#### Chapter | fourteen

## Memory Reconsolidation, Trace Reassociation and the Freudian Unconscious

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Memory is a fundamental biological function; we need to have long-term memories to find the best food and sex and to be safe from danger. Memories also shape our identity. We could say that we are who we are because of the conscious experience of thinking, the present physical and psychical state and our memories of the past. As Augustine (Confessions, Book 11, Chapter 20) said,

There is nothing like future and past.... There is only the presence of the past, the presence of the presence, and the presence of the future. These three I see in the soul, but I cannot see them independent of it: Present is the memory of the past, present is the perception of the presence, and present is the expectation of the future.

Why do we have memories? To retain and progressively modify our behavior under the impact of experience. Memory is indispensable in all behavior, making it at once consistent and modifiable. Thus, memory seems the critical function designed to replay the past so that it can integrate selected aspects of it into present behavior. Often, however, our behavior does not act in the best or healthiest mode. In certain cases, it may become mildly or even severely pathological, as in the case of mental malfunctioning and disorders such as anxiety, depression,

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obsession, and compulsion. The mechanisms underlying these psychopathologies are still in large part elusive to our scientific understanding. The impact that mental health disorders have on our society in the United States and throughout the world has long been underestimated. Data collected by the massive Global Burden of Diseases Study (2009) conducted by the World Health Organization, the World Bank, and Harvard University reveal that mental illness, including suicide, accounts for more than 20% of the burden of disease in established market economies, such as the United States. This is more than the disease burden caused by all types of cancers.

For many mental health pathologies, "talking" therapies, in which the patient re-evokes memories and experiences and is guided toward new perceptions and reprocessing of the past, are recommended, especially for the treatment of stress, mood, and anxiety disorders. These types of therapies have their roots in psychoanalysis.

Psychoanalysis has undoubtedly had a profound influence on many aspects of 21<sup>st</sup>-century culture. As a general theory of individual human behavior, psychoanalytic hypotheses enrich and are enriched by the study of the biological and social sciences, behavior, history, philosophy, art, and literature. As a developmental theory, psychoanalysis contributes to child psychology, education, and family studies. In mental health, it is the basis of all other dynamic approaches to therapy. Although during the past century psychoanalysis has undergone numerous theoretical modifications, there has been, unfortunately, very little rigorous validating research to prove the basic assumptions of psychoanalytic theories. Nevertheless, the insights or theories of psychoanalysis constitute the foundation of most psychotherapeutic approaches employed in general psychiatric practice, in child psychiatry, and in individual, family, and group therapies.

The main goal of psychoanalysis is the examination of the complex relationship between body, brain, and mind and the comprehensive understanding of the role of emotions in health as well as in medical illness. It is centered on the observation that individuals are often unaware or "unconscious" of many of the factors that determine their emotions and behavior. Freud was the preeminent pioneer in understanding the importance of the unconscious. Through his extensive work with patients and his theoretic elaboration, he provided evidence that factors which influence thought and action exist outside of awareness, that unconscious conflicts play a part in determining both normal and abnormal behavior, and that the unconscious past shapes the present.

What is the unconscious process that according to Freud controls the individual behaviors and being? How is it formed, and how is it that through recollection, memory reactivation, and memory updating psychopathologies can be alleviated?

### 14.1 DIFFERENT TYPES OF UNCONSCIOUS PROCESSES

To begin to address the previous questions, it is important that we first clarify that the unconscious according to Freud (Freudian unconscious) is distinct

from other nonconscious processes of the brain and mind, such as the cognitive unconscious.

The cognitive and the Freudian unconscious (FU) refer to distinct processes and are connected merely by the use of the word "unconscious." Indeed, the notion of mental processes that do not reach consciousness has been entertained by a variety of psychological theories (Gazzaniga, Ivry, & Mangun, 2008). Recent neurobiological studies, particularly with neuroimaging approaches, have provided evidence for the existence of unconscious neural processing (Hassin, Uleman, & Bargh, 2005). The type of unconscious processes that were tested in these studies, and that have been dubbed the "cognitive unconscious" (CU) (Kihlstrom, 1987), comprised phenomena such as subliminal perception (Kouider & Dehaene, 2007) and other mechanisms of perception and information processing (Driver & Vuilleumier, 2001; Vuilleumier & Pourtois, 2007) including blindsight (Cowey & Stoerig, 1991; Weiskrantz, 1996), which do not lead to a conscious experience (Dehaene, Changeux, Naccache, Sackur, & Sergent, 2006). These phenomena are operated by input—output mechanisms that are common with those that underlie conscious phenomena (Lau & Passingham, 2007; Rees, 2007; Snodgrass & Shevrin, 2006). However, this type of unconscious does not correspond to what the psychoanalytical theory has postulated on the basis of its clinical practice and theoretical elaborations.

The FU refers to the ensemble of feelings, thoughts, urges, and memories that, outside of consciousness, influence our behavior and experiences. The Freudian hypothesis of the existence of unconscious processes has emerged from the clinical perspective provided by the analysis of dreams (Freud, 1900/1953), parapraxis, slip of the tongue/pen, and neurotic symptoms (Freud, 1915b/1957, pp. 186-189), and the FU operates according to principles that are distinct from those that characterize conscious processes or the CU. A first approach to define the principles of FU functions was based on the analysis of the mental operations of dreaming (Freud, 1915b/1957), during paradoxical sleep (Hobson & Pace-Schott, 2002; Mancia, 2004; Nielsen, 2000; Takeuchi, Miyasita, Inugami, & Yamamoto, 2001). The mental events that emerge in this state are devoid of temporal and spatial dimensions; for example, events can occur simultaneously in different locations and times without any apparent contradiction (Freud, 1901/1960). In the FU, like in dreams, emotional contents can be associated with one another without any logical connection; furthermore, contents can be substituted and displaced (Freud, 1900/1953, pp. 277-508). Although the existence of the FU has nourished an extensive theoretical and clinical psychoanalytical literature, and provided heuristically valid elaborations concerning the determinants of mental life such as drives and repression mechanisms, to date there is no neurobiological explanation for its existence. Some insightful reflections have been proposed (Kandel, 1998, 1999; Shulman & Reiser, 2004), as illustrated by the emergence of the new discipline of neuropsychoanalysis (Solms, 2004). To quote Freud (1920/1955),

The deficiencies in our description would probably vanish if we were already in a position to replace the psychological terms by physiological or chemical ones.... Biology is truly a land of unlimited possibilities. We may expect it to give us the most surprising information and we cannot guess what answers it will return to the questions we have put to it. (p. 60)

Contemporary neuroscience appears to be ready to begin addressing the biological bases of the FU.

