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Use of Neurobiological Modeling to Establish the Structure of the Psychotherapy Situation

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Abstract

The resolution of attachment deficits in psychotherapy may require modification of the psychotherapeutic context. The neurobiological basis of attachment requires a specific brain-to-brain or unconscious-to-unconscious "listening" process. To remedy attachment failure – caused by a neurobiological deficit related to the failure of unconscious communication – therapy needs to be restructured to facilitate communication between the therapist's unconscious and that of the patient. When successful, the process can be likened to the alteration of a radio signal transmitter of a radio signal – the patient's unconscious – so that the receiver – the therapist's unconscious – can receive it successfully. In this way, the attachment established between therapist and patient can reverse the original attachment failure and resolve the psychopathology. This article illustrates this process, based on neurobiological deficits in post-traumatic stress disorder (PTSD), where the wiring of the brain fails because of an overgrowth of the amygdala-based fear mastery center, and an underdevelopment of the hippocampus-based neuronal plasticity center. Usually, these centers are balanced so that individuals can evaluate the masterability of their fear; patients with PTSD, however, have an overwhelming fear response to all circumstances, regardless of actual masterability. To counteract this phenomenon, patients are treated by two therapists: One to whom they present a masterable fear, the "hippocampal therapist," and another to whom they present an unmasterable fear, the "amygdala therapist." In this way they learn to master manageable fears without interference by overwhelming fears.

Keywords: ***Separation bonding; Brain-to-brain communication; Hippocampal; and Amygdala therapists.***

1. Introduction

While the two fundamental post-traumatic stress disorder (PTSD) treatment modalities, psychotherapy, and psychopharmacotherapy, have demonstrated significant efficacy, there are marked limitations to their success, and neither have been shown to approach the cause of the illness they are treating. This may limit their long-term efficacy, especially post treatment, and degree of effectiveness, even in the short term. A metanalysis [1] of the long-term efficacy

of psychotherapy for PTSD, spanning all research-controlled trials with a documented effect size, found that all trials demonstrated efficacy, including eye movement desensitization and reprocessing, exposure-based therapy, cognitive behavior therapy, supportive counseling, and psychopharmacotherapy. The mean effect size across treatments was 1.88 for pretreatment to follow-up, and 17 for post treatment to follow-up. Steenkamp et al. [2] estimated that approximately 60% to 72% of military patients retained PTSD diagnosis after treatment, while Watkins et al. [3] showed that, although current PTSD treatments have significant efficacy, more research is needed to improve clinical outcomes in populations with limited efficacy. These findings suggest that, although established treatments have significant efficacy, it is not sustained or improved upon by patients after the conclusion of treatment. This is likely because these treatments are not aimed at addressing the underlying basis of the disorder: if they were, they would begin a process of change and growth that would last longer beyond the end of the treatment.

The efficacy of current psychotherapy and psychopharmacotherapy approaches have not been established for more deeply traumatized patients. Current evidence suggests that they do not produce long-lasting, profound changes resulting in a resolution of the condition, aiming instead for symptomatic improvement. Hence, there is a need for treatment that not only alleviates the symptoms but also reverse the underlying deficit. Such treatment will lead to a more profound and longer lasting improvement that can produce intrapsychic structural changes in how the individual deals with stress, compared to current symptom management-based treatments. Further, a more in-depth treatment model can restart an arrested developmental process to facilitate progressive gains long after the treatment ends and could be successfully applied to patients who are not able to benefit from current treatments. This will allow access to effective treatment to a wider range of patients and reduce the high drop-out rate among PTSD patients. [4] Even though this type of therapy may require a longer time and be more expensive, actual savings related to regained productivity and quality of life may more than compensate for this. The attachment model provides a useful framework for addressing the neurobiological roots of PTSD and possibly facilitating a more in-depth resolution of the condition. This paper aims to demonstrate such a model, explain its underlying mechanisms, and demonstrate its utility with a case presentation.

2. Clinical Impact Statement

There are limitations to the efficacy of current behavior-based psychotherapy treatments for PTSD, possibly as they do not address the underlying neurobiological deficits perpetuating the problem. This can be resolved by utilizing an attachment-based therapy aimed at resolving the underlying neurobiological deficits likely causing the disorder. By conceptualizing neurobiological deficits as related to an overgrowth of the amygdala-based fear center blocking the adjacent hippocampus-based neuronal plasticity center, we can determine a treatment structure to reverse the deficit. By dividing the treatment between two therapists – one the fear-based amygdala therapist, and the other the growth-promoting hippocampal therapist – and instructing the patient to only focus their fear on the first, this deficit can be resolved. Thus, the patient brings fear-based material only to the amygdala therapist and discusses growth-promoting material only with the hippocampal therapist. In this manner, the patient whose all-consuming PTSD symptoms prevents an attachment with any one therapist, can form an attachment with the whole treatment team. To demonstrate this process, I discuss a case where the model was used successfully after behavioral and psychopharmacotherapy had failed.

3. Theoretical Framework

Bowlby [5] established the concept of attachment to address what he considered a deeper aspect of psychopathology than what was currently being addressed by psychodynamic-based therapies of his day. While his colleagues were addressing the problems, patients had in coping with their environment, Bowlby [6] was focused on the deeper, biologically-based attachments he believed would serve as a potential secure base from which later capacities to adapt would evolve. He posited that the individual's instinctual development is fostered by the mother during this attachment period. Through her attachment with her infant, the mother facilitates the emergence of those instincts and, when she does not, there can be a failure of their development.

