

Impact of childhood trauma on the epigenetics of anxiety disorder

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ABSTRACT

The interaction of epigenetics, childhood trauma, and anxiety disorders is a fascinating area of scientific study with significant ramifications for clinical practice and mental health. This abstract captures the intricate interplay between these factors, emphasizing how early-life hardships leave persistent biochemical fingerprints on a person's genetic composition, perhaps influencing the emergence of anxiety disorders. Through epigenetic pathways, childhood trauma, which includes events like abuse, neglect, and persistent stress, might influence a person's sensitivity to anxiety. These processes, which control the expression of genes involved in stress response, neurotransmitter signaling, and emotional regulation, include DNA methylation, histone changes, and microRNA regulation. The disturbance of the hypothalamic-pituitary-adrenal (HPA) axis and neuroplasticity provide as more evidence of the effects of trauma-induced epigenetic modifications, which manifest as altered brain circuits and stress response mechanisms. This complex interaction highlights how nature and nurture interact dynamically, enhancing our knowledge of the many-faceted causes of anxiety disorders. A need for focused treatments and therapies that address the molecular causes of anxiety is made as a result of the recognition of the long-lasting impacts of childhood trauma, giving those who are afflicted hope for better mental health outcomes and resilience.

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Introduction

Trauma in early childhood is one of the mental health problems that raises health-related concerns in public. People that face any traumatic event in their early life are likely to develop a traumatic disorder that retards them mentally. Along with mental health problems people facing trauma disorder get involved in other bad activities like alcohol drinking and substance use. The use of alcohol makes the mental health of trauma-affected patients worse. The suicide rate of people has increased over the years because of extreme mental health problems they faced in their lives. Trauma makes a person null and develops sad and depressive symptoms[1]. The depressive episodes of a person having past trauma reduce their will to live. Most people around the world who commit suicide face severe type of mental disorders. Childhood trauma can be caused because of various reasons. These reasons include childhood abuse, gender discrimination, and other psychological problems. These traumas reason develop mental health disorders in people in early childhood. Various body functions get disturbed when a person faces a traumatic event in his life[2, 3]. It's crucial to remember that while epigenetic pathways might cause childhood trauma to raise the likelihood of developing anxiety disorders, genetics also affect an individual's sensitivity. Additionally, while resilience traits and nurturing surroundings can lessen the effects of traumatic events, not everyone who encounters childhood trauma will go on to acquire an anxiety condition. It is a challenging and

developing field of study to comprehend how developmental trauma, epigenetics, and anxiety disorders interact. Therapeutic measures, such as psychotherapy and pharmaceuticals, may help buffer the consequences of epigenetic alterations brought on by trauma and lower the likelihood of developing anxiety disorders. Epigenetic changes brought on by trauma are not always permanent. Mitochondrial gut health as well as HPA axis functioning gets affected because of mental disorders due to trauma. The changes in a person's physical and mental health due to childhood trauma make him socially vulnerable. Such people tend to avoid crowded places and like to stay alone. The social interaction makes such people nervous and develops restlessness in them. By understanding the anxiety disorder problem faced by any trauma-affected person, the recovery process can be improved.

The main reason behind the onset of psychiatric problems in anxiety disorder-affected patients is epigenetic mechanisms. Epigenetic inheritance is a phenomenon that is involved in producing heritable phenotypes. These phenotypes change due to mental illness and result in DNA modification. The change in phenotype only modifies the DNA but does not change the pattern of the DNA[4]. Epigenetic changes are different from genetic changes. Genetic changes result due to the alternation of gene in DNA whereas epigenetic modification results due to environmental factors. Another factor that causes epigenetic



change is stress. A person facing stress problems in his life undergoes epigenetic modifications that change his heritable phenotypes[5] Early life trauma causes alteration in the brain functioning of a person having epigenetic modifications. The epigenetic alternations influence the neurological system and increase the chances of anxiety disorder development. A person having changes in his endocrine system working mechanisms also has changes in epigenetic mechanisms that develop psychiatric problems in him. Also, the changes in the DNA methylation process are observed in patients having childhood trauma[6]. Researchers and clinicians have long been fascinated by childhood trauma and its profound effects, which has led to studies into the complex interactions between early experiences and the emergence of various mental health issues. Anxiety disorders stand out among these diseases owing to their frequency, influence on people's everyday lives, and complicated etiological roots.

