Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index *eISSN: 3107-0426*

Original article

Childhood Maltreatment Effects on the Serotonin Transporter Gene and Its Relation to Anxiety and Depression

Mohamed Alwash[1], Ahmed Alsharksi*[2], Abdalla Ali[3]

[1]*Department of Genetics and Biotechnology, Faculty of Science, Misurata University, Libya*
[2]*Department of Nursing Technology, Higher Institute of Science and Technology, Tawargha, Libya.*
[3]*Department of Chemistry, Faculty of Education, Misurata University, Libya.*
***Corresponding Email**. ahmedalsherkas87@gmail.com*

***Keywords**:*
Childhood Maltreatment, SLC6A4, 5-HTTLPR Polymorphism, Serotonin Transporter Gene, Depression, Anxiety, Gene–Environment Interaction.

ABSTRACT

Childhood maltreatment is a major public health concern and represents one of the strongest environmental risk factors associated with the development of psychological disorders, including depression and anxiety. Increasing evidence suggests that genetic susceptibility may influence individual responses to adverse childhood experiences. The serotonin transporter gene (SLC6A4) contains a functional length polymorphism in its promoter region, known as **5-**HTTLPR, which affects serotonin transporter expression and has been implicated in emotional regulation, stress response, anxiety, and depression, particularly among individuals exposed to childhood adversity. This study aimed to investigate the association between childhood maltreatment and symptoms of depression and anxiety among young adults. Additionally, the study explored the relationship between childhood maltreatment and family-related factors, including parental marital status and socioeconomic and educational conditions during childhood, within the context of genetic vulnerability associated with the serotonin transporter system. A cross-sectional study was conducted among 65 randomly selected participants (30 males and 35 females) aged 20–30 years in Misurata, Libya, from 21 June to 6 July 2023. Data were collected using a self-administered questionnaire to assess the severity of childhood maltreatment. Depressive symptoms were evaluated using the Beck Depression Inventory (BDI), and anxiety symptoms were assessed using a self-report questionnaire. Associations between childhood maltreatment, psychological outcomes, and demographic variables were analyzed. Childhood maltreatment was significantly associated with increased symptoms of depression and anxiety among participants. However, no significant association was observed between childhood maltreatment and parental marital status or the family's educational and economic status during childhood. Childhood maltreatment is strongly associated with depression and anxiety symptoms in young adults. These findings support the importance of considering both environmental adversity and genetic susceptibility factors, including serotonin transporter gene variation (5-HTTLPR), in understanding individual differences in vulnerability to stress-related psychological disorders.

Introduction

Childhood Maltreatment

Childhood maltreatment is a significant public health concern and one of the strongest risk factors for current and subsequent psychopathology, adverse health outcomes, and impaired development. It includes various forms of abuse and neglect, including physical abuse, sexual abuse, emotional maltreatment, and physical neglect [1]. Physical abuse refers to situations in which a caregiver or responsible adult intentionally inflicts physical injury on a child through non-accidental means, excluding culturally accepted physical modifications such as circumcision and ear piercing. Sexual abuse involves situations in which a caregiver or responsible adult engages in, or attempts to engage in, sexual activity with a child for sexual gratification or financial benefit. This includes individuals who have authority, responsibility, or power over the child, such as relatives, acquaintances, or other trusted adults. Physical neglect occurs when a caregiver fails to provide the minimum required care necessary to meet a child's basic needs, including adequate nutrition, clothing, housing, hygiene, and medical or dental care. In addition, lack of supervision represents a failure to ensure the child's safety and provide appropriate monitoring according to the child's emotional and developmental needs, including exposure to unsafe environments and inadequate substitute care. Emotional maltreatment occurs when a caregiver persistently interferes with a child's fundamental emotional needs through harmful behaviors, such as rejection, lack of acceptance, restriction of age-appropriate autonomy, and impairment of psychological security and emotional stability [1].

Early adverse childhood experiences may produce long-lasting effects on brain function and increase vulnerability to psychiatric disorders later in life. Childhood adversity and early-life stress have been associated with alterations in serotonin (5-HT) function and changes in serotonergic signaling pathways. These alterations may result from changes in receptor availability and/or impaired receptor function, contributing to increased susceptibility to stress-related psychological disorders [2,3]. Therefore, the present study aimed to investigate the association between childhood

***Received**: 07-06-2026 - **Accepted**: 04-08-2026 - **Published**: 15-08-2026*

Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index eISSN: 3107-0426

maltreatment and symptoms of depression and anxiety among young adults, and to examine the potential role of the serotonin transporter gene (SLC6A4/5-HTTLPR) in relation to psychological outcomes. Additionally, this study evaluated the relationship between childhood maltreatment and selected family-related factors, including parental marital status and socioeconomic and educational conditions during childhood. Furthermore, gender differences in exposure to childhood maltreatment were examined.

