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Attachment Styles and Traumatic Responses: Exploring the Impact of Parental Interaction on Child Development and Coping Mechanisms

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Abstract

This article explores the intricate relationship between attachment styles formed during early childhood and the subsequent responses to traumatic events, particularly the death of a parent. Drawing on the theoretical framework of attachment theory and incorporating contemporary research, the paper discusses how parental interactions shape the neural circuitry of infants and children, influencing their ability to form secure or insecure attachments. These attachment styles, in turn, play a critical role in determining the child's coping mechanisms when faced with trauma. This paper focuses on trying to understand how attachment theory is connected to the reaction to trauma with a highlight on the four major styles of attachments which are secure, anxious, avoidant, and disorganized to mention but a few, and how they influence stress and adversity in children. Attachment theory holds that human beings' ability to form affectional bonds in infancy determines their patterns of relatedness across the life cycle. The type of attachment that is secure usually supports healthy adaptation and good coping mechanisms regardless of the trauma in the childhood of the child. While secure attachment mostly facilitates favorable trauma-related outcomes, anxious or avoidant attachment can exacerbate or alter the responses. The caregiving system that is avoidant attachment has implications of autonomous self-functioning which has features of suppression of the emotional response and poor search for emotional support during stress. From the principles of developmental psychology and trauma theory, the paper also focuses on the major significance of the child's early caregivers' interactions that define the resilience and vulnerability factor. This knowledge is therefore critical in designing specific interventions based on the improvement of coping behaviors and emotional regulatory systems of

children who have been exposed to trauma. Finally, we have the synthesis of new knowledge about the role of secure attachment relationships as its fundamental element in shaping adaptive traumatization and psychological development. The article also delves into the physiological processes involved in emotional regulation and the role of cortisol in disrupting attachment. Finally, the implications of these findings for therapeutic interventions and the challenges of addressing prolonged grief and traumatic responses in clinical settings are considered.

Keywords

Attachment Styles, Traumatic Response, Parental Interaction, Child Development, Emotional Regulation, Cortisol, Grief Coping, Neurobiology, Therapeutic Interventions

1. Introduction

The years of early childhood are the most important for forming the further emotional and psychological development of a person [1]. In John Locke's philosopher view the human mind at birth is a tabula rasa or blank paper on which details and frameworks of thinking and behavior are written by experiences [2]. In this context, parental interactions are the most critical determinant because they contribute to the development of attachment styles that define a child's ability to cope with emotional adversity throughout their lives, including the loss of a parent [2].

Attachment theory formulated by John Bowlby postulates that relationships with the first figures in the child's life are essential for the formation of the child's security. These bonds or attachment styles are divided into two groups, namely: secure attachment style, avoidant attachment style, and ambivalent attachment style [3]. In secure attachment, the child has learned to depend on the caregivers because they have been responsive to his needs. On the other hand, avoidant attachment stems from caregiving that is characterized by emotional unavailability, hence the child is forced to become independent and emotionless. Children in ambivalent attachment are not sure of the availability of caregivers and therefore they are insecure [4]. The theoretical perspective discussed by John Bowlby is the attachment theory, which namely explains how interactions between caregivers and children influence the human's emotional and psychological well-being, especially in stress and trauma situations. According to Bowlby, attachment is the unique partnership between a child and his or her caregiver which determines how the child will address love relationships and aggression as well as how he or she handles stress in the future. This theoretical framework is important for an exploration of how attachment organization elaborates and influences children's responses to trauma and their posttraumatic symptomatology in terms of secure, anxious, avoidantly, and disorganized patterns.

From attachment theory as postulated by Bowlby, the child uses the primary caregiver as a haven of safety from whom he or she ventures out into the world to play, or goes whenever he or she feels hurt. This provides them with a secure environment and assurance that makes a child develop a secure attachment, which is important for a child's psychological and emotional well-being. Specifically, when a child is traumatized, it was found that children who are securely attached are likely going to show signs of security. Are likely to turn to protection and rely on malingering and seeking support in a reasonable manner reflecting healthy coping with stress. One gets to know how to manage stressful events and create a positive self-concept from being securely attached.

Preschool children characterized by insecure patterns of attachment—anxious, avoidant, or disorganized—are doomed to confront major difficulties in coping with trauma. Anxious attachment mainly manifests by increased attention to the caregivers' accessibility and the importance of the attachment relationships, and it leads to heightened anxiety levels and poor stress regulation. These children may become overwhelmed by traumatic events and may develop a tendency to worry or cling a great deal because the attachment system never 'shuts down' when threats are perceived.

Whereas, avoidant attachment refers to a situation where the defendants are emotionally detached and completely rely on themselves. In traumatic events, avoidantly attached children are likely to dampen his or her feelings and refrain from seeking comfort. This suppression may result in putting up negative coping mechanisms as their defense mechanism such as denial or lack of feeling which interferes with the effective handling of trauma.

Insecure attachment defined by Mary Ainsworth as a residual state of unpredictability is linked with disorganized and frightening caregiver behavior which leads to, correspondingly, disorganized and nonpredictable ways of coping with stress. In children with disorganized attachment, the patterns of behavior are mixed up and such children are disoriented in friendship. The experiences of the traumatized child may be characterized by confusion, fear, and the absence of rational treatment and management that may help him or her handle events effectively [5].

Using Bowlby's attachment theory as the frame of reference, it is possible to view the effects of parental behavior on child development and reactions to trauma. Such insights help to capture how the secure and insecure patterns of attachment affect the manifestation of coping patterns and subsequently create efficient approaches to helping children suffering from trauma. Promoting secure base relationships and attending to the distinct needs of children with insecure organized attachment patterns will positively impact one's ability to adapt and regulate after trauma with additional benefits for general psychological functioning [6].

