

WHEN THE PAST IS ALWAYS PRESENT

A MODEL FOR THE FORMATION AND EXTINGUISHING OF TRAUMATIC MEMORIES

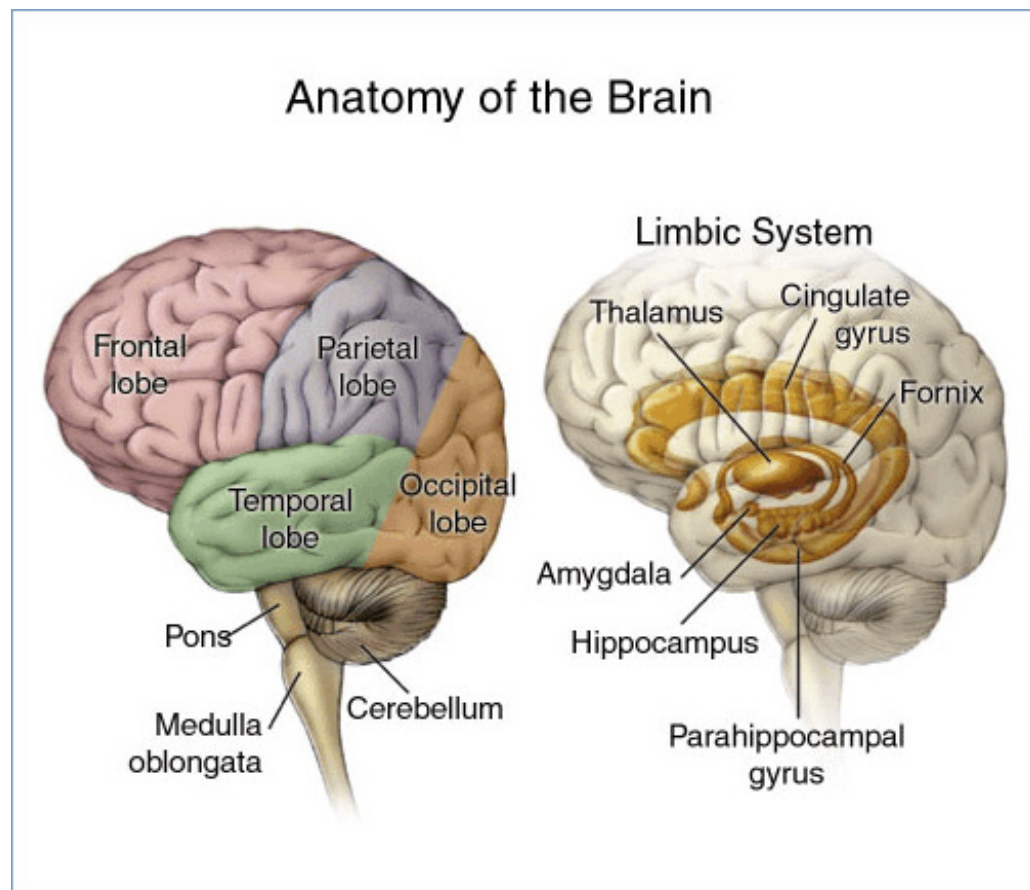
This talk was inspired by energy psychology and is offered as another way to view EP

There are haunted souls. Haunted by memories of things past that won't go away. Some of these memories are available to the conscious mind while others are hidden in the realm of the subconscious only to arise in our dream world. They activate emotional and physical responses that are pathological in intensity and duration. Over time, they produce a maladaptive response that impairs judgment, alters mood, is experienced as pain and diminishes the ability to see new ways to solve problems.

These immutable memories can affect every aspect of life.

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The limbic system drives survival behavior and is involved with emotion, learning and memory



It is believed that these memories activate an area of the brain called the limbic system. This part of the brain is the generator of our emotional life. The limbic system is part of the old brain that evolved before reptiles and amphibians, functionally designed to ensure survival. It's why we are here. In the face of danger the limbic system prepared us to flee, or if necessary, send a message that one was prepared to fight. Once the danger had passed and we were safe, the passage of time would normally diminish the relationship between the memory of the event and the emotions it generated. For humans, at least, this is not always true.

What happens inside our brain that allows these memories to haunt us? The answer lies in a process called traumatization. We define traumatization neurobiologically as:

TRAUMATIZATION

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the process that permanently encodes and consolidates a link between emotional, cognitive and somatic components of the traumatizing event within the limbic system. Subsequent activation of the limbic system by any of these components retrieves part or all of the memory causing the release of stress hormones. Furthermore, after activation, this memory must become labile, subject to de-novo protein synthesis for re-consolidation.

We avoid here the ambiguity of what is a trauma and choose to define it operationally. The process of traumatization requires both an inescapable, fearful or highly unpleasant event and a permissive neurobiological landscape to produce a permanent link. During this talk, the questions that I would like to address include:

GOALS

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---For the purposes of this talk what types of traumatic memories are there?

---What is required for traumatization?

---How does it produce an immutable pathway linking the memory of the event with physical, cognitive and emotional components?

—What disorders are related to traumatization?

---Why does the link between memory and emotion become labile during reactivation?

---What therapies are available to de-link this encoded memory?

Clinically, we see that components of the event can be divided into two main groups. When accessible to conscious retrieval these are simply called **traumatic memories**. These memories are bound into a coherent whole. The thought and the emotional response are linked. Emotional and physical responses that are not linked to a cognitive image are called **dissociated traumatic memories**. It becomes puzzling to the individual when they feel or experience something that is not related to anything they are aware of. These memories are stored in our subconscious, not available to conscious scrutiny. For this talk, we use the word ‘subconscious’ to mean mental content, generated by internal or external cues that are not consciously registered. When activated, these subconscious dissociated memories can cause somatic, cognitive and emotional responses, exactly as memories that *are* available to consciousness.

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TRAUMATIC MEMORIES

Traumatic Memory– Conscious thought that activates the limbic system. This is best seen in phobias

Dissociated Traumatic Memory– Subconscious stimuli that activates the limbic system. This is best seen in chronic pain.

After a traumatic event is consolidated, the memory is considered to be located at many places in the brain. This consolidation process is complex and poorly understood.

