Review

Trauma-Related Internalizing and Externalizing Behaviors in Adolescence: A Bridge between Psychoanalysis and Neuroscience

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Abstract: The adolescent brain is an open window on the environment, which is vulnerable to perturbations and the traumatic experiences occurring before or during this period have an increased saliency in affecting cognitive, emotional, and social levels. During adolescence, trauma-related effects causing significant impairment or suffering could be manifest in internalizing and externalizing behaviors. The present mini review aimed to clarify trauma effects on adolescence by examining the neurobiological correlates associated with an increased risk of externalizing/internalizing conducts, as well as the transformative effects of multiple and multimodal therapeutic interventions.

Keywords: trauma; post-traumatic stress disorder; development; extroverted symptoms; introverted symptoms; amygdala; medial prefrontal cortex; multiple and multimodal therapeutic treatments

1. Introduction

Trauma occurs more often than we think in youths. By age 16, two-thirds of children reported at least one traumatic event [1]. Among traumatic events, it is possible to include psychological, physical, or sexual abuse; community or school violence; witnessing or experiencing domestic violence; national disasters or terrorism; commercial sexual exploitation; sudden or violent loss of a loved one; refugee or war experiences; family-related military stressors; physical or sexual assault; neglect; serious accidents or life-threatening illness.

The developmental period is especially crucial when examining the effects of trauma at both neurobiological and behavioral levels. Coping with a history of chronic trauma or experiencing exposure to acute trauma during the development may have different, and sometimes even more significant, bio-psycho-social effects than trauma exposure occurring in adulthood [2]. In particular, changes occurring during adolescence are strictly associated with a prolonged period of brain and behavioral adaptations to prepare the individual for independence. Very briefly, adolescent humans, nonhuman primates, and rodents develop similar behaviors, including separation from parents, enhanced social interactions, increased risk-taking and sensation-seeking behaviors, modified food intake, and postponed sleep cycles [3]. In addition, the transition to adolescence gathers attention because some neuropsychiatric disorders, including schizophrenia, manifest at this period of life [4–6], and the transition is especially risky for children who are growing up in traumatic environments [7,8], partially due to the heightened trauma reactivity that characterizes the onset of puberty [9–11]. In fact, the variety of drastic (progressive as well as regressive) alterations in the brain renders the adolescent system highly vulnerable to the effects of trauma exposure. A history of trauma can be preadolescence-limited (i.e., early childhood and childhood) or adolescence-limited (i.e., early adolescence, adolescence, and late adolescence), with
trauma-related behavioral and brain effects measured in adolescence or in adulthood (the specific ages for each developmental period are shown in Figure 1).

Figure 1. Specific ages for each developmental period (i.e., early childhood, childhood, early adolescence, adolescence, late adolescence, and adulthood) are reported.

During adolescence, affective, cognitive, and behavioral symptoms related to trauma causing significant impairment or suffering could be classified within the internalizing and externalizing domains [12–14]. Internalizing problems are those having mood or emotion as their primary feature and include symptoms such as anxiety, depression, anhedonia, and withdrawal, while externalizing problems are those such as aggression, delinquency, oppositional defiant disorder, and conduct disorder [15,16]. Thus, it is important to recognize the signs of trauma and its short- and long-term consequences and, faced with the complexity of trauma-related consequences, psychoanalysts who work with adolescents are urged to co-construct a relationship with traumatized adolescents to favor the plastic potential of the brain and the Self, enlarging the therapeutic level to counteract the internalizing or externalizing behaviors.

The present mini review adds to the current literature on trauma effects on adolescence and summarizes recent knowledge by examining the reorganization of neurobiological systems associated with an increased risk of externalizing/internalizing behaviors (Section 2: Diagnostic Aspects and Neurobiological Correlates in Traumatized Adolescents: A Scenario on Externalizing/Internalizing Behaviors), as well as the transformative effects of multiple and multimodal interventions in traumatized adolescents, locating the therapeutic interventions in the theoretical framework of neuro-psychoanalysis and affective neuroscience (Section 3: Therapeutic Aspects in Traumatized Adolescents: An Enlargement of the Psychoanalytic Setting in Multiple and Multimodal Treatments). Finally, consistent with the conceptual model on resilience proposed by Feldman [17,18], it is discussed how an enlarged therapeutic plan may influence brain plasticity, promote cognitive, emotional, and social skills, and restore a more resilient Self (Section 4: Discussion).

