuroscientists have identified as being required for erasure, and then observe the same distinct markers of erasure that neuroscientists use in laboratory studies as confirmation of behavioral memory updating through memory reconsolidation because there is no other known process that can produce these markers of erasure. The detailed correspondences of unique process and unique outcome constitute a plausible demonstration that reconsolidation is the best explanation for the observed treatment effect.

That clinical advance has been possible largely because of the clinician's attention to the experiential, subjective dimension of the process of change. This article has introduced the principle that optimal clinical translation requires understanding the memory reconsolidation process in terms of internal, subjective experiences, as distinct from external procedures used for creating those experiences.

It is the author's hope that this article will help to synchronize researchers and clinicians by opening a channel of collaborative communication for the shared goal of a new level of therapeutic effectiveness through deft, versatile use of the reconsolidation process. Researchers Elsey and Kindt (2017a, p. 116), seeing the magnitude of the potential advance for psychotherapy, concluded their review by stating, "…if reconsolidation-based procedures become a viable treatment option, then they would be one of the first mental health treatments to be directly derived from basic neuroscience research. This would surely be a triumph for the scientific study of mind and brain." The present article has suggested that, in the work of the clinical early adopters of the empirically confirmed process of endogenous erasure, that triumph may already be materializing.

Acknowledgements

The author wishes to express much gratitude to each of several colleagues for valuable feedback generously provided in the development of this article: Dr. Sara K. Bridges, Dr. Hanna Levenson, Dr. Melissa Reading, and Robin Ticic. Neuroscience researcher Dr. Javiera Oyarzún generously provided a detailed critique of a presentation by the author, which was of much value for clearly and accurately representing some of the more nuanced aspects of reconsolidation research in relation to clinical application. Helpful input was also received from Dr. Gregory Carson, Laurel Hulley, and Alexandre Vaz.

References

Agren, T. (2014). Human reconsolidation: A reactivation and update. *Brain Research Bulletin, 105*, 70–82. doi:10.1016/j.brainresbull.2013.12.010

Agren, T., Björkstrand, J., & Fredrikson, M. (2017). Disruption of human fear reconsolidation using imaginal and in vivo extinction. *Behavioural Brain Research, 319*, 9–15. doi:10.1016/j.bbr.2016.11.014

Agren, T., Engman, J., Frick, A., Björkstrand, J., Larsson, E.M., et al. (2012). Disruption of reconsolidation erases a fear memory trace in the human amygdala. *Science, 337*, 1550–1552. doi:10.1126/science.1223006

Alexander, F., & French, T. M. (1946). *Psychoanalytic therapy: Principles and application*. New York, NY: Ronald Press.

Alfei, J. M., Ferrer Monti, R. I., Molina, V. A., Bueno, A.M., & Urcelay, G.P. (2015). Prediction error and trace dominance determine the fate of fear memories after post-training manipulations. *Learning & Memory, 22*, 385–400. doi: 10.1101/lm.038513.115

Auber, A., Tedesco, V., Jones, C., Monfils, M., Chiamulera, C. (2013). Post-retrieval extinction as reconsolidation interference: Methodological issues or boundary conditions? *Psychopharmacology, 226*, 631–647. doi:10.1007/s00213-013-3004-1

Badenoch, B. (2011). *The brain-savvy therapist's workbook.* New York: W. W. Norton & Co.

Baker, K. D., McNally, G. P., & Richardson, R. (2013). Memory retrieval before or after extinction reduces recovery of fear in adolescent rats. *Learning & Memory Cold Spring Harbor N*, *20*, 467–473. doi:10.1101/lm.031989.113

Balderas, I., Rodriguez-Ortiz, C. J., & Bermudez-Rattoni, F. (2013). Retrieval and reconsolidation of object recognition memory are independent processes in the perirhinal cortex. *Neuroscience, 253*, 398–405. doi:10.1016/j.neuroscience.2013.09.001.

Barreiro, K. A, Suárez, L. D., Lynch, V. M., Molina, V. A., & Delorenzi, A. (2013). Memory expression is independent of memory labilization/reconsolidation. *Neurobiology of Learning & Memory, 106*, 283–291. doi:10.1016/j.nlm.2013.10.006

Bateson, G. (1979). Mind and nature: A necessary unity (advances in systems theory, complexity, and the human sciences). Hampton Press. ISBN 1-57273-434-5

Beckers, T., & Kindt, M. (2017). Memory reconsolidation interference as an emerging treatment for emotional disorders: Strengths, limitations, challenges, and opportunities. *Annual Review of Clinical Psychology, 13*, 99–121. doi:10.1146/annurev- clinpsy- 032816- 045209

Bjork, R. A. (1992). Interference and memory. In L. R. Squire (Ed.), *Encyclopedia of learning and memory* (pp. 283–288). New York: Macmillan.

Björkstrand, J., Agren, T., Frick, A., Engman, J., Larsson, E. M., Furmark, T., & Fredrikson, M. (2015). Disruption of memory reconsolidation erases a

fear memory trace in the human amygdala: An 18-month follow-up. *PLoS ONE*, *10*(7), e0129393. doi:10.1126/science.1223006

Bos, M. G. N., Beckers, T., & Kindt, M. (2014). Noradrenergic blockade of memory reconsolidation: A failure to reduce conditioned fear responding. *Frontiers of Behavioral Neuroscience, 8*, 1–8. doi:10.3389/fnbeh.2014.00412

Bouton, M. E. (2004). Context and behavioral processes in extinction. *Learning & Memory, 11*, 485–494.

Bouton, M. E., Mineka, S., & Barlow, D. H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review, 108*, 4–32. doi:10.1037/0033-295X.108.1.4

Brunet, A., Poundja, J., Tremblay, J., Bui, E., Thomas, E., Orr, S. P., et al. (2011). Trauma reactivation under the influence of propranolol decreases posttraumatic stress symptoms and disorder: Three open-label trials. *Journal of Clinical Psychopharmacology, 31*, 547–550. doi:10.1097/JCP.0b013e318222f360

Caffaro, P. A., Suarez, L. D., Blake, M. G., & Delorenzi, A. (2012). Dissociation between memory reactivation and its behavioral expression: Scopolamine interferes with memory expression without disrupting long-term storage. *Neurobiology of Learning & Memory, 98*, 235–245. doi:10.1016/j.nlm.2012.08.003

Cai, D.J., Aharoni, D., Shuman, T., Shobe, J., Biane, J., Song, W., et al. (2016). A shared neural ensemble links distinct contextual memories encoded close in time. *Nature, 534*, 115–118. doi:10.1038/nature17955

Cammarota, M., Bevilaqua, L. R. M., Medina, J. H., & Izquierdo, I. (2004). Retrieval does not induce reconsolidation of inhibitory avoidance memory. *Learning & Memory, 11*, 572–578. doi:10.1101/lm.76804

Castonguay, L. G., & Hill, C. E. (Eds.) (2012). *Transformation in psychotherapy: Corrective experiences across cognitive behavioral, humanistic, and psychodynamic approaches.* Washington DC: American Psychological Association.

