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REVIEW

Epigenetics and immunology: Under-recognized aspects of suicidality

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Abstract

Suicidality is a complex phenomenon influenced by genetic, environmental, and epigenetic factors. Current tools to estimate suicide risk are insufficient, and there is an increasing need for reliable biomarkers to complement clinical approaches. Growing evidence suggests that immune system dysregulation contributes to the pathophysiology of psychiatric disorders and suicidal behavior. Epigenetic mechanisms, including DNA methylation, histone modifications, and non-coding RNAs, regulate gene expression and may act as a bridge between environmental stressors and (neuro)inflammatory responses. In this review, we examine the evidence of peripheral and central inflammation in suicide completers and individuals with suicidal behavior. Next, we review current knowledge from various studies on suicide-associated epigenetic alterations. Furthermore, we evaluate the mechanisms by which early life adversity and chronic stress contribute to suicide diathesis, focusing on their association with epigenetic modifications and inflammatory pathways. We also examine future prospects and limitations of immunology-related biomarkers and the possibilities of therapeutic interventions targeting the immune system and epigenetic regulation. While challenging, research on epigenetic and immune alterations in suicidality shows promise for identifying suicide risk subtypes and advancing personalized psychiatry.

Key Words: Suicide; Suicidal behavior; Inflammation; DNA methylation; Histone tail modification; Non-coding RNA; Multi-omics; Adverse childhood experiences

Core Tip: Suicidality is heterogeneous and cannot be fully accounted for by psychiatric comorbidities. To understand its biological background, it is important to consider epigenetic regulation as a mediator of suicide risk in response to environmental stimuli, particularly early life adversity. Chronic inflammation has been implicated in neuropsychiatric disorders, and further research should examine whether suicidality is associated with a specific inflammatory profile. The interactions between immune system dysregulation, stress response, and epigenetics hold promise for biomarker discovery but are insufficiently explored. To accelerate the development of targeted suicide prevention strategies, stringent sample selection and integrative multi-omics approaches should be applied.

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INTRODUCTION

Suicide is a major global public health challenge, accounting for more than 720000 deaths each year[1]. Suicide attempts may be up to 20 times more frequent and present a major risk factor for suicide[2]. Suicidal behavior, which includes preparatory behaviors, suicide attempts, and completed suicide, is a complex and poorly understood phenomenon[2]. While commonly associated with an underlying psychiatric disorder, most notably major depressive disorder (MDD), bipolar disorder, substance use disorder, and schizophrenia[3,4], approximately 10% of suicide completers do not have a psychiatric diagnosis at the time of their death[2]. Additionally, few psychiatric patients die due to suicide, making psychiatric diagnosis a poor predictor of suicide[5].

The stress-diathesis model describes suicide as a result of an interaction between environmental stressors and a susceptibility (diathesis), independent of psychiatric disorders[6]. While many molecular and physiological changes have been linked to suicidality, including inflammation, glial dysfunction and disruptions in neuroplasticity, neurotransmission, the hypothalamic-pituitary-adrenal (HPA) axis, and neurotrophic signaling, the biological correlates of this diathesis are still not fully understood[7]. Figure 1 summarizes environmental, genetic, and epigenetic influences on the pathophysiological changes associated with suicide.

Suicidal behavior exerts a heavy emotional and financial toll on society. However, suicide prevention efforts remain inadequate[8]. Various factors, including stigma, insufficient prediction power of clinical assessments, as well as the lack of reliable biomarkers of suicide risk, present challenges in implementing effective interventions[3,9]. Furthermore, suicidality research faces many obstacles, including the translational limitations of animal models[10], unavailability of *in vivo* brain sampling in humans, the high prevalence of psychiatric comorbidities accompanying suicidality, and limited access to patients' past medical and personal history, which contributes to variabilities in study designs[11]. Nevertheless, technological advancements have made it possible to investigate various aspects of the neurobiological background of suicidal behavior, which may provide tools for improving suicide prevention practices[2].

While computationally demanding, the multi-omics approach has enabled simultaneous investigation of multiple potential biomarkers[12,13]. Promising targets have emerged in immune response and inflammation pathways, both peripherally and centrally[13]. In many cases, the mechanistic roles of putative biomarkers remain poorly understood[9]. The rapidly growing research on epigenetics has great potential to advance our understanding of suicidality, as it may provide a link between genetic predisposition and environmental influences[4]. While rarely examined in combination, the intersection of epigenetic regulation and immune system dysfunction may represent a critical dimension of suicidality that warrants further study. Epigenetic modifications may regulate immune system responses, while dysregulated immune responses may feedback to alter epigenetic marks, influencing suicide diathesis.

In this review, we critically examine current research on epigenetic and immune alterations in suicidality, covering preclinical and clinical studies. We aimed to highlight the evidence for the interplay between epigenetic modifications and (neuro)inflammation. Furthermore, we assessed the relevance for biomarker discovery and future therapeutic implementation, while also describing the pitfalls of past research and providing recommendations for future research.

Genetic background of suicidal behavior

Assessing an individual's suicide risk is challenging, as suicidal behavior is etiologically heterogeneous and is determined by biological characteristics, comorbid psychiatric diagnoses, personality traits, as well as economic, sociodemographic, and environmental influences[4]. Various models explaining suicidal behavior commonly classify risk factors into distal and proximal and, in some cases, developmental[4]. Distal factors, such as family history, early life adversity (ELA), and genotype, are thought to increase predisposition; proximal factors, which include recent life events, current psychiatric disorders, substance use, and the availability of means, are viewed as precipitants or triggers[7].

The genetic contribution to suicidality was first suggested by epidemiologic studies of twins, families[14], and adoptees [15]. Many studies associated the familial aggregation of impulsive-aggressive personality traits with an increased suicide risk[16,17]. The heritability of suicidal behavior was estimated at approximately 40%, independent of familial clustering of psychiatric disorders[18]. Early genetic research into suicide neurobiology utilized candidate gene associations that targeted neurotransmitter systems, most notably the serotonergic and, to a lesser extent, the dopaminergic and