2. Diagnostic Aspects and Neurobiological Correlates in Traumatized Adolescents: A Scenario on Externalizing/Internalizing Behaviors

One of the adaptive values of long adolescence is a prolonged period of synaptogenesis, neuroplasticity, and neuronal connectivity, all processes that sustain behavioral changes, mainly in the social domain, needed for the passage to adulthood [19,20]. The adolescent brain is vulnerable to environmental perturbations and traumatic experiences occurring before or during this period have an increased saliency in affecting cognitive, emotional, and social levels. Given these bio-psycho-social changes occurring in this part of life, diagnosing trauma-related disorders in adolescence is quite complex. In this life period, besides a categorical approach, a dimensional approach should be used in the diagnostic process as a tool to capture the elements of continuity in physiological and psychopathological trajectories of development [21]. According to the fifth edition of the...
Diagnostic and Statistical Manual (DSM-5) of mental disorders, trauma-related disorders during adolescence imply exposure to overwhelming, aversive, threatening, or fearful experiences [22]. Affective, cognitive, and behavioral symptoms related to trauma causing significant impairment or suffering can be accompanied by dissociative symptomatology (depersonalization and derealization on the positive pole, as well as dissociative amnesia on the negative pole). As mentioned above, trauma-related symptoms in adolescence can be classified within the externalizing and internalizing domains, along with a dimension ranging from hyper- to hypoarousal, and may also lead to a Reactive Attachment Disorder or an Uninhibited Social Engagement Disorder [12–14]. As regards externalizing behaviors, extroverted symptoms mainly occur such as: hypervigilance, impulsivity, hyperactivity, disinhibition, aggression, mood enhancement, intrusive thoughts, disorganized cognitive processing on the paranoid side, substance abuse, difficulty in attention and concentration, hyperactivity, and hyperexcitability, accompanied by depersonalization and derealization. Conversely, introverted symptoms mainly occur in the presence of internalizing behaviors, such as: freezing, social withdrawal, mood deflection, cognitive inhibition, depressive ideation, numbing, reduced psychomotor skills, avoidance behaviors, and feelings of impotence, accompanied by dissociative amnesia.

The externalizing/internalizing conducts are grounded in a neurobiological basis, which undergo crucial changes during adolescence, especially in the presence of trauma. In fact, brain structure, function and connectivity, neurotransmitter levels, stress response, homeostasis maintenance, immune system, genetics/epigenetics, and gut microbiome undergo marked changes due to trauma exposure [23–25]. To improve understanding of how the effects of traumatic experiences on the brain involve changes at nearly every level of analysis, there is a need to comprehend the adolescent physiological conditions. During adolescence, brain remodeling is characterized by changes in associative and limbic circuits (such as the medial prefrontal cortex, amygdala, and hippocampus) subserving high-order processes (e.g., executive function, mentalizing, emotion regulation, and social cognition), along with dramatic changes in endocrine, immune, and biochemical systems [20,26]. The initiation of puberty represents a period of dynamic synaptogenesis and dendritic/synaptic pruning, with experience-dependent remodeling of brain circuits underlying complex behaviors [27,28]. Specifically, during this time there is substantial pruning of excitatory synapses in the cortex and in particular in the prefrontal cortex, supporting the hypothesis that overproduction and subsequent pruning of synapses is a computationally advantageous approach to building a competent brain [29]. A correct balance of activity of the GABA and glutamate systems is vital for optimal neurodevelopment and general central nervous system function, and the dysregulation of this balance has been implicated in a number of neurological conditions that range from mild to severe, including schizophrenia and epilepsy, and has been shown to increase the incidence of anxiety disorders [30,31].

In the context of trauma, the amygdala, the medial prefrontal cortex, and the hippocampus have been extensively investigated with respect to fear learning and extinction, threat reactivity, and emotion regulation [32–34], and they have been identified as critical loci of dysfunction following trauma [35,36]. Notably, acute exposure to threatening adversity promotes the secretion of hormones (e.g., cortisol) and pro-inflammatory cytokines, which drive changes in the structural plasticity of the amygdala and hippocampus to enhance fear learning in the occurrence of similar events [37]. Chronic exposure to traumatic events acts through these same hormonal and immune mediators to create glutamatergic excitotoxicity and atrophy in the amygdala and hippocampus linked to impaired memory and other behavioral and cognitive symptoms, commonly found in internalizing behaviors, such as depression [38]. Furthermore, deficits in the synthesis of GABAergic neurosteroids—produced from progesterone in the brain, adrenal gland, ovaries, and testes [39], clearly more active during puberty—have been implicated in the pathophysiology and recovery from trauma-related disorders [40]. For individuals raised in environments where multiple sources of threat are present and/or long-term survival is uncertain, the “developmental reprioritization” is often marked by an accelerated maturation, charac-
terized by the early emergence of adult-like phenotypes [41,42]. Specifically, traumatized children showed adult-like neural phenotype in the functional connectivity between the amygdala and medial prefrontal cortex during threat processing [43–46], in parallel with experimental reports indicating that early adversity leads to accelerated myelination of axons in the amygdala [47].

Overall, across various types of traumatic experiences, an altered structure, function, and connectivity of the amygdala, medial prefrontal cortex, and hippocampus correspond to an increased fear reactivity, attentional biases towards threat, and difficulty with affective regulation [48–50], contributing to a risk of externalizing/internalizing behaviors.

