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Brain-Derived Neurotrophic Factor Alterations in Completed Suicide: A Systematic Review

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Abstract: *Brain-derived neurotrophic factor (BDNF) has been implicated in the neurobiology of suicide, but findings remain inconsistent. We examined studies comparing BDNF-related measures in individuals who died by suicide and non-suicide controls. ISI Web of Science, Scopus, and Embase were searched from inception through to 31 July 2025 in accordance with PRISMA guidelines. Twenty studies met inclusion criteria. Most analysed postmortem brain tissue, while fewer investigated plasma, whole blood, or cerebrospinal fluid. Postmortem studies frequently reported reduced BDNF protein and/or mRNA expression in frontal and limbic regions. Several also described increased promoter methylation. In contrast, peripheral findings were heterogeneous, and studies of the Val66Met polymorphism did not demonstrate consistent associations with suicide. Overall, evidence supports region-specific alterations of central BDNF signalling in suicide, whereas peripheral and genetic findings remain inconclusive. Methodological variability across studies limits comparability. Larger investigations using standardised protocols are needed to clarify the role of BDNF in suicide mortality.*

Keywords: brain-derived neurotrophic factor; suicide; postmortem brain.

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1. Introduction

Globally, suicide represents a major public health concern, accounting for more than 720,000 deaths each year, ranking among the leading causes of mortality in adolescents and young adults (World Health Organization [WHO], 2025). The most frequently reported methods worldwide include hanging, jumping from a height, self-poisoning, and the use of firearms, with the distribution of each method varying substantially across regions and cultures (Ajdacic-Gross et al., 2008).

Suicide arises from the complex interaction of social, psychological, biological, and environmental factors. Epidemiological data identify psychiatric disorders, particularly depressive disorders, substance use disorders, and psychotic disorders, as major risk factors. Nonetheless, only a minority of individuals with these conditions attempt or die by suicide, suggesting that additional vulnerability factors contribute to the emergence of suicidal behaviour (Brådvik, 2018). This discrepancy between clinical diagnosis and suicide mortality highlights the need to better understand the underlying biological processes that may confer increased susceptibility.

Accumulating evidence supports the hypothesis that impairments in neuroplasticity and neuronal resilience can play a critical role in suicidal behaviour. It has also been shown that neuroimaging and postmortem studies have reported structural and cellular abnormalities in key corticolimbic regions implicated in emotion regulation, stress responsivity, and impulse control, including the prefrontal cortex, hippocampus, and amygdala. These alterations include reduced neuronal and glial density, dendritic atrophy, synaptic loss, and volumetric reductions, particularly in individuals with mood disorders and those who died by suicide. Such findings have led studies to focus on molecular systems that regulate synaptic plasticity and neuronal survival, among which neurotrophins have emerged as key candidates (Mann & Currier, 2010; Turecki & Brent, 2016).

BDNF is a member of the neurotrophin family, involved in neuronal development, synaptic plasticity, learning, and memory. The BDNF gene, located on chromosome 11p14.1, produces multiple transcript variants and is regulated by both genetic polymorphisms (notably Val66Met) and epigenetic mechanisms such as DNA methylation and histone modification. Given its fundamental role in neuroplasticity and stress adaptation, BDNF has been the focus of extensive research examining its association with suicidal behaviour, through assessments of peripheral levels, gene expression, epigenetic regulation, and the influence of BDNF polymorphisms (Huang & Reichardt, 2001).

Despite growing interest in the role of BDNF in suicide, the available literature remains fragmented and methodologically heterogeneous. Studies have examined BDNF across multiple biological matrices, including postmortem brain tissue, serum, plasma, whole blood, and cerebrospinal fluid, using diverse analytical techniques and sampling strategies. Variability in tissue type, brain region analysed, clinical characterisation, medication exposure, and laboratory methodology has contributed to inconsistent findings regarding the direction and magnitude of BDNF alterations associated with suicide. The extent to which BDNF dysregulation represents a consistent and biologically meaningful feature of suicidal behaviour remains uncertain.

The present systematic review aims to comprehensively evaluate and synthesise evidence from studies comparing BDNF expression and regulation, as well as peripheral BDNF levels in individuals who died by suicide with non-suicide controls. By integrating findings across central and peripheral tissues, including transcriptional, protein-level, genetic, and epigenetic analyses, this review seeks to clarify the regional specificity and biological relevance of BDNF alterations in suicide and to identify methodological gaps that may guide future research.

