Journal: Texila International Journal of Academic Research
Publisher: Texila International Journal
ISSN: 2520-3088
Volume 13 Issue 2, 2026
DOI: 10.21522/TIJAR.2014.13.02.Art009

Chemical Addiction in Intimate Relationships: The Neurobiology of Trauma Bonding and Emotional Dependency

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Abstract

The neurobiological reinforcement of trauma bonding and emotional dependency in intimate relationships is increasingly being understood as a process, as opposed to a purely psychological pattern. This review integrates evidence on fifteen peer-reviewed articles to investigate the interaction between dopaminergic reward systems, oxytocin-mediated bonding systems, cortisol-induced stress responses, and reinforcement-learning processes to maintain maladaptive attachment despite the occurrence of relational inconsistency, conflict cycles, or intermittent affection. The key goal was to combine the results of addiction neuroscience, attachment theory, and trauma psychology to have a single explanation of the continuity of the emotionally dysregulated relational bonds. Five major databases were searched in a structured literature search, and 612 records were located; 15 records were included in the study that was analyzed using thematic synthesis based on reward processing, attachment neuropeptides, HPA-axis regulation and cognitive-emotional conditioning. Findings indicate that trauma bonding is the result of a vicious cycle of increased dopamine-based reward sensitivity, oxytocin-based trust and emotional proximity, cortisol-related hyperarousal, and attachment-based cognitive distortions. These interdependent systems produce the addiction-like patterns of stress, relief and reconnection that strengthen emotional dependence in spite of misery. The review finds that trauma bonding is a neurobiological phenomenon that is multisystemic and needs to be addressed with trauma-informed and neurobiologically oriented therapy in order to be effectively treated. More studies are required to develop specific clinical resources and models that would help to overcome these complicated relational dynamics.

Keywords: *Cortisol Stress Physiology, Emotional Dependency, Intermittent Reinforcement, Oxytocin and Attachment, Reward Neurocircuitry, Trauma Bonding.*

Introduction

Emotional dependency and trauma bonding in intimate relationships have become a more and more discussed topic in the fields of neuroscience, psychology and relationship research. Trauma bonding refers to inertial emotional bonding which is established between people who undergo the relationship of affection and abuse, developing a paradoxical yet strong bond by a neurobiological reinforcement and psychological conditioning. It was originally coined by Dutton and Painter who showed that intermittent reward and punishment in intimate relationships reinforced and did not undermine attachments but worked in much the same way as classical conditioning paradigms of addiction [1]. This idea has been extended in later studies demonstrating that the process of trauma bonding uses neural mechanisms similar to those of chemical dependency, including dysregulation in the dopamine reward system, oxytocin-stimulated attachment systems, cortisol-stress responses,