Following this pioneering viewpoint, neurobiologists have pointed out a number of features of attachment, in which the mother's unconscious communication abilities with her infant foster the development of instinctual gains. Feldman, [7] for example, developed the concept of biobehavioral synchrony to describe a mode of communication between mother and infant and highlighted a specific form of communication forming the basis of the bond between them, which, in turn, fosters the infant's motivation. When this intuitive synchrony is not present, the motivation also lacks. Lenzi et al. [8] provided evidence for a similar attachment-based system, predicated on an unconscious-to-unconscious mode of communication of fear between mother and infant. This form of attachment enables infants to form a bond with their mothers that supports their ability to master fear. While an unconscious communication mode between mother and infant, termed biobehavioral synchrony, [7] is responsible for the attachment necessary to master pleasurable experiences emanating primarily from the nucleus accumbens brain region, the mastery of fear is derived from the adjacent amygdala region. Each of these systems is based on a different level of the brain-to-brain connectivity between infant and mother. These findings suggest that the therapist could employ unconscious communication between patient and therapist to stimulate these forms of attachment when they were failing in other conditions. For example, in patients who had not had an effective unconscious-to-unconscious relationship with their mother in which their fear was addressed, such a relationship could be reconstructed in psychotherapy, reversing a potential attachment failure.

Although therapists are currently not focused on communication with their patients on this unconscious-to-unconscious basis, it is conceivable that they can do so. The advantages of doing this, as Uhlhaas et al. [9] have demonstrated, consist in stimulating the maturation and restructuring of the patient's neuronal networks and releasing instincts that have laid dormant for a lifetime. If indeed the failure of maturation of the neuronal network of a patient was the reason for the deficit, this form of communication in psychotherapy might remediate it. This paper discusses how such a neuronal deficit might occur in one type of condition, PTSD, and how to develop the unconscious-to-unconscious form of communication needed in psychotherapy to remediate it.

I will provide evidence that doing so results in a resumption of neuronal plasticity and simultaneously in a much more complete resolution of the patient's former symptoms compared to previous psychotherapy processes. What distinguishes this form of therapy for PTSD from those that preceded it is that the patient, through this attachment-based therapy, can learn to master fear, which may be a basic vulnerability that generated the PTSD in the first place, providing a more pervasive solution to their problem.

The ability to master fear-based situations using this model can extend far beyond the therapy, as patients can use their newly developed neuronal plasticity based on what they learned in the therapy sessions. When successful, this type of treatment will enable patients to experience what Bowlby [5] termed an "instinctual releaser," to successfully master fear-generating situations that they had never encountered before. That is because they have established a neurobiological solution for a problem and thus become motivated to master progressively more types of fear-based situations.

Although Bowlby considered the bond between mother and infant to be uniform, recent neurobiological research supports multiple forms of attachment, each emanating from a different brain node. These nodes are arranged in a hierarchical manner. Feldman [10] outlined this hierarchy. One of those nodes, which she felt was part of the "reward" network, was that of the amygdala/hippocampus region, which is associated with the management of fear. This brain node is activated when young children are separated from their mother. Moriceau and Sullivan [11] demonstrated the activation of this brain node in the rat pup when it is ready to leave the nest. During the first 12–15 days post-partum, the rat pup's amygdala is not activated, fear is not experienced, and the rat pup forms a basic bond with its mother. It is contact with the mother – her odor, and her presence in the nest – that constitutes the necessary mode of communication during this initial form of attachment. [12].

During the second phase, as the rat pup begins to move out of the nest to explore its world, the amygdala or fear center becomes activated as it is neurobiologically needed to enable the pup to master danger in its explorations. Moriceau and Sullivan [11] and Moriceau et al. [13] provide evidence that, during this second developmental phase in the rat pup, the mother's role changes as she enables her pup, through her presence to help it master fear. She does so by providing the pup with an experience of safety by protecting it when it encounters situations of unmanageable danger and allowing it to master ones where the danger level is lower. The result of this type of bond is manifested in the reduction of the pup's corticosterone level, generating a reduction in the amygdala-based activity level. This process is a purely instinctual one. The mother rat responds to her pup's level of fear providing it with an experience of relative safety in the context of danger. Not only does the rat pup experience the emergence of fear in the face of a predator's odor, but the amygdala, which has been dormant before that period, becomes activated as does the emergence of corticosterone to fight potential bodily danger. [11], [13].

These researchers also showed that this second fear-based system is an attachment system because, just as the primary attachment system that preceded it, it is a proximity-seeking system, in which the pup seeks direct contact with the mother. The point these researchers have demonstrated is that the mother rat is operating by instinct: sensing her pup's level of biological danger and reducing it with her presence at just the right times. Furthermore, both periods are guided by "sensitive periods" during the initial phase, which lasts 10 days, and the second phase, which immediately follows it. The mother's presence during the critical second fear-based period was demonstrated to reduce fear in the presence of a predator's odor. During the initial period, that same odor did not induce fear and the mother's presence did not result in any response. In addition, Rickenbacher et al. [14] showed that the hormone oxytocin, which was critical in promoting basic rat pup attachment emanating from the hippocampus, is also generated by the amygdala during the second phase to support mother-rat–pup attachment. Vrticka et al. [15] provided evidence for this same attachment-based fear

process occurring in humans. Kirsch et al. [16] showed an intranasal oxytocin modulated fear response in humans obtained by attenuating the activity of the amygdala.

