

# **A Model for Disrupting an Encoded Traumatic Memory**

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*Post Traumatic Stress Disorder (PTSD) is a chronic and sometimes progressive illness. It has been hypothesized that PTSD is encoded in such a way that retrieval of a traumatic memory not only causes the individual to experience fear, but also reconsolidates the linkage between the memory and the fear response, thus preventing desensitization. Recent work on conditioned fear however, has shown that reactivation of these consolidated memories returns them to a protein synthetic dependent state that makes the linkage subject to disruption.*

*This paper describes a theoretical model for the surprising effectiveness of a therapy for PTSD. It is proposed that, after activation of the fear response, tapping on certain areas of the body*

*increases serotonin release. This increase in serotonin appears to disrupt the linkage between the thought and the emotional response. Using this approach, other disorders such as phobias, certain types of chronic pain and other pathologically encoded negative emotive states may also be curable.*

KEY WORDS: Amygdala, Glutamate, PTSD, Tapping, GABA, Serotonin, Nor-epinephrine, Locus Coeruleus

## **INTRODUCTION**

Since not everyone who experiences a trauma develops PTSD, one needs to postulate that the event have meaning for the individual and that a specific biological landscape of the brain at the time of the trauma is necessary. For the individual, this permissive biological landscape is a consequence of the transactions between genetics and how life's events are experienced (*Garpenstand & Annas & Ekholm & Orelan & Fredrickson 2001*)

(*Rau & Decole & Faneslow 2005*). Although the brain needs to alert us when a fearful situation occurs, it is characteristic that, for individuals with untreated PTSD, the fear response does not diminish over time. Consequently, ongoing activation of the fear response by conscious/subconscious stimuli associated with the event leads to chronic distress.

Over the last several years, individuals presented to our practice with a variety of psychological problems, including PTSD. Some were initially treated using a technique (see below) that involves tapping on areas of the skin (*Callahan 1985, 2000, 2001, Craig*). For those treated, the simplicity and ease of administration of this approach did not interfere with further evaluation and treatment. This therapy, developed by Roger Callahan and others, calls for bringing the event to conscious awareness thereby re-activating distress. Subsequent application of non-specific sensory input, literally, gentle tapping on the patient's face, body and hands, produces a remarkable reduction in distress. Our hypothesis is that

after activation by a retrieved trauma, tapping disrupts the amygdala pathway that directs the fear response, thus preventing subsequent responsiveness. The memory of the trauma loses its power to evoke distress.

From an observational point of view, when tapping is applied, it appears that a dimmer switch is being thrown. After a successful treatment, as measured by a decreasing SUD (Subjective Units of Distress is a scale from 0-10, as reported by patient, where 0 is none and 10 is extreme distress (*Wolpe 1958*)), thoughts that had been clear were less so. Not only was the ability to generate these images diminished, the response to the thought was gone, often for good. Experience with this technique has been quite remarkable and, if the hypothesis is correct, a new theoretical model can be explored for the treatment of PTSD.

## Post-Traumatic Stress Disorder

PTSD produces symptoms that are reflective of a chronically activated limbic system. We propose, that after abnormal encoding --for which we have only slight clues about the neurobiological landscape necessary (*Yehuda 2004*)-- an amygdala-based pathway that is critical for sustaining PTSD, is produced. A retrieved memory (or associations) of the traumatic event becomes the conditioned stimulus (**CS**). Retrieval of the traumatic event activates neurons located in the lateral nucleus (**LA**) of the amygdala. An event specific pathway then leads to activation of neurons in the basolateral nucleus (**BLA**). The **BLA** neurons then forward this information to the central nucleus (**Ce**) of the amygdala and fear is produced. (Fig 1).

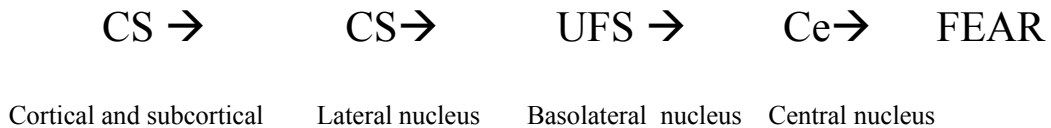


Fig. 1

Proposed Pathway for Generation of Fear

The most powerful threat to survival is that of being killed. The retrieved memory of that event then becomes the conditioned stimulus (**CS**). All sensory input at the time of the event becomes part of this encoding. Only a small sensory portion of the event is needed to activate this pathway. This threat can be perceived either on the conscious or subconscious level. An event specific pathway then activates an area in the **BLA** that contains hard-wired survival pattern recognition areas representing unconditioned fear stimuli (**UFS**). These stimuli, such as heights, dark places, open spaces, loud noises, fear of being killed are survival based. In the final irreversible step, the activated **UFS** sends a signal to the central nucleus of the amygdala and a fear response is produced.