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COMPONENTS OF TRAUMATIC MEMORIES AND STIMULI

Cognitive
Emotional
Somatosensory

This memory can be activated by cognitive, emotional or somatosensory stimuli that can be conscious or subconscious, associated or related and can produce a response that is cognitive, emotional or somatic.

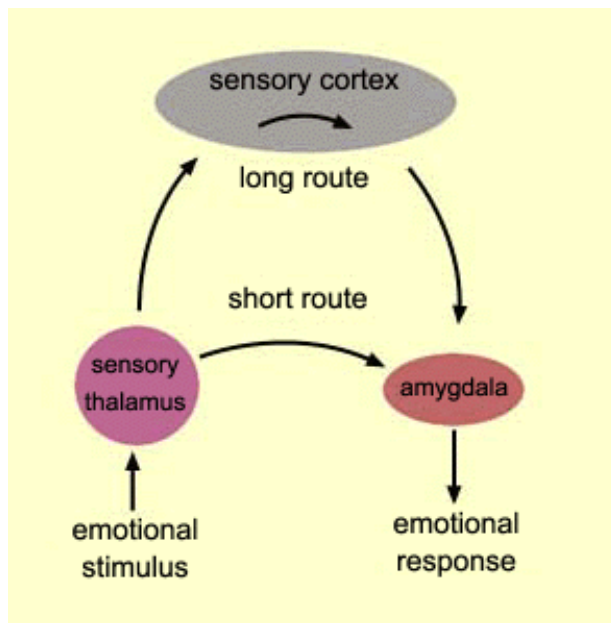
The issue of what is available and what is dissociated from consciousness is not well understood

It is remarkable of traumatic memories that acute anxiety for example can be devoid of a cognitive source such as in panic disorder, a dissociated traumatic memory. Chronic psychogenic pain is always devoid of its traumatic origin. What is also remarkable about traumatization is that somatosensory stimuli can produce affect and even event retrieval. For example, someone who has had a fear of

roller coasters may experience fear when there is turbulence on a plane and they get the same feeling in the stomach. For this talk, we will not be concerning ourselves with how or what does or does not become dissociated. There are theories that involve hippocampal binding and the like but it remains speculative. We will not be concerned about the details of this process except to note again that all three components of the traumatizing event, cognitive, emotional and somatosensory can act as stimuli and be the consequence of retrieval of the memory.

Traumatization is a remarkable event. It is not the norm. For example, estimates for individuals experiencing severe trauma suggest that about 15% will develop PTSD. This is a somewhat misleading number and suggests that the remainder have no effect at all. This is clearly not so. People can be traumatized but do not meet the criteria of PTSD. To understand what is different about traumatization we should first look at the normal functioning during a fearful event.

An event occurs. Sensory information enters the brain and heads to an area called the thalamus. Under normal conditions the sensory input is parceled off to areas of the hippocampus and higher cortical brain



areas where decisions are made. Through a complex evaluation, involving cortical and sub-cortical areas, lasting but milliseconds, an

Slide 6 Information processed by the sensory cortex uses multimodal pathways and involves an assessment by the orbito-frontal cortex. Information that goes directly to the amygdala produces an immediate response without evaluation.

assessment is made and a physiological and emotional

response is generated. These are experienced both physically and mentally. However, sometimes, especially if survival is perceived to be at stake, the input may be directly routed from the thalamus to an area of the brain that prepares one for action. No cognitive processing here, just prepare. Preparation requires a coordinated effort and if survival truly is at stake, one really doesn't want to have to think about getting ready. This preparatory process involves the activation of an area well suited to coordinate this task. This area is a small almond shaped area deep in the limbic system and is called the amygdala. Once activated, arousal occurs and there is potential for traumatization.

Under normal functioning the amygdala coordinates a response; emotions are writ large on our body and are experienced as feelings and prepare us for action that need to be taken to resolve a situation. It is in

the amygdala that the linkages producing traumatization are believed to reside. In its simplest form, if an event is traumatizing then the linkage is forged.

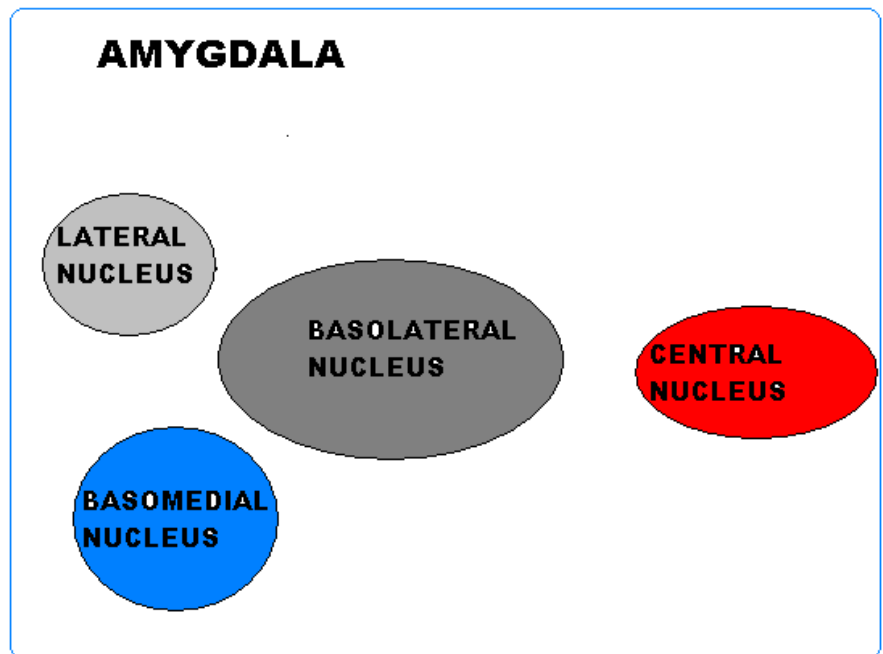
Traumatization

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Event → Traumatization occurs → Chronic Emotional/Painful State.