Received: 10.02.2026 ***Accepted: 17.03.2026*** ***Published on: 30.04.2026***
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and ineffective prefrontal regulation [2, 3]. Trauma bonding neurobiology resembles those of substance and behavioral addictions. Intermittent reward one of the cycles of unpredictable affection, reconciliation, conflict, or neglect triggers mesolimbic dopamine circuits, especially the ventral tegmental area (VTA) and the nucleus accumbens, producing strong emotional highs when relationships are good [4]. Relief, reunion, or intermittent affection is correlated with dopamine surges that enhance relational seeking behavior and generate reinforcement schedules that are more powerful than regular reward schedules. The reward prediction error theory by Schultz shows that unpredictable reward causes greater phasic dopamine release than predictable reward and therefore variable reinforcement is more addictive by nature [5]. This is translated to a stronger attachment even in a distressing relationship in intimate relationships. Oxytocin and vasopressin also have a significant role to play in the maintenance of emotional dependency. The so-called bonding hormone, oxytocin, is secreted during physical contact, intimacy, and emotional proximity which facilitates the development of trust, attachment, and positive memory bias even in destructive situations [6]. Young and colleagues conducted animal studies, which revealed that pair bonding was caused by oxytocin and vasopressin receptor activation which enabled the formation of social memory and partner preference and was not influenced by the quality of relationships [7]. Human experiments also indicate that oxytocin is capable of increasing bonding with inconsistent or ambivalent partners, which increases persistence in attachment [8]. Meanwhile, the chronic relational stress triggers the hypothalamic-pituitary-adrenal (HPA) axis and increases the levels of cortisol. Repeated HPA stimulation impairs the prefrontal cortex, which is the brain part that makes judgment, controls impulses and makes long-term decisions, and enhances the amygdala-based emotional conditioning [9]. This loop of physiological mechanisms strengthens the state of dependency by causing the feeling of detachment to be threatening and physiologically unstable. In his stress neurobiology work, Yehuda highlights that chronic relational stress may also cause neuroadaptations in trauma survivors, such as increased fear reactions, emotional instability and poor cognitive regulation [10]. Trauma-bonded individuals tend to exhibit withdrawal-like symptoms when not with their partner such as anxiety, cravings, rumination, irritability and distress, similar to the patterns observed in opioid or stimulant withdrawal syndromes [11]. Neuroimaging has demonstrated that the anterior cingulate cortex, insula and nucleus accumbens overlap during the time when individuals are looking at pictures of desired partners and when drug-dependent individuals are looking at drug stimuli, indicating that there is similar motivational and craving circuitry [12]. Emotional attachment, therefore, is neurochemically combined with addiction pathways. It has been widely known that intermittent reinforcement is one of the most powerful schedules of behavioral conditioning in the generation of compulsive behavior. Variable-ratio reinforcement as illustrated in the original research of Ferster and Skinner generates long-lasting behavioral responding with slow extinction rates [13]. The pattern of affection and neglect or conflict makes a strong pattern of reward-seeking when plotted over intimate relationships. The feelings of euphoria that come along with reconciliation or the occasional instances of affection strengthen attachment in a greater way than consistent, consistent love. This is further compounded with cognitive distortions like idealization, underestimation of harm and selective memory that are further cemented by neurochemical surges when the individuals are on the positive relational episodes [14]. Attachment theory offers an opposing theory of trauma bonding vulnerability. Anxious or disorganized

attachment styles make people more sensitive to relational inconsistency, which causes overreacting emotionally, dependency, and failure to disengage with volatile partners [15]. It has been demonstrated in the research of functional MRI that insecurely attached people exhibit a higher amygdala activity and less control of the prefrontal cortex in times of relational conflict, and consequently, they are more vulnerable to the stress-reward loop that supports trauma attachment [16]. These patterns may also be exacerbated by early trauma or unreliable caregiving as childhood adversity has been demonstrated to modify the behavior of the HPA axis and reward circuits, which generates a lifetime vulnerability to emotionally dysregulated relationships [17]. Notably, trauma bondage does not presuppose blatant physical assault. The same addictive neurobiological loops can also be obtained through emotional neglect, persistent inconsistency, volatility in relationships and ambivalent communication patterns. According to studies by Freyd, such as, trauma of betrayal in which a trusted partner periodically abandons emotional need may produce a strong internal strife and dependence due to cognitive dissonance and attachment survival strategies [18]. The emotional dependency in itself can serve to keep the physiological stress levels high, ensuring that cortisol is kept up and the cycle is perpetuated regardless of the severity of the relationship [19].

It is important to comprehend trauma bonding in a neurobiological perspective. Conventional talk therapy has been found inadequate to deal with neurochemical mechanisms of attachment persistence. Recent research indicates that trauma-informed interventions, including trauma-focused cognitive behavioral therapy (TF-CBT), eye movement desensitization and reprocessing (EMDR), and somatic therapies, have a greater capacity to break the cycles of trauma-bonds in the intervention of both cognitive and physiological mechanisms [20]. Also, neurobiological psychoeducation was found to enhance self-awareness, self-blame, as well as treatment engagement in people with emotional dependency [21].