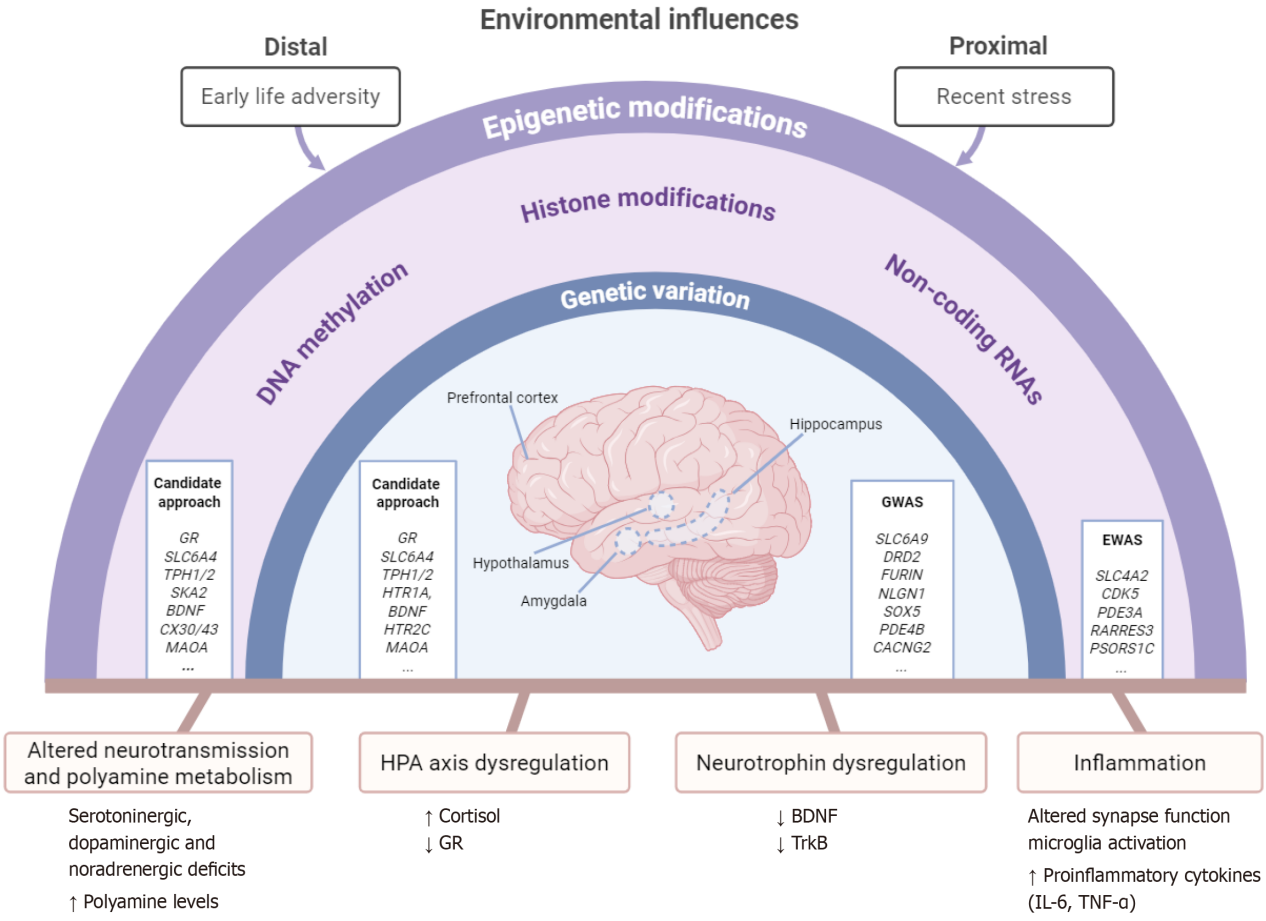

Figure 1 Genetic, epigenetic and environmental factors influence the pathophysiology of suicidality. Brain areas most commonly examined in postmortem suicidality research are labeled in the center of the image. Some of the genes associated with suicidality *via* targeted and (epi)genome-wide approaches are listed in the boxes to the left and right of the center. In the upper part of the image, environmental influences that act distally or proximally to alter suicide risk are represented. The interaction between the environment, genetic predisposition and epigenetic mechanisms drives the development of molecular and functional alterations associated with suicidality, including the disruption of key neurobiological processes like neurotransmission, neurotrophin signaling, stress and inflammatory responses (presented in the bottom of the image). GWAS: Genome-wide association study; EWAS: Epigenome-wide association study; HPA: Hypothalamic-pituitary-adrenal; GR: Glucocorticoid receptor; TrkB: Tropomyosin receptor kinase B; BDNF: Brain-derived neurotrophic factor; IL-6: Interleukin 6; TNF-α: Tumor necrosis factor alpha; SLC6A4: Solute carrier family 6 member 4; SLC4A2: Solute carrier family 4 member 2; SLC6A9: Solute carrier family 6 member 9; TPH1/2: Tryptophan hydroxylase 1/2; SKA2: Spindle and kinetochore associated complex subunit 2; CX30/43: Connexin 30/43; MAOA: Monoamine oxidase A; HTR1A: Serotonin 1A receptor; HTR2C: Serotonin 2C receptor; DRD2: Dopamine receptor 2; FURIN: Paired basic amino acid cleaving enzyme; NLGN1: Neuroligin-1; SOX5: SRY-box transcription factor 5; PDE4B: Phosphodiesterase 4B; CACNG2: Calcium voltage-gated channel auxiliary subunit gamma 2; CDK5: Cyclin-dependent kinase 5; PDE3A: Phosphodiesterase 3A; RARRES3: Retinoic acid receptor responder 3; PSORS1C: Psoriasis susceptibility 1 candidate 1. Created in BioRender. Šmon J. (2025) https://BioRender.com/kzls5et.

noradrenergic systems. While these studies identified variations in tryptophan hydroxylase 1 and serotonin transporter gene[19,20] as contributors to suicide risk, it was clear that suicidality is a polygenic condition, and this approach proved insufficient in determining its etiology.

The development of high-throughput genomic technologies led to the emergence of an unbiased approach[21]. With the ability to simultaneously examine numerous genetic variants in large cohorts, many genome-wide association studies (GWAS) have identified novel loci associated with suicide risk[22-24], further supporting the estimated heritability of suicidality[2]. These loci are often intergenic, associated with inflammatory pathways and the immune response, as well as with cell interactions, neurodevelopment, and synaptic plasticity[25]. While glucocorticoids, whose levels are regulated by the HPA axis, exert immunomodulatory functions in peripheral tissues and in the brain, the target candidate approach rarely focused directly on immunology-related targets[26]. GWAS results thus provided a new perspective on the role of immunity in neuropsychiatric disorders, suggesting that the genetic variability in immune and inflammatory pathways may present a vulnerability factor for suicidality[27].

The largest GWAS meta-analysis to date comprised 22 cohorts of various ancestries and connected 12 risk loci to suicidal behavior[28]. These loci were brain-enriched and overlapped with antidepressant targets[28]. However, due to modest loci effect size, the polygenic risk scores obtained by GWAS currently have low predictive power, and therefore lack clinical utility[29]. Furthermore, suicide and/or suicidal behavior were rarely examined independent of one another or independent of psychiatric disorders, which may have contributed to the scarcity of significant findings and inconsistent results of these studies[25,30,31]. While the genetics of suicide death and suicide attempt appear to strongly overlap, a locus mapped to neuroligin-1, involved in synapse function, may be specific to suicide[32]. Expanding GWAS

study results with other modalities, such as measuring expression of genes surrounding the loci of interest, may prove beneficial in elucidating the functional role of these variants in suicidality[33]. Regarding the immune alterations in suicidality, combining GWAS with epigenetic approaches, such as genome-wide DNA methylation studies[34], is likely to provide insight into the mechanisms by which environmental exposures, including ELA, exert their effects on immune and stress-response genes[35]. Findings on the heritability of suicidal behavior underscore the need to further investigate the immunological dimension of suicidality, including the differences that may distinguish it from various neuropsychiatric conditions[36].

SUICIDALITY AND IMMUNE SYSTEM DYSREGULATION

Association between inflammation and depression

As implied by extensive evidence from genetic and epigenetic studies, many neuropsychological disorders, including depression and suicidality, are connected to alterations in the innate and adaptive immune systems, both peripherally and centrally[37-40]. Recently, a bidirectional relationship has been proposed between inflammation and depression in which both conditions exacerbate one another[41]. The ability of inflammation to trigger depressive symptoms was already identified in the 1990s through observations of patients receiving immunotherapy. As an adverse effect of interferon (IFN) treatment, up to 30%-70% increased risk of depression was observed[42], which did not always subside with treatment discontinuation. The effect of IFN on mood disorders was linked to its stimulation of pro-inflammatory cytokine production, with subsequent tryptophan depletion, excess reactive oxygen species formation and HPA axis dysregulation[43]. Various genetic variants were associated with IFN treatment-induced depression, including variants in genes coding for tryptophan-degrading enzyme, serotonin receptor[44], and glucocorticoid receptor (GR)[45]. The incidence of comorbid depression is elevated in patients with autoimmune disorders and other diseases associated with chronic inflammation[46], and increased CD4+/CD8+ T cell ratio was linked to depression[47]. However, increased anti-inflammatory processes have also been reported[40]. These findings led to further inquiries about the relationship between depression and inflammation, including the effects of cytokines that are specific for the central nervous system (CNS). It has been proposed that ELA and psychosocial stress may induce inflammatory responses, which alter microglial function and synaptic remodeling, thus influencing the predisposition to suicidal behavior[37].

Some studies suggest that the relationship between inflammation and depression is sex-specific[48], and the association between depression and elevated inflammatory marker levels seems to be stronger in females than males[49]. However, with the majority of postmortem suicidality research focusing solely on male subjects, these differences are likely underexplored. While women are twice as likely to develop depression compared to men[50] and more frequently display suicidal ideation (SI) and attempt suicide, men more often die due to suicide[51]. As early as neonatal development, males and females show differential expression of estrogen receptor alpha in sexually dimorphic brain regions, which is linked to epigenetic regulation (most notably, DNA methylation patterns in gene promoters). To study sex differences in depression susceptibility, mouse models have been developed that show sex-specific responses to stress. Reducing DNA methyltransferase 3A levels, whose overexpression is linked to stress, in the nucleus accumbens of female mice made them resilient to depression-like behavior, and their gene expression profile became more similar to their male counterparts[52]. In the prefrontal cortex of MDD patients, the long non-coding (lncRNA) RNA FEDORA, which can promote depression-like behaviors in female mice, was upregulated in women but not in men[53]. Increased testosterone levels have been implicated in suicide risk in both men and women, and sex hormones have an immune-modulatory role, with estrogen possessing both anti-inflammatory and pro-inflammatory properties, and testosterone exerting mainly an anti-inflammatory effect by inhibiting the expression of pro-inflammatory interleukins[54]. In depressed men, inflammation has been linked to more pronounced structural brain changes than in women[55]. While there is a lack of epigenetic studies in humans that have specifically examined sex differences in suicidality in association with inflammatory profiles, a recent postmortem study identified sex-specific gene expression patterns in suicide completers, with alterations in immune response - related gene expression being more pronounced in women than in men[56].

Cytokines and suicidality

Cytokines are small signaling proteins that are involved in cell-cell communication. They are produced and secreted by different cell types but are particularly important for immune cells, which use cytokines as messenger molecules. There are over 300 different cytokines, which are divided into chemokines, lymphokines, IFNs and growth factors. They can have pro-inflammatory or anti-inflammatory effects, and their action can promote or inhibit inflammatory reactions. Even very low concentrations of cytokines can have strong effects[57]. They act as messengers over shorter distances by binding to a cell surface receptor, which triggers signaling pathway cascades within the cell. A single type of cytokine can bind to different receptors, and different types of cytokines can also bind to the same type of receptor. When the same type of cytokine binds to different types of receptors, each binding event can lead to a different effect depending on the receptor type. Cytokine function is therefore complex and contributes to the maintenance of homeostasis[58].