Mahler et al. [17] introduced the concept of separation and individuation into the mental health community, demonstrating several phases in the process. She proposed several phases of the process, beginning with the hatching phase, which she demonstrated in normal children be at approximately 5–9 months, in which children seek to explore the world away from their mother. During the second phase, which she showed as occurring from 9–14 months, children have more prolonged periods in which they experiment exploring the world without the mother's presence. During the following phase, which she termed the rapprochement phase and lasts from 14–24 months, children develop a fear of abandonment by the mother, thus they check-in with her regularly as independence proceeds. In the last of her phases, which occurs by age 24 months, children develop the concept of object constancy: they believe their mother can be present when they explore their world without having to check whether this is true.

These observations are consistent with an attachment-based neurobiological substructure of the type just described. Furthermore, a separation bonding failure could occur at the neurobiological attachment-based level, explaining failures of attachment as they might be manifested in a failure of the separation-individuation process. If the mother has a failure of separation bonding at a neurobiological level, she has not facilitated the instinctual-based, unconscious fear-based communication, and the child might be too fearful to successfully navigate the separation-individuation processes, or, in more instances, may appear to navigate them but be vulnerable to stresses later in life, because of a partially unsuccessful separation bonding experience.

Separation bonding is the neurobiological process by which the mother shifts unconscious communication with her infant from facilitating safety to facilitating mastery of fear. She becomes unconsciously attuned to the level of her infant's fear and provides protection when the fear level becomes too great. Addressing attachment process failure is very suitable for psychotherapy treatment at the attachment level, as it can repair the underlying attachment deficits and resolve later failures due to excessive stress later in life.

Current treatment of PTSD focuses strictly on the current situation's stress, without considering the underlying attachment issue that made the individual vulnerable in the first place. Although more recent treatment processes begin to address the underlying deficit that makes a patient vulnerable to PTSD, this is the first study which focuses on the underlying neurobiological problem that results from the failure of attachment and uses the treatment to resolve it. By focusing on this underlying process, the resolution of the disorder may be more pervasive and longer lasting and extend development long beyond the duration of the treatment. I hope to demonstrate this process in the case presentation to follow.

3.1 Failure of separation bonding as a precursor for ptsd vulnerability

Cloitre et al., [18] in a study of women with PTSD related to childhood abuse, examined a randomly selected group to address interpersonal skills deficits likely emanating from the period prior to the onset of their current trauma. In one group, the women worked on skills training to reprogram their ability to cope with stress, after which they received

trauma-based treatment. They were compared to a group receiving trauma treatment only. The group with both components had the longest lasting remission, suggesting that early deficits from trauma or neglect had a marked effect on the PTSD symptoms. These patients did better when the impact of prior trauma on their adaptive capacity was treated. Iwaniec et al. [19] identified early childhood factors which may predispose children to later trauma-related illnesses. Among the factors identified were working models of the self, behavior and coping strategies, and availability of supportive relationships.

Owen [20], [21] comprehensively discussed the impact of early attachment disorders as a significant contributor to the genesis of PTSD disorders. There is evidence that vulnerability to PTSD symptoms may emanate from early attachment deficits. Stovall-McClough and Cloitre [22] interviewed adult female childhood abuse survivors and assessed the extent of early attachment deficits, dissociative symptoms, and PTSD genesis later in life. They found a 7.5-fold increase in the likelihood of being diagnosed with PTSD in individuals who had earlier attachment deficit and a history of early childhood abuse. O'Connor and Elklit [23] found a direct association between pathological attachment styles and PTSD symptoms in 328 Danish young adults. Barazzone et al. [24] conducted a metanalysis of studies on the relationship between attachment and the effects of trauma on the genesis of PTSD found an important link between attachment failures and the genesis of PTSD in those who had been traumatized. Although failure in early attachment has been identified as a vulnerability to the later development of PTSD, no specific attachment factor has been identified which can be remedied by psychotherapy to reduce the vulnerability to the disorder.

At the brain wave level, neurobiologists have demonstrated that the mastery of fear involves the process of contextualizing fear-based experiences. Stujenske et al. [25] and Lisman and Jensen [2] demonstrated that, when slow theta waves (4–12 Hz), associated with electrical activity of safety emanating from the hippocampus, were emitted, organized faster gamma waves were emitted predominantly from the amygdala and represented potentially fear-inducing perceptions; this resulted in contextualized fear. When the gamma waves were nested in the theta waves, safety was also experienced. Conversely, when the gamma waves were not nested, only fear was experienced. We can consider this contextualized fear as fear that can be understood and mastered. The neurobiological hypothesis linking inadequate contextualized fear to the genesis of PTSD fits with the neurobiology of the condition, [27] namely that the fear center, the amygdala, has an overgrowth due to massive uncontextualized fear, while the hippocampus, the center responsible for contextualized fear, shrinks.

This neurobiological hypothesis supports the concept of a failure of separation bonding leading to the disorder. The mother's presence in helping her children master fear generating exploration of their environment leads to contextualized fear or masterable fear. The mother, through her unconscious awareness of the fear level experienced by her infants helps them to distinguish fear that is masterable from that which is not. Failure of this attachment process leads to the opposite: inability to contextualize fear and vulnerability to PTSD when faced with later life stress. Research has shown that this process operates primarily at a neurobiological level, not at a psychological level. The patient with an attachment failure has an excessive amygdala-based response to any potential fear-generating perception. Since the problem emanates at a neurobiological level, it cannot be reversed by a primarily psychological approach, because the

neurobiological equipment which may reverse it is not able to use that psychological approach. Successful bonds between mothers and children are unconscious, not conscious.

Mothers unconsciously sense the fear level of her children and respond accordingly, to help the children recognize their own fear level and to determine when it is or when it is not masterable, facilitating their safe exploration. The mother who responds not to the child's level of fear but to her own, based on her conscious expectations of what the child's level of fear should be in a particular situation, is not attached at this level and her response leads to failure. That is because she is not helping her children learn how to master their own fear level but their mother's. That is true whether the mother is overly frightened or not frightened enough. We can see how this hypothesis fits with Mahler's descriptive stages. At first, during the hatching phase, children have to experience a flood of fear when their amygdala becomes activated as they begin to explore their world. As the process continues, they recognize the invaluable contribution of their mother in contextualizing that fear. At some point, the contextualization process becomes internalized; thus, they do not need their mother's presence anymore to contextualize fear for them. This separation bonding hypothesis, with its underlying neurobiological basis, presents a considerable advantage for its use in psychotherapy. This neurobiological condition may be the substratum responsible for Mahler's successful separation/individuation schema. Because of their underlying neurobiological deficit, it is impossible for PTSD patients to distinguish masterable and unmasterable fear, unless the therapist shows them how to do it. Since it is a neurobiological deficit, providing them with insight will not suffice, because they do not have a frame of reference of safety into which to put contextualizable, masterable fear. They interpret every experience not in terms of its potential masterability, but in terms of how to avoid the danger, which precludes mastery. Furthermore, they develop a defense in the form of dissociation to protect themselves from their fear, which prevents the formation of a fear mastery ability. This defense emanates from the anterior cingulate, which becomes hypertrophied, blocking fear impulses from reaching the prefrontal cortex. [28] For patients with a basic attachment deficit in the process of fear mastery, a neurobiological receptor for mastering fear must be established before the psychological process of mastering fear-inducing situations can be successfully implemented.

This neurobiological structure must be developed in patients who do not have it, by focusing on the underlying neurobiological deficit and creating the attachment which can generate it. The purpose of this type of treatment will be to make them aware of the possibility of contextualizable fear, to develop the neurobiological equipment to accomplish this task, and to help them distinguish between contextualizable and non-contextualizable fear. The only way this can be done is to create two therapists, one of which will help them develop the neurobiological equipment to have the capacity to contextualize fear, while the other therapist helps them avoid danger that is not contextualizable. In the one therapist's office, only contextualizable fear is permitted, so that situations can provide the frame of reference that they lack.

It is the patients' ability to distinguish the functions of both of their therapists that enables them to form the separation bond. In treating PTSD patients who do not have a frame of reference of potential safety, this must be established separately before they can deal successfully with contextualizing fear in that frame of reference. In this treatment process, the patient is instructed to use the "amygdala therapist" for highly fear-promoting experiences, and to use the

"hippocampus therapist" for less fear-promoting experiences. If this is done, the patient forms an immediate attachment to both therapists, because they are providing what the patient's mother could not. The therapists together provide a neurobiologically-informed model of the deficit with their alteration of the structure of the treatment situation. It is akin to setting up a receiver to hear the patients' unconscious brain wave signals with their unconscious and help the patients set up a separate brain category, related to contextualizable fear.

To these PTSD patients, all perceptual experiences have been perceived as dangerous. They now have the opportunity to learn how to distinguish different levels of fear. This shift in the treatment structure supports the development of the plasticity-based neuronal growth to create a new possible structure, in which the possibility of masterable fear is borne and can flourish, without the interference of overwhelming fear intrusions, which would flood that system and make it unworkable.

Such an experience not only resolves the failure of attachment and enables the mastery of new potentially traumatic situations, but also completely resolves the dissociation-related deficits in PTSD. The major caveat is that we are not used to shifting the structure of the psychotherapy situation to resolve patient problems. We use the context of the material within the therapy to accomplish that goal. As Bowlby was developing the attachment theory, Winnicott [29] was establishing the concept of psychotherapy as a "holding environment" in which he viewed the psychotherapist as simulating the mother's caregiving for her infant. By providing the patient with consistent understanding in a framework that supported that understanding, psychotherapy was essentially providing the patient with an analogous situation to the one that failed in the original maternal caretaking. Winnicott was advocating a pronounced shift in the treatment setting, asserting that the structure of that setting, as opposed to the content provided by the therapist, could be used to remediate early environmental failures. In developing his attachment theory, Bowlby had the same aim. Psychotherapy had to transition from just providing insight to providing patients with a form of personal gratification missing from early infancy. Since Bowlby was writing about the same shift in psychotherapy as Winnicott, it is possible that modifying psychotherapy based on the findings of the same discipline (neurobiology) that was stimulated by Bowlby's work may offer us a model for correcting the deficit in the language structure used by patients with PTSD. If we use the structural deficit in these patients' brains as a basis for altering the structure of psychotherapy to correct the language dysfunction, are we not providing the patient with the same type of corrective experience envisioned by Winnicott and Bowlby? In the case to be presented, I discuss that the patient felt he could trust his psychotherapy for the first time immediately after the two-person psychotherapy model was implemented. He developed an immediate attachment to his psychotherapists which persisted. As a result, he was also able to show his extensive real fears to his therapists for the first time. That is because patients with PTSD do not show their real fears because the attachment failure has caused them to not show it to anyone, as no one has responded to it before. Instead, they develop dissociation defenses to protect their prefrontal cortex from the impact of this excessive fear. Once we have a structure of the treatment that the patient can see as facilitating the resolution of the excessive fear, the dissociations resolve. We cannot use insight, the fundamental therapeutic instrument for psychotherapy, because the patient's dissociation prevents it. It has been shown that, in PTSD, there is a predominance of non-contextualized fear, which correlates with overactivation of the amygdala and/or under-activation of the hippocampus/prefrontal lobe; we have seen both conditions in PTSD patients.

Dissociation uses the prefrontal cortex and anterior cingulate for the wrong purpose. Instead of the amygdala providing a warning signal to the hippocampus prefrontal cortex, to stimulate an adaptation process, dissociation stops that process by shutting off the fear system as the anterior cingulate stops access to the prefrontal lobe. Without access, there is little capacity for insight; lack of capacity for insight strengthens uncontextualized fear, which eventually breaks through the dissociation barrier, resulting in periods of rage, panic, and depression.

To date, the use of neurobiological modeling in psychotherapy to resolve this problem has been minimal. The fundamental basis of psychotherapy—that insight, a psychological construct, is the driver of psychotherapeutic rather than neurobiological change—remains unchanged. Current theory holds that bringing unconsciously established sequences into consciousness allows patients to use insight to adopt more adaptive and satisfying behaviors, thoughts, and feelings. Yet with the presence of dissociation there is a chance that we may never be able to bring them into consciousness.

In studies of dissociative identity disorder, Reinders et al. [30] recorded subjective emotional reactions such as fear, sensorimotor reactions such as restlessness, cardiovascular responses (heart rate, blood pressure, and heart rate variability), and cerebral activation patterns in dissociative-disorder patients under different mental states. They read a story from the patient's life that pertained either to their trauma or to a nontraumatic autobiographical event. When in their neutral mental state, patients reacted to the story of their traumatic experience as if it were a neutral memory and claimed not to recall it. When in their traumatized personality state, they had a significant subjective and cardiovascular reaction to the traumatic memory and a different cerebral activation pattern, and they remembered the event. In a literature review on the neurobiology of dissociative states in borderline personality disorder, dissociative identity disorder, and dissociative-subtype PTSD patients, Krause-Utz et al. [31] concluded that dissociation may be a protective strategy to cope with overwhelming emotions in traumatic/stressful situations, and that the cost of the dissociation is a disruption of mental functions that are crucial to the development of identity, self-control, and emotion regulation. The dissociative process structure and function is relevant not only to the conscious/unconscious dichotomy, but to the unconscious itself. Therefore, it may not be possible to bring it into consciousness. According to Lyons-Ruth et al., [32] based on their extensive research on dissociative disorder patients, the trauma of infancy, believed by some to be the source of dissociative disorders, may not be the most prominent cause of dissociations. Rather, they found that the most common cause was the emotional unavailability of patients' mothers. Thus, when reporting the incidence of trauma in their lives, patients with dissociative disorders may not have access to the real memories of that trauma, probably because the dissociation process renders the recollection impossible. Can other types of brain-related processes be utilized as the basis for change in psychotherapy when the genesis of insight is not feasible? Adult brain neurogenesis and neuronal plasticity, brain mechanisms involved in the development of mental conditions that may be affected by psychotherapy, are potential candidates.

Adult neurogenesis is the continuous generation of new neurons in discrete regions of the brain throughout life, which is now regarded as the fundamental mechanism of neuronal plasticity. [33] That this process may fail in mental disorders has recently become an area of active investigation. The effects of neurogenesis failure in PTSD have also been documented. Efficiently processing fear-related environmental cues is a product of the hippocampus cortical network,

which participates in assessing fear-generating stimuli related to the production of adult-borne hippocampal dentate granule cells. This mechanism has been shown to be adversely affected by stress. [34] As a result of this loss of neuronal cells, Krystal et al. [35] cited evidence that a "synaptic disconnection syndrome" develops that would require new neuron formation for resolution. These authors proposed using ketamine to stimulate neurogenesis. Furthermore, Mahan et al. [36] cited evidence that fear conditioning may cause an abnormal increase in amygdala neurogenesis by stimulating brain-derived neurotrophic factor, which can promote those neurons' growth, consolidating fear generation through hypertrophy of the amygdala cells involved in fear generation. The result is a decrease in the hippocampus prefrontal cortex assessment of fear sensitivity and an increase in the amygdala fear-generation system. This process is consistent with observations of an atrophied or constricted hippocampus and a hypertrophied amygdala in these patients. In considering neuronal plasticity as a potential perspective for correcting attachment failure deficits, psychotherapy may facilitate that plasticity.

Meyer [37] cited evidence that the ability to learn a new attachment through psychotherapy can reverse neuronal deficits. She cited an example of a college professor who had a stroke that affected his ability to speak, walk, and perform other common activities who regained his cognitive and motor function through learning new attachment patterns. The author asserted that attachment deficits may impair neuronal plasticity, resulting in mental disorders, and it may be necessary to achieve a new attachment through psychotherapy to reconstitute neuronal plasticity capacity lost because of the original attachment failure.