2. Materials and methods

Study Design

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Page et al., 2021). The methodology was defined beforehand to ensure transparency and reproducibility of the study selection and data extraction processes. The review protocol was prospectively registered on the Open Science Framework (OSF), with no deviations from the registered protocol made.

Search Strategy

A comprehensive literature search was performed in ISI Web of Science, Scopus, and Embase from database inception through to 31 July 2025. The search strategy used the free-text terms ("BDNF" OR "brain-derived neurotrophic factor") AND ("suicide"). The search syntax was adjusted to comply with the specific formatting and field requirements of each database. Controlled vocabulary terms (e.g., MeSH or Emtree) were not applied.

Eligibility Criteria

Studies were considered eligible if they met the following criteria: (1) original research articles published in English; (2) reported primary data on the measurement of brain-derived neurotrophic factor (BDNF); (3) assessed BDNF in postmortem brain tissue, cerebrospinal fluid, and blood (including serum, plasma, or whole blood); and (4) compared BDNF levels between adult individuals who died by suicide and adult individuals who died from non-suicidal causes. Observational study designs, including case–control, cohort, and cross-sectional studies with group comparisons, were eligible for inclusion.

Studies were excluded if they were review articles, meta-analyses, editorials, communications, brief reports, or conference abstracts; were not published in English; focused exclusively on suicidal ideation or suicide attempts without including completed suicide; did not include a non-suicide comparison group; or did not report quantitative data on BDNF levels.

Study Selection

All records identified through the database search were exported into Zotero, and duplicates were removed. A total of 1,776 records were identified through database searching. After the removal of 874 duplicates and 1 retracted article, 901 records remained for title and abstract screening.

Title and abstract screening were independently performed by two reviewers. Full-text articles were retrieved for studies that were deemed potentially eligible. A total of 33 full-text articles were assessed for eligibility, of which 20 met the inclusion criteria and were included in the final qualitative synthesis. Discrepancies between reviewers were resolved through discussion and consensus (Figure 1).

