

# We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

5,800

Open access books available

142,000

International authors and editors

180M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index  
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?  
Contact [book.department@intechopen.com](mailto:book.department@intechopen.com)

Numbers displayed above are based on latest data collected.  
For more information visit [www.intechopen.com](http://www.intechopen.com)



## Chapter

# New Diagnosis and Treatment Approaches to Post-Traumatic Stress Disorder

*Nevzat Tarhan, Muhsin Konuk, Mesut Karahan, Öznur Özge Özcan, Sibel Öztürk Ayvaz, Gökben Hızlı Sayar, Nurper Ülküer, Hazal Ayas and Feride Zeynep Güder*

## Abstract

Post-traumatic stress disorder (PTSD) is a mental health condition and disorder causing psychological deterioration triggered by terrifying events or traumatic experiences either by experiencing or by witnessing it. Though many people have common feelings, PTSD symptoms vary from one person to another. So it is strongly recommended to focus on new diagnostic and therapeutic methods relying and structured on a neurobiological dimension by collecting and processing neuroimaging data. It is crucial to make a profound analysis of PTSD in terms of its ontological, biological, developmental, psychological, and sociological aspects. Both with the new treatment opportunities and involvement of in silico-based artificial intelligence applications, new psychotherapy techniques and new discourses in digital media will be possible. Within the scope of the study, ontological discussions are followed and juxtaposed by Neuro-Biological Perspectives on Genomics and Epigenomics as well as the clinical and neuro-imaginative perspectives and clinical overviews of PTSD. Besides, the neuro-developmental views in the context of children along with adverse childhood experiences (ACE) and their relation to PTSD are analyzed by emphasizing the significance of brain development. Sociological aspects of PTSD in the digital habitus are collocated to develop unique therapy approaches that embrace sociological perspectives of Information Society.

**Keywords:** PTSD, ontology, neurobiology, genomics, epigenomics, neuro-imaginations, ACE, toxic stress, cyber violence and traumas

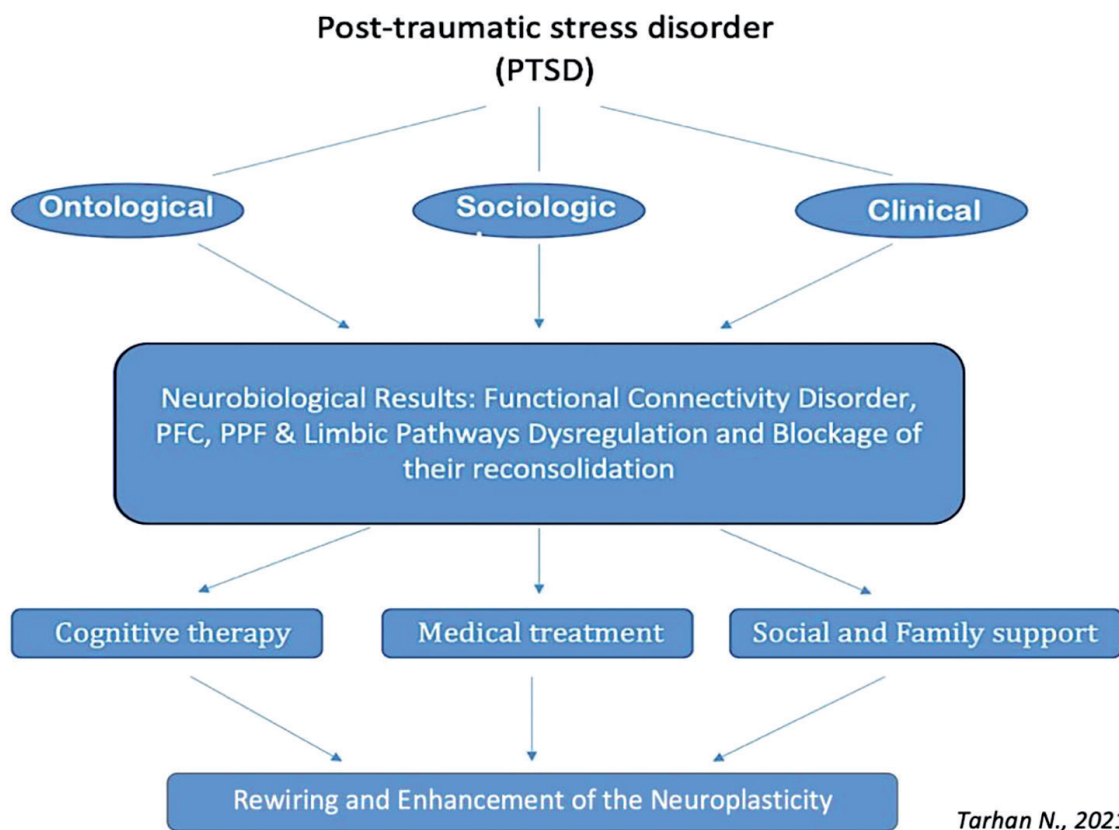
## 1. Introduction

Tsunami-like digital revolutions cause a sharp transformation of the world from modern society to Information society that eventually changed the scientific paradigms and approaches. Therefore, there appear several problems related to major clinical problems. Similarly, there emerge gaps between the cathartic effect of clinical interviews and therapeutic alliance. This gap also exists in the deeper understanding

of the real experiences of the client and the help of the therapist. This chapter discusses the new diagnosis and treatment opportunities of post-traumatic stress disorder (PTSD) that appeared in stunning psychotherapy techniques and approaches and the changing role of psychiatrists. Since the shift in society and technological advancements doubles the burden of psychiatrists to a large extent, conventional diagnoses and therapies for PTSD do not work properly. It seems inevitable that recent developments and challenges surpass conventional approaches to PTSD that can easily miss embracing the overwhelming realities that those people experience.

This study, therefore, aims to explore and exemplify new diagnostic and therapeutic approaches to PTSD cases by embracing the digital revolutions of society with their novel implications and insights. To search for new working diagnoses and treatment opportunities, PTSD issues are analyzed by different angles and multi perspectives developed by certain disciplines. For Tarhan, the algorithm for the diagnosis and treatment process of PTSD has three main dynamics—ontological, sociological, and clinical (see **Figure 1**). These three dynamics are expanded by two more additional dynamics. Recent research on genomics and epigenomics in neuro-biological perspectives along with clinical and neuro-imaging perspectives are presented that they conglomerate new insights and implications for PTSD. They are explained in detail in the second and third parts of the chapter, respectively.

The underlying reason for designing this chapter in five subsequent parts comes from the need to shed some new light on PTSD from different angles. The latest technological innovations in genomics, epigenomics in neuro-biological perspectives, and clinical and neuro-imaging perspectives challenge the psychiatrists' role as they are expected to update their reference frames related to PTSD for diagnosis and



**Figure 1.**  
*The algorithm for the diagnosis and treatment process of PTSD.*

treatment alternatives. Therefore, the study presents five interdisciplinary domains that are correlated with each other thematically as they particularly aimed to answer the question of what possible new approaches we have in terms of PTSD.

To pursue the goals mentioned above, the first part of the chapter starts with an ontological discussion that analyzes PTSD in terms of epidemiology and existential questions. In addition to those dimensions, the heavy impact of Coronavirus Disease (COVID-19) on PTSD cases and its sociopolitical consequences are portrayed in detail. This first part also throws a question as to whether post-traumatic growth is possible or not. It underlines that ontological well-being should not be ignored as healthy mindsets that eventually produce a healthy society. Ontological well-being and positive psychology are given as the ultimate aims of the science that would work for the benefit of the whole society counting PTSD patients. However, it is a challenging standpoint to offer a therapy that can work for the whole society. In the PTSD context, if social psychiatry utilizes cutting-edge approaches by wisely utilizing the technological advances of the cyber era, the mental and social well-being of society can be reached.

The second part follows the ontological discussions with Neuro-Biological Perspectives on Genomics and Epigenomics in PTSD. This part also discusses current Genome-Wide Association Studies and current Epigenome-Wide Association Studies. PTSD can occur at the organic, cellular, and molecular level due to the effect of an external event such as psychological trauma, as well as inherited from generation to generation. In PTSD, genetic and epigenetic studies are prioritized based on biological research because they are promising in elucidating molecular functioning and finding biomarkers. The goal of these studies is to lay the groundwork for new and preventive treatments to ameliorate the symptoms and the disease. In this context, there is current evidence for the potential of current genetic and epigenetic studies from the biological risk factors of PTSD.

In a similar vein, the third part of the chapter portrays the clinical and neuro-imaging perspectives and clinical overviews of PTSD. This part argues the practical psychological treatments, such as neuromodulatory and neurobiological treatments. This part presents the Post-Traumatic Stress Disorder Checklist for DSM-5 as a self-report measure to evaluate the presence and severity of PTSD symptoms.

The fourth part expands the topic by adding the neuro-developmental perspective of PTSD, particularly in the context of children. Here, PTSD is analyzed by emphasizing the significance of Brain Development. This part is followed by a detailed explanation of adverse childhood experiences (ACE) and their relation to PTSD.

The fifth and final part of the chapter reviews PTSD in its correlation to the new paradigms and changes in Information Society. This part brings forward those transformations of society and media that necessitate searching for new discourses and alternative digital therapies for PTSD. Here, within the context of Attachment Theory, this final part warns of the potential evil that is inherent in new media, particularly in Digital Habitus and Dark Web. It would be wise to be cautious toward the widespread acts of cyber violence. It highlights the significance of a new emotional repertoire that can be identified as new types of traumas seen in digital habitus. In conclusion, since new trauma types emerged on cyber platforms, the mission and goal of social psychiatry are recommended to adopt these changes. Therefore, keeping pace with these transformations is widening the job definition of contemporary psychiatrists. Searching for new ways and counter activities to prevent society from demonic sides of social media ultimately shaped the agenda of psychiatrists.

## **2. Ontological discussions**

*Trauma* can be roughly defined as an event that threatens the physical and psychological integrity of the person. However, the traumatic effect changes from one person to another. While the death of a cat has a traumatic effect on some people, the most severe war conditions do not have a traumatic effect on others. In the first step, therefore, we need to find the underlying reasons for these discrepancies. Why do these individual differences arise? It is worth explaining that the meaning ascribed to the event or a philosophy of life that is not afraid of death has an anti-traumatic effect.

Some basic information regarding the understanding of PTSD can be summarized as follows—PTSD is one of the few disorders among the Diagnostic and Statistical Manual of Mental Disorders (DSM) diagnostic categories regarding the etiology. Therefore, it is relatively easy and unproblematic to diagnose a PTSD case. The trauma mentioned there may appear in different situations and features. They can be concrete, obvious, extreme, unusual, unexpected, unforeseen, etc. The fact that the event's cause is not obscure ensures that the discomfort it causes is both predictable and fully comprehensible.

In the shock of the event, we can interpret cognitive blockage in many people as the brain shutting itself off to mental stimuli due to excessive adrenocorticotrophic hormone (ACTH) and cortisol release. Later on, in other words, over-aroused state, avoidance behavior related to the event, insomnia, re-experiencing (Flashback), having nightmares are common symptoms. Even when awake, the person remembers the traumatic event over and over. S/he continues to experience the event that happened 10 years ago as if it happened yesterday. Since s/he feels like s/he is reliving the event, s/he tries to avoid the reminders.

Another definition of PTSD in DSM appears as follows: “The main feature of post-traumatic stress disorder is to experience the event of actual or threatened death, serious injury, or a threat to the physical integrity of the person, or the death or threat of death of another person. Extremely traumatic experiences, such as witnessing an event that poses a threat to one's life, injury or physical integrity, or learning that a family member or other relative has unexpectedly died or has been killed by being exposed to violence, was seriously injured or is under threat of death or injury. The development of specific symptoms following a source of traumatic stress. The person's reactions to the event in question are extreme fear, helplessness, or horror” [1].

### **2.1 Epidemiology**

Studies conducted with large populations affected by the devastating earthquake, wars, and involvement in concentration camps found PTSD development risk between 20 and 50%. In one study conducted, data were analyzed from 26 population surveys in the World Health Organization World Mental Health Surveys. A total of 71,083 respondents aged 18+ participated. The cross-national lifetime prevalence of PTSD was 3.9% in the total sample and 5.6% among the trauma-exposed [2].

The experience of trauma is by no means exceptional, with all of its disruptive, jarring, deeply traumatic, intolerable, and “extremely terrifying, helpless, or terrifying” qualities. Moreover, both the frequency of PTSD that develops after a traumatic

event and the acute stress disorder that occurs immediately after the trauma is often closely related to the threat to the person's psychological integrity.

It does not seem possible to establish a relationship between the nature of the trauma and the developing pathological picture that would require us to refer to the importance and severity of the current trauma. Of course, we should also note that different psychopathological conditions, such as post-traumatic depression and substance abuse, can occur utterly independent of PTSD. So, when we consider all these, we need to argue that the non-traumatic factors that determine the emergence of PTSD are essential enough and need to be carefully investigated [3].

## **2.2 Existential dimension**

It is the ontological and cultural dimension that does not attract much attention from non-traumatic factors. Believing in and taking shelter in an unseen reality that knows everything, controlling the existence of belief in God when they feel helpless, powerless, and weak increases resilience to trauma. The conception of guardian angels, the Holy Spirit, or absolute monotheism (Tawhid) can be mental-sheltering. This approach, which changes our perceptions toward resilience, is also used in third-wave psychotherapies (Mindfulness, Metacognitive therapies) [4].

When mainly dealing with why evil exists, the Theodicy discipline proposes that it is significant to attribute more positive meanings to evil instead of ascribing it as a punishment. For the Positive Psychology approach, perceptions can change in the direction of endurance. Philosophers develop more or less similar ideas. Epicurus alleges that the Gods do not interfere with the earth, so evil belongs to the Gods. The motivation to enjoy is a sufficient measure for man. Giving the example of his famous cave allegory, Plato declares that God is absolute good and this world is not real life. While Kant says that evil has nothing to do with God, Leibniz claims that evil is for the benefit of good. For Comte, if he cannot prevent evil, God is helpless.

Unlike all these thinkers mentioned above, existential philosophy accelerated ferocious competition with the proposition that "God does not exist or cannot be proven, man's purpose is to seek self-interest and freedom in the world, and he must be selfish." As a result, many scholars, such as Nietzsche, Sartre, Kierkegaard, and Dostoevsky, changed the purpose, meaning, and values of life formed by human values in the name of hedonism and freedom. They even found Hegel and Kant to be prescriptive. On the other hand, Karl Popper said that if there is no evidence in epistemology, falsification is required. That is, it cannot be proven that God does not exist. Analytical philosopher Alvin Plantinga, on the other hand, argued that evils are necessary not because God does not exist but because God gives free will.