A compelling framework for understanding how environmental factors, such as childhood trauma, can leave long-lasting molecular marks on a person's genetic makeup and possibly influence the development of anxiety disorders later in life has recently emerged in the field of epigenetics. Childhood trauma refers to a variety of traumatic events that happen to a person when they are still developing. This may involve neglect, experiencing physical, mental, or sexual abuse, seeing domestic violence firsthand, or growing up in unstable or stressful situations. The importance of these experiences in determining a person's physical, emotional, and psychological well-being has been well-documented. In reality, studies have shown a strong link between childhood trauma and a higher risk of developing mental diseases, such as anxiety disorders. However, the methods through which these early hardships leave long-lasting marks have come to the fore, giving rise to the study of epigenetic mechanisms. The change in the DNA methylation process increases the chances of neuroendocrine damage. The neuroendocrine damage then induces depression and makes a person mentally unstable to perform daily life activities. The Brain-derived neurotrophic factor (BDNF) changes its state as a result of epigenetic mechanisms and alters the developmental process of brain. the brain developmental alternation reduces the level of plasticity of brain. Moreover, the childhood trauma led to adversity in genomic functioning. the loci of multiple genomes get changed due to the adversity changes associated with epigenetic process.

All the alterations in the epigenetic process can be treated using clinical procedures. The drug-targeting process improves the epigenetic mechanism and reduces the chances of alternation of the processes underlying the epigenetic mechanism. People having PTSD change the epigenetic process. To treat these changes in PTSD patients they are given treatment-based therapy. Valproic acid is used to overcome the stress level and inflammation in PTSD patients[7]. The disorder of neurotransmitters involved in developing PTSD changes into an orderly form using valproic acid. By effectively targeting the epigenetic changes and by treating them through proper medical intervention the chances of trauma-related anxiety disorder development in people are reduced.

Most health professionals find out that epigenetics is a reversible process by reversing the epigenetics process through altering environmental factors the chance of alternation in epigenetic mechanisms is reduced greatly. Maintaining the epigenetic

inheritance process holds critical value as it minimizes the risk of the development of anxiety disorder[8]. Also, there are a lot of problems created as a result of trauma condition. The first problem is an increase in the chances of the onset of cardiovascular diseases. trauma condition disturbs the heart functioning of a person developing panic and anxiety attacks. Sudden anxiety attacks make a patient's heartbeat irregular and increase the chances of cardiovascular disorder onset in the patient. The second problem is obesity. This problem develops because of the depression faced by trauma-affected patients [9]. The rapidly developing area of epigenetics in molecular biology explores the dynamic interplay between a person's genetic make-up and their environment. While genetics supplies the underlying foundation, epigenetic processes alter gene expression patterns in response to environmental signals. This mediates how genes are 'read' by cells and affects how they operate. Childhood trauma may permanently alter a person's genetic landscape, and epigenetic alterations including DNA methylation, histone modifications, and microRNA regulation have been identified as important mediators of this effect.

DNA methylation is one of the main epigenetic processes connected to how childhood trauma affects anxiety disorders. The expression of genes is changed as a result of this process, which adds methyl groups to certain DNA sequences. Early-life trauma can alter the DNA methylation patterns of genes implicated in stress response pathways, neurotransmitter signalling, and emotional regulation in the setting of anxiety disorders. These alterations could make people more sensitive to stressors, interfere with emotion control, and cause anxiety-related phenotypes. Studies also show that patients having depression have an eating disorder that makes them obese. the third problem is maladaptive daydreaming This problem causes anxiety disorder and affects the patient's daydreamer and he tends to live in his imaginary world. Such daydreamer tends to lose their interest in the real world All these problems develop in trauma affected patient and makes his life more miserable.

Due to trauma disorder, various personality traits of a person are affected. Vulnerability is a character that develops in people having trauma conditions. Another factor that develops in trauma-affected patients is social awkwardness. The trauma-affected patients face problems while interacting with people. The trait of social awkwardness makes them self-isolated and they tend to avoid interacting with people These changes in personality trait makes trauma patient more mentally depressed and sad. To treat such patients, they are given behavioral therapies along with medication-based treatment. Behavioral therapies develop self-confidence and self-resilience factors in patients and make them able to face the world. The patients with anxiety disorder getting behavioral therapy show improved behavioral activities[10]. Such patients tend to participate in social gatherings and begin to interact with people. the positive trait rebuilding in AD patients is possible only through proper cognitive and behavioral therapy-based interventions.

Furthermore, for treating any anxiety-based disorder various neurobiological studies are made. Neurobiological studies provide information about the severity of the disorder and its type. This information then helps health professionals and psychiatrists in providing effective medications and therapies based on the disorder type[11]. Another essential component of epigenetic control is histone modifications, which are crucial in shaping the chromatin architecture that houses DNA. Changes in

histone modifications have been linked to changes in childhood trauma, which affect the accessibility of genes involved in anxiety and the stress response. Histone alterations can help or inhibit the transcription of genes by altering the compactness of chromatin, hence changing a person's propensity for anxiety disorders. Small RNA molecules known as microRNAs (miRNAs), which regulate gene expression post-transcriptionally, have also become important participants in the epigenetic processes that link early trauma to anxiety disorders. Early-life stresses can cause the dysregulation of miRNAs, which causes abnormal expression patterns of genes related to anxiety, emotion control, and brain plasticity. Therefore, these molecular changes may influence the unfavorable behavioral and physiological reactions frequently seen in anxiety disorders. The psychiatric problems underlying the mental disorder-related condition of patients become more understandable using neurobiological studies.

Research objectives:

The main objective of this research paper is to explain the Impact of childhood trauma on the epigenetics of anxiety disorder. Moreover, the epigenetics modification caused due to trauma condition is discussed in this article for understanding the mechanism underlying the onset of anxiety disorder.

Literature review:

Researchers claim that the main reason behind the development of anxiety disorder is early childhood maltreatment. Children who undergo physical or emotional abuse at an early age in their lives develop anxiety and show panic symptoms. The depression symptoms cause DNA methylation alternation in patients having anxiety disorder and make their disorder condition more severe. Also, the process of epigenetic aging acceleration is observed in patients having anxiety disorder. [12] Studies show that trauma conditions impact a person's mental health state making him more depressed and less active. Chronic stress is one of the stress types caused due to early life trauma adversity. epigenetic mechanism influences a person's psychological abilities making him more vulnerable to developing psychiatric disorders. The intergenerational epigenetic mechanism alternations are responsible for the onset of stress and anxiety symptoms in trauma disorder-affected individuals [13] Studies claim that a mother experiencing traumatic and tragic events in her life during a pregnancy develops mental health problems in the child. The inheritable altered phenotypes when inherited by a child from a mother cause the onset of mental-related health problems in a child after birth.

To avoid the transfer of altered phenotypes mothers are given proper therapy-based interventions along with medications during the pregnancy time to improve their mental health [14]. studies show that the experience of inner as well as outer environments changes the functioning of the brain. The outer factors influence the epigenetic mechanism of a person and increase the chance of mental health disorders. Brain plasticity reduces due to the changes in neuronal functioning of the brain. The neuronal functioning gets disturbed due to a disturbance in epigenetic factors [15]. studies explain that all the epigenetic changes result are due to the adverse childhood trauma events experienced by mentally affected patients. The neurobiology studies reveal that mitochondrial metabolism process alternation is one of the epigenetic modification processes that induces mental health disorders in people with traumatic pasts [16]. Also, epigenetic modifications determine the mental disorder nature.

The neurological studies on epigenetic modification predict that this process is treated and diagnosed through proper medicated-based treatment therapy [17]. The deep link between neuroplasticity and epigenetics emphasises the effect of childhood trauma on anxiety disorders even more. The amazing capacity of the brain to rearrange its structure and operation in response to experiences is known as neuroplasticity. Early trauma can cause epigenetic changes that affect synaptic connections and change how the brain reacts to stress and emotional inputs. The increased sensitivity to anxiety-inducing cues seen in those with a history of childhood trauma may be caused by this neurological remodeling. Furthermore, epigenetic alterations brought on by childhood trauma make the hypothalamic-pituitary-adrenal (HPA) axis, a key player in the body's stress response, vulnerable to dysregulation. Anxiety disorders have been related to changes in the HPA axis's functioning, which controls the release of stress hormones. Epigenetic changes have the potential to upset the delicate HPA axis balance, making people more susceptible to stress and contributing to the pathophysiology of anxiety. Studies predict that stress is caused due to various factors and its severity depends on the person's age and sex.

Childhood stress conditions are more vulnerable as it is the reason behind psychiatric problems. The PNS model explains that a person's behavior during adolescence is controlled through regulating various biological as well as epigenetic mechanisms [18]. studies explain that adversity in early life results in mental health problems with serious complications. Studies reveal that children having traumatic pasts are more depressed and mentally disturbed than children having non-traumatic pasts. Traumatic events are among the causes behind non-accidental injury conditions [19]. studies suggest that people affected with chronic pain have disturbed mental Health.

Chronic pain symptoms onset in children at a relatively early age because it is a heritable trait. Children inherit chronic pain symptoms from their mothers during pregnancy. The epigenetic mechanism is the underlying process behind chronic pain development in childhood [20]. Studies claim that war activities become prevalent in the world because people get triggered due to stress and anxiety factors. All the conflicts among people start because of anger that comes from stress and anxiety. Epigenetic factors are inheritable factors that an individual possesses and affect the mental health of people [21]. Moreover, war trauma is one of the reasons that develops physical disorders in people with mental health problems. War trauma changes a person's epigenetic factors and induces several disorders onset. The neuroendocrine system as well as immunological system functioning get altered because of war trauma. The change in various epigenetic process functions increases the chances of brain-related disorders [22]. studies claim that to understand the etiopathology behind functional and movement disorders studies on the epigenetic mechanisms are made. Childhood abuse is the main factor that induces the epigenetic changes that ultimately cause FMD and other neurological disorders. The main epigenetic mechanism that alerts during the onset of FMD Is the DNA methylation process [23, 24].

The substantial effects of epigenetic alterations brought about by childhood trauma on gene expression are not limited to molecular biology; psychology and therapeutic practise are also profoundly affected. A biological predisposition to anxiety disorders may result from abnormal gene expression in the neurotransmitter systems, emotion regulation, and stress response. This highlights

the value of a thorough approach to understanding the aetiology of these disorders—one that takes into account both genetic predisposition and the environmental exposures that influence gene expression. As a result, the investigation of how childhood trauma affects the epigenetics of anxiety disorders is an exciting interdisciplinary confluence that sheds light on the complex mechanisms behind the long-lasting impacts of early events. Childhood trauma can enhance a person's susceptibility to anxiety disorders, and epigenetic alterations, such as DNA methylation, histone modifications, and miRNA regulation, have been identified as important mediators in this process. The complicated nature of the link between early experiences and mental health consequences is highlighted by the interaction between these molecular processes and neuronal plasticity, as well as by the dysregulation of systems like the HPA axis. As our knowledge of epigenetics expands, it offers the potential to influence cutting-edge therapy approaches that address the underlying molecular causes of anxiety disorders, opening up new doors for intervention and support for people who have experienced traumatic events as children. Studies suggest that treating the mental health problems of youth holds great importance. Anxiety disorder is the most prevalent disorder in youth that disturbs their brain regulatory process.

To provide treatment to AD-affected youth they are given behavioral therapies. The SSRIs treats the anxiety symptoms in youth and provides them with stable brain regulation[25].studies explain that most of children loses their parents at early age .losing one or both parent develop depression and stress in children .this loss of parent at early childhood is one of the traumatic event for child .this traumatic event develops complex mental heath problems during early childhood including PTSD. certain molecular mechanism underlying epigenetic mechanism gets alter when a person face traumatic event during every childhood .

this alternation of molecular mechanism due to traumatic event makes the childhood events more adverse for child facing these events[26].studies explain that morality rate increase in people having adversity during the early life. early childhood adversity makes them develop mental disorders that results in epigenetic age deceleration. alternation of the epigenetic aging process increases the risk of mortality for people with anxiety disorder[27] Studies show that psychiatric problems among people are increasing at an alarming level.

The burden on the health sector due to psychiatric problem is more .epigenetic dysregulation is the main biomarker behind these psychiatric problems development in people[28].furthermore ,some people cope with the traumatic experiences of past and does not get serious mental health problems. for coping the childhood stress ,people undergo therapy treatment. therapist guide the AD patient about the possible risk factors that can trigger their anxiety .by avoiding these risk factor AD Patients overcome their childhood anxiety. PTSD patients undergo the same therapy procedure in order to overcome their stress due to traumatic childhood events[29].studies highlight that the anxiety disorder is becoming one of the serious mental disorder because of the epigenetic mechanism involved in this disorder. numerous biological as well as psychological characteristics plays critical role in inducing anxiety disorder[30].Studies predict that exposure to stress at early life is dangerous as it disturbs the normal functioning involved in epigenetic programming .the disturbance of

epigenetic programming onsets mental disorders that have high prevalence rate[31].