In the context of neuropsychiatry, various brain regions, including the hypothalamus, hippocampus and amygdala, both react to and produce cytokines[37], which can also act as messengers between the peripheral and central immune systems[59]. The brain is separated from the peripheral immune system by the blood-brain barrier (BBB), which normally prevents peripheral immune cells from entering the brain under physiological conditions. Additional neuroimmune response mechanisms have evolved in the brain that are based on glial cells and have partially taken over the role of immune cells in the brain. It is not yet fully understood to what extent cytokines can enter the brain from other parts of

the body, but there are known examples of cytokines that can cross the BBB (either due to increased BBB permeability or specialized transporters)[60].

Impaired cytokine function is associated with several mental and neurological disorders such as depression, bipolar disorder, schizophrenia and Alzheimer's disease, as well as suicidality[61]. Polymorphisms in interleukin genes are associated with completed suicide[62]. Notably, increased levels of pro-inflammatory cytokines, especially tumor necrosis factor α (TNF-α) and interleukin-6 (IL-6), have been consistently linked to depression in human studies[4,63,64]. Regarding suicidality, the inflammatory markers most strongly associated with increased risk are IL-6 and C-reactive protein (CRP), which were both elevated in the plasma of suicide attempters[65]. Plasma IL-6 is also associated with increased impulsivity and aggression, which are behavioral risk factors for suicidality[66].

Inflammatory cytokines are released by innate immune cells, including macrophages and microglia, typically in response to the binding of pathogen-associated molecules (*e.g.*, lipopolysaccharide) to toll-like receptors (TLRs) and subsequent activation of the nuclear factor kappa B pathway[67]. IL-1β, a key cytokine that stimulates the production of other pro-inflammatory cytokines, also requires activation by the inflammasome, which is induced by extracellular ATP accumulation, tissue damage[37], and accumulation of protein aggregates[68]. Cytokines are also associated with neurotransmitter function, as they affect neurotransmitter synthesis, release and reuptake, thereby significantly shaping communication pathways in the brain. They are involved in the regulation of dopamine and glutamate[69]. Excessive cytokine signaling in the brain promotes corticotropin-releasing hormone (CRH) release and may have deleterious effects on neurogenesis, memory formation, circadian rhythms[37] and brain-derived neurotrophic factor (BDNF) signaling[70]. Furthermore, cytokine-induced increase in indoleamine 2,3-dioxygenase activity shifts tryptophan metabolism from serotonin production towards the kynurenine pathway, which increases the levels of the neurotoxin quinolinic acid and lowers serotonin levels[69,71]. Alterations in kyreunine metabolism in cerebrospinal fluid (CSF) of *post-mortem* brains and plasma from living subjects have been associated with depression and suicide[72-74].

Glial dysfunction and suicidality

Morphological and functional alterations in glial cells have also been implicated in suicidality[37]. Glial and neuronal cells can produce cytokines and they also have receptors on their surfaces to which cytokines can bind[75]. Aside from their role in immunity, microglia contribute to the regulation of BBB, neurodevelopment and synapse remodeling, and are therefore involved in the processes of learning and memory[76]. Upon activation, microglia alter their shape and start releasing inflammatory mediators, which can be either pro- or anti-inflammatory[77]. Peripheral inflammation can trigger neuroinflammation *via* HPA axis activation or T-cell effects on glia[78]. Serum markers of neuroinflammation, including neuron specific enolase and glial fibrillary acidic protein, have been associated with suicidality[79]. Some studies have documented increased levels of activated microglia in *post-mortem* brain of depressed suicide completers compared to controls[80,81].

Evidence for immune alterations in suicidality from biomarker analyses

Studies investigating genetic predisposition to cytokine-related genes are scarce. Kaushik *et al*[82] analyzed five single nucleotide polymorphisms (SNPs) in the *IL-1β*, *IL-4*, *IL-6* and *IL-10* genes in a study group of Indian suicide completers and controls (n = 490). Significant differences between the two groups were observed for genetic variation in *IL-1β* (rs16944), *IL-4* (rs2070874), and *IL-6* (rs1800795)[82]. Noroozi *et al*[62] genotyped three SNPs within the *IL-8* gene, which codes for an interleukin with neuroprotective function, in individuals who attempted suicide, suicide completers, and healthy controls. The T allele of rs4073 was significantly associated with suicide attempts, and specific *IL-8* haplotypes showed distinct associations with method and severity of suicidal behavior[62]. Focusing on gene expression, Rengasamy *et al*[83] aimed to differentiate between suicide attempters, suicide ideators, and healthy controls based on cytokine gene expression. While they found a significant association between the severity of depression and expression of IL-10 and TNF-α, such results were not identified for the severity of SI[83].

A larger amount of data is provided by studies that examined cytokines as biomarker molecules, accessible in both living and *post-mortem* samples (Table 1)[84-93]. Most of the studies described in Table 1 use a targeted approach, examining selected cytokines previously associated with neuroinflammation and mental/neurological disorders. As blood is a relatively accessible tissue, the studies predominantly investigated cytokine concentration in peripheral blood as the sample of choice. Only a few studies use CSF, which is a more invasive procedure for the patient. Brain tissue is only available for *post-mortem* studies. Suicidality is a complex phenomenon often associated with other mental disorders, which also have an impact on study participants. Table 1 shows that the groups studied are quite heterogeneous and exhibit various mental disorders in combination with suicidality. A distinction must be made for the term suicidality, as its meaning can range from SI, behavior, attempt to completed suicide. With such complex behaviors, we can also expect differences in the time course of the behavior, which could also likely affect the temporal course of the cytokines present and measured. This may represent one of the possibilities for study results that are sometimes difficult to compare. Examples of such cytokines are the pro-inflammatory IL-2 and the anti-inflammatory IL-10. Studies of both cytokines show mixed results, with some researchers describing decreased levels[84-86] and others reporting increased levels[87,88]. For example, Tian *et al*[87] observed increased IL-2 Levels, while Yang *et al*[84] and Hoprekstad *et al*[85] observed decreased IL-2 Levels. Looking more closely, multiple differences between studies can be observed. Tian *et al*[87] examined first-episode drug-naïve MDD patients, while Hoprekstad *et al*[85] specifically observed schizophrenia spectrum disorder patients on antipsychotic therapy. Differences can also be observed in the severity or definition of suicidality of the studied patients: SI in the past week, Tian *et al*[87] *vs* low suicide risk - mild suicide risk - moderate suicide risk - severe suicide risk, Yang *et al*[84] *vs* low - high risk, Hoprekstad *et al*[85].

Table 1 Overview of recently published studies examining cytokines in various disease states involving or associated with suicidality

Studied biomarker	Tissue	Subjects	Number	Method	Main findings	Ref.
IL-1β, IL-2, IL-6, IL-8, IL-10, IFN-γ, TNF-α, CRP, CXCL-1, CCL2	Blood	MDD patients and healthy controls	*n* = 260	ELISA	Association between suicide risk and increased levels of IL-6, CRP, TNF-α, CXCL-2, and IFN-γ. Association between suicide risk and decreased levels of IL-2 and IL-8	Yang *et al*[84], 2024
CRP, TNF-α, IL-2, IL-6	Blood	MDD patients with severe or mild suicidal symptoms, healthy controls	*n* = 179	ELISA	Association between severe suicidal symptoms and increased levels of CRP and TNF-α	Chen *et al*[90], 2024
IL-1β, IL-2, IL-6, IL-8, IL-10, TNF-α	Blood	First-episode drug-naïve MDD patients: With suicidal ideation and without suicidal ideation	*n* = 88	Luminex	Association between suicidal ideation and increased levels of IL-2	Tian *et al*[87], 2024
IL-1β, IL-2, IL-4, IL-6, IL-10, IFN-γ, TNF-α	Blood	Schizophrenia spectrum disorder patients: Suicidality low risk and suicidality low risk	*n* = 141	Luminex	Association between suicidality and decreased levels of IL-2 and IL-10	Hoprekstad *et al*[85], 2024
TNF-α, IL-1β, IL-6	Blood	BD patients with or without suicide attempt, healthy controls	*n* = 64	Luminex	Association between suicidal behavior and increased levels of IL-6	Jiang *et al*[91], 2022
IL-1β, IL-2, IL-4, IL-6, IL-8, IL-10, IL-12p70, IL-13, IFN-γ, TNF-α	Blood	Mood and anxiety disorders patients	*n* = 66	V-PLEX	Association between suicidal risk and increased levels of IL-6. Association between suicidal risk and decreased levels of IL-8	Keaton *et al* [92], 2019
IL-1β, IL-2, IL-4, IL-5, IL-6, IL-8, IL-10, IL-12P70, IL-17 A, IL-17 F, IL-22, TNF-α, TNF-β, IFN-γ	Blood	Depression disorder patients with or without suicidal behavior	*n* = 82	Flow cytometry	Association between suicidal ideation and increased levels of TNF-α	Liu *et al*[89], 2024
40 chemokines (Bio-Plex Pro™ Human Chemokine 40-Plex Panel)	Brain (dorsolateral prefrontal cortex)	Suicide completers and control	*n* = 39	Bio-Plex	Association between completed suicide and decreased levels of CCL1, CCL8, CCL13, CCL15, CCL17, CCL19, CCL20, CXCL11, and IL-10. Association between completed suicide and increased levels of IL-16	Shinko *et al* [86], 2020
IL-1β, IL-2, IL-4, IL-6, IL-8, IL-10, IL-13, TNF-α VEGF	Blood, CSF	Suicide death subjects and non-suicidal death subjects	*n* = 65	Luminex	Association between suicide and increased levels of plasma IL-6 and IL-10. Association between suicide and decreased levels of CSF VEGF	Kaushik *et al* [88], 2025
IL-1β, IL-6, IL-8, TNF-α	CSF	Suicide attempters and healthy control subjects	*n* = 110	ELISA	Association between suicide attempt/Level of suicide violence method and increased levels of IL-6	Lindqvist *et al* [93], 2009