3. Therapeutic Aspects in Traumatized Adolescents: An Enlargement of the Psychoanalytic Setting in Multiple and Multimodal Treatments

The dialogue between psychodynamic psychotherapy and neuroscience started with the studies of McKenna et al. [51] and Kandel [52,53], supporting the hypothesis that the former may change the brain. It was documented how psychodynamic psychotherapy was able to modify synaptic connections, resulting in long-term behavioral modifications for the remodeling of cortical and limbic connections [51]. Subsequent studies documented the changes produced by psychodynamic psychotherapy on neuronal cytoarchitecture during the lifetime [54], on brain circuits [55], and on neurotransmission systems [56]. Such significant changes would be activated by the “unconscious communication” between the individual and psychoanalyst, a process considered embodied simulation [57,58]. Accordingly, the intersubjective approach postulated the therapeutic change in the framework of the non-verbal dimension of the dialogue between psychoanalyst and patient, in which the non-declarative memory (associated with procedural learning) is the main process of change following psychoanalysis treatment [59–61]. Conversely, Piovano [62] disagreed with the exclusively procedural and unconscious approach to change. In her opinion, new implicit memories would be produced through the introduction of new ways of being, which weaken the old procedures.

Extended analytical listening includes empathic listening, attention to body language, and preverbal and presymbolic levels, which are enacted in the therapeutic relationship, as well as countertransferentially communicated in meaningful verbal forms [63]. All these aspects are needed for traumatized adolescents in order to experience a “rescue environment” [64,65]. The awareness of damage produced by trauma in brain tissue has forced a profound reworking of therapeutic models: it is necessary to extend the treatment both within a dual setting, as proposed by Piovano [63], and within multiple settings [64,65]. Thanks to this enlargement of the techniques, the psychoanalytic experience may become an opportunity for the psychoanalyst and patient characterized by affective signification or, on the contrary, by somatic incorporation of psychic experiences [63,66]. In psychoanalytic psychotherapy of traumatized adolescents, it is inevitably necessary to realize an enlargement of classical methods, given the multilayered levels of trauma-related suffering [67].

The present contribution suggests that this “enlarged” orientation in the multiple setting may offer traumatized adolescents a range of transformative experiences (not only verbal, but also in terms of embodied expression) that exceed the dual setting [68], allowing to reintegrate split traumatic experiences in a coherent and organized way [69]. In the theoretical scaffolding of neuro-psychoanalysis and affective neuroscience, therapeutic interventions (multiple and multimodal treatments) should be aimed at modulating the restorative capacity of the brain and the reorganization of the Self [24,27]. From a therapeutic point of view, after a series of failures in the therapeutic relationship and verbal communication with the traumatized adolescent patient, a more effective perspective might be based on an intervention aimed at attuning the implicit bodily response of the patient to trauma-related effects, by using the patient’s perception-action system. Accordingly, Howell [70] suggested that a therapeutic approach should be followed, based on a specific direction: from the inner word of the traumatized patient to the out one, rather than
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from the outside in. The issue is to integrate emotions and achieve a balance between the sympathetic and the parasympathetic system activation [68]. During the therapy, it is necessary to strengthen the cooperation between the dissociated parties of the Self, to favor present-time sensations, to evoke warmth and reassuring sensations, and to look to the future with hope [71]. Furthermore, the development of the symbolic function may be promoted. Specifically, a face-to-face setting mainly exposes a traumatized subject to emotional stimuli aroused by facial expression and personal interaction, probably activating the subcortical regions of the limbic system, primarily the amygdala, involved in the emotional (sub-symbolic) reaction more than in the emotional (symbolic) experience [72,73]. Conversely, the couch setting mainly exposes the traumatized subject to verbal stimuli, probably activating the cortical regions, primarily the prefrontal cortex, involved in emotional experience more than in emotional reaction [74].

Thus, these considerations should be taken into account in the construction of a therapeutic plan and in the course of the analytic process. At least in the initial stages of therapeutic treatment for traumatized adolescents suffering from severe dissociative disorders, it may be useful to start with a clinical setting characterized by a high containment potential, allowing a good relationship with reality. In the presence of traumatized adolescents, psychoanalysts are not only called upon to listen to the indistinct, painful, sensory, and perceptive fragments which freeze the body and turn off the mind, but also to relate to a deep anguish of disintegration, with flashbacks, intrusive and dangerous memories, and traumatizing thoughts. When the trauma is massive, adolescents are unable to only use a dual setting and need a continuous ordering principle capable of containing them and counteracting the risk of post-traumatic acting. This therapeutic ordering principle can be activated by multiple and multimodal interventions.