Serotonin

5-Hydroxytryptamine (5-HT), commonly known as serotonin, is a monoamine neurotransmitter synthesized in serotonergic neurons of the central nervous system. It plays an important role in regulating multiple behavioral, physiological, and cognitive functions, including memory, mood, emotions, sleep, appetite, wakefulness, and temperature regulation. Serotonin is synthesized from the essential amino acid tryptophan through a series of enzymatic reactions [4].

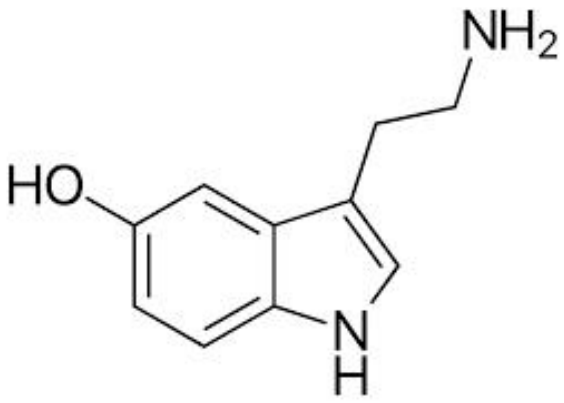

Figure 1, The molecular structure of serotonin [26]

Serotonin Transporter

Serotonin is removed from the synaptic cleft through reuptake by the serotonin transporter (5-HTT), which is expressed on presynaptic serotonergic neurons. The serotonin transporter protein is encoded by the SLC6A4 gene located on chromosome 17 and plays an essential role in regulating serotonergic neurotransmission [5,6]. Genetic variations within the serotonin transporter gene can influence serotonergic activity and have been associated with differences in personality traits and vulnerability to mental health disorders [7].

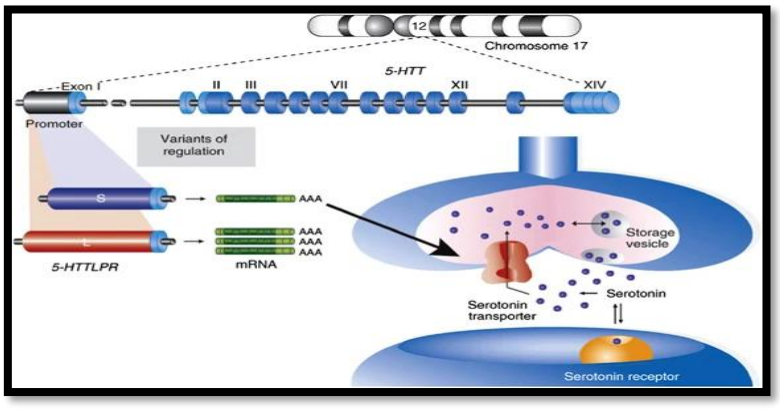

Figure 2, Representation of the long/short polymorphism of the 5-HTT gene and serotonin release, reception, and recycling in neurons [5]

The promoter region of the serotonin transporter gene (5-HTTLPR) contains a 44-base pair insertion/deletion polymorphism that influences transcriptional efficiency. The two common alleles, long (L) and short (S), produce the same protein; however, the long allele is associated with higher transcriptional activity and increased expression of serotonin transporter mRNA and protein compared with the short allele [7]. The short allele, which is associated with reduced serotonin transporter activity, has been linked to increased susceptibility to depression and suicidal ideation. Previous studies have suggested that the serotonergic system contributes to the pathophysiology and treatment of depression through regulatory effects on other neurobiological pathways [8]. Furthermore, approximately 10% of the variance in anxiety-related traits has been attributed to genetic variation involving serotonin transporter function [7]. Individuals carrying one or two copies of the

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*

Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index eISSN: 3107-0426

short 5-HTTLPR allele have demonstrated increased amygdala reactivity to threatening stimuli compared with individuals homozygous for the long allele. These findings suggest that 5-HTTLPR polymorphism contributes to differences in emotional processing and stress responsiveness [6]. Conversely, individuals carrying longer alleles may show greater bias toward processing positive information and reduced attention toward negative stimuli [7].