Schore [38] noted that dissociation is a prominent symptom in patients who suffered from early trauma, which impaired the right, attachment-related brain function. This situation impairs unconscious communication with others, which, in turn, impairs the person's capacity for attachment. Schore suggested that psychotherapy can be designed to reverse this impairment in right brain function and thus reverse the dissociation. Addressing the right brain in psychotherapy activates memories in the form of enactments that can be resolved in the treatment process. In this sense, psychotherapy is restoring neuronal plasticity. Thus, insight may not be a possible way to resolve an underlying separation bonding deficit. However, restructuring the psychotherapy situation so that the therapists can "listen" to the unconscious of the patient with their own unconscious may be able to generate the attachment between them that is needed. Creating a psychotherapy structural model to correct the original deficit—as I am demonstrating in the case of a patient with PTSD—may facilitate this listening process.

3.2 Resolving dissociation through psychotherapy while stimulating neuronal plasticity

The neurobiology of dissociation regarding the brain circuit associated with fear—the amygdala-hippocampus prefrontal lobe circuit—is common in PTSD patients and is reflected in their inability to access normally accessible information and control motor processes. Dissociation also includes involuntary intrusions of sensory, affective, and cognitive information into conscious awareness or behaviors. [39] Dissociations are a biological defense mechanism for coping with an overwhelming threat that cannot be avoided. [40] It is geared to "contain" the fear level of the patient by limiting its impact on the sensitive regions of the cerebral cortex, but it simultaneously confirms to the patients that their fear is not masterable by their cortex and that is why it has no access to that brain region. Sierra and Berrios [41] described dissociation as a disconnection between the corticolimbic brain regions that process fear emotions and the

amygdala fear-generating center. Studies have shown that the anterior cingulate, which is the conduit between the lower and upper centers, is shut off. The prefrontal cortex, having not received fear signals from the amygdala, exhibits increased activity, [42] which either directly or through the anterior cingulate dampens activity in the amygdala. Further evidence supports the idea that this negative feedback loop also shuts off the hippocampus during dissociation. [43] Farina et al. [44] describe the many forms that trauma-based dissociation may take, with the common factor being their disintegrative potential in the process of coordinating various aspects of the person's psychological function.
The implication of these dissociation processes may be that their integration may not be possible without addressing the underlying neurobiological deficits that have generated them in the first place. It also outlines the extensiveness of dissociation processes in those who have experienced trauma. Dissociation, according to these authors, is not a unique rare aspect of traumatic experience, but a very common factor connected with most traumatized individuals. In fact, the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) describes a recognized subtype of PTSD, the dissociative subtype.

It must be emphasized that this adaptation process is entirely neurobiological; it does not reach the level of psychological experience. Because of this, it may not be possible to resolve it using a psychological treatment strategy, because the deficit is not in the content that the patient communicates, but in the process or mode of communication itself. It is the structure of the "language-forming process" that is deficient: patients who dissociate form their communication using the underlying brain deficit. Thus, when the therapist speaks with them, patients translate what they hear into the same "language" they use to experience every communication. Since they are forming communication based on their "language of fear," they will interpret any communication, including communication from the therapist, in that same language.

Many researchers have demonstrated the connection between attachment and dissociation. Liotti [45] provided evidence that inconsistent mother-infant attachments were associated with the development of dissociation defenses to facilitate adaptation to a world they experienced as very frightening. Barach [46] demonstrated that patients with dissociative identity disorder had extreme detachment or emotional responsiveness, similarly to children who faced the loss of their primary caretaker. Barach noted that children of unresponsive caretakers were also likely to engage in dissociative or detached behaviors. Lyons-Ruth et al. [47] postulated that stress and trauma in infancy may be directly associated with inconsistent maternal behaviors in which the mother's emotional availability and her affective signals of that availability may have been traumatic to the child. This inconsistency may result in traumatic experiences that are less overwhelming and may not lead to dissociation, or they may lead to partial dissociations depending on later life experiences. The functional interrelationship between basic attachment failures and later adaptational experiences suggests that reversing patients' underlying "language failure" as we facilitate a more normalized communication of fear may simultaneously restore a more normalized attachment. The quality of the attachment between therapist and patient would be based on the normally balanced communication of fear that should have occurred in the patient's infancy. To overcome this problem, psychotherapy must correct the underlying brain problem involved in the emotional language formation deficit, which is a deficit in the structure of the neurobiological system patients use to create this faulty language. Psychotherapy must be geared to shift patients from a language of fear to a more balanced language—one that places fear and contextualized fear in a more normal equilibrium. Since dissociation is the brain's adaptive

mechanism that perpetuates this fear language, by shifting the language to a more balanced fear/contextualized fear language, we can simultaneously eliminate the dissociation process. In the case presentation to follow I have shown that as the separation bond was established with her therapists, the patient was able to not only relax the dissociation, but to then benefit from insight which was precluded by the dissociation. That is another reason why using this type of modeling of the psychotherapy situation is so essential: because it enabled the use of insight which was impossible prior to the resolution by the dissociation, which eliminated the use of insight-based psychotherapy.