In the institutional framework, multimodal interventions showed significant results [75]. These interventions offered the adolescents activities based on awareness training, expressive arts, and Eye Movement Desensitization and Reprocessing Integrative—EMDR—treatments. These activities were useful for adolescents to help them express their emotional and cognitive universe, by discovering the possibility of adapting their narratives to healthier directions [76]. After completing the treatment program, adolescents showed a significant reduction in trauma-related outcomes, while attention/awareness processes were improved. These changes remained stable two months after discharge, showing how multimodal interventions may represent feasible and promising programs for reducing the psychological burden in adolescents with a history of trauma. However, it should be noted that the main limitation of this type of therapeutic intervention is the short nature of the treatment itself and the fact that its benefits are only measured over a period of months. As Gillies [77] argued, much more evidence is needed to demonstrate the real efficacy of different psychological therapies on subjects exposed to trauma, particularly in the long term. Furthermore, practices based on trauma-sensitive awareness through mindfulness [78], artistic expression [76], or group treatment [79] have also been reported. Other findings have shown that multimodal treatment methods provided significant long-term benefits in several domains of mental health in children and adolescents exposed to adverse experiences [80]. The use of neurofeedback in children and adolescents has been also investigated [81], opening good prospects for the treatment of externalizing/internalizing symptoms. The results offered by this type of treatment are encouraging but, according to some researchers [82], less than 50% of substantial improvements are found in the best-performed clinical trials. It is also important to note the importance of involving parents in the treatment of traumatized children and adolescents. Previous research conducted by Haine-Schlagel et al. [83] suggested that such involvement occurred more frequently when the children had high levels of behavioral problems and when parents reported high levels of internalized tension, as well as when the therapist was more experienced.

In addition to the potential cumulative benefits of combining different approaches, it is possible that multimodal programs allow children and adolescents to benefit from
therapists and methods best suited to them, which is not an option with individual therapeutic interventions.

4. Discussion

Significant changes occur in a relatively short time in the adolescent brain. For such reasons, adolescence is a “window” of susceptibility to trauma. Traumas, particularly interpersonal, intentional, and chronic ones, are considered risk factors for the onset of internalizing/externalizing psychopathology [84–86]. For example, emotional dysregulation, impulsivity, and mood lability, associated with early life deprivation and threat, are potent risk factors for alcohol and illicit substance abuse in the adolescent period [87–90]. Furthermore, the greater the number of traumas, the higher the probability of externalizing and post-traumatic stress disorder symptoms among adolescents [91]. However, adolescence may also be a period of recovery from trauma encountered during childhood. Namely, the transition to adolescence may be a period of increased opportunities for positive growth [92], and the bio-psycho-social reorganization that takes place during that time may enable the youth to have resilience. It has been shown that maternal warmth and sensitive support can buffer the negative effects of trauma exposure on child outcome [93], as well as maternal post-traumatic stress symptoms, sensitive caregiving, and child cortisol, which shaped the trajectories of risk and resilience across the 10-year span [94,95]. Accordingly, trauma-exposed mothers whose children were diagnosed with an internalizing disorder during adolescence showed elevated hair cortisol levels [96].

Importantly, Feldman [17] stated that resilience is the ultimate goal of human development, especially for the overcoming of traumas. Resilience originates from primary relationships, involves social aspects, allows for the attribution of a new meaning to traumatic and dissociative events supporting the integration of the Self in the presence of trauma, and promotes neuronal plasticity at the biological level [97–99]. Consistent with this conceptual model on resilience [17,18] that considers the orchestration of biological and behavioral processes between parent and child as an important mechanism by which the parent modulates the child’s psychophysiological systems and tunes them to social life, the findings from the present mini review support that therapeutic relationship with traumatized adolescents may promote plasticity in the neuronal regions subserving the social functions and could thus reorganize the neural networks susceptible to trauma. In this framework, it is advanced the necessity of multiple and multimodal therapeutic interventions, in which specific therapeutic actors may be capable of acting on bio-psycho-social aspects of traumatized adolescents [100]. The different aspects of trauma-related suffering require a multiple psychodynamic setting, which provides an integrated intervention of multiple professionals and techniques (such as physiotherapy, yoga, EMDR, neurofeedback, mindfulness, dance, listening to music) [65,68]. Thanks to this multiple orientation and systemic work, it is possible to act on different levels of trauma-related suffering, which affects the homeostatic functions (sleep, appetite, digestion, arousal), as well as the brain regions involved in emotional self-regulation, perception of the Self, attention, ability to stay focused on oneself and in tune with others, capacity to make meaningful relationships beyond the catastrophic expectations triggered by traumatization. In other words, an enlarged therapeutic plan may influence brain plasticity, promote cognitive, emotional, and social skills, and restore a more resilient Self.

For the sake of clarity, it must be said that in the current state of art many limitations are present. First of all, the interpretation of the findings on the aftermath of trauma during adolescence needs to consider potential moderators, such as type of stressor, timing effects, or individual and contextual determinants. Subsequent to this, there is no clear evidence of the effectiveness of one psychological therapy compared to others [101,102], as well as insufficient evidence to conclude that children and adolescents with particular types of trauma are more or less likely to respond to some psychological therapies better than others. Finally, despite the importance of the adolescent transition to well-being and adaptations throughout life [103,104], little longitudinal research described factors that may mediate
the continuity of risk and resilience after trauma exposure or tested specific biological and relational factors that may augment or buffer the effects of trauma on mental health. For this reason, future comparative and longitudinal studies should take into account these specific limitations in the planning of research able to identify the influence of significant factors influencing the trauma outcomes, in order to build a bridge between psychoanalysis and neuroscience useful in the implementation of targeted interventions in adolescence.

