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Structural and Functional Neuroimaging in Major Depressive Disorder with Suicidal Ideation: A Review

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Abstract

Major depressive disorder with suicidal ideation (MDD-SI) represents a clinically high-risk subtype of depressive disorders characterized by marked neurobiological heterogeneity and has become a major focus of suicide prevention and precision intervention research. In recent years, neuroimaging studies have increasingly demonstrated that patients with MDD-SI exhibit distinct structural and functional brain alterations compared with depressed patients without suicidal ideation. These abnormalities primarily involve the prefrontal–cingulate–limbic system, the default mode network (DMN), and reward-related circuits. With the growing number of longitudinal neuroimaging studies, accumulating evidence suggests that biological treatments—particularly electroconvulsive therapy (ECT)—not only lead to significant reductions in depressive symptoms and suicidal ideation but also induce measurable structural and functional plasticity in these key brain regions and networks.

Structural MRI studies indicate that, at baseline, patients with MDD-SI commonly show gray matter abnormalities in regions such as the anterior cingulate cortex, prefrontal cortex, and hippocampus. Following ECT treatment, regionally selective structural changes have been observed, most notably hippocampal volume increases and structural recovery in the anterior cingulate cortex and ventromedial prefrontal cortex. Functional neuroimaging studies further demonstrate that treatment-related reductions in suicidal ideation are accompanied by enhanced prefrontal–limbic regulatory control, normalization of anterior cingulate cortex function, and suppression of excessive self-referential processing within the DMN. Collectively, these findings suggest that treatment-related neuroimaging changes may constitute an important neural substrate underlying the improvement of suicidal ideation.

This review summarizes current structural and functional neuroimaging findings in patients with MDD-SI before and after treatment, with particular emphasis on treatment-related plasticity in key brain regions and networks. Furthermore, an integrative conceptual framework—linking baseline structural abnormalities, treatment-induced neuroplasticity, and the alleviation of suicidal ideation—is proposed to provide insights for future research on imaging-based predictive biomarkers and the neurobiological mechanisms underlying treatment response.

Keywords: major depressive disorder, suicidal ideation, neuroimaging, functional magnetic resonance imaging, therapy

1. Introduction

Major depressive disorder (MDD) is a mood disorder characterized by persistent depressed mood and/or a marked loss of interest or pleasure. It is one of the most representative and disabling conditions within the spectrum of depressive disorders (Association, 2013; Malhi & Mann, 2018).

Major depressive disorder (MDD) shows considerable clinical heterogeneity (Kessler et al., 2003). According to symptom severity, MDD can be classified as mild, moderate, or severe; based on clinical features, it may also present with psychotic features, anxious distress, mixed features, or somatic symptoms (Association, 2013). Major depressive disorder (MDD) is strongly associated with an increased risk of suicide, with approximately 60% of suicide deaths linked to depressive disorders (Turecki & Brent, 2016). Suicidal ideation and a history of suicide attempts are important indicators of disease severity and poor prognosis (Mann et al., 2005).

Major depressive disorder with suicidal ideation (MDD-SI) refers to a clinical condition in which individuals meeting the diagnostic criteria for major depressive disorder experience persistent or recurrent suicidal thoughts during depressive episodes (Association, 2013; Turecki & Brent, 2016). According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) (Association, 2013), suicidal ideation is one of the core symptoms of a major depressive episode and includes both passive wishes for death and active suicidal thoughts, such as recurrent thinking about suicide methods or plans.