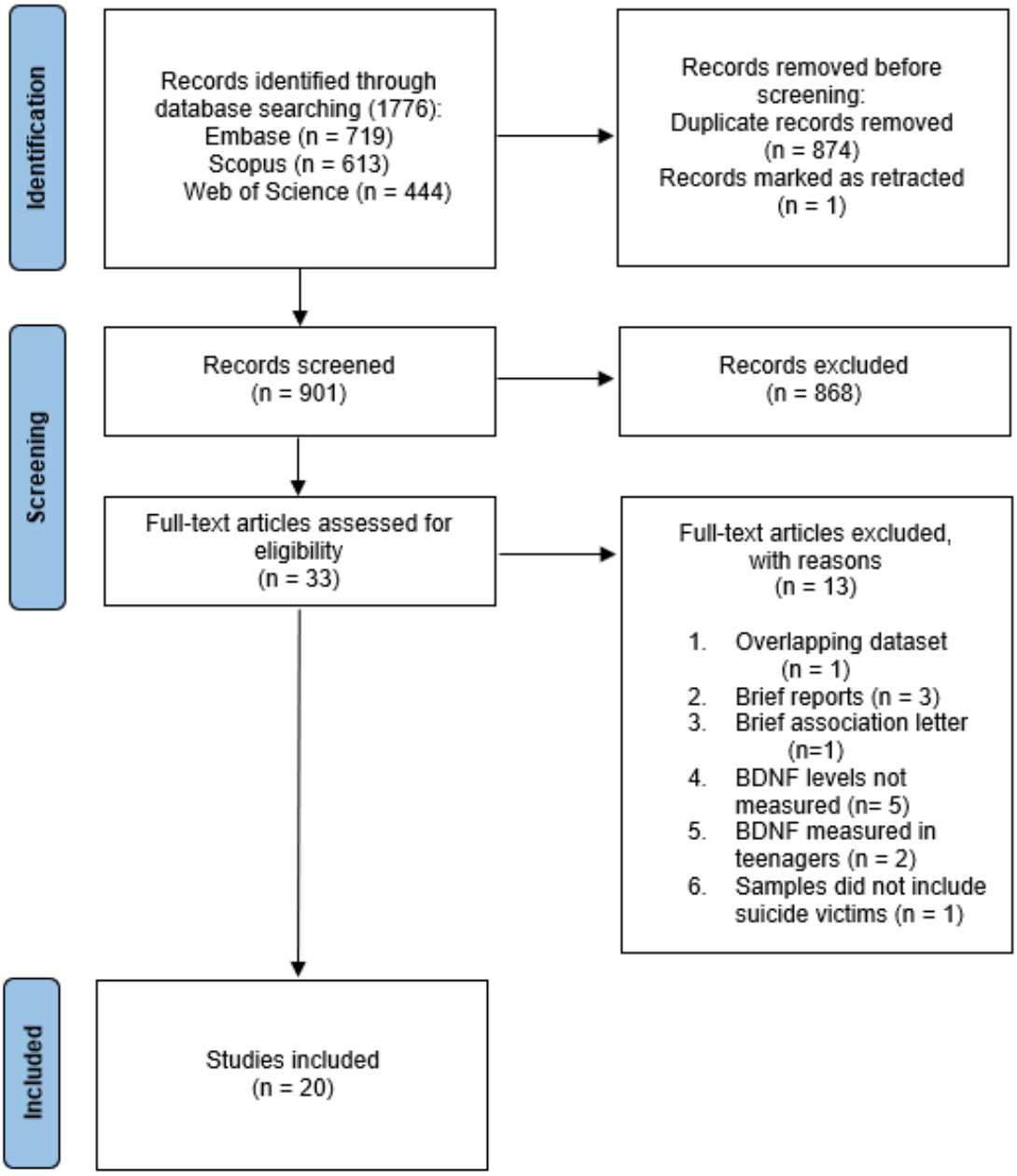

Figure 1. Search strategy following PRISMA criteria (Page et al., 2021)

Data Extraction

Data extraction was conducted independently by two reviewers using a standardised data extraction form developed specifically for this review. The following variables were collected from each included study: first author and year of publication; study design; sample size of suicide and control groups; demographic characteristics; psychiatric diagnoses, where available; method of suicide; type of biological sample analysed; brain region examined in postmortem investigations; method used for BDNF measurement; type of outcome assessed (e.g., protein levels, mRNA expression, epigenetic modifications, genetic polymorphisms); and principal findings.

Data Synthesis

Given the anticipated heterogeneity across studies in terms of biological matrices (postmortem brain tissue, cerebrospinal fluid, serum, plasma, whole blood), brain regions examined, laboratory methodologies, population characteristics, and outcome measures, findings were synthesised qualitatively, and a quantitative meta-analysis was not performed.

Studies were grouped according to the type of biological sample analysed and, where applicable, by specific brain region. Within each category, results were summarised with respect to the direction and magnitude of BDNF alterations in suicide decedents compared with non-suicide controls. Methodological differences and potential sources of variability across studies were examined to provide a structured narrative synthesis of the available evidence.

Results

This section summarises the study selection process, the characteristics of the included studies, and the principal findings across biological matrices.

Study Selection

A total of 1,776 records were identified through database searching, including 719 records from Embase, 613 from Scopus, and 444 from Web of Science. After removal of 874 duplicates and one retracted article, 901 records remained for title and abstract screening.

Following the title and abstract evaluation, 868 records were excluded. Thirty-three full-text articles were assessed for eligibility. Thirteen studies were excluded at this stage for the following reasons: one overlapping dataset, three brief reports, one brief association letter, five did not measure BDNF levels, two measured BDNF levels in teenagers, and one did not include samples from suicide victims.

Ultimately, twenty studies met the inclusion criteria and were included in the final qualitative synthesis. The study selection process is summarised in the PRISMA flow diagram (Figure 1).

Characteristics of Included Studies

The twenty included studies were published between 2003 and 2023 and were conducted across multiple countries, including the United States, Canada, India, Slovakia, Slovenia, Poland, Spain, Hungary, Switzerland, Turkey and China.

Across the included studies, the total number of suicide decedents was 1883, and the total number of non-suicide controls was 1516. Sample sizes varied considerably, ranging from 6 to 517 participants. All included studies were quantitative observational investigations measuring biological variables; no purely qualitative research designs were identified.