Freud distinguished two types of nonconscious mental processes: the primary process, which corresponds to what we defined as FU, and the secondary process, which operates according to logical processes that are common to conscious processes.

Furthermore, according to Freud, at least three mechanisms can be responsible for the production of unconscious processes. The first one can be related to the direct unconscious inscription of traces following a given experience. This type of unconscious can, for example, be produced by subliminal perceptual processing leading to the establishment of traces that do not reach consciousness (Del Cul, Baillet, & Dehaene, 2007). This type of unconscious may be akin to the CU and could be considered as being part of what Freud defined as the latent unconscious or preconscious (Freud, 1923/1961, p. 15). He also called it "unbemerkt," meaning "not noticed" (i.e., by consciousness), hence not reaching consciousness (Freud, 1923/1961, p. 16) (Figure 14.1). This allowed him to distinguish this latent unconscious/preconscious (unbemerkt, Figure 14.1) from what he called "unbewusst," which we now refer to as FU (Figure 14.1).

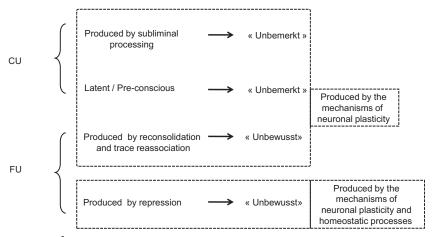


FIGURE 14.1 Summary of the key concepts differentiating the cognitive unconscious (CU) from the Freudian unconscious (FU). *Unbemerkt*, not noticed; *Unbewusst*, not known.

Freud's theory proposed two mechanisms to explain the unbewusst (Figure 14.1): one formed by the mechanism of repression and another one for which he had no explanation. Although the former mechanism still represents the historical definition of the unconscious for psychoanalytical theory, Freud hinted that not all unconscious processes could be accounted for by the mechanism of repression (Freud, 1915b/1957, p. 166); he discussed other mechanisms in particular in Ego and Id (Freud, 1923/1961, p. 18). Specifically, he proposed the possible existence of "the third unconscious," considering that it would be an addition to the "preconscious" (unbemerkt) and to the one formed by repression (unbewusst), for which he could not suggest a mechanism nor could he assess its importance in the overall economy of the unconscious (Freud, 1923/1961, pp. 13-18).

In this chapter, we propose a model that may explain the FU, particularly the "third unconscious," based on three fundamental principles of neuroscience and physiology: synaptic plasticity, trace reassociation, and homeostatic processes. On the basis of this model, we speculate that the dynamic processes of memory formation, retrieval, and updating critically contribute to the formation of FU, which includes its psychopathologies as well as their psychoanalytic treatment.

#### 14.2 MEMORY TRACES ACCORDING TO FREUD

At the end of the 1800s and beginning of the 1900s, Freud elaborated on the process of memory and memory traces in numerous writings. Starting with his early Studies on Hysteria (Breuer & Freud, 1895/1955), in which he described and discussed the processes of memory and trauma, Freud recognized the existence of a complex network of associations that creates our memories and therefore our identity, which obviously includes our pathologies. Freud was inspired by his early work On Aphasia (Freud, 1891/1953), in which he emphasized that the periphery of the body is represented topographically in the brain, and then extended this idea to a complex picture of the formation of words, objects, and their associations. He therefore created a model derived from the spatial and topographical arrangement of the nervous system, which he used as one of the organizing frameworks of psychoanalytic theory. This schematic, spatial arrangement of the mental apparatus was seen as a hierarchical structure of agencies, functions, and organizations that all derived from the combination of simple connections and processes of transference, translation, and transformation.

Freud believed that mnemic traces (Erinnerungsspur) are unconscious, whereas the memories of these traces (Erinnerungen) are conscious and, based on a hierarchical organization, proposed that the psychical material of hysteric cases is organized by collections of memories or "themes." Each theme is organized in three strata. First, it is arranged linearly as a bundle of documents. Second, it is stratified concentrically around a pathogenic nucleus, which, as Freud writes, "consists of those recollections (of experiences

or trains of thoughts) in which the traumatic moment peaked or the pathogenic idea found his purest form." On a third order, according to thought content, there is a nonlinear organization that "contains nodal points at which two or more treads meet, then continue as a single group ... several treads either running independently or in places connected by side-paths—flow into the nucleus. In other words often ... a symptom ... is multiply determined or overdetermined" (Breuer & Freud, 1895/1955, pp. 288-290). Based on such concentrically organized, stratified themes or memories, Freud's topographical and spatial models of mental organization emerged.

Hence, Freud's hypothesis of memory includes the idea of networks of associations and representations, part of which become at times active (perhaps the equivalent of conscious) and part that remain inactive (or unconscious). According to Freud, an important aspect of memory is the passage of time because he hypothesized that a sequence of associations that are nonlinearly connected is formed over time and that memories are contained in multiple representations.

Similarly, in *The Aetiology of Hysteria*, Freud (1896/1962) described memories as chains of associations with convergences and divergences through nodal points like a complex network. In addition, in a letter to Fliess, Freud (1950/1985) described memory as a process of continuous elaboration:

Our psychic mechanism has come into being by a process of stratification: the material present in the form of memory traces being subjected from time to time to a rearrangement in accordance to fresh circumstances—to a retranscription.... Memory is present not once but several times over.... Successive registrations represents the psychic achievements of successive epochs of life.... At the boundary between two such epochs a translation of the psychic material must take place. (In psychoneuroses such translation does not take place in case of some material.)

Interestingly, he then added, "A failure of translation—this is what is known clinically as 'repression.' The motive for it is always a release of the unpleasure that would be generated by the translation."