Anxiety

Anxiety disorders are complex psychiatric conditions characterized by excessive worry, fear, and hyperarousal that can interfere with daily functioning. These disorders are associated with several psychosocial factors, including socioeconomic disadvantage, occupational stress, relationship difficulties, trauma, and interpersonal conflict, suggesting that stressful life events contribute substantially to their development. Biological mechanisms underlying anxiety disorders involve, in part, alterations in the hypothalamic–pituitary–adrenal (HPA) axis, which regulates physiological responses to stress and maintains homeostasis [9]. Anxiety disorders are among the most prevalent psychiatric disorders worldwide, affecting approximately one-quarter of individuals during their lifetime. Although the etiology of anxiety disorders remains incompletely understood, familial and genetic factors have been recognized as important contributors. Advances in molecular genetics have facilitated the identification of potential genetic factors involved in anxiety susceptibility; however, identifying specific causal genes remains challenging due to the complex nature of these disorders [10]. Generalized anxiety disorder (GAD) is characterized by excessive and persistent anxiety and worry about multiple aspects of everyday life, including health, family, work, and financial issues. Traumatic experiences and stressful life events, such as childhood abuse, bereavement, divorce, and major life transitions, may contribute to the onset or worsening of GAD symptoms [11]. GAD is associated with substantial clinical burden due to delayed diagnosis, frequent comorbidity with depressive disorders, other anxiety disorders, and trauma-related disorders. Genetic studies have investigated several candidate genes involved in anxiety susceptibility, including genes related to serotonergic signaling, stress regulation, and neuroplasticity [12]. Two important polymorphisms within the serotonergic system have been frequently investigated in relation to anxiety and depression. The first is the 5-HTTLPR polymorphism located in the promoter region of the serotonin transporter gene (SLC6A4), which consists of long (L) and short (S) alleles. The short allele has been associated with altered stress sensitivity and increased vulnerability to psychological stress. The second is the rs6295 polymorphism in the serotonin receptor gene HTR1A, which has been associated with anxiety and depressive symptoms [9]. Anxiety and fear are normal adaptive emotional responses to threatening situations. However, in anxiety disorders, these responses become excessive or prolonged, resulting in significant impairment of daily functioning [13].

Depression

Depression is a common and disabling psychiatric disorder that affects mood, cognition, behavior, and physical health. It is characterized by persistent sadness, loss of interest, reduced energy, sleep disturbances, and impaired ability to experience pleasure [14]. The common features of depressive disorders include depressed mood, emotional emptiness, irritability, and associated cognitive and somatic changes that significantly impair social and occupational functioning [15]. Environmental risk factors contributing to depression include poverty, adverse family experiences, unhealthy relationships, violence, divorce, childhood maltreatment, and other stressful life events. These factors may interact with biological and genetic vulnerabilities to increase the risk of depressive disorders [16]. Prospective studies have demonstrated that childhood maltreatment significantly increases the risk of depression during adulthood. Global estimates indicate substantial prevalence rates of childhood maltreatment experiences, including physical neglect, physical abuse, emotional abuse, and emotional neglect [17]. The severity and timing of childhood trauma appear to influence the risk of recurrent or chronic depression, increased suicidality, somatic symptoms, and impaired psychological functioning [18]. The human serotonin transporter gene contains two major polymorphisms: a variable-number tandem repeat (VNTR) polymorphism located in intron 2 (5-HTT-VNTR) and a variable insertion/deletion polymorphism in the promoter region (5-HTTLPR). These polymorphisms influence serotonin transporter expression and may contribute to individual differences in vulnerability to depression and stress-related disorders [8].

Literature Review

Previous studies have investigated the relationship between childhood maltreatment, serotonin transporter gene polymorphisms, and the development of depression and anxiety disorders. These studies have provided evidence supporting the contribution of gene–environment interactions to individual differences in vulnerability to psychological disorders. A study investigating the interaction between stressful life events and serotonin transporter polymorphism included 549 male and female twins with a mean age of 34.9 years. The study assessed major depressive episodes and generalized anxiety symptoms and demonstrated that individuals carrying two short (S) alleles of the 5-HTTLPR polymorphism were more sensitive to the depressogenic effects of stressful life events compared with individuals carrying one or two long (L) alleles. However, serotonin transporter genotype did not significantly modify the effect of stressful life events on generalized anxiety symptoms [19]. A study involving 118 young adults examined the effects of early family environment, current adversity, and serotonin transporter promoter polymorphism on depressive symptoms. The findings

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*

Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index eISSN: 3107-0426

showed that early family stress was significantly associated with depressive symptoms. Furthermore, individuals homozygous for the short allele of 5-HTTLPR exhibited greater depressive symptoms following adverse experiences, while supportive environments appeared to reduce vulnerability to psychological distress [20]. A longitudinal cohort study of 309 adolescents and young adults evaluated the association between 5-HTTLPR polymorphism, environmental adversity, depression, and anxiety. The results demonstrated that both family adversity and stressful life events were strongly associated with depressive and anxiety symptoms. Genetic variation in the serotonin transporter gene influenced individual responses to environmental stressors, supporting the role of gene–environment interactions in psychiatric vulnerability [21].

A population-based study involving 942 older adults investigated the association between adverse childhood experiences, serotonin transporter genotype, and late-life depression. Childhood traumatic experiences were associated with approximately a twofold increased risk of depression. Significant risk factors included childhood poverty, parental mental illness, excessive physical punishment, verbal abuse, humiliation, and mistreatment by adults outside the family. Interactions between childhood adversity and 5-HTTLPR genetic variation were also observed [22]. A study involving 363 healthy individuals investigated the effects of childhood maltreatment and serotonin transporter genetic variants on anxiety sensitivity. Participants were assessed using the Childhood Trauma Questionnaire and genotyped for functional serotonin transporter variants, including 5-HTTLPR and rs25531. The results demonstrated a significant gene–environment interaction between serotonin transporter variants and childhood maltreatment, particularly affecting the somatic component of anxiety sensitivity [23].