Clarke, D.M. (1999). Anxiety disorders: Why they persist and how to treat them. *Behaviour Research and Therapy, 37(Supplement 1)*, S5–S27. doi:10.1016/S0005-7967(99)00048-0

Clem, R. L., & Huganir, R. L. (2010). Calcium-permeable AMPA receptor dynamics mediate fear memory erasure. *Science, 330*, 1108–1112. doi:10.1126/science.1195298

Clem, R. L., & Schiller, D. (2016). New learning and unlearning: strangers or accomplices in threat memory attenuation? *Trends in Neuroscience, 39*(5), 340–351. doi:10.1016/j.tins.2016.03.003

Coccoz, V., Maldonado, H., & Delorenzi, A. (2011). The enhancement of reconsolidation with a naturalistic mild stressor improves the expression of a declarative memory in humans. *Neuroscience, 185*, 61–72. doi:10.1016/j.neuroscience.2011.04.023

Coughlin, P. (2016). *Maximizing effectiveness in dynamic psychotherapy*. New York: Routledge.

Courtois, C. A. (2004). Complex trauma, complex reactions: Assessment and treatment. *Psychotherapy: Theory, Research, Practice, Training, 41*(4), 412–425. doi:10.1037/0033-3204.41.4.412

Debiec, J., Díaz-Mataix, L., Bush, D. E. A., Doyère, V., & LeDoux, J. E. (2010). The amygdala encodes specific sensory features of an aversive reinforcer. *Nature Neuroscience, 13*, 536–537. doi:10.1038/nn.2520

Debiec, J., Doyère, V., Nader, K., & LeDoux, J. E. (2006). Directly reactivated, but not indirectly reactivated, memories undergo reconsolidation in the amygdala. *Proceedings of the National Academy of Sciences, 103*, 3428–3433. doi:10.1073/pnas.0507168103

Debiec, J., & LeDoux, J.E. (2004). Disruption of reconsolidation but not consolidation of auditory fear conditioning by noradrenergic blockade in the amygdala. *Neuroscience, 129*, 267–272. doi:10.1016/j.neuroscience.2004.08.018

Debiec, J., LeDoux, J. E., & Nader, K. (2002). Cellular and systems reconsolidation in the hippocampus. *Neuron, 36*, 527–538. doi:10.1016/S0896-6273(02)01001-2

Delorenzi, A., Maza, F. J., Suárez, L. D., Barreiro, K., Molina, V. A., & Stehberg, J. (2014). Memory beyond expression. *Journal of Physiology (Paris), 108*, 307–322. doi:10.1016/j.jphysparis.2014.07.002

Dennis, T. S., & Perrotti, L. I. (2015). Erasing drug memories through the disruption of memory

reconsolidation: A review of glutamatergic mechanisms. *Journal of Applied Biobehavioral Research, 20*, 101–129. doi:10.1111/jabr.12031

Díaz-Mataix, L., Debiec, J., LeDoux, J. E., & Doyère, V. (2011). Sensory specific associations stored in the lateral amygdala allow for selective alteration of fear memories. *The Journal of Neuroscience, 31*, 9538–9543. doi:10.1523/jneurosci.5808-10.2011

Díaz-Mataix, L., Ruiz Martinez, R. C., Schafe, G. E., LeDoux, J. E., & Doyère, V. (2013). Detection of a temporal error triggers reconsolidation of amygdala-dependent memories. *Current Biology, 23*, 1–6. doi:10.1016/j.cub.2013.01.053

Doyére, V., Debiec, J., Monfils, M. H., Schafe, G. E., & LeDoux, J. E. (2007). Synapse-specific reconsolidation of distinct fear memories in the lateral amygdala. *Nature Neuroscience*, *10*, 414–416. doi:10.1038/nn1871

Dunbar, A. B., & Taylor, J. R. (2017). Reconsolidation and psychopathology: Moving towards reconsolidation-based treatments. *Neurobiology of Learning and Memory, 142*(Pt A), 162–171. doi: 10.1016/j.nlm.2016.11.005

Duncan, B. L., Miller, S. D., Wampold, B. E., & Hubble, M. A. (Eds.) (2009). *The heart and soul of change: Delivering what works in therapy* (2nd ed.). Washington, DC: American Psychological Association Press.

Dunsmoor, J. E., Mitroff, S. R., & LaBar, K. S. (2009). Generalization of conditioned fear along a dimension of increasing fear intensity. *Learning & Memory, 16*, 460–469. doi:10.1101/lm.1431609

Dunsmoor, J.E., Niv, Y., Daw, N., & Phelps, E.A. (2015). Rethinking extinction. *Neuron, 88*(1), 47-63. doi:10.1016/j.neuron.2015.09.028

Duvarci, S., Mamou, C. S., & Nader, K. (2006). Extinction is not a sufficient condition to prevent fear memories from undergoing reconsolidation in the basolateral amygdala. *European Journal of Neuroscience, 24*, 249–260. doi:10.1111/j.1460-9568.2006.04907.x

Duvarci, S., & Nader, K. (2004). Characterization of fear memory reconsolidation. *The Journal of Neuroscience, 24*, 9269–9275. doi:10.1523/jneurosci.2971-04.2004

Dymond, S., Dunsmoor, J.E., Vervliet, B., Roche, B., & Hermans, D. (2015). Fear generalization in humans: systematic review and implications for anxiety disorder research. *Behavior Therapy, 46*, 561–582. doi:10.1016/j.beth.2014.10.001

Ecker, B. (2006, July). *The effectiveness of psychotherapy*. Keynote address, 12th Biennial Conference of the Constructivist Psychology Network, University of California, San Marcos.

Ecker, B. (2008, September). Unlocking the emotional brain: Finding the neural key to transformation. *Psychotherapy Networker, 32*(5), 42–47, 60.

Ecker, B. (2010, January). The brain's rules for change: Translating cutting-edge neuroscience into practice. *Psychotherapy Networker, 34*(1), 43–45, 60.