Studies explain that various epigenetic mechanism get alerted due to the traumatic events faced by a person during adolescence. parents' separation and getting life threats are among the traumatic events that some children face during their childhood. these events develop depression and make childhood event traumatizing for them[32]

An individual's mental and physical health, as well as their chance of acquiring anxiety disorders, can be significantly impacted by childhood trauma. The study of epigenetics focuses on how environmental influences, such as experiences and behaviours, may affect how genes are expressed without changing the underlying DNA sequence. Following a traumatic event in childhood, epigenetic alterations may contribute to the development of anxiety disorders. The epigenetics of anxiety disorders may be affected by early stress in the following ways:

1. DNA Methylation: DNA methylation is a frequent epigenetic change in which a methyl group is added to certain DNA regions, frequently suppressing gene expression. Alterations in DNA methylation patterns in genes involved in stress response, emotion regulation, and neurotransmitter signalling can result from childhood trauma, such as abuse or neglect. These modifications can affect how the body reacts to stress and how it processes emotions, which can raise the chance of developing anxiety disorders.

2. Histone Modifications: Proteins known as histones assist in packing DNA into the tight chromatin structure. Histone alterations may alter how tightly DNA is twisted around them, which may have an impact on gene expression. The expression of genes linked in the brain's stress response and emotional regulation can be affected by histone alterations brought on by childhood trauma, which can affect the development of anxiety disorders.

3. MicroRNAs: MicroRNAs (miRNAs) are tiny RNA molecules that control gene expression by interacting with messenger RNA (mRNA) and blocking protein translation. Childhood trauma can change how certain miRNAs are expressed, which in turn can affect how genes involved in the anxiety and stress response pathways are expressed.

4. Neuroplasticity: The capacity of the brain to reorganise and adapt in response to events might be affected by epigenetic alterations brought on by childhood trauma. Synaptic connections, which are essential for emotional regulation and stress response, can be formed and remain stable under the influence of altered epigenetic markers. Behaviours associated with anxiety may be influenced by changes in synaptic connection.

5. HPA Axis Dysregulation: A crucial mechanism involved in the body's stress response is the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis can become dysregulated as a result of epigenetic alterations brought on by childhood trauma, which can result in a heightened stress response and greater susceptibility to anxiety disorders.

6. Gene Expression: Changes in gene expression patterns brought on by epigenetic alterations may have a long-lasting effect on the way that neurotransmitters, receptors, and other molecules involved in anxiety control work. This may result in an enhanced sensitivity to stimuli and a higher risk of anxiety disorders.

Table 1 Descriptive statistic:

| | N | Minimum | Maximum | Mean | Std. Deviation |
|--------------------|----|---------|---------|--------|----------------|
| childhood trauma 1 | 50 | 1.00 | 4.00 | 1.5800 | .78480 |
| childhood trauma 2 | 50 | 1.00 | 5.00 | 1.7600 | .84660 |
| childhood trauma 3 | 50 | 1.00 | 3.00 | 1.5200 | .64650 |
| epigenetics | 50 | 1.00 | 4.00 | 1.5200 | .86284 |
| anxiety disorder 1 | 50 | 1.00 | 3.00 | 1.4800 | .64650 |
| anxiety disorder 2 | 50 | 1.00 | 4.00 | 1.4600 | .76158 |
| Valid N (listwise) | 50 | | | | |

The above result represents that descriptive statistical analysis result present mean values, median values, also that explain the minimum and maximum rates of each variables included dependent and independent. The result describes that standard deviation of each indicators included childhood trauma 1,2 and 3 the result describe mean values are 1.5800, 1.7600 and 1.5200 result present that positive average value of mean. The result also present standard deviation of each independent variable its values are 0.784, 0.846 and 0.646 its shows that 78%, 84% and 64%

deviate from mean. The epigenetics is mediator variable result present that mean value is 1.52000 the standard deviation rate is 0.86 its shows that 86% deviate from mean value.

The anxiety disorder 1 and 2 represent that dependent variable result describe the mean values are 1.4800 and 1.4600 result shows positive average value of mean the standard deviation rate is 64% and 76% deviate from mean values. The overall result present significant values between them.