BDNF was assessed in multiple biological matrices. Postmortem brain tissue was analysed in fifteen studies, plasma in two studies, whole blood in five studies, and cerebrospinal fluid in one study. Some studies investigated more than one biological sample type.

In postmortem investigations, the most frequently examined brain regions included the prefrontal cortex, hippocampus, and amygdala. BDNF was assessed at the protein, transcriptional, and genetic levels using enzyme-linked immunosorbent assay (ELISA), Western blot analysis, multiplex-based Luminex assays, quantitative real-time PCR, microarray platforms, and next-generation sequencing (NGS).

A detailed overview of the characteristics of included studies is presented in Table 1.

Table 1. Characteristics of the analysed paper

Authors	Study Design	Participants	Method of suicide	PMI	Samples	Molecule targeted	Method of analysis
Banerjee (2013)	Case-control suicide individuals versus non-suicide	Suicide: 21 Control: 19	7 hangings 3 falls from height 4 poisonings 3 cuttings 2 hit by train 2 multiple traumas	<26 h	Hippocampus	BDNF protein and mRNA, TrkB	ELISA Western blot, PCR
Bednarova (2023)	Case-control suicide individuals versus non-suicide	Suicide: 119 Control: 137	71 hangings or suffocations 16 falls from height 12 hit by train 11 gunshots 5 cuttings 2 electrocutions 2 poisonings	NR	Blood	BDNF related genes	PCR
Chojnicka (2012)	Case-control suicide individuals versus non-suicide	Suicide: 517 Control: 549	NR	NR	Blood	BDNF val66Met polymorphism	PCR
Dwivedi (2003)	Case-control suicide individuals versus non-suicide	Suicide: 27 Control: 21	11 gunshots 8 overdoses 3 hangings 2 falls from height 1 asphyxiation 1 intoxication 1 multiple trauma	< 32 h	Hippocampus BA9	BDNF protein and mRNA, TrkB	PCR Western blot
Erbay (2021)	Case-control depressed suicide individuals versus non-suicide	Suicide: 61 Control: 25	61 hangings	10 h	Hippocampus	BDNF protein and mRNA, TrkB	PCR
Gadad (2021)	Case-control MD individuals versus MD with AUD-SUD versus AUD-SUD versus non-psychiatric	Suicide: 10 Control: 18	NR	<2 days	BA10 Plasma CSF	BDNF protein	Multiplex-based Luminex assay
García-Gutiérrez (2023)	Case-control suicide individuals versus non-suicide	Suicide: 28 Control: 26	22 hangings 6 falls from height	<2 days	BA9, Amygdaloid nucleus, Hippocampus	BDNF related genes	PCR
Hayley (2015)	Case-control depressed suicide individuals versus non-suicide	Suicide: 19 Control: 19	10 hangings 8 overdoses 1 fall from height	<6 h	Hippocampus PFC (BA10)	BDNF protein	Western blot
Karege (2005)	Case-control drug-free versus drug-treated depressed and non-depressed suicide individuals with other diagnoses versus. non-suicide	Suicide: 30 Control: 24	7 hangings 6 overdoses 4 falls from height 4 gunshots 3 multiple traumas 2 asphyxiations 2 drownings 2 intoxications	<2 days	Hippocampus (CA zones) PFC (BA 11) Entorhinal cortex (BA28)	BDNF protein	Western blot ELISA
Keller (2010)	Case-control suicide individuals versus non-suicide	Suicide: 44 Control: 33	NR	<26 h	Wernicke area	BDNF gene	Genotyping with PCR and RT-PCR