A risk-enriched study of 273 women examined whether serotonin transporter polymorphism moderated the relationship between childhood maltreatment and chronic depression. Childhood maltreatment was assessed using standardized interviews, and depressive episodes were evaluated prospectively. The findings showed that the short allele of 5-HTTLPR significantly increased vulnerability to chronic depression among individuals exposed to childhood maltreatment, supporting a genetic contribution to stress-related depressive outcomes [24]. A longitudinal investigation examining participants from childhood to adulthood demonstrated that maltreatment and less efficient forms of the serotonin transporter promoter polymorphism contributed to increased risk of depression. The findings supported a gene–environment interaction model in which childhood maltreatment combined with genetic susceptibility increased vulnerability to depressive symptoms across development [25]. Another investigation examined the interaction between childhood trauma and low-expression 5-HTTLPR genotypes in relation to suicidal behavior among individuals with substance dependence. The results showed that individuals with greater childhood trauma exposure and low-expression serotonin transporter genotypes had an increased risk of suicide attempts, suggesting that genetic factors may influence the psychological consequences of early-life adversity [26].

A comprehensive review of generalized anxiety disorder genetics highlighted the contribution of serotonergic and stress-related pathways to anxiety vulnerability. Candidate genes, including SLC6A4, HTR1A, MAOA, BDNF, and genes involved in stress regulation, have been investigated in relation to anxiety phenotypes. The review emphasized the importance of gene–environment interactions involving childhood trauma and stressful life events in the development of anxiety disorders [12].

A genetic study investigating anxiety-related traits in healthy individuals analyzed polymorphisms involved in HPA-axis regulation and serotonergic signaling. DNA samples from 72 healthy participants were examined for variants including NR3C1, FKBP5, OXTR, SLC6A4 (5-HTTLPR), and HTR1A. The findings suggested that specific genetic variants may contribute to differences in anxiety susceptibility, with some effects influenced by sex differences [9].

Methods

Study Design and Participants

A cross-sectional survey study was conducted among 65 randomly selected participants (30 males and 35 females) aged 20–30 years in Misurata, Libya, between 21 June and 6 July 2023. Participants were assessed for childhood maltreatment experiences, depressive symptoms, and anxiety levels using structured self-administered questionnaires. Additional demographic information, including parental marital status and the family's economic and educational status during childhood, was collected. All participants provided informed consent before participation.

Assessment of Childhood Maltreatment

Childhood maltreatment was assessed using a 63-item self-report questionnaire designed to evaluate the severity and frequency of adverse childhood experiences, including different forms of abuse and neglect.

Assessment of Depression

Depressive symptoms were assessed using the Beck Depression Inventory (BDI). The BDI is a 21-item self-report instrument widely used to measure the severity of depressive symptoms. Based on the total score, participants were classified into different categories, including no depression, mild mood disturbance, moderate depression, severe depression, and extreme depression.

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*

Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index *eISSN: 3107-0426*

Assessment of Anxiety

Anxiety symptoms were evaluated using a 58-item self-report questionnaire. The obtained scores were categorized into five levels of anxiety severity: low, mild, moderate, severe, and extreme anxiety.

Statistical Analysis

Data were analyzed using descriptive and inferential statistical methods. Percentages were calculated to describe categorical variables. The normality of data distribution was assessed using normality tests. The Spearman rank correlation coefficient (Spearman's rho) was used to evaluate the strength and direction of associations between childhood maltreatment scores and psychological outcomes, including depression and anxiety. A p-value <0.05 was considered statistically significant.

Results

Classification of participants by gender

The study included 65 participants (30 males at a rate of 46.2, 35 females at a rate of 53.8 females) (Table 1).

Table 1, Classification of participants by gender.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Female	35	53.8	53.8	53.8
	Male	30	46.2	46.2	100.0
	Total	65	100.0	100.0	

About 4 participants had dead fathers (6.2%), and participants (93.8 %) did not (Table 2). One participant had a dead mother (1.5%), and 64 participants (98.5%) did not (Table 3).

Table 2, Classification of participants by the death of a father.

The death of a father					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	4	6.2	6.2	6.2
	No	61	93.8	93.8	100.0
	Total	65	100.0	100.0	

Table 3, Classification of participants by the death of a mother

The death of a mother					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	1	1.5	1.5	1.5
	No	64	98.5	98.5	100.0
	Total	65	100.0	100.0	

Only 1 participant had divorced parents (1.5%), compared to 64 participants (98.5%) who did not have divorced parents (Table 4).