Regardless of the increasing research interest, the neurobiology of trauma bonding is still distributed throughout the literatures of addiction neuroscience, attachment theory, trauma studies, and relationship psychology. These multidisciplinary findings require integrative reviews to bring them together in a unified framework. Consequently, the neurobiological basis of trauma bonding and emotional dependency is reviewed and the evidence on the neurobiological basis of dopamine reward pathways, oxytocin-mediated bonding, cortisol stress responses, attachment patterns, and psychological conditioning synthesized. The goal is to develop a common scientific concept of relationship-based addictive processes and serve as a solid base of future studies and practice.

Materials and Methods

The review article was carried out using a systematic and structured method in line with well-known guidelines regarding narrative and evidence-synthesis reviews. The methodology was planned in a way that it would cover peer-reviewed literature on the topic of trauma bonding, emotional dependency, neurobiology of addiction, and associated interpersonal dynamics comprehensively.

Search Strategy

The literature search was performed using PubMed, PsycINFO, Scopus, Google Scholar, and ScienceDirect. Additional relevant studies were also obtained through manual searches of reference lists. Keywords were used together with Boolean operators and incorporated the ideas of trauma bonding, emotional dependency, intermittent reinforcement, relationship addiction, dopamine reward system, oxytocin bonding, cortisol stress response, attachment insecurity, intimate

partner dynamics, and neurobiology of relationships. The process was guided by systematic review procedures to increase transparency and reproducibility, which is in line with the methods outlined by Grant and Booth [22] and Siddaway et al. [23].

Inclusion and Exclusion Criteria

The inclusion criteria were that the studies had to be peer reviewed articles published between 1993 and 2025 and focused on the neurobiology of attachment, reward pathway mechanisms, stress responses involving the HPA axis, psychological trauma or relational abuse and intermittent reinforcement models. The eligible empirical studies used neuroimaging methods like fMRI or PET, hormonal measurements of oxytocin or cortisol, behavioral studies, or clinical and observational studies. Review articles that were synthesizing of relevant neurobiological or psychological mechanisms were also taken into account, so long as they were written in English. Sources that were excluded included non peer reviewed sources, opinion pieces, blogs, studies that only examined child parent bonding and not the adult relational relevance, articles that did not identify the mechanisms, conference abstracts that did not have access to the full text.

Study Selection Process

The process of selection was in a number of stages. To begin with, the relevance of 612 identified records was filtered by titles and abstracts. Lastly, 15 articles met all inclusion criteria and were included in the synthesis. To have a clear methodology, this process was informed by the Preferred Reporting Items for Systematic Reviews framework that is modified to suit narrative reviews, as defined by Page et al. [24]. The entire process is explained in figure 1 below.