Here, Freud introduced the idea that memories are permanent modifications of the central nervous system but remain in a dynamic state and are continuously updated, unless they are pathogenic. According to his view, pathogenic memories that remain in the unconscious—as might be the case for highly traumatic memories, which lead to mental disorders such as post-traumatic stress disorder (PTSD) and conversion or dissociative disorders (hysteria)—do not change over time (no transcription or retranscription), most likely because of repression. This may perhaps be translated into neuroscientific terms as follows: These memories are not retrieved and therefore not reactivated. perhaps because of a blockade due to repression exerted on the retrieval process per se. Hence, they cannot undergo either reconsolidation or updating.

In the Project for a Scientific Psychology, Freud (1950/1966) also elaborated on the cellular mechanisms of memories and proposes that the mnemic trace, a metapsychological construct, results from the facilitation of neurons and the involvement of a differential system of inscription of perceptions so that consciousness and perception are physically distinct from the unconscious and the lasting quality of mnemic traces. He gave a detailed description of what he hypothesized to be the cellular substrates of memory. In Chapter 3 of the Project, Freud wrote,

We assume that these neurons (the  $\psi$  neurons) are permanently altered by the flux of excitation; or rather, if we introduce the contact barrier theory, that their contact barriers are in a state of permanent alteration.... This alteration must depend on the fact that the contact barriers become more capable of conduction and less impermeable, that is, more similar to those of the  $\varphi$  system. We shall describe this situation of the contact barriers as their degree of facilitation. We may therefore state that memory is represented by facilitations that exist between the  $\psi$ neurons. (p. 299)

This is a surprisingly accurate definition of what was discovered in the 1970s and called long-term potentiation, which is now believed to represent the cellular mechanisms underlying memory formation (Bliss & Collingridge, 1993).

From the many letters to Wilhelm Fliess, we know that Freud was interested in understanding the neurobiological bases of psychological functions. However, his enthusiasm turned into frustration because the neuroscience of the late 19th century and early 20th century was too rudimentary to allow for a test of his hypotheses. He decided not to finish the book and even wanted the manuscript to be destroyed. The work remained unpublished until 1950, when it was translated into English with the title Project for a Scientific Psychology (Freud, 1950/ 1966). These hypotheses were not abandoned but instead reformulated in *Inter*pretation of Dreams (Freud, 1900/1953), in which he wrote,

We may describe as a memory trace and to the function relating to it we give the name of memory.... Memory traces can only consist in permanent modifications of the elements of the system. But, as already pointed out elsewhere, there are obvious difficulties involved in supposing that one and the same system can accurately retain modifications of its elements and yet remain perpetually open to the reception of fresh occasions for modifications.... We must therefore assume that the basis of association lies in the mnemonic systems.... Our memories—not excepting those which are mostly deeply stamped in our minds—are in themselves unconscious. They can be made conscious; but there can be no doubt that they can produce all their effects while in an unconscious condition. (pp. 538–539)

These conclusions provide important concepts concerning the definition and functioning of memories; that is, memories are built on permanent modification. However, they become active only partially at one time, and only the active parts are open to receiving and making new associations.

It is also important to remember that, as mentioned previously, in addition to the formulation of the theoretical framework of how memories exist in the psychic apparatus and how they can be supported by cellular substrates, Freud elaborated the definition and concepts of memories embedded in a context of trauma. He therefore provided a theoretical view of memories created, stored, and living in traumatic experiences. Freud elaborated on traumatic memories in *Studies on Hysteria* (Breuer & Freud, 1895/1955), in which he wrote,

A memory of a psychical trauma enters the great complex of associations, it comes alongside other experiences, which may contradict it, and it is subjected to rectification by other ideas.... In this way a normal person is able to bring about the disappearance of the accompanying affect through the process of association. To this we must add the general effacement of impressions, the ... forgetting.

On the other hand, the memories of ... hysterical phenomena persist for a long time with astonishing freshness and ..., unlike other memories of their past lives, are not at the patient's disposal.... These memories constitute an exception in their relation to all the wearing-away processes.... These memories correspond to traumas that have not been sufficiently abreacted; ... and we find at least 2 sets of conditions under which the reaction to the trauma fails to occur: ... In the first group, ... the nature of the trauma excluded a reaction (loss of a loved person or social circumstances). The second group of conditions are determined by the psychical state in which the patient received the experiences (severely paralyzing fright, semi-hypnotic twilight state of daydreaming, auto-hypnosis).... Both these conditions that the psychical trauma cannot be disposed. In the first group the patient is determined to forget the experience and exclude them from association; in the second group, the associative working-over fails to occur because there is no associative connection between the normal state of consciousness and the pathological ones in which the ideas made their appearance. (pp. 9-11)

Therefore, here, in agreement with his writings to Fliess, Freud stresses that traumatic memories are incredibly strong, less flexible than other normal memories, less susceptible to forgetting, and excluded from the activation by retrieval.

# 14.3 MEMORY TRACES, CONSOLIDATION, AND RECONSOLIDATION ACCORDING TO NEUROSCIENCE AND POTENTIAL LINKS TO PSYCHOANALYSIS

In neuropsychological terms, a memory trace is an *engram*, a hypothetical means by which information is stored as biophysical or biochemical change in the brain (and other neural tissue) in response to external stimuli. It remains unclear which biophysical or biochemical mechanisms underlie or

represent an engram. However, a great deal of experimental evidence from both humans and experimental animals throughout the past century has demonstrated that biological changes do indeed occur in the brain following new learning; these changes are required for maintaining information over time or, in other words, to store memories.

Electrophysiological studies show that newly learned information is encoded in the brain as patterns of neuronal activity (Eichenbaum, 2004). With time, this information is transformed into more persistent modifications, which seem to be engrained in molecular or structural forms such as structural modifications of existing synapses or formation of new ones (synaptic plasticity). This process of transforming the activity induced by new learning into stable, long-lasting modifications has been termed memory consolidation (McGaugh, 2000). An important feature of the memory consolidation process is that for a limited time after learning, the new trace is labile because it can easily be disrupted by several types of interfering events. In fact, experiments that began at approximately the end of the 1800s and beginning of the 1900s, but then increased significantly in the past 50 years, have shown that if a new memory is exposed to challenges such as brain trauma, seizure, a second learning event, or pharmacological treatments of many sorts, it fades away, and recall tests at later times show amnesia. This has been found in a multitude of types of memories and animal species including humans (Squire, Stark, & Clark, 2004). With time, however, the memory becomes increasingly stable until it is fully insensitive to disruption or consolidated. Indeed, if the interfering challenge is presented sometime after the memory is formed, no effect is seen, and the memory survives perfectly. Hence, there is an opportunity for disrupting newly formed memories immediately after they are formed and for a limited time. How long does this time window of opportunity last? The answer to this is still debated. General interfering events, such as traumas or brain lesions, suggest that memory consolidation takes a relatively long time, which although variable in different memories, can take several years in humans. On the other hand, pharmacological and molecular interferences, such as an acute blockade of de novo protein synthesis, disrupt memories only if applied soon after training, but they are ineffective a few hours or days later. This temporal dichotomy seems to be due to different phases of the overall consolidation process (Alberini, 2011; see also Chapter 5).