BD: Bipolar disorder; CCL: Chemokine C-C motif ligand; CRP: C-reactive protein; CSF: Cerebrospinal fluid; CXCL: Chemokine (C-X-C motif) ligand; ELISA: Enzyme-linked immunosorbent assay; IFN: Interferon gamma; IL: Interleukin; MDD: Major depressive disorder; TNF: Tumor necrosis factor; VEGF: Vascular endothelial growth factor.

Looking more broadly, the occurrence and representation of cytokines can also be influenced by pharmacological treatment. Studies suggest that antidepressant use can reduce cytokine levels, while some antidepressants (such as mirtazapine) increase cytokine production[94]. Cytokine expression is also controlled by the biological circadian rhythm - this can be observed by disease severity or symptoms in some chronic inflammatory diseases. This indicates that the time of day when the sample is taken may potentially affect cytokine levels[95]. In addition, numerous environmental, socio-psychological and biological factors are also associated with changes in cytokines. All of these factors further increase the non-specificity of differences in cytokines across subjects[94].

Nevertheless, what does appear to be frequently observed are increased levels of pro-inflammatory cytokines such as IL-6, CRP and TNF. IL-6, which shows the most conclusive results (Table 1), is a major mediator of the acute phase reaction and acts directly on cells to synthesize CRP, complement components, and other acute phase proteins. Excessive IL-6 activation has also been observed in other chronic inflammatory diseases[96]. A disrupted balance between pro- and anti-inflammatory cytokines influences HPA dysregulation, as cytokines can affect the hypothalamus, stimulating the HPA, releasing CRH, adrenocorticotropic hormone and glucocorticoids[89]. Additionally, cytokines affect neurotransmission, altering the levels of both neurotransporters and neurotransmitters[97], and dysregulate neurogenesis[98].

As suicidality studies *per se* are relatively scarce, it is worth examining studies on depression, as they are repeatedly studied together. Coexistence of MDD and systemic immune activation (changes in inflammatory markers levels,

immune cell count and antibody titers) has already been shown in several studies. Köhler *et al*[64] performed the most extensive meta-analysis of 82 studies on depression, cytokine and chemokine levels, and determined increased levels of IL-6, TNF, IL-10, sIL-2, C-C motif ligand (CCL) 2, IL-13, IL-18, IL-12, IL-1 receptor antagonist, and soluble TNF receptor 2, and decreased levels of IFN-γ in MDD patients compared to healthy controls. When studying the effect of antidepressant treatment on base levels of inflammatory biomarkers, it was found that antidepressants can decrease IL-6, IL-10, TNF-α and CCL2 Levels[99,100]. While these findings hold implications for future therapeutic opportunities, deeper investigation into the regulatory mechanisms of (neuro)inflammation in depression and suicide is needed.

EPIGENETIC MECHANISMS IN SUICIDALITY

As genetic characterization could not provide a comprehensive understanding of the neurobiological basis for suicide diathesis, including the contribution of immunity and inflammation towards suicide risk, research has expanded towards other molecular factors, including epigenetic processes[2]. Epigenetic alterations are defined as heritable DNA modifications that regulate gene function and do not involve nucleotide sequence changes[101]. Environmental signals, including social environment, can trigger epigenetic responses, which include DNA methylation, histone modifications and non-coding RNA (ncRNA)-mediated changes of gene activity[101]. In suicidality, epigenetic modulation has been proposed to mediate the connection between genetic predisposition and environmental influences, most notably ELA[35], and may be involved both in distal (stable epigenetic modifications) and proximal (dynamic epigenetic modifications) factors[34]. While epigenetic changes may dysregulate immune system function, inflammation can in turn induce further epigenetic alterations, influencing the vulnerability to psychiatric disorders. For example, increased gastrointestinal permeability and subsequent alterations in microbiota-derived metabolites with immunomodulatory functions may influence brain neurogenesis and contribute to depression through epigenetic modifications[102]. Figure 2 represents the proposed interplay between different types of epigenetic alterations and the immune system, with its consequent contribution to suicide risk.

DNA methylation

Most epigenetic studies in suicidality have focused on DNA methylation, which is usually viewed as a stable epigenetic mark and thus reflective of long-term effects of environmental influences[34]. To date, various studies have been conducted on *post-mortem* brain tissue or blood, utilizing both candidate gene and -omics approaches[103-112], and in some cases multi-omics[113]. Common candidate genes include those involved in the HPA axis, neurotransmission and neurotrophin signaling[34]. In a study that did not account for the subjects' history of ELA, differences in promotor methylation of the *BDNF* gene were found in Wernicke's area of suicide completers compared to controls[114]. Hypermethylation of *BDNF* and the glucocorticoid receptor gene (*NR3C1*) promoters has been linked to increased risk of depression[115]. In the hippocampus and prefrontal cortex of adolescent suicide completers, a correlation between altered DNA methylation and decreased GR expression was found, as well as altered expression of methylating and demethylating enzymes[116]. In a recent epigenome-wide meta-analysis, differences in DNA methylation patterns were observed in the prefrontal cortex and cerebellum of suicide completers compared to controls[104]. A recent epigenome-wide association study on blood samples from military veterans associated four differentially methylated genomic sites with suicide attempts[105]. Genetic loci that were connected to suicidality in various DNA methylation studies commonly correspond to processes involved in (neuro)inflammation[117], cognition[106], cell cycle[111] and intercellular communication[107], cell structure regulation[112], transcription and expression regulation[108], synapse function and neurodevelopment[104,118], the polyamine system[109], the HPA axis[118] and glutamate signaling[110]. With regards to inflammatory markers, there is more data on DNA methylation patterns in depression than suicidality; for example, a genome-wide DNA methylation study with weighted gene co-methylation network analysis returned a depression-associated co-methylation module in which the pathways related to immune function were enriched. The results could be further linked to telomere length and IL-6 Levels[119]. Furthermore, a methylome-wide association analysis showed enrichment in autoimmune disease and inflammation, and the DNA methylation profile appeared stable as a longitudinal disease status biomarker[120]. On suicidality, a recent integrative transcriptome-methylome study by Sha *et al*[117] analyzed transcription and DNA methylation profiles in *post-mortem* brain of suicide completers and controls, identifying 40 differentially methylated regions that mapped to 7 immune-related genes: *ARPC2, CX3CL1, PSMB2, RNF41, RSF1, SPN* and *USP14. NPAS4*, a calcium-dependent transcription factor that regulates genes linked to neuroprotection and homeostasis of excitatory-inhibitory balance, was downregulated in suicide completers. Furthermore, the relationship between *NPAS4* methylation and expression was altered in suicide completers compared to controls[117]. This is interesting, as NPAS4 also regulates BDNF - the neuronal growth factor responsible for neuronal growth, development, and plasticity[117]. It is worth noting that sample heterogeneity (including suicide methods) and small group sizes, as well as differences in utilized methods, encumber result interpretation and comparison between studies.