There are many emotions that emanate from the amygdala, including fear, anger, grief, guilt, jealousy, and so on. In addition, pain and other somatic symptoms that are encoded with the traumatization can also be expressed. Each has its own unique efferent neuronal pathways from the amygdala. It is for us to understand the process consequences of traumatization so we can begin to approach de-traumatizing the memory, that is

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Nuclei of the amygdala



de-link the memory
from its emotional,
physical and

cognitive components. In order to do this, a detailed look at the amygdala is necessary.

The amygdala is made of several nuclei; it is most commonly described as having a lateral nucleus, basolateral nucleus, basal and medial nucleus and the central nucleus. These nuclei have been broken down to even smaller areas but for our purposes we can use this model. Information from all over the brain enters the Lateral nucleus. It is here that the process of traumatization begins. Information is directed to the Basolateral nucleus (BLA) which functions to provide a unique pathway to prepare us. One of the functions of the amygdala is to prepare the individual for survival activities. Under stressful conditions neurons, activated by pathways arising in the BLA, enter the central nucleus and via efferent pathways cause the release of stress hormones and other chemicals that act to prepare and protect the body for action. These hormones produce a number of physiological changes that increase heart rate, increase blood flow to muscles, decrease blood flow to skin, alter immune functioning, change bowel function, dilate pupils, and produce an extreme focus of attention. These prepare us. It is the central nucleus of the amygdala that has efferents to many brain systems including memory systems.

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Efferents of the Central Nucleus

Ce of Amygdala

→ *Anterior pituitary* → *cortisol*

→ *Locus coeruleus* → *nor epinephrine*

→ *lateral hypothalamus* → *epinephrine*

→ *Basal nucleus* → *acetyl choline* → *intra Ce*
Opioids

The amygdala also receives information back from these systems and they influence each other in a feedback loop.

Events that arouse fear and defensive rage have been clearly associated with the amygdala. Fortunately, a large body of animal research has studied the reaction of the amygdala when fear is produced. Smaller amounts of animal research have studied the amygdala when rage is produced. It is in these two emotional responses that we can study the parameters that lead to traumatization. Rage and fear are the emotional precursors to the actions of what Cannon has described as fight or flight behavior. In the non-primate world, the animal either escapes or is killed. In primates, survival of a fear and rage response without fight or flight is also possible.

The attendant physical changes that prepare for flight as mentioned above can be accounted for by the initial amygdala activation. On the other hand rage and other emotional and somatic states have specific muscular, cognitive, and visceral peculiarities and require additional areas of the amygdala and brain to be activated. For rage, activation of additional efferent neurons produce an arching of the back, clenched jaw muscles, bearing of teeth, increased tension in the head, neck, and buttock muscles. We postulate that since rage and fear are the most primitive of emotions and, as such, are critical for survival they must also be the major sources of traumatization. Using the research about conditioned fear and its effect on amygdala function allows us to begin to model traumatization and its memory consolidation.

The amygdala produces a fear response by activating the efferent neurons from the central nucleus of the amygdala. This activation occurs because the thalamus/cortex/hippocampus has sent a signal that activates an unconditioned fear stimulus in the amygdala. Thus for any stimulus that activates an unconditioned fear stimulus (UFS) in the amygdala:

Pathway of Rage Stimuli

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Fear→ *collateral circumstances*—→*Basal nucleus*→ *Rage of amygdala*

The expression of rage is driven by collateral circumstances at the time of the event. For the purposes of traumatization we can make the distinction between defensive rage and predatory rage. Predatory rage is posturally different and involves action and as such cannot lead to traumatization, in and of itself.

Fear and rage are the consequence of amygdala activation. The motivation to action is also part of the coordinating function of the amygdala and takes place in other parts of the brain.

Not only does the amygdala produce emotions, but it is also the site where traumatizing memories leading to the pathological encoding of physical sensations occur as well. Under normal conditions, initiating consolidation of traumatic memories in the **amygdala** makes sense from an evolutionary point of view. It is here we want to begin the process. Once consolidated, initiation of a subsequent response also begins here. This allows us, very rapidly if necessary, to increase our chances of survival if we ran into similar circumstances. One would want the area that stores the linkage between memory and physiological state to be the same as that coordinates behavior and activates appropriate bodily responses. This **consolidation** of the traumatic memory in the amygdala has been shown to require both protein synthetic and neurochemical

processes. The amygdala also mediates memory storage in other parts of the brain as well. Thus, emotionally arousing events are better recalled than neutral ones, and this effect is absent in subjects with amygdala lesions. While the details are complicated for memory consolidation, we can probe the amygdala based consolidation of a **traumatic memory** consolidation by studying the effects of conditioned fear on experimental animals. It is important to understand though that the amygdala is only part of the consolidation process.

Using conditioned fear experiments we can study speed of acquisition of the response, consolidation, extinction and many other variables of learning and memory. They experiments include simple Pavlovian (show light/get footshock) and passive avoidance (latency to stepping off a vibrating platform onto a floor that is electrified). For our discussion, what evidence is there that the amygdala is the site where memory initiating consolidation is begun? What evidence do we have that the amygdala is the place for storing the pathways that ultimately lead to emotions, behavior and somatosensory experiences?

Studies of the effects of brain lesions published over six decades have suggested that the amygdala is involved with consolidating memory of fear and rage. The first studies, where bilateral temporal lobes were removed that contained the amygdala, demonstrated the inability of the animal to show fear or to generate anger. Beginning in the 1980's better techniques and molecular probes such as drugs that blocked (antagonists) and facilitated (agonists) neurotransmission and protein synthesis allowed for a more detailed investigation of emotionally based memory consolidation. The data suggest that simple neurotransmitters and neuromodulators enhance or inhibit consolidation of memories in the BLA. These include: nor epinephrine, glutamate, opioids, cortisol and acetyl choline.

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Neurochemicals that Facilitate Consolidation

Glutamate

Nor-epinephrine

Acetyl Choline

Neurochemicals that Inhibit Consolidation

GABA

Opioids

Cortisol

Highly localized infusions of these chemicals suggest that the BLA is selectively involved with the modulation of memory responsiveness. The adjacent central nucleus, the site of efferent signals, does not appear to be directly involved with consolidation of a fear memory, although the view that the Ce may be involved with neuronal plasticity is changing.