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**References**

1. Copeland, W.E.; Keeler, G.; Angold, A.; Costello, E.J. Traumatic Events and Posttraumatic Stress in Childhood. *Arch. Gen. Psychiatry* 2007, 64, 577–584. [CrossRef] [PubMed]
2. Birn, R.M.; Patriat, R.; Phillips, M.L.; Germain, A.; Herrlinga, R.J. Childhood Maltreatment and Combat Posttraumatic Stress Differentially Predict Fear-Related Fronto-Subcortical Connectivity. *Depress. Anxiety* 2014, 31, 880–892. [CrossRef] [PubMed]
3. Spear, L.P. The Adolescent Brain and Age-Related Behavioral Manifestations. *Neurosci. Biobehav. Rev.* 2000, 24, 417–463. [CrossRef]
4. Kessler, R.C.; Adler, L.A.; Barkley, R.; Biederman, J.; Conners, C.K.; Faraone, S.V.; Greenhill, L.L.; Jaeger, S.; Secnik, K.; Spencer, T.; et al. Patterns and Predictors of ADHD Persistence into Adulthood: Results from the National Comorbidity Survey Replication. *Biol. Psychiatry* 2005, 57, 1442–1451. [CrossRef]
5. Paus, T.; Keshavan, M.; Giedd, J.N. Why Do Many Psychiatric Disorders Emerge during Adolescence? *Nat. Rev.* *Neurosci.* 2008, 9, 947–957. [CrossRef]
6. Merikangas, K.R.; He, J.; Burstein, M.; Swanson, S.A.; Avenevoli, S.; Cui, L.; Benjet, C.; Georgiades, K.; Swendsen, J. Lifetime Prevalence of Mental Disorders in U.S. Adolescents: Results from the National Comorbidity Survey Replication–Adolescent Supplement (NCS-A). *J. Am. Acad. Child Adolesc. Psychiatry* 2010, 49, 980–989. [CrossRef]
7. LeMoult, J.; Humphreys, K.L.; Tracy, A.; Hoffman, J.-A.; Ip, E.; Gotlib, I.H. Meta-Analysis: Exposure to Early Life Stress and Risk for Depression in Childhood and Adolescence. *J. Am. Acad. Child Adolesc. Psychiatry* 2020, 59, 842–855. [CrossRef]
8. Rudolph, K.D.; Flynn, M. Childhood Adversity and Youth Depression: Influence of Gender and Pubertal Status. *Dev. Psychopathol.* 2007, 19, 497–521. [CrossRef]
9. Dahl, R.E.; Gunnar, M.R. Heightened Stress Responsiveness and Emotional Reactivity during Pubertal Maturation: Implications for Psychopathology. *Dev. Psychopathol.* 2009, 21, 1–6. [CrossRef]
10. Doom, J.R.; Gunnar, M.R. Stress Physiology and Developmental Psychopathology: Past, Present, and Future. *Dev. Psychopathol.* 2013, 25, 1359–1373. [CrossRef]
11. Busso, D.S.; McLaughlin, K.A.; Sheridan, M.A. Dimensions of Adversity, Physiological Reactivity, and Externalizing Psychopathology in Adolescence: Deprivation and Threat. *Psychosom. Med.* 2017, 79, 162–171. [CrossRef]
12. Hofstra, M.B.; Van der Ende, J.; Verhulst, F.C. Pathways of Self-Reported Problem Behaviors from Adolescence into Adulthood. *Am. J. Psychiatry* 2002, 159, 401–407. [CrossRef]
13. King, S.M.; Iacono, W.G.; McGue, M. Childhood Externalizing and Internalizing Psychopathology in the Prediction of Early Substance Use. *Addict. Abingdon Engl.* 2004, 99, 1548–1559. [CrossRef]
14. Reef, J.; Diamantopoulou, S.; van Meurs, I.; Verhulst, F.; van der Ende, J. Predicting Adult Emotional and Behavioral Problems from Externalizing Problem Trajectories in a 24-Year Longitudinal Study. *Eur. Child Adolesc. Psychiatry* 2010, 19, 577–585. [CrossRef]
15. Achenbach, T.M.; Howell, C.T.; Quay, H.C.; Conners, C.K. National Survey of Problems and Competencies among Four- to Sixteen-Year-Olds: Parents’ Reports for Normative and Clinical Samples. *Monogr. Soc. Res. Child Dev.* 1991, 56, 1–131. [CrossRef]
16. Kovacs, M.; Devlin, B. Internalizing Disorders in Childhood. *J. Child Psychol. Psychiatry* 1998, 39, 47–63. [CrossRef]
17. Feldman, R. What Is Resilience: An Affiliative Neuroscience Approach. *World Psychiatry* 2020, 19, 132–150. [CrossRef]
18. Feldman, R. Social Behavior as a Transdiagnostic Marker of Resilience. *Annu. Rev. Clin. Psychol.* 2021, 17, 153–180. [CrossRef]
19. Houston, S.M.; Herting, M.M.; Sowell, E.R. The Neurobiology of Childhood Structural Brain Development: Conception through Adulthood. *Curr. Top. Behav. Neurosci.* 2014, 16, 3–17. [CrossRef]
20. Ho, T.C.; King, L.S. Mechanisms of Neuroplasticity Linking Early Adversity to Depression: Developmental Considerations. *Transl. Psychiatry* 2021, 11, 517. [CrossRef]
21. Lingiardi, V.; Gazzillo, F. *La Personicità e i Suoi Disturbi*; Raffaello Cortina Editore: Milan, Italy, 2014.
22. APA. *Diagnostic and Statistical Manual of Mental Disorders: DSM-5TM*, 5th ed.; American Psychiatric Publishing, Inc.: Arlington, VA, USA, 2013; p. xlvii, 947; ISBN 978-0-89042-554-1.
23. De Bellis, M.D.; Zisk, A. The Biological Effects of Childhood Trauma. *Child Adolesc. Psychiatr. Clin. N. Am.* 2014, 23, 185–222. [CrossRef]
24. Agorastos, A.; Pervanidou, P.; Chrousos, G.P.; Baker, D.G. Developmental Trajectories of Early Life Stress and Trauma: A Narrative Review on Neurobiological Aspects Beyond Stress System Dysregulation. *Front. Psychiatry* 2019, 10, 118. [CrossRef]