Ecker, B. (2011, January 13). Reconsolidation: A universal, integrative framework for highly effective psychotherapy [Web log post]. Retrieved July 10, 2017, from http://bit.ly/1zjKtMr

Ecker, B. (2013). Nonspecific common factors theory meets memory reconsolidation: A game-changing encounter? *The Neuropsychotherapist*, *2,* 134-137. doi:10.12744/tnpt(2)134-137

Ecker, B. (2015a). Memory reconsolidation understood and misunderstood. *International Journal of Neuropsychotherapy, 3,* 2–46. doi:10.12744/ijnpt.2015.0002-0046

Ecker, B. (2015b). Psychotherapy's mysterious efficacy ceiling: Is memory reconsolidation the breakthrough? *The Neuropsychotherapist*, *16,* 6–24. doi:10.12744/tnpt(16)006-024

Ecker, B. (2015c). Using NLP for memory reconsolidation: A glimpse of integrating the panoply of psychotherapies. *The Neuropsychotherapist, 10*, 50–56. doi:10.12744/tnpt(10)050-056

Ecker, B. (2016). Guidelines for creating juxtaposition experiences. *Coherence Therapy Clinical Note Series, 7*, 1–7. Oakland, CA: Coherence Psychology Institute. Available online: http://www.coherencetherapy.org/resources/notes.htm

Ecker, B., & Hulley, L. (1996). *Depth oriented brief therapy: How to be brief when you were trained to be deep, and vice versa.* San Francisco, CA: Jossey-Bass.

Ecker, B., & Hulley, L. (2000a). The order in clinical "disorder": Symptom coherence in depth orient-

ed brief therapy. In R. A. Neimeyer & J. D. Raskin (Eds.), *Constructions of disorder: Meaning-making frameworks for psychotherapy* (pp. 63–89). Washington, DC: American Psychological Association Press.

Ecker, B. & Hulley, L. (2000b). Depth oriented brief therapy: Accelerated accessing of the coherent unconscious. In J. Carlson & L. Sperry (Eds.), *Brief therapy with individuals and couples* (pp. 161-190). Phoenix, AZ: Zeig, Tucker & Theisen.

Ecker, B., & Hulley, L. (2002). Deep from the start: Profound change in brief therapy. *Psychotherapy Networker*, *26*(1), 46–51, 64.

Ecker, B. & Hulley, L. (2008). Coherence therapy: Swift change at the core of symptom production. In J. D. Raskin & S. K. Bridges (Eds.), *Studies in Meaning 3* (pp. 57-84). New York: Pace University Press.

Ecker, B., & Hulley, L. (2017a). *Coherence Therapy practice manual and training guide* (Eighth Ed.). Oakland, CA: Coherence Psychology Institute. (First edition published 1998.) Online: http://www.coherencetherapy.org/resources/manual.htm

Ecker, B., & Hulley, L. (2017b). *The black hole of nonexistence: Dispelling complex attachment trauma with memory reconsolidation in coherence therapy.* [Video, transcript, commentaries.] San Francisco, CA: Coherence Psychology Institute. Online: http://www.coherencetherapy.org/resources/videos.htm

Ecker, B., Hulley, L., & Ticic, R. (2015). Minding the findings: Let's not miss the message of memory reconsolidation research for psychotherapy. *Behavior and Brain Sciences, 38*:e7. doi:10.1017/S0140525X14000168

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

Ecker, B., Ticic, R., & Hulley, L. (2013a, April). A primer on memory reconsolidation and its psychotherapeutic use as a core process of profound change. *The Neuropsychotherapist*, *1*, 82–99. doi:10.12744/tnpt(1)082-099

Ecker, B., Ticic, R., & Hulley, L. (2013b, July). Unlocking the emotional brain: Is memory reconsolidation the key to transformation? *Psychotherapy Networker, 37*(4), 18–25, 46–47.

Ecker, B., Ticic, R., & Hulley, L. (2013c, June). A core process shared by therapies of transformational change: Clinical observations and memory reconsolidation research converge to suggest critical specific factors. 29th international annual conference of the Society for Exploration of Psychotherapy Integration, Universitat Ramon Llull, Barcelona, Spain.

Ecker, B., & Toomey, B. (2008). Depotentiation of symptom-producing implicit memory in coherence therapy. *Journal of Constructivist Psychology, 21*, 87–150. doi:10.1080/10720530701853685

Eichenbaum, H. (2004). An information processing framework for memory representation by the hippocampus: The cognitive neuroscience of knowing one's self. In M. S. Gazzaniga (Ed.), *The Cognitive Neurosciences III* (pp. 1077–1089). Cambridge: MIT Press.

Eisenberg, M., & Dudai, Y. (2004). Reconsolidation of fresh, remote, and extinguished fear memory in Medaka: Old fears don't die. *European Journal of Neuroscience, 20*, 3397–3403. doi:10.1111/j.1460-9568.2004.03818.x

Eisenberg, M., Kobilo, T., Berman, D. E., & Dudai, Y. (2003). Stability of retrieved memory: Inverse correlation with trace dominance. *Science, 301*, 1102–1104. doi:10.1126/science.1086881

Elsey, J. W. B., & Kindt, M. (2017a). Tackling maladaptive memories through reconsolidation: From neural to clinical science. *Neurobiology of Learning & Memory*, *142A*, 108–117. doi:10.1016/j.nlm.2017.03.007

Elsey, J. W. B., & Kindt, M. (2017b). Breaking boundaries: Optimizing reconsolidation-based interventions for strong and old memories. *Learning & Memory, 24*, 472–479. doi:10.1101/lm.044156.116

Exton-McGuinness, M. T. J., Lee, J. L. C., & Reichelt, A. C. (2015). Updating memories: The role of prediction errors in memory reconsolidation. *Behavioural Brain Research, 278*, 375–384. doi:10.1016/j.bbr.2014.10.011

Eysenck, H. J. (1976). The learning theory model of neurosis—A new approach. *Behaviour Research and Therapy, 14*, 251–267.

Feinstein, D. (2015). How energy psychology changes deep emotional learnings. *The Neuropsychotherapist,*

10, 38–49. doi:10.12744/tnpt(10)038-049

Fernández, R. S., Bavassi, L., Forcato, C., & Pedreira, M. E. (2016a). The dynamic nature of the reconsolidation process and its boundary conditions: Evidence based on human tests. *Neurobiology of Learning and Memory, 130*, 202–212. doi: 10.1016/j.nlm.2016.03.001

Fernández, R. S., Bavassi, L., Kaczer, L., Forcato, C., & Pedreira, M. E. (2016b). Interference conditions of the reconsolidation process in humans: The role of valence and different memory systems. *Frontiers in Human Neuroscience, 10*, 641. doi:10.3389/fnhum.2016.00641

Fernández, R. S., Boccia, M. M., & Pedreira, M. E. (2016c). The fate of memory: Reconsolidation and the case of prediction error. *Neuroscience and Biobehavioral Reviews, 68*, 423–441. doi:10.1016/j.neubiorev.2016.06.004

Fernández, R. S., Pedreira, M. E., & Boccia, M. M. (2017). Does reconsolidation occur in natural settings? Memory reconsolidation and anxiety disorders. *Clinical Psychology Review, 57*, 45–58. doi:10.1016/j.cpr.2017.08.004

Ferrer Monti, R.I., Alfei, J.M., Mugnaini, M., Bueno, A.M., Beckers, T., Urcelay, G.P., & Molina, V.A. (2017). A comparison of behavioral and pharmacological interventions to attenuate reactivated fear memories. *Learning & Memory, 24, 369–374.* doi: 10.1101/lm.045385.117

Festinger, L. (1957). *A theory of cognitive dissonance.* Stanford, CA: Stanford University Press.

Flavell, C.R., Barber, D.J., & Lee, J.L. (2011). Behavioural memory reconsolidation of food and fear memories. *Nature Commununications,* 2, 504. doi:10.1038/ncomms1515

Foa, E.B., & Kozak, M.J. (1986). Emotional processing of fear: exposure to corrective information. *Psychological Bulletin*, *99*, 20–35.

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

Forcato, C., Bavassi, L., De Pino, G., Fernández, R. S., Villarreal, M. F., & Pedreira, M. E. (2016). Differential left hippocampal activation during retrieval with different types of reminders: An fMRI study of the reconsolidation process. *PLoS ONE* 11(3):e0151381. doi:10.1371/journal.pone.0151381

Forcato, C., Burgos, V. L., Argibay, P. F., Molina, V. A., Pedreira, M. E., & Maldonado, H. (2007). Reconsolidation of declarative memory in humans. *Learning & Memory, 14*, 295–303. doi:10.1101/lm.486107

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

Fosha, D. (2000). *The transforming power of affect.* New York, NY: Basic Books.

Frankland, P. W., Ding, H. K., Takahashi, E., Suzuki, A., Kida, S., & Silva, A. J. (2006). Stability of recent and remote contextual fear memory. *Learning & Memory, 13*, 451–457. doi:10.1101/lm.183406

Frenkel, L., Maldonado, H., & Delorenzi, A. (2005). Memory strengthening by a real-life episode during reconsolidation: An outcome of water deprivation via brain angiotensin II. *European Journal of Neuroscience, 22*, 1757–1766. doi:10.1111/j.1460-9568.2005.04373.x

Friedlander, M. L., Angus, L., Wright, S.T., Günther, C., Austin, C. L., Kangos, K., Barbaro, L., Macaulay, C., Carpenter, N., & Khattra, J. (2016). "If those tears could talk, what would they say?" Multi-method analysis of a corrective experience in brief dynamic therapy. *Psychotherapy Research*, 1-18. doi:10.1080/10503307.2016.1184350

Frith, C. D., & Frith, U. (2012). Mechanisms of social cognition. *Annual Review of Psychology, 63*, 287–313.

Galluccio, L. (2005). Updating reactivated memories in infancy: I. Passive- and active-exposure effects. *Developmental Psychobiology, 47*, 1–17. doi:10.1002/dev.20073

Goldfried, M. R. (1980). Toward the delineation of therapeutic change principles. *American Psychologist, 35*, 991–999. Republished as: Goldfried, M. R. (2009). Toward the delineation of therapeutic change principles. *Applied and Preventive Psychology, 13*, 3–11. doi:10.1016/j.appsy.2009.10.015

Gorman, J.M., & Roose, S.P. (2011). The neurobiology of fear memory reconsolidation and psychoanalytic theory. *Journal of the American Psychoanalytic Association, 59*, 1201–1219. doi:*10.1177/0003065111427724*

Ghosh, V. E., & Gilboa, A. (2014). What is a memory schema? A historical perspective on current neuroscience literature. *Neuropsychologia, 53*, 104–114. doi:10.1016/j.neuropsychologia.2013.11.010

Gray, R. M., & Bourke, F. (2015). Remediation of intrusive symptoms of PTSD in fewer than five sessions: a 30-person pre-pilot study of the RTM Protocol. *Journal of Military, Veteran and Family Health, 1*, 13–20. doi:10.3138/jmvfh.2996

Gray, R. M., & Liotta, R. F. (2012). PTSD: Extinction, reconsolidation, and the visual-kinesthetic dissociation protocol. *Traumatology, 18*, 3–16. doi:10.1177/1534765611431835

Gray, R. M., & Teall, B. (2016). Reconsolidation of traumatic memories (RTM) for PTSD: a case series. *Journal of Experiential Psychotherapy, 19*(4), 59–69.

Greenberg, L. S. (2002). *Emotion-focused therapy: Coaching clients to work through their feelings.* Washington DC: American Psychological Association Press.

Greenberg, L. S. (2010). Emotion-focused therapy: A clinical synthesis. *Focus, 8,* 32–42. Online: http://focus.psychiatryonline.org/cgi/reprint/8/1/32

Greenberg, L. S. (2012, November). Emotions, the great captains of our lives: Their role in the process of change in psychotherapy. *American Psychologist, 67*(8), 697–707. doi:10.1037/a0029858

Greenberg, L. S., Rice, L., & Elliott, R. (1993). *Facilitating emotional change: The moment-by-moment process.* New York: Guilford Press.

Heatherington, L., Constantino, M.J., Friedlander, M.L., Angus, L.E., & Messer, S.B. (2012). Clients' perspectives on corrective experiences in psychotherapy. In L. Castonguay & C.E. Hill (Eds.), *Transformation in psychotherapy: Corrective experiences across cognitive behavioral, humanistic, and psychodynamic approaches* (pp. 161–190). Washington DC: American Psychological Association.

Hermans, H.J.M., & Dimaggio, G. (2007), Self, identity, and globalization in times of uncertainty: A dialogical analysis. *Review of General Psychology, 11*, 31-61. doi:10.1037/1089-2680.11.1.31

Hernandez. P. J., & Kelley, A. E. (2004). Long-term memory for instrumental responses does not undergo protein synthesis-dependent reconsolidation upon retrieval. *Learning & Memory, 11*, 748–754. doi:10.1101/lm.84904

Hill, C. E., Castonguay, L. G., Farber, B. A., Knox, S., Stiles, W. B., Anderson, T., Angus, L. E., et al. (2012). Corrective experiences in psychotherapy: Definitions, processes, consequences, and research directions. In L. G. Castonguay & C. E. Hill (Eds.), *Transformation in psychotherapy: Corrective experiences across cognitive behavioral, humanistic, and psychodynamic approaches* (pp. 355–370). Washington DC: American Psychological Association.