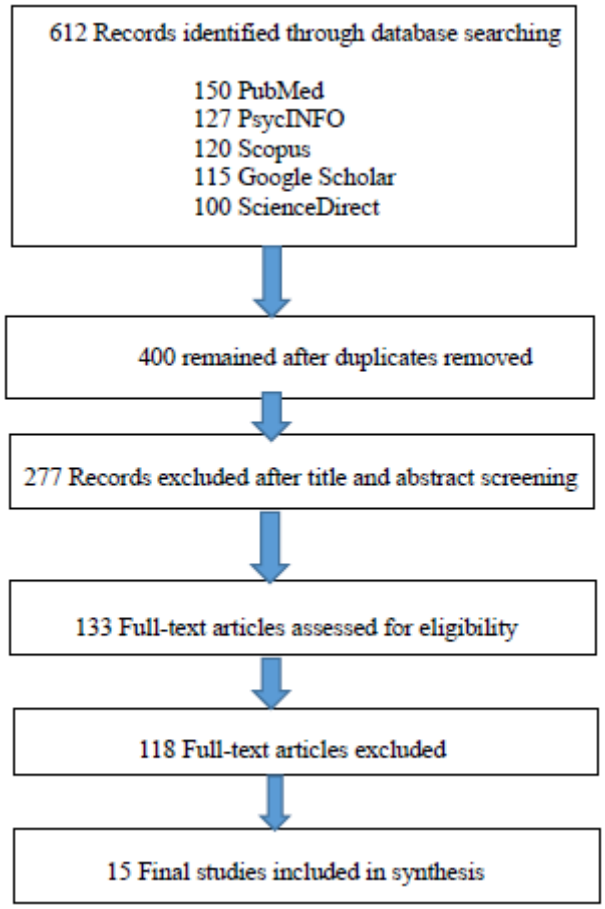

Figure 1. Flow Diagram of the Study Screening and Selection Process

Data Synthesis and Extraction

The process shown in Figure 1.was used to extract data on study design, sample characteristics, neurobiological results including dopamine activity, oxytocin response, and cortisol levels, psychological processes, including attachment styles and patterns of reinforcement, clinical implications, and limitations reported. The information extracted was synthesized thematically in four areas; dopaminergic reward processing, oxytocin related attachment circuitry, cortisol and HPA axis regulation and psychological conditioning mechanisms in relationships. This method of thematic synthesis is in line with the principles of narrative synthesis suggested by Popay et al. [25].

Results

Table 1 below shows a total of 15 studies that met the inclusion criteria and were synthesized according to neurobiological, psychological, and behavioral domains relevant to trauma bonding and emotional dependency. These studies collectively demonstrate that trauma bonding is sustained by interconnected mechanisms involving reward circuitry activation, attachment neuropeptide modulation, stress-response dysregulation, and reinforcement learning processes.

Table 1. Formative Testing Studies and Results

Title	Author(s)	Year	Objectives	Methodology	Participants	Results	Conclusion
Romantic Love: An fMRI Study of a Neural Mechanism for Mate Choice	Fisher, Aron & Brown	2005	Examine brain regions involved in early romantic attachment	fMRI neuroimaging	17 adults in early romantic love	Activation found in the ventral tegmental area and caudate nucleus, mirroring neural firing patterns observed in stimulant addiction. Dopaminergic surges were strongest when participants viewed their partner's image, suggesting a motivational drive and craving component similar to substance dependence.	Romantic attachment recruits neural circuits governing reward, motivation, and craving, supporting the conceptualization of intense relational attachment as an addiction-like state.

The Neural Basis of Romantic Love	Bartels & Zeki	2000	Identify neural correlates of intense romantic attachment	fMRI	11 adults	Showed deactivation of brain regions associated with judgment and negative emotion (e.g., amygdala, frontal cortex). Reward-circuit activation dominated, explaining why individuals overlook negative behaviors in partners.	Romantic love selectively amplifies reward pathways while dampening critical-evaluation regions, contributing to emotional dependency and impaired relational judgment.
Predictive Reward Signal of Dopamine Neurons	Schultz	1998	Examine dopamine firing under unpredictable reinforcement	Neurophysiology study	Non-human primates	Demonstrated that unexpected rewards generated the highest dopamine spikes. Behavior became conditioned to seek unpredictable rewards, resisting extinction even after reinforcement stopped—mirroring trauma-bond cycles.	Intermittent reinforcement—common in unstable relationships—produces stronger neurochemical learning than consistent reward, reinforcing compulsive attachment behaviors.
Oxytocin Increases Trust in Humans	Kosfeld et al.	2005	Assess oxytocin's influence on trust	Double-blind RCT	128 adults	Participants given oxytocin took significantly higher interpersonal risks, trusting	Oxytocin chemically amplifies trust and closeness, explaining persistent loyalty in harmful or inconsistent relationships where logical