Accumulating evidence indicates that suicidal ideation not only reflects the severity of depression but also represents a key clinical indicator for predicting suicidal behavior and adverse outcomes (Ribeiro, Huang, Fox, & Franklin, 2018). Compared with depressed patients without suicidal ideation (MDD-nSI), individuals with MDD-SI generally show more severe depressive symptoms, particularly depressed mood, hopelessness (Ribeiro et al., 2018) and anhedonia (Ballard et al., 2017), and are often accompanied by pronounced negative cognitive biases, such as rumination, feelings of worthlessness, and excessive guilt. Epidemiological studies indicate that approximately 40%–70% of patients with major depressive disorder experience suicidal ideation at some point during their lifetime (Cai et al., 2021). Persistent or recurrent suicidal ideation is widely regarded as one of the most robust risk factors for suicide attempts and suicide deaths (Turecki & Brent, 2016). Depressive disorders are also among the psychiatric conditions most strongly associated with suicide, and more than half of individuals who die by suicide have a history of depressive disorders (Turecki & Brent, 2016).

The assessment of suicidal ideation varies across studies, and commonly used instruments include the Beck Scale for Suicide Ideation (BSSI) (Beck, Kovacs, & Weissman, 1979), the Columbia-Suicide Severity Rating Scale (C-SSRS) (Posner et al., 2011) and suicide-related items from depression rating scales(Guo et al., 2024), such measurement heterogeneity may affect the comparability of findings across studies.

Increasing evidence suggests that suicidal ideation is not merely an epiphenomenon of depression severity but may represent a depressive subtype with relatively distinct psychological and neurobiological underpinnings (Schmaal et al., 2020). Neuroimaging studies have shown that patients with MDD accompanied by suicidal ideation exhibit distinct structural and functional abnormalities in the prefrontal–limbic system, emotion regulation networks, and reward-related brain regions (Schmaal et al., 2020). These alterations may be closely associated with impaired impulse control, dysregulation of negative emotions (Turecki & Brent, 2016), and pessimistic expectations about the future (Ribeiro et al., 2018). Therefore, investigating major depressive disorder with suicidal ideation as a distinct research focus may help to better understand the heterogeneity of depressive disorders and provide important insights into the neural mechanisms underlying suicide risk.

The treatment of major depressive disorder should follow a stratified, individualized, and sequential framework of comprehensive interventions (Lam et al., 2024). Treatment decisions should take into account multiple factors, including symptom severity, the presence of psychotic features or suicide risk, previous treatment response, medical and psychiatric comorbidities, and patient preferences (Health & Excellence, 2022). Overall, pharmacotherapy constitutes the first-line intervention for patients with moderate-to-severe and severe major depressive disorder. In cases

where symptoms are severe, rapid treatment response is required, or pharmacological treatment is ineffective, intensified biological treatments such as electroconvulsive therapy (ECT) may be considered (Lam et al., 2024). Throughout the treatment process, continuous management across the acute, continuation, and maintenance phases is emphasized to achieve symptom remission and reduce the risk of relapse (Bauer et al., 2015).

With the development of longitudinal neuroimaging studies, accumulating evidence suggests that biological treatments not only significantly alleviate depressive symptoms and suicidal ideation but also induce structural and functional plasticity in key brain regions. Therefore, systematically summarizing the structural and functional neuroimaging findings in patients with major depressive disorder with suicidal ideation before and after treatment is of great importance for understanding the neural mechanisms underlying suicidal ideation. This review aims to summarize the neuroimaging abnormalities observed in patients with MDD-SI and to highlight treatment-related imaging changes and their potential neural mechanisms, with the goal of providing insights for future research on imaging-based predictive biomarkers and precision interventions.

2. Neuroimaging Characteristics of MDD-SI

2.1 Structural Neuroimaging Alterations

Structural abnormalities are primarily distributed within the prefrontal–limbic system, which plays a crucial role in key psychological processes such as emotion regulation, impulse control, and value evaluation (Schmaal et al., 2020).

The prefrontal cortex is one of the most consistently reported abnormal regions in studies of MDD-SI (Schmaal et al., 2020). Several voxel-based morphometry (VBM) and cortical thickness studies have demonstrated reduced gray matter volume or cortical thickness in the dorsolateral prefrontal cortex (DLPFC), orbitofrontal cortex (OFC), and ventromedial prefrontal cortex (vmPFC), with these alterations being particularly pronounced in patients with MDD who have suicidal ideation or a history of suicidal behavior (Ding et al., 2015). These regions play a critical role in emotion regulation, impulse control, and future value evaluation (Haber & Knutson, 2010), and structural impairments in these areas may reduce individuals' cognitive control over negative emotions and suicidal impulses (Turecki & Brent, 2016).

The anterior cingulate cortex (ACC) (Xia, Wu, Wang, Zhou, & Zhang, 2025), particularly the rostral and dorsal ACC, is widely considered a key neural hub integrating emotional processing and cognitive control (Shackman et al., 2011). Structural MRI studies have reported a higher prevalence of ACC gray matter reduction or cortical thinning in patients with MDD-SI compared with depressed patients without suicidal ideation (H. Li et al., 2021; Wagner et al., 2011). Structural abnormalities of the ACC may be associated with increased feelings of hopelessness, impaired regulation of emotional conflict, and suicide-related rumination (Mann, 2003).

Within the limbic system, structural alterations of the amygdala (Cong et al., 2022) and hippocampus (Xu et al., 2023) have been repeatedly reported in patients with MDD-SI. Some studies have found that depressed patients with suicidal ideation or a history of suicide attempts exhibit reduced amygdala volume (Cong et al., 2022), suggesting abnormalities in emotional reactivity and threat processing. Meanwhile, hippocampal volume reduction is also commonly observed (Gosnell et al., 2016), which may be associated with chronic stress (Sapolsky, 2000), dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis (Pariante & Lightman, 2008), and negative biases in emotional memory (Gotlib & Joormann, 2010).

In addition to cortical and limbic structures, several studies have also reported volumetric alterations in subcortical regions such as the striatum (Ho et al., 2021), thalamus (Kang et al., 2020), and insula (Schmaal et al., 2020). These regions are involved in reward processing, interoception, and the perception of distress. Overall, structural abnormalities in patients with MDD-SI are unlikely to occur in isolation but rather reflect a broader imbalance between emotion regulation and cognitive control networks (Marchand et al., 2012).

2.2 Functional Neuroimaging Alterations

Functional neuroimaging studies indicate that patients with MDD-SI exhibit significant functional abnormalities in neural networks involved in emotion regulation (V. C. Chen et al., 2021), cognitive control (Ouyang et al., 2022), self-referential processing (Ouyang et al., 2022), and

reward processing (Qiao et al., 2020).

In both task-based and resting-state functional magnetic resonance imaging (fMRI) studies, patients with MDD-SI commonly exhibit reduced regulatory control of the prefrontal cortex over the limbic system (Pu et al., 2015). Specifically, decreased activity has been observed in the dorsolateral prefrontal cortex (DLPFC) and the ventromedial/orbitofrontal prefrontal cortex (vmPFC/OFC) (Kim et al., 2017), whereas the amygdala shows hyperactivation in response to negative emotional stimuli (Victor, Furey, Fromm, Ohman, & Drevets, 2010). Compared with depressed patients without suicidal ideation (MDD-nSI), individuals with MDD-SI more frequently exhibit weakened or dysregulated functional connectivity between the prefrontal cortex and the amygdala (Li et al., 2022), which may contribute to heightened negative emotional responses and reduced impulse control.

The anterior cingulate cortex (ACC) is one of the most frequently reported abnormal regions in functional neuroimaging studies of MDD-SI (M. Zhang et al., 2025). Studies have shown that patients with MDD-SI exhibit abnormalities in resting-state activity of the ACC (M. Zhang et al., 2025) as well as in its functional connectivity with the prefrontal cortex and limbic system (Du et al., 2017). Network abnormalities involving the dorsal ACC (dACC), which have been associated with suicidal behavior, may be related to dysfunction in conflict monitoring and error detection processes (Minzenberg, Lesh, Niendam, Cheng, & Carter, 2016).

Resting-state fMRI studies have demonstrated significant abnormalities in the default mode network (DMN) in patients with MDD-SI, particularly increased functional connectivity among the medial prefrontal cortex (mPFC), posterior cingulate cortex (PCC), and precuneus (M. Zhang et al., 2025). Compared with depressed patients without suicidal ideation (MDD-nSI), individuals with MDD-SI show more pronounced DMN hyperactivity or enhanced internal connectivity (Wei et al., 2018). These abnormalities are thought to be closely associated with excessive self-focus, rumination, and negative self-evaluation (Hamilton, Farmer, Fogelman, & Gotlib, 2015), and may represent an important neural basis underlying the persistence of suicidal ideation.

In task-based fMRI studies involving reward processing and decision-making, suicide-related depressive populations (including individuals with suicidal ideation and suicide-related subgroups) commonly exhibit reduced responsiveness in the ventral striatum, orbitofrontal cortex (Jollant, Lawrence, Olié, Guillaume, & Courtet, 2011), and other prefrontal regions. Compared with depressed patients without suicidal ideation (MDD-nSI), individuals with MDD-SI show blunted neural responses to positive stimuli and reward feedback. These findings suggest that anhedonia and impaired future value evaluation may play a key role in the development of suicidal ideation (Schmaal et al., 2020).

3. Treatment-Related Neuroimaging Changes in MDD-SI

3.1 Treatment-Related Structural Brain Changes

In recent years, longitudinal structural MRI studies have indicated that some depression- and suicide-related structural abnormalities in patients with MDD-SI may be reversible following effective treatment (Tendolkar et al., 2013), suggesting that structural brain plasticity may represent an important neural basis underlying clinical improvement, particularly the alleviation of suicidal ideation. Current evidence is mainly derived from studies of electroconvulsive therapy (ECT), whereas structural neuroimaging studies examining pharmacological treatments with suicidal ideation as an outcome remain relatively limited (Vieira, Faria, Ribeiro, Picó-Pérez, & Bessa, 2023).

The hippocampus represents one of the most consistently reported regions exhibiting treatment-related structural changes (Wilkinson, Sanacora, & Bloch, 2017). Multiple longitudinal MRI studies have demonstrated that hippocampal volume significantly increases following ECT in patients with MDD, including those with suicidal ideation (Nordanskog et al., 2010; Tendolkar et al., 2013), and the magnitude of this increase has been associated with improvements in certain clinical symptoms, including reductions in suicidal ideation (Nordanskog, Larsson, Larsson, & Johanson, 2014). Given that patients with MDD-SI often exhibit more pronounced hippocampal atrophy(Xu et al., 2023), this “reverse plasticity” has been suggested to reflect the restorative effects of ECT on stress-related neurotoxicity and impaired neurogenesis (Duman & Aghajanian, 2012).

In the anterior cingulate cortex (ACC) (Pirnia et

al., 2016) and certain prefrontal regions, such as the orbitofrontal cortex (OFC) (Gbyl et al., 2019), increases in gray matter volume or cortical thickness have also been observed following ECT. These regions represent key structural nodes that show the most pronounced differences between patients with MDD-SI and those without suicidal ideation (MDD-nSI) (Yi et al., 2025). The treatment-related structural changes in these areas suggest that ECT may reduce the persistence of suicide-related thoughts by reshaping critical hubs involved in emotion regulation and cognitive control (Pirnia et al., 2016; Yi et al., 2025).

Notably, ECT-related structural changes are not diffusely distributed across the entire brain but rather exhibit regional specificity (Mulders et al., 2020), primarily involving the prefrontal–cingulate–limbic system (Pirnia et al., 2016). Some studies have suggested that patients with more pronounced baseline structural abnormalities (e.g., smaller hippocampal volume (Joshi et al., 2016) tend to show greater structural recovery and clinical improvement following ECT. This finding may have potