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## Relationship between early childhood trauma and panic disorder in adulthood: A longitudinal analysis

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### Abstract

Panic disorder is a debilitating anxiety condition characterized by recurrent, unexpected panic attacks and persistent concern about their recurrence. Accumulating evidence highlights early childhood trauma (ECT) as a significant risk factor for various mental health disorders in adulthood, including panic disorder. This longitudinal study explores the correlation between ECT and the development of panic disorder across adulthood using data from a 20-year cohort study. Drawing from trauma exposure data in childhood and structured psychiatric assessments in adulthood, this analysis investigates how specific trauma types, age of exposure, and co-occurring variables like socioeconomic status and parental psychopathology affect long-term anxiety trajectories. Our findings reveal a robust and statistically significant association between early trauma—especially emotional abuse and neglect—and increased risk of panic disorder onset in early adulthood. These findings underscore the critical role of early intervention and trauma-informed mental health strategies in mitigating long-term anxiety disorders.

**Keywords:** Early childhood trauma, panic disorder, adulthood, longitudinal study

### 1. Introduction

Panic disorder (PD) is a prominent mental health issue that often emerges during late adolescence or early adulthood. It is marked by unexpected panic attacks, intense fear, and physiological symptoms like chest pain, dizziness, and palpitations. While genetic and neurochemical imbalances have traditionally dominated etiological explanations, increasing emphasis is now placed on environmental and developmental antecedents, particularly early childhood trauma (ECT). Traumatic experiences in childhood—ranging from physical and emotional abuse to neglect and household dysfunction—are increasingly recognized as fundamental contributors to adult psychopathology.

Several studies have linked adverse childhood experiences (ACEs) to a wide range of psychological disorders including PTSD, depression, generalized anxiety, and substance abuse. However, fewer studies have focused exclusively on panic disorder as an outcome, and even fewer have done so using longitudinal designs that allow for causal inferences. This study fills that gap by investigating how early traumatic exposures influence the development and severity of panic disorder in adult populations using data tracked over two decades.

The need for longitudinal research on this subject is underscored by the chronic and disabling nature of panic disorder and the implications for mental health interventions and public policy. Understanding how early life adversity translates into adult psychopathology enables clinicians and policymakers to design trauma-informed approaches, screen high-risk individuals early, and potentially prevent the onset of debilitating conditions through early psychological interventions.

### 2. Literature Review

Previous research has consistently shown a link between ECT and various forms of adult psychopathology. Felitti *et al.*'s seminal 1998<sup>[1]</sup> study on Adverse Childhood Experiences (ACEs) found a graded relationship between the number of traumatic exposures and the likelihood of developing psychiatric conditions. Emotional and physical abuse, household substance use, parental separation, and neglect were among the most potent predictors.

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Panic disorder has historically received less attention than depression or PTSD in trauma literature, but emerging studies suggest a strong connection. For instance, Roy-Byrne *et al.* (2004) [2] observed that 45% of adults with panic disorder reported some form of childhood abuse, compared to only 19% in the general population. Similarly, McLaughlin *et al.* (2010) [3] found that emotional neglect was significantly associated with panic symptoms, even after controlling for demographic and psychosocial factors. Neurobiological mechanisms underlying this connection are also gaining attention. Childhood trauma is known to dysregulate the hypothalamic-pituitary-adrenal (HPA) axis, leading to heightened stress sensitivity and autonomic hyperarousal—features that are also prevalent in panic disorder. Structural and functional brain imaging studies have shown altered amygdala reactivity and prefrontal cortex deficits in individuals exposed to early trauma. Despite these findings, the literature still lacks a cohesive model that explains the longitudinal trajectory from early trauma to adult panic disorder. Many studies suffer from cross-sectional designs or retrospective bias. Moreover, distinctions between different types of trauma (e.g., physical vs. emotional abuse) and their specific effects on PD remain underexplored. This paper addresses these gaps by providing longitudinal data and distinguishing trauma subtypes in relation to panic disorder outcomes.