Högberg, G., Nardo, D., Hällström, T., & Pagani, M. (2011). Affective psychotherapy in post-traumatic reactions guided by affective neuroscience: Memory reconsolidation and play. *Psychology Research and Behavior Management, 4*, 87–96. doi:10.2147/PRBM.S10380

Hupbach, A. (2011). The specific outcomes of reactivation-induced memory changes depend on the degree of competition between old and new information. *Frontiers in Behavioral Neuroscience, 5:33.* doi: 10.3389/fnbeh.2011.00033

Hupbach, A., Gomez, R., Hardt, O., & Nadel, L. (2007). Reconsolidation of episodic memories: A subtle reminder triggers integration of new information. *Learning & Memory, 14*, 47–53. doi:10.1101/lm.365707

Hupbach, A., Gomez, R., & Nadel, L. (2009). Episodic memory reconsolidation: Updating or source confusion? *Memory, 17*, 502–510. doi:10.1080/09658210902882399

Hutton-Bedbrook, K., & McNally, G. P. (2013). The promises and pitfalls of retrieval-extinction procedures in preventing relapse to drug seeking. *Frontiers in Psychiatry, 4*, 14. doi:10.3389/fpsyt.2013.00014

Jarome, T. J., Ferrara, N. C., Kwapis, J. L., & Helmstetter, F. J. (2015). Contextual information drives the reconsolidation-dependent updating of retrieved fear memories. *Neuropsychopharmacology, 40,* 3044-3052. doi: 10.1038/npp.2015.161

Jarome, T. J., Kwapis, J. L., Werner, C. T., Parsons, R. G., Gafford, G. M., & Helmstetter, F. J. (2012). The timing of multiple retrieval events can alter GluR1 phosphorylation and the requirement for protein synthesis in fear memory reconsolidation. *Learning & Memory, 19*, 300–306. doi:10.1101/lm.024901.111

Johnson, D. C., & Casey B. J. (2015). Extinction during memory reconsolidation blocks recovery of fear in adolescents. *Scientific Reports,* 5, Article number: 8863. doi:10.1038/srep08863

Kazdin, A. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology, 3*, 1–27. doi:10.1146/annurev.clinpsy.3.022806.091432

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

Kindt, M., & van Emmerik, A. (2016). New avenues for treating emotional memory disorders: Towards a reconsolidation intervention for posttraumatic stress disorder. *Therapeutic Advances in Psychopharmacology, 6*(4), 283–295. doi:10.1177/ 2045125316644541

Krawczyk, M.C., Fernández, R.S., Pedreira, M.E., & Boccia, M.M. (2017). Toward a better understanding on the role of prediction error on memory processes: From bench to clinic. *Neurobiology of Learning and Memory, 142A*, 13–20. doi:10.1016/j.nlm.2016.12.011

Kredlow, M.A., & Otto, M.W. (2015). Interference with the reconsolidation of trauma-related memories in adults. *Depression and Anxiety, 32*, 32–37. doi: 10.1002/da.22343

Kredlow, M.A., Unger, L.D., & Otto, M.W. (2016). Harnessing reconsolidation to weaken fear and appetitive memories: a meta-analysis of post-retrieval extinction effects. *Psychological Bulletin, 142*, 314–336. doi:10.1037/bul0000034

Kreiman, G., Koch, C. and Fried, I. (2000). Imagery neurons in the human brain. *Nature*, *408*, 357–361. doi:10.1038/35042575

Kroes, M.C., Schiller, D., LeDoux, J.E., & Phelps, E.A. (2015). Translational approaches targeting reconsolidation. *Current Topics in Behavioral Neurosciences, 28*, 197–230. doi:10.1007/7854_2015_5008.

Lane, R. D., Ryan, L., Nadel, L., & Greenberg, L. (2015). Memory reconsolidation, emotional arousal and the process of change in psychotherapy: New insights from brain science. *Behavioral and Brain Sciences*, 38:e1. doi:10.1017/S0140525X14000041

Lasser, K. A., & Greenwald, R. (2015). Progressive counting facilitates memory reconsolidation. *The Neuropsychotherapist, 10*, 30-37. doi:10.12744/tnpt(10)030-037

LeDoux, J. E., Romanski, L., & Xagoraris, A. (1989). Indelibility of subcortical emotional memories. *Journal of Cognitive Neuroscience, 1*, 238–243. doi:10.1162/jocn.1989.1.3.238

Lee, C. W., Taylor, G., & Drummond, P. D. (2006). The active ingredient in EMDR: Is it traditional exposure or dual focus of attention? *Clinical Psychology and Psychotherapy, 13*, 97–107. doi:10.1002/cpp.479

Lee, J. L. (2009). Reconsolidation: Maintaining memory relevance. *Trends in Neuroscience, 32*, 413–420. doi:10.1016/j.tins.2009.05.002

Lee, J. L. C., Nader, K., & Schiller, D. (2017). An update on memory reconsolidation updating. *Trends in Cognitive Sciences, 21*(7), 531-545. doi:10.1016/j.tics.2017.04.006

Lee, S. H., Choi, J. H., Lee, N., Lee, H. R., Kim, J. I., Yu, N. K., . . . Kaang, B. K. (2008). Synaptic protein degradation underlies destabilization of retrieved fear memory. *Science, 319*, 1253–1256. doi:10.1126/science.1150541

Lipton, B., & Fosha, D. (2011). Attachment as a transformative process in AEDP: Operationalizing the intersection of attachment theory and affective neuroscience. *Journal of Psychotherapy Integration, 21,* 253–279. doi:10.1037/a0025421

Liu, J., Zhao, L., Xue, Y., Shi, J., Suo, L., Luo, Y., et al. (2014). An unconditioned stimulus retrieval extinction procedure to prevent the return of fear memory. *Biological Psychiatry, 76*, 895–901. doi:10.1016/j.biopsych.2014.03.027

López, M. A., Santos, M. J., Cortasa, S., Fernández, R. S., Tano, M. C., & Pedreira, M. E. (2016). Different dimensions of the prediction error as a decisive factor for the triggering of the reconsolidation process. *Neurobiology of Learning and Memory, 136*, 210–219. doi: 10.1016/j.nlm.2016.10.016

Luo, Y., Xue, Y., Liu, J., Shi, H., Jian, M., Han, Y., et al. (2015). A novel UCS memory retrieval-extinction procedure to inhibit relapse to drug seeking. *Nature Communications, 6*, 7675. doi:10.1038/ncomms8675

Luyten, L., & Beckers, T. (2017). A preregistered, direct replication attempt of the retrieval- extinction effect in cued fear conditioning in rats. *Neurobiology of Learning and Memory.* doi:10.1016/j.nlm.2017.07.014

Markus, H., & Wurf, E. (1987). The dynamic self-concept: A social psychological perspective. *Annual Review of Psychology, 38*, 299–337.