Histone modifications

To date, few studies have examined posttranslational histone modifications in connection to suicidality. Histones are proteins involved in chromatin structure and gene transcription regulation[121]. Post-translational modifications of histone tails influence the level of chromatin compaction, and thus, the accessibility of DNA for transcriptional machinery. Specific histone modification patterns are associated with different genomic regions: Dimethylation and trimethylation of histone 3 Lysine 4 (H3K4) is associated with active promoters, while dimethylation and trimethylation of H3K9 and H3K27 are associated with repressed promoters[7]. Epigenetic regulation of BDNF *via* histone modifications

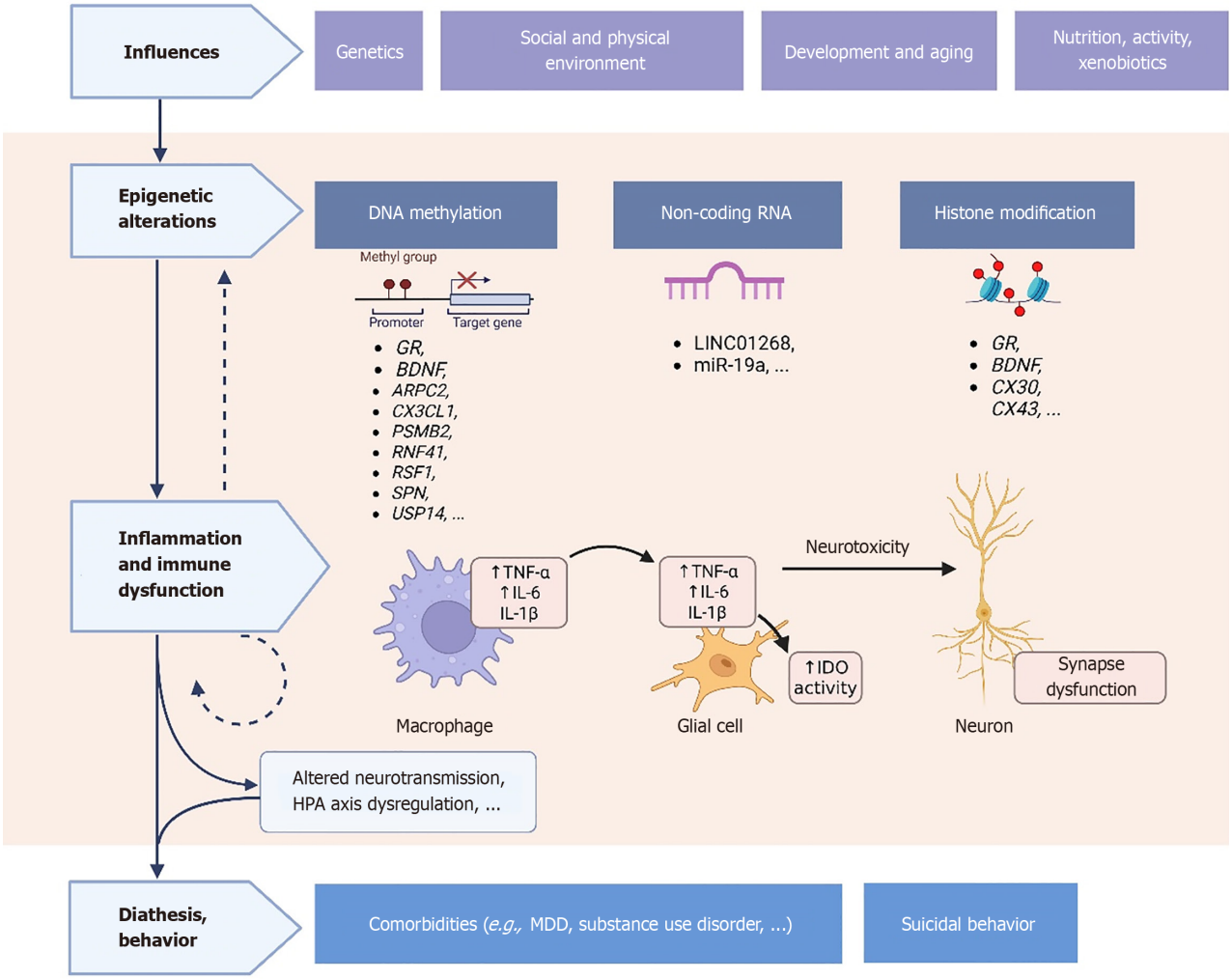

Figure 2 Schematic diagram: Interactions between epigenetics and the immune system in suicidality. This conceptual diagram, based on the synthesis of current literature, illustrates how various exogenous and endogenous factors influence the epigenetic state that contributes to suicide diathesis and suicidality itself. Epigenetic mechanisms regulate the expression of genes that are directly or, more commonly, indirectly involved in the immunity of peripheral and central tissues, which can also communicate with one another (*e.g.*, macrophage - glial cell interactions). As represented by the figure below the "DNA methylation" box, DNA methylation in promoter regions is associated with the repression of transcription. Genes and RNAs listed below the three epigenetic mechanisms (DNA methylation, non-coding RNAs and histone modifications) have been implicated in various methodologically different studies, including those with epigenome-wide approaches. In suicidality, epigenetic alterations may promote glial dysfunction and cytokine dysregulation, which has wide-reaching effects, impacting neuron function, neurotransmission and hypothalamic-pituitary-adrenal axis. Inflammatory processes may exacerbate each other (represented by the dashed circle) and contribute to the biological correlate of suicide diathesis, promoting the development of psychiatric disorders and influencing behavior. As represented by the dashed line, the immune system can also affect epigenetics, providing potential for both regulation and exacerbation. GR: Glucocorticoid receptor; BDNF: Brain-derived neurotrophic factor; ARPC2: Actin-related protein 2/3 complex subunit 2; CX3CL1: Chemokine (C-X3-C motif) ligand 1; PSMB2: Proteasome 20S subunit beta 2; RNF41: Ring finger protein 41; SPN: Sialophorin; USP14: Ubiquitin specific peptidase 14; IL: Interleukin; TNF-α: Tumor necrosis factor alpha; IDO: Indoleamine 2:3-dioxygenase; CX30: Connexin 30; CX43: Connexin 43; HPA: Hypothalamic-pituitary-adrenal; MDD: Major depressive disorder. Created in BioRender. Šmon J. (2025) https://BioRender.com/bdjmxj3.

has been implicated in rodent models of depression[122].

In humans, studies focusing on specific genes have connected histone modifications in *post-mortem* brain samples to suicidality, with a various degree of success. In the orbitofrontal cortex, increased H3K27 trimethylation in suicide completers corresponded to decreased expression of the astrocyte-expressed tropomycin receptor kinase B gene (*TrkB.T1*), which functions as a BDNF receptor[123]. Disturbances in astrocytic networks were connected to increased H3K9me3 in connexin 30 (CX30) and 43 (CX43) promoters in the prefrontal cortex[124]. In the frontal cortex and hippocampus of suicide completers, increased H3K27me2 and decreased H3K9/14ac were linked to deceased BDNF levels[125], which are associated with a proinflammatory profile[126]. Alterations in polyamine metabolism have been implicated in suicide neurobiology[127] and the overexpression of *OAZ1*, a gene involved in the polyamine system, was linked to increased H3K4me3 in its promoter region in the prefrontal cortex of suicide completers[121]. A recent study that utilized chromatin immunoprecipitation followed by high-throughput DNA sequencing demonstrated decreased H3K14ac levels in the hippocampus of suicide completers compared to controls, which associated with the differential expression of *ADORA2A*, *B4GALT2* and *MMP14*[128]; ADORA2A has been implicated in mood disorders[129], MMP14 in inflammation[130], while B4GALT was associated with suicide in blood proteomics[131].

ncRNAs

NcRNAs are a heterogenous group of functional RNA molecules that are not translated into proteins but participate in post-transcriptional regulation of gene expression[132]. In neuropsychiatry, the most examined ncRNA type are microRNAs (miRNAs), followed by lncRNAs, while other types are understudied[133].

LncRNAs are RNA molecules that are over 200 base pairs long, abundant in the brain and less evolutionarily conserved than other ncRNAs. They have been implicated in numerous inflammatory diseases[134], which makes them interesting candidates for suicide research. However, they are difficult to study due to the variety of effects they can exert on gene expression and are therefore poorly examined[7]. Downregulation of certain lncRNAs has been found in peripheral blood mononuclear cells of MDD patients compared to controls, with the presence of SI and previous suicide attempt(s) associated with greater alterations[135]. Furthermore, the use of violent methods for suicide has been associated with alterations in the brain expression levels of the human-specific lncRNA LINC01268, which may regulate genes associated with immunity[136], and LOC285758, which maps next to *MARCKS*, a gene upregulated in suicide. Recently, a novel lncRNA regulating *MAOA* gene expression was characterized in the brain of suicide completers, and its upregulation was associated with increased impulsivity and aggression[137,138]. In another study, altered expression of 23 LncRNAs, as well as their overlapping or antisense protein coding genes, was found in the rostral anterior cingulate cortex of depressed suicide completers compared to controls. Many of their corresponding genes were associated with the innate immune response, specifically IFN signaling[139].

MiRNAs are noncoding RNA molecules approximately 22 nucleotides long that regulate numerous cellular functions *via* binding to partially complementary mRNA targets and repressing translation[140]. Seventy percent of the total human miRNAs are estimated to be neuronal miRNAs, which makes them interesting targets for neuropsychiatric research[141]. Proper miRNA function is important for neurodevelopment, and altered miRNA expression has been associated with neurodegenerative and psychiatric disorders including depression, (neuro)inflammation and suicidal behavior[142,143]. Importantly, animal model studies have shown that ELA may contribute to suicidal behavior in adults by inducing abnormal miRNA expression[144], which may be detected in brain and blood[145,146].