In the animal model, memory for different types of training involve different brain regions and the amygdala also regulates memory consolidation, that is synaptic plasticity at other brain sites such as the hippocampus, hypothalamus, cingulate, insular and orbito-frontal cortices. In addition, and to make things even more confusing, infusions of antagonists and agonists of a variety of neurochemicals directly to other areas of the brain can also influence consolidation of fear based memories. Thus, while the BLA is clearly a locus of consolidation, it is

not the only one. Nonetheless, we speculate that is a locus activated very early in memory consolidation. For this talk however we will consider just the BLA.

Given this simple overview, the question of how and why a traumatizing event creates a **permanent link, either conscious or subconscious of the emotional, physical, and cognitive states that produce the memory** needs to be addressed. Since clearly not everyone exposed to a fear or rage generating event develops traumatization, it has been speculated that traumatization requires a suitable landscape of the brain. In addition, the threatening event must be perceived as inescapable, one in which the individual feels helpless and powerless, without the ability to run or to fight. This threat does not have to be for one's life, for humans with their big prefrontal cortexes can experience threat to lifestyle or other loss and become traumatized. Thus, events that can become traumatic memories are those that do not have resolution and can include things that lead to pathological guilt, grief, jealousy and so on. The argument can be made that some events are so powerful that almost anyone would be traumatized. This we know is not true. Therefore, traumatization must occur at a singular moment in a person's life. The stage must be set, that is the amygdala, by the conjoined product of long term, short term and hormonal and genetic influences must be permissive for traumatization to occur. My experience has taught me that a pre-morbid anxiety state or prior stressful events alters the threshold to traumatization. This anxiety state can be the result of previous traumas, an inherently low threshold to stressors or hormonal changes such as those seen in puberty. These stressors produce in the amygdala a state that makes an event more likely to produce a traumatic memory. This kindling reflects the maladaptive response to previous stressors and if the stressor is chronic and inescapable, serotonin, a calming neurotransmitter is decreased. There is much data that confirms that patients who suffer from traumatization have

lower than normal levels of serotonin. Is this a consequence of the traumatization or a state that allows for the generation of a traumatic memory? It is why the SSRI's are useful in treating trauma related disorders such as panic disorder and PTSD as well as depression. The role serotonin plays in both the landscape and the threshold to traumatization is complex, but, it is my belief that it is low serotonin level that sets the stage for traumatization. In addition to the landscape, there are three other factors that appear to be critical to traumatization and are reflective of the more cortical aspects of the event. These include a sense of helplessness, the meaning of the event to the individual and the degree to which one perceives that life is at stake.

Since we never forget, a traumatic memory, be it simple or dissociated must activate the limbic system each and every time a link to the event, be it conscious or subconscious is experienced. One of two possible explanations exists for the permanence of this memory → activation relationship. Either an event related memory path is created that is immutable and prevents the relationship from being extinguished or the memory link between the stimulus and the activation of the response is reconsolidated every time it is activated. The data suggests that reconsolidation is the mechanism. This reconsolidation is a survival mechanism designed to insure that what it was that happened before will never happen again. A further discussion of reconsolidation is needed here.

Re-consolidation is the event that occurs after consolidation has taken place. It involves activation of the amygdala by one, two, or all of the three components of a traumatic memory; the three components being cognitive, emotional and somatosensory. The data strongly suggests that reconsolidation does not recapitulate consolidation. Several studies have demonstrated that different glutamate receptors and different temporal issues are involved with each. It is actually possible to show that these two processes are dissociable.

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TRAUMATIC MEMORY REQUIRES RECONSOLIDATION

Only when a traumatic event is consolidated and is able to be re-consolidated can we call this process traumatization. The first question that I will address is ‘why does the memory become labile and subject to disruption’? I speculate that re-consolidation improves the chance of survival. It is clear that consolidation is a different process than re-consolidation. Several studies have demonstrated that different glutamate receptors and temporal issues are involved in each. Thus, the purpose of re-consolidation is to allow for other stimuli that could possibly cue for the event be incorporated into the consolidated system efficiently as possible. Metaphorically, it is like having an upside down bucket with water flowing on it. If one considers that the inside of the upside down bucket is the consolidated memory system. After the initial event and traumatization there is only one hole on the bottom of the bucket where water can enter the consolidated memory system. Through the process of generalization we can add holes. This is an extremely effective way of making sure that any aspect of the traumatizing event that can alert us to danger becomes incorporated. One does not have to go through the whole process of consolidation. As we shall see later, in studying phobias which I consider a traumatization, if one develops a fear of crossing a particular bridge, it soon generalizes to other bridges. We speculate that generalization occurs via the process that is part of reconsolidation. Thus, one might prefer to call reconsolidation both a mechanism for new learning, certainly a survival mechanism that would be useful if one wanted to avoid a fear producing issue and remembering. Thus, a new stimulus that is reconsolidated on the activated consolidated memory adds a new conditioned stimulus that can activate the entire downstream process. Once a memory is activated, all stimuli that affect the outcome are

reconsolidated. We believe that this occurs in the BLA nucleus of the amygdala. The activation of the UFS in the BLA and its efferent outflow to the Ce therefore provides two locations to intervene to inhibit a retrieved memory from producing a response. One pathway involves inhibiting the flow from the UFS to the Ce. Alternatively, preventing UFS activation also constitutes a potential method of disrupting the traumatic memory.

I would like to take some questions if there are any and then take a break. In the next session I will discuss disorders that represent the consequences of traumatization and are what I consider to be amygdala based disorders.