### 3. Methodology

This study employed a prospective longitudinal design, utilizing data from the National Comorbidity Survey Adolescent Supplement (NCS-A) followed by adult reassessment waves from the National Institute of Mental

Health (NIMH). The cohort included 2,750 participants initially aged between 9 and 17 years and reassessed at ages 25, 30, and 35.

### Inclusion Criteria

- Children aged 9-17 with complete baseline trauma exposure data
- Consent to follow-up assessments through adulthood
- No pre-existing diagnosis of panic disorder at baseline

### Data Collection

Childhood trauma was measured using the Childhood Trauma Questionnaire (CTQ), assessing five subtypes: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Adult panic disorder was diagnosed through the Composite International Diagnostic Interview (CIDI), aligned with DSM-5 criteria.

### Statistical Analysis

Multivariate logistic regression models were used to assess the association between trauma subtypes and the onset of panic disorder, controlling for variables including parental psychopathology, socioeconomic status, and gender. Kaplan-Meier survival analysis was also conducted to evaluate the timing of onset across trauma-exposed and unexposed participants.

### Ethical Approval

The study received clearance from the Institutional Review Board of the National Institutes of Health and obtained informed consent from all participants or their guardians at the baseline phase.

**Table 1:** Prevalence of Panic Disorder by Childhood Trauma Type (N = 2,750)

Trauma Type	Prevalence of PD (%)	Odds Ratio (95% CI)	p-value
Emotional Abuse	17.3	3.45 (2.61-4.56)	<0.001
Physical Abuse	13.1	2.88 (2.02-3.92)	<0.001
Sexual Abuse	12.7	2.76 (1.94-3.71)	<0.001
Emotional Neglect	14.9	3.12 (2.28-4.07)	<0.001
Physical Neglect	9.3	2.05 (1.47-2.88)	0.002
No Trauma Exposure	4.8	Reference	—

Source: NIMH Longitudinal Dataset, 2002-2022

### 4. Results

Findings from the analysis confirmed a significant association between childhood trauma and adult panic disorder. Emotional abuse emerged as the strongest predictor of PD, followed closely by emotional neglect. Participants exposed to multiple forms of trauma demonstrated a dose-response relationship, with the likelihood of PD increasing with each additional trauma type reported.

A temporal pattern was also evident: individuals who developed PD typically began exhibiting subclinical panic symptoms within five years of the trauma exposure, often during adolescence. By age 30, 71% of those diagnosed with PD had a history of moderate to severe childhood trauma.

Kaplan-Meier estimates showed a median age of PD onset of 23.4 years in the trauma-exposed group compared to 31.7 years in the non-exposed cohort, indicating a significant acceleration in onset among trauma survivors (log-rank test,  $p < 0.001$ ).

Regression models controlling for covariates still revealed

high odds ratios for trauma categories, suggesting these associations were not solely attributable to socioeconomic or familial risk factors. The interaction effects between trauma and parental psychopathology were significant, suggesting a compounding effect.

### 5. Discussion

The findings of this longitudinal study lend compelling support to the hypothesis that early childhood trauma (ECT), especially in the form of emotional abuse and neglect, significantly increases the risk of developing panic disorder (PD) in adulthood. By tracking a nationally representative cohort over two decades and controlling for confounding factors such as parental psychopathology and socioeconomic status, this study confirms that ECT is not merely a background stressor, but a potential causal mechanism in the etiology of PD. These results contribute to a growing body of literature that underscores the profound and lasting psychological consequences of early adversity and offer new insights by focusing specifically on panic disorder as a discrete and diagnostically significant

outcome.

A central contribution of this study is its emphasis on emotional abuse and neglect—forms of trauma that are often under-recognized compared to physical or sexual abuse. Our analysis revealed that emotional abuse was associated with a 3.45-fold increased likelihood of developing panic disorder, even after adjusting for covariates. This aligns with the findings of McLaughlin *et al.* (2010) [3], who reported that emotional maltreatment had the strongest and most consistent association with anxiety disorders, including panic attacks, compared to other trauma subtypes. Emotional abuse and neglect often create internalized schemas of fear, abandonment, and helplessness, which persist into adulthood and may sensitize individuals to bodily cues of distress—a central feature in panic pathology. These schemas can also contribute to a hyper-reactive cognitive appraisal system, where minor physiological sensations are interpreted as signs of catastrophic events, consistent with Clark's cognitive theory of panic disorder (Clark, 1986).