This paper seeks to briefly discuss how these styles of attachment are not simply psychological phenomena but are part of the neurological makeup of the child. At

the core of this discourse, is the focus on the nervous system that influences emotional and stress responses, including the vagal nerve, the amygdala, and the hypothalamic-pituitary-adrenal (HPA) axis [7]. There is the vagal nerve which is part of the parasympathetic nervous system that is involved in the regulation of the stress response mechanism and a return to the state of rest. The amygdala which helps in the processing of emotions such as fear and pleasure works hand in hand with the frontal cortex in the processing of the stimuli both at the conscious and unconscious levels. On the other hand, the HPA axis has the role of synthesizing and releasing cortisol which is very vital in the stress response system [8]. Stress regulation as well as responses to trauma are two areas where both the vagus nerve as well as the amygdala come into play when dealing with attachment styles. Knowledge of these components will enhance the understanding of the neurological process of human attachment, which was theorized by John Bowlby. Notably, the vagal nerve influences the person's emotional behavior by controlling the stress response as well as supporting the sensations of protection and serenity. When it comes to the vagal functions of attachment styles, certain considerations will be useful. In more secure attachment, children are capable of a more optimal vagal regulation which will help them go back to baseline after exposure to stress or trauma. This competence is related to the stimulation they get from their primary parent, with the right and consistent stimulation which maintains the child's internal organization. The vagus nerve when well-developed helps in the management of one's emotions and whenever something tragic happens the child is easily relieved of lots of stress. On the other hand, children with insecure attachment including the anxious or avoidant attachment would demonstrate an unhealthy vagal flexibility. For example, children with anxious attachment styles could be easily overstimulated, and therefore, have perceived problems with self-soothing since their vagal tone does not dampen down their stress response. This kind of situation may result in continued caution and an increased tendency to respond emotionally. Likewise, disoriented children with avoidant attachment may have tended to dampen their stress biological reactions, which may lead to vagal unresponsiveness that can make it difficult for such children to be calm after stress.

The amygdala is a round group of neurons in the temporal part of the brain and is involved with emotion, particularly the feeling of fear and threats. It is a very important part of the brain that is involved in processing stressful or traumatic stimuli and can be referred to as an emotional alarm that triggers such reactions as fear and anxiety. The component related to attachment styles is very important in the case of the amygdala. There is another perspective by which securely attached children appear to have a better-regulated amygdala such that they experience stress in a more balanced manner and can regulate fear and anxiety. This regulation is also partly because of the kind of caregivers, who ensure that the household provides stable functioning of the amygdala. Instead, children who have insecure attachment strategies can have intensified or dysregulated amygdala activity. For example, children with an anxious-ambivalent attachment may have

an over-aroused amygdala and thus will be more sensitive to stress and more prone to panic attacks. Their amygdala may be overdeveloped mainly because they had occasional or erratic caregiving experiences. On the other hand children with avoidant attachment bring about interactivity on the amygdala which leads to low sensitivity to stress and in most cases, they are likely to deny feelings. The knowledge of the functioning of the vagus nerve and amygdala can be useful for the perception of the effects of attachment behaviors and reactions to trauma. Secure attachment helps regulate the interaction between the systems that were described neurobiologically, to protect the vulnerable child and help him or her effectively manage stressors. The two main forms of insecure attachment, namely anxious and avoidant attachment, can destabilize this balance and infuse stress and trauma management. In light of these neurobiological factors, it is less confusing as to how to structure support and treatment to enhance children's emotional functioning and modify traumatic responses depending on their attachment organization.

Another important concern of this paper is that of reviewing how the disruption of such systems, especially by high cortisol levels may impede the proper unfolding of attachment relationships and result in long-term psychological repercussions. For instance, children who have been exposed to chronic stress or trauma or the loss of a parent may develop an overactive stress response system; the typical patterns of attachment are disrupted and insecure attachment styles are the end product. These disruptions can present in multiple ways such as: difficulty in managing emotions, social withdrawal, and learning disability that shape the way the individual handles loss and trauma in adulthood.

Further, this paper also focuses on Mary Ainsworth's study of attachment theory which helps to define how one's responses to mourning and grieving are influenced by one's relationship with the caregivers in the early years of life. Ainsworth's "Strange Situation" experiment that highlighted secure, avoidant, and ambivalent attachment patterns are interpreted about how these patterns affect responses to parental death. Thus, the children with secure attachments are likely to be better equipped to cope with the grief in a healthy manner because their sense of security has been established earlier. While the securely attached could easily work through their grief and be ready to move on, the avoidant or ambivalent might have more difficulties, possibly developing prolonged or complicated grief that might hinder their everyday functioning.

Following these connections, the paper also reflects upon the general impact on therapeutic practices. Knowledge of neurobiology and psychopathology of attachment and trauma is beneficial for creating a specific treatment approach to improving the experience of loss and trauma for clients with various types of attachment. For instance, psychodrama and drama therapy are described as methods that may be useful in aiding traumatized people in rewriting their traumatic memories and regaining control over the feeling processes.

In conclusion, this paper aims to synthesize data originating from neurobiology,

psychology, and clinical practice to provide a synthesis of how the attachment formed within infancy impacts the clients' reactions to trauma and grief. Consequently, this study will seek to explore how such biological processes and developmental patterns can affect one's capacity to benefit from therapy as an adult, and thus add to the existing debates on the effects that early childhood experiences may have on one's overall emotional and psychological wellbeing later in life.

2. Attunement vs. Attachment

Attunement is a bidirectional process between the parent and the infants or children. It is a measure of how receptive one person is to the emotions of another in a relationship. Parental attunement is the ability of the parent to understand their infant or child's physiological and psychological needs, significantly reducing trial and error in determining the appropriate mode of action to meet their infant or child's demand [9]. Despite serving the same evolutionary goal as attunement, attachment does not require the ability of the parent to sense the infant's or child's emotions. Evolution prewiring attachment and attunement within the infants' or children's neural circuitry allows them to activate a complementary neural circuitry within their caregiver. The function of the complementary neural circuitry is to provide psychological and physical comfort to the child or infants. **Figure 1** depicts the impact of attachment styles during the child's development.