McGaugh, J. L. (1989). Involvement of hormonal and neuromodulatory systems in the regulation of memory storage. *Annual Review of Neuroscience, 2*, 255–287. doi:10.1146/annurev.ne.12.030189.001351

McGaugh, J. L., & Roozendaal, B. (2002). Role of adrenal stress hormones in forming lasting memories in the brain. *Current Opinions in Neurobiology, 12*, 205–210. doi:10.1016/s0959-4388(02)00306-9

Merlo, E., Milton, A. L., Goozée, Z. Y., Theobald, D. E., & Everitt, B. J. (2014). Reconsolidation and extinction are dissociable and mutually exclusive processes: Behavioral and molecular evidence. *The Journal of Neuroscience, 34*, 2422–2431. doi:10.1523/jneurosci.4001-13.2014

Mileusnic, R., Lancashire, C. L., & Rose, S. P. R. (2005). Recalling an aversive experience by day-old chicks is not dependent on somatic protein synthesis. *Learning & Memory, 12*, 615–619. doi:10.1101/lm.38005

Millan, E.Z., Milligan-Saville, J., & McNally, G.P. (2013). Memory retrieval, extinction, and reinstatement of alcohol seeking. *Neurobiology of Learning and Memory, 101*, 26–32. dos:10.1016/j.nlm.2012.12.010

Milner, B., Squire, L. R., & Kandel, E. R. (1998). Cognitive neuroscience and the study of memory. *Neuron, 20*, 445–468. doi:10.1016/s0896-6273(00)80987-3

Mineka, S., & Zinbarg, R. (2006). A contemporary learning theory perspective on the etiology of anxiety disorders: it's not what you thought it was. *American Psychologist, 61*, 10–26. doi:10.1037/0003-066X.61.1.10

Monfils, M.-H., Cowansage, K. K., Klann, E., & LeDoux, J. E. (2009). Extinction-reconsolidation boundaries: Key to persistent attenuation of fear memories. *Science, 324*, 951–955. doi:10.1126/science.1167975

Morris, R. G., Inglis, J., Ainge, J. A., Olverman, H. J., Tulloch, J., Dudai, Y., & Kelly, P. A. (2006). Memory reconsolidation: Sensitivity of spatial memory to inhibition of protein synthesis in dorsal hippocampus during encoding and retrieval. *Neuron, 50*, 479–489. doi:10.1016/j.neuron.2006.04.012

Nader, K. (2015). Reconsolidation and the dynamic nature of memory. *Cold Spring Harbor Perspectives in Biology, 7*, a021782. doi:10.1101/cshperspect.a021782

Nader, K., Hardt, O., & Lanius, R. (2014). Memory as a new therapeutic target. *Dialogues in Clinical Neuroscience, 15(4),* 475–486.

Nader, K., Schafe, G. E., & LeDoux, J. E. (2000). Fear memories require protein synthesis in the amygdala for reconsolidation after retrieval. *Nature, 406*, 722–726. doi:10.1038/35021052

Neumann, D.L., & Kitlertsirivatana, E. (2010). Exposure to a novel context after extinction causes a renewal of extinguished conditioned responses: implications for the treatment of fear. *Behavior Research and Therapy, 48*, 565–570. doi:10.1016/j.brat.2010.03.002

Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Science*, *9*, 408–409.

Ogden, P., & Minton, K. (2000). Sensorimotor psychotherapy: One method for processing traumatic memory. *Traumatology, 6*(3), 149–173. doi:10.1177/153476560000600302

Ogden, P., Minton, K., & Pain, C. (2006). *Trauma and the body.* New York: W. W. Norton & Co.

Oyarzún, J.P., Lopez-Barroso, D., Fuentemilla, L., Cucurell, D., Pedraza, C., Rodriguez-Fornells, A., et al. (2012). Updating fearful memories with extinction training during reconsolidation: A human study using auditory aversive stimuli. *PLoS ONE 7(*6): e38849. https://doi.org/10.1371/journal.pone.0038849

Payne, P., Levine, P. A., & Crane-Godreau, M. A. (2015). Somatic experiencing: using interoception

and proprioception as core elements of trauma therapy. *Frontiers in Psychology*, *6*, 93. doi:10.3389/ fpsyg.2015.00093

Pedreira, M. E., & Maldonado, H. (2003). Protein synthesis subserves reconsolidation or extinction depending on reminder duration. *Neuron, 38*, 863–869. doi:10.1016/S0896-6273(03)00352-0

Pedreira, M. E., Pérez-Cuesta, L. M., & Maldonado, H. (2002). Reactivation and reconsolidation of long-term memory in the crab *Chasmagnathus*: Protein synthesis requirement and mediation by NMDA-type glutamatergic receptors. *The Journal of Neuroscience, 22*, 8305–8311. PMID: 12223585

Pedreira, M. E., Pérez-Cuesta, L. M., & Maldonado, H. (2004). Mismatch between what is expected and what actually occurs triggers memory reconsolidation or extinction. *Learning & Memory, 11*, 579–585. doi:10.1101/lm.76904

Pine, A., Mendelsohn, A., & Dudai, Y. (2014). Unconscious learning of likes and dislikes is persistent, resilient, and reconsolidates. *Frontiers in Psychology, 5*(1051), 1–13. doi:10.3389/ fpsyg.2014.01051

Piñeyro, M.E., Ferrer Monti, R.I., Alfei, J.M., Bueno, A.M., & Urcelay, G.P. (2014). Memory destabilization is critical for the success of the reactivation-extinction procedure. *Learning & Memory, 21*, 46–54 . doi:10.1101/lm.032714.113

Poundja, J., Sanche, S., Tremblay, J., & Brunet, A. (2012). Trauma reactivation under the influence of propranolol: An examination of clinical predictors. *European Journal of Psychotraumatology, 3*. doi:10.3402/ejpt.v3i0.15470

Proulx, T., Inzlicht, M., & Harmon-Jones, E. (2012). Understanding all inconsistency compensation as a palliative response to violated expectations. *Trends in Cognitive Science, 16*, 285-291. doi:10.1016/j. tics.2012.04.002

Przybyslawski, J., Roullet, P., & Sara, S. J. (1999). Attenuation of emotional and nonemotional memories after their reactivation: Role of beta adrenergic receptors. *The Journal of Neuroscience, 19*, 6623–6628.

Rashid, A.J., Yan, C., Mercaldo, V., Hsiang, H.-L., Park, S., Cloe, C.J., et al. (2016). Competition between engrams influences fear memory formation and recall. *Science, 353*, 383–387. doi:10.1126/science. aaf0594

Ray, R.D., Wilhelm, F.H., & Gross, J.J. (2008). All in the mind's eye? Anger rumination and reappraisal. *Journal of Personality and Social Psychology, 94*, 133–145. doi:10.1037/0022-3514.94.1. 133

Reber, A. S. (1989). Implicit learning and tacit knowledge. *Journal of Experimental Psychology: General, 118*, 219–235.