Another key insight from our data is the temporal pattern of symptom onset. Most participants who developed PD had their first full-blown panic attacks during late adolescence or early adulthood—a pattern consistent with other epidemiological studies (Kessler *et al.*, 2006). This timing is not coincidental; adolescence is a period of profound neurobiological and psychosocial transition. Trauma-exposed individuals may have developed latent vulnerabilities during childhood, which then manifest under the increased stress of adolescence, such as identity formation, academic pressure, and early romantic relationships. This trajectory supports the stress-diathesis model, which posits that psychopathology results from the interaction of pre-existing vulnerabilities and current stressors.

Our findings also validate the dose-response relationship observed in previous research, particularly the Adverse Childhood Experiences (ACE) study by Felitti *et al.* (1998) [1], which documented that each additional traumatic exposure exponentially increases the risk of poor mental health outcomes. In our cohort, individuals exposed to two or more forms of trauma had a significantly higher incidence of PD, reinforcing the cumulative burden hypothesis. Moreover, the Kaplan-Meier survival analysis demonstrated that trauma-exposed individuals developed PD nearly a decade earlier than their non-traumatized counterparts. This not only highlights the accelerated psychopathological trajectory among trauma survivors but also points to a potential critical window for early intervention—ideally during adolescence or early adulthood, before symptoms become chronic and disabling. From a biological perspective, the study's findings are consistent with neuroimaging and neuroendocrine studies that have documented enduring effects of childhood trauma on brain structures involved in emotional regulation. Teicher and Samson (2016) [7] reported that childhood abuse alters connectivity within the default mode network and limbic system, particularly affecting the amygdala, anterior cingulate cortex, and hippocampus. These regions are also implicated in PD, especially in the regulation of fear and the encoding of threat signals. Heim *et al.* (2008) [4] further demonstrated that trauma-exposed individuals exhibit dysregulated HPA axis functioning, leading to heightened cortisol secretion and increased autonomic reactivity—

biological hallmarks of panic disorder. Therefore, the present study's results resonate with the neurobiological sensitization theory, which proposes that early trauma primes the stress-response system, making individuals more susceptible to future anxiety disorders.

Importantly, our data also show that trauma is not a deterministic factor. Not every individual exposed to ECT developed PD, suggesting the presence of resilience factors. These may include supportive adult relationships, personal coping skills, or access to therapeutic resources. However, the presence of parental psychopathology emerged as a critical moderating factor in our analysis. Children raised by parents with mood or anxiety disorders had a significantly higher risk of developing PD themselves, even after accounting for trauma exposure. This supports findings by Nomura *et al.* (2007), who identified intergenerational transmission of affective disorders as a significant risk mechanism, mediated partly through parenting quality and family environment. Our results emphasize the gene-environment interaction model, where inherited predispositions interact with environmental stressors to produce complex outcomes.

Our study also revealed gender-specific patterns, with females showing higher rates of panic disorder following trauma exposure. This aligns with epidemiological trends reported by McLean and Anderson (2009), who suggested that gender differences in emotional processing, hormonal reactivity (e.g., estrogen modulation of the HPA axis), and socialization patterns may contribute to increased vulnerability in females. While our study did not aim to dissect these mechanisms in depth, the gender discrepancy reinforces the need for gender-sensitive mental health strategies, particularly in trauma-focused therapies.

Comparing our results to other longitudinal studies also offers useful contrasts. For example, the Dunedin Multidisciplinary Health and Development Study (Caspi *et al.*, 2003) linked early adversity to a broad range of internalizing disorders, but did not isolate panic disorder specifically. Similarly, the Great Smoky Mountains Study (Costello *et al.*, 2002) reported elevated rates of psychiatric symptoms among maltreated children but emphasized depression and conduct disorders. Our study fills this gap by focusing specifically on panic disorder, a condition often overshadowed by more prominent post-traumatic sequelae like PTSD.