Table 4, Classification of participants by divorced parents

Table: Divorced					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	1	1.5	1.5	1.5
	No	64	98.5	98.5	100.0
	Total	65	100.0	100.0	

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*

Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index *eISSN: 3107-0426*

Also 1 participant had separated parents (1.5%), compared to 64 participants (98.5%) who did not have separated parents (Table 5).

Table 5, Classification of participants by separated parents

Separated					
		Frequency	**Percent**	**Valid Percent**	**Cumulative Percent**
Valid	Yes	1	1.5	1.5	1.5
	No	64	98.5	98.5	100.0
	Total	65	100.0	100.0	

Concerning another marriage, 3 participants had another marriage for their parents (4.6 %), compared to 62 participants (95.4 %) who did not have one (Table 6).

Table 6, Classification of participants by another marriage for parents

Another Marriage					
		Frequency	**Percent**	**Valid Percent**	**Cumulative Percent**
Valid	Yes	3	4.6	4.6	4.6
	No	62	95.4	95.4	100.0
	Total	65	100.0	100.0	

Classification of participants by economic status of the family during childhood

21 participants had a monthly income of 500 or more, 21 participants had a monthly income of 1000 or more, and 23 participants had a monthly income of 3000 or more.

Table 7, Participants classified by economic status

Table: economic status					
		Frequency	**Percent**	**Valid Percent**	**Cumulative Percent**
Valid	500 or more	21	32.3	32.3	32.3
	1000 or more	21	32.3	32.3	64.6
	3000 or more	23	35.4	35.4	100.0
	Total	65	100.0	100.0	

Classification of participants by educational status of the family during childhood

Most fathers were educated, while only one were illiteracy (Table 8)

Table 8, Classified participants by educational status of the father

The education status of the father					
		Frequency	**Percent**	**Valid Percent**	**Cumulative Percent**
Valid	Illiteracy	1	1.5	1.5	1.5
	Primary school	7	10.8	10.8	12.3
	Preparatory school	17	26.2	26.2	38.5
	Secondary school	9	13.8	13.8	52.3
	Bachelor	19	29.2	29.2	81.5
	Master, doctorate	12	18.5	18.5	100.0
	Total	65	100.0	100.0	

Classification of participants by educational status of the mother

Most mothers were educated, while 5 were illiterate (Table 9)

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*

Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index *eISSN: 3107-0426*

Table 9, Classification of participants by educational status of the mother

The educational status of the mother					
		Frequency	**Percent**	**Valid Percent**	**Cumulative Percent**
Valid	llliteracy	5	7.7	7.7	7.7
	Proficient in reading and writing	4	6.2	6.2	13.8
	Primary school	22	33.8	33.8	47.7
	Preparatory school	18	27.7	27.7	75.4
	Secondary school	7	10.8	10.8	86.2
	Bachelor	6	9.2	9.2	95.4
	Master, doctorate	3	4.6	4.6	100.0
	Total	65	100.0	100.0	

Classification of Participants by the Depression Scale

Among the four levels of depression, the category that recorded the highest value was Mild mood disturbance, with a number of 30 at a rate of 46.15%, and the lowest value was recorded at the level of severe depression, with a number of 7 at a rate of 10.76% (Table 10).

Table 10, classification of participants by category on the Depression Scale.

		Frequency	**Percent**
Valid	No depression	11	16.92
	Mild mood disturbance	30	46.15
	moderate depression	17	26.15
	severe depression	7	10.76
	Total	65	100

Classification of participants by the anxiety scale

Among the three levels of anxiety, the category that recorded the highest value was Mild anxiety, with 29 at a rate of 44.61%, and the lowest value was recorded at the level of severe anxiety, with 8 at a rate of 12.30% (Table11).

Table11, Classification of participants by the Anxiety Scale

		Frequency	**Percent**
Valid	weak anxiety	28	43.07
	Mild anxiety	29	44.61
	moderate anxiety	8	12.30
	Total	65	100

Determine the link between childhood maltreatment and the death of a father

From Table 12, we notice that the relationship between Childhood maltreatment and the death of a father is not statistically significant; that is, there is no significant relationship between them.

Table12, The link between childhood maltreatment and the death of a father

Correlations				
			Childhood maltreatment	**the death of a father**
Spearman's rho	Childhood maltreatment	Correlation Coefficient	1.000	0.109-
		Sig. (2-tailed)	.	0.386
		N	65	65

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*

Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index eISSN: 3107-0426

	The death of a father	Correlation Coefficient	0.109-	1.000
		Sig. (2-tailed)	0.386	.
		N	65	65

Determine the link between childhood maltreatment and the death of a mother

From the data in (Table,13), we notice that the relationship between Childhood maltreatment and the death of a mother is not statistically significant; that is, there is no significant relationship between them.