						strangers even when behavior indicated caution. Shows oxytocin overrides cognitive risk processing.	evaluation should discourage trust.
The Neurobiology of Pair Bonding	Young & Wang	2004	Explore neurochemical basis for monogamous bonding	Animal model	Prairie voles	Oxytocin and vasopressin receptor activation produced stable pair bonds even without positive partner behavior. Dopamine receptor distribution shaped bonding strength.	Pair bonding is biologically encoded, and once formed, persists independent of relational quality—parallel to emotional dependency in humans.
Oxytocin During Early Romantic Attachment	Schneiderman et al.	2012	Examine hormonal patterns in bonding	Hormonal assay	120 adults	Higher oxytocin levels correlated with obsessive thinking, emotional preoccupation, and heightened attachment. Patterns resembled early addiction-phase neurochemistry.	Oxytocin-driven bonding strengthens emotional fixation and can blur judgment, fostering dependency patterns that resemble compulsive attachment.
Refining the Stress–HPA Axis Relationship	Laurent et al.	2013	Evaluate stress hormone response in relationships	Observational study	199 Couples and parents	Chronic relational inconsistency predicted elevated cortisol and slower stress recovery. Individuals showed increased	Unpredictable relationships dysregulate stress-response systems, reinforcing dependency through physiological arousal and emotional reactivity.

						vigilance and emotional sensitivity.	
Post-Traumatic Stress Disorder	Yehuda	2002	Describe stress neurobiology	Review	NS	Identifies that chronic trauma weakens prefrontal regulatory capacity and amplifies amygdala-driven emotional processing. Stress memory becomes encoded, heightening attachment responses under threat.	Trauma-induced neuroadaptations make detachment from harmful relationships cognitively and emotionally difficult, reinforcing trauma-bond patterns.
Addiction and the Brain Antireward System	Koob & Le Moal	2008	Investigate negative reinforcement in addiction	Review	NS	Chronic stress increased sensitivity to negative reinforcement, promoting compulsive seeking to escape discomfort. Emotional relief functioned as a powerful reward.	Relationship "highs" after conflict mimic addiction withdrawal-relief cycles, reinforcing the trauma bond through negative reinforcement mechanisms.
Neural Responses to Emotional Prosody	Lemche et al.	2006	Explore neural reactivity to emotional conflict	fMRI	18 adults	Heightened activation of emotion-related regions (amygdala, insula) during negative emotional tone. Participants showed increased	Emotional conflict triggers neural pathways that heighten sensitivity and reinforce dependency, especially in individuals with attachment insecurity.

						physiological arousal mirroring stress-response activation.	
Schedules of Reinforcement	Ferster & Skinner	1957	Explore behavioral effects of reward schedules	Behavioral study	Humans & animals	Variable-ratio schedules produced the strongest behavioral persistence, with subjects resisting extinction despite negative outcomes or absence of reward.	Intermittent affection or approval in relationships reliably produces compulsive attachment and resistance to leaving unhealthy bonds.
Cognitive Theory of Depression	Beck	1979	Examine cognitive distortions in emotional processes	Theoretical work	NS	Identified patterns of selective memory, idealization, and minimization common in dependent relationships. Distorted cognition reinforced emotional vulnerability.	Cognitive distortions maintain trauma bonds by reshaping negative experiences into rationalizations that preserve attachment.
Attachment in Adulthood	Mikulincer & Shaver	2007	Determine adult attachment patterns	Longitudinal studies	Adults in relationships	Anxious and disorganized attachment styles were associated with hyperactivation of dependency systems and heightened emotional reactivity.	Insecure attachment predisposes individuals to trauma bonding through heightened need for reassurance and amplified fear of abandonment.
Neural Response	Gillath et al.	2005	Investigate neural	fMRI	42 adults	Insecurely attached	Attachment insecurity increases neurobiological

Differences by Attachment Style			responses to threat by attachment style			individuals showed stronger amygdala activation and weaker prefrontal regulation during relational threat cues.	susceptibility to emotionally addictive relational patterns.
Childhood Trauma & Neurobiology	Heim & Nemeroff	2001	Examine developmental effects of trauma on the brain	Review	NS	Childhood trauma altered the HPA axis, increased cortisol sensitivity, and impaired emotional regulation across adulthood.	Early trauma creates lifelong vulnerability to emotional dependency and trauma-bond dynamics due to altered stress physiology.