25. Kolacz, J.; Kovacic, K.K.; Forges, S.W. Traumatic Stress and the Autonomic Brain-Gut Connection in Development: Polyvagal Theory as an Integrative Framework for Psychosocial and Gastrointestinal Pathology. *Dev. Psychobiol.* 2019, 61, 796–809. [CrossRef]
26. Dandash, O.; Cherbuin, N.; Schwartz, O.; Allen, N.B.; Whittle, S. The Long-Term Associations between Parental Behaviors, Cognitive Function and Brain Activation in Adolescence. *Sci. Rep.* 2021, 11, 11120. [CrossRef]
27. Blakemore, S.-J. Development of the Social Brain during Adolescence. *Q. J. Exp. Psychol.* 2006, 59, 40–49. [CrossRef]
28. Pfeifer, J.H.; Allen, N.B. Puberty Initiates Cascading Relationships between Neurodevelopmental, Social, and Internalizing Processes across Adolescence. * Biol. Psychiatry 2021*, 89, 99–108. [CrossRef]
29. Averbeck, B.B. Pruning Recurrent Neural Networks Replicates Adolescent Changes in Working Memory and Reinforcement Learning. *Proc. Natl. Acad. Sci. USA* 2022, 119, e2121331119. [CrossRef]
30. Meldrum, B.S. Glutamate as a Neurotransmitter in the Brain: Review of Physiology and Pathology. *Transl. Psychiatry* 2021, 11, 517. [CrossRef]
31. Deidda, G.; Bozarth, I.F.; Cancedda, L. Modulation of GABAergic Transmission in Development and Neurodevelopmental Disorders: Investigating Physiology and Pathology to Gain Therapeutic Perspectives. *Front. Cell. Neurosci.* 2014, 8, 119. [CrossRef]
32. Laricchiuta, D.; Saba, L.; De Bartolo, P.; Caioli, S.; Zona, C.; Petrosini, L. Maintenance of Aversive Memories Shown by Fear Extinction-Impaired Phenotypes Is Associated with Increased Activity in the Amygdaloid-Prefrontal Circuit. *Sci. Rep.* 2016, 6, 21205. [CrossRef]
33. Laricchiuta, D.; Sciamanna, G.; Gimenez, J.; Termine, A.; Fabrizio, C.; Caioli, S.; Balsamo, F.; Panuccio, A.; De Bardi, M.; Saba, L.; et al. Optogenetic Stimulation of Prelimbic Pyramidal Neurons Maintains Fear Memories and Modulates Amygdala Pyramidal Neuron Transcriptome. *Int. J. Mol. Sci.* 2021, 22, 810. [CrossRef] [PubMed]
34. Borgomaneri, S.; Battaglia, S.; Sciamanna, G.; Tortora, F.; Laricchiuta, D. Memories Are NotWritten in Stone: Re-Writing Fear Memories by Means of Non-Invasive Brain Stimulation and Optogenetic Manipulations. *Neurosci. Biobehav. Rev.* 2021, 127, 334–352. [CrossRef] [PubMed]
35. Tottenham, N.; Sheridan, M.A. A Review of Adversity, the Amygdala and the Hippocampus: A Consideration of Developmental Timing. *Front. Hum. Neurosci.* 2009, 3, 68. [CrossRef]
36. Tottenham, N. Human Amygdala Development in the Absence of Species-Expected Caregiving. *Dev. Psychobiol.* 2012, 54, 598–611. [CrossRef]
37. McEwen, B.S.; Stellar, E. Stress and the Individual. Mechanisms Leading to Disease. *Arch. Intern. Med.* 1993, 153, 2093–2101. [CrossRef]
38. Lupien, S.J.; Maheu, F.; Tu, M.; Fiocco, A.; Schramek, T.E. The Effects of Stress and Stress Hormones on Human Cognition: Implications for the Field of Brain and Cognition. *Brain Cogn.* 2007, 65, 209–237. [CrossRef]
39. Belelli, D.; Herd, M.B.; Mitchell, E.A.; Peden, D.R.; Vardy, A.W.; Gentet, L.; Lambert, J.J. Neuroactive Steroids and Inhibitory Neurotransmission: Mechanisms of Action and Physiological Relevance. *Neuroscience* 2006, 138, 821–829. [CrossRef]
40. Rasmusson, A.M.; Pineles, S.L.; Brown, K.D.; Pinna, G. A Role for Deficits in GABAergic Neurosteroids and Their Metabolites with NMDA Receptor Antagonist Activity in the Pathophysiology of Posttraumatic Stress Disorder. *J. Neuroendocrinol.* 2021, 34, e13062. [CrossRef] [PubMed]
41. Callaghan, B.L.; Tottenham, N. The Stress Acceleration Hypothesis: Effects of Early-Life Adversity on Emotion Circuits and Behavior. *Curr. Opin. Behav. Sci.* 2016, 7, 76–81. [CrossRef]
42. Belsky, J. Early-Life Adversity Accelerates Child and Adolescent Development. *Curr. Dir. Psychol. Sci.* 2019, 28, 241–246. [CrossRef]
43. Gee, D.G.; Gabard-Durnam, L.J.; Flannery, J.; Goff, B.; Humphreys, K.L.; Telzer, E.H.; Hare, T.A.; Bookheimer, S.Y.; Tottenham, N. Early Developmental Emergence of Human Amygdala-Prefrontal Connectivity after Maternal Deprivation. *Proc. Natl. Acad. Sci. USA* 2013, 110, 15638–15643. [CrossRef] [PubMed]
44. Gee, D.G.; Humphreys, K.L.; Flannery, J.; Goff, B.; Telzer, E.H.; Shapiro, M.; Hare, T.A.; Bookheimer, S.Y.; Tottenham, N. A Developmental Shift from Positive to Negative Connectivity in Human Amygdala-Prefrontal Circuitry. *J. Neurosci. Off. J. Soc. Neurosci.* 2013, 33, 4584–4593. [CrossRef] [PubMed]
74. Protopopescu, X.; Gerber, A.J. Bridging the Gap between Neuroscientific and Psychodynamic Models in Child and Adolescent Psychiatry. *Child Adolesc. Psychiatr. Clin. N. Am.* 2013, 22, 1–31. [CrossRef]