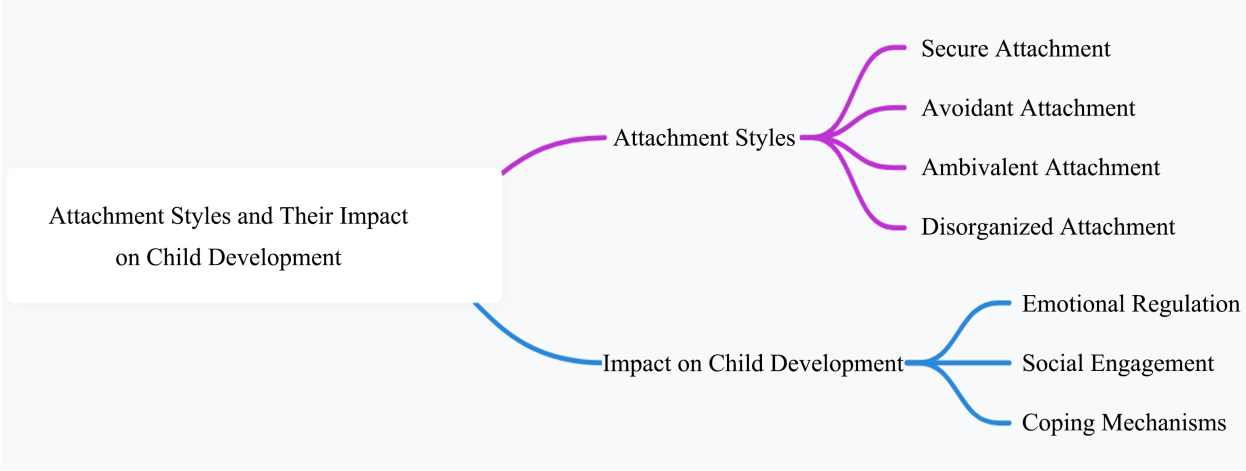

Figure 1. Attachment styles impact on the development of a child.

3. Parental Interaction Teaches the Child and Infant to Self-Regulate

The parent providing comfort and care to the infants or child teaches them how to regulate emotion by stimulating the development of vagal nerves and mirror neuron neural networks and the interconnection of ventral vagal nerves and mirror neurons. Regulating emotion refers to but is not limited to infants or children discovering what facial expressions can cause harm and which one will not via

learning and mirroring their parent's facial expression. Darwin argued that some facial expressions are universal and that emotions are biological processes communicated via facial expressions. Some of these facial expressions are signals of danger, such as when a dog shows their teeth or when a bird puffs up. In contrast, other facial expressions can signal safety, security, or love. Mirroring their parents' facial expressions allows them to communicate and develop more profound and complex communication skills. The ventral vagal nerves and mirror neurons produce and learn how to interpret complex facial expressions [10]. As infants or children interact with their parents, the ventral vagal nerves form new neural connections and become more myelinated as they age. At the same time, mirror neurons form new connections during this process.

4. Vagal Nerve's and Amygdala's Role in Learning and Regulating Emotions

Currently, the vagus nerve, which belongs to the groups of the autonomic nervous system is often discussed in conjunction with the regulation of emotions. Although such theoretical perspectives and indirect findings point to a relationship between vagal nerve activity and effects, a direct evidence base is still scarce. The vagus nerve is a part of the body's parasympathetic division which deals with the "rest and digest function of the body. It affects many other functions affecting the heart rate, digestion, and respiratory rate among others. Based on theoretical formulations, it was presumed that cranial vagal outflow could be involved in the regulation of emotions by altering the physiological substrates that underpin the effect [11]. For instance, enhanced vagal tone means fewer than thirty-five heartbeats per minute and higher emotional regulation implying that a well-functioning vagus nerve might bolster emotional stability.

Especially, the vagal tone has been investigated with emotional regulation. Vagal tone is a measure of the level of activity of the parasympathetic nervous system, of which HRV is a measure. People with higher HRV have reduced emotional problems resulting in better psychological well-being and improved vagal function in the case of the nervous system [12]. However, while there are some theoretical links between the vagal nerve and emotional regulation, there is very little concrete evidence for the actual processes involved in this area. Some investigations were undertaken to investigate the role of vagal tone on emotional processes but most of them use mediate indicators and correlation data.

As mentioned above, a large number of papers investigate Heart Rate Variability (HRV) as an index of vagal tone. Some research has shown that there is a positive significant relationship between HRV and positive affect and a negative significant relationship between HRV and negative affect which includes anxiety and depression. Given that higher HRV is associated with better self-regulation, these results imply that vagal activity of the heart could be associated with self-regulation of emotions, however, HRV is a complex measure including not only vagal tone but respiratory rates and overall balance of the sympathetic and

parasympathetic systems. Therefore, even though HRV gives valuable information, it nakedly does not give proof of the involvement of the vagus nerve in managing an emotional state [13].

Vagus nerve stimulation is a clinical practice applied in epilepsy and depression thus giving a more direct way of investigating the effects of the vagus nerve. Several studies have also demonstrated the effectiveness of VNS in enhancing the mood and cognition of depressed patients. This gives insight that the vagus nerve might be implicitly involved in the regulation of emotions. However such studies are very often compromised by an intricate integration of functions that implicate the numerous neurobiological systems involved, particularly when it comes to the analyses of the effects of the stimulation of the vagus nerve on the regulation of the emotional processes.

Interventions following formal experimental designs that alter activity in the vagal nerve by ways such as transcutaneous vagus nerve stimulation (tVNS) or breathing practice yielded inconsistent outcomes. A few investigations proposed that Seriant Vagus Nerve Stimulation may affect mood and emotional processing in the individual, this is based on the fact that tVNS may have disparities in outcomes amongst clients [14]. Furthermore, most of these studies are conducted with a small number of participants and or with methodological flaws, which makes interpreting study results a challenge.

Evidence that the vagal nerves play a role in learning is due to their effects on the amygdala. Some research reported that stimulating rat and human vagus nerves resulted in increased retention, and another study has indicated that stimulating the vagal nerve resulted in the release of epinephrine within the amygdala [15]. Epinephrine seems to increase memory through its action on the amygdala, and blocking epinephrine from binding to receptors within the amygdala negated the increase in memory. Despite some experts arguing that the vagus nerve is responsible for learning, others do not agree. Anatomists agree that electrically stimulating the vagal nerve would result in elevated levels of interleukin (IL)-1β within the hippocampus [16]. Interleukin (IL)-1β plays a role in learning, and there is a positive correlation between learning and elevated levels of interleukin (IL)-1β [17].