Table13: The link between childhood maltreatment and the death of a mother

Correlations				
			childhood maltreatment	**the death of a mother**
Spearman's rho	childhood maltreatment	Correlation Coefficient	1.000	0.137
		Sig. (2-tailed)		0.278
		N	65	65
	The death of a mother	Correlation Coefficient	0.137	1.000
		Sig. (2-tailed)	0.278	.
		N	65	65

Determine the link between childhood maltreatment and economic status

From the data in (Table ,14), we notice that the relationship between Childhood maltreatment and economic status is not statistically significant; that is, there is no significant relationship between them.

Table 14, The link between childhood maltreatment and economic status.

Correlations				
			Childhood maltreatment	**Economic status**
Spearman's rho	Childhood maltreatment	Correlation Coefficient	1.000	0.036
		Sig. (2-tailed)	.	0.778
		N	65	65
	Economic status	Correlation Coefficient	0.036	1.000
		Sig. (2-tailed)	0.778	.
		N	65	65

Determine the link between childhood maltreatment and the educational status of the family during childhood

From the data, there is no relationship between abuse and education for the mother's education and the father's education, as the correlation coefficient was weak and not statistically significant (Sig > 0.05) (Table15).

Table 15. The link between childhood maltreatment and educational status of the family during childhood

Correlations					
	Childhood maltreatment			**Educational status of the father.**	**Educational status of the mother**
Spearman's rho		Correlation	1.000	0.094	0.116

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*

Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index eISSN: 3107-0426

	Childhood maltreatment	Coefficient			
		Sig. (2-tailed)	.	0.456	0.359
		N	65	65	65
	Educational status of the father.	Correlation Coefficient	0.094	1.000	0.387
		Sig. (2-tailed)	.456	.	0.001
		N	65	65	65
	Educational status of the mother.	Correlation Coefficient	0.116	0.387	1.000
		Sig. (2-tailed)	0.359	0.001	
		N	65	65	65

Correlation is significant at the 0.01 level (2-tailed).

Determine the link between depression and anxiety toward child maltreatment

Through the Spearman correlation coefficient between treatment, depression, and anxiety, we find that the correlation coefficient is positive, and this indicates the direct relationship between abuse and both anxiety and depression, while the correlation coefficient, which is of a significant level (0.01), indicates that there is a strong relationship between childhood maltreatment and Depression in adults also indicates a strong relationship between childhood maltreatment and anxiety in adults (Table 16, Table 17).

Table 16, The link between depression and anxiety toward child maltreatment

Correlations					
			Childhood maltreatment	**Depression**	**Anxiety**
Spearman's rho	Childhood maltreatment	Correlation Coefficient	1.000	0.384	0.484
		Sig. (2-tailed)		0.002	0.000
		N	65	65	65
	Depression	Correlation Coefficient	0.384	1.000	0.252
		Sig. (2-tailed)	0.002		0.043
		N	65	65	65
	Anxiety	Correlation Coefficient	0.484	0.252	1.000
		Sig. (2-tailed)	0.000	0.043	
		N	65	65	65

***. Correlation is significant at the 0.01 level (2-tailed).*
**. Correlation is significant at the 0.05 level (2-tailed).*

Table 17, The link between depression and anxiety toward child maltreatment

	Depression	**Anxiety**
childhood maltreatment	0.384	0.484

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*

Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index eISSN: 3107-0426

Determine gender differences in childhood maltreatment

From the obtained data, it became clear that the data follows a normal distribution, so parametric statistics were used, where the T-test was used to find out whether there is a difference in the opinions of males and females in the context of childhood maltreatment (Table 18). The paired-samples t-test was used to compare the means of two measurements.

Table 18, Tests of Normality for Gender differences in childhood.

Tests of Normality						
	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
childhood maltreatment	0.119	65	0.023	0.925	65	0.001

a. Lilliefors Significance Correction

H0: The mean of the male answers = the mean of the female answers
H1: The mean of the male answers ± the mean of the female answers

A comparison is made between the mean ranks in (Table 21). We find that there is a difference in the ranks, where females had 37.39 while the males had 27.88. From the table, looking at the T-test, we find that it is not significant, as it was less than (0.05), and this indicates the acceptance of the alternative hypothesis, meaning that the mean answers of males are not equal to the mean answers of females, meaning that there are gender differences in child maltreatment.

Table 19, Descriptive Statistics for Gender difference in childhood

Descriptive Statistics								
	N	Mean	Std. Deviation	Minimum	Maximum	Percentiles		
						25th	50th (Median)	75th
childhood maltreatment	65	1.6639	0.23784	1.18	2.19	1.5238	1.6667	1.8333
gender	65	1.4615	0.5024	1.00	2.00	1.0000	1.0000	2.0000

Table 20, Mann-Whitney Test.

Ranks				
	Gender	N	Mean Rank	Sum of Ranks
childhood maltreatment	Female	35	37.39	1308.50
	Male	30	27.88	836.50
	Total	65		

Table 21, Test Statistics for gender differences in childhood.