However, a number of relatively recent studies, extending previous observations first published in the 1960s, showed that memory consolidation is not based on a unique, single process of molecular consolidation, and that once stabilized against these interferences, memories can again revert to a labile state for a limited period of time if retrieved or reactivated. These reactivated memories over time once again become stable and insensitive to disruption—a process that is detailed later and that has been termed reconsolidation (Alberini, 2005; Alberini, Milekic, & Tronel, 2006; Dudai, 2004; Nader, 2003; Sara, 2000). The chapters of this book summarize the studies and debated questions that remain to be addressed in this fascinating field.

These findings on memory reconsolidation revolutionized the way we think about long-term memory formation, storage, recall, and stability, or actually the unstable, dynamic nature of memory traces. Knowing that memories after retrieval are fragile, changeable, and disruptable is important for many reasons. For example, in addition to gaining a better understanding of mental processes, this knowledge provides an opportunity to develop more accurate therapeutic protocols in mental health, including psychoanalysis and psychotherapy, that specifically target the intrinsic features and mechanisms of mnemonic processes.

Following the rediscovery of memory reconsolidation, a few studies went on to examine the effect of employing behavioral or the combination of behavioral and pharmacological methods for treating psychopathologies such as PTSD and addiction (Suris, Smith, Powell, & North, 2012; see Chapters 5, 10, 12, and 13).

Our intent here is to discuss the role of trace reactivation in psychoanalysis. Specifically, we elaborate on how trace reactivation is important in psychoanalytic treatment and how it may represent a model for explaining the formation and expression of unconscious processes such as those that characterize the FU.

In 1914, Freud published *Remembering, Repeating and Working-Through*, which clearly established his position on analytic technique, namely that the cathartic method had yielded to the associative method. Freud emphasized that treatment needs to involve real psychic work for the patient for whom passive hypnosis is no longer clinically effective. The goal is to remember and "to fill in gaps in memory," as Freud states, and to "overcome resistances due to repression" (Freud, 1914/1958, p. 148).

What mechanisms are targeted when the patient, in the psychoanalytic setting, goes through the process of remembering and working-through? Obviously, the work of the analyst is to facilitate the re-evoking or reactivation of memories and promote the elaboration and the filling of the gaps. However, remembering, as Freud said and as we can see now in neuroscientific terms, is not a straightforward process.

During psychoanalysis, the subject undertakes the process of becoming aware (or conscious) of the underlying sources of his or her unconscious behavior, both intellectually and emotionally, by re-experiencing them and by redirecting the emotions toward the analyst and then reprocessing them in a new mode. Thus, remembering and elaborating past memories in the new analytic setting is a key component of the psychoanalytic process, whether used to learn about the mind and its functioning or to alleviate disturbances in therapeutic processes. But how does this happen? Why is it that re-experiencing emotions and recalling the past in the new, present setting guided by the analyst allows the subject to recognize his or her unconscious patterns of behavior and ultimately change them to better deal with the realities of adult life? The answer to this question is both important and complex because it is multifaceted.

According to neuroscientific knowledge, and particularly to the emerging view that normal memories exist in a very dynamic state, we can suggest that

psychoanalytic therapy and working-through critically implicate new encoding, consolidation of new traces, and reconsolidation of retrieved memories, all of which would provide the tools to fill the gaps and emotionally re-tune and redirect the personal experience. Specifically, with the rediscovery of memory reconsolidation, a great deal of enthusiasm has been directed to this memory process, and major roles for reconsolidation have been hypothesized in psychoanalysis (Bleichmar, 2010; Gorman & Roose, 2011). We note here, for the purpose of discussion, that it is debatable whether or not reconsolidation is a mechanism for memory updating outside of adding onto the same experience (see Tronel et al. (2005) and Chapter 5). Hence, although the discovery of reconsolidation critically shifted the way we think about the dynamic nature of long-term memory formation and storage, it is still unclear whether it is reconsolidation that plays a major role in the dynamic rewriting of memory traces or whether instead it is the formation of new memories, which therefore undergo new consolidation processes and, hence, exist in parallel to the old memories. Furthermore, as suggested by reconsolidation studies in animals and humans (see Chapters 5 and 10), the reconsolidation of declarative memories may be limited by time, thus only affecting recent and not yet consolidated memories. As such, it might not be the reconsolidation process that in the psychoanalytic process has the main role of updating memories in changing the representation of experiences. Furthermore, retrieval per se does not weaken or disrupt memories but, rather, can strengthen the memory via reconsolidation. Indeed, if no interference occurs within the time window of fragility, the memory reconsolidates and likely becomes stronger and more long-lasting (see Chapters 3, 5, and 10). On the other hand, we suggest that the new perceptions present in the psychoanalytic treatment, and importantly, the new affect and emotional state of the present while recalling the past, do indeed provide an opportunity for changing consolidated memories via new memory traces (updating) or even, in certain conditions, weakening recent memory traces by interfering with their reconsolidation (extinction or new learning during reconsolidation; see Chapters 8 and 9).

It is also possible, as discussed in Chapter 5, that the reconsolidation of emotional memories that have a more implicit rather than declarative nature may not be (or may be less) restricted by the age of the memory. If this is the case (which still needs to be proven), then reconsolidation, together with the consolidation of new traces, may play a more important role in psychoanalytic settings. This understanding will be important because we do not yet know whether unconscious memory traces are more mechanistically similar to implicit or explicit memories or whether they follow different rules. Along the same lines, another very important question that needs to be addressed, and that is relevant for discussing the role of memory stages and processes in psychoanalysis, is whether or not the memories formed during development follow similar or different rules as those formed in adulthood. Because most studies on the mechanisms of memory consolidation and reconsolidation

have been carried out in adults, it is important to question whether the same knowledge applies during development. Studies on consolidation and reconsolidation during developmental stages are greatly needed.

