Review
Ανασκόπηση

Neurobiology of emotional trauma

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An emotional trauma may induce a cascade of neurobiological events that have long-lasting consequences even altered gene expression. Early abuse and neglect can deregulate the child's developing neurobiological system by reducing its resistance to stressful events, leading to later problems of emotional regulation. Children who have been subjected to physical or emotional abuse tend to pay more attention to signs that contain anger and are hypersensitive to threat. Scar hypothesis and the theories of behavioural sensitization or electrophysiological kindling suggest that emotional traumas may leave traces that persist even after remission of depression, and render individuals vulnerable to the onset of new episodes, even under the influence of only moderate psychosocial stress. Unfavorable early social experiences, such as emotional abuse or institutionalization can affect the structure and function of the prefrontal cortex. Exposure to repeated emotional stressors, even in the absence of post-traumatic stress disorder (PTSD) diagnoses, has been shown to produce increased synapse formation and dendritic growth in basolateral amygdala, dendritic retraction in the hippocampus, and anxiety-like behavior against specific triggers, such as phobia of open spaces. During the narration of an emotionally traumatic event, there is activation of the limbic system, the right amygdale, the orbitofrontal cortex and the anterior cingulate gyrus. In addition, there is an activation of the anterior insula, which records the physical impact of negative emotions, and the anterior and medial temporal cortex, which are involved in negative emotions. Neuroimaging studies in PTSD patients have found hypoactivity in the frontal lobe, anterior cingulate and thalamic areas, indicating the effects of PTSD on executive function, attention and cognitive, memorial, and affective and somatosensory integration. One of the most replicated findings in studies involving PTSD patients is the decreased activation of the dorsolateral prefrontal cortex. Studies have also found a negative correlation between the dorsolateral prefrontal cortex and amygdala activation. A recent meta-analysis revealed structural brain abnormalities associated with PTSD and emotional trauma and suggested that global brain volume reductions can distinguish PTSD from major depression. Neuroimaging studies of successful eye movement desensitization and reprocessing (EMDR) treatment have consistently shown that patients exhibited increased frontal lobe activation. Moving beyond diagnostic boundaries, focusing on the causal interplay between specific traumatic processes and using standardized measures, are useful directions for future research in memory, emotion and emotional trauma.

Key words: Emotion, emotional trauma, post-traumatic stress disorder, prefrontal cortex, amygdala, scar hypothesis.

Emotional trauma

Childhood trauma is a significant risk factor for later development of serious mental illness, such as major depression or schizophrenia.[1,2] High risk for psychosis individuals with childhood emotional trauma showed high comorbidity with mood and anxiety disorders.[3] Apart from being at increased risk of depression, people with a history of childhood emotional trauma are also more likely to have an early onset, and increased chronicity and comorbidity.[4] Early abuse and neglect can deregulate the child's developing neurobiological system, by reducing its resistance to stressful events, and leading to later problems of emotional regulation.[5] People experiencing childhood and adolescent abuse and neglect tend to enter environments that put them at a higher risk of adversity during adulthood.[6] Many abuse cases can be identified as cases suffering from post-traumatic stress disorder (PTSD), which has been described as an anxiety and memory disorder, characterized by the person's inability to "integrate" the traumatic event into consciousness. PTSD manifests with dramatic symptoms of hyperarousal, intrusive memorial recollections, nightmares, and somatosensory flashbacks. These intrusive fragments may be visual, olfactory, auditory, kinaesthetic or visceral.[1] Emotional trauma has serious implications on a brain's executive functionality, resulting in the development of falsifications, distortions and deregulations of cognitive functions.[7] The abused child becomes hypervigilant for dangerous signals, magnifies non-verbal signs at the expense of verbal, and shows a selective attention to non-verbal cues, such as facial expressions. These children tend also to have difficulty in regulating their emotions, to withstand unpleasant feelings and to feel positive emotions.[8]

Neurobiological research suggests childhood abuse is more likely to affect brain-derived neurotrophic factor expression, leading to dysregulation of the HPA axis and associated dopamine changes.[9,10] Sympathetic nervous system activation could result in anger or fear, with congruent cognitive processing (e.g., hypervigilance and threat-focused reasoning), and aggressive, dependent or avoidant behavior.[11] In contrast, dissociative detachment response, ranging from emotional numbing to more intense experiences of derealization and depersonalization and interpersonal passivity may be manifestations of parasympathetic nervous system activity.[12,13] Excessive and prolonged stress hormone release weakens the structures involved in explicit memory in several ways. Damage to the hippocampus can disrupt the neuronal circuits, fragmenting various parts of traumatic experiences.[14] This can also be caused by dissociation, when individuals may focus on non-traumatic aspects of the environment or images, as a way to escape.[15] Damage to the hippocampus and the orbitofrontal cortex can prevent the encoding of cognitive and autobiographical memory.[16] A situation that is perceived as "dangerous", such as a person that resembles an offender or a startling sound in the middle of the night, activates the hypersensitive amygdala to initiate an alarm reaction and a normal stress response accompanied by a feeling of fear. In this case, the brain can revert to a more "undifferentiated" emotional state, during which the affective components are disconnected and no longer recognizable. This chaotic situation may alternatively be dissociated, subsequently leading to emotional numbness, or invading into consciousness from time to time as an overwhelming emotion.[17]

Emotional traces

Research has shown that the causal role of emotional trauma during severe life events is smaller in recurrence than in first-onset episode of depression.[18] This observation was described by Post (1992)[19] as a process of behavioural "sensitization"and electrophysiological "kindling", suggesting that emotional traumas connected with depressive episodes may leave some traces that persist after remission, and render individuals vulnerable to the onset of new episodes, even under the influence of only moderate or no psychosocial stress. This is the basis of the scar hypothesis of depression,[20] which assumed that "something", presumably encoded at the biological level, increases the likelihood of future vulnerability. Meanwhile, a broad variety of domains are described, such as psychosocial, cognitive, emotional, which may be involved in this vulnerability.[21]

The gradually increasing scar obviously functions as an additional risk factor and as the catalyser of a vicious circle for the ultimate development of clinical depression. Likewise, "scarring" residual symp-

toms after the depressive episode constitute a risk factor for relapse. Negative emotionality and low self-esteem can be perceived as potential risk factors, related to negative environmental events, and at the same time induce risk for further development of scars.[22] Evidence suggests that psychosocial stress activates the HPA axis and, in turn, the dopamine and serotonin systems, where exaggerated effects have been observed in individuals who experienced childhood adversity.[23] Moreover, early adversity may lead to the formation of negative schemas of the self and the others, and it may be associated with faulty responses to environmental stimuli, such as informational processing bias for negative or irrelevant stimuli.[24]

Two types of sensitization mechanisms have been described: one related to the stressor and one to the affective episode. An emotional trauma may induce a cascade of neurobiological events that have long-lasting consequences such as altered gene expression. In this way, the individual may end up more vulnerable and exhibit altered behavioural responses to stress because of the sensitization by previous stressors.[19,25] In conclusion, both stressors and negative mood states may be able to produce scars that are relevant to the vulnerability for depression. Additionally, these processes may work together in producing vulnerability and are probably gradually inducing an increased amount of scar over periods of time. Some evidence exists that the process of kindling involved in the risk for new depressive episodes reaches consolidation at a certain point after which the amount of acquired vulnerability stabilizes.[26] Based on the associative network theory, we would predict that not using the connections between negative concepts will weaken the strength of the associations, increase the threshold for activation and thus decrease the vulnerability, via reducing the previously developed scarring. At the level of dealing with stressful situations, it has been found that the experience of positive emotions during stressors decreases the level of stress sensitivity. Moreover, positive emotions neutralize the expression of genetic risk associated with increased daily life stress sensitivity, through the decrease in previous amount of scarring.[22]

In the field of epigenetics, the S-allele of the 5-HTTLPR, by 44 bp shorter than the L allele, was found to be associated with increased probability of depression when there is history of stressful factors.[27] However, a recent meta-analysis of 31 data groups from 38,802 individuals showed that the S-allele of the 5-HTTLPR gene does not significantly increase the risk of depression after a stressful event, or this may be the case in some cases only.[28] The authors concluded that if an interaction exists in which the S allele of 5-HTTLPR increases risk of depression only in stressed individuals, then it must be of modest effect size and only observable in limited situations.

Emotional trauma and psychosis

There are concerns about the under-detection of trauma in persons with psychosis or other severe mental illnesses, although there is evidence for a clear link between childhood adverse experiences and psychotic symptoms, with different proposed pathways. First is that psychosis is the result of childhood adversity.[29] Other possible pathways are that psychosis is a dimension of PTSD resulting from trauma,[30] or that trauma is a result of psychotic symptoms or involuntary treatment experiences.[31] Moreover, Lu et al (2017)[32] suggested that the experiences both of psychotic symptoms and psychiatric treatment, potentially traumatic, can be a powerful barrier to engaging people in mental health services and facilitating recovery.

Up to 80% of at clinical high risk for psychosis youth endorse a lifetime history of childhood traumatic events and victimization, while several studies have shown that the experience of childhood trauma predicts psychosis onset among high risk individuals.[3] Neuroimaging studies (i.e., magnetic resonance imaging; functional magnetic resonance imaging; positron emission tomography; diffusion tensor imaging; multimodal) have revealed that in those high risk individuals who converted to psychosis, functional changes in striatal dopamine synthesis and release were observed.[33,34] Oswald et al (2014)[35] found that perceived stress partially mediated the association between childhood adversity and ventral striatal dopamine responses.

There are some similarities between the symptoms of PTSD and psychosis. Hallucinations in psycho-

sis are analogous to the experience of flashbacks, intrusive images and bodily sensations associated with PTSD as they both present in visual, auditory, or tactile modalities. Suspiciousness in psychosis resembles the hypervigilance in PTSD and negative symptoms in psychosis resembles the avoidance behaviors in PTSD.[3,36] Morrison et al (2003)[37] proposed an integrative model of the spectrum of trauma reactions outlining three routes between trauma and psychosis: (1) trauma may lead to psychosis, (2) psychosis and related experiences can themselves give rise to PTSD, and (3) both psychosis and PTSD may lie on a spectrum of shared reactions to emotional trauma.

Stevens et al (2017)[38] proposed the following clinical manifestations relating to traumatic vulnerability, trigger, and treatment implications: (1) The "classical" association between trauma and psychosis, by referring to childhood trauma leading to an overt traumatic and coexisting schematic vulnerability.[39] A meta-analysis supported the existence of traumatic psychosis by suggesting that if childhood abuse was eradicated, then one-third of adult psychosis would not occur.[29] (2) Symptoms appear to have a chronic and pervasive vulnerability that is genetic and/or organic in nature.[40] (3) The symptoms of PTSD are developing prior to the onset of psychotic symptoms. This type of psychosis, similar to the traumatic psychosis subgroup, is triggered by trauma, but the symptoms may be a response to an initial trauma of any nature (rather than re-traumatization) and the symptoms of PTSD predate the emergence of the symptoms of psychosis.[41] (4) There is a triggering of PTSD, as a result of acute psychosis and could also relate to previous traumas that have been suppressed, where re-traumatization occurs.[42]

Neuronal networks

Planning, judgment, decision making, set shifting, anticipation and reasoning are cognitive processes required for the successful completion of any complex behavioral or cognitive task.[43] Also required in this context is the suppression of unnecessary input and output, as well as the inhibition of inappropriate responses. The amygdala, which is fully developed during adolescence, is essential for the decoding of emotions. On the contrary, the system of critical reasoning –prefrontal cortex (PFC)– matures slowly up until early adulthood. This maturation process is identified with an anterior-posterior direction of maturation in adolescent brain mentioned above. Therefore, there is a lack of balance between the ("reasonable") prefrontal cortex, which is still developing, and the ("impulsive") amygdala system in the adolescent brain. Thus, we would suggest that emotions and feelings outweigh critical thinking and logical decisions, even though teenagers may be aware of the imminent danger.[44]

The prefrontal cortex-PFC appears to have two overlapping and interconnected neural networks: one involving the orbitofrontal cortex, which plays a role in emotional and motivational aspects of reward expectancy, and the other involving the dorsolateral prefrontal cortex (DLPFC), hypothesized to subserve working memory and related cognitive processing of reward expectancy.[45] In resolving a complex cognitive problem, such as reconstructing a past memory, selecting words to express a thought or attempting to identify a person, a set of interconnected cortical areas can execute an extremely rapid evaluation of a vast informational landscape, while considering numerous goals, constraints, scenarios and hypotheses, until the entire system settles into the solution to the cognitive problem.

The distribution of attention within the extra personal space is coordinated by a large-scale network built around the frontal eye fields, the intraparietal sulcus, and in the cingulate gyrus. The frontal eye fields and the posterior parietal cortex as a network appear to be responsible for spatial attention, the midtemporal and temporopolar cortices for face and object recognition, the amygdala and the hippocampo-entorhinal complex for the emotion-memory network, Wernicke's area and Broca's area for the language network, and the prefrontal cortex and posterior parietal cortex for the working memory-executive function network. The activation of visual areas is related to the fact that visual image recollection involves the same area as visual perception. This activation of the visual areas has also been observed during induced recollection of images when observing repulsive photographs. Emotional trauma is accompanied by a reduction in associative process involving words and abstract concepts.[46]

The thalamus has a major role in interconnections of the above functions. Most of the thalamic subnuclei have almost no connections among each other. Obviously, they fulfil the important role of setting co-activation boundaries for separating the activity of one network from the activity of others. Interconnected cortical areas send interdigitating projections to the striatum. Since the striatum receives cortical inputs without, however, projecting back to the cerebral cortex, it could serve the role of an efference synchronizer for the coordination of the outputs of cortical areas in a given network.[47]

Neuroanatomy

Exposure to acute emotional stress, in the absence of diagnoses of PTSD, has been shown to increase spine synapse formation in basolateral amygdale, which is associated with anxiety-like and avoidant behaviors. Exposure to repeated emotional stressors, in the absence of PTSD diagnoses, has been shown to produce even greater synapse formation in basolateral amygdala, increased dendritic growth in basolateral amygdala, dendritic retraction in the hippocampus, and anxiety-like behavior against specific triggers, such as phobia of open spaces. These changes are assumed to be related to anxiety symptoms, avoidance, hypervigilance and overconsolidation, as well as intrusion of traumatic memories.[48] Ganzel et al (2007)[49] used fMRI to study the reaction to images of fearful vs neutral subjects in people who were at different distances from the point of disaster on September 11, 2011. More than 3 years after the event, in response to viewing fearful faces, bilateral amygdala activity was found to be significantly higher in those who were within 1.5 miles of the point of destruction, compared to those who were living farther away at that time.

Neuroimaging studies in patients with PTSD have found hypoactivity in frontal lobe, anterior cingulate and thalamic areas, indicating the effects of PTSD on executive function, attention and cognitive, memorial, and affective and somatosensory integration. On the contrary, hyperactivation of temporal and limbic structures apparently reflects arousal, hypervigilance and overconsolidation/intrusion of traumatic episodic memory. Patients suffering from PTSD exhibit reduced thalamic activation, consequently resulting in impairments in somatosensory, cognitive, memorial, and interhemispheric integration.[50] Although some researchers have found that traumatic memories are retrieved differently than are emotional memories, others have demonstrated that the phenomenological characteristics of these memory types are highly similar.[51]

Given the thalamus' pivotal role in interconnection functions between several structures, the consequences of this lowered thalamic activation are impairments in the functional connectivity of various neuronal networks, evidenced by the following: (1) Failure of somatosensory integration, manifested by fragmentation with respect to olfactory, auditory memories, and taste memories, visual flashbacks, and disturbing kinesthetic sensations. (2) Failure of cognitive integration, manifested by self-blame and shame. (3) Failure of memory integration, manifested by overconsolidated episodic memory, coupled with impaired semantic memory. (4) Failure of hemispheric dynamic integration, manifested by marked hemispheric laterality and its concomitant kindled and hyperemotional state of the nervous system.[52]

Hemispheric laterality studies have consistently shown that in patients with PTSD there is a marked increased activation in the right hemisphere as compared to the left hemisphere. There has been a growing consensus that interhemispheric coherence is regulated by the corpus callosum, which facilitates the synchronous oscillation of bilateral neural networks and their functional connectivity. As noted above, the thalamus is the central structure that mediates the synchronous oscillation and must, therefore, be critically involved in the synchronous oscillatory functioning of the corpus callosum.[47,50]

One of the most replicated findings in studies involving PTSD patients is the decreased activation of the dorsolateral prefrontal cortex, BA 9 and 11, a structure known to serve several key emotion modulating processing functions. The emotional activity that the frontal lobes are required to modulate seems to be partly mediated by an overactive amygdala in PTSD patients.[53] Lowered activation of the left dorsolateral prefrontal cortex in patients suffering from PTSD is consistent with the hyperarousal response that is noted. Studies have found a negative correlation between the dorsolateral prefrontal cortex and amygdala activation.[54,55]

Eye movement desensitization and reprocessing therapy (EMDR) has been shown to improve the hyperarousal response and to repair cognitive, emotional and somatosensory integration.[56] The mechanisms of this repair are still being investigated. EMDR's ability to mediate the activation of the specific ventrolateral and the nonspecific centrolateral thalamic nuclei allows for the repair of synchronous oscillation and, therefore, of a thalamo-cortical temporal binding. Clinically, this may mediate improvement of the integration of somatosensory, cognitive, and memorial function. The right-sided lateralization seems to be improved with EMDR. Additionally, the ventrolateral thalamic nucleus activates the dorsolateral prefrontal cortex, which is the most consistent finding of EMDR neuroimaging, further facilitating the "integration" of traumatic memories into neocortical networks. Actually, neuroimaging studies of successful EMDR treatment have consistently shown that the patients exhibited increased frontal lobe activation.[56–58]

Neuroimaging

Unfavorable early social experiences, such as abuse, deprivation of nurturer, institutionalization, on having depressed mother, affect the structure and function of the prefrontal cortex (PFC)[59] while the parental socioeconomic status during childhood was found to affect the executive control systems of the prefrontal cortex.[60] Flashbacks are painful and sometimes difficult to be expressed in words. It is well known that the Broca's area is involved in speech production and inner speech.[61] In PET-scan studies, Broca's area appears "silent" during the traumatic image recollection, while on the contrary, it activates when listening to a neutral narration. This reduced activity or "silence" of the Broca's area in patients with PTSD is interpreted as a manifestation of the weakened associative process while reliving the traumatic events.[62]

Brain activity have been also studied with PET-scan, in people who heard a recorded narration of the traumatic event, compared to a neutral personal narration. During the narration of an emotionally traumatic event, there was activation of the limbic system, the right amygdale, the orbitofrontal cortex and the anterior gyrus cingulate.[63] The orbitofrontal cortex is involved in physical and emotional experience as well as in the integration of memories. The anterior gyrus cingulate is involved in the cognitive recollection of images, emotions and physical representations.[64] During the narration of the traumatic event, there was also activation of the anterior area of the anterior insula, which records the physical impact of negative emotions, as well as the anterior and medial temporal cortex, two areas which are involved in anxiety and other negative emotions, while the activation had spread to the secondary visual cortex.[65]

There are indications that PTSD is associated with variation in the volume of various brain structures. A large number of MRI studies have focused on how and how much the brain is affected by the particular disorder. The brain areas of great importance are primarily the hippocampus, which is responsible for memories, and the amygdala, responsible for emotional reactions. Different meta-analyses in PTSD patients have found a significant decrease in hippocampus volume[66–69] but not in amygdala volume,[70,71] in comparison to healthy subjects. Another meta-analysis studied different brain areas and found significant decrease in hippocampus and amygdala volume.[72] In a recent meta-analysis, which included almost all the above studies (fMRI region-of-interest and voxel-based morphometry (VBM) studies in PTSD), found that patients with PTSD compared with all control subjects had reduced brain volume, intracranial volume, and volumes of the hippocampus, insula, and anterior cingulate. The VBM meta-analysis revealed prominent volumetric reductions in the medial prefrontal cortex, including the anterior cingulate. Compared with region-of-interest data from patients with major depressive disorder, those with PTSD had reduced total brain volume, and both disorders were associated with reduced hippocampal volume. The authors suggested that global brain volume reductions can distinguish PTSD from major depression.[73]

Conclusion

Summarizing, multiple models including the gene–environment interaction, the stress-vulnerability and the stress-sensitivity hypotheses, have been cited to explain the link between emotional trauma and later mental disorders, with emphasis on both cognitive

processes and neurobiological mechanisms, like the hypothalamic-pituitary-adrenal axis. Activation of the amygdala, the anterior gyrus cingulate and the orbitofrontal cortex, with right-hemisphere dominance, are the most reliable findings in patients with emotional trauma, while thalamus has a pivotal role in these interconnections. Clinicians should be able to determine whether emotional trauma is a significant centrepiece or a complicating factor of the presenting problem. Moving beyond diagnostic boundaries and focusing on the causal interplay between specific traumatic processes and symptoms is a useful direction for future research in memory, trauma, and emotion. Finally, standardized clinical and neuroimaging measures would help to compare different factors, like the brain connectivity and the number or the age of occurrence of traumatic events, in order to find whether there is a specific brain area, brain function, or a "critical period" for childhood emotional trauma.[3,72,74–76]

Νευροβιολογία του συναισθηματικού τραύματος

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Το συναισθηματικό τραύμα μπορεί να προκαλέσει μια αλληλουχία νευροβιολογικών γεγονότων με μακροχρόνιες συνέπειες, όπως την τροποποίηση έκφρασης των γονιδίων. Η πρώιμη κακοποίηση και παραμέληση μπορεί να απορρυθμίσει την ανάπτυξη του νευροβιολογικού συστήματος του παιδιού με αποτέλεσμα τη μείωση της αντίστασης στα στρεσογόνα γεγονότα και την ανάπτυξη προβλημάτων σχετικών με τη ρύθμιση του συναισθήματος. Τα παιδιά που έχουν υποστεί σωματική ή συναισθηματική κακοποίηση τείνουν να δείχνουν μεγαλύτερη ευαισθησία στην απειλή και προσοχή σε σημάδια που περιέχουν θυμό. Η υπόθεση της ουλής (scar hypothesis) και οι θεωρίες συμπεριφορικής ευαισθητοποίησης και ηλεκτροφυσιολογικής αυτοανάφλεξης (behavioural sensitization and electrophysiological kindling) υποδεικνύουν ότι το συναισθηματικό τραύμα μπορεί να προκαλέσει ίχνη που επιμένουν μετά την αποδρομή της κατάθλιψης και καθιστούν τα άτομα ευάλωτα στην επανέναρξη ενός νέου επεισοδίου, ακόμη και μετά από επίδραση ενός μικρού στρεσογόνου γεγονότος. Πρώιμες επιβαρυντικές εμπειρίες, όπως συναισθηματική κακοποίηση ή ιδρυματισμός βρέθηκε να επηρεάζουν τη δομή και τη λειτουργία του προμετωπιαίου φλοιού. Έκθεση σε συνεχή συναισθηματικά γεγονότα, ακόμη και με την απουσία διάγνωσης μετατραυματικής διαταραχής (PTSD), έδειξε να προκαλεί αυξημένο σχηματισμό συνάψεων και δενδριτών στην πλαγιοβασική αμυγδαλή, δενδριτική ανάσχεση στον ιππόκαμπο και συμπεριφορά άγχους σε ειδικές περιστάσεις, όπως φοβία σε ανοικτά μέρη. Στη διάρκεια αφήγησης ενός συναισθηματικά τραυματικού γεγονότος υπάρχει ενεργοποίηση του μεταιχμιακού συστήματος, της δεξιάς αμυγδαλής, του κογχομετωπαίου φλοιού και του πρόσθιου προσαγωγίου. Επιπλέον, υπάρχει ενεργοποίηση της πρόσθιας νήσου, η οποία ενέχεται στην επισήμανση των σωματικών επιπτώσεων των αρνητικών συναισθημάτων, καθώς και του πρόσθιου και μέσου κροταφικού φλοιού, που ενέχονται στα αρνητικά συναισθήματα. Οι νευροαπεικονιστικές έρευνες σε ασθενείς με PTSD βρήκαν υποδραστηριότητα σε μετωπιαίο φλοιό, πρόσθιο προσαγώγιο και θαλαμικές περιοχές, γεγονός που υποδεικνύει τις επιδράσεις του συναισθηματικού τραύματος στην ολοκλήρωση της εκτελεστικής, της μνημονικής και σωματοαισθητικής λειτουργίας. Ένα από τα πλέον επιβεβαιωμένα ευρήματα στις έρευνες με ασθενείς με PTSD είναι η μειωμένη ενεργοποίηση του πλαγιοραχιαίου προμετωπιαίου φλοιού. Οι έρευνες επίσης έδειξαν αρνητική συσχέτιση ανάμεσα στην ενεργοποίηση του πλαγιοραχιαίου προμετωπιαίου φλοιού και της αμυγδαλής. Νευροαπεικονιστικές έρευνες σε άτομα με επιτυχή έκβαση θεραπείας με EMDR έδειξαν αυξημένη ενεργοποίηση του μετωπαίου λοβού. Πρόσφατη μετα-ανάλυση ανέδειξε δομικές εγκεφαλικές

ανωμαλίες σε άτομα με PTSD και συναισθηματικό τραύμα, υποδεικνύοντας ότι ο συνολικός όγκος εγκεφάλου μπορεί να διαχωρίσει τα άτομα με PTSD και τα άτομα με κατάθλιψη. Η διερεύνηση πέρα από διαγνωστικές δεσμεύσεις, η εστίαση στην αιτιώδη σύνδεση μεταξύ των ειδικών τραυματικών διαδικασιών και η χρησιμοποίηση τυποποιημένων μετρήσεων, αποτελούν χρήσιμες κατευθύνσεις για τη μελλοντική έρευνα της μνήμης, του συναισθήματος και του συναισθηματικού τραύματος.

Λέξεις ευρετηρίου: Συναίσθημα, συναισθηματικό τραύμα, μετατραυματική διαταραχή, προμετωπιαίος φλοιός, αμυγδαλή, υπόθεση ουλής.

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