Test Statistics	
	childhood maltreatment
Mann-Whitney U	371.500
Wilcoxon W	836.500
Z	2.021-
Asymp. Sig. (2-tailed)	0.043

a. Grouping Variable: gender

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*

Libyan Med J. 2026;18(8):460-471
https://doi.org/10.69667/lmj.26802
Libyan Medical Journal
https://lmj.ly/index.php/ojs/index eISSN: 3107-0426

Discussion

This study investigated the association between childhood maltreatment and psychological outcomes, including depression and anxiety symptoms, as well as its relationship with selected family-related factors, including parental marital status and socioeconomic status during childhood. The findings of the present study showed that there was no significant association between childhood maltreatment and parental marital status-related factors, including parental death, divorce, separation, or remarriage. These findings suggest that changes in family structure alone may not be sufficient predictors of childhood maltreatment within the studied population. Although family instability has been widely considered an important risk factor for adverse childhood experiences, previous studies have reported inconsistent findings. Previous research has demonstrated that family dysfunction, parental conflict, and stressful home environments may increase the risk of child maltreatment.

Family difficulties may influence parenting behaviors and increase parental stress, which can contribute to neglect or abuse. However, the absence of a significant association in the current study may be explained by differences in cultural background, family support systems, social networks, and characteristics of the studied sample. Childhood maltreatment is considered a complex phenomenon resulting from interactions between multiple factors, including parental psychological characteristics, social environment, economic conditions, and child-related factors rather than a single family characteristic [24,25]. Similarly, the present study did not identify a significant relationship between childhood maltreatment and parental socioeconomic status during childhood. Although socioeconomic disadvantage has frequently been reported as a risk factor for child maltreatment, financial difficulties may act indirectly through increased parental stress, reduced social support, and impaired family functioning.

Economic status alone may not fully explain the occurrence of maltreatment because other factors, such as parental mental health, parenting practices, emotional availability, and family relationships, may play important roles. In contrast, the current study demonstrated a significant positive association between childhood maltreatment and symptoms of depression and anxiety among young adults. Participants who reported higher levels of childhood maltreatment showed increased depressive and anxiety symptoms. These findings are consistent with previous studies demonstrating that adverse childhood experiences are strongly associated with long-term psychological consequences, including mood disorders, anxiety disorders, and emotional dysregulation [7,17]. The relationship between childhood maltreatment and later psychological disorders may be explained by long-lasting effects of early-life stress on neurobiological systems involved in emotional regulation.

Childhood adversity has been associated with alterations in the hypothalamic–pituitary–adrenal (HPA) axis, serotonergic neurotransmission, and stress-response pathways. These biological changes may increase vulnerability to depression and anxiety during adolescence and adulthood [11,18]. The serotonergic system has received considerable attention in explaining individual differences in response to childhood adversity. The serotonin transporter gene (SLC6A4) and its functional polymorphism 5-HTTLPR have been suggested to influence susceptibility to stress-related psychiatric outcomes. Individuals carrying the short allele may exhibit altered serotonin transporter expression and increased sensitivity to environmental stressors. Previous studies have reported interactions between childhood maltreatment and 5-HTTLPR variation in predicting depression and anxiety symptoms, supporting a gene–environment interaction model [18,22]. The findings of this study are also consistent with previous evidence indicating that childhood abuse and neglect increase the risk of depressive symptoms and anxiety disorders in adulthood.

Early traumatic experiences may affect emotional processing, cognitive development, and stress regulation, resulting in persistent psychological vulnerability. Therefore, early identification of children exposed to maltreatment and providing psychological support may reduce the long-term consequences of adverse childhood experiences. The present study has several implications. Understanding the relationship between childhood maltreatment and mental health outcomes may assist healthcare professionals in identifying individuals at increased risk of depression and anxiety. Furthermore, prevention programs targeting vulnerable families and improving parental awareness may contribute to reducing childhood maltreatment and its long-term effects.

Conclusion

The present study demonstrates that childhood maltreatment is significantly associated with increased symptoms of depression and anxiety among young adults. These findings highlight the long-term psychological consequences of adverse childhood experiences and support the important role of early-life stress in increasing vulnerability to mental health problems [1,7,6]. Although no significant association was observed between childhood maltreatment and parental marital status or family socioeconomic status during childhood, the results suggest that childhood maltreatment is a complex phenomenon influenced by multiple biological, psychological, and environmental factors.