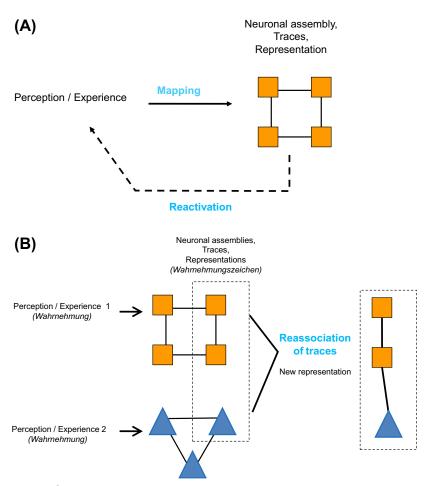
Despite all these questions that remain to be addressed, we believe that, as mentioned previously, consolidation of new traces together with updating of old memories via consolidation, as well as reconsolidation of recent traces, may all contribute to the complex process that occurs in psychoanalytic settings. Next, we propose a mechanistic hypothesis that may explain such a process.

#### 14.4 SYNAPTIC PLASTICITY AND TRACE REASSOCIATION: A WORKING MODEL FOR THE FREUDIAN UNCONSCIOUS

As mentioned previously, experience activates specific synaptic connections and therefore leaves a trace, or engram, in the neuronal networks through the mechanisms of synaptic plasticity (Morris et al., 2003; Neves, Cooke, & Bliss, 2008). The concept of "traces" as the neural counterparts of an encoded experience is represented in Figure 14.2A. Thus, an initial experience will provide an initial pattern of activated synapses; this pattern can be reactivated during recall, upon which the trace becomes prone to modifications (Figure 14.2A) (Braitenberg & Schüz, 1998; Buzsaki & Draguhn, 2004; Fuster, 2006; Gelbard-Sagiv, Mukamel, Harel, Malach, & Fried, 2008; Sakurai, 1999; Sutherland & McNaughton, 2000). In other words, real experiences as well as imaginary events will lead to the production of a trace and therefore of a mental reality through the mechanisms of synaptic plasticity. These mechanisms are common to both CU and FU. In the following section, we propose that trace reassociation is a key mechanism that distinguishes the FU from the CU. Memory traces may only partially maintain direct relationships between experience and representations because memory is dynamic and adaptive and builds on constructions based on selective attention, selective encoding, consolidation, and editing. However, such disconnections between experiences and traces would still contribute to conscious mental processes and, if nonconscious, would occur according to the logic of the secondary process.

Let's consider the possibility that parts of the neuronal assemblies that map for a given perceptual experience (Experience 1 in Figure 14.2B) can reassociate with elements of neuronal assemblies of a different experience (Experience 2, Figure 14.2B). The mechanisms that may drive this reassociation of traces are discussed later. This reassociation of traces will lead to the establishment of a new trace, built from elements of the traces left by the original experiences, producing a novel neuronal assembly and hence a novel representation (Figure 14.2B).

The mechanism of trace reassociation will introduce a discontinuity with the original experiences. Indeed, although primary traces are in a direct relationship with the original experience, the mechanism of trace reassociation produces a new set of traces, which is no longer in a direct relationship with



**FIGURE 14.2** (A) Perceptual experiences are mapped by the mechanisms of neuronal plasticity onto neuronal assemblies (traces). Reactivation of a given pattern of neuronal assemblies will provide a representation of the initial experience. (B) Neuronal assemblies, or parts thereof, encoding different perceptual experiences (perception/experience 1 and 2) can reassociate, leading to the establishment of a new neuronal assembly and hence a new representation. The geometrical forms represent cells and the connecting lines synaptic connections that would therefore constitute the neuronal assemblies.

the original experience, although it engages parts of the original traces. We hypothesize that the mental reality is constituted of original and reassociated traces, the latter possibly contributing at least in part to the unconscious as defined by Freud. Indeed, the idea that traces (which Freud called *Wahrrnehmungszeichen*) left by perception (*Wahrnehmung*; Figure 14.2B) can reassociate to establish new traces is a notion initially proposed by Freud in 1895 in the *Project* (1950/1966) and in 1896 in a letter to Fliess (1950/1985).

This notion of trace reassociation can be revisited in light of the recent experimental evidence concerning activation and reactivation of traces during

memory formation and recall that we reviewed previously. To summarize this concept briefly, newly learned information becomes a stable memory through memory consolidation. However, stabilized memories, in some circumstances, can become labile if their trace is reactivated. Retrieval results in the reactivation of the memory trace and, importantly, also mediates the formation of new associations. Hence, new as well as reactivated traces can in principle also reassociate with other reactivated traces. We speculate that this integration of traces within other reactivated and temporally dissociated or partial neuronal traces may lead to a condition in which a discontinuity in content and time may exist between the original trace and the new reassociated traces (Figure 14.2B).

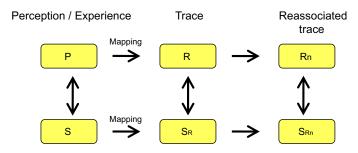
From this point of view, these types of reassociated traces do not constitute a factual memory representation, even though they were initially generated by an experience. One can view these new representations constituted from the reassociation of traces as a mechanism that produces unconscious mental representations that may contribute to the establishment of the FU. However, if representations produced by trace reassociation reach consciousness, they might result in what has been defined as "memory distortions," which include false memories, intrusions, and confabulations. For example, inadequate binding of representations of a learned or reactivated event can result in memory failure, in which fragments of an episode are retrieved but there is no recollection of how or when the fragments were acquired.

To recapitulate, one can posit that an internal reality is created by the mechanisms of synaptic plasticity underlying trace inscription, reactivation, and elaboration through reassociations. A discontinuity between experience and traces may emerge through the mechanism of trace reassociation and provides a potential mechanism for the establishment of the FU. In other words, we posit that the discontinuity and synchronic reassociation of traces represent the basic mechanisms of what Freud defined as the primary process. We further conclude that the FU, although being constituted by reassociated traces, is not a factual memory but, rather, an elaboration of an internal reality that is created and maintained by long-term brain plasticity mechanisms.