Most suicidality studies have focused on miRNA expression in the prefrontal cortex of *post-mortem* brain, where global downregulation of miRNA was reported in suicide completers compared to controls[147], as well as increased expression of certain miRNAs, including those presumably involved in serotonin receptor 5-HT1AR signaling pathway[148] and TNF-α upregulation[149]. Downregulation of the miRNA miR-218 that targets deleted in colorectal cancer, which associates with susceptibility to depression after ELA exposure in mice[145,146], was found in suicide completers with MDD compared to controls[145,146,150]. Furthermore, differential regulation of miRNA was also identified in the locus coeruleus, the major site of noradrenaline synthesis in the brains of depressed suicide completers compared to controls [151]. MiRNAs can also travel *via* extracellular vesicles (EVs); in EVs isolated from *post-mortem* brain tissue, dysregulation of miRNAs associated with neurotransmission and synapse function was found in suicide completers[152]. In another recent study, miRNA levels in EVs from the CSF of suicide completers were compared to controls, and two differentially expressed miRNAs were found, which target *SLC6A4* and *TNF□α* expression[153].

Some studies have reported similar miRNA expression patterns in blood and brain tissue, which is promising because the detection of miRNAs in peripheral samples has greater potential for clinical implementation[154]. Downregulation of miR-1202, a brain-enriched miRNA that targets the metabotropic glutamate receptor 4 transcript, was found both in the prefrontal cortex of suicide completers with MDD and in blood samples from MDD patients, and may be predictive of antidepressant response[155]. In living subjects, expression of miRNA in the blood of patients with treatment-resistant depression was compared to that of controls, and the downregulation of two miRNAs, let-7b and let-7c, was identified as a possible biomarker[156]. In the peripheral blood of depressed patients, increased levels of TNF-α associated with a pro-inflammatory miRNA profile (upregulated miR-342, and downregulated miR-146a and miR-155) compared to controls [157]. Another recent study associated elevated serum levels of miR-30a, miR-30e, and miR-200a in suicidal MDD patients with acute suicide risk compared to MDD patients without acute suicide risk. Early growth response protein 1 was indicated as a transcriptional regulator of these miRNAs[158]. Furthermore, there is evidence that some miRNA levels are responsive to antidepressant treatment, which makes them an attractive biomarker for monitoring treatment success[159]. However, due to miRNA pleiotropy, selection of miRNAs that could function as suicide biomarkers is difficult[160], and *in silico* analysis algorithms are being developed for selection of brain-expressed miRNAs that target suicidality- and inflammation-associated genes[161].

STRESS, TRAUMA, AND THE IMMUNE SYSTEM; TRIGGERS AND MEDIATORS OF EPIGENETIC-IMMUNE DYSREGULATION

Stress, trauma, and inflammation in suicide

To further understand the mechanisms by which the epigenetic-immune interaction contributes to suicidality, we must consider the context of stress and trauma as predisposing factors. When CNS homeostasis is disrupted, there is a neuroinflammatory response, which is appropriate as long as it does not cause a chronic inflammatory state[162]. As the immune system is closely linked to the stress response, it is not surprising that suicidality research has correlated dysregulated stress responses with increased inflammation[163].

In the context of psychoneuroimmunology, the most often studied cytokines are IL-6, TNF, IL-1b, and INFs, associated with inflammation, and IL-10 which is associated with an anti-inflammatory effect[40]. Several of these were changed in suicide completers and attempters[164], as demonstrated in Table 1, and in most cases, they have also been linked to

stress. Preclinical studies have demonstrated increased brain IL-1β levels in response to stress[165], as well as the reversal of depressive-like behaviors after administration of IL-1β antagonists in rodents[166]. Furthermore, the activation of the hippocampal melacontin receptor MC4R reversed the deleterious effects of IL-1β on cognitive function[167]. In suicidal adolescents, ELA was associated with altered expression of glucocorticoid response elements in peripheral mononuclear cells[168].

Stress can be perceived as acute psychosocial stress, chronic social stress or ELA, which have all been linked to suicidality. Acute stress is associated with glucocorticoids and microglia activation, which leads to cytokine synthesis. In the case of ELA, the abnormal cortisol stress response and elevated levels of pro-inflammatory biomarkers such as CRP, IL-6 and TNF-α are present in subjects with chronic stress, GR resistance is observed. This resistance can also be induced by inflammatory cytokines, and elevated levels of IL-6 have been associated with chronic stress[163]. Events like traumatic brain injury (TBI) are also linked to immune system activation and are associated with microglia activation and increased inflammatory factor levels, such as IL-1 and TNF-α[169]. In a large study of TBI patients, the odds of dying due to suicide were three times higher in TBI patients than controls[170]. TNF-α levels positively correlated to SI[171].

Epigenetics as a mechanistic link between stress, inflammation and suicidality

Exposure to ELA has been robustly associated with an increased risk of maladaptive behavioral phenotypes[172], psychiatric disorders[173] and suicidality[174], as well as with structural and functional alterations in the brain[175] and HPA and immune system dysregulation[36]. The long-lasting effects of stress, including the influence of early-life social environment on stress reactivity in adulthood, has been extensively demonstrated in animal studies[176-179] and translated to humans[177,180-182].

As previously noted, poor adaptation to stress and dysfunction of the stress axis can be associated with changes in the regulation of certain genes. The candidate genes of the HPA axis, *SKA2, FKBP5, NR3C1* and *CRH* genes, are of particular interest; of the latter, *SKA2* has been linked to lifetime trauma history. The most evident results, however, are available for GR, which has an anti-inflammatory effect after cortisol binding. For this gene, lower expression[183] was associated with stress in rodents and humans due to increased DNA methylation[178]. In adult rats, insufficient maternal care promoted exaggerated stress response by inducing methylation changes in the *NR3C1* promoter in the hippocampus [177]. Furthermore, altered histone acetylation and transcription factor binding to the *NR3C1* promoter was observed [178]. Most importantly, however, the DNA methylation patterns could be modified by exposure to a different environment[178]. In adult male rats exposed to chronic stress, an altered methylation pattern was found in the promoters of glucocorticoid-responsive genes in the prefrontal cortex[179]. In rodents, these epigenetic changes could be pharmacologically reversed[178,179]. Epigenetic modifications are not concentrated only at single gene promoters but may be found in large clusters of over a hundred kilobase pairs; chromosomal regions containing synaptogenesis-related genes appeared to be highly influenced by variations in maternal care[184]. Recent studies also suggested the possibility of transgenerational inheritance of stress-induced epigenetic alterations, which may explain a portion of familial suicide risk that cannot be attributed to genetics or the environment[185,186]. Prenatal maternal stress in rats induced alterations in fetal brain development across four generations, as shown by DNA methylation, miRNA, and mRNA profiling in the fetal cortex. Interestingly, effects were more pronounced in the third and fourth generations, suggesting a cumulative effect[186]. In another study, pre-reproductive stress in female adolescent rats influenced the behavior and expression of CRH receptor 1 across the first and second generation offspring, with sex-specific effects on DNA methylation patterns in amygdala[185].

Similar effects of ELA and trauma have been observed in humans[180,181]: In suicide completers with a history of childhood abuse, decreased hippocampal expression and increased methylation in the *NR3C1* promoter was reported [182,187]. Furthermore, intrauterine stress induced by maternal stress can dysregulate the HPA axis[188], activate the immune system and may present a risk factor for the development of depression[189]. A similar picture of increased DNA methylation levels of *NR3C1* in the hippocampus was also observed in those who died by suicide when compared with the DNA methylation status of a control group of those who died from other causes. The effect was especially strong in those who died by suicide and who had experienced childhood abuse, suggesting that DNA methylation changes acquired in childhood remained active in adulthood. They also detected decreased levels of *NR3C1* expression, which is consistent with expectations, as increased levels of DNA methylation often lead to reduced gene expression[187,190]. An epigenome-wide association study identified the interactions between methylation patterns in the *SKA2* gene, whose product interacts with GR, and traumatic experiences as predictive of lifetime suicide risk[111]. This result was confirmed by candidate gene studies[191,192].

While it is well established that HPA axis dysfunction accompanies prolonged inflammation, there is little research on epigenetic alterations targeting immune-related genes in association with ELA and psychopathologies. Aside from stress-related genes, epigenetic mechanisms also regulate the expression of cytokines and TLRs, and environmental stressors such as ELA may contribute to a pro-inflammatory phenotype. Reduced *IL-6* promoter methylation has been associated with childhood trauma in a cohort of African American men[193]. Lam *et al*[194] examined peripheral blood DNA methylation patterns in over 14000 gene promoters from individuals with variations in early-life socioeconomic status. DNA methylation patterns were connected to leukocyte composition, demographics, cortisol levels, early-life poverty and psychosocial stress in adulthood. Interestingly, DNA methylation patterns were associated with inflammatory responses of peripheral blood mononuclear cells to *ex vivo* TLR stimulation, while the relationship to gene expression was weaker, which may suggest that epigenetic changes induced by ELA may create a poised transcriptional state, priming the immune system for altered response to future environmental challenges[194].