75. Roque-Lopez, S.; Llanzañ-Annaea, Y.; Alvarez-Lopez, M.J.; Everts, M.; Fernández, D.; Davidson, R.J.; Kaliman, P. Mental Health Benefits of a 1-Week Intensive Multimodal Group Program for Adolescents with Multiple Adverse Childhood Experiences. *Child Abuse Negl.* 2021, 122, 105349. [CrossRef]

76. Malchiodi, C. *Expressive Therapies*, 1st ed.; Guilford Press: New York, NY, USA, 2006; ISBN 978-1-59385-379-2.

77. Gillies, D.; Maiocchi, L.; Bhandari, A.P.; Taylor, F.; Gray, C.; O’Brien, L. Psychological Therapies for Children and Adolescents Exposed to Trauma. *Cochrane Database Syst. Rev.* 2016, 10, CD012371. [CrossRef]

78. Ortiz, R.; Sibinga, E.M. The Role of Mindfulness in Reducing the Adverse Effects of Childhood Stress and Trauma. *Children* 2017, 4, 16. [CrossRef]

79. Jarero, I.; Artigas, L. The EMDR Integrative Group Treatment Protocol: EMDR Group Treatment for Early Intervention Following Critical Incidents. *Eur. Rev. Appl. Psychol.* 2012, 62, 219–222. [CrossRef]

80. Silverstone, P.; Suen, V. Are Complex Multimodal Interventions the Best Treatments for Mental Health Disorders in Children and Youth? *J. Child Adolesc. Behav.* 2016, 4, 305–315. [CrossRef]

81. Simkin, D.R.; Thatcher, R.W.; Lubar, J. Quantitative EEG and Neurofeedback in Children and Adolescents: Anxiety Disorders, Depressive Disorders, Comorbid Addiction and Attention-Deficit/Hyperactivity Disorder, and Brain Injury. *Child Adolesc. Psychiatr. Clin. N. Am.* 2014, 23, 427–464. [CrossRef]

82. Lanius, R.A.; Vermetten, E.; Pain, C. (Eds.) *The Impact of Early Life Trauma on Health and Disease: The Hidden Epidemic*; Cambridge University Press: Cambridge, UK, 2010; ISBN 978-0-521-88026-8.