As I hope to demonstrate in the case presentation, once the capacity for insight develops, and the hierarchy of the patient's psyche changes, psychotherapy becomes more insight based. Since dissociation is a neurobiological and not a psychological defense, the resolution of that deficit is one example of the resumption of neuronal plasticity through the attachment process. A second example is the ability of the patient to then form communication modes which are more understandable and to use insight- based psychotherapy, which they could not before this. The use of modifying the structure of the psychotherapy situation as has been done in this case is applicable to all other levels of psychopathology emanating from different brain nodes. The reason for this is because the attachment level is one in which primary perceptual and perceptual motor experiences are translated from brain-wave-based neurobiological experience into meaningful psychological ones. To accomplish this, the therapists must communicate unconscious to unconscious with their patients and change the structure of the psychotherapy situation, altering it to accommodate the underlying brain-wave-based unconscious communication.

4. Case Material

Harry was a 27-year-old man who was initially referred because of depression. However, he also had symptoms of PTSD. He was constantly preoccupied with death and had numerous flashbacks of traumatic experiences in his life, including being molested by his mother's best friend, his parents' alcoholism and constant screaming fights, the traumatic deaths from ovarian cancer of his mother and two of his sisters, and his own trauma of suffering from and resolving testicular cancer. He had constant anxiety, fearing a recurrence of his cancer, and often wished he could die so he could join his deceased relatives. In the past, when he told his mother about the sexual molestation, the mother accused him of lying. Neither parent ever attempted to protect Harry from danger, so he grew up constantly frightened. Despite this, he graduated from high school, left home, and obtained employment as a clerk in a stock brokerage firm, where he was very successful.

He developed PTSD symptoms after medical treatment had resolved his cancer. He was so preoccupied with the trauma of a potential recurrence that he needed a referral for psychotherapy treatment. At first, I tried working with him myself using a combination of psychotherapy and Buspar for anxiety, Prazosin for his flashbacks and traumatic dreams, and Fluoxetine for depression. When he started treatment, he had been abusing alcohol to cope with these fears, but I was able to help him stop. However, the medication and psychotherapy had only minimal effects, and his fears and depression continued unabated. He appeared to be in a near-constant dissociative state, and I did not feel I was connecting with him at all.

I decided to split the treatment, with me taking care mostly of medication management and the other therapist providing the psychotherapy. Although Harry said he liked this arrangement, it did not appear to be working either; he did not seem to be connecting with either one of us. The other therapist used cognitive behavioral and exposure-based therapy. Although superficially cooperative, his dissociations were extensive, and he appeared to be only going through the motions of treatment with no real engagement. The medications did not appear to be helpful, nor did the psychotherapy seem to matter to the patient; the other therapist's interventions also did not have any real effect on his symptoms. His fear of death and at the same time his wish to die and join his deceased relatives were extreme and appeared to worsen even as the treatment progressed. I felt that, unless we did something to improve his connection with us, we would lose him.

I applied my understanding of the neurobiology of PTSD symptoms like Harry's in developing a more effective intervention. One day, the other therapist consulted me in despair. Harry, she felt, was on the verge of killing himself and his anxiety was "through the roof." His desire to join his deceased relatives had become magnetic. He had told us of a dream in which there were blood and guts sprawled all over the countryside. The other therapist felt she was unable to reach him, something I had been experiencing as well. I suggested we meet with him together. In that session, I discussed the idea of dividing the treatment. I would be the amygdala therapist discussing his fears and concerns, while the second therapist would be the hippocampus therapist, discussing issues that would generate little if any fear. My purpose was to separate the overgrown amygdala-based fear-generating mechanism from the hippocampus-based context-generating mechanism, with the goal of helping Harry develop a capacity to formulate his experiences in terms of safe contexts that did not overwhelm him with fear. In this way, he could learn to develop contextual fear as opposed to just pure unmanageable fear. I explained to Harry that the second therapist and I needed a way to connect with him to provide a safe space away from the constant dangers he was experiencing. I suggested that, in his meetings with the second therapist, he would only discuss issues unrelated to danger, while he would discuss his concerns about danger and death exclusively with me. Both of us would provide him with psychotherapy. Even though no one beside myself in the room understood why I had suggested this, it appeared to everyone, including Harry, to be just the right thing.

Harry's treatment shifted almost immediately. His preoccupation with suicide and extreme danger subsided and the traumatic dreams decreased. Instead, he started to experience real fear at an intensity he had not been able to share with anyone previously. The dissociation that had blocked the emergence of that fear had begun to subside. At that moment, we could feel his fear in a way we had not been able to when it was blocked by his dissociations. He later recalled that, at that moment, his attachment with us began, and it would preoccupy his life for a number of years.
He learned to extend his sense of safety from the second therapist's office to his home, where he would sit for long hours just experiencing "safety," something he could not have done before. He had formed an attachment with us based on our providing him with safety in the face of danger – what I call a separation bond. Through the emergence of this bond, Harry learned to shift his experiences from pure fear to context-based fear by learning to develop a safe context in which to examine his experience of fear. As an example of this, toward the end of his first year of treatment, Harry had a dream in which he was closeted at home with his recently deceased sister. Although it was frightening to him, there was a "safe" context (his home) in which he could view the experience, as opposed to having the dream about blood and guts spilled all over the countryside, as he did before we started this treatment strategy. In addition, he was able to expose

his fear to us in a way he said he had not been able to before, and this helped him learn to organize it. We could become aware of the fear that was ignored by his parents because they were so preoccupied with their own fears that they never considered his.