Heidegger, in 1966, accepted "time" as the most fundamental ontological category in the philosophical field. Today, people emotionally become vulnerable to traumatic experiences when existentialist philosophers Camus and Sartre defend absurdism by saying there is no meaning in life. Positive psychology, for this reason, has tried to fill the gap of this meaninglessness and meet the need to search for meaning. Today, when metacognitive genes related to the search for the meaning of life are mentioned, evidence has been sought against the approach of absurdism that reduces resilience to trauma. Psychological well-being is discussed in subjective, relational, semantic and temporal, and existential dimensions. The positive psychology literature confirms the importance of psychological well-being for resilience, so ontological well-being should not be ignored [5].

Ontological well-being, apart from subjective well-being, is the evaluation of life. Here, one's own life as a project should be examined together within the contexts of "past," "future," and "present." The meaningful combination of past, present, and future is the main focus of the evaluation of life [6].

People feel the need for a solid belief. The statement, "I believe, therefore I am," has been an area in which neuroscientists present their evidence [7, 8]. Being able to connect to that feeling in situations that one cannot control and cannot afford is considered in trauma therapies. For this reason, knowing the ontological dimension in the protection and prevention of PTSD and providing resilience training are recommended by Seligman under the name of the PERMA model.

### **2.3 COVID-19 impact**

According to the March 2021 news in New York Times, the increase of existential questioning with the effect of social trauma globally draw our attention, particularly to the establishment of "Ministries of Loneliness" in England and Japan and on the search for solutions to suicide epidemics. The Ministry of Loneliness has an important mission as the existential needs of individual members of society have to be truly met by the systems. They can open new ways for the people who face trauma and allow them truly benefit from the effects of new diagnostic and therapeutic approaches. The existence of the ministry is highly significant, particularly in the case of the suicide epidemic. It can prevent suicidal people's feeling isolated and self-destructive action.

For this reason, studies on reorganizing the meaning of life and lifestyle have increased with the effect of social trauma. In a similar vein, Üsküdar University Senate reflected their studies and published a manifesto on Earth Day on April 22, 2021, to increase the resistance of world societies to trauma after the Pandemic and lead life toward the better tried to announce it globally. Üsküdar University here aims to help people who had a traumatic experience on a wider scale.

### **2.4 Can trauma have sociopolitical consequences?**

After the cold war, the world became unipolar. As a result, global trends toward social justice have declined. Therefore, it is necessary to avoid the emergence of a new wealth hostility and to minimize opportunity and income inequality. For this reason, it is the right place to commemorate Marx and Engels together and talk about that extraordinary passage from the Communist Manifesto:

"Wherever the bourgeoisie has taken over, it has put an end to all feudal, patriarchal, rural relations. It has ruthlessly cut off the tangled feudal ties that bind man to his 'natural superiors,' leaving no other bond between man and man than pure self-interest, solid 'cash payment.' It has drowned the divine ecstasy of religious bigotry, the chivalric spirit, and petty-bourgeois sentimentality in the icy waters of selfish calculation. He has transformed personal dignity into exchange value and has replaced the innumerable freedoms so hard-won with that single, ruthless freedom, the freedom to trade. In short, it has replaced the exploitation of hidden religious and political illusions with open, indecent, direct, and brutal exploitation."

What will close that trauma bracket is obvious—the struggle of the poor/ oppressed for liberation, equality, and freedom. Since the oppressed/poor are naked,

all organizations and ties have collapsed, they are alone and helpless; since there is no light left for their hope, all ears to hear their voices are deaf [3].

## **2.5 Is post-traumatic growth possible?**

As the sociopolitical consequence of the trauma, the global justice movement for worldwide peace must be initiated. We conducted a study in our field to turn trauma into an opportunity and presented it to the scientific world as evidence. In April 2020, 6318 cases were screened in all provinces of Turkey on Pandemic Fears, Anxiety, and Maturation, and there was a significant increase in six questions in the post-traumatic growth scale [9]. The primary aim of this investigation was to understand whether these findings are permanent after the Pandemic is over. These six questions are as follows:

1. The priority of the things I care about in life has changed, 59%
2. My interest in spiritual issues increased, 49%
3. I realized that I could handle the weaknesses, 56%
4. I can accept the events as they are, 56%
5. I started to give more importance to my social relations, 48%
6. I understand the value of the things I have, 74%

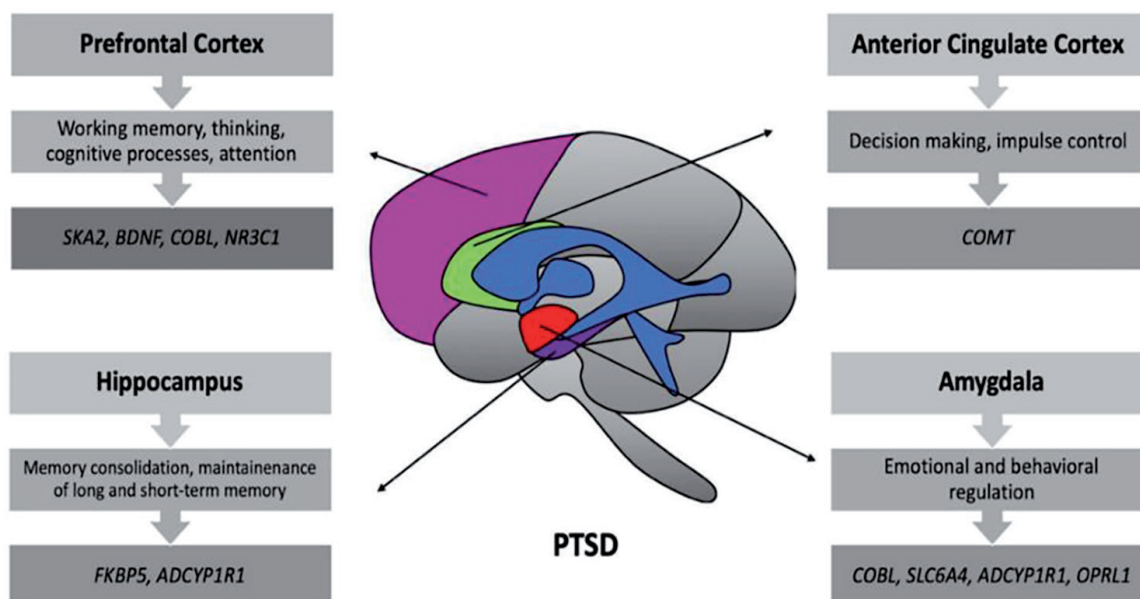
As a result, the causality relationship between Existence and Trauma draws attention. In addition, although 2 years have passed since COVID-19, it continues to force societies as a global social trauma. There are precursors to social crises that may occur. Such as migration and increase in mental disorders as post-pandemic. The rise of individual armament globally is worrying. We must find solutions so that there is no new break and disappointment in humanity. In conclusion, within the scope of this study, it is decided to present the aforementioned global well-being manifesto that will contribute to humanity's search for meaning and solution. In addition to this, it is lucid that the role of social scientists here plays a crucial role. For a better world, media, social and political scientists and leaders take several factors into consideration. Their function to lead to global peace and mental health is explained further in part six.

## **3. Neuro-biological perspective**

### **3.1 Focus on genomics and Epigenomics in post-traumatic stress disorder**

Post-traumatic stress disorder (PTSD) is a multifactorial disease characterized by structural, metabolic, and molecular changes in various brain regions and neural circuits, such as the limbic system, hippocampal region, and prefrontal cortex (in **Figure 2**), which regulate neurobehavioral functions [10]. Epigenetic and genetic current studies are included in this section. PTSD can occur at the organic, cellular, and molecular level due to the effect of an external event such as psychological trauma, as





**Figure 2.**

*Current candidate genes associated with different brain regions and neuro-behaviors in PTSD [10].*

*Abbreviations: post-traumatic stress disorder (PTSD), catechol-O-methyltransferase (COMT), Cordon-Bleu WH2 Repeat Protein (COBL), Solute Carrier Family 6 Member 4 (SLC6A4), pituitary adenylate cyclase-activating polypeptide 1 receptor (ADCYP1R1), Opioid-Related Nociceptin Receptor 1 (OPRL1), FK506 binding proteins (FKBPS), Spindle And Kinetochore Associated Complex Subunit 2 (SKA2), Brain-Derived Neurotrophic Factor (BDNF), Nuclear Receptor Subfamily 3 Group C Member 1 (NR3C1).*

well as being inherited from generation to generation. In PTSD, genetic and epigenetic studies are prioritized based on biological research because they are promising in elucidating molecular functioning and finding biomarkers. The goal of these studies is to lay the groundwork for new and preventive treatments to ameliorate the symptoms that cause the disease. In this context, there is the current evidence for the potential of current genetic and epigenetic studies from the biological risk factors of PTSD.

Thousands or even hundreds of thousands of single nucleotide polymorphisms (SNPs) with a polygenic background are the genetic basis of PTSD according to Genome-wide association study (GWAS) summary statistic [11]. Considering the studies on twins with a traumatic history for hereditary dimensions, PTSD is inherited from 30% males and 70% females, SNPs play a major role in this hereditary process from women [12]. The heritability of PTSD following trauma has been demonstrated, but biological variations have not yet been fully defined. Elucidating the biological mechanisms underlying PTSD may contribute to a more accurate diagnosis and development of swelling-specific treatment interventions. Among the biological processes involved in PTSD and related conditions, this section focuses on epigenetic and genetic mechanisms. Genomic and epigenomic studies in large groups are valuable. Loci most recently examined in large-scale GWAS and Epigenome-wide association studies (EWAS) became candidate biological markers for PTSD.

### 3.1.1 Current genome-wide association studies

Specific PTSD genetic variants that contribute to genetic studies have been most extensively researched and are currently known in the monoaminergic neurotransmission and hypothalamic-pituitary-adrenal (HPA) axis [13, 14]. The most frequently studied serotonin transporter gene (SLC6A4) polymorphisms in the

monoaminergic system were associated with PTSD and identified with a prevalence of 45% in Europeans is the S allele frequency of SLC6A4. Association studies of 5-hydroxytryptamine 5-HT (5HTTLPR) and PTSD have been inconclusive, and a recent meta-analysis of 12 studies found no evidence of association overall, but the S allele was associated with PTSD in samples classified as having high trauma exposure [15]. Nominally significant associations between PTSD symptoms and many neurotransmitter-related genes, including 5-hydroxytryptamine (serotonin) 2A receptor gene (HTR2A), Solute Carrier Family 6 Member 3 (SLC6A3), Dopamine Receptor D3 (DRD3), Neuropeptide Y (NPY) Cannabinoid Receptor 1 (CNR1), and Regulator of G Protein Signaling 2 (RGS2) have been investigated [16–18].

One of the largest polymorphism studies of the Nurse's Health Study II, which included 845 PTSD cases and 1693 trauma-exposed controls, examined 3742 single nucleotide polymorphisms (SNPs) spanning more than 300 genes, but no gene was of clinical significance [19]. Meta-analysis and GWAS studies take an agnostic approach to the discovery of risk loci by comparing the frequencies of hundreds of thousands of SNPs and other genetic markers from the whole genome with those of controls, at least an update five gene markers are promising, including Zinc Finger DHHC-Type Palmitoyltransferase 14 (ZDHHC14), Parkinson Protein 2 E3 Ubiquitin Protein Ligase (PARK2), Kazrin, Periplakin Interacting Protein (KAZN), TMEM51 antisense RNA 1 (TMEM51-AS1), and Zinc Finger Protein 813 (ZNF813) [20–22]. The latest Military cohort study (29,539 PTSD cases and 166,145 controls) reported that Zinc Finger Protein 140 (ZNF140) is upregulated in blood, and Small Nuclear Ribonucleoprotein U11/U12 Subunit 35 (SNRNP35) is downregulated in the dorsolateral prefrontal cortex in Military PTSD [23]. Duncan et al. investigated strong evidence of overlapping SNPs and multi-loci risk between PTSD and schizophrenia (from 20,730 individuals) via 11 genome-wide case-control molecular genetic studies [24]. Chen et al. found two loci including chr10\_6953246\_D and rs2311207 that were associated with the severity of PTSD symptoms [25]. Other genome-wide significant loci were Ankyrin Repeat Domain 55 (ANKRD55) (rs1595))2 and Zinc Finger Protein 626 (ZNF626) on chromosome 19, moreover, the ANKRD55 gene was also related to rheumatoid arthritis and psoriasis that are additionally seen in patients with PTSD [26]. Maihofer et al. also found loci on four genes: Gamma-Aminobutyric Acid Type B Receptor Subunit 1 (GABBR1), Forkhead box protein P2 (FOXP2), Family with Sequence Similarity 120A (FAM120A), and ADP Ribosylation Factor Guanine Nucleotide Exchange Factor 2 (ARFGEF2) which had genome-wide significant ( $p < 5 \times 10^{-8}$ ) from African American ancestry and the external Million Veteran's PTSD [27]. Pooler also discovered two SNPs; rs13160949 on chromosome 5 ( $p = 7.33 \times 10^{-9}$ ) and SNP rs2283877 on chromosome 22 ( $p = 2.55 \times 10^{-8}$ ) which have been firstly investigated in PTSD [28]. SNP rs267943 is located on chromosome 5 in the intron of the death-associated protein 1 (DAP1) gene had the strongest association from 396 chronic PTSD patients (Thai Tsunami survivors) and 457 controls [29]. Large-scale genome studies have identified heterogeneous and numerous SNPs and genes at multiple loci. Successful polygenic prediction models can be discovered in the future by increasing the number of current and large-scale studies. Current candidate genes associated with different brain regions and neuro-behaviors in PTSD are given in **Figure 2**.

### 3.2 Current epigenome-wide association studies

To better observe the Gene-Trauma Correlations in PTSD, epigenetic studies are also important to investigate the effects of environmental factors. Epigenome-wide

association studies (EWAS) have identified epigenetic mechanisms for PTSD due to alteration of gene expression modifications without changing the genetic code. Epigenetic studies are carried out due to traumatic memory in the hippocampal region, frontal cortex associations, and extreme fear in the limbic system. An important regulation of gene function and phenotypic expression occurring in the understanding of PTSD occurs at the level of epigenetic regulation. Epigenetic changes include DNA methylation, histone modifications, and non-coding RNAs.