This paper is concerned with how this pathological encoding is sustained in PTSD and offers a model for disrupting the amygdala pathway from the **CS** to the **UFS** thus, providing for a cure.

One possibility for the maintenance of PTSD lies in abnormal glutamate functioning in the basolateral nucleus of the amygdala

(BLA) (*Liang & Hu & Chang 1996*) (*Reidel & Platt & Micheau 2003*). Once encoded in this way, glutamate, along with other neurotransmitters, such as nor-epinephrine (NE), are released into the amygdala during activation and, in PTSD, initiate a protein synthetic process. It appears the glutamate and nor-epinephrine activate a “reloading” of the pathway and hence, immortalize it. Recent research suggests that this process can be interrupted. For example, using an animal model for conditioned fear, (*Duvarci & Nader 2004*) (*Nader & Schafe & LeDoux 2000*) infusion of a protein synthesis inhibitor (anisomycin) into the BLA of a rat after fear activation, permanently abrogates the fear response. Under these conditions the amygdala-based link between the CS and the fear response is not reconsolidated

### **Treatment of PTSD**

Here, we use a method of therapy (*Callahan 2001*) to inhibit glutamate/nor epinephrine function. As mentioned above, this involves thought activation of the event followed by sensory

stimulation, and is outlined below. It normally takes about 2-3 minutes to perform this therapy once the response has been activated by thought.

### **DISRUPTING CS → UFS → FEAR IN THE AMYGDALA**

The traumatic event is brought to consciousness and a distress response as measured by an SUD is obtained.

*The individual then*

*Taps over the eye 5x*

*Taps under the eye 5x*

*Taps 5x under axilla*

*Taps 5x under the sternal notch*

*A Gamut procedure follows, the individual taps behind the fifth knuckle on the back of the hand and*

*Closes eyes*

*Opens eyes*

*Looks down to the left then the right*

*Makes big circle with the eyes both ways*

*Hums a tune*

*Counts backwards from 5*

*Hums a tune*

The individual then repeats the tapping and Gamut sequence and

an SUD is taken. The sequence is repeated until the SUD goes to zero or cannot be lowered further. The technique seems peculiar. It remains unclear as to what part of the process is therapeutic though research suggests that the tapping is the critical sensory input. Either client or practitioner can perform the tapping. There is no data to support either as more effective. The therapy can be used on both simple and complex traumas, as well as phobias and other negative emotive states though the time frame for treatment will vary. (*Andrade & Feinstein 2003*)

## **DISCUSSION OF THEORETICAL MODEL**

As mentioned above, after activation of a conditioned fear by a stimulus that produces fear, a protein synthetic process that involves the amygdala is necessary for reconsolidation of the **CS→UFS** linkage and continued expression. Our hypothesis is that for PTSD, tapping after reactivating a traumatic event also disrupts this pathway.

We propose that tapping de-links the **CS→UFS** pathway

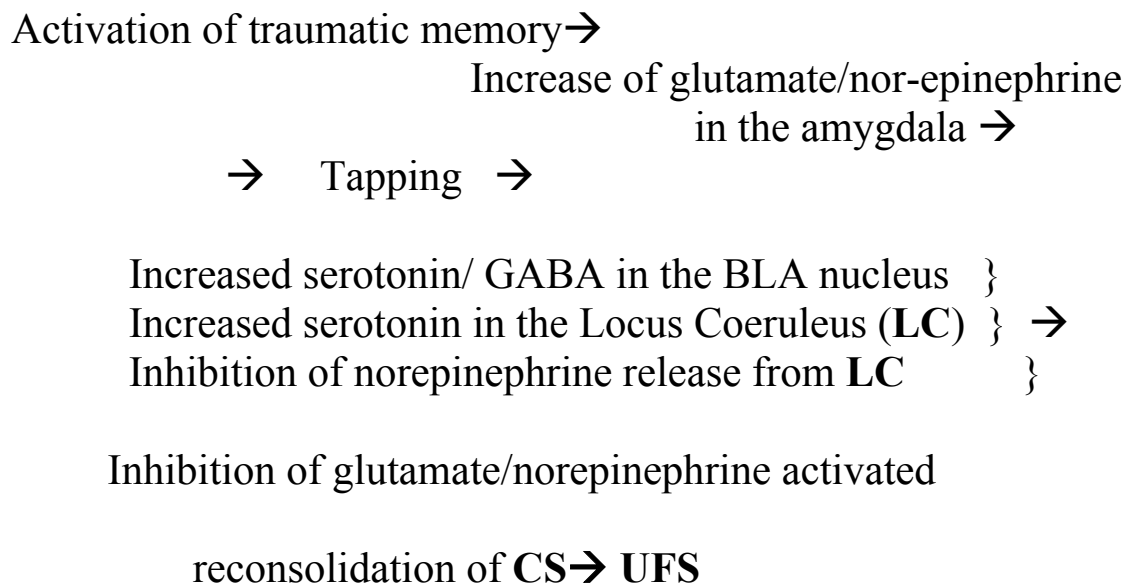
via the involvement of neurotransmitters. Consistent with this hypothesis, a metabotropic glutamate antagonist, AP5 (*Jerusalinsky & Ferreira & Walz & Da Silva & Bianchin & Ruschel & Zanatta & Medina & Izquierdo 1992*) appears to be able to produce amnesia for step down passive avoidance learning, a fear paradigm, when infused into the amygdala and hippocampus at 0 minutes after training. Later infusions at 90 and 180 minutes post training have no effect. These results suggest that AP5 inhibits an initiating process that is crucial to post-training memory reconsolidation. Other glutamate antagonists when infused at later times do produce an amnesic affect, suggesting interference in other reconsolidation processes. In addition, propranolol is known to cause a deficit in fear retention after a fear memory is retrieved (*Debiec & LeDoux 2004*). We suggest that de-linking the **CS**→**UFS** pathway also requires inhibiting beta-adrenergic activity in the amygdala.