In this part of the talk I would like to list five disorders that are amygdala based. An amygdala based disorder is one in which the primary pathology lies within. These diseases are all the consequence of traumatization but encoded in different ways. Being amygdala based, they are susceptible to

de-linking if one can inhibit reconsolidation. I would like to discuss three of five disorders in more detail and show how the amygdala is involved with the pathogenesis of the disease. These are phobias, PTSD and chronic psychogenic pain. In the phobic disorder fear is generated by essentially neutral objects or situations. Then why are we afraid? The answer as we shall see lies in its encoding. For psychogenic pain both the emotional and cognitive aspects of the trauma are separated from the pain. Recently a patient presented with severe hand pain. This was ultimately shown to be related to a traumatic taxi accident in London 15 years previously and she was returning to London to live. Treatment of the traumatic memory of the accident instantaneously removed the pain. Here the pain was completely dissociated from the event. Finally, PTSD can have the cognitive, emotional or somatosensory dissociated depending on the event. Panic disorder, although not a subject for today's discussion similarly has emotion without cognition. Pathological emotions also share a traumatic encoding. While other disorders can be considered as arising from a traumatizing event, such as dissociative identity disorder or borderline personality disorder, they are much more complex and involve aspects of character that probably do not originate in the limbic system.

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Amygdala based disorders

Phobias Panic

PTSD Pathological Emotions Pain

PHOBIAS

Phobias provide a wonderful model for the simplest form of amygdala based traumatization. They produce fear to objects and situations that are not inherently threatening. There is no evolutionary advantage to be terrified of public speaking or escalators, or the number 13. Certainly airplanes, cars, tunnels and bridges did not exist during early human evolution, so why do some people become terrified at the mere thought of

these objects. They are the easiest to cure because the emotional response they produce is often readily available by mere imaginal activation. If you have a phobia about crossing a bridge, you just think 'bridge' and you have a response. If one stops thinking about the bridge, the response is extinguished. This imaginal activation suggests that thought can be considered a sense, just like vision or hearing in its ability to activate the amygdala.

Phobias are characterized by a persistent, irrational and excessive fear of objects or situations. Since there is no real imminent danger associated with these objects or situations, they may be considered to be conditioned stimuli. But since phobias do not remit with time and can generalize, they should be considered to be undergoing reconsolidation, and hence meet our definition of traumatization.

Phobias are learned and as such are fundamentally different from responses to innate (unconditioned) fear stimuli (UFS). As described earlier, a fear response always occurs by exposure to an innate fear stimulus. These stimuli are non-specific but fall into broad categories that can be applied to many situations. Such stimuli are reflective of the many ways we can be killed, and are hard wired in the brain and include: fear of the unknown (novel situations), heights (falling), closed spaces, (being trapped), **being unable to run**, open places, (no place to hide), creepy crawly slithery things (land based predators) and something coming out of our visual field (air based predator). We experience fear before these stimuli can produce a full assessment. Details are unimportant, only avoidance is. We are ready and we don't yet have an idea what we are going to do.

Now if the amygdala has been kindled, by whatever mechanism, we propose the following process produces of a phobia.

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You are riding along in a car and go over a bridge and you look down and see the height (this is a UFS). It is the height that produces an innate fear response and you are unable to escape because you are riding in a car. However, since you are consciously aware that you are crossing a bridge, the bridge then becomes associated with the fear response. Thus, just like in classical conditioning, when you bring a bridge to mind you become fearful. Since the amygdala is kindled this experience causes a traumatization to occur in the amygdala and a phobia is formed. This requires a specific glutamate activated pathway. Thus, different phobias require different pathways. So, for example, there are different pathways that produce fear of elevators and fear of spiders. If the phobia generalizes, that is, fear is generated for all bridges, would need to de-link only the concept of going over a bridge to treat the phobia. I always try to have the patient remember the first incident that produced the phobia. This sometimes leads to interesting areas. For example, in treating fear of flying, which is a phobia, I have found that many individuals have had a bad experience on a roller coaster when they were young. This makes total sense if one considers the visceral stomach response seen both with the roller coaster and turbulence. Thus, the fear of falling and the visceral response to plummeting as we go over the top of the roller coaster is the same as the visceral response to turbulence. This somatic experience is the same as that produced by turbulence. The fear associated with the roller coaster is now experienced on the plane producing a new fear, fear of flying. This is generalization by somatic association. It is interesting that the individual who suffers from the fear of flying rarely if ever makes the connection to the roller coaster. Of course there are other mechanisms that apply, eg, closed spaces, fear of falling, for example, that are UFS's but I thought the visceral example to be instructive.

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Phobia Generation

Innate Unconditioned Fear Stimulus → *Conditioned Stimulus* →
(Height) (Bridge)

Traumatic Memory Consolidation → *Retrieval of Memory* → *Fear* →
(Bridge = Fear) (Bridge)

Re-consolidation and Generalization

Normal fear producing memories, can be forgotten or at least not produce a fear response when recalled. If there is traumatization, the memory retrieval produces fear and reconsolidates all stimuli that have become associated with this pathway.

PTSD

PTSD is a disorder that is associated with the fear of either being killed or severely injured-- or of witnessing something horrific. PTSD presents with a wide variety of symptoms

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and as in phobias not everyone exposed to a fearful or unpleasant event develops PTSD. We hypothesize that PTSD too, is an amygdala based disorder and we propose that reconsolidation of the traumatic memory is critical and sustains the disorder. While other events can be traumatizing (producing a permanent link in the amygdala) a diagnosis of PTSD has specific criteria. It is thought by some to have at least part of the traumatizing event dissociated. We believe that it is this dissociated part sustains the individual in an activated state since avoidance is not possible. This dissociated state can appear in our dreams, as intrusive thoughts or reactions to situations without our being aware of their impact on our

minds. The symptoms are those of a brain continually activated. Unlike phobias where we are **cognitively** aware of the non-threatening nature of the event although we are experiencing fear, PTSD is always **cognitively** experienced as life threatening. Furthermore, phobias are cognitively driven, that is shut off the sensory input about the phobic object or situation and the fear resolves, this is clearly not so with PTSD.

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PTSD Symptoms

Re-experiencing the trauma

Emotional numbing

Avoidance

Increased arousal

Dissociation makes the treatment of PTSD particularly difficult. However, some PTSD patients do remember the event and for those, simple de-linking is possible. However, many individuals with PTSD also have other psychological difficulties and these will need to be addressed as well.

After encoding of the trauma in the same amygdala nuclei used by phobic encoding, a retrieved traumatic or subconscious dissociated traumatic memory can activate the limbic system. The dissociated memory along with other aspects of the traumatizing event produces ongoing activation of the limbic system sustaining a chronic stress response that further kindles the amygdala and worsens the already difficult situation.