Conflict of interest. Nil

References

1. Zeanah CH, Humphreys KL. Child abuse and neglect. J Am Acad Child Adolesc Psychiatry. 2018;57(9):637-44.

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*

2. Roy A, Hu XZ, Janal MN, Goldman D. Interaction between childhood trauma and serotonin transporter gene variation in suicide. Neuropsychopharmacology. 2007;32(9):2046-52.
3. Jonnakuty C, Gragnoli C. What do we know about serotonin? J Cell Physiol. 2008;217(2):301-6.
4. McCormack K, Newman TK, Higley JD, Maestripieri D, Sanchez MM. Serotonin transporter gene variation, infant abuse, and responsiveness to stress in rhesus macaque mothers and infants. Horm Behav. 2009;55(4):538-47.
5. De Neve JE. Functional polymorphism (5-HTTLPR) in the serotonin transporter gene is associated with subjective well-being: evidence from a US nationally representative sample. J Hum Genet. 2011;56(6):456-9.
6. Miller JM, Kinnally EL, Ogden RT, Oquendo MA, Mann JJ, Parsey RV. Reported childhood abuse is associated with low serotonin transporter binding in vivo in major depressive disorder. Synapse. 2009;63(7):565-73.
7. Comtois-Cabana M, Barr E, Provençal N, Ouellet-Morin I. Association between child maltreatment and depressive symptoms in emerging adulthood: the mediating and moderating roles of DNA methylation. PLoS One. 2023;18(1):e0280203.
8. Kapfhammer HP. Somatic symptoms in depression. Dialogues Clin Neurosci. 2006;8(2):227-39.
9. Cui R. Editorial: A systematic review of depression. Curr Neuropharmacol. 2015;13(4):480.
10. Neumeister A, Young T, Stastny J. Implications of genetic research on the role of serotonin in depression: emphasis on the serotonin type 1A receptor and the serotonin transporter. Psychopharmacology (Berl). 2004;174(4):512-24.
11. Lindholm H, Morrison I, Krettek A, et al. Genetic risk-factors for anxiety in healthy individuals: polymorphisms in genes important for the HPA axis. BMC Med Genet. 2020;21:184.
12. Smoller JW, Block SR, Young MM. Genetics of anxiety disorders: the complex road from DSM to DNA. Depress Anxiety. 2009;26(11):965-75.
13. Gottschalk MG, Domschke K. Genetics of generalized anxiety disorder and related traits. Dialogues Clin Neurosci. 2017;19(2):159-68.
14. Shimada-Sugimoto M, Otowa T, Hettema JM. Genetics of anxiety disorders: genetic epidemiological and molecular studies in humans. Psychiatry Clin Neurosci. 2015;69(7):388-401.
15. Chand SP, Arif H, Kutlenios RM. Depression (Nursing). In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [cited 2026 Aug 12]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK559035/
16. Šalamon Arčan I, Kouter K, Videtič Paska A. Depressive disorder and antidepressants from an epigenetic point of view. World J Psychiatry. 2022;12(9):1150-68.
17. Conejero I, Olié E, Calati R, Ducasse D, Courtet P. Psychological pain, depression, and suicide: recent evidences and future directions. Curr Psychiatry Rep. 2018;20(5):33.
18. Kendler KS, Kuhn JW, Vittum J, Prescott CA, Riley B. The interaction of stressful life events and a serotonin transporter polymorphism in the prediction of episodes of major depression: a replication. Arch Gen Psychiatry. 2005;62(5):529-35.
19. Taylor SE, Way BM, Welch WT, Hilmert CJ, Lehman BJ, Eisenberger NI. Early family environment, current adversity, the serotonin transporter promoter polymorphism, and depressive symptomatology. Biol Psychiatry. 2006;60(7):671-6.
20. Laucht M, Treutlein J, Blomeyer D, et al. Interaction between the 5-HTTLPR serotonin transporter polymorphism and environmental adversity for mood and anxiety psychopathology: evidence from a high-risk community sample of young adults. Int J Neuropsychopharmacol. 2009;12(6):737-47.
21. Ritchie K, Jaussent I, Stewart R, et al. Association of adverse childhood environment and 5-HTTLPR genotype with late-life depression. J Clin Psychiatry. 2009;70(9):1281-8.
22. Klauke B, Deckert J, Reif A, et al. Serotonin transporter gene variation and childhood maltreatment in anxiety sensitivity. J Neural Transm. 2011;118:1101-8.
23. Cutuli JJ, Raby KL, Cicchetti D, Englund MM, Egeland B. Contributions of maltreatment and serotonin transporter genotype to depression in childhood, adolescence, and early adulthood. J Affect Disord. 2013;149(1-3):30-7.
24. Brown GW, Ban M, Craig TK, Harris TO, Herbert J, Uher R. Serotonin transporter length polymorphism, childhood maltreatment, and chronic depression: a specific gene-environment interaction. Depress Anxiety. 2013;30(1):5-13.
25. Albalawi FM, et al. Childhood trauma interacts with low expressing 5-HTTLPR genotypes to increase risk of suicidal behavior. [unverified journal details; please verify from original source].
26. Naeem M, Chadeayne AR, Golen JA, Manke DR. Crystal structure of serotonin. Acta Crystallogr E Crystallogr Commun. 2022;78(4):365-8.

Received: *07-06-2026 -* ***Accepted:*** *04-08-2026 -* ***Published:*** *15-08-2026*