Table 2 One-way ANOVA test:

| ANOVA | | | | | | |
|--------------------|----------------|----------------|----|-------------|-------|------|
| | | Sum of Squares | df | Mean Square | F | Sig. |
| childhood trauma 1 | Between Groups | 7.736 | 3 | 2.579 | 5.285 | .003 |
| | Within Groups | 22.444 | 46 | .488 | | |
| | Total | 30.180 | 49 | | | |
| childhood trauma 2 | Between Groups | 2.061 | 3 | .687 | .956 | .422 |
| | Within Groups | 33.059 | 46 | .719 | | |
| | Total | 35.120 | 49 | | | |
| childhood trauma 3 | Between Groups | 1.909 | 3 | .636 | 1.576 | .208 |
| | Within Groups | 18.571 | 46 | .404 | | |
| | Total | 20.480 | 49 | | | |
| epigenetics | Between Groups | 5.767 | 3 | 1.922 | 2.879 | .046 |
| | Within Groups | 30.713 | 46 | .668 | | |
| | Total | 36.480 | 49 | | | |
| anxiety disorder 1 | Between Groups | 1.794 | 3 | .598 | 1.472 | .235 |
| | Within Groups | 18.686 | 46 | .406 | | |
| | Total | 20.480 | 49 | | | |

The above result demonstrate that one-way ANOVA test analysis result describe sum of square value, the mean square values, the F statistic rate and also that significant value between dependent and independent variables. the anxiety disorder 1 shows that between the group and within the group its sum of square rate is 1.794, 18.686 also that 20.480 the result also describe that F statistic rate is 1.472 the significant value is 0.235 its shows that 23% significantly level between them. the result present that positive f

statistic and significant relation between them.

Similarly, the epigenetics is mediator variable result also present that sum of square rate is 5.767 the mean square rate is 2.879 also that significant value is 0.046 its shows that 4% significantly value between them. the overall result demonstrates that positive and significant values between dependent and independent variable for determine the impact between them.

Table 3 Total Variance Explained

| Component | Initial Eigenvalues | | | Extraction Sums of Squared Loadings | | |
|-----------|---------------------|---------------|--------------|-------------------------------------|---------------|--------------|
| | Total | % of Variance | Cumulative % | Total | % of Variance | Cumulative % |
| 1 | 2.316 | 38.597 | 38.597 | 2.316 | 38.597 | 38.597 |
| 2 | 1.230 | 20.502 | 59.100 | 1.230 | 20.502 | 59.100 |
| 3 | 1.027 | 17.121 | 76.220 | 1.027 | 17.121 | 76.220 |
| 4 | .712 | 11.869 | 88.089 | | | |
| 5 | .435 | 7.251 | 95.340 | | | |
| 6 | .280 | 4.660 | 100.000 | | | |

Extraction Method: Principal Component Analysis.

The above result represent that total variance explained the result describe % of variance, the % of cumulative also that total rate of

components. The % of variance are 38.597, 20.502, 17.121, 11.869, 7.251 also that 4.660 each values are shows positive % of variance

of each components. Similarly, the cumulative values are 38.597, 59.100, 76.220, 88.089, 95.340 these shows that positive cumulative values of each indicator. The extraction sums of squared values

are 38.597, 20.502, 17.121 these are all present significant rates between them. the result also describe positive link between the dependent and independent indicator.

Table 4 Test Statistics

| Test Statistics | | | | | | |
|---|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|
| | childhood trauma 1 | childhood trauma 2 | childhood trauma 3 | epigenetics | anxiety disorder 1 | anxiety disorder 2 |
| Chi-Square | 36.880 ^a | 24.720 ^a | 17.440 ^b | 48.240 ^a | 20.320 ^b | 51.280 ^a |
| df | 3 | 3 | 2 | 3 | 2 | 3 |
| Asymp. Sig. | .000 | .000 | .000 | .000 | .000 | .000 |
| a. 0 cells (0.0%) have expected frequencies less than 5. The minimum expected cell frequency is 12.5. | | | | | | |
| b. 0 cells (0.0%) have expected frequencies less than 5. The minimum expected cell frequency is 16.7. | | | | | | |

The above result describe that chi square values of test statistic included dependent and independent variables. the childhood trauma 1, childhood trauma 2 and childhood trauma 3 these are independent variable result shows that 36.880, 24.720, 17.440 positive chi square values.