Disruption of the amygdala-based CS→UFS link by activation of the serotonergic system appears to be the common factor involved. Stutzman (*Stutzman & LeDoux 1999*) has shown that inhibition of glutamate activity in the lateral nucleus of the amygdala can be produced by serotonergic activation of GABAergic neurons. These neurochemicals have been shown to prevent learning and re-learning of fear responses as well as the response to fear stimuli. Increased serotonin also acts on glutamate receptors in the locus coeruleus (*Aston-Jones & Akaoka & Charney & Chouvet 1991*) to inhibit release of noradrenaline. It is interesting that this inhibition occurs only when the animal is in an activated state, that is when there are increased glutamate levels in the locus coeruleus.

These data provide for a parsimonious analysis as serotonin acts both in the amygdala and the locus coeruleus on glutamate activated systems. This inhibition of reconsolidation does not interfere with retrieval of the memory, just its role as a

precursor to activation of the **CS→UFS** pathway. The specificity derives from activating a unique **CS→UFS** pathway. As all associated stimuli converge on the **LA**, once the **CS→UFS** connection is de-linked, no further effects from the traumatic memory should be seen. (Fig. 2)

### **INHIBITING RECONSOLIDATION**



**Fig. 2**

Tapping increases serotonin and disrupts reconsolidation by inhibiting  
the protein synthetic process

The decrement in SUD during tapping appears to arise from decreased outflow for the central nucleus due to increased serotonergic activity causing a decrease in nor-epinephrine release from the locus coeruleus. Its permanence reflects that the linkage between the **CS** and the **UFS** once broken cannot be restored unless the exact neurobiological moment of trauma encoding is recreated. The end result is emotional amnesia for the event, similar to the effect seen with anisomycin where we speculate that the **CS→UFS** linkage is broken by inhibiting protein synthesis. (*Durvaci 2004*).

Finally we must consider the transduction event that causes the rise in serotonin. We can only speculate that the tapping involves serotonergic pathways. Much research has been done on acupuncture (*Wenhe & Yucun 1981*), massage (*Field & Hernandez-Reif & Diego & Schanberg & Kuhn 2005*) and light, in this regard (*Magnusson & Boivan 2003*). The effects of these techniques for relaxation, pain control and mood suggest activation of the serotonergic raphe nuclei. In the laboratory, stimulation of the

raphe nuclei ablated the learned fear response in a step down passive avoidance paradigm (*Fibeger & Lepiane & Phillips 1978*).

### **Summary :**

Thought activation of the abnormally encoded amygdala based **CS→UFS** linkage increases glutamate and nor-epinephrine in the amygdala. The increase of these neurochemicals sets the stage for the protein synthetic reconsolidation of the **CS→UFS** linkage.

Tapping, which would never occur under natural conditions of survival, takes advantage of the moment when this connection is susceptible to disruption. We speculate that tapping or other sensory input causes a rise in serotonin, releases GABA in the amygdala and also interferes with glutamate activation of the locus coeruleus and release of nor-adrenaline and subsequent protein

synthesis. This therapy does not concern itself with the neurobiological landscape that existed during encoding, nor does it require the therapist to evaluate content. The key is to activate the pathway that produces the fear response in the amygdala making it labile and subject to disruption. This model allows us to test these ideas. Infusion of serotonin releasing agents (not reuptake inhibitors) should produce results similar to tapping.

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