Epigenetic influences on stress, neuroinflammation and depression

The risk for psychiatric disorders, including depression, is increased by stressful events, particularly ELA, and it can be further associated with a more damaging disease course and with poor treatment outcome and suicidality[173], which is closely intertwined with epigenetic alterations and (neuro)inflammation. In a study by Miller and Cole[195], healthy adolescents with a high risk of depression were followed for 2.5 years. Neuroinflammation became present in subjects exposed to ELA, such as parental separation, low socioeconomic status, and familial psychopathology. Elevated levels of IL-6 and CRP were present in their blood. On the other hand, there were no changes in these biomarkers among the subjects not experiencing adversities[195]. When looking at DNA methylation, several genes that are associated with inflammatory and immune processes, such as *LTA, GFI1, ARID5B, CD52, TNFSF13, SLFN13, LST1, TNFRSF1A, CCRL2*, and *IL-32*, as well as NF receptor and TNF signaling pathway, were found differentially methylated in ELA patients that received trauma-focused psychotherapy. These findings supported the hypothesis of inflammatory/immune pathophysiology in MDD[196]. In addition to DNA methylation, miRNAs may be potential biomarkers for treatment-resistant depression in association with stressful life events. Candidates range from miR-29a, miR-125b, miR-126, miR-146a, miR-195, and let-7f implicated in inflammation and miR-15a associated with ELA, to miR-146a negatively correlated with recent stressful life events. MiR-15a, miR-29a, miR-126, miR-195, and let-7f levels changed during psychotherapy, and miR-29a also functioned as a response predictor[197]. Such findings highlight the potential of translating the knowledge on epigenetic and immune alterations in depression and suicidality into biomarker-driven strategies for suicide risk prediction and alleviation.

PREVENTIVE STRATEGIES AND THERAPEUTIC INTERVENTIONS

Biomarkers for suicide risk estimation

With increasing insight into the neurobiological background of suicidality, new diagnostic and therapeutic avenues are possible. Identification of objective biological markers associated with suicide risk is particularly promising, as it could lead to the development of minimally invasive risk stratification tools and increase the prediction value of clinical assessments[4]. However, no such quantifiable indicators are currently available[198]. Possible targets include the molecular components of neurotransmitter systems, the HPA axis, the immune response and inflammation, as well as endocannabinoids and lipids[198]. As some of these targets may be affected on different levels (genetic and epigenetic alterations, as well as altered mRNA or protein concentrations), multi-omic research has the potential to facilitate biomarker discovery[13]. Epigenetic markers of immune response are particularly compelling candidates, as they reflect the interplay between genetic vulnerability and environmental exposures[103], and are important in mediating the influence of ELA and stress on suicide risk[199]. Furthermore, inflammation may induce epigenetic changes[200], which has therapeutic implications, especially for antidepressant non-responders. Figure 3 highlights the potential future applications of peripheral immunology and/or epigenetics- related biomarkers in assessing suicide risk and predicting effective treatment.

Regarding epigenetic biomarkers of neuroinflammation in the context of suicidality, ncRNAs are especially promising due to their accessibility in peripheral body fluids[141,198], high specificity for cell of origin and possibly being in comparison to novel protein biomarkers, cheaper to detect[201]. In a randomized clinical trial, some miRNAs in combination with clinical testing, have predicted worsening of SI in patients receiving antidepressant treatment[202]. However, miRNA detection has certain limitations: Levels of neuronal miRNAs in peripheral tissues largely reflect the integrity of the BBB, which may be altered in various inflammatory conditions. Furthermore, it is difficult to identify the freely circulating miRNAs' tissue of origin. Therefore, EV-derived miRNAs may be advantageous because they can be traced to their (*e.g.*, neuronal) tissue of origin *via* surface protein markers[203]. Exosomal miRNAs are secreted physiologically in response to stress[204] and as previously mentioned, their CSF levels have been linked to suicide in *post-mortem* studies[153]. However, their levels appear to be low in peripheral fluids, requiring sensitive detection methods[205], which raises the question of cost-effectiveness. Further studies examining EV-derived miRNA in peripheral fluids of living subjects with suicide risk, in combination with inflammation marker assessment, are necessary to evaluate their biomarker potential.

Many studies have also examined protein expression levels as potential suicidality biomarkers[9]. For example, an analysis of matched *post-mortem* blood and brain samples of depressed patients associated the differential expression of four genes with suicide completion, of which two (decreased *SOX9* and increased *PER3* levels) showed the same pattern of change in both tissues[206]. A recent study investigated the genetic basis of SI in patients with MDD *via* a weighted gene co-expression network analysis on peripheral blood RNA-sequencing data. These results suggest that gene expression regulation, immune response, and inflammation may be linked to SI among MDD patients, while innate immunity, mitochondrial function, and neurodegeneration are more broadly associated with MDD[207].

Peripheral markers of inflammation are consistently being reported as possible biomarkers of suicide risk[208]. Elevated neutrophil-to-lymphocyte ratio, primarily driven by higher neutrophil counts, has recently emerged as a potential marker of suicidality[209]. A recent 2-year follow-up study evaluated 39 molecular analytes in peripheral blood of patients with mood disorders. Elevated CRP and plasma thrombospondin-2 were linked to recent suicide attempts, while low plasma serotonin levels were linked to SI. Thrombospondin-1 and platelet-derived growth factor were predictive of future suicidal events. Interestingly, there was no overlap between markers of past and future suicidal events, suggesting the possibility of distinct molecular components influencing SI development of and attempt[210]. Abnormal expression of inflammation-related genes in monocytes was found in MDD patients, and patients with upregulated gene profiles had a higher prevalence of ELA and high suicide risk. These alterations were not observed in