Mirror neurons, responsible for executing a particular facial expression, also fire when the infant or child observes their parent execute it. As the infants or children interact with the parent and get better at discriminating and understanding emotion via facial expression, their mirror neurons that interpret and express these same emotions form more elaborate and complex neural networks. The infant or child learns to predict the intent of facial expression due to the fine-tuning of mirror neurons and learns to regulate their emotions due to the correct developmental track of synaptogenesis and myelination of the ventral vagal nerves. The ability of ventral vagal nerves to inhibit the HPA axis from releasing cortisol is not as effective in an infant relative to an adult. The role of different nerves including vagal, mirror and HPA axis has been demonstrated in **Figure 2**.

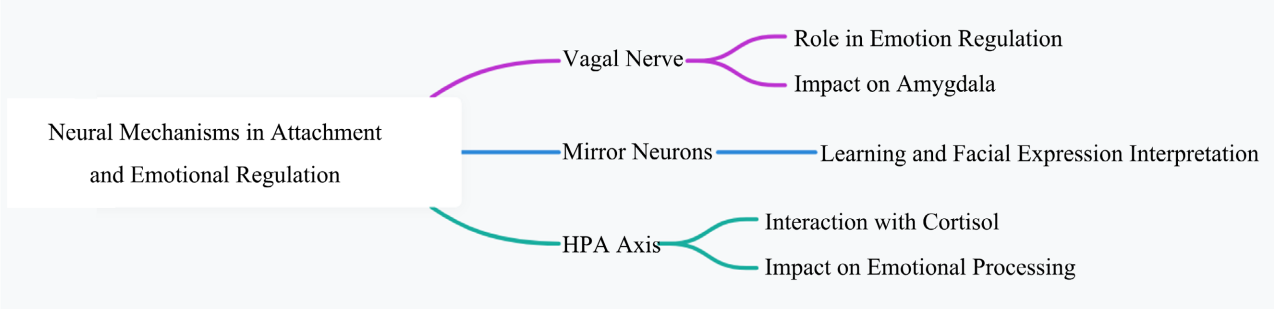

Figure 2. Neural mechanism and their impacts.

5. Vagal Nerve Activation and Its Role in Modulating the HPA Axis and Emotional Processing

The mechanism of action in which the ventral vagal nerve inhibits the HPA axis is by turning on the low-tone parasympathetic nervous system. It is essential to note that the ventral vagal nerves have dual functions, as the ventral vagal nerves can also activate corticotrophin-releasing neurons within the paraventricular nuclei. Electrical stimulation of vagal nerves results in elevated levels of interleukin (IL)-1β and corticotrophin-releasing hormones within the hypothalamus [18] [19]. Increased levels of interleukin (IL)-1β within the hypothalamus can stimulate the HPA axis, with the PVN implicated as one of the mediating variables [20] [21].

In vivo, gut serotonin can stimulate the vagal nerve to transmit neural signals within the brain [22] [23]. Serotonin can also stimulate the HPA axis to release cortisol by directly stimulating AgRP and POMC neurons within the paraventricular nuclei of the hypothalamus; the stimulation of AgRP and POMC neurons results in the activation of the HPA axis [24] [25]. Serotonin stimulates serotonergic axons inside the amygdala and affects how the amygdala unconsciously and consciously processes environmental stimuli [26] [27].

6. Unconscious and Conscious Processing of Stimulus

The amygdala unconsciously processes stimuli before it sends the information to the frontal lobe for conscious processing. Primary emotions are primal and responsible for the unconscious processing of stimuli. Secondary emotion within the frontal lobe is responsible for the conscious processing of stimuli. Secondary emotions are different from primary emotions in that secondary emotions are learned, while primary emotions are in the superposition of being innate and learned. As a result, the infant's amygdala is entirely responsible for processing the infant's or child's world until the frontal lobe undergoes years of neuroplasticity to be able to carry out the same function. Neuroscientists once believed that the amygdala only regulates emotion related to the fight-and-flight response, but they now know that it can regulate social cognition [28]. For example, the amygdala plays a role in learning positive and negative facial expressions [29].

Suppose the facial expression is happy (*i.e.*, positive), associated with safety and

security. In that case, the brain's ventral tegmental area produces dopamine, which travels to the mesolimbic system to activate the nucleus accumbens [30]. The nucleus accumbens processes the positive facial expression and simultaneously learns whether it is rewarding [31]. If the stimuli are rewarding, the infant or child will seek out such facial expressions from their caregivers in the future and avoid the aversive ones. Once the nucleus accumbens processes the stimuli, it sends them to the amygdala for further processing. Once the amygdala decides that the facial expression is positive, it sends the information to the left frontal medial cortex for processing, and it activates the ventral vagal nerve of the parasympathetic nervous system. The activation of the ventral vagal nerves activates social engagement. Social engagement plays a role in building attachment with their caregivers and activating other higher brain processes.

In contrast, if the facial expression is sadness, fear, or anger (*i.e.*, negative facial expression), it would be stressful stimuli. The nucleus accumbens can identify aversive facial expressions as a stress stimulus. The nucleus accumbens then sends the information to the amygdala for further processing. The amygdala, identifying the information as aversive, sends the information to the right frontal medial cortex for further processing and activates the sympathetic nervous system (fight or flight response).

Wadsworth *et al.* (2019) stated that stress could coactivate the sympathetic-adrenomedullary and hypothalamic-pituitary axis. The coactivation of the sympathetic-adrenomedullary axis and the hypothalamic-pituitary axis or any one of the sympathetic nervous systems to a traumatic event makes it more likely that a person will interpret a stimulus as being aversive and more likely another traumatic response to a crisis event because of somatic markers [32]. Somatic markers refer to autonomic physiological and psychological arousal that aids the person in deciding. Primary and secondary emotions are somatic marker types that influence how the person views and interprets the stimulus. If the somatic marker results in the person viewing the stimulus as aversive, neuronal action potential will occur within the paraventricular nuclei. The action potential of neurons within the paraventricular nuclei classically stimulates the SAMs axis and/or HPA axis. Short-term stress activates the sympathetic-adrenomedullary axis, while long-term stress activates the hypothalamic-pituitary axis. Activating the paraventricular nuclei triggers the release of corticotropin-releasing hormone that eventually makes its way within the portal vein, and the portal vein conveys it to the anterior pituitary. Once within the pituitary, the corticotropin-releasing hormone triggers the anterior pituitary to release ACTH. ACTH then travels within the systematic circulation that eventually reaches and triggers the zona fasciculate of the adrenal cortex to release and produce cortisol.