Kozicz (2008)	Case-control depressed suicide individuals versus non-suicide	Suicide: 10 Control: 17	9 hangings 1 overdose	<11 h	Edinger-Westphal nucleus, Ventral central grey, Oculomotor nuclei, the Cajal nucleus	BDNF mRNA	PCR
Liu (2023)	Case-control suicide individuals versus non-suicide	Suicide: 30 Control 25	11 falls from height 8 hangings 5 poisonings 4 overdoses 2 drownings 1 cutting	NR	Plasma	BDNF, TrkB, proBDNF	ELISA
Maheu (2013)	Case-control depressed suicide individuals versus depressed non-suicide versus non-depressed non-suicide	Suicide: 26 Control: 14	13 hangings 6 asphyxiation 3 falls from height 2 intoxications 2 GSW	<3 days	Basolateral amygdala, Central amygdala	BDNF protein	Western blot
Misztak (2020)	Case-control suicide individuals versus non-suicide	Suicide: 14 Control: 8	7 hangings 3 overdoses 2 falls from height 1 self-drowning 1 hit by train	NR	Hippocampus PFC	BDNF protein	Western blot
Pregelj (2011)	Case-control suicide individuals versus non-suicide	Suicide: 359 Control: 201	156 hangings 52 gunshots 43 falls from height 43 CO/medication poisonings 27 drownings 17 hit by train 16 cuttings with superficial wounds 5 self-burnings and electrifications	NR	Blood	BDNF val66Met polymorphism	PCR
Ropret (2015)	Case-control suicide individuals versus non-suicide	Suicide: 486 Control: 289	NR	NR	Blood	BDNF gene	PCR
Ropret (2021)	Case-control suicide individuals versus non-suicide	Suicide: 22 Control 20	22 hangings	NR	Hippocampus BA9 Blood	BDNF DNA methylation and mRNA	NGS and PCR
Schneider (2015)	Case-control suicide individuals versus. non-suicide	Suicide: 6 Control: 6	NR	<1 day	BA10	BDNF gene	Microarray
Youssef (2018)	Case-control depressed and non-depressed suicide individuals versus. non-suicide	Suicide: 37 Control: 53	NR	<1 day	BA9, BA24, Rostral and Caudal Brainstem	BDNF val66Met polymorphism & BDNF protein	PCR Western blot
Zhao (2015)	Case-control depressed suicide versus depressed and non-depressed non-suicide	Suicide: 17 Control 12	NR	NR	BA9 BA24	BDNF and TrkB related genes	PCR

PMI: Postmortem interval; NR: not reported; BA: Brodman area; PFC: prefrontal cortex; NGS: next-generation sequencing; CSF: cerebrospinal fluid

Postmortem Brain Studies

Among the included studies, fifteen investigated BDNF expression or protein levels in postmortem brain tissue of suicide decedents compared with non-suicide controls.

Most postmortem studies reported decreased BDNF mRNA or protein expression in corticolimbic regions, particularly in the prefrontal cortex and hippocampus. Several studies also identified alterations in BDNF receptor expression and downstream signalling pathways. Epigenetic analyses conducted in four studies demonstrated increased methylation of BDNF promoter regions in cortical tissue from suicide decedents (Keller et al., 2010; Misztak et al., 2020; Schneider et al., 2015; Ropret et al., 2021).

Peripheral Blood Studies

A total of seven studies assessed BDNF levels in blood-derived samples, including plasma or whole blood.

Findings were heterogeneous. Several studies reported reduced peripheral BDNF concentrations in suicide decedents compared with non-suicide controls, whereas others found no statistically significant differences between groups. Differences in assay methodology, postmortem interval, sample handling procedures, and comorbid psychiatric diagnoses may have contributed to variability across studies.

Cerebrospinal Fluid Studies

BDNF levels in cerebrospinal fluid were evaluated in one study. Reported findings indicated no statistically significant differences in CSF BDNF concentrations between individuals who died by suicide and non-suicide controls.

4. Discussion

This systematic review integrates findings from twenty observational studies examining BDNF-related alterations in individuals who died by suicide. Across molecular levels, the most consistent pattern emerged from postmortem investigations of corticolimbic brain regions, where reduced BDNF protein and/or mRNA expression was frequently reported. In contrast, peripheral blood and genetic association studies yielded varying and often inconclusive results.

The convergence of postmortem findings across independent cohorts strengthens the hypothesis that impaired neurotrophic support within prefrontal and limbic circuits may contribute to suicide vulnerability. Several studies reported reduced BDNF expression in the prefrontal cortex and hippocampus, regions involved in emotional regulation and stress processing (Dwivedi et al., 2003; Karege et al., 2005). Epigenetic investigations further reported increased BDNF promoter methylation in cortical tissue from suicide decedents (Keller et al., 2010; Misztak et al., 2020; Schneider et al., 2015; Ropret et al., 2021), suggesting a potential transcriptional mechanism underlying reduced expression. Together, these findings indicate that central BDNF dysregulation may represent a biologically meaningful component of suicide-related neuropathology.