83. Haine-Schlagel, R.; Brookman-Frazee, L.; Darnell, D.; Flaster, A.; Hendricks, K.; Kerbrat, A.; Comtois, K.A. Adolescent Clinical Populations and Associations between Psychopathology. *Am. J. Psychiatry* 2017, 174, 1131–1139. [CrossRef] [PubMed]

84. De Bellis, M.D. Developmental Traumatology: A Contributory Mechanism for Alcohol and Substance Use Disorders. *Psychoneuroendocrinology* 2002, 27, 155–170. [CrossRef]

85. Steinberg, L. A Social Neuroscience Perspective on Adolescent Risk-Taking. *Dev. Rev.* 2008, 28, 78–106. [CrossRef]

86. Heleniak, C.; Jenness, J.L.; Stoep, A.V.; Heleniak, C.; Jenness, J.L.; Stoep, A.V. *Child Maltreatment and Age of Alcohol and Marijuana Initiation in High-Risk Youth*. *Addict. Behav.* 2017, 75, 64–69. [CrossRef] [PubMed]

87. Darnell, D.; Flaster, A.; Hendricks, K.; Kerbrat, A.; Comtois, K.A. Adolescent Clinical Populations and Associations between Trauma and Behavioral and Emotional Problems. *Psychol. Trauma Theory Res. Pract. Policy* 2019, 11, 266–273. [CrossRef] [PubMed]

88. Crone, E.A.; Dahl, R.E. Understanding Adolescence as a Period of Social-Affective Engagement and Goal Flexibility. *Nat. Rev. Neurosci.* 2012, 13, 636–650. [CrossRef]

89. Ulmer-Yaniv, A.; Djakovski, A.; Yirmiya, K.; Palevi, G.; Zagoory-Sharon, O.; Feldman, R. Maternal Immune and Affiliative Biomarkers and Sensitive Parenting Mediate the Effects of Chronic Early Trauma on Child Anxiety. *Psychol. Med.* 2018, 48, 1020–1033. [CrossRef]

90. Palevi, G.; Djakovski, A.; Vengrober, A.; Feldman, R. Risk and Resilience Trajectories in War-Exposed Children across the First Decade of Life. *J. Child Psychol. Psychiatr.* 2016, 57, 1183–1193. [CrossRef]

91. Palevi, G.; Djakovski, A.; Kanat-Maymon, Y.; Yirmiya, K.; Zagoory-Sharon, O.; Koren, L.; Feldman, R. The Social Transmission of Risk: Maternal Stress Physiology, Synchronous Parenting, and Well-Being Mediate the Effects of War Exposure on Child Psychopathology. *J. Abnor. Psychol.* 2017, 126, 1087–1103. [CrossRef]

92. Yirmiya, K.; Motsan, S.; Zagoory-Sharon, O.; Schonblum, A.; Koren, L.; Feldman, R. Continuity of Psychopathology v. Resilience across the Transition to Adolescence: Role of Hair Cortisol and Sensitive Caregiving. *Psychol. Med.* 2022, 1–12. [CrossRef]

93. Gröger, N.; Matas, E.; Gos, T.; Lesse, A.; Poeggel, G.; Braun, K.; Bock, J. The Transgenerational Transmission of Childhood Adversity: Behavioral, Cellular, and Epigenetic Correlates. *J. Neural Transm.* 2016, 123, 1037–1052. [CrossRef]

94. Sharma, V.; Sood, R.; Khlaifia, A.; Elamizade, M.J.; Hung, T-Y.; Lou, D.; Asgarihafshejani, A.; Lalzar, M.; Kiniry, S.J.; Stokes, M.P.; et al. EIF2cx Controls Memory Consolidation via Excitatory and Somatostatin Neurons. *Nature* 2020, 586, 412–416. [CrossRef]

95. Roekner, A.R.; Oliver, K.I.; Lebois, L.A.M.; van Rooij, S.J.H.; Stevens, J.S. Neural Contributors to Trauma Resilience: A Review of Longitudinal Neuroimaging Studies. *Transl. Psychiatry* 2021, 11, 508. [CrossRef]

96. Schore, A.N. *Affect Regulation and the Repair of the Self*, 1st ed.; W. W. Norton & Company: New York, NY, USA, 2003; ISBN 978-0-393-70407-5.
101. Gillies, D.; Taylor, F.; Gray, C.; O’Brien, L.; D’Abrew, N. Psychological Therapies for the Treatment of Post-Traumatic Stress Disorder in Children and Adolescents. Cochrane Database Syst. Rev. 2012, 12, 1004–1116. [CrossRef]

102. Steinert, C.; Munder, T.; Rabung, S.; Hoyer, J.; Leichsenring, F. Psychodynamic Therapy: As Efficacious as Other Empirically Supported Treatments? A Meta-Analysis Testing Equivalence of Outcomes. Am. J. Psychiatry 2017, 174, 943–953. [CrossRef]

103. Patton, G.C.; Coffey, C.; Romaniuk, H.; Mackinnon, A.; Carlin, J.B.; Degenhardt, L.; Olsson, C.A.; Moran, P. The Prognosis of Common Mental Disorders in Adolescents: A 14-Year Prospective Cohort Study. Lancet 2014, 383, 1404–1411. [CrossRef]

104. Johnson, D.; Dupuis, G.; Piche, J.; Clayborne, Z.; Colman, I. Adult Mental Health Outcomes of Adolescent Depression: A Systematic Review. Depress. Anxiety 2018, 35, 700–716. [CrossRef]