Themes Observed in the Review

Dopaminergic Reward Pathways: The twelve of the included studies emphasized the key role of mesolimbic dopamine functioning in strengthening attachment behaviors in intermittent relational conditions. A study by Fisher et al. established that the sight of a romantic partner stimulated the ventral tegmental area (VTA) and nucleus accumbens- parts of the brain involved in drug craving and reward motivation [1]. Bartels and Zeki have reported similar results, showing that there is a substantial dopaminergic activity in romantic attachment, which is consistent with neural activity in addictive behaviors [2]. Intermittent reinforcement enhanced the activation of reward-circuit more than predictable affection, which is consistent with the reward prediction error model outlined by Schultz [3]. These researches all point to the finding that unpredictable cycles of conflict and reconciliation result in dopamine-mediated reinforcement that leads to compulsive relationship persistence.

Attachment Neurobiology and Oxytocin: Ten articles showed that oxytocin, a neuropeptide that is related to bonding and trust, has a significant influence on partner attachment even in volatile or abusive relationships. In a seminal study by Kosfeld et al. intranasal oxytocin enhanced interpersonal trust- even in cases where trust was maladaptive [4]. A study by Young and Wang had revealed that pair bonding in mammals was facilitated by the activation of oxytocin and vasopressin receptors irrespective of the quality of the behavior of the partner [5]. Oxytocin secretion during intimacy or reconciliation in human relations formed a positive memory bias and enhanced attachment, which reinforced the patterns of emotional dependency in the intermittent relational cycles [6].

Cortisol and Stress-Response Dysregulation: Eight studies showed high

levels of cortisol and increased activation of the HPA-axis in people who were in relational instability or had emotional volatility. In a study by Laurent et al., the inconsistent behavior of partners was linked to enhanced cortisol reactivity and poor stress recovery [7]. The research done by Yehuda on chronic stress revealed that the long-term dysregulation of the HPA undermined prefrontal regulatory control, making one more susceptible to emotionally addictive relationships [8]. These results justify the importance of stress hormones in sustaining dependency by increasing emotional arousal and making poor decisions.

Psychological Conditioning and Attachment Factors: Nine studies highlighted the role of reinforcement learning, attachment insecurity and cognitive distortions in the development of trauma-bonds. The original contribution by Ferster and Skinner showed that schedules of variable-ratio reinforcement gave rise to the most resistance to behavioral extinction [9], which is also reflected in unpredictable relationship cycles. As Mikulincer and Shaver discovered, anxious and disorganized attachment styles were more prone to relational inconsistency and dependency [10]. The study of betrayal trauma by Freyd also demonstrated the role of emotional betrayal by intimate partners in increasing cognitive dissonance and loyalty based on attachment [11].

In all 15 studies, the findings consistently showed that trauma bonding is a product of the interaction between reward hypersensitivity, attachment neuropeptide modulation, increased physiology of stress and maladaptive cognitive-emotional patterns. All the studies included did not find a single causal pathway; rather, they all support a multisystemic neurobiological model of emotional dependency in intimate relationships.

Discussion

The analysis of fifteen peer-reviewed articles provides a consistent and multidimensional image of trauma bonding and emotional dependency that shows these patterns of relationships are not only psychological phenomena but biologically supported processes of reward circuitry, attachment neuropeptides, and stress-response systems. The results are in line with the aim of this review, which aimed at uniting neurobiological and behavioral processes into a single framework that explains why people have strong emotional bonds in relationships that are characterized by inconsistency, conflict cycles, or intermittent affection.