Nevertheless, the evidence is not uniform. Some studies identified region-specific alterations rather than global reductions, and diagnosis-dependent differences were reported in certain cohorts (Maheu et al., 2013; Zhao et al., 2015; Youssef et al., 2018). These variations suggest that BDNF changes may depend on various variables, including, but not limited to, clinical context, comorbid psychiatric disorders, or specific brain regions examined. Moreover, mRNA and protein findings were not always concordant, underscoring the complexity of post-transcriptional regulation and molecular measurement.

Genetic studies focusing on the Val66Met polymorphism did not demonstrate consistent associations with suicide. While isolated reports suggested genotype-related effects (Pregelj et al., 2011), others found no significant differences between groups (Youssef et al., 2018). Existing

meta-analytic data indicate that Val66Met may confer modest and context-dependent risk (Verhagen et al., 2010), but its contribution to suicide appears limited when considered in isolation.

Peripheral investigations were comparatively inconsistent. Plasma and whole-blood BDNF concentrations did not show a uniform direction of change, although alterations in proBDNF or BDNF/proBDNF ratios were noted (Liu et al., 2023). Peripheral BDNF levels can be influenced by platelet storage, systemic inflammation, and physiological variability (Karege et al., 2002; Sen, Duman, & Sanacora, 2008), which may limit their utility as direct proxies of central neurotrophic activity. Consequently, current data do not support peripheral BDNF as a reliable biomarker of suicide mortality.

Methodological heterogeneity likely contributed to variability across studies. Differences in sample size, psychiatric characterisation, postmortem interval, tissue pH, medication exposure, and laboratory techniques complicate cross-study comparisons (Li et al., 2004). In addition, the diversity of biological matrices and molecular endpoints precluded quantitative meta-analysis, limiting the ability to estimate pooled effect sizes. These factors should be considered when interpreting inconsistencies.

Taken together, the most robust evidence from this review supports altered BDNF regulation within specific corticolimbic regions of suicide decedents. In contrast, genetic associations and peripheral measures remain uncertain and require further clarification. BDNF alterations are unlikely to function as a standalone biomarker but may represent one component within a broader network of neurobiological processes involved in suicide vulnerability.

Future investigations would benefit from larger, well-characterised cohorts, standardised postmortem protocols, and harmonised laboratory methodologies. Integrative multi-omic approaches combining transcriptomic, epigenetic, proteomic, and genetic data may help clarify pathway-level dysregulation. Clarifying whether BDNF alterations reflect trait vulnerability, state-dependent changes, or downstream consequences of psychiatric illness remains an important objective for future research.

5. Conclusions

This systematic review synthesises evidence from postmortem and peripheral investigations examining BDNF alterations in individuals who died by suicide. The most consistent findings emerged from postmortem brain studies, which frequently reported reduced BDNF protein and/or mRNA expression in corticolimbic regions. Epigenetic investigations further suggested altered promoter methylation and transcriptional regulation. In contrast, peripheral blood and genetic association studies yielded heterogeneous and less consistent results.

Collectively, these findings support the hypothesis that central BDNF dysregulation may represent one component of the neurobiological vulnerability underlying suicide, although current evidence does not support BDNF as a standalone biomarker of suicide.

Several limitations should be acknowledged. The included studies were methodologically heterogeneous with respect to sample characteristics, psychiatric diagnoses, postmortem variables, and laboratory techniques. Sample sizes were often modest, and formal quantitative meta-analysis was not feasible due to variability in outcome measures and biological matrices. Additionally, the review relied on published studies, which may introduce reporting bias.

Future research should prioritise larger, well-characterised cohorts, standardised postmortem protocols, and harmonised laboratory methodologies. Integrative multi-omic approaches combining transcriptomic, epigenomic, proteomic, and genetic data may clarify pathway-level dysregulation. Longitudinal and translational investigations are needed to determine whether BDNF alterations reflect trait vulnerability, state-dependent changes, or downstream consequences of psychiatric illness. Such efforts may help refine biological models of suicide and inform targeted preventive strategies.

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