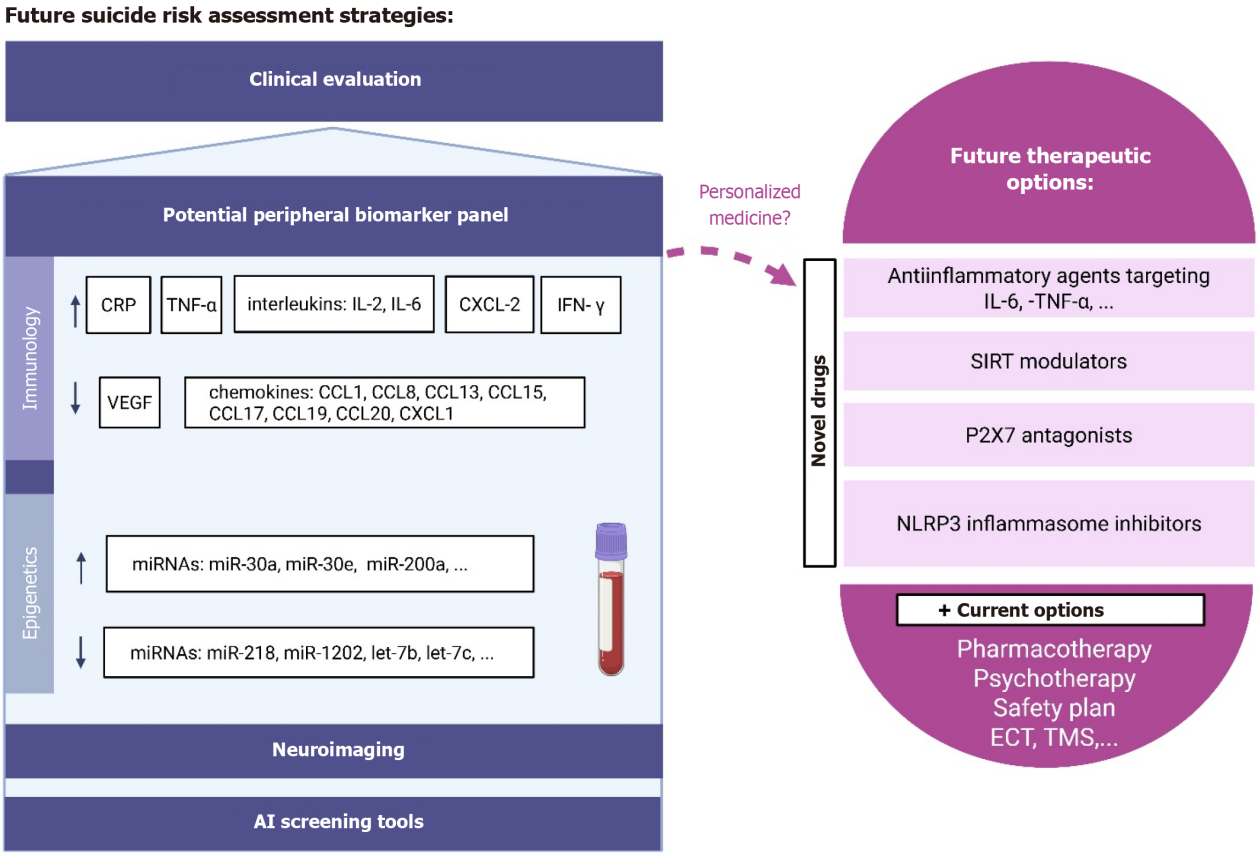

Figure 3 The role of biomarkers in suicide risk assessment and future therapeutics. This conceptual illustration, based on the synthesis of recent literature, demonstrates the possible future directions for suicide risk assessment and personalized treatment strategies. On the left side, a potential future suicide risk assessment strategy is presented, in which various tools (peripheral blood biomarkers, neuroimaging, artificial intelligence technology) may aid the clinician in evaluating suicide risk. Increased or decreased levels of some of the potential immunological (interleukins, chemokines, *etc.*) and epigenetic biomarkers, are listed, based on reported associations in suicidality and depression. On the right side, possible future therapeutic options are presented for suicide risk alleviation and/or the treatment of underlying psychiatric disorders, that may be used in combination with established treatment options. Immune modulators and anti-inflammatory agents are listed, but not epigenetic modifiers, as there is stronger implication for the clinical implementation of the former. Blood-based biomarker measurement in peripheral blood, *e.g.*, *via* enzyme-linked immunosorbent assay or multi-marker panels, could contribute to individualized risk profiling and thus enable personalized therapy. AI: Artificial intelligence; CRP: C-reactive protein; TNF-α: Tumor necrosis factor alpha; VEGF: Vascular endothelial growth factor; IL: Interleukin; CCL: Chemokine C-C motif ligand; CXCL: Chemokine (C-X-C motif) ligand; ELISA: Enzyme-linked immunosorbent assay; IFN-γ: Interferon gamma; SIRT: Sirtuin; P2X7: Purinoceptor 7; NLRP3: NLR family pyrin domain containing 3; ECT: Electroconvulsive therapy; TMS: Transcranial magnetic stimulation. Created in BioRender. Šmon J. (2025) https://BioRender.com/jm04fz5.

controls with a history of ELA, suggesting that inflammation specifically occurs in MDD-predisposed individuals exposed to trauma[211].

Methodological limitations

Despite the increasing attempts at elucidating molecular changes associated with suicide risk, including epigenetic and immune alterations, there are currently many drawbacks that limit the utility of these findings. Among studies, there are pronounced differences in cohorts and methodologies, as well as the criteria for assessing suicidal behavior (ideation, attempts, differences in means used for suicide)[9]. The influence of sex, age, psychiatric and other comorbidities, medications, socioeconomic environment and other confounding factors may contribute to the heterogeneity of participants and the lack of replicable results by obscuring true biomarker effects[4]. For example, some potential biomarkers seem to be sex-specific, such as *SAT1* expression in blood[206]. It is challenging to distinguish molecular aspects unique to suicidality from the influence of disorders commonly associated with it, including MDD and substance abuse[4], as most suicide completers have comorbid psychiatric diagnoses[2]. Similarly, many studies suffer from small sample sizes and therefore low statistical power. Furthermore, most potential biomarkers lack accuracy and most notably specificity; for example, blood levels of CRP and IL-6 are associated with suicide risk[65], however, they are also elevated in numerous other disorders and show high variability among individuals[212]. While peripheral tissues and fluids, including blood, saliva and hair, may present possible biomarker sources[213], most suicidality research is performed on *post-mortem* brain, which undergoes rapid degradation, and variations in the *post-mortem* interval present an important confounding factor[11]. It is also not yet clear how *post-mortem* brain findings translate to other *post-mortem* or living tissues, such as venous blood which is the preferred clinical sample due to ease of access, or even among different brain regions[11]. Furthermore, *post-mortem* samples do not provide longitudinal insight into the development of suicidality, or real-time assessment of suicide risk. This is problematic, as identifying suicide risk factors that characterize the early

stages in the progression from predisposition towards suicidal behavior is crucial for timely intervention[2]. To overcome at least some of these challenges, future research should prioritize large, well-characterized cohorts, standardized methodologies and, if possible, longitudinal designs. Complementing *post-mortem* studies with approaches that target living participants, and integrating multi-omics approaches with clinical and behavioral data, would enhance replicability and predictive value, which would facilitate the discovery and clinical implementation of reliable suicidality biomarkers.

Due to the heterogeneity of suicidality, characterization of distinct biological subtypes (*e.g.*, the impulsive and planned behavior subtypes) would be an important future goal in suicidality research, and continuing with integrative studies is important for the realization of personalized medicine in the psychiatric field[214]. Statistical and machine learning approaches may aid in subtype identification[215]. Controlling for confounding factors is a challenging but important step in planning future research to increase reproducibility. With technological advancement, it is likely that a panel of multiple biomarkers, including immune and epigenetic alterations, in concert with clinical assessment and computerized adaptive tests, will be used for risk stratification in the future[4]. Combining clinical and biochemical modalities with brain imaging studies such as functional magnetic resonance imaging would further contribute to improved risk assessment, however, cost- and time-effectiveness may present barriers to their clinical implementation[216].

Therapeutic opportunities and future directions

Despite methodological challenges in suicidality research and the current lack of reliable biomarkers, the growing insights into the inflammatory background of suicidality are driving the development of novel therapeutic strategies for alleviating suicide risk and treating suicide-associated pathologies, most notably, MDD. High inflammation and insufficient anti-inflammatory response have been associated with resistance to antidepressant treatment in MDD[40]. In randomized clinical trials, various anti-inflammatory agents, including omega 3 fatty acids, non-steroidal anti-inflammatory drugs (NSAIDs), minocycline and statins have exhibited limited efficacy in alleviating depressive symptoms, with differences in patient subpopulations[217]. Furthermore, glial dysfunction and neuroinflammation are being targeted *via* purine receptor antagonists, which also mediate IL-1β release[218], and inflammasome inhibitors[219]. Among more promising potential anti-inflammatory agents for depression treatment are sirtuin modulators[220], which have recently shown promise in Phase 1 clinical trial[221]. While anti-inflammatory drugs are increasingly being explored in the treatment of depression[38], it is more difficult to assess their potential in suicide prevention, as subjects with increased suicide risk are rarely included in clinical trials[222]. Still, a reduction in SI was noted in patients with depression that were taking NSAIDs[223]. Certain medications used in depression treatment, *e.g.*, ketamine, also exert an anti-inflammatory effect, especially in patients with higher inflammation levels, and a rapid reduction of suicidal thoughts has been observed in patients treated with ketamine[224]. Glutamatergic system modulation and the activation of the neuroprotective aspects of the kynurenine pathway are thought to contribute to the anti-inflammatory effects of ketamine. Therefore, clinical trials are examining the potential of dual (antidepressant and anti-inflammatory) therapy in treatment-resistant depression[225]. It is likely that future therapeutic strategies will utilize a similar, but increasingly personalized antidepressant and anti-inflammatory combination approach.

Despite the growing evidence of epigenetic dysregulation in suicidality, therapeutic approaches targeting epigenetic modifications have not yet been examined due to their effects on global gene expression and safety concerns. Currently, the use of demethylating agents and histone deacetylase inhibitors is limited to hematologic malignancies[226]. However, animal studies have suggested that histone deacetylase inhibitors may have antidepressant-like effects in animals, most likely by upregulating *BDNF* expression[227]. Furthermore, epigenetic alterations may help in predicting treatment efficacy[228]. Non-pharmacological approaches, including psychotherapy[197] and lifestyle interventions such as mindfulness, exercise and diet, may also exert anti-inflammatory effects *via* epigenetic mechanisms, highlighting the importance of lifestyle medicine in treatment and prevention of depressive symptoms[229]. In MDD patients, aerobic exercise was shown to influence the concentration and miRNA cargo of EVs, stimulating their neuroprotective effect[230].

Recent advancements in artificial intelligence (AI) methods have shown promise for improving suicidality risk assessment[231]. However, current machine learning approaches are limited, as they do not include neurobiological and neuroimaging data[232]. The development of AI-driven models harnessing large-scale datasets, including multi-omics data, may facilitate the discovery of complex relationships between molecular alterations and suicidal behaviors[233]. In future research, a collaborative, interdisciplinary approach is required to adequately address the heterogeneity of suicidality, as well as to ensure adherence to ethical standards in utilizing large datasets[231].

CONCLUSION

Suicidality is a complex and multifactorial phenomenon. There is increasing evidence of the involvement of epigenetic and immune alterations in the development of suicide risk, particularly in mediating the influences of stress and early-life adversity. While the interplay between epigenetic regulation and (neuro)inflammatory processes, including cytokine imbalance and glial dysfunction, remains underexplored in neuropsychiatric research, there is compelling evidence that advancing our understanding of this field may enable the development of personalized approaches to suicide risk assessment, monitoring and treatment of psychiatric comorbidities. Continued interdisciplinary research integrating multi-omic approaches, applying well-characterized cohorts, is essential to ensure reproducibility of findings.

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