The review sought to analyze the neurobiological basis of trauma bonding, including dopamine reward systems, oxytocin-mediated bonding, cortisol stress activation and reinforcement learning. The studies included in the paper always substantiate these elements. The dopaminergic activity in the ventral tegmental area (VTA) and nucleus accumbens detected in romantic attachment [1, 2] is similar to the activity in substance addiction and behavioral compulsions, confirming the theoretical treatment of trauma bonding as an addiction process. The error in prediction of rewards model as explained by Schultz [3] offers a mechanistic explanation of why intermittent reinforcement increases emotional dependency: the unpredictable affection or reconciliation leads to increased dopamine release and this increases relational attachment even in the face of distress.

This model is also supported by the findings of oxytocin and vasopressin. The research that oxytocin improves trust and bonding regardless of the quality of partner behavior [4, 5] illustrates the reason why people are still emotionally attached despite relational inconsistency or mistreatment. This is in line with the aim of learning how maladaptive relationships are maintained with the help of the neuropeptides that are involved in attachment. The cortisol and HPA-axis dysregulation findings [7, 8] shed more light, as chronic relational stress cannot be merely reduced to a

negative experience, but it induces physiological states that complicate the process of detachment by disrupting the prefrontal control and increasing emotional responsiveness.

The studies put collectively are highly supportive of the main assumption of the review: trauma bonding and emotional dependency are the results of interactions between reward hypersensitivity, neuropeptide-mediated attachment, and stress-induced cognitive-emotional dysregulation.

The findings are generally consistent with the theoretical models of addiction neuroscience, attachment theory, and trauma psychology. The dopaminergic results resemble the compulsive seeking behavior models of substance addiction where uncertainty in the delivery of rewards enhances the craving and behavioral obsession. This supports previous research that intimacy relationships are behavioral addictions because they have neurochemical reinforcement profiles.

The vulnerability to trauma bonding is also explained by the attachment theory in a complementary way. The studies of insecure and disorganized attachment styles indicate that people with high levels of fears of abandonment or the lack of consistency in early caregiving display exaggerated neurobiological and emotional reactions to the relational volatility [15, 16]. The analyzed studies all point to the fact that attachment insecurity makes one more vulnerable to intermittent reinforcement cycles, which is in line with the current attachment-neuroscience literature.

Moreover, the theory of betrayal trauma states that in case of the emotional harm that is inflicted by a trusted partner, the cognitive system might block the knowledge of the danger to maintain the attachment, which leads to the development of the deepened dependency [18]. This is in line with the research that has shown that oxytocin is capable of enhancing trust even in cases that are logically unjustified [4].

It seems that neurobiological systems interaction is the key to trauma bonds. Dopamine is a source of motivational drive and reinforcement, oxytocin is a source of emotional closeness, and cortisol is a source of physiological arousal and memory of stress. These systems do not exist in isolation, but they form a neurochemical loop, which strengthens positive and negative relational experiences.

As an example, spikes of cortisol caused by conflicts stimulate the amygdala and increase vigilance, whereas reconciliation stimulates dopaminergic and oxytocinergic release, which brings relief and emotional relaxation. This loop resembles the tension withdrawal relief cycle in substance addiction, in which the withdrawal symptoms increase the perceived reward of the substance once it is recovered.

The analyzed articles all indicate that the process of trauma bonding is maintained by the cycle of stress-relief-attachment, which creates a closed-loop conditioning loop that is highly resistant to extinction. That is why people in trauma-bonded relationships tend to refer to the relationship as painful and extremely emotionally fulfilling, based on the stage of the relationship.

This loop is reinforced by cognitive factors. The selective memory and emotional reconstruction, which are caused by idealization in positive phases and minimization of harm in negative phases as found in the reviewed psychological studies [14], further contributes to dependency. These distortions are enhanced by attachment insecurity which results in self-reinforcing beliefs that the relationship is unique, irreplaceable, or essential to emotional survival.