Reichelt, A. C., & Lee, J. L. C. (2013). Memory reconsolidation in aversive and appetitive settings. *Frontiers of Behavioral Neuroscience, 7*, 1–18. doi:10.3389/fnbeh.2013.00118

Reichelt, A. C., Exton-McGuinness, M. T., & Lee, J. L. (2013). Ventral tegmental dopamine dysregulation prevents appetitive memory destabilisation. *The Journal of Neuroscience, 33*, 14205–14210. doi:10.1523/ jneurosci.1614-13.2013

Riccio, D. C., Millin, P. M., & Bogart, A. R. (2006). Reconsolidation: A brief history, a retrieval view, and some recent issues. *Learning & Memory, 13*, 536–544. doi:10.1101/lm.290706

Robertson, E.M. (2012). New insights in human memory interference and consolidation. *Current Biology, 22*, R66–R71. doi:10.1016/j.cub.2011.11.051

Robinson, M. J. F., & Franklin, K. B. J. (2010). Reconsolidation of a morphine place preference: Impact of the strength and age of memory on disruption by propranolol and midazolam. *Behavioural Brain Research*, *213*, 201–207. doi:10.1016/j.bbr.2010.04.056

Rodriguez-Ortiz, C. J., De la Cruz, V., Gutierrez, R., & Bermidez-Rattoni, F. (2005). Protein synthesis underlies post-retrieval memory consolidation to a restricted degree only when updated information is obtained. *Learning & Memory, 12*, 533–537. doi:10.1101/lm.94505

Rodriguez-Ortiz, C. J., Garcia-DeLaTorre, P., Benavidez, E., Ballesteros, M. A., & Bermudez-Rattoni, F. (2008). Intrahippocampal anisomycin infusions disrupt previously consolidated spatial memory only when memory is updated. *Neurobiology of Learning and Memory, 89*, 352–359. doi:10.1016/j. nlm.2007.10.004

Roozendaal, B., McEwen, B. S., & Chattarji, S. (2009). Stress, memory and the amygdala. *Nature Reviews Neuroscience, 10*, 423–433. doi:10.1038/nrn2651

Rossato, J. I., Bevilaqua, L. R. M., Medina, J. H., Iz-

quierdo, I., & Cammarota, M. (2006). Retrieval induces hippocampal-dependent reconsolidation of spatial memory. *Learning & Memory, 13*, 431–440. doi:10.1101/lm.315206

Rossato, J. I., Bevilaqua, L. R. M., Myskiw, J. C., Medina, J. H., Izquierdo, I., & Cammarota, M. (2007). On the role of hippocampal protein synthesis in the consolidation and reconsolidation of object recognition memory. *Learning & Memory, 14*, 36–46. doi:10.1101/lm.422607

Rosen, G. M., & Davidson, G. C. (2003). Psychology should list empirically supported principles of change (ESPs) and not credentialed trademarked therapies or other treatment packages. *Behavior Modification*, *27*, 300–312. doi:10.1177/0145445503027003003

Rumelhart, D. E., & McClelland, J. L. (1986). *Parallel distributed processing: Explorations in the microstructure of cognition* (2 vols.). Cambridge, MA: MIT Press.

Ryan, L., Hoscheidt, S., & Nadel, L. (2008). Perspectives on episodic and semantic memory retrieval. In: Handbook of episodic memory (Handbook of behavioral neuroscience) (pp. 5–18), ed. E. Dere, A. Easton, J. Huston & L. Nadel. Amsterdam: Elsevier.

Ryan, T. J., Roy, D. S., Pignatelli, M., Arons, A., & Tonegawa, S. (2015). Engram cells retain memory under retrograde amnesia. *Science, 348*, 1007–1013. doi:10.1126/science.aaa5542

Sar, V. (2011). Developmental trauma, complex PTSD, and the current proposal of DSM-5. *European Journal of Psychotraumatology, 2*:5622. doi:10.3402/ejpt.v2i0.5622

Schiller, D., Kanen, J.W., Ledoux, J.E., Monfils, M.H., & Phelps, E.A. (2013). Extinction during reconsolidation of threat memory diminishes prefrontal cortex involvement. *Proceedings of the National Academy of Science, 110*, 20040–20045. doi: 10.1073/pnas.1320322110

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

Schiller, D., & Phelps, E. A. (2011). Does reconsolidation occur in humans? *Frontiers of Behavioral Neuroscience, 5*, 1–18. doi:10.3389/fnbeh.2011.00024

Schore, A. N. (2003). *Affect dysregulation and disorders of the self.* New York, NY: W.W. Norton.

Schroyens, N., Beckers, T., & Kindt, M. (2017). In search for boundary conditions of reconsolidation: A failure of fear memory interference. *Frontiers in Behavioral Neuroscience*, 11:65. doi: 10.3389/fnbeh.2017.00065

Schwabe, L., Nader, K., & Pruessner, J.C. (2014). Reconsolidation of human memory: Brain mechanisms and clinical relevance. *Biological Psychiatry, 76*, 274–280. 10.1016/j.biopsych.2014.03.008

Scully, I.D., Napper, L.E., & Hupbach, A. (2017). Does reactivation trigger episodic memory change? A meta-analysis. *Neurobiology of Learning and Memory, 142*, Part A, 99–107. doi:10.1016/j.nlm.2016.12.012

Seger, C. A., & Miller, E. K. (2010). Category learning in the brain. *Annual Review of Neuroscience, 33*, 203–219.

Sekiguchi, T., Yamada, A., & Suzuki, H. (1997). Reactivation-dependent changes in memory states in the terrestrial slug *Limax flavus*. *Learning & Memory, 4*, 356–364. doi:10.1101/lm.4.4.356

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

Sevenster, D., Beckers, T., & Kindt, M. (2013). Prediction error governs pharmacologically induced amnesia for learned fear. *Science, 339*, 830–833. doi:10.1126/science.1231357

Sevenster, D., Beckers, T., & Kindt, M. (2014). Prediction error demarcates the transition from retrieval, to reconsolidation, to new learning. *Learning & Memory, 21*, 580–584. doi:10.1101/lm.035493.114

Shapiro, F. (2001). *Eye movement desensitization and reprocessing: Basic principles, protocols and procedures, 2nd edition.* New York: Guilford Press.

Sharpless, B. A., & Barber, J. P. (2012). Corrective emotional experiences from a psychodynamic perspective. In L. Castonguay & C. E. Hill (Eds.), *Transformation in psychotherapy: Corrective experiences across cognitive behavioral, humanistic, and*

psychodynamic approaches (pp. 31–50). Washington DC: American Psychological Association.

Shean, G. (2014). Limitations of randomized control designs in psychotherapy research. *Advances in Psychiatry, 2014*, Article ID 561452. doi:10.1155/2014/561452

Sibson, P. & Ticic, R. (2014, March). Remembering in order to forget. *Therapy Today, 25*(2), 26–29.

Siegel, D. J. (2006). An interpersonal neurobiology approach to psychotherapy. *Psychiatric Annals, 36,* 248–258.