Moreover, many earlier studies relied on cross-sectional or retrospective designs, which are susceptible to recall bias. The prospective nature of our study, involving structured diagnostic interviews and standardized trauma assessments across multiple time points, enhances its methodological robustness and supports causal inferences. This design aligns with methodological standards proposed by Pine and Klein (2015) for developmental psychopathology studies.

Despite the strengths of our study, it is necessary to acknowledge certain limitations. Although longitudinal, our findings are constrained by self-report instruments for trauma exposure, which, while validated, still depend on retrospective memory. There is also the issue of cultural specificity—our cohort was primarily based in Western populations, and trauma perception and expression of panic symptoms may differ across cultural contexts. Future research should explore these dynamics in diverse populations and consider other moderators such as religious coping, access to mental health services, and sociopolitical

factors.

Another avenue for future exploration is the role of protective interventions, including cognitive-behavioral therapy (CBT), family therapy, and trauma-informed educational practices, in altering the trajectory from trauma to PD. The emerging field of preventive psychiatry offers promising models for early intervention, such as resilience training programs in schools and routine trauma screenings in pediatric care.

In conclusion, our findings contribute robust, longitudinal evidence that early trauma—especially emotional maltreatment—has a lasting impact on adult mental health, manifesting significantly in the form of panic disorder. This reinforces the conceptualization of PD not merely as a neurochemical imbalance or acute stress reaction, but as the possible culmination of long-term developmental vulnerabilities shaped by early adversity. By integrating biological, psychological, and social perspectives, this study helps shift the understanding of panic disorder toward a more nuanced, developmental framework, emphasizing prevention, early identification, and personalized care.

## 6. Conclusion

This longitudinal study provides compelling evidence that early childhood trauma (ECT)—particularly emotional abuse and neglect—plays a significant role in the development of panic disorder (PD) in adulthood. By examining a large, representative cohort over two decades, the research confirms that adverse experiences during formative years are not only associated with increased risk but also contribute to earlier onset and more severe forms of panic-related psychopathology. The findings reinforce the growing recognition that panic disorder is not simply a reaction to isolated stressors but often represents the delayed psychological impact of developmental adversity.

The study contributes to the literature in several meaningful ways. It emphasizes the importance of distinguishing between trauma subtypes, highlighting that emotional abuse—often minimized or overlooked in clinical settings—has one of the strongest predictive values for PD. It also advances our understanding of the cumulative nature of trauma, demonstrating that individuals with multiple adverse experiences face a significantly heightened risk. Moreover, it illustrates the importance of contextual moderators such as parental mental health and socioeconomic status, suggesting that trauma does not occur in isolation but within complex ecological systems that influence long-term outcomes.

From a clinical and public health perspective, these findings underscore the urgent need for early identification of at-risk children and the implementation of trauma-informed interventions. Screening for emotional maltreatment in pediatric and school settings, integrating psychological support within primary care, and educating caregivers about the long-term effects of emotional abuse could substantially reduce the burden of anxiety disorders in the population. Mental health practitioners should consider trauma histories, especially emotional abuse and neglect, when diagnosing and treating panic disorder to develop more personalized and effective treatment plans.

Furthermore, the results advocate for a shift in the conceptualization of panic disorder—from a purely symptomatic diagnosis to one rooted in developmental history. Recognizing the etiological influence of early

adversity can inform preventative strategies and therapeutic models that not only treat symptoms but address their underlying causes.

In summary, childhood trauma—particularly in the emotional domain—casts a long shadow over adult mental health. Understanding and addressing this relationship can pave the way for more compassionate, effective, and preventive approaches to managing panic disorder, ultimately fostering resilience and recovery across the lifespan.

## Conflict of Interest

Not available

## Financial Support

Not available

## 7. References

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