#### 14.5 HOMEOSTATIC PROCESSES AND SOMATIC **STATES**

We thus propose that one of the mechanisms through which a component of the FU is established is trace reassociation. As a next step, the nature of the principle(s) that determines the reassociation of traces has to be established. We posit here that the pleasure principle, which is central to Freudian theory, could be one such principle (Freud, 1920/1955, pp. 7–11). With the pleasure principle, Freud postulated that certain aspects of mental life are guided by pleasure-seeking behaviors originating from the unconscious. The principle of pleasure, which supposedly governs mental functions, was also viewed by Freud as a principle of non-displeasure. In addition, in the face of clinical phenomena that indicate a compulsive tendency to repeat unpleasant experiences, Freud recognized the "beyond the pleasure principle." Pleasure and displeasure appeared to be linked, a system of displeasure being triggered and existing in parallel with that of pleasure. Thus, the Freudian pleasure principle is essentially a non-displeasure principle, encompassing those physiological processes that maintain bodily homeostasis.

To address the physiological processes at the basis of the pleasure principle, we consider the theory of emotions as proposed by William James (1890/1950) and its renaissance in the light of contemporary neuroscientific evidence by Antonio Damasio (1994) leading to the somatic markers theory. The fundamental idea is that perceptions, particularly those charged with emotional tones, are associated with a particular somatic state (Figure 14.3). Thus, whereas a perception is emotionally neutral, its somatic state, such as increased heart rate and respiration, will determine its emotional tone, such as fear, rage, or pleasure. For example, on the basis of clinical observations, it has been suggested that the anticipation of a given somatic state is a critical determinant of decision making (Bechara, Damasio, Tranel, & Damasio, 1997). Indeed, the determining factor will be the anticipation of the least unpleasant somatic state that will result from enacting the decision process (Damasio, 1996). This process implies that mental representations are associated with representations of somatic states (Figure 14.4). Interestingly, the Freudian concept of "drive" (Freud, 1915a/1957, p. 122), which is taken as a concept at the interface between the mental and the somatic, resonates with this notion (Figure 14.4). Indeed, a given somatic state S in Figure 14.4 will be perceived through the interoceptive nervous system (Craig, 2003) and will trigger physiological regulatory mechanisms aimed at maintaining homeostasis. Similar regulatory mechanisms will also be triggered by the reactivation of its mapping (S<sub>R</sub> in Figure 14.4). Because the somatic state S, or its mapping as S<sub>R</sub>, is associated



P: perception/experience

R : representation of P (neuronal assembly)

S: somatic state

S<sub>R</sub>: representation of S (neuronal assembly)

**FIGURE 14.3** Perceptual experiences (P) and their associated somatic state (S) are mapped as traces (R and  $S_R$ , respectively) by the mechanisms of synaptic plasticity. These traces can undergo the process of reassociation and yield new sets of traces ( $R_n$  and  $S_{rn}$ ).

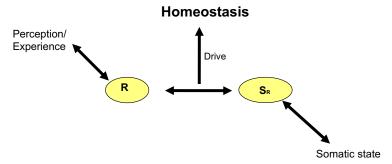


FIGURE 14.4 Representations of perceptual experiences (R) are associated with representations of somatic states (S<sub>R</sub>). A given somatic state or the reactivation of its mapping (S and S<sub>R</sub>, respectively) is likely to correspond to a departure from the physiological state of homeostasis. A breach in somatic homeostasis will be perceived as unpleasant because it may signal a threat to the organism's integrity. Because the somatic state, or its mapping as  $S_R$ , is associated with a representation R of a given experience, the return to a homeostatic state of the body can be operated only through the enactment of an action related to the content of the representation R. The association of R with  $S_R$  defines the notion of "drive" and results in an action whose aim is to re-establish homeostasis.

with a representation R of a given experience (Figure 14.4), the return to a homeostatic state of the body can be operated only through the enactment of an action related to the content of the representation R. The association of R with S<sub>R</sub> defines the notion of "drive" and results in an action whose aim is to re-establish homeostasis (Figure 14.4). In Freudian theory, the aim of the "drive" is to discharge the internal excitation and to return to a previous state (Freud, 1920/1955, pp. 34–43). Thus, in our view, the Freudian notion of "drive" represents at the level of the FU a parallel with the notion of "decision" taken from a cognitive perspective. Indeed, the anticipation of pleasure and unpleasure as defined in the somatic markers theory (Damasio, 1996) provides a basis to appraise the pleasure principle of Freudian theory (Freud, 1920/ 1955).

The question of the pleasure principle is intimately related to the psychoanalytic concept of repression. In the Freudian theory, the aim of repression is to prevent unpleasure by removing unwanted, unpleasant representations from consciousness. According to Freud, repression provides one of the mechanisms for the generation of the unconscious (Freud, 1915b/1957; Freud, 1915c/1957, pp. 146-158; Freud, 1923/1961, pp. 13-18). A role of the dorsolateral prefrontal cortex in voluntary suppression of unwanted memories has been proposed (Anderson et al., 2004). Whether this suggestion is relevant to unconscious repression as postulated by Freud remains to be demonstrated.

#### 14.6 THREE DISTINCT MECHANISMS OF THE FU

Based on our previous discussion, we propose that the unconscious (*unbewusst*) that could not be explained by mechanisms of repression in Freudian terms

(third unconscious; Figure 14.4) could be produced by the reassociation of traces. As we have seen, representations of experiences are associated with representations of particular somatic states. Through the process of trace rearrangement, traces encoding both the experience and the associated somatic state become changeable, creating a potential for reassociation. Anticipation of the least unpleasant somatic state will be a key determinant of the nature of the reassociation. Thus, the pleasure principle and its homeostatic function may play a major role in the process of trace reassociation and may contribute to one of the mechanisms for the establishment of the FU.