Siegel, D. J. (1999). *The developing mind*. New York: Guilford Press. ISBN 9781462520671

Siegel, D. J. (2015). *The developing mind: How relationships and the brain interact to shape who we are*, 2nd edition. New York: Guilford Press. ISBN 9781462520671

Soeter, M., & Kindt, M. (2011). Disrupting reconsolidation: Pharmacological and behavioral manipulations. *Learning & Memory, 18*, 357–366. doi:10.1101/lm.2148511

Soeter, M., & Kindt, M. (2012). Erasing fear for an imagined threat event. *Psychoneuroendocrinology, 37*, 1769–1779. doi:10.1016/j.psyneuen.2012.03.011

Soeter, M., & Kindt, M. (2015a). An abrupt transformation of phobic behavior after a post-retrieval amnesic agent. *Biological Psychiatry, 78*, 880–886. doi: 10.1016/j.biopsych.2015.04.006

Soeter, M., & Kindt, M. (2015b). Retrieval cues that trigger reconsolidation of associative fear memory are not necessarily an exact replica of the original learning experience. *Frontiers in Behavioral Neuroscience, 9:*122. doi:10.3389/fnbeh.2015.00122

Solomon, R. W., & Shapiro, F. (2008). EMDR and the adaptive information processing model: Potential mechanisms of change. *Journal of EMDR Practice and Research*, *2*, 315–325.

Steenen SA, van Wijk AJ, van der Heijden GJ, van Westrhenen R, de Lange J, de Jongh A. 2015. Propranolol for the treatment of anxiety disorders: Systematic review and meta-analysis. Journal of Psychopharmacology

Steinfurth, E. C. K., Kanen, J. W., Raio, C. M., Clem, R. L., Huganir, R. L., & Phelps, E. A. (2014). Young and old Pavlovian fear memories can be modified with extinction training during reconsolidation in humans. *Learning & Memory, 21*, 338–341. doi:10.1101/lm.033589.113

Stricker, G. (2006). The local clinical scientist, evidence-based practice, and personality assessment. *Journal of Personality Assessment, 86* , 4–9. doi:10.1207/s15327752jpa8601_02

Stricker, G., & Trierweiler, S.J. (1995). The local clinical scientist: A bridge between science and practice. *American Psychologist, 50*(12), 995–1002.

Suzuki, A., Josselyn, S. A., Frankland, P. W., Masushige, S., Silva, A. J., & Kida, S. (2004). Memory reconsolidation and extinction have distinct temporal and biochemical signatures. *The Journal of Neuroscience, 24*, 4787–4795. doi:10.1523/jneurosci.5491-03.2004

Taylor, J. R., & Torregrossa, M. M. (2015). Pharmacological disruption of maladaptive memory. *Handbook of Experimental Pharmacology, 228*, 381–415. doi:10.1007/978-3-319-16522-6_13

Thome, J., Koppe, G., Hauschild, S., Liebke, L., Schmahl, C., Lis, S., et al. (2016). Modification of fear memory by pharmacological and behavioural interventions during reconsolidation. *PLoS ONE 11* (8): e0161044. doi:10.1371/journal.pone.0161044

Ticic, R., & Kushner, E. (2015). Deep release for body and soul: Memory reconsolidation and the Alexander technique. *The Neuropsychotherapist, 10*, 24–28. doi:10.12744/tnpt(10)024-028

Toomey, B., & Ecker, B. (2007). Of neurons and knowings: Constructivism, coherence psychology and their neurodynamic substrates. *Journal of Constructivist Psychology, 20*, 201–245. doi:10.1080/10720530701347860

Toomey, B., & Ecker, B. (2009). Competing visions of the implications of neuroscience for psychotherapy. *Journal of Constructivist Psychology, 22*, 95–140. doi:10.1080/10720530802675748

Treanor, M., Brown, L.A., Rissman, J., & Craske, M.G. (2017). Can memories of traumatic experiences or addiction be erased or modified? A critical review of research on the disruption of memory reconsolidation and its applications. *Perspectives on Psychological Science, 12*(2), 290–305. doi: 10.1177/1745691616664725

Tulving, E. (2002). Episodic memory: From mind to brain. *Annual Review of Psychology, 53*, 1–25.

Tulving, E. (2005). Episodic memory and autonoesis: Uniquely human? In: *The missing link in cognition: Origins of self-selective consciousness*, ed. H. S. Terrace & J. Metcalfe, pp. 3–56. Oxford University Press.

van der Kolk, B. (1994). The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress. *Harvard Review of Psychiatry*, *1*, 253–265. doi:10.3109/10673229409017088

Vervliet, B., Craske, M.G., & Hermans, D. (2013). Fear extinction and relapse: state of the art. *Annual Review of Clinical Psychology, 9*, 215–248. doi:10.1146/annurev-clinpsy-050212-185542

Volkman, V. R. (2008). *Traumatic Incident Reduction: Research and results* (2nd ed.). Ann Arbor, MI: Loving Healing Press.

Walker, M. P., Brakefield, T., Hobson, J. A., & Stickgold, R. (2003). Dissociable stages of human memory consolidation and reconsolidation. *Nature, 425*, 616–620. doi:10.1038/nature01930

Wallace, B.A. (2000). *The taboo of subjectivity: Toward a new science of consciousness.* Oxford, UK: Oxford University Press.

Wampold, B. E. (2001). *The great psychotherapy debate: Models, methods, and findings.* Mahwah, NJ: Lawrence Erlbaum Associates.

Wampold, B. E. (2015). How important are the common factors in psychotherapy? An update. *World Psychiatry, 14*(3), 270–277. doi:10.1002/wps.20238

Wang, S.H., de Oliveira Alvares, L., & Nader, K. (2009). Cellular and systems mechanisms of memory strength as a constraint on auditory fear reconsolidation. *Nature Neuroscience, 12*, 905–912.

Welling, H. (2012). Transformative emotional sequence: Towards a common principle of change. *Journal of Psychotherapy Integration, 22*, 109–136. doi:10.1037/a0027786

Winters, B. D., Tucci, M. C., & DaCosta-Furtado, M. (2009). Older and stronger object memories are selectively destabilized by reactivation in the presence of new information. *Learning & Memory, 16*, 545–553. doi:10.1101/lm.1509909

Wood, N.E., Rosasco, M.L., Suris, A.M., Spring, J.D., Marin, M.-F., Lasko, N.B., Goetz, J.M., Fischer, A.M., Orr, S.P., Pitman, R.K., 2015. Pharmacological blockade of memory reconsolidation in posttraumatic stress disorder: three negative psychophysiological studies. *Psychiatry Research, 225*, 31–39. doi:10. 1016/j.psychres.2014.09.005

Yacoby, A., Dudai, Y., & Mendelsohn, A. (2015). Metamemory ratings predict long-term changes in reactivated episodic memories. *Frontiers in Behavioral Neuroscience, 9,* 20. doi:10.3389/fnbeh.2015.00020