7. The Production and Release of Cortisol

ACTH binding to and activating MC2R receptors helps with the production of cortisol, which involves the downstream activation of PKA and cAMP [33]. The

ACTH stimulation of cortisol also involves increasing the amount of LDL receptors on the surface of cells within the zona fasciculata and zona glomerulosa [34]. LDL binding to LDL receptors permits cholesterol to enter the cell. The conversion of cholesterol to cortisol is not a zero-order reaction. It is not zero-order because the reaction rate depends on the concentration of substrates and enzyme availability, following Michaelis-Menten kinetics. As the concentration of cholesterol increases, the rate of conversion to cortisol will increase until the enzyme becomes saturated. This saturation means that, at high substrate concentrations, the reaction rate reaches a maximum and does not increase further. The rate of converting cholesterol into pregnenolone, which is regulated by CYP11A1, is influenced by the availability of cholesterol within the mitochondria. Steroid Acute Regulatory Protein (StAR) and Translocator Protein (TSPO) are involved in transporting cholesterol into the mitochondria but do not directly affect the enzymatic conversion process. They are essential for providing the substrate necessary for the reaction to occur [35] [36]. Once cholesterol is within the mitochondria, CYP11A converts it into pregnenolone [37]. Then, 3β-hydroxysteroid dehydrogenase (3β-HSD) converts pregnenolone into progesterone. Some Pregnenolone and progesterone produced within the zona glomerulosa eventually enter the zona fasciculata [38]. CYP17 converts pregnenolone into 17-hydroxypregnenolone. 3β-hydroxysteroid dehydrogenase then converts 17-hydroxypregnenolone into 17-hydroxyprogesterone. CYP21 then converts 17-hydroxypregnenolone into 11-deoxycortisol. CYP11B1 converts 11-deoxycortisol into cortisol within the mitochondria of zona fasciculata cells. Cortisol then exits the mitochondria into the bloodstream. Cortisol then travels the systematic circulation and can turn off the production of cortisol by inhibiting corticotrophin-releasing hormones via its action on neurons within PVN, inhibiting the release of cortisol via preventing the pituitary gland from releasing ACTH [34]. Cortisol can also cause dysregulation of the sympathetic nervous system by its action on the amygdala if the negative feedback loop is not working properly [39]. The overproduction of cortisol can result in an increased cortisol level, and an increased cortisol level lowers someone's threshold to experience traumatic events, decreases someone's resilience in recovering from trauma, and can disrupt the attachment process within children.

8. An Elevated Cortisol Level Can Disrupt the Attachment Process

The dysregulation of the sympathetic and parasympathetic nervous systems can negatively impact development, such as the ability to form attachments with the caregiver. The parasympathetic and sympathetic nervous system elevating visceral response (Somatic markers) can negatively impact the ability of infants or children to form an attachment with their caregiver. Elevated visceral response (such as somatic markers associated with fear) sends information regarding parental interaction to the right ventral frontal medial cortex. The amygdala sending

information to the right ventral frontal medial cortex can result in the infant or child associating parental interaction with fear (Table 1). While infants' or children's brains are not born with the innate ability to experience fear at a young age, infants or children can experience fear, as Simonelli A. (2013) reported that infants can experience post-traumatic stress disorder. It is also important to note that the parent should expose their infants or children to fear as it is a necessary part of development [40]. However, they should also consider their age when exposing them to fear, as exposing them too soon can cause dysregulation of the vagus nerve and sympathetic nervous system. The vagus nerve and sympathetic nervous system dysregulation can harm the attachment process due to elevated cortisol levels. Harming the attachment process can have negative developmental consequences. However, the infant's and child's brains are resilient in that they can still form an attachment with an abusive caregiver due to having the ability to form abuse-related attachment relationships.

Table 1. Impact of parental interaction on child development.

Aspect of Development	Role of Parental Interaction	Neural Mechanisms Involved	Long-term Impact on Child Behavior
Emotion Regulation	Teaches self-regulation of emotions	Vagal Nerve, Mirror Neurons	Better emotional control, resilience
Social Engagement	Facilitates building of social bonds	Ventral Vagal Nerve, Amygdala	Strong interpersonal relationships
Cognitive Development	Supports learning and cognitive growth	Prefrontal Cortex, Hippocampus	Enhanced learning abilities, curiosity

The neurochemical that is responsible for infants or children forming attachments with their caregivers regardless of the quality of care and abuse is norepinephrine [41]. The painful and non-painful maternal interaction with the infants or child stimulates locus coeruleus to activate the olfactory bulb via the production and release of norepinephrine. During parental interaction, parental odor stimulates the olfactory bulb to relay neural signals, resulting in neuroplasticity of neural circuitry, a neural representation of the caregiver and their odor. Any interaction with the caregiver results in the release of norepinephrine—the release of norepinephrine results in the stimulation of the olfactory bulb. Once the olfactory bulb is activated, high cortisol levels result in the olfactory bulb and the anterior and posterior piriform cortex activating the amygdala, resulting in the infants or children associating the parent with fear.

In contrast, considering that the infants' or children's cortisol levels are too low for the olfactory bulb and the anterior and posterior piriform cortex to activate the amygdala would result in the infant or child identifying and learning the caregiver's odor without associating it with fear. The infant or child does not associate the parent with fear because the amygdala receives neural stimulation; however,

neuroplasticity responsible for associating the parent with fear does not occur due to the infant or child's naturally occurring low cortisol level and parental interaction reducing cortisol level. An excellent example of this is avoidance learning. During the critical attachment period, painful stimuli cannot interrupt the bond between the mother and the infant or child [41]. However, after the attachment period has ended and elevated cortisol levels, avoidance learning can result in the infant or child avoiding the parent due to associating the parent with pain and fear.