Through the mechanism of trace reassociation, new representations are created that, although integrating elements of the original experience, are distinct from the original representation. As previously discussed, through this process, a discontinuity between experience and its inscription within a trace network is created that might form one of the basic mechanisms for the FU. This implies that the FU, or at least the component generated by trace reassociation, is not a pure memory trace. Furthermore, the possibility to associate parts of traces left by distinct experiences and to integrate them into a new and unique one (Figure 14.1B) could be viewed as a mechanism to create what Freud described as the process of condensation. According to the Freudian theory, condensation is a process through which a unique representation embeds several associative chains (Freud, 1923/1961, p. 18). Freud viewed this process as the basis of the mental activity operating during dreaming (dream-work) and other manifestations of the unconscious. Typically, in condensation, multiple dream-thoughts are combined into a single element of the manifest dream; this would explain how people and places tend to meld into composite figures in our dreams (Freud, 1900/1953, pp. 279-304). Another mechanism discussed by Freud is displacement; in psychoanalytical terms, the "intensity" associated with a representation is displaced onto another (Freud, 1900/1953, pp. 305–309). A typical example is provided by an unjustified fear (phobia) of neutral objects or situations, such as phobias of harmless animals or of confined spaces. In our model, we propose that a somatic state associated with a given representation can be displaced and associated with another representation through the mechanisms of trace reassociation. This could thus lead to associations that have not been experienced and therefore are part of the FU.

A combined analysis of the Freudian theory and contemporary neuroscience, focused on two cardinal physiological mechanisms—namely synaptic plasticity underlying trace reassociation and homeostatic processes related to somatic states—provides a heuristic within which to identify different kinds of unconscious processes (Ansermet & Magistretti, 2007): (1) the CU, (2) the unconscious produced by repression, and (3) the unconscious resulting from trace reassociation and discontinuity. The proposed classification may provide a framework to orient clinical work. Indeed, clinical interventions should aim at bringing to consciousness the latent form of unconscious, promote the interpretation of the unconscious produced by repression, and use the potential for change offered by the unconscious resulting from trace reassociation.

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#### REFERENCES

- Alberini, C. M. (2005). Mechanisms of memory stabilization: are consolidation and reconsolidation similar or distinct processes? Trends in Neurosciences, 28(1), 51-56.
- Alberini, C. M. (2011). The role of reconsolidation and the dynamic process of long-term memory formation and storage. Frontiers in Behavioral Neuroscience, 5(12). http:// dx.doi.org/10.3389/fnbeh.2011.00012.
- Alberini, C. M., Milekic, M. H., & Tronel, S. (2006). Mechanisms of memory stabilization and de-stabilization. Cellular and Molecular Life Sciences, 63(9), 999-1008.
- Anderson, M. C., Ochsner, K. N., Kuhl, B., Cooper, J., Robertson, E., Gabrieli, S. W., et al. (2004). Neural systems underlying the suppression of unwanted memories. Science, *303*(5655), 232–235.
- Ansermet, F., & Magistretti, P. (2007). Biology of Freedom: Neural Plasticity, Experience and the Unconscious. New York: Other Press.
- Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. Science, 275(5304), 1293-1295.
- Bleichmar, H. (2010). On: memory in a labile state: therapeutic application. International Journal of Psychoanalysis, 91(6), 1524-1526.
- Bliss, T. V., & Collingridge, G. L. (1993). A synaptic model of memory: long-term potentiation in the hippocampus. Nature, 361(6407), 31-39.
- Braitenberg, V., & Schüz, A. (1998). Cortex: Statistics and Geometry of Neural Connectivity. Berlin: Springer.
- Breuer, J., & Freud, S. (1955). Studies on hysteria. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. II. London: Hogarth Press. (Original work published 1895).
- Buzsaki, G., & Draguhn, A. (2004). Neuronal oscillations in cortical networks. Science, *304*(5679), 1926–1929.
- Cowey, A., & Stoerig, P. (1991). The neurobiology of blindsight. Trends in Neurosciences, *14*(4), 140–145.
- Craig, A. D. (2003). Interoception: the sense of the physiological condition of the body. Current Opinion in Neurobiology, 13(4), 500-505.
- Damasio, A. (1994). Descartes' Error: Emotion, Reason and the Human Brain. New York: Putnam.
- Damasio, A. R. (1996). The somatic marker hypothesis and the possible functions of the prefrontal cortex. Philosophical Transactions of the Royal Society of London - Series. B: Biological Sciences, 351(1346), 1413–1420.
- Dehaene, S., Changeux, J. P., Naccache, L., Sackur, J., & Sergent, C. (2006). Conscious, preconscious, and subliminal processing: a testable taxonomy. Trends in Cognitive Sciences, 10(5), 204-211.
- Del Cul, A., Baillet, S., & Dehaene, S. (2007). Brain dynamics underlying the nonlinear threshold for access to consciousness. PLoS Biol, 5(10), e260.
- Driver, J., & Vuilleumier, P. (2001). Perceptual awareness and its loss in unilateral neglect and extinction. Cognition, 79(1-2), 39-88.
- Dudai, Y. (2004). The neurobiology of consolidations, or, how stable is the engram? Annual Review of Psychology, 55, 51-86.