Thus, in PTSD not only are simple traumatic memories encoded but also dissociated traumatic memories. The peculiar thing about dissociated traumatic memories is that they cannot be activated by conscious thought. Thus, the effects of their activation, as seen in the slide, remain mysterious to the individual. While PTSD is encoded like phobias, that is, during a traumatization, it is unlike phobias as there is ongoing activation because

avoidance of those dissociated memories and limbic system activation is not possible. What evidence do we have that dissociated memories can cause problems? If it cannot be brought to mind does it really do harm? To answer this question we need to discuss another amygdala based disorder.

The third amygdala based disorder that I will discuss is chronic pain.

PAIN

Pain does not normally arise from memory. Thinking about a previously painful experience never reproduces the pain. Victims of torture do not re-experience the pain when it is brought to mind. They may become very distressed, but it does not reproduce the pain. Thus the ability to re-experience pain not only requires that the pain be stored as a dissociated memory (we can't associate the event with the pain we are feeling) but it must also be co-encoded with the trauma and be activated only by subconscious stimuli. Chronic pain that is un-anatomical in distribution, where there is no peripheral lesion and that resists traditional treatment should be considered of psychogenic origin. It is proposed that since the pain is co-encoded with the trauma the amygdala plays an important role in its expression. The idea that pain can originate in the brain has been around since the time of Charcot, Breuer and Freud. They called it hysterical pain, an unfortunate and pejorative descriptor. The brilliant insights and research of John Sarno, who uses the term tension myositis syndrome to describe the physiology of psychogenic pain, suggest that unresolved rage is at the root. Sarno considers this to be a centrally mediated vasoconstriction that produces ischemia in tendons and nerves. Robert Scaer in his book, *The Body Bears The Burden*, explains psychogenic pain as a consequence of a **procedural** memory being encoded with a trauma such as a fear producing motor vehicle accident. In addition, sensory changes including alterations of skin temperature, changes in sweating can also be observed.

In either encoding state, defensive rage or fear, pain which may be part of the event is often not experienced due to endogenous opioids that are released during the traumatizing moment. Thus, not only is the pain

dissociated during the initial encoding event (e.g. not experienced) subsequent stimuli that re- produces pain are dissociated from conscious awareness.

Encoding States of Trauma and the process of Traumatization

As mentioned above there are several criteria that are necessary for traumatization. The first is that the landscape be such that the amygdala has been kindled, either by previous experience or hormones or an inherent genetic sensitivity. Secondly, the event must cause great distress and the person must perceive that his life and/or his attendant family, friends and lifestyle is in danger, a fear response is generated and thirdly, escape is impossible. Defensive rage and a fear response are closely related emotions and thus are very similar neurochemically.

Bracha and coworkers reformulation of the fear survival response provides a sequence as to the potential timing of the traumatic encoding. First there is a freeze response or freezing. This is what animals do when they are exposed to danger, such as when feeding in an open field. If a stimuli, either conscious or subconscious alerts the animal to a potential predator a freeze response occurs and attention is focused on the location of the stimulus. From an evolutionary point of view this makes sense since a predator scans for movement. If a predator appears we must act to either try to escape or fight. Just prior to action, the emotions of fear and defensive rage are engaged. It is clear from Sarno's, Levine's and Scaer's work that when escape is not possible and fighting seems futile, a potential for traumatization is realized. From a neurobiological point of view, cortisol, nor epinephrine, endorphins and glutamate are all released in the limbic system. We speculate that if escape is accomplished and a safe place is found or the predator is killed or driven off serotonin is released globally into the brain.

If fear or rage is activated and continued flight or fight cannot occur, in some cases the animal enters into a state of tonic immobility. Bracha calls this state fright.

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Response to Stimuli

Freeze→ (rage/anger)→Fight or Flight→ Fright

Tonic immobility may enhance survival when a predator, thinking that his prey is dead temporarily loosens its grip, thus providing the prey an opportunity to escape. In humans, this may be described as dissociated state, that is, awareness of the event from an observer vantage point, not as a participant. It is here, in this state, we speculate that dissociation occurs. This state is also nature's way of being merciful; the animal is dissociated as long as signals that escape is not possible persist. These four responses freeze, fear and defensive rage, fight/ flight and fright have different neurobiological states and it is in one of these that traumatization, the permanent encoding, takes place.

Mechanisms of traumatization

Traumatization occurs because of events that produce changes in the amygdala. These events can only be encoded if the landscape of the amygdala is kindled. Kindling has genetic, hormonal and environmental influences. These include previous trauma, family history of traumatization, other underlying psychological problems such as Obsessive Compulsive disorder and generalized anxiety and puberty. This sets the stage for the event. An event is more likely to cause traumatic injury if

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Event Related Issues That Increase Risk of Traumatization

- The event produces intense emotion*
- The event is perceived as inescapable*
- There is perceived threat to oneself or others*
- There is perceived responsibility*
- There is no motoric component*

they ultimately involve issues of survival. If the perception during the event is one of helplessness and powerlessness, and the behavioral component the fight or flight does not occur, traumatization is possible. The first step in traumatization requires a link between the event as it enters the LA of the amygdale and ultimately links with and unconditioned fear stimulus. The second step involves the UFS sending efferent signals to the central nucleus that activates the stress system. This causes the release of nor epinephrine from the locus coeruleus. The nor epinephrine enters the LA and elsewhere in the brain and produces a glutamate driven protein synthetic cascade that consolidates the memory of the event and sets the stage for subsequent reconsolidation. As mentioned above, resolution of an event is critical to avoid encoding the memory as a traumatization. If action, either flight or fight does not occur, serotonin will not be released and nor epinephrine will encode this as a traumatization. This may help explain the repetition compulsion seen in traumatization. The individual relives the event in the hope of changing the outcome. Unfortunately, if the individual survives without the motoric component, the stimuli cause a release of nor-epinephrine and the pathway self reconsolidates after each activation, insuring its immortality. Without serotonin, every emotionally arousing experience would encode permanently. Serotonin at the time of the event, allows us forget. Since we cannot repeat the event in reality we must rely on our imagination to recreate the event so that we as therapists can provide a different ending. Thus, we need to have an outside force to change the outcome.