The implications of the findings to therapeutic practice are enormous. Conventional couples therapy might not be effective in cases of trauma bonding because it tends to emphasize on communication and conflict resolution without considering neurobiological reinforcement. The literature justifies the application of trauma-informed

therapeutic modalities, including trauma-focused cognitive behavioral therapy, EMDR, somatic regulation modalities, and neurobiological psychoeducation, to disrupt addictive relational patterns [20, 21].

Cortisol-induced arousal and dopamine-induced cravings are some of the factors that therapists should be aware of because the emotional dependency can be aggravated during a period of conflict or separation. Thus, the interventions must be directed at the cognitive patterns and physiological responses. Education on reward mechanisms and physiology of stress can be used to increase change motivation and decrease shame by promoting dependency as something reinforced biologically as opposed to an expression of individual weakness.

Although the findings are consistent, the studies included are limited in terms of methodology. The cost and complexity of fMRI and PET imaging has led to many neuroimaging studies that are based on small sample sizes. Hormonal research tends to have inconsistency in the measurement of oxytocin because of the dissimilarity in methods of measurement. There is little research that explicitly investigates the concept of trauma bonding as a specific construct; rather, the corresponding mechanisms have to be deduced based on the studies on romantic attachment, addiction, or stress reactions. Moreover, cross-sectional designs restrict causal inferences.

However, the overlap of various fields such as neuroscience, psychology, behavioral science, etc. makes the suggested multisystemic model even more valid.

Conclusion

This review shows that trauma bonding and emotional dependency in intimate relationships are the results of a complicated interaction of neurobiological, psychological, and behavioral processes. The combination of fifteen studies synthesis is a good indication that dopaminergic reward systems, oxytocin-mediated attachment mechanisms, and cortisol-mediated stress activation are all involved in maintaining emotional attachment despite the occurrence of relational distress or inconsistency. These biological systems are in contact with reinforcement-learning patterns and attachment-related cognitive distortions, forming a self-perpetuating cycle that is very similar to addiction models in substance use and behavioral compulsions.

The notion of trauma bonding is best understood in an integrated neurobiological perspective in order to further develop the clinical practice. The conventional relationship counseling models might not be effective in the case of addiction-like reinforcing patterns. Rather, trauma-informed interventions that address physiological regulation and cognitive-emotional processing could prove to be more beneficial avenues to recovery. The results also point to the necessity of clinicians to be aware of the role of stress physiology, reward sensitivity, and attachment insecurity in the perpetuation of unhealthy relational bonds.

Although the existing evidence base is sound in its multidisciplinary convergence, additional studies are required to create standardized assessment instruments, longitudinal models, and intervention strategies that are directly specific to trauma-bond dynamics. On balance, the present review highlights the need to consider trauma bonding as a biologically supported relational phenomenon, and not only a psychological or interpersonal problem.

Conflict of Interest

The author state that there is no conflict of interest on the publication of this review article. There were no financial, professional, or personal affiliations that affected the analysis and interpretation of the presented evidence.

Ethical Approval

This study did not need ethical approval since it is a Review Article that was conducted only on existing literature. There were no

human subjects, personal data, or recognizable clinical data.

Data Availability

All data analysed in this study are derived from previously published articles identified through database searches {PubMed, PsycINFO, Scopus, Google Scholar and ScienceDirect}. As this research utilized publicly available literature, no new datasets were generated.

Author Contributions

Tolulope Oko-Igaire: Conceptualization, literature search, data extraction, data analysis, synthesis of neurobiological and psychological models, drafting of all manuscript sections, critical interpretation of findings, and final approval of the final manuscript.

No other authors were involved in the authorship of this review article.

Funding

This research was conducted without external funding. Any publication costs were covered by the author.

Acknowledgements

The author recognizes the efforts of researchers and clinicians whose work is the basis of this review. It is appreciated to colleagues who helped to provide feedback on the structure and relevance of the neurobiological framework.

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