During his sessions with the second therapist, Harry discussed how he was turning his home into a safe environment. He discussed how he wanted to be left alone to feel safe and how he was coping with the idea of not being able to work any longer. With me, he discussed being upset with his wife's drinking, as well as his fears of getting testicular cancer again and being traumatized by his brother's recent death. In one interaction approximately two years into his treatment, his wife called me to say he was having suicidal thoughts, so I had him taken to a hospital. When I saw him in hospital the next day, he told me he was fine and I should release him, but I did not as I could sense the intensity of fear he had, and I sent him to an inpatient unit instead. He became furious with me for doing this but later agreed that he benefited from being there, because he found there were many people like himself whose parents had neglected them. In the sessions that followed his hospitalization, we were able to reconstruct that he "expected" me to ignore his fears like his parents did, even though he was not aware of them, and he shared that his suicide attempt happened because his fear level had been very high.

Once we had established that the second therapist and I were not going to ignore his fears and that he had a safe environment in his treatment to express them, his growth expanded. He began to learn how to express his own fear—a new mode of relating for him. Gradually, his fears became contextual fears that he could frame in a way that allowed him to get help with them and gradually solve them himself. For example, when his brother died, he was able to deal with his grief about him without being with him physically. He did not rush across the country, as this would have been imprudent. His brother had only a day or two left to live when Harry had found out about the condition, so his brother would not even have been able to recognize him. He felt that, since it would have been only terror of failing his brother that made him go, he decided it was not in his best interests to do so. That type of decision would not have been possible for him a year or two earlier.

Harry was gradually able to move away from the two-person treatment model and see me exclusively. Psychotropic medication that had never worked before now had a markedly positive effect, and we were able to reduce the dosages. His dissociations ceased entirely. As his treatment progressed, we shifted from helping him express fear and contextualize it in a way he could manage (which is the goal of separation bonding) to a strategy of helping him become more flexible in applying his fear-based solutions. To accomplish this second goal, the dissociations had to disappear, because he had to be aware of the real perceptual input from which the fear arose. Otherwise, being flexible would just augment the goal of the dissociations, which was to further diminish the impact of the traumatic fear experiences. He was now able to use interpretation and support that was not available to him at the beginning of his treatment. For example, he indicated that he was too fearful to leave his house without his wife. We worked on getting him to leave for short periods of time. When he was invited to his daughter's house for a visit, he was fearful, and we examined what was the worst thing that could happen to him. My role shifted from helping him express his fears and assessing whether or not he could master them to becoming his advocate, encouraging him to try to do new things in new situations. This

meant doing things he could not do before, and helping him try out new strategies for approaching new situations to modulate his fears, using a cognitive appraisal strategy as outlined in Schore's [39] affect regulation theory.

I was far from impartial. I was invested in his success, which is what made this strategy work. Moreover, I was helping him form a sense of his own self-direction and skills, and to seek out his own desired role in life. I helped him see that he survived his cancer because he, unlike his siblings or mother, had the good judgment to seek out and follow medical advice. He was able to realize he was interested in being a leader who initiated actions to help others. Instead of seeing this as a weakness, he could reframe it as a strength.

When Harry discovered that his daughter had advanced breast cancer, he was traumatized. He told me that, if his daughter died, he would not want to live either. In my role as advocate, however, I said that, to help his daughter and others, he had to promise me that, no matter what happened to his daughter, he would live on.

Harry was able to see the wisdom in what I was saying. He also understood that I was involved and that it was important for me that he would survive. He told me this made a big difference to him. I was now establishing a bond of adaptation with him that emanated from our previous separation bond. As a result, Harry was able to speak with his daughter daily about how the daughter felt. At first, his daughter wanted to deny that any of this was bothering her. Furthermore, the daughter's friends called Harry to tell him that he was not being helpful by encouraging the daughter to talk to him about her condition; they encouraged a more arms-length approach, but I insisted he was doing the right thing. He told me how much comfort he found in my viewpoint. When his daughter's white blood cell count was so low that her chemotherapy had to be interrupted, Harry was so upset, he thought about suicide. However, he agreed not to further consider this option. Harry provided his daughter with empathy and support and, with my help, he was able to go through the phases of coping with the death of his daughter, finally becoming able to accept it and moving on with his life. This shows that the patient was able to use a single therapist offering insight and support to deal with issues that would have been too traumatic for her to handle had we not previously addressed the PTSD symptoms.

5. Conclusions

This case illustrates the use of a neurobiological model of a proposed deficit of a patient with PTSD to inform a psychotherapy strategy. As predicted, as soon as I proposed this model to Harry, he immediately dropped the dissociative barrier that had limited his ability to communicate his fear. The bond of separation he formed with his two therapists enabled him to develop a capacity for contextualized fear, which resolved both his dissociations and his PTSD. He was able to develop a sense of self and cope with the grief of losing his daughter through establishing his ability to deal with loss, which entails having a separation bond.

There is exponentially growing interest in integrating neuroscience into psychotherapy, and many authors have emphasized the importance of doing so. Javanbakht and Alberini [48] indicate that this approach could exponentially increase the formulation of new hypotheses and ways to approach mental conditions that would not be accessible without that research. Fuchs [49] emphasized the role of neuroplasticity in brain function as a way of expanding psychotherapy processes, and Thomas [50] focused on the neurobiological research as a system which can be integrated

with the social system to expand our methods for psychotherapy. This article is an example of this type of expansion, by demonstrating a neurobiological model for the treatment of PTSD.

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