Animal research generally suggests that stress-induced epigenetic modification following environmental stress may affect stress-response functions as mediated by gene expression, HPA axis. Epigenetic factors, such as DNA methylation, have been shown to modulate the influence of the environment on gene expression [30]. McNerney et al. showed that the hippocampal volume/glucocorticoid receptor (GR) gene methylation interaction is an indicator of PTSD symptoms in 67 Veteran Patients [31]. Although animal and small sample epigenetic studies give clues about multiple genes and analysis, a major challenge for these studies is controlling the wide variety of stress factors that subjects are exposed to throughout their lives, and also they must be significant in EWAS measures. Hjort et al. reported that offspring of 72% of 117 mothers with PTSD had higher cortisol levels and differential methylation in candidate genes [NR3C1, 5-Hydroxytryptamine Receptor 3A (HTR3A), and BDNF] but the level of methylation differences did not reach epigenome-wide corrected significance levels [32]. Recent Epigenome-wide meta-analysis of military and civilian PTSD reported low DNA methylation in the four CpG regions of the Aryl-hydrocarbon repressor (AHRR) from blood DNA samples of 1896 PTSD patients [33]. Epigenetic meta-analysis of civilian PTSD (545 study participants) also found differential methylations in two CpG sites including NRG1 (cg23637605) and HGS (cg19577098) [34]. Interestingly, Yang et al. conducted two new different epigenetic biotypes for PTSD (G1 and G2). The G2 biotype has been associated with an increased risk of PTSD. The G1 biotype had higher polygenic risk scores and higher DNA methylation [35]. Logue et al. reported an epigenome-wide significant association with cg19534438 in the gene G0S2 (G0/G1 switch 2) and replicated it in other military cohorts. Although cg04130728 in Carbohydrate Sulfotransferase 11 (CHST11) had no genome-wide association, was significantly associated with PTSD in brain tissue (mostly prefrontal cortex) [36]. A longitudinal epigenome-wide association study identified three epigenome-wide significant CpGs, the intergenic CpG cg05656210 and Mitotic Arrest Deficient 1 Like 1 (MAD1L1) (cg12169700) and HEXDC (cg20756026).

Interestingly, cg12169700 was located within the same linkage disequilibrium block as a recently identified PTSD-associated (rs11761270) SNP in MAD1L1 [37]. In a meta-analytical review by Wolf et al., sex and immunity were strongly associated with the age of DNA methylation. However, they noted the lack of research into the underlying biological mechanisms [38]. In a multi-ethnic meta-analysis study (30,000 PTSD cases and 170,000 controls), non-coding RNAs such as Long Intergenic Non-Protein Coding RNA 2335 (LINC02335), microRNA 5007 (MIR5007), transcribed ultra-conserved region 338 (TUC338), (Long Intergenic Non-Protein Coding RNA 2571) (LINC02571), Long Intergenic Non-Protein Coding RNA 458 (LINC00458), microRNA 1297 (MIR1297) and Long Intergenic Non-Protein Coding RNA 558 (LINC00558) and PARK2 gene are involved in dopamine regulation, is associated with PTSD [20].

These studies support epigenetic differences in those with PTSD but it is also difficult to understand how persistent epigenomic changes affect a person's response to a traumatic event, and specifically to the molecular landscape of the brain. For this

reason, it is inevitable to encounter multiple epigenetic effects in many parts of the brain and that these have not yet found their place in translational medicine. Current epigenetic studies are focused on research on blood DNA, and analysis of postmortem data from different brain regions can be used to understand how epigenetic regulation works in PTSD at a circuit, brain region, or whole-brain level [10]. Consequently, since the biological studies of PTSD are heterogeneous, it has not yet taken its place in translational medicine for a definitive diagnosis. More research with larger sample groups is needed in the biological diagnosis and treatment of PTSD.

### **3.3 Neuro-biological perspective**

#### *3.3.1 Fear and stress network in animal models*

The paucity of human studies investigating the neurobiological mechanisms of PTSD mirrored the understanding of this disease in animal models. Dysregulations of fear and stress-focused inflammatory responses detected in various brain regions have emphasized the importance of central nervous system centers that regulate fear memories (i.e., amygdala) and in response to acute or chronic stress response (i.e., the hypothalamus) since it began to be detected in PTSD patients. While animal studies continue to investigate fear-related processes for the amygdala, the medial prefrontal cortex (mPFC) and the hippocampus, interactions of the lateral (LA) (acquisition of fear and extinction concerning learning) and central nuclei (CeA) (behavioral expression of conditional fear) of the amygdala's nuclei regulating the inhibitory and excitatory effects of fear have been identified [39]. Connections between the hippocampus and the amygdala, particularly the LA, appear to be essential for the acquisition and reinforcement of contextual fear. At this point, it is thought that the somatosensorial projection of the hippocampus to the amygdala triggers contextual fear memory and may trigger fear-related learning through the LA nucleus. In addition, other evidence suggests that projections from the hippocampus to the mPFC can innervate neurons in the prelimbic (PL) and infralimbic (IL) regions that are active during fear and stress in animal models. PL and IL regions were important by creating neuronal potentials after the mPFC learned stress and conditioned fear on rodents, especially PL activity is responsible for regulating fear while its expression [40]. For instance, Richter-Levin developed a PTSD model in which animals are conditioned to pair a water-associated zero maze (WAZM) with underwater trauma that might be related to Amygdala LA and CeA nuclei. The remainder of underwater trauma rather than swimming stress, additional evidence of increased ERK phosphorylation (pERK) in the ventral dentate gyrus and basolateral amygdala [41]. Considering animal models of electrocution, this model is used more in learning and memory mechanisms than in PTSD, although it is associated with contextual reminders of trauma (associative fear) and ambiguous stimuli in a new setting (non-associative fear). Likewise, single long-term stress patterns were associated with neuronal apoptosis and dysregulation of autophagy in the hippocampus, amygdala, and prefrontal cortex (PFC), consistent with the findings in PTSD patients in terms of neurobiological background [42]. The social and psychological stressors animal model was mostly used for the PTSD behavioral measurements. In contrast, the social defeat stress (SDS) model was associated with optogenetic modulation of neuron projections to/from the ventromedial prefrontal cortex, ventral tegmental area, nucleus accumbens, and dorsal raphe nucleus in parallel with the PTSD clinic. Interestingly, amygdala-mPFC neuroadaptation was discovered in

resting-state functional magnetic resonance imaging (rsfMRI) findings from Long-Evans rats exposed to the cat collar in predator-associated animal models [43].

### *3.3.2 Neurochemical and synaptical background*

Serotonin (5-HT) is an important neurotransmitter for PTSD, targeting GABAergic neurons in response to fear-related acute stress in the amygdala, hippocampus, and ventromedial prefrontal cortex (vmPFC) regions. Clinical and animal studies have shown that symptomatic reduction associated with the use of antidepressants and/or anxiolytics in the treatment of PTSD is associated with stimulation and interaction of 5-HT<sub>1A</sub>, 5-HT<sub>1B</sub>, and 5-HT<sub>2A</sub> or 5-HT<sub>2C</sub> receptors. Sullivan et al. demonstrated positron emission tomography (PET) results of PTSD-like animals found higher 5-HT<sub>1A</sub> neuronal binding in all brain regions except the hippocampus and higher serotonin concentration in raphe nuclei compared to the healthy group [44]. Murrough et al. showed low 5-HT<sub>1B</sub> receptor density in the amygdala and anterior cingulate cortex (ACC) in PTSD patients [45]. The majority of the overactive noradrenergic activity associated with PTSD is due to the interaction of peripheral catecholamine (epinephrine, norepinephrine, and dopamine), transporter and receptor systems. In an animal and replicated study in humans, the high synaptic activity of norepinephrine (NE) in PTSD patients was detected in PFC projection areas. NPY also inhibits NE release and is found in high concentrations in the hippocampal and amygdala regions, it is associated with the projection of emotional values to memory and plays a role in the neurobiology of PTSD. Although intranasal NPY treatment reduces symptoms in many animal models of PTSD, efforts to develop NPY receptor-related pharmacological agents have failed [46]. Glutamate is an integral part of the learning, memory, and plasticity process. The glutamatergic system is studied as ionotropic and metabotropic. The PFC is transferred from the other to the amygdala and the bases of the whole brain regions to the amygdala are transmitted by glutamatergic contents and abnormal glutamate levels in PFC and N-methyl-D-aspartate (NMDA) receptor density in the hippocampus that is associated with synaptic plasticity underlying learning and memory, also have been reported in acute stress animal models. Especially metabotropic glutamate receptors have related with PTSD symptoms, high glutamate levels in the lateral temporal cortex and lower levels in ACC have been demonstrated. Research is ongoing that injection of subanesthetic doses of ketamine into rat brains increases glutamatergic neuronal activity in the PFC, which NMDA antagonists trigger learning and fear-related plasticity when examining the link between the glutamate system and dissociative symptomatology. Animal studies have shown that ketamine administration increases glutamate neurotransmitter levels and thus stimulates BDNF signaling, neurogenesis, and synaptogenesis [47]. GABA plays an important role in spatial and long-term memory, and directly in fear memory, in relation to neurogenesis in the hippocampal region. Fang et al. reported increased dysregulation of anxiety and fear memory with increased active GABAergic neurons in the CeA region of the amygdala in the single prolonged stress (SPS) animal model [48]. Behind the neurobiological mechanisms of PTSD, neuronal cell membrane damage due to stress and fear has also been researched. This damage is usually caused by oxidative stress-related free radicals (reactive oxygen species, for example, nitric oxide, glutathione, and hydrogen peroxide) damage to the cell membrane. In a recent study, Michels et al. found high higher levels of  $\gamma$ -amino butyric acid GABA and glutathione in PTSD patients via single-voxel proton magnetic resonance spectroscopy (MRS) in the dorsolateral prefrontal cortex (DLPFC) and ACC [49].

PTSD is also related to abnormal activity of the dopaminergic system, which has a mesolimbic pathway that is related to fear conditions and high plasma dopamine concentration was reported in PTSD patients. However, the dopamine metabolism of PTSD is unclear, so the genetic background is more studied. Most of these neurobiological explanations are accompanied by synaptic losses underlying PTSD. The clinical behavioral reflections (i.e., social disinhibition, apathy, attention and memory disorders, etc.) of these synaptic losses in various parts of the brain are tried to be explained. As a result of stress, disruption of intracellular signaling may result in a decrease in glutamate receptors and shrinkage of dendrite horns in postsynaptic neurons. The synaptic degeneration hypothesis is the basis of many neurodegenerative psychiatric disorders. Results of a postmortem pilot study reported that PTSD patients were immature, as the dendrites evaluated in vmPFC tissues were smaller in their spines compared to the control group [50]. In addition, neuroimaging studies conducted in PTSD were associated with volumetric and neuronal connectivity deficiencies in cortical areas and their resulting loss of cognitive functions in PTSD clinics. In particular, losses in dendritic connections are predominantly in hippocampal regions associated with neuroplasticity, resulting in chronic or acute stress-related learning disabilities. In short, the perspective on neuroplasticity has been developed by investigating neurochemical and receptor interactions in various brain regions of PTSD. In this context, antidepressants used clinically for PTSD may contribute to clinical improvement by promoting synaptic plasticity with this neurobiological infrastructure. In addition, inferences about synaptic connectivity based on neuroimaging methods are still unclear but may reveal various risks. Due to the limited knowledge about the neurobiology of PTSD, the inadequacy of the findings from animal stress models for the pathophysiology prevents us from making definite conclusions about the clarity of the applications for the clinical treatment of this disease. As a result, PTSD has been scientifically investigated with behavioral consequences related to neurobiological, genetic, and epigenetic, literature discussions continue especially in terms of both neuroscientific and clinical aspects. The importance of neurochemical, biological, and brain-regional neurologic interactions in human and animal models remains a mystery, and further studies need to unlock this mystery.

#### 4. Clinical overview of PTSD

A cluster of psychiatric symptoms that persist for more than 1 month following a trauma, causing distress or a decrease in functionality in social, occupational, or other important areas of life is called PTSD [51].

**Trauma content:** There is actual or intimidating death, serious injury, or sexual assault. The person may have experienced this event directly and witnessed it. It may be the death of a family member or friend or learning that he or she has experienced trauma with a high probability of death. Persistent encounter with the adverse consequences of traumatic events (occupational exposure).

**What happens?** Recurrent involuntary distressing memories of traumatic events and recurrent distressing dreams involving these memories. Feeling as if traumatic events are recurring, dissociative reactions. Experiencing excessive or extended distress or physiological responses at exposure to stimuli that symbolize or evoke traumatic events.

**What are the avoidance behaviors?** Efforts to avoid and avoid distressing memories, feelings, and thoughts associated with the traumatic event. Avoidance or efforts to

avoid people, places, conversations, activities, objects, and situations that may evoke distressing memories, feelings, and thoughts associated with the traumatic event.

**What is observed in cognitions and mood following the trauma?** Inability to remember the trauma, Negative beliefs, and expectations about self, others, or the world; Blaming self or others about the cause and consequences of the traumatic event, Persistent negative emotional states (e.g., fear, horror, anger, guilt, and shame); Decreased interest and participation in important activities; Feelings of detachment or alienation from others; Inability to constantly experience positive emotions (such as happiness and love).

**What are the changes in arousal and the reactions?** Verbal or non-verbal aggressive, angry behavior toward people or objects, outbursts of anger; Acting without restraint or engaging in self-destructive behavior; being alert all the time; Exaggerated startle response: It occurs in 88% of patients. Increased heart rate, greater skin conductance responses, and slower skin conductance in response to startling stimuli are well-defined findings [51, 52]. Focusing difficulties: The reason for the decrease in attention resources is the basic loss of sensory mechanisms before attention [52]. The dissociative subtype emphasizes a closure or blunted response to traumatic stressors characterized by dissociation [51–53]. The person constantly or recurrently experiences one of the following symptoms in response to the triggering factor:

1. Self-alienation (depersonalization): persistent or recurrent experiences in which the person feels detached from his mental processes or body, looking at them as if he were an outside observer (e.g, the sensation that he is in a dream; the sensation that he or his body is unreal, or that time is running slowly).
2. Unreality (derealization): persistent or recurrent experiences of feeling that the world or environment around the person is unreal or somewhat distorted).

**What is Delayed Onset PTSD?** If the symptoms are not fully appeared at least 6 months after the traumatic event (even if some symptoms start in a short time), it is called delayed-onset PTSD.

#### **4.1 Diagnosis**

In the face of severe stress, information processing is impaired, and it is not possible to resolve the traumatic event. An unintegrated traumatic experience can be easily aroused and affect daily life. Painful experiences cannot be suppressed or excluded. In experiences recorded with anxiety/fear, stimuli that stimulate one of the emotion-thought elements activate all of them. This general arousal and the unorganized cognitive processing behind it are considered as the source of symptoms, such as arousal, memory disorders, and impulsivity in PTSD. The individual who encounters the trauma first experiences confusion. This unprepared/unconditioned situation changes in the next step. By using the lived experiences before the trauma, the trauma is perceived as if it had been encountered before. The same emotional and physical reactions are given in the previous cases. This is a highly learned behavior. However, since this behavior is not suitable for the new situation, it is not an appropriate response and the answers become complex. Increasing confusion also increases anxiety. To diagnose PTSD, valid, objective/empirical methods other than previous trauma have not been defined. The diagnosis depends on the clinical interview. The

use of check-lists without recourse to clinical interviews may lead to the loss of significant clinical information that may be essential in the holistic provision of therapy and clinical care. To be diagnosed with PTSD, an adult must have all of the following for at least 1 month after a traumatic event: At least one re-experiencing symptom, one avoidance symptom, two arousals, and reactivity symptoms, and two cognition and/or mood symptoms.

The Post-traumatic Stress Disorder Checklist for DSM-5 (PCL-5) is a self-report measure to evaluate the presence and severity of PTSD symptoms (**Table 1**).

## **4.2 Factors facilitating the occurrence and persistence of PTSD**

Inability to explain and share the effects of trauma, severity, and frequency of dissociative reactions during or immediately after trauma, childhood physical abuse, genetic predisposition, family history of psychopathology and PTSD, being a woman, excess physiological response during the traumatic event, acute stress disorder and early PTSD symptoms, previous psychiatric disease history, low socioeconomic level, and low education level, temporal intensity, and duration of trauma, memory disorders, soft neurological signs, low IQ, childhood attention deficit hyperactivity disorder symptoms are the factors related with increased risk or chronicity of PTSD [52].

PTSD is associated with many comorbidities besides causing disability on its own. Major depressive disorder, generalized anxiety disorder, alcohol and substance use disorders can be listed as the main comorbid conditions. The high-stress level accompanying PTSD increases the risk for many systemic diseases, such as hypertension, diabetes, and asthma [55].

One of the important comorbidities of PTSD is a borderline personality disorder. Borderline personality disorder causes the person to become prone to experiencing traumatic events by distorting the perception of risk. On the other hand, PTSD symptoms deepen the loss of functionality associated with a personality disorder.

### *4.2.1 Acute and chronic PTSD*

If PTSD symptoms are present within 3 months following the trauma, it is defined as acute PTSD; and if symptoms persist for more than 3 months, it is defined as chronic PTSD [55].

### *4.2.2 Complex PTSD*

PTSD is single-event trauma from traumatic experiences, such as rape, physical assault, or war. However, the traumatic event might be prolonged chronic victimization, such as interpersonal violence. Over time, chronic traumatization, often of an interpersonal nature, such as multiple and/or long-term developmentally negative traumatic events, came to be used to describe the term “complex trauma” [56].

### *4.2.3 Course and prognosis*

The clinical course and outcome of PTSD vary depending on the factors before, during, and after the trauma. The nature of the symptoms observed after trauma,



In the past month, how much were you bothered by:	Not at all	A little bit	Moderately	Quite a bit	Extremely
1. Repeated, disturbing, and unwanted memories of the stressful experience?	0	1	2	3	4
2. Repeated, disturbing dreams of the stressful experience?	0	1	2	3	4
3. Suddenly feeling or acting as if the stressful experience were actually happening again?	0	1	2	3	4
4. Feeling very upset when something reminded you of the stressful experience?	0	1	2	3	4
5. Having strong physical reactions when something reminded you of the stressful experience?	0	1	2	3	4
6. Avoiding memories, thoughts, or feelings related to the stressful experience?	0	1	2	3	4
7. Avoiding external reminders of the stressful experience?	0	1	2	3	4
8. Trouble remembering important parts of the stressful experience?	0	1	2	3	4
9. Having strong negative beliefs about yourself, other people, or the world?	0	1	2	3	4
10. Blaming yourself or someone else for the stressful experience or what happened after it?	0	1	2	3	4
11. Having strong negative feelings, such as fear, horror, anger, guilt, or shame?	0	1	2	3	4
12. Loss of interest in activities that you used to enjoy?	0	1	2	3	4
13. Feeling distant or cut off from other people?	0	1	2	3	4
14. Trouble experiencing positive feelings	0	1	2	3	4
15. Irritable behavior, angry outbursts, or acting aggressively?	0	1	2	3	4
16. Taking too many risks or doing things that could cause you harm?	0	1	2	3	4
17. Being "super alert" or watchful or on guard?	0	1	2	3	4
18. Feeling jumpy or easily startled?	0	1	2	3	4
19. Having difficulty concentrating?	0	1	2	3	4
20. Trouble falling or staying asleep?	0	1	2	3	4
<i>Criterion B (1–5)—at least one ≥2 Criterion C (6–7)—at least one ≥2 Criterion D (8–14)—at least one ≥2 Criterion E (15–20)—at least one ≥2.</i>					
<i>Mild 0–20; Moderate 20–40; Severe 40–60; Extreme 60–80.</i>					

**Table 1.**  
The PTSD Checklist for DSM-5 (PCL-5) [54].

the prognosis of the disease, or the information obtained from follow-up studies conducted at different periods makes it difficult to define a specific clinical situation for the course of the disease. PTSD starts when trauma is encountered or within the next few years, symptoms increase in the next few years and continue by drawing a plateau. Symptoms may fluctuate over time and intensify during stressful periods. Approximately 30% of patients show complete improvement, 60% have mild to moderate symptoms, and 10% have symptoms that remain unchanged or worsen. It is common for those who benefit from treatment to reappear after years of being exposed to a serious stressor.

### **4.3 Neuroimagination studies**

Several neuroimaging studies have been implemented to investigate the pathophysiology of PTSD. Some symptoms associated with PTSD are related to changes in brain structure and function [57]. Brain regions implicated in the development of PTSD include the hippocampus, amygdala, and medial prefrontal cortex [58].

Advanced neuroimaging techniques contributed to our understanding of the possible pathophysiology of PTSD. The results of neuroimaging studies point to the importance of the hippocampus in PTSD. Exposure to chronic stress results in disturbances in memory function and neural damage to the hippocampus. The HPA axis controls stress response in the body by producing cortisol. The neural damage might be related to high levels of glucocorticoids, changes in serotonergic function, inhibition of neurogenesis in the hippocampus, or inhibition of brain-derived neurotrophic factors [59].

The magnetic resonance imaging (MRI) studies in PTSD consistently revealed reduced hippocampal and inferior temporal cortex volumes. The decreased volume of the inferior temporal cortex was inversely correlated with anxiety levels in PTSD [60]. Other neural structures often implicated in the pathophysiology of PTSD include the amygdala and prefrontal cortex. Amygdala is the integrative center for emotions, emotional behavior, and motivation. Functional magnetic resonance imaging (fMRI) studies with PTSD patients present increased activity in the amygdala in response to threat stimuli compared [57]. However, investigation of a large sample of nearly a hundred PTSD patients was characterized by reduced amygdala volumes [61].

Several methods have been used to study the pathophysiology of PTSD. Many neural networks and pathways that play a role in PTSD have been revealed, and these pathways can be studied in-depth due to the advances in techniques for neuroimaging.

### **4.4 PTSD treatment strategies**

PTSD is associated with functional impairment and comorbidity. Therefore, early diagnosis and appropriate treatment are essential in PTSD. Existing treatment guidelines for the treatment of PTSD disorder generally aim to—reduce PTSD symptoms or achieve remission, loss of diagnosis, treatment of comorbid medical and psychiatric diseases, improvement of quality of life, correction of impairment in functional areas, return to work or duties. Treatment guidelines include psychological, pharmacological, and neuro-modulatory treatments [55]. However, a major limitation must be recognized: the current therapies described for PTSD are based on western cultures and modern technologies, and many of these approaches do not easily apply to rural communities in low- and middle-income countries. Clinicians

or psychotherapists should, therefore, adopt psychotherapeutic strategies that are appropriate to the cultures in which they work.

#### **4.5 Pharmacological interventions**

It includes the use of various psychotropic drugs to target the core symptoms of PTSD. Medications that target key symptoms of PTSD, including intrusions, avoidance, negative changes in cognition and mood, and changes in arousal and responsiveness, include selective serotonin reuptake inhibitors, serotonin and norepinephrine reuptake inhibitors, atypical antipsychotics,  $\beta$ -blockers, and sleep medications (e.g.,  $\alpha$ -blockers, nabilone, hypnotics). Pharmacological treatments include antidepressants (e.g., sertraline), antipsychotics (e.g., risperidone), anticonvulsants (e.g., topiramate), hypnotics (e.g., zopiclone), and mood stabilizers (e.g., lithium), mood stabilizers; adrenergic agents; benzodiazepines; and other pharmacological agents [55].

Selective serotonin reuptake inhibitor (SSRI) stands out among pharmacological treatments because it is effective in most PTSD symptoms, easy to use, and has low side-effect profiles. They are the most valid and widely used drugs for the treatment of re-experiencing, avoidance, emotional blunting, and hyperarousal symptoms. SSRIs have been found to be effective in PTSD in double-blind, placebo-controlled randomized trials.

Mood stabilizers have the effect of reducing the sensitization of the limbic system, which develops in the first weeks and months after the traumatic event. Lamotrigine was found to be effective in re-experiencing and avoiding symptoms of PTSD. Studies are reporting that lithium, valproic acid, carbamazepine, oxcarbazepine, and gabapentin are effective. It has been found that propranolol, a beta-blocker, has positive effects on nighttime nightmares, remembering repetitive anxiety-provoking situations, jumping, sleep disturbances, and self-esteem.

#### **4.6 Psychological interventions**

Psychological treatments for PTSD are mainly in the form of cognitive-behavioral therapy. Cognitive processing therapy, trauma-focused cognitive behavioral therapy, and long-term exposure are largely within the framework of cognitive and behavioral therapy. Among the Cognitive Behavioral Therapies, especially Exposure Therapy and Systematic Desensitization techniques are successful in trauma treatment. In both techniques, it is aimed to desensitize the person and gradually reduce the traumatic effects by enabling the person to face the images and situations related to the trauma in a systematic and controlled manner.

Interpersonal psychotherapy was also found to be promising in recent research. Interpersonal psychotherapy is a form of attachment-based therapy. The patient is shown his / her own needs, and the support he/she needs. The client is taught how to get the support he/she needs from those around him/her. Thus, he/she will be able to recognize the attachment needs that have become active due to the trauma and will be able to provide appropriate social support for himself/herself.

During the traumatic event, the individual is exposed to intense fear and anxiety. The traumatic event cannot be processed by the brain as it should. The traumatic memory, which cannot be processed adequately and appropriately, disturbs the individual over time. Eye Movement Desensitization and Reprocessing (EMDR), developed by Shapiro and used in the treatment of PTSD, activates both halves of the

brain through two-way eye stimulation and ensures healthy processing of the traumatic memory. With the EMDR method, the traumatic memory with high emotional intensity for the individual loses its vitality and the individual's hypersensitivity disappears.

#### **4.7 Neuromodulation interventions**

Neuro-modulatory treatments are viable treatment options for many psychiatric disorders. After U.S. Food and Drug Administration (FDA) approval of transcranial magnetic stimulation (TMS) as an option for treating depression, researchers also tried to use repetitive transcranial magnetic stimulation (rTMS) for depressive symptoms of PTSD [62]. rTMS and transcranial direct current stimulation (tDCS) are frequently employed as adjunctive options to pharmacotherapy for the treatment of several psychiatric disorders including PTSD. Several studies also investigated the potential of rTMS and tDCS in the treatment of PTSD to decrease the overactivity of the amygdala. The results of the studies revealed that both high-frequency and low-frequency rTMS can significantly reduce PTSD symptoms. rTMS may, therefore, be an effective add-on treatment option for treatment-resistant PTSD [63].

Deep TMS is a drug-free and non-surgical intervention, it does not require anesthesia. During the application, the patient is awake and conscious. The target area in the brain is physically stimulated by sending magnetic pulses under the skull with a mechanism placed in the skull. Thus, neurons working with electrical activity are activated by magnetic stimulation. It has been reported that recalling traumatic memories with activation and talking about it in the presence of an expert significantly reduces the burden of trauma.

The treatment method, which is based on the electrical processing of the data of the brain and bodywork and presenting it as feedback to the person, is called neurobiofeedback training. Thus, the patient learns to consciously control his/her own brain activity and bodily functions, such as breathing, muscle tension, and heart rate. Promising results are obtained in PTSD with the use of neurobiofeedback together with pharmacotherapy and psychotherapies.

### **5. Neuro-developmental perspective of PTSD**

Human development starts from conception and continues until the end of life. Along this developmental pathway, earlier years witness the highest speed and the most complex changes. Moreover, recent neurological research studies have concluded that the human brain is the fastest developing organ in the first years of life. Not only physically that the child's brain reaches its almost full size by age four, but also by making almost 700 million snaps connections every second completing at least 80% of its functioning capacity. This makes neurological development the most decisive developmental process in the early years [64]. It also points to the fact that ACE become the major cause of long-term emotional problems, including PTSD.

Worldwide, children are often exposed to serious traumatic events, such as war, displacement, famine, and violence, that all disrupt a child's secure family structure and lead to long-term stress. Mental health problems affect around 10–20% of the child population worldwide [65]. Trauma is common in children and adolescents

and may lead to PTSD. PTSD refers to maladaptive responses to at least one severe, threatening event (serious injury, threatened death, or sexual violence) by DSM-5, and the stress response, emotion regulation problems, and threat learning are indicated as common diagnostic symptoms of PTSD are; intrusion, avoidance, negative alterations in cognition and mood and arousal [66]. These cognitive symptoms have led researchers to examine the neurodevelopmental dimension of PTSD in the light of neuroscience studies.

According to the recent neurodevelopmental research results psychological as well as physiological responses to traumatic events such as being unable to bond with primary caregiver might lead to trauma having a long-term neurological impact on a child's psycho-social development and neurological functions [67, 68]. Such psychological problems, referred to as PTSD, are often associated with multiple psycho-social problems ranging from delinquency, poor academic performance to, alcohol and substance abuse, and even to suicidal attempts. Moreover, children exposed to traumatic events will have emotional, social, and physical developmental problems later in life [69]. It was observed that children exposed to traumatic events performed lower performance on cognitive and intellectual abilities than the children without a diagnosis of PTSD [70]. Besides, according to research results verbal and nonverbal intellectual capacity, mean IQ scores, language delay, sensory processing, memory, aggressive behavior, visual processing, affect, and behavior problems can be seen in children [71].

Neuroimaging research with PTSD indicates both functional and structural abnormalities in the front limbic area responsible for emotion regulation and threat processing. Such as decreased gray matter volume in ventromedial prefrontal and dorsal anterior cingulate cortex seen in structural analyses and hyperactivation of the insula, amygdala, and mid anterior cingulate cortex, smaller frontal-occipital circumference seen in functional analyses [72, 73]. Both structural and functional differences are also observed in the prefrontal cortex and limbic system (hippocampus and amygdala). Therefore, memory, emotion and excite function problems may accord exposure to stress [74].

## **5.1 Brain development**

Most recent scientific studies on brain development reveal the fact that early experiences shape the architecture of a child's brain having a long-term impact on a child's social and emotional well-being [1]. A child's social, cognitive, and emotional development is heavily dependent on the quality of interaction between child and "significant" adult (e.g., mother, father. Caregiver. whoever s/he is bonded with). Neurodevelopmental studies claim that bonding problems and parenting inconsistencies might cause long-term mental health problems [75]. Although, the first years are critical for life-long success and healthy physical and mental development for the rest of their life, having a baby makes a significant change in the lives of parents. Lower stress of families is associated with lower stress levels and normal brain development in children [76].

Neurological development of the brain does not take place in a vacuum nor by itself. It is highly dependent on external stimulations and interactions. In other words, a child's early experiences shape the brain architecture from the beginning [1] and leave footprints that last a lifetime. Brain developmental functions also have critical moments providing "windows of opportunities" or challenges for specific developmental pathways (**Figure 3**).

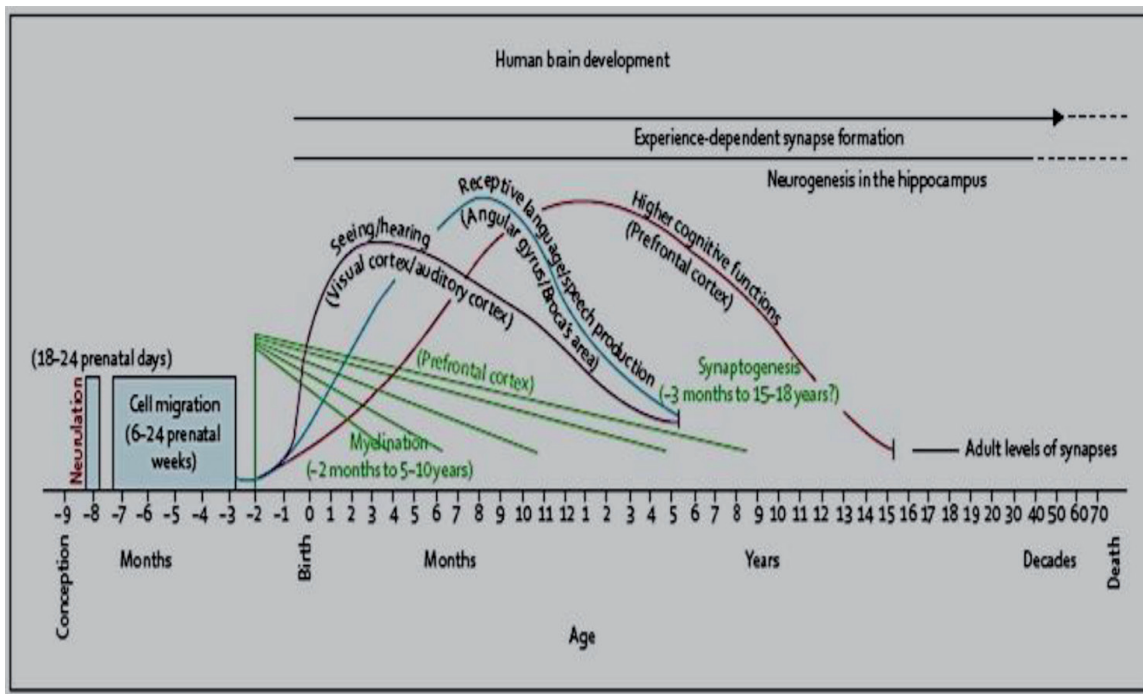
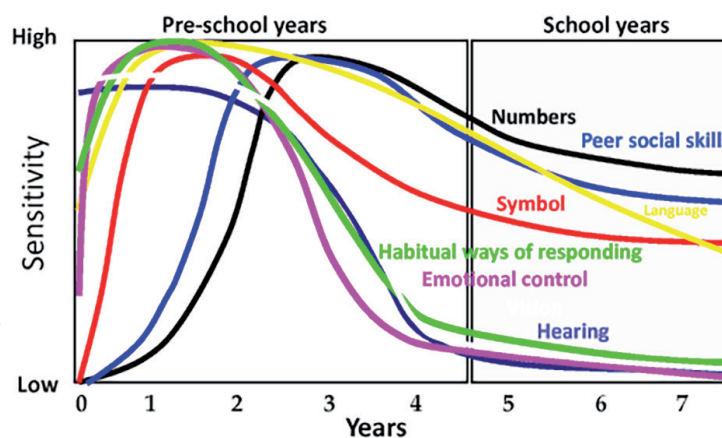


Figure 3.  
 Human brain development [1].

### Sensitive Periods in Early Brain Development



Graph developed by Council for Early Child Development (ref: Nash, 1997; Early Years Study, 1999; Shonkoff, 2000.)

Figure 4.  
 Sensitive periods in early brain development [77].

Among all critical and sensitive periods, the first 3 years of life seem to be the most critical for emotional control responding to positive as well as adverse experiences as demonstrated in **Figure 4** [77].

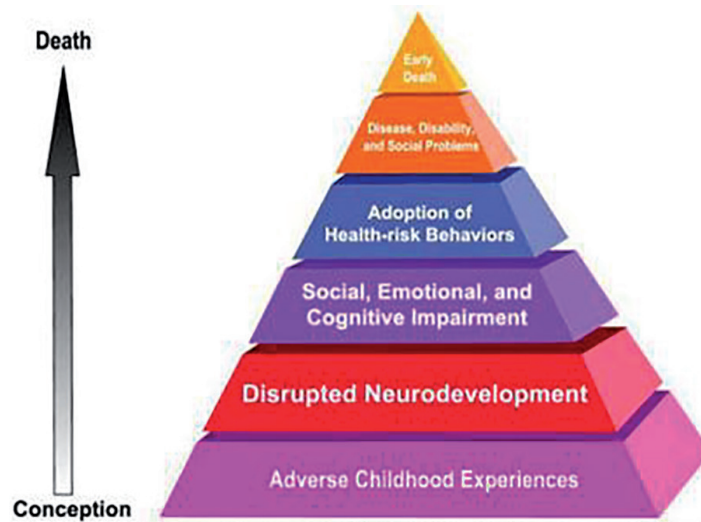
Human brain development takes more years to reach maturity compared to other species. Although this long way to maturity is adaptive for the human species, the adolescent period is the second most important part of this journey of maturity because of its adaptive values of plasticity [78]. Therefore, the adolescent period is also sensitive and critical for stress factors. According to research about brain development, it claimed that exposure time to stress plays an important role in brain structure, the adolescent stage is more vulnerable to stress compared to the adulthood stage of development.

## 5.2 PTSD as a result of adverse childhood experiences (ACE)

Adverse childhood experiences (ACE), as demonstrated in **Figure 5**, can disrupt neurodevelopment causing social, emotional, and cognitive impairment in children that lead to the adoption of health-risk behavior leading to adulthood illnesses including cardiovascular diseases, sleep disorders, obesity, and the like [79].

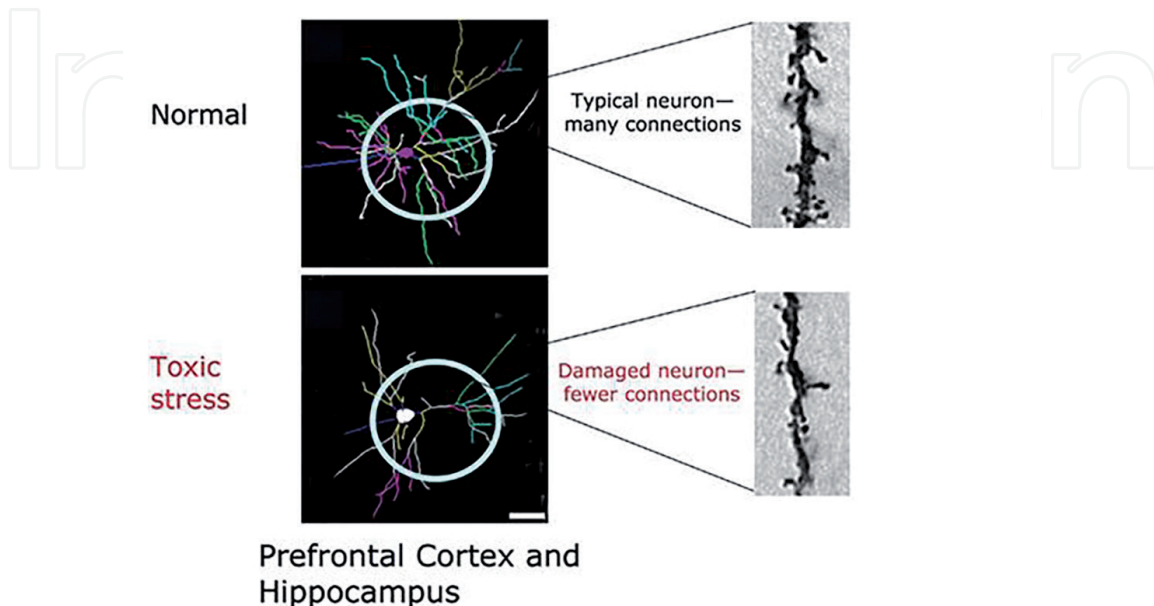
Toxic stress is known as one of the most ACE in leaving almost irreversible damage in a child's brain as given in **Figure 6**.

Weems's model, based on evolutionary presentative, emphasizes that the stress may lead to delay, accelerate, or prolonged developmental process according to the adaptive importance of these changes. According to the model, the developmental



**Figure 5.** Mechanisms by which ACE influences health and well-being throughout the lifespan [79].

## Persistent Stress Changes Brain Architecture



**Figure 6.** Persistent stress changes brain architecture [15].

timing of stress exposure plays an important role in how the brain responds to the stressor, also they claimed that age and maturation are critical for amygdala volumes, which are responsible for emotion regulation [80]. Also, it is known that the prefrontal cortex and amygdala connection develop through childhood to adulthood and become stronger [79].

Coping with stress, if the stress is moderate level, is important for the healthy development of children and adolescents. In contrast, if the stress level is high, long term, and hard to cope with it, may cause damage to brain structural development, which is called toxic stress. Toxic stress defines as the activation of the human body's stress reaction system frequently and hardly reacting to long-term stressful stimuli or long-term activation of stress response to a stressful event with a failure of the human body. Toxic stress may cause prolonged physiologic and psychologic abnormalities, such as organ dysfunction or brain functional abnormalities. Abuse, violence, neglect, food scarcity are common types of toxic stress sources All these sources can also be considered as risk factors in the development of PTSD.

Toxic stress also negatively affects the neuroendocrine-immune system. Such abnormalities on cortisol levels might also be observed. Moreover, toxic stress preventing neural connections can have an adverse impact on brain architecture that impacts planning, reasoning, emotional and behavioral control areas. It is also claimed that these responses may play a role in psychopathological disorders including psychosis, depression, and PTSD [81].

Parenting style is considered as one of the major factors having an impact on the psycho-social development of young children as well as adolescents. It is known that parenting style plays an important role in the etiology of psychopathology in children and adolescents, both as a genetic factor and an environmental factor from a transgenerational perspective [82]. Parenting style can also be a protective factor when it has a positive role, whereas when it has a negative role, it is considered as a risk factor for the development of psychopathologies, such as depression, anxiety, obsessive-compulsive disorder, and schizophrenia. Also, according to evocative gene-environment correlations, symptoms of adolescent psychological disorders may affect parental psychological status [83]. Further, some studies have found that attachment styles are associated with both psychopathological and cognitive problems in adolescents, such as psychosomatic complaints, anxiety, verbal aggression, attention-seeking behavior, and thinking problems [84]. In conclusion, exposure to the traumatic event, negative parenting attitudes, or negative attachments may also have a negative effect on brain development.

All these developmental risk factors, evaluated from an evolutionary perspective, adaptational sensitivity in both young children and adolescents, from the perspective of toxic stress, exposure to toxic stress in the early developmental period, from the perspective of family attitudes, children growing up in an overprotective or authoritarian and inconsistent family structure, when faced with traumatic stress, are all considered risk factors for developing PTSD.

Developmental models of PTSD argue that an individual's biological, cognitive, and psycho-social characteristics are more decisive factors than the traumatic event itself in the emergence of PTSD. Such PTSD models, pointing to the importance of developmental processes of each individual argue that the brain will be affected by the traumatic event in a neurodevelopmental state. Likewise, it is thought that the neurodevelopmental state of the individual's brain is an important determinant in the development of PTSD reacting to the traumatic event.



According to neurodevelopmental research, the duration, intensity, timing of a traumatic event is important on how the brain and development will affect the event. Also, genetic vulnerabilities may affect the damage size, such as a genetically vulnerable child may develop psychopathology while a hardier child may not [73].

Preventing, early intervention and rehabilitation techniques are important for protecting the child from PTSD effects, especially neurodevelopmental damages. If possible, preventing children from possible traumatic experiences should be the first step. Later, for children who have been exposed to the traumatic event, early intervention and immediate action to remove the child from the traumatic environment should be the second step, finally, rehabilitation, such as psycho-social support mechanisms and programs, plays an important role in preventing neurodevelopmental damages. As a promising research Nelson et al. [85], studied with 136 abandoned children to examine their brain development, cognitive functioning, social and physical growth with 12 years' study. According to results, compared with children in foster care, the institutionalized children showed severe impairment in IQ and brain development, along with psychological disorders [85]. This study confirms that even if the child is exposed to a traumatic event, immediate action to remove the child from the traumatic environment afterward supports the reduction of traumatic effects and abnormal brain development.

## **6. New paradigms and changes in information society and PTSD**

### **6.1 Transformations of society and media**

The hegemony of the digital culture has changed and transformed the structure of society dramatically. The characteristics of those changes have a snowball effect on the layers of society. It is presumed that when people change, so are immediate consequences, and impacts are followed. The transformation of society ultimately dominates the way we think, feel, behave, and the way we use language. Nevertheless, the concepts, terminologies, and issues that are used in the present academia are now much more blurred compared to the clarity of the old modern days. Hence, creating a common and mutual language is plausible to comprehend the mental problems and the society with inclusive perspectives and views of digital habitus. Keeping all these digital reformations in mind, scholars try hard to keep pace with stunning developments. It seems inevitable that all academic references and mental archives should be re-read and digested accordingly.

For the well-being of public health, traumas should not be confined to individual levels. People in all walks of life are likely to suffer from PTSD. On a much broader scale, to help the victims of PTSD, diagnosis and therapy alternatives can be presented to society as a whole. If new diagnostic and therapeutic approaches regarding PTSD cases focus solely on personal levels, psychiatrists can easily fall into the trap by ignoring its widespread effect on society and on the culture of the individual that s/he lives. It is, therefore, significant to design unique therapy alternatives for PTSD patients by taking the cultural, social, and geographical facts into account. In addition to these alternatives, the experts should focus on cultural differences as well as individual differences. For example, the socio-cultural development in Turkey is in an eclectic form where the transition process from the traditional to the modern and postmodern period does not work in line. For this reason, the specific conditions of Turkish society and culture should be taken into account in social interpretations of individual problems.

Owing to the particular reasons mentioned above, social psychiatry should work for the benefit of the whole society counting PTSD patients. It is a challenging standpoint to offer a therapy that can work for the whole society. In the PTSD context, if social psychiatry utilizes cutting-edge approaches by wisely utilizing the technological advances of the cyber era, the mental and social well-being of society can be reached. It is, therefore, recommended to use new communication media, such as webinars, supervisions via the Internet, mental health apps, and developing “mind wares”. These new discourses will automatically maximize the impact of social psychiatry particularly for PTSD cases [86].

## **6.2 Media and trauma**

Media and television have a dangerous role as they can easily traumatize and continually retraumatize people with the vivid and graphic and horrifying pictures and videos that are broadcast on newscasts. They usually warn before they expose people pictures and videos full of violence. This is media violence. This warning cannot prevent people from trauma. So, they keep traumatizing people visually. Almost less than five decades before the digital reformation took place, George Gerbner, proposed in his Cultivation Theory that television negatively affect the mind of people [87]. TV news and programs usually exaggerate the violence of the outside world and depict the whole world as a bad, mad and cruel one. When particularly old people are exposed to media too long, they start to perceive the world as full of traumas, chaos, and tragedies. This is called Mean World Syndrome. As a result of that syndrome, three types of behavior or mood appeared: aggressive, depressive, and escaping. Unlike the world in which Gerbner lived, mass media today diversify with new technological opportunities within digital systems and affect the social sphere in a much shorter time and on a larger scale. While it is necessary to find an extra effort and time to encounter the mass media of the modern era, such as watching television, in the postmodern era, digital forms of communication tools have infiltrated the fabric of daily life. Now, without a special time requirement, individuals encounter multi-media messages in any part of daily life.

This infiltration of media in postmodern culture leaves us nowhere to escape. The risks of trauma are now scattered everywhere. Simmel associates the identity and mental problems of modern society with the over stimulus in the city life in his article *Metropolis and Mental Life* [88]. Whereas, in the postmodern age, we face a shift of paradigms as the rules of the game had changed. In the postindustrial society, identity and mental problems have different characteristics. Bernard Stiegler has correlated emotional problems with the digital revolution, as an inundation that carries with traumas, or at the very least the tensions.

The tsunami-like transformation of the communication landscape caused the sharp transformation from modern life to Information society that largely affected identity formation as the fragmented and episodic one [89]. People, for example, lost their feelings of belonging, and even their authentic ideology amidst uncertainty, which can be defined as social autism. The reasons highlighted here doubled the burden of psychiatrists in the post-capitalist/post-truth era. Conventional therapies, therefore, can miss embracing the overwhelming realities that those people experience.

## **6.3 Searching new discourses in digital therapies for PTSD**

Digital media are now full of spiritual platforms providing solutions for people in trouble or suffering psychological disorders. There appeared new types of

narration that quickly become popular in cyberspace. Some approaches like collective healing streams which offer to heal the past wounds of the society may have genuine reasons and philosophy behind them [90]. However, they are not free from problems. Disinformation is ubiquitous and misleading. Therapy-based applications and contents addressing PTSD are largely held in esoteric and unprofessional ways. Unfortunately, their popularity can be hazardous and confusing regarding a large number of PTSD cases.

When the whole world was haunted by the chaotic and sudden emergence of the COVID-19 pandemic, people were not ready to cope with this storm fully. This chaos and crisis have their unique problems. Many people suffer from that not only because they try to avoid being infected but also to escape from the harsh and odd situations that affected them mentally. It seems that it is hard to find isolation from this undefined fear atmosphere for everyone. People from all walks of life have suffered from the burden of digital works, ambiguity, and economic recession. As Chul Han discusses in *The Scent of Time*, the perception of time and its management worsened the situation [91]. While digital violence and crimes have been increasing, people have been pushed to live by the strict structures of discipline society and capitalism [92].

In these dark times, Turkish popular media resort to products that contain psychological consultancy contents. Here we come across a newly fledgling genre about digital media that works for Psychological Consultancy [93]. Some digital therapy contents, for example, act like postmodern witchcraft as they underestimate the real role of designing a therapy. These forms, which are grown in popular culture, also have an important function in maintaining the capitalist system. Expertise knowledge in the improvement of life created a new field outside of scientific knowledge and created a mystical field in postmodern culture. In places where modernization of society does not develop well, these forms of knowledge replace rational and scientific knowledge and present themselves in a form of reality.

Therefore, it takes professional psychiatrists to end this widespread cacophony and reverse the negative situation to positive ones [94]. If wisely used, digitalization gives several opportunities to implement new techniques regarding PTSD cases. Digital therapy sessions, awareness-raising activities can reach millions of people online. People can take online therapies or watch videos related to their problems. This is the natural outcome of the advent of the Internet and changing communication environments that widen the possibilities of civil protection services and emergencies [95].

As stated earlier in the study, changes in society challenge psychiatrists to diagnose and offer therapy by adding new perspectives to their conventional practices. Psychiatrists' role has changed as they now have to adapt these changes into their therapeutic diagnosis to follow the recent changes in society and find proper treatment. The pros of advanced technology should be used for the benefit of the people on the whole. It is true, digitalization challenges the psychiatrist to take PTSD cases by taking and addressing full sides of problems but digital narration opportunities allow them to lead fresh approaches in their discourse. Bennegadi, here, throws further questions: "Does the presence of a digital tool complicate the notion of empathy? Is confidentiality guaranteed? Is nonverbal language considered? And finally, how do we define the role of digital as a transitional element in the relationship?" [86]. The frame of these questions indicates that this is just the beginning of a new era. The methodology and their discourse that are shaped by the hegemony of the digital habitus must be arranged to offer the maximum of possibilities to the citizens in the art and the way of preserving their well-being.

## 6.4 Attachment theory and PTSD in digital habitus

Social media has been described as a double-edged sword. Beck's suggestions about The Risk Society have lost their validity. It is apparent that the types of risks that modern societies experience should be adapted to the de facto of the Information society. The risks are now scattered everywhere. There is no secure shelter in Information Age where people hide and protect themselves. The possibility of surviving without experiencing trauma is now a dream [96].

Bourdieu as the father of the term habitus declared that human beings are conditioned by their habitus [97]. Information society now created the term digital habitus where people now have to learn how to express constellations of new or previously unrecognized feelings, sensations, thoughts, and traumas to build an emotional repertoire, which assists them in emotional regulation. This is important because naming and expressing new experiences allows people to claim convenient agency in dealing with them.

To survive in the cyber world, we need to ensure that are we corresponding. Emotional and social attachments can create our little hells. Therefore, Attachment Theory can be reread to prevent dangerous attachments that can take place in digital areas. Attachment theory signifies the importance of our social interactions among trusted ones. In a similar vein, social baseline theory argues that social relationships play an important role in the well-being of society. Attachment theory argues that many people internalize attachment representations, such that mental representations of attachment figures acquire comparable soothing effects. So we humans learn from an early age to seek refuge in trusted others in times of need; caregivers provide us with food, nurture, and protection when we are vulnerable [98].

## 6.5 Cyber violence, dark web, and new emotional repertoire

Susan Sonntag underlines the importance of a basic human trait, such as empathy, in her book *On Regarding the Pain of Others*. Exposing visual violence in a voyeuristic gaze causes us to lose the feelings of empathy for others [99]. Unfortunately, indifference and showing no empathy toward the victims are now the new realities of society. The aestheticizing of the violence can decrease the real emotional reaction can cause paralysis of emotion. Here Baudrillard defines excessive and improper uses of violent images that cause "implosion of meaning." It is a kind of aphasia. This feeling of insensitivity, which is the general loss of meaning experienced in the social sphere, is read by Baudrillard as a feature of mass society. While the concept of the mass in the Frankfurt School was considered as a passive entity in communication studies, Baudrillard transformed the mass into an entity that could not be influenced by any information and on which no information could create effect [100].

While the general loss of meaning in the social field drags the mass into a sense of numbness, the tragedy, chaos, or violence experienced in the social field disappears without the slightest effect on the individual. This seemingly numbing state allows the legitimation and spread of violence as an impulse on every individual who constitutes the social sphere. In the social sphere, the individual gradually becomes the dynamo of a mechanism that produces or legitimizes violence, while the normative sphere of the society recedes. Reading, interpreting, and using technical tools from a critical perspective without being fetishized in the digital society will protect individuals from the possible negative effects of these tools.

When the September 11, 2001 attacks happened in New York, the collapse of the buildings and their visuals shocked the whole world. People who watched the planes plunging into the towers witnessed the commencement of a new era. This new era showed the vulnerability of the security of systems. Here, as explained above by Baudrillard's terminology implosion of meaning, people who watched the shootings of collapsing and jumping people from the towers are appalled by the heaviness of the tragedy. They were emotionally paralyzed. Feeling acedia or aphasia are two similar traumatic outcomes when exposed to violent visuals. As we can see, continual exposure to a persisting stress source or income has created new types of patients who have PTSD. To put in other words, PTSD in the postmodern society is the outcome of the post-modern city life we all witnessed.

To make things worse, capitalism eliminates the possibility of building a healthy community to get rid of the troubles. The fact that postmodern urban life forms are generally shaped in the axis of the dynamics of the capitalist system. As a result, elements such as competition, individualism, and hedonism become more visible in the cultural field. In this spatial practice, where the social collectivity is replaced by the hedonistic tendencies of the consumption culture, interpersonal communication forms are gradually falling apart [101]. In addition to the aforementioned paralysis of emotions caused by postmodern cultural, social, and economic problems, the COVID-19 pandemic time has appeared as a chaotic example for the present situation. People have begun to struggle with so many things with many new unknown emotions and trauma types that emerged on social media platforms. These unknown terminologies now become a big part of our emotional repertoire. They are multifarious such as thumbnailing, trolling, body shaming, gaslighting, cancel culture or de-platforming, #metoo campaign, toxic masculinity, stress to feed on social media, hashtagging posts, feeling acedia, losing the spatial sense in virtual and augmented reality, being immersed in the metaverse, having been cringed on social media. The list can be endless when it comes to the new phobia types of which may not be classified under the DSM-5 criteria yet to be accepted as legitimate trauma types [102]. Digital habitus is considered as the underlying reason of the problem cause or triggers many new phobias, such as netless phobia, fear of missing out (FOMO), nomophobia, or the feeling of being stalked by unknown gazes due to synoptic surveillance. The dark side of the digital world as we can see here turn many ordinary people's life into hell. Cyber violence is reinforced by professional criminals who used random pictures of people in deep fake porno. It takes a second to be the victim of deep fake porno. Hence, so-called naïve nudity trend-sending naked pictures to your beloved can be a real trauma for many young people. Similarly, some rather new terminologies, such as crowdsourcing or hive mind activities, can serve just for big data and nobody can guess the real outcome of those digital practices.

It follows that toward a more peaceful and healthy society, we need people who are emotionally healthy as well as mature. For many PTSD patients, time freezes. The past events occasionally haunt them. A similar haunting process can be seen in the Virtual Reality universe. The artistic use of visuals and space in VR technologies can cause new trauma in which time has been expanded as if it is a "duree" experience [103]. Aestheticizing the violence in those arts can trigger fears and worsen the situation. So all these new encounters have brought a new level of violence types in the new media. As it has been presented, each new media and innovation comes with its drawbacks.

Reversing this more positively, media content and AI should collaborate. Psychiatrists, therefore, here must carefully read the signs of digital habitus problems

to address the issue by implementing new technologies wisely. One must be aware of the fact that not every innovation has positive effects on society. Reading, interpreting, and using technical tools adopting a critical perspective and without fetishizing things in the digital society will protect individuals from the possible negative effects of these tools.

## **6.6 Counter activities to prevent society from cyber violence**

Prevention of PTSD should start by preventing and monitoring the activities of people on Digital Media. What we need is counter activities to stop the evil deeds and narrations all over the world. A narcissist can disguise himself/herself in social media groups with a hidden agenda camouflaging in amicable chats, can easily take advantage of PTSD people's vulnerability. They can easily hunt a new dependent person as his/her victim. A cyber sadist invites his/her victim in his digital tower-like Marquis de Sade captured and imprisoned his victims in his tower. Similarly, illegal groups can find their supporters from this digital bowl. Tracking the digital activities of potential criminals, sadists or schizophrenics can save many people's life. However, all these counter activities are hot topics and controversial as they are against the freedom of communication and privacy. These surveillance and counter-violence discussions are beyond the scope of this study. Pursuing digital technologies to find new ways to reach PTSD can be incomplete if people are not warned against the demonic uses of social media. As explained above, some narcissist people can find their victims via social media platforms. New media here not only digitalize the mindset of people but also offer new facets of crime, violence, and even terrorism. Now cybercrimes, digital violence, data mining, fake accounts, identity stealing, illicit money flows, the resonance of terrorists can cause new traumas in the cyber world. Counter activities to raise awareness of the malicious use of social media is surpassing the job description of social psychiatrists. To fight the root of the problems sometimes can minimize the number of people who would be traumatized in the cyber world. To create a peaceful society, one has a peaceful mindset that is free from restlessness, such as exaggerated startle responses, flashbacks, nightmares, and hypervigilance. Genuine and meaningful communication to lead a meaningful life should not be taken for granted. To provide sensitivity, it is necessary to construct a collective language in the social field by professionals working in the field of communication science and experts in the field of psychology. They should work together to build a new language that prevents social polarization. In addition to the polarized ideologies, digital media have also problematic discourse and contents. Aggressive language in social media is contagious. People who are traumatized, victimized, killed, raped, exiled had not escaped the same pattern of violent discourse. The wild and violent human nature has necessitated certain psychological support systems to cure the victims of tragic events. It is assumed that no society is immune from traumas, and postmodern societies are no exception. Depression, unhappiness, and the symptoms of burnout are highly correlated with the traumatic issues within the scope of postmodern society [91].

## **7. Conclusion**

As stated above, this study brings Information Age traumas and psychological disorders to the forefront. First trying to find an answer to the questions that why and what types of traumas happen in the Information Age. And, secondly, what changes

should be implemented in the discourse and the methodology of the psychiatrists. Trying to answer these questions can provide us a chance not only to define the problem accurately but also to seek valid discourses for the psychiatrists to be used in PTSD cases, particularly in postmodern society. Within the scope of the study, collecting and processing neuroimaging data or utilizing the latest AI techniques can be given as an example of designing new diagnostic and therapeutic methods that rely on neurobiological dimensions. In addition to these new approaches, sociological aspects of PTSD in the digital habitus can be added to develop unique therapy approaches that embrace sociological perspectives of Information Society with a full trajectory of healing practices and a chance of addressing PTSD in its full spectrum.

In conclusion, postmodern city life has worsened the situation of the Information Age regarding PTSD. It affects not only daily life, work-life, education, and academic life, but also affects the total health of society. A person who suffers from PTSD has a lot of issues to cope with. Since the main foci of this study are to explore and exemplify new diagnostic and therapeutic approaches to PTSD cases, a profound analysis of PTSD in terms of its biological, sociological, developmental, psychological, and even ontological aspects are provided by embracing the digital revolutions of the society with its novel implications and insights. To address the issue with its full sides and angles, new treatment opportunities are portrayed as a sine qua non for contemporary psychiatrists.

### **Conflict of interest**


The authors declare no conflict of interest.

### **Author details**

Nevzat Tarhan\*, Muhsin Konuk, Mesut Karahan, Öznur Özge Özcan, Sibel Öztürk Ayvaz, Gökben Hızlı Sayar, Nurper Ülküer, Hazal Ayas and Feride Zeynep Güder  
Uskudar University, Istanbul, Turkey

\*Address all correspondence to: [nevzat.tarhan@uskudar.edu.tr](mailto:nevzat.tarhan@uskudar.edu.tr)

### **IntechOpen**

© 2022 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. 

## References

- [1] American Psychiatric Association. *The Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Washington: American Psychiatric Association Publishing; 2013
- [2] Koenen KC, Ratanatharathorn A, Ng L, McLaughlin KA, Bromet EJ, Stein DJ, et al. Posttraumatic stress disorder in the world mental health surveys. *Psychological Medicine*. 2017;47(13):2260-2274
- [3] Özmen E. Bir İktidar Pratiği Olarak Psikiyatri ve Psikoloji: Travma Örneği [Internet]. 2013 . Available from: <https://birikimdergisi.com/guncel/715/bir-iktidar-pratigi-olarak-psikiyatri-ve-psikoloji-travma-ornegi> [Accessed: September 19, 2021]
- [4] Tarhan N. *Çağın Vicdanı Bediüzzaman*. İstanbul: Nesil Publishing; 2012
- [5] Tarhan N. In: Tatlı A, Görmez İ, editors. *Hakkın Dilinden Ontoloji*. İstanbul: Üsküdar University Publishing; 2020
- [6] Şimşek ÖF. Happiness revisited: Ontological well-being as a theory-based construct of subjective well-being. *Journal of Happiness Studies*. 2009;10:505-522
- [7] Tanrıdağ O, İnaniyorum O. *Halde Varım*. İstanbul: Üsküdar University Publishing; 2017
- [8] Tarhan N. *Travmayı Anlamak*. In: *Travma Psikoloji Kongresi; İstanbul. 7-8 March 2020; İstanbul*. Available from: <https://www.nevzattarhan.com/travmayi-anlamak.html>
- [9] Hızlı Sayar G, Ünübol H, Tarhan N. Fears, concerns and maturation during the Covid-19 Pandemia (in Turkish). İstanbul: Üsküdar University Publications; 2020
- [10] Nisar S, Bhat AA, Hashem S, Syed N, Yadav SK, Uddin S, et al. Genetic and neuroimaging approaches to understanding post-traumatic stress disorder. *International Journal of Molecular Sciences*. 2020;21(12):4503. DOI: 10.3390/ijms21124503
- [11] Misganaw B, Guffanti G, Lori A, Abu-Amara D, Flory DJ, SBPBC, et al. Polygenic risk associated with post-traumatic stress disorder onset and severity. *Translational Psychiatry*. 2019;9:165. DOI: 10.1038/s41398-019-0497-3
- [12] True WR, Rice J, Eisen SA, Heath AC, Goldberg J, Lyons MJ, et al. A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. *Archives of General Psychiatry*. 1993;50(4):257-264. DOI: 10.1001/archpsyc.1993.01820160019002
- [13] Dunlop BW, Wong A. The hypothalamic-pituitary-adrenal axis in PTSD: Pathophysiology and treatment interventions. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*. 2019;89:361-379. DOI: 10.1016/j.pnpbp.2018.10.010
- [14] Aliev G, Beeraka NM, Nikolenko VN, Svistunov AA, Rozhnova T, Kostyuk S, et al. Neurophysiology and psychopathology underlying PTSD and recent insights into the PTSD therapies—A comprehensive review. *Journal of Clinical Medicine*. 2020;9(9):2951. DOI: 10.3390/jcm9092951
- [15] Gressier F, Calati R, Balestri M, Marsano A, Alberti S, Antypa N, et al. The 5-HTTLPR polymorphism and



posttraumatic stress disorder: A meta-analysis. *Journal of Traumatic Stress*. 2013;**26**(6):645-653. DOI: 10.1002/jts.21855

[16] Amstadter AB, Koenen KC, Ruggiero KJ, Acierno R, Galea S, Kilpatrick DG, et al. Variant in RGS2 moderates posttraumatic stress symptoms following potentially traumatic event exposure. *Journal of Anxiety Disorders*. 2009;**23**(3):369-373. DOI: 10.1016/j.janxdis.2008.12.005

[17] Beins EC, Beiert T, Jenniches I, Hansen JN, Leidmaa E, Schrickel JW, et al. Cannabinoid receptor 1 signaling modulates stress susceptibility and microglial responses to chronic social defeat stress. *Translational Psychiatry*. 2021;**11**(1):164. DOI: 10.1038/s41398-021-01283-0

[18] Wolf EJ, Mitchell KS, Logue MW, Baldwin CT, Reardon AF, Aiello A, et al. The dopamine D3 receptor gene and posttraumatic stress disorder. *Journal of Traumatic Stress*. 2014;**27**(4):379-387. DOI: 10.1002/jts.21937

[19] Solovieff N, Roberts AL, Ratanatharathorn A, Haloosim M, De Vivo I, King AP, et al. Genetic association analysis of 300 genes identifies a risk haplotype in SLC18A2 for post-traumatic stress disorder in two independent samples. *Neuropsychopharmacology*. 2014;**39**(8):1872-1879. DOI: 10.1038/npp.2014.34

[20] Nievergelt CM, Maihofer AX, Klengel T, Atkinson EG, Chen C-Y, Choi KW, et al. International meta-analysis of PTSD genome-wide association studies identifies sex- and ancestry-specific genetic risk loci. *Nature Communications*. 2019;**10**(1):4558. DOI: 10.1038/s41467-019-12576-w

[21] Sheerin CM, Lind MJ, Bountress K, Nugent NR, Amstadter AB. The genetics

and epigenetics of PTSD: Overview, recent advances, and future directions. *Current Opinion in Psychology*. 2017;**14**:5-11. DOI: 10.1016/j.copsy.2016.09.003

[22] Wilker S, Schneider A, Conrad D, Pfeiffer A, Boeck C, Lingenfelder B, et al. Genetic variation is associated with PTSD risk and aversive memory: Evidence from two trauma-exposed African samples and one healthy European sample. *Translational Psychiatry*. 2018;**8**:251. DOI: 10.1038/s41398-018-0297-1

[23] Huckins LM, Chatzinakos C, Breen MS, Hartmann J, Klengel T, da Silva Almeida AC, et al. Analysis of genetically regulated gene expression identifies a prefrontal PTSD gene, SNRNP35, specific to military cohorts. *Cell Reports*. 2020;**31**(9):107716. DOI: 10.1016/j.celrep.2020.107716

[24] Duncan LE, Ratanatharathorn A, Aiello AE, Almli LM, Amstadter AB, Ashley-Koch AE, et al. Largest GWAS of PTSD (N=20 070) yields genetic overlap with schizophrenia and sex differences in heritability. *Molecular Psychiatry*. 2018;**23**(3):666-673. DOI: 10.1038/mp.2017.77

[25] Chen C-Y, Stein M, Ursano R, Cai T, Gelernter J, Heeringa S, et al. Genome-wide association study of posttraumatic stress disorder symptoms in two cohorts of United States Army soldiers. *European Neuropsychopharmacology*. 2017;**27**:S472. DOI: 10.1016/j.euroneuro.2016.09.552

[26] Stein MB, Chen CY, Ursano RJ, Cai T, Gelernter J, Heeringa SG, et al. Genome-wide association studies of posttraumatic stress disorder in 2 cohorts of US army soldiers. *JAMA Psychiatry*. 2016;**73**(7):695-704. DOI: 10.1001/jamapsychiatry.2016.0350

- [27] Maihofer A, Strom N, Mattheisen M, Torres K, Stein M, Ressler K, et al. Refined PTSD phenotyping identifies additional GWAS risk variants and broader domains underlying risk to psychopathology. *Biological Psychiatry*. 2020;**87**(9):S51-S52. DOI: 10.1016/j.biopsych.2020.02
- [28] Pooler T. Genome-wide association study on the sleep symptom of post-traumatic stress disorder [thesis]. Minneapolis: Walden University; 2015
- [29] Thavichachart N, Mushiroda T, Thavichachart T, Charoensook O, Prasansuklab A, Rutchatajumroon P, et al. Genome-wide association study in Thai tsunami survivors identified risk alleles for posttraumatic stress disorder. *Open Journal of Genetics*. 2015;**5**:43-57. DOI: 10.4236/ojgen.2015.52004
- [30] Lee RS, Sawa A. Environmental stressors and epigenetic control of the hypothalamic-pituitary-adrenal axis. *Neuroendocrinology*. 2014;**100**(4):278-287. DOI: 10.1159/000369585
- [31] McNerney MW, Sheng T, Nechvatal JM, Lee AG, Lyons DM, Soman S, et al. Integration of neural and epigenetic contributions to posttraumatic stress symptoms: The role of hippocampal volume and glucocorticoid receptor gene methylation. *PLoS One*. 2018;**13**(2):e0192222. DOI: 10.1371/journal.pone.019222
- [32] Hjort L, Rushiti F, Wang SJ, Fransquet P, Krasniqi SP, Çarkaxhiu SI, et al. Intergenerational effects of maternal post-traumatic stress disorder on offspring epigenetic patterns and cortisol levels. *Epigenomics*. 2021;**13**(12):967-980. DOI: 10.2217/epi-2021-0015
- [33] Smith AK, Ratanatharathorn A, Maihofer AX, Naviaux RK, Aiello AE, Amstadter AB, et al. Epigenome-wide meta-analysis of PTSD across 10 military and civilian cohorts identifies methylation changes in AHRR. *Nature Communications*. 2020;**11**(1):5965. DOI: 10.1038/s41467-020-19615-x
- [34] Uddin M, Ratanatharathorn A, Armstrong D, Kuan PF, Aiello AE, Bromet EJ, et al. Epigenetic meta-analysis across three civilian cohorts identifies NRG1 and HGS as blood-based biomarkers for post-traumatic stress disorder. *Epigenomics*. 2018;**10**(12):1585-1601. DOI: 10.2217/epi-2018-0049
- [35] Yang R, Gautam A, Getnet D, Daigle BJ, Miller S, Jett M. Epigenetic biotypes of post-traumatic stress disorder in war-zone exposed veteran and active duty males. *Molecular Psychiatry*. 2020;**26**:4300-4314. DOI: 10.1038/s41380-020-00966-2
- [36] Logue MW, Miller MW, Wolf EJ, Huber BR, Morrison FG, Zhou Z, et al. An epigenome-wide association study of posttraumatic stress disorder in US veterans implicates several new DNA methylation loci. *Clinical Epigenetics*. 2020;**12**(1):46. DOI: 10.1186/s13148-020-0820-0
- [37] Snijders C, Maihofer AX, Ratanatharathorn A, Baker DG, Boks MP, Geuze E, et al. Longitudinal epigenome-wide association studies of three male military cohorts reveal multiple CpG sites associated with post-traumatic stress disorder. *Clinical Epigenetics*. 2020;**12**:11. DOI: 10.1186/s13148-019-0798-7
- [38] Wolf EJ, Maniates H, Nugent N, Maihofer AX, Armstrong D, Ratanatharathorn A, et al. Traumatic stress and accelerated DNA methylation age: A meta-analysis. *Psychoneuroendocrinology*. 2018;**92**:123-134. DOI: 10.1016/j.psychneuen.2017.12.007

- [39] Quinones MM, Gallegos AM, Lin FV, Heffner K. Dysregulation of inflammation, neurobiology, and cognitive function In PTSD: An integrative review. CABN. 2020;**20**(3):455-480. DOI: 10.3758/s13415-020-00782-9
- [40] Giustino T, Maren S. The role of the medial prefrontal cortex In the conditioning and extinction of fear. *Frontiers in Behavioral Neuroscience*. 2015;**9**:298. DOI: 10.3389/fnbeh.2015.00298
- [41] Maren S, Phan KL, Liberzon I. The contextual brain: Implications for fear conditioning, extinction and psychopathology. *Nature Reviews Neuroscience*. 2013;**14**:417-428. DOI: 10.1038/nrn3492
- [42] Zheng S, Han F, Shi Y, Wen L, Han D. Single-prolonged-stress induced changes in autophagy-related proteins beclin-1, LC3, and p62 in the medial prefrontal cortex of rats with post-traumatic stress disorder. *Journal of Molecular Neuroscience*. 2017;**62**:43-54. DOI: 10.1007/s12031-017-0909-x
- [43] Liang Z, King J, Zhang N. Neuroplasticity to a single-episode traumatic stress revealed by resting-state fMRI in awake rats. *NeuroImage*. 2014;**103**:485-449. DOI: 10.1016/j.neuroimage.2014.08.050
- [44] Sullivan GM, Ogden RT, Huang YY, Oquendo MA, Mann JJ, Parsey RV. Higher in vivo serotonin-1a binding in posttraumatic stress disorder: A PET study with [11C]WAY-100635. *Depression and Anxiety*. 2013;**30**(3):197-206. DOI: 10.1002/da.22019
- [45] Murrugh JW, Czermak C, Henry S, Nabulsi N, Gallezot JD, Gueorguieva R, et al. The effect of early trauma exposure on serotonin type 1B receptor expression revealed by reduced selective radioligand binding. *Archives of General Psychiatry*. 2011;**68**(9):892-900. DOI: 10.1007/10.1001/archgenpsychiatry.2011.91
- [46] Sabban EL, Alaluf LG, Serova LI. Potential of neuropeptide Y for preventing or treating posttraumatic stress disorder. *Neuropeptides*. 2015;**56**:19-24. DOI: 10.1016/j.npep.2015.11.004
- [47] Krystal JH, Abdallah CG, Averill LA, Kelmendi B, Harpaz-Rotem I, Sanacora G, et al. Synaptic loss and the pathophysiology of PTSD: Implications for ketamine as a prototype novel therapeutic. *Current Psychiatry Reports*. 2017;**19**(10):1-11. DOI: 10.1007/s11920-017-0829-z
- [48] Fang Q, Li Z, Huang GD, Zhang HH, Chen YY, Zhang LB, et al. Traumatic stress produces distinct activations of GABAergic and glutamatergic neurons in amygdala. *Frontiers in Neuroscience*. 2018;**12**:1-13. DOI: 10.3389/fnins.2018.00387
- [49] Michels L, Schulte-Vels T, Schick M, O’Gorman RL, Zeffiro T, Hasler G, et al. Prefrontal GABA and glutathione imbalance in posttraumatic stress disorder: Preliminary findings. *Psychiatry Research: Neuroimaging*. 2014;**224**(3):288-295. DOI: 10.1016/j.pscychresns.2014
- [50] Young KA, Thompson PM, Cruz DA, Williamson DE, Selemon LD. BA11 FKBP5 expression levels correlate with dendritic spine density in postmortem PTSD and controls. *Neurobiology of Stress*. 2015;**2**:67-72. DOI: 10.1016/j.ynstr.2015.07.002
- [51] American Psychiatric Association. *The Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Washington:

American Psychiatric Association  
Publishing; 2013. pp. 271-272

[52] Wisco BE, Pugach CP, Nomamiukor FO. Sadness and depression in PTSD. In: Tull M, Kimbrell N, editors. *Emotion in Posttraumatic Stress Disorder: Etiology, Assessment, Neurobiology, and Treatment*. 3rd ed. Academic Press; 2020. pp. 89-116. DOI: 10.1016/B978-0-12-816022-0.00004-1

[53] Courtois CA, Elhai JD, Ford JD, Grasso DJ. *Posttraumatic Stress Disorder Scientific and Professional Dimensions*. Elsevier Academic Press; 2009. DOI: 10.1016/C2013-0-19408-1

[54] Weathers FW, Litz BT, Keane TM, Palmieri PA, Marx BP, Schnurr PP. *The PTSD Checklist for DSM-5 (PCL-5)* [Internet]. Scale Available From: The National Center for PTSD; 2013. Available from: <https://www.ptsd.va.gov/> [Accessed: September 15, 2021]

[55] Forman-Hoffman V, Cook Middleton J, Feltner C, Gaynes BN, Palmieri Weber R, Bann C, et al. *Psychological and Pharmacological Treatments for Adults With Posttraumatic Stress Disorder: A Systematic Review Update*. Rockville: Agency for Healthcare Research and Quality; 2018. DOI: 10.23970/AHRQEPCCER207

[56] O'Shea BG. *Healing Complex Posttraumatic Stress Disorder: A Clinicians Guide*. New York: Springer; 2021. DOI: 10.1007/978-3-030-61416-4

[57] Bremner JD. *Neuroimaging in posttraumatic stress disorder and other stress-related disorders*. *Neuroimaging Clinics of North America*. 2007;17:523-538

[58] Bremner JD. *Functional neuroimaging in post-traumatic stress disorder*. *Expert Review of Neurotherapeutics*. 2007;7:393-405

[59] Fennema-Notestine C, Stein MB, Kennedy CM, Archibald SL, Jernigan TL. *Brain morphometry in female victims of intimate partner violence with and without posttraumatic stress disorder*. *Biological Psychiatry*. 2002;52:1089-1101

[60] Kroes MC, Rugg MD, Whalley MG, Brewin CR. *Structural brain abnormalities common to posttraumatic stress disorder and depression*. *Journal of Psychiatry & Neuroscience*. 2011;36(4):256-265

[61] Karl A, Schaefer M, Malta LS, Dörfel D, Rohleder N, Werner A. *A meta-analysis of structural brain abnormalities in PTSD*. *Neuroscience and Biobehavioral Reviews*. 2006;30(7):1004-1031

[62] Liu JJW, Nazarov A, Easterbrook B, Plouffe RA, Le T, Forchuk C, et al. *Four decades of military posttraumatic stress: Protocol for a meta-analysis and systematic review of treatment approaches and efficacy*. *JMIR Research Protocols*. 2021;10:10. DOI: 10.2196/33151

[63] Kan RB, Zhang B, Zhang J, Kranz GS. *Non-invasive brain stimulation for posttraumatic stress disorder: A systematic review and meta-analysis*. *Translational Psychiatry*. 2020;10:168

[64] National Research Council (US) and Institute of Medicine (US) Committee on Integrating the Science of Early Childhood Development. In: Shonkoff JP, Phillips DA, editors. *From Neurons to Neighborhoods: The Science of Early Childhood Development*. Washington: National Academies Press; 2000. DOI: 10.17226/9824

[65] Kieling C, Baker-Henningham H, Belfer M, Conti G, Ertem I, Ulkuer N, et al. *Child, and adolescent mental health worldwide: Evidence for action*.

Lancet. 2011;22:1515-1525. DOI: 10.1016/S0140-6736(11)60827-1

[66] American Psychiatric Association. Desk Reference to the Diagnostic Criteria from DSM-5. Arlington: American Psychiatric Association Publishing; 2013. DOI: 10.1176/appi.books.9780890425596

[67] Bremner JD. Does Stress Damage the Brain?: Understanding Trauma-Related Disorders from a Mind-Body Perspective. New York: W.W.Norton; 2005

[68] Creeden K. The neurodevelopmental impact of early trauma and insecure attachment: Re-thinking our understanding and treatment of sexual behavior problems. Sex Addict Compulsivity. 2004;11(4):223-247

[69] Putnam FW. The impact of trauma on child development. Juvenile & Family Court Journal. 2009;57(1):1-11. DOI: 10.1111/j.1755-6988.2006.tb00110.x

[70] Warshaw M, Fiermann E, Pratt L, Hunt M, Yonkers K, Massion AO, et al. Quality of life and dissociation in anxiety disorder patients with histories of trauma or PTSD. The American Journal of Psychiatry. 1993;150(10):1512-1516. DOI: 10.1176/ajp.150.10.1512

[71] Richardson M, Henry J, Black-Pond C, Sloanne M. Multiple types of maltreatment: Behavioral and developmental impact on children in the child welfare system. Journal of Child and Adolescent Trauma. 2008;1:317-330. DOI: 10.1080/19361520802505735

[72] Herring RJ. Trauma, PTSD, and the developing brain. Current Psychiatry Reports. 2017;19(10):69-78. DOI: 10.1007/s11920-017-0825-3

[73] Perry BD. Childhood experience and the expression of genetic potential: What

childhood neglect tells us about nature and nurture. Brain and Mind. 2002;3:7-100. DOI: 10.1023/A:1016557824657

[74] Weems CF, Russell JD, Neill EL, McCurdy BH. Annual research review: Pediatric posttraumatic stress disorder from a neurodevelopmental network perspective. Journal of Child Psychology and Psychiatry. 2019;60(4):395-408. DOI: 10.1111/jcpp.12996

[75] Winston R, Chicot R. The importance of early bonding on the long-term mental health and resilience of children. London Journal of Primary Care. 2016;8(1):12-14. DOI: 10.1080/17571472.2015.1133012

[76] Iruka IU, Sim J, Fort A. Black Parents and their Babies: Attending to the First 1,000 Days. Chapel Hill, NC: Equity Research Action Coalition at the UNC Frank Porter Graham Child Development Institute; 2021. Available from: <https://fpg.unc.edu/publications/black-parents-and-their-babies-attending-first-1000-days>

[77] National Scientific Council on the Developing Child. Excessive Stress Disrupts the Architecture of the Developing Brain: Working Paper 3. Updated Edition. 2005/2014. Available from: <http://www.developingchild.harvard.edu> [Accessed: November 15, 2021]

[78] Tottenham N, Galván A. Stress and the adolescent brain: Amygdala-prefrontal cortex circuitry and ventral striatum as developmental targets. Neuroscience and Biobehavioral Reviews. 2016;70:217-227. DOI: 10.1016/j.neubiorev.2016.07.030

[79] Kimberg L, Wheeler M. Trauma and trauma-informed care. In: Medical Management of Vulnerable and Underserved Patients: Principles,

- Practice, and Populations. 2nd ed. New York: McGraw-Hill Medical Publishing Division; 2019. DOI: 10.1007/978-3-030-04342-1\_2
- [80] Weems CF. Severe stress and the development of the amygdala in youth: A theory and its statistical implications. *Developmental Review*. 2017;**46**(1):44-53. DOI: 10.1016/j.dr.2017.08.001
- [81] Hillary AF. Toxic stress: Effects, prevention and treatment. *Children*. 2014;**1**(3):390-402. DOI: 10.3390/children1030390
- [82] Raffagnato A, Angelico C, Fasolato R, Sale E, Gatta M, Miscioscia M. Parental bonding and children's psychopathology: A transgenerational view point. *Children*. 2021;**8**(11):1012. DOI: 10.3390/children8111012
- [83] Avinun R, Knafo A. Parenting as a reaction evoked by children's genotype: A meta-analysis of children-as-twins studies. *Personality and Social Psychology Review*. 2014;**18**(1):87-102. DOI: 10.1177/1088868313498308
- [84] Lacasa F, Ochoa S, Balluerka N, Mitjavila M. The relationship between attachment styles and internalizing or externalizing symptoms in clinical and nonclinical adolescents. *Anales de Psicología*. 2015;**31**(2):422-432. DOI: 10.6018/analesps.31.2.169711
- [85] Nelson C, Furtado E, Fox N, Zeanah C. The deprived human brain. *American Scientist*. 2009;**97**(3):222-229. DOI: 10.1511/2009.78.222
- [86] Bennegadi R. Social psychiatry In the era of cyber age and globalization: Threatened, empowered, or both? *World Social Psychiatry*. 2019;**1**:62-66
- [87] Shanahan J, Morgan M. Television and its Viewers Cultivation Theory and Research. Cambridge: Cambridge University Press; 2004
- [88] Simmel G. *The Metropolis and Mental Life*. New York: Free Press; 1950. pp. 409-424
- [89] Foshay R. *The Digital Nexus: Identity, Agency, and Political Engagement*. Athabasca: AU Press; 2016. DOI: 10.15215/aupress/9781771991292.01
- [90] Hübl T, Avritt JJ. *Healing Collective Trauma: A Process for Integrating our Intergenerational & Cultural Wounds*. Colorado: Boulder; 2020
- [91] Han B-C. *The Scent of Time, a Philosophical Essay on the Art of Lingerin*. Cambridge: Polity Press; 2017
- [92] Han B-C. *The Burnout Society*. Stanford: Stanford University Press; 2015
- [93] Güder FZ. Psychological consultancy through digital media in times of Covid-19 pandemic: An analysis of the TV series "red room" and "apartments of innocents". In: IFIG 8th International Communication Days, Crisis Communication in the Digital Age Symposium; 26-28 May 2021; İstanbul
- [94] Tarhan N. *Toplum Psikolojisi ve Empati: Sosyal Şizofreniden Toplumsal Empatiye*. İstanbul: Timaş Yayınları; 2016. pp. 82-83
- [95] Öngün E. Renaming citizenship: An evolution from social citizenship to digital citizenship. In: Öngün E, Pembecioğlu N, Gündüz U, editors. *Handbook of Research on Digital Citizenship and Management during Crises*. USA: IGI Global; 2021. pp. 1-16. DOI: 10.4018/978-1-7998-8421-7
- [96] Beck U. *Pioneer in Cosmopolitan Sociology and Risk Society*. Springer Briefs on Pioneers in Science

and Practice; 2014. p. 18. DOI:  
10.1007/978-3-319-04990-8

[97] Costa C. Bourdieu, Habitus and Social Research the Art of Application. 1st ed. New York: Palgrave Macmillan; 2015

[98] Bryant RA. Social attachments and traumatic stress. *European Journal of Psychotraumatology*. 2016;7(1):29065. DOI: 10.3402/pt.v7.29065

[99] Sontag S. *Regarding the Pain of Others*. New York: Picador; 2003

[100] Baudrillard J. *Simulacra and Simulation (the Body in Theory: Histories of Cultural Materialism)*. Michigan: University of Michigan Press; 1994

[101] Cem T. 6th International Communication Days Digital Transformation Symposium, Üsküdar University, Faculty of Communication; 02-03 May 2019; İstanbul, Turkey

[102] Zecher JL. *Acedia: The Lost Name for the Emotion We're All Feeling Right Now* [Internet]. 2020. Available from: <https://theconversation.com/acedia-the-lost-name-for-the-emotion-were-all-feeling-right-now-144058>. [Accessed: October 23, 2021]

[103] Restrepo JAF. Durée and temporality: A defense of Bergson's conception of time. *Discusiones Filosoficas*. 2015;16(27):49-61. DOI: 10.17151/difil.2015.16.274