Hence, secure attachment can easily be observed in the child when the caregivers always meet the child's stress regulation needs. It reduces cortisol release during stress thus it leads to proper regulation of emotions about stress. On the other hand, people with Insecure attachment may have received rather inconsistent or otherwise, non-optimal care during early childhood and, thus may have dysregulated cortisol [42]. In addition, the extended duration of cortisol in the body among insecurely attached persons has negative implications for their health since they are exposed to the following illnesses: anxiety and depression, as well as an ineffective immune response. We have seen that this dysregulation aids in creating a recycling pattern of stress and maladaptive attachment leading to poor formation of attachment bonds in adulthood. Here we have demonstrated how cortisol and attachment styles point to early caregiver relationships as having a potential for influencing both the stress reactivity and emotions of an individual, which in terms has potential implications for the mental and general health of an individual.

9. Mary Ainsworth's Attachment Style

Another essential developmental psychological approach to classify attachment style among children due to parental interaction comes from Mary Ainsworth. Mary Ainsworth addressed how different types of parental interaction can result in different attachment styles via her strange situation experiment [43]. She experimentally determined if the children would cling to their mother or play with toys and other children in the room, how the children would interact with a stranger without the presence of their mother, and how the children would reunite with the mother post her departure. She developed a tripartite classification. The tripartite classification is three different types of attachment patterns. One type of attachment is secure attachment. Children who exhibit secure attachment were upset when their mother left. They transitioned from negative to positive emotions when their mother returned and explored and played with other children and toys when their mother was present.

Children she classified as avoidant attachment did not seek the mother's comfort or show negative emotions such as anger or fear when their mother left the room [43]. She labeled children as having ambivalent attachment styles when they showed anxiety when the mother was present, did not cry or protest when the mother left, and their mother returning to the room did not comfort them (**Table 2**).

Table 2. Comparison of attachment styles.

Attachment Style	Key Characteristics	Parental Interaction	Child's Behavior in Strange Situation	Coping Mechanisms for Grief
Secure	Comfortable with intimacy; Trusting	Responsive and nurturing	Upset when mother leaves; Comforted upon return	Healthy processing; Open to seeking support
Avoidant	Emotionally distant; Avoids closeness	Unresponsive and distant	Indifferent to mother's departure and return	Suppresses emotions; Avoids seeking help
Ambivalent	Anxious; Uncertain about relationships	Inconsistent; Sometimes nurturing, sometimes not	Anxiety even when mother is present; Difficult to console	Clings to others; Struggles with prolonged grief

10. Mary Ainsworth Attachment and Grief

Parental interaction with the child that shapes the child's neural circuitry will result in one of the attachment styles discussed supra, which influences how the child projects what they learned from interacting with their parent to other people and situations later in life. Attachment style affects how they will grieve their parent's death. Grief is a normal psychological reaction to losing a loved one [44]. However, prolonged grief is when grief lasts longer than twelve months for adults and six months for children under the age of 18. Symptoms associated with prolonged grief must negatively impact the person's ability to function at school, work, and home. The symptoms associated with prolonged grief must also be aberrant for their culture. Common symptoms include feeling that a fragment of their identity has also died with their parent, negative emotions related to their parent's death, and avolition (American Psychiatric Association, 2013). Examples of some of these negative emotions are emotional numbness, shock, disbelief, and denial.

Researchers have demonstrated that Mary Ainsworth's tripartite classification is essential in dealing with grief. Therapists should consider a person's attachment style when selecting coping mechanisms for grief. For example, individuals with an anxious attachment style experience severe shame and guilt when dealing with grief, while individuals with an avoidant attachment type experience complicated grief [45]. Complicated grief is prolonged grief. *Prolonged grief* is persistent grief accompanied by maladaptive behaviors and thoughts. Huh *et al.* (2018) stated that individuals' avoidant attachment style had higher levels of shame and guilt when grieving due to relying excessively on problem-focused coping. Unlike individuals with avoidant attachment styles, anxious attachment styles did not vary in their coping mechanisms for dealing with their grief, and their coping mechanisms did not moderate their grief response [45]. In the 1980s, Bowlby identified grief among individuals with anxious and avoidant attachment styles. Individuals with avoidant attachment styles during grief do not seek help from others, avoid others

during their grief, and are individualistic when trying to regulate their emotions during grief. In contrast, individuals with an anxious attachment style seek others out for acceptance, help to cope with their grief, and worry that their significant other will abandon them during times of need for emotional support [4]. The link between traumatic grief and attachment style is well demonstrated in **Figure 3** below.

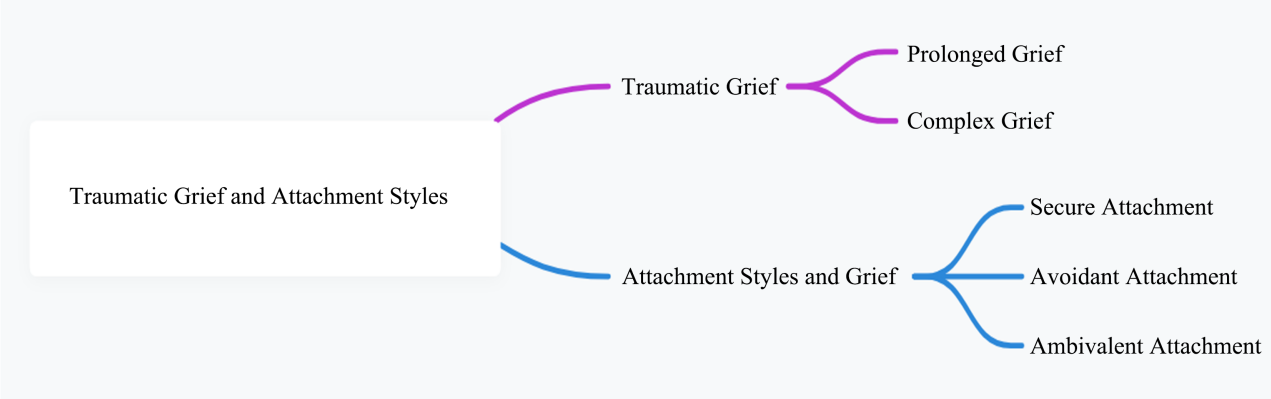

Figure 3. Traumatic grief linked with attachment styles.