Therapy for Amygdala based Traumatic disorders

One therapeutic approach to decrease the response is to modulate outflow from the BLA to the central nucleus. One of the classic methods of treating a conditioned fear is by the process of extinction training whereby a conditioned animal (one repeatedly exposed to a light followed by a foot shock and who subsequently responds with fear to the light) exposed to the light without the foot shock. This causes a decrease and eventually an extinction of the fear response. Extinction does not appear to be simple forgetting. This is important in the traumatization issue because without an intervention of some sort, simple forgetting of a traumatization is not possible. Having said that, I suspect there are people who spontaneously overcome phobias and even more serious traumas, but I argue that it is not simple forgetting. Extinction training involves new learning, but not extinguishing the connection located in the BLA between the stimulus and the UFS. Thus, if extinction **training** is carried out (non-reinforced Cs's are presented) so that the CS no longer produces the fear, spontaneous recovery (recovery of the fear response over time), renewal (recovery of response when the light, the CS, is presented in a novel environment and reinstatement (recovery of the response after presentation of the light under the same conditions where the response was forged) can occur.

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After extinction training is carried out

Spontaneous recovery

Renewal
Reinstatement

can still occur

Recently, LeDoux and others have looked at another way of inhibiting the response produced by a retrieved memory using localized chemical interventions. Instead of a slow decrement in responding due to non-reinforcement, animals who were conditioned to freeze (a fear response) in response to a light (the conditioned stimulus associated with a foot-shock), subsequently and permanently no longer did so when a protein synthesis inhibitor was infused into BLA immediately after activation of conditioned stimulus (the light). It appeared that the fear response did not re-consolidate after activation. This is remarkable and was not predicted by traditional theory. This result suggests that activation of the fear by the conditioned stimulus places the link between the retrieved memory and the response in a protein synthetic dependent state and subject to disruption. To study this process we can use fear conditioning as a model to help us understand the process of inhibiting reconsolidation.

For example, using another fear conditioning model, passive step down avoidance, electrical activation of the dorsal raphe, the site of the origin of the brain serotonin, also permanently abrogated subsequent responding. This is the critical experiment in understanding de-traumatization and will be discussed a little later. In addition, infusion of propranolol, a blocker of nor epinephrine, into the lateral nucleus of the amygdala also extinguished the response to the conditioned stimulus after activation. Recent research on

PTSD has confirmed these findings in humans given propranolol after imaginal activation of the traumatizing event.

The effects of traumatization are a consequence of amygdala activation. Outflow from the Ce produce emotional, cognitive and physical effects. Therapy therefore must consist of either decreasing the outflow of the BLA to the Ce or by directly inhibiting the re-consolidation of the stimulus to the UFS located in the BLA.

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Regardless of the therapeutic approach, activation of the link between the traumatic memory and the UFS is the first critical step as this initiates glutamate dependent protein synthesis. Affect activation by retrieval of a traumatic memory is therefore a marker for protein synthesis and therapies given at that time can be used to learn new responses or directly inhibit reconsolidation.

These results suggest four critical aspects of a fear memory. Firstly, the reconstitution of the linkage between the retrieved memories with the UFS is the key process in the initiation of reconsolidation. Secondly that reconsolidation involves nor epinephrine and glutamate as well as protein synthesis. Thirdly, there is a cortical mechanism that can inhibit Ce outflow and finally this hitherto for immutable link can be broken.

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Amygdala in Retrieval of Traumatic Memory

Retrieved traumatic memory* → *UFS*-----> *Emotion
BLA* *Ce
Amygdala

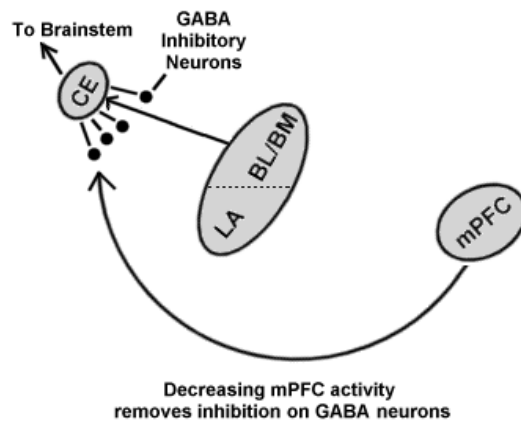
The amygdala receives many afferent connections that can modulate functioning and the prefrontal cortex is one of the most important. The prefrontal cortex is part of our decision making centers.

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It can inhibit BLA output from activating the Ce. As in extinction training repeated exposure to the traumatizing object or memory has been shown to alter the effect of the trauma. It appears that this new learning activates intercalated neurons in the amygdala which block the outflow from the BLA to the Ce leaving the pathway in the BLA intact. This then explains the why the response can return. For humans, the technique of desensitization may work the same way. This approach works for phobias. Desensitization, in safe surroundings, is used in a technique called Traumatic Incident Reduction used for PTSD. Another similar exposure technique is V/K dissociation. In each case the emotions associated with the event are activated and new responses to the memory are learned.

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Medial Prefrontal Cortex in Extinction



However, using the prefrontal cortex as in ‘talk therapy’ requires that the problem be brought to conscious awareness. This may not be possible if the individual has dissociated the traumatic memory.

In the treatment of chronic pain, Sarno avoids the activation of the primary event and tries to diffuse the fear that the pain is sinister in nature. He focuses on the pain and tries to re-frame the fear it generates. If the pain cannot be resolved or it returns to the same place or another part of the body, he recommends psychotherapy to uncover and remove the initiating anger that was co-encoded. It is this dissociated anger that must be brought conscious awareness. Since the cause of the anger is often dissociated, then the discovering the connection between the anger and the event becomes the object of the therapy.

In treating pain Scaer and Levine propose that traumatization is due to an incomplete fear response, encoding a trauma as a procedural memory. If we can find a way to complete it, that is, change the outcome, there will be a de-linking of the connection between the traumatizing event and the pain. Levine's method involves engaging in process whereby an individual experiences 'a felt sense'. Levine calls this somatic experiencing. Thus, the somatosensory part of the traumatic memory is used to activate re-consolidation. The individual can then be imaginably guided to a safe place at which time the brain releases serotonin.