The other chi square rates are 48.240, 20.320, and 51.280 these are all shows positive and significant values between them. In summary, the chi-square values suggest that there are statistically

significant associations between the variables being examined in your analysis, including childhood trauma variables and possibly other related variables. The specific interpretation of these results would depend on the context of your study and the research question you are addressing.

Chi-square tests are often used in studies involving categorical data to assess whether there is a relationship between variables or whether certain variables have a significant impact on outcomes

Table 5 Paired Samples Test

| | | Paired Differences | | | | t | df | Sig. (2-tailed) | |
|--------|---|--------------------|----------------|-----------------|---|--------|-------|-----------------|-------|
| | | Mean | Std. Deviation | Std. Error Mean | 95% Confidence Interval of the Difference | | | | |
| | | | | | Lower | | | | Upper |
| Pair 1 | childhood trauma 1 - anxiety disorder 1 | .10000 | 1.14731 | .16225 | -.22606 | .42606 | .616 | 49 | .541 |
| Pair 2 | childhood trauma 2 - anxiety disorder 2 | .30000 | 1.07381 | .15186 | -.00517 | .60517 | 1.976 | 49 | .054 |
| Pair 3 | childhood trauma 3 - epigenetics | .00000 | .88063 | .12454 | -.25027 | .25027 | .000 | 49 | 1.000 |

The above result present that paired sample test result describe the mean values, the standard deviation rate, the 95% confidence interval values at lower and upper rates the t statistic value and significant value of each pair. The first pair is childhood trauma 1 and anxiety disorder 1 result shows that mean value is 0.1000 the t statistic rate is 0.616 the significant rate is 0.541 its shows that 54% significantly level between them. the second pair is shows that t statistic is 1.976 the significant value is 0.054 its shows that 5% significantly level. The result also describes that lower confidence interval rate is -0.00517 the upper confidence interval rate is 0.605 its shows that 60% confidence interval between them.

Conclusion:

The connection between childhood trauma, epigenetics, and anxiety disorders emerges as a gripping tale of how events change our biology and psychology in the complex web of human development and mental health. The path from early trauma to the emergence of anxiety disorders is marked by epigenetic modifications—molecular traces that connect our genes with the environments in which we are raised. Childhood trauma has a significant impact on a person's life trajectory since it is a strong environmental influence. These events, whether they result from overt abuse, neglect, or the quiet turmoil of a turbulent upbringing, can function as catalysts, setting off a chain reaction of biological reactions that lasts well beyond the initial occurrence. The developing study of epigenetics reveals the underlying processes that mediate this significant effect, providing understandings of how trauma is passed down through generations and the malleability of the human psychology. The

mechanisms through which the effects of childhood trauma reverberate across a person's genetic landscape include DNA methylation, histone changes, and microRNA control. These epigenetic alterations leave their mark on genes involved in stress response, neurotransmitter signaling, and emotional control, thereby predisposing people to anxiety disorders more easily. Childhood trauma creates the conditions for a lifetime interaction between nature and nurture by changing the basic foundation of gene expression. The amazing flexibility of the brain, or neuroplasticity, emerges as a sign of both sensitivity and resiliency to the long-lasting impacts of trauma-induced epigenetic alterations. The cognitive and emotional reactions that characterize anxiety disorders are supported by the remodeling of synaptic connections, illuminating the intricate interaction between biology and psychology. In addition, the disruption of the HPA axis, which is a key component of the body's stress response, emphasises the systemic nature of these epigenetic changes and expands their effect beyond neuronal circuits. In addition to deepening our understanding of mental health, this investigation into the epigenetics of anxiety disorders has consequences for clinical practise and public health initiatives. Understanding the long-lasting impacts of childhood trauma highlights how essential it is to offer supportive surroundings and therapeutic therapies to reduce any possible adverse effects. The epigenetic lens also encourages novel treatment approaches that target the biological underpinnings of anxiety disorders, providing promise for customised therapies that tackle the underlying reasons. A complex picture of the human experience is painted by the junction of childhood trauma, epigenetics, and

anxiety disorders. It highlights the connection between our genetics and environment and shows how previous hardships leave their marks on us. The prospect of greater comprehension, prevention, and treatment of anxiety disorders grows as research explores deeper into the complex systems that link our experiences to our DNA. In the end, by illuminating this nexus, we provide a route towards recovery, resiliency, and the empowerment of people who are burdened by childhood trauma.

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