11. Death of a Parent Can Be a Crisis Event

The death of one's parent can be a crisis event. The death of one's parent is a crisis event because their parent's death can be unpredictable and tax their resources [46]. The death of a parent can tax a child or adult's resources due to financial costs from all situations related to their parent's death and loss of financial and emotional support from the parent. The loss of social support from the deceased parent makes the child or adult more susceptible to producing a traumatic response from future crisis events [47]. Crisis events typically involve grief. However, crisis events do not always involve traumatic responses. Crisis events only involve traumatic responses when the SAMS and HPA axis are activated. The child may experience traumatic responses if their parent's death was from a traumatic event such as gun violence and other trauma responses producing nonviolent crisis events [48]. If the person experiences grief and traumatic response simultaneously in response to the crisis, the condition is termed traumatic grief [49]. Differentiating traumatic responses from grief can be challenging because they involve pain, anger, and guilt. Clinicians differentiate traumatic response from grief in that only traumatic response includes the feeling of terror and the inability to talk about the feelings or event responsible for their traumatic emotion. A traumatic response from a crisis event can result in the child or person developing post-traumatic stress disorder, Developmental traumatic disorder, or Complex post-traumatic disorder.

12. Billable and Non-Billable Trauma-Related Disorders

Developmental trauma disorder is the child version of Complex post-traumatic

disorder. Children with developmental traumatic disorder, adults with Complex post-traumatic disorder, and individuals with post-traumatic stress disorder share three core symptoms. The three core symptoms are re-experiencing, avoidance, and hyperarousal (Kaminer *et al.*, 2005). Children with developmental traumatic disorder and adults with Complex post-traumatic disorder also share three unique additional symptoms that depart from post-traumatic stress disorder. The three additional symptoms are failure to regulate their emotions, identity problems, and building close relationships [46]. Clinicians may want to substitute post-traumatic stress disorder for developmental, traumatic, and complex post-traumatic disorder because they cannot get paid for nonbillable diagnoses. A non-billable diagnosis is a medical condition not listed within the DSM5 or ICD-11. During such substitution, medical treatment for the three core symptoms will not raise a red flag when substituting post-traumatic stress disorder for developmental, traumatic disorder, or Complex post-traumatic disorder because the said treatment is consistent across all three disorders. However, clinicians rendering medical treatment to someone with PTSD for the three additional symptoms unique to developmental and complex traumatic disorders can result in a red flag due to the fraud and abuse law outlined by CMS. CMS made it clear that it is fraudulent to bill Medicare and Medicaid for unnecessary medical treatment, and providing treatment for symptoms not associated with PTSD to someone with the condition can be viewed as unnecessary medical care. Two examples of fraud and abuse discussed infra are Healthcare fraud, 18 U.S.C. § 1347, and false claim, 31 U.S. Code § 3729.

13. Misrepresentation of Information to Get Money from a Healthcare Benefit Program

Health care fraud, 18 U.S.C. § 1347, is when someone collects money from a government health care benefit program under pretense. Common examples of healthcare fraud are not charging providers deductibles, diagnosing someone with a diagnosis they do not have, and unbundling (Findlaw.com, n.d.). Unbundling is to bill the health care benefit programs separately for each medical procedure because it would cost more than bundling all the bills within a single bill. Mr. Hong (United States v. Hong, 2019) could not bill for acupuncture and massage therapy because Medicare did not cover them. To get reimbursed by Medicare, he substituted the ICD-11 code for massage and acupuncture therapy with the ICD-11 code for physical therapy. Substituting massage therapy and acupuncture code with physical therapy code constitutes misrepresentation of information under the pretense to get money from a health care benefit program. The False Claim Act, 31 U.S. Code § 3729, is the tool the DOJ uses to get restitution for any damages associated with health care fraud. Restitution that the perpetrator can repay the U.S. government is $10,000 for each false claim and treble damage (United States v. Hong, 2019). Treble damage is the violator repaying three times the amount of money they fraudulently billed the government health care benefit program.

Substituting PTSD for Complex or developmental traumatic disorders differs from substituting massage and acupuncture therapy for occupational therapy because massage and acupuncture therapy are listed within the ICD, while Complex and developmental traumatic disorders are not. The other difference is that mental health professionals may approve of using PTSD because Complex and developmental post-traumatic disorders are not within the ICD or DSM 5. Another significant difference is that the DSM-5 and ICD-11 allow for PTSD. Suppose the person has additional symptoms associated with Complex or developmental post-traumatic disorders. In that case, the clinician can use V codes within the DSM 5 and Z codes within the ICD-11 to treat symptoms that depart from PTSD. The V and Z codes aim to provide medical care to clients that cannot be pigeonholed to a specific diagnosis [50].

14. Psychopharmacology for Trauma-Related Disorders

Olanzapine is a fat-soluble drug that clinicians use to treat people with PTSD, Complex post-traumatic disorders, and developmental traumatic disorder partly due to its effects of reducing cortisol levels [51]. While the mechanism of olanzapine reducing cortisol levels is multifactorial, researchers believe that olanzapine lowers cortisol levels by preventing the hypothalamus from releasing corticotropin-releasing hormone due to blocking serotonin from binding to serotonin receptors within the hypothalamus [22]. The elevation of cortisol level may result in the person being in an aroused state that makes it more likely the person may relive the traumatic event and more likely another traumatic response, so olanzapine lowering cortisol level would make it less likely the person relive another traumatic event, another traumatic response to a crisis event, and less likely to be hyper-aroused. There is also reasonable implication of using olanzapine to facilitate parental attachment with infants or children with elevated cortisol levels due to a trauma response. However, psychopharmacological research within this area is lacking. Olanzapine's mechanism of action of reducing cortisol levels has to do with its ability to out-compete dopamine for dopamine receptors within the ventral tegmental area, nucleus accumbent, and mesolimbic system. While taking olanzapine, a trauma response is less likely due to dopamine not being able to activate the necessary amount of dopamine receptors that are responsible for activating the neuron within the mesolimbic pathway that activates the nucleus accumbens, which in turn activates the amygdala, and amygdala in turns activates the HPA and SAMs axis. Olanzapine also inhibits the sympathetic nervous system via antagonizing serotonin [24]. Olanzapine antagonizing serotonin receptors also stabilizes the person's mood because serotonin stimulates the sympathetic nervous system. An overactive parasympathetic nervous system can cause low serotonin levels, and low serotonin levels can cause depression [52]. Therapeutic interventions used for traumatic-related disorders including psychotherapy and psychopharmacology are displayed in **Figure 4**.