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Summary of Traumatization Therapy

Somatic experiencing works here

Retrieved Traumatic Memory → // **Fight of Flight**

EFT and EMDR work here

Cognitive therapy and psychotherapy work here

→ **Emotional response**

It seems that for each therapy so far described, one needs to track down the event that was traumatically encoded. This is also true for tapping. It has been proposed that tapping or any soothing sensory stimulation that can raise serotonin can effectively inhibit re-consolidation of an activated emotional state. How does this occur?

Retrieval of a traumatic memory releases glutamate in the locus coeruleus

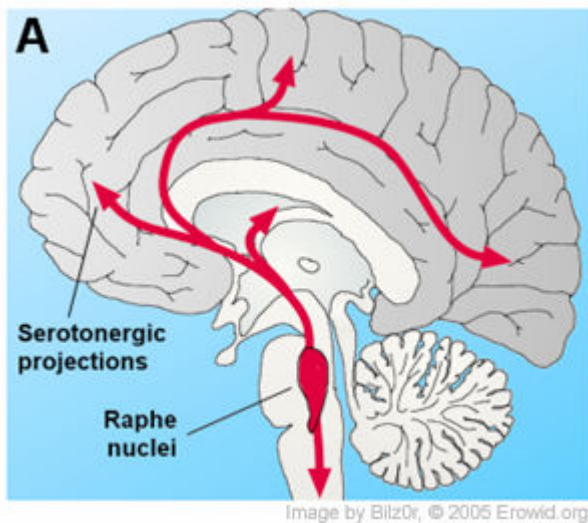
Slide 24 **Glutamate activation**

Glutamate → Specific Pathway in BLA → Protein Synthesis

Glutamate → Locus Coeruleus → Nor-epi Release into Lateral Nucleus

that subsequently releases nor epinephrine into the lateral nucleus and begins the reconsolidation of the specific pathway in the BLA between the memory the UFS. The activation of the UFS leads to a stress response mediated by the limbic system. Without intervention re-consolidation occurs. However, under appropriate sensory stimulation (tapping massage, eye movement acupressure and others) a generalized acute release of serotonin via ascending pathways from an area called the raphe nuclei occurs. This release is non-specific and global and it unrelated to the context or content of the memory.

Slide 25 Ascending serotonin projections. Serotonin is a modulator of information processing. It is generally considered a neuromodulator and such has stores of serotonin that are released continually. When activated via tapping the dorsal raphe releases a large amount of serotonin globally. It's effect operates through the antagonist of glutamate, GABA.



Memory retrieval releases glutamate in the prefrontal cortex, the hippocampus, the locus coeruleus and the BLA. While it has been shown

that a propranolol can inhibit re-consolidation of an activated traumatic memory, tapping does not cause release of such substances. One mechanism by which change can be effected is that serotonin is released by tapping. Through serotonin receptors on GABA inhibitory neurons, nor epinephrine release from the locus coeruleus and glutamate driven protein synthesis in the BLA occurs. The key here is that glutamate activation of protein synthesis is event specific.

If one can eliminate the relationship between the event and the UFS an emotional response can not be produced and the event no longer has power. In principle, de-linking a phobia, removing the emotion to a traumatic event or eliminating a painful condition that was encoded with a trauma uses the common mechanism of inhibiting the completion of glutamate activated protein synthesis. Indeed, any traumatic memory or its encoded procedural component can use tapping because it interferes at the common boundary of traumatic memory and the final common pathway leading to a chronic stress response, be it emotional, cognitive or somatosensory. In summary

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Neurobiology of Tapping

Activation of traumatic memory→ Increase of Glutamate and Nor-epi→

Sensory Stimulation (Tapping, EMDR)→

Increased Serotonin→

Inhibition of Reconsolidation

TAPPING

Based on the mechanism of its encoding, we have suggested that an activated traumatic memory (be it physical, cognitive or emotional) can be disrupted by tapping. This model provides an outline that addresses the permanence, specificity, the need and skill to uncover dissociated memories and their links, the temporal relationship between activation of the affect and a successful treatment. Thus, the specificity and permanence is due to the mechanism that produces a glutamate, event specific protein synthetic reconsolidation. Nor-epinephrine initiates this process leading to new learning and a re-consolidation of the linkage between the retrieved memory and the UFS. In addition, decreased outflow from the Ce as modulated via the prefrontal cortex, allows for a decrement in distress during treatment. This model does not require specific areas to tap. Indeed, one could consider more soothing ways of increasing serotonin, such as stroking the head, rubbing the temples and so on.

Finally, there are a number of disorders that are modulated by the amygdala but are not linked by trauma directly. They may however be a manifestation of the maladaptive response to the trauma. These include OCD, addictive cravings, generalized anxiety, and depression.

As dissociated traumatic memories have the home in the subconscious, other modalities that do not necessarily require activation such as hypnosis and biofeedback, massage acupuncture may be complimentary to tapping. Regardless of the technique used, tapping de-links the memory and the unresolved initially encoded state. Resolution by motoric action as in somatic experiencing, and tapping and Eye movement desensitization response extinguishes the ghosts by inhibiting re-consolidation. It stops the need to re-enact, to get it right or to end it. These approaches exorcize the immortalized ghosts. Tapping on the appropriate retrieved memory extinguishes it for good.

Thus, we have a simple neurobiological explanation of many of the observed responses to tapping after affect activation.

There are several interesting other aspects. For example, extinction by classical non-reinforcement is different from using protein synthesis inhibitors thus suggesting the two pathways: one from the prefrontal and one directly in the BLA. The other aspect is that the amygdala modulates memory activation all over the brain. It may be that instead of just having the serotonin work locally in the amygdala, it works globally throughout the brain where the other aspects of the activated memory are undergoing glutamate induced protein synthesis and can be de-linked. It may be that for emotionally tagged items the memory begins with thought, then amygdala activation which sets the emotional tone throughout the body.