- Eichenbaum, H. (2004). Hippocampus: cognitive processes and neural representations that underlie declarative memory. Neuron, 44(1), 109-120.
- Freud, S. (1953). On Aphasia, a Critical Study. London: Imago. (Original work published
- Freud, S. (1953). The interpretation of dreams. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vols. IV-V. London: Hogarth Press. (Original work published 1900).
- Freud, S. (1955). Beyond the pleasure principle. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. XVIII (pp. 1-64). London: Hogarth Press, (Original work published 1920).
- Freud, S. (1957a). Instincts and their vicissitudes. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. XIV (pp. 109-140). London: Hogarth Press, (Original work published 1915).
- Freud, S. (1957b). The unconscious. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. XIV (pp. 166-215). London: Hogarth Press, (Original work published 1915).
- Freud, S. (1957c). Repression. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. XIV (pp. 1141–1158). London: Hogarth Press, (Original work published 1915).
- Freud, S. (1958). Remembering, repeating and working-through. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. XII (pp. 145-156). London: Hogarth Press, (Original work published 1914).
- Freud, S. (1960). The psychopathology of everyday life. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. VI (pp. 1–279). London: Hogarth Press, (Original work published 1901).
- Freud, S. (1961). The Ego and the Id. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. XIX (pp. 1-59). London: Hogarth Press, (Original work published 1923).
- Freud, S. (1962). The aetiology of hysteria. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. III (pp. 187–221). London: Hogarth Press, (Original work published 1896).
- Freud, S. (1966). Project for a scientific psychology. In J. Strachey (Ed.), The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. I (pp. 281-397). London: Hogarth Press, (Original work published 1950).
- Freud, S. (1985). Letter to Wilhelm Fliess, December 6, 1896. In J. M. Masson (Ed.), The Complete Letters of Sigmund Freud to Wilhelm Fliess: 1887–1904 (pp. 207–215). Cambridge, MA: Harvard University Press, (Original work published 1950).
- Fuster, J. M. (2006). The cognit: a network model of cortical representation. *International* Journal of Psychophysiology, 60(2), 125-132.
- Gazzaniga, M. S., Ivry, R. B., & Mangun, G. R. (2008). Cognitive Neuroscience: The Biology of the Mind (3rd ed.). New York: Norton.
- Gelbard-Sagiv, H., Mukamel, R., Harel, M., Malach, R., & Fried, I. (2008). Internally generated reactivation of single neurons in human hippocampus during free recall. Science, *322*(5898), 96–101.
- Global Burden of Diseases Study. (2009). Operations Manual: Final Draft. Retrieved from http://www.globalburden.org/GBD\_Study\_Operations\_Manual\_Jan\_20\_2009.pdf.
- Gorman, J. M., & Roose, S. P. (2011). The neurobiology of fear memory reconsolidation and psychoanalytic theory. Journal of the American Academy of Psychoanalysis, 59(6), 1201-1220.
- Hassin, R. R., Uleman, J. S., & Bargh, J. A. (Eds.). (2005). The New Unconscious. Oxford: Oxford University Press.
- Hobson, J. A., & Pace-Schott, E. F. (2002). The cognitive neuroscience of sleep: neuronal systems, consciousness and learning. Nature Reviews Neuroscience, 3(9), 679-693.
- James, W. (1950). The Principles of Psychology. New York: Dover, (Original work published 1890).

- Kandel, E. R. (1998). A new intellectual framework for psychiatry. American Journal of Psychiatry, 155(4), 457-469.
- Kandel, E. R. (1999). Biology and the future of psychoanalysis: a new intellectual framework for psychiatry revisited. American Journal of Psychiatry, 156(4), 505-524.
- Kihlstrom, J. F. (1987). The cognitive unconscious. Science, 237(4821), 1445–1452.
- Kouider, S., & Dehaene, S. (2007). Levels of processing during non-conscious perception: a critical review of visual masking. Philosophical Transactions of the Royal Society of London - Series B: Biological Sciences, 362(1481), 857-875.
- Lau, H. C., & Passingham, R. E. (2007). Unconscious activation of the cognitive control system in the human prefrontal cortex. Journal of Neuroscience, 27(21), 5805-5811.
- Mancia, M. (2004). The dream between neuroscience and psychoanalysis. Archives italiennes de biologie, 142(4), 525-531.
- McGaugh, J. L. (2000). Memory—A century of consolidation. Science, 287(5451), 248-251.
- Morris, R. G., Moser, E. I., Riedel, G., Martin, S. J., Sandin, J., Day, M., et al. (2003). Elements of a neurobiological theory of the hippocampus: the role of activity-dependent synaptic plasticity in memory. Philosophical Transactions of the Royal Society of London - Series B: Biological Sciences, 358(1432), 773-786.
- Nader, K. (2003). Memory traces unbound. Trends Neuroscience, 26(2), 65-72.
- Neves, G., Cooke, S. F., & Bliss, T. V. (2008). Synaptic plasticity, memory and the hippocampus: a neural network approach to causality. Nature Reviews Neuroscience, 9(1), 65 - 75.
- Nielsen, T. A. (2000). A review of mentation in REM and NREM sleep: « Covert » REM sleep as a possible reconciliation of two opposing models. Behavioral and Brain Sciences, 23(6), 851-866, discussion 904-1121.
- Rees, G. (2007). Neural correlates of the contents of visual awareness in humans. Philosophical Transactions of the Royal Society of London - Series B: Biological Sciences, *362*(1481), 877–886.
- Sakurai, Y. (1999). How do cell assemblies encode information in the brain? Neuroscience & Biobehavioral Reviews, 23(6), 785-796.
- Sara, S. J. (2000). Retrieval and reconsolidation: toward a neurobiology of remembering. Learning & Memory, 7(2), 73-84.
- Shulman, R. G., & Reiser, M. F. (2004). Freud's theory of mind and functional imaging experiments. Neuro-Psychoanalysis, 6(2), 133-164.
- Snodgrass, M., & Shevrin, H. (2006). Unconscious inhibition and facilitation at the objective detection threshold: replicable and qualitatively different unconscious perceptual effects. Cognition, 101(1), 43-79.
- Solms, M. (2004). Freud returns. Scientific American., 290(5), 82-88.
- Squire, L. R., Stark, C. E., & Clark, R. E. (2004). The medial temporal lobe. Annual Review of Neuroscience, 27, 279-306.
- Suris, A., Smith, J., Powell, C. M., & North, C. S. (2012). Interfering with the reconsolidation of traumatic memory: sirolimus as a novel agent for treating veterans with posttraumatic stress disorder. Annals of Clinical Psychiatry. In press.
- Sutherland, G. R., & McNaughton, B. (2000). Memory trace reactivation in hippocampal and neocortical neuronal ensembles. Current Opinion in Neurobiology, 10(2), 180-186.
- Takeuchi, T., Miyasita, A., Inugami, M., & Yamamoto, Y. (2001). Intrinsic dreams are not produced without REM sleep mechanisms: evidence through elicitation of sleep onset REM periods. Journal of Sleep Research, 10(1), 43-52.
- Tronel, S., Milekic, M. H., & Alberini, C. M. (2005). Linking new information to a reactivated memory requires consolidation and not reconsolidation mechanisms. PLoS Biol, 3(9), e293.
- Vuilleumier, P., & Pourtois, G. (2007). Distributed and interactive brain mechanisms during emotion face perception: evidence from functional neuroimaging. Neuropsychologia, *45*(1), 174–194.
- Weiskrantz, L. (1996). Blindsight revisited. Current Opinion in Neurobiology, 6(2), 215-220.