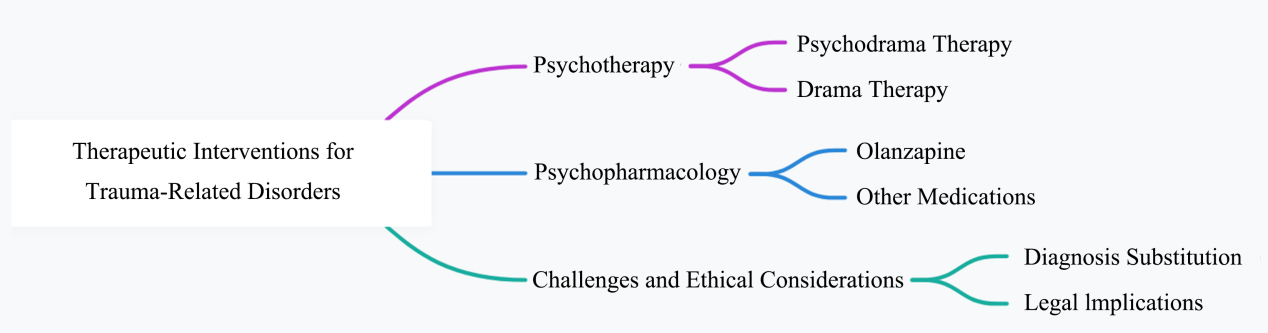

Figure 4. Therapeutic interventions applied for trauma disorders.

15. Psychotherapy

Psychodrama and drama therapy are tools a therapist can use to help clients relive their traumatic events by acting. The goal of having the client relive their traumatic event is for them to regain mastery of their emotions. Gaining mastery of the traumatic event is controlling their traumatic response, such as dealing with stimuli that remind them of the traumatic event and regaining control of their visceral emotions. Teaching the client how to act out positive emotions is essential when using psychodrama and drama therapy. Getting some clients to act out certain emotions may be difficult due to their gender and cultural background. In some cultures, it may be frowned upon for males to show emotion in which they are vulnerable and need help [53] [54]. As a result, it may be challenging to get them to perform an act in which they were vulnerable, overcome such vulnerability, and the positive emotion they felt after such accomplishment. Another reason the therapist should teach the client how to act before having the client act out aversive emotion is due to the Yerkes-Dodson Law. According to Yerkes-Dodson Law, people can do simple tasks during high emotional arousal, and people can only do complex tasks under low emotional arousal [55]. Using Yerkes-Dodson law, if acting is not easy, the client cannot act out intended emotions when reliving the traumatic event or being hyper-aroused due to sympathetic nervous system activation. Once the client learns how to act, they will find it easier to control their facial expression during elevated arousal to act out the intended emotion the therapist wants them to act out. Intended emotion can be aversive or pleasant. Aversive emotions are usually negative emotions or emotions related to the traumatic event in some way, while pleasant emotions are happy emotions. The therapist can teach the client how to self-regulate by making the client act out traumatic events that produce aversive emotions and then make them act out positive events in their memory (psychodrama therapy) or TV shows (drama therapy) [56] [57].

Acting out emotions helps the person regain function and understanding of their emotion, facial expressions, and social situations related to their parent's death. Acting helps the person gain control of their emotion due to consciously activating neural circuitry for the intended emotion. The intended emotion will compete with the neural circuitry for the emotion they are experiencing. The motor cortex consciously activates the vagus nerves to stimulate facial muscles to

make associated facial expressions of the intended emotion, activating neural circuitry for the intended emotion. Neural circuitry for the intended emotion refers to mirror neurons and neurons within the subcortical cortex. Stimulating the mirror neurons within the amygdala and neurons within the subcortical cortex will activate the neural circuitry of the intended emotion and the person genuinely and unconsciously expressing the intended emotion [55]. The subcortical cortex involves structures such as the basal ganglia and subtantia nigra and is responsible for the involuntary control of facial expression. The therapist should gradually use psychodrama and drama therapy to expose the client to aversive emotion and thought related to their parent's death when the client is ready, and only after teaching them how to control their visceral emotion by acting. Psychodrama and drama therapy are vital to getting the client ready for therapy and closing the client up after therapy. The therapist can use psychodrama and drama therapy to disengage the client's sympathetic nervous system by acting after they have relived the traumatic event at the end of therapy.

16. Conclusions

The findings presented in this paper reveal that an infant's attachment is a vital determinant of her or his responses to traumatic occurrences such as the loss of a parent. This paper discusses an understanding of the neurobiological components of the vagal nerve, amygdala, and cortisol about emotional regulation and the formation of attachment patterns. Interference of these processes due to the increased cortisol levels during traumatic events can result in enduring emotional and psychological conditions such as Prolonged or Traumatic Grief.

Drawing from Mary Ainsworth's framework of attachment style, it is possible to understand how people have different ways of coping with grief and trauma. A secure attachment, which is brought about by parental care promotes healthy ways of dealing with grief and trauma while avoidant or ambivalent attachment to the parents predisposes a person to adopt unhealthy ways of coping with grief and trauma. These findings underscore the need for early intervention and specialized treatments that incorporate an individual's attachment pattern in the treatment of mourning and trauma-related disorders.

For clinical practice, knowledge about attachment styles and traumatic responses can help to improve the therapeutic process. The combination of psychodrama, drama therapy, and psychopharmacological methods, linked with the utilization of emotional regulation and attachment repair, may be seen as a potential way to regain control over one's emotional state and prevent future crises. This paper encourages further research on the time effects of attachment styles on emotional health and the establishment of proper therapeutic interventions that will consider the patients' attachment styles.

Thus, this study advances knowledge of humans' early development and how the experiences of attachment and trauma shape their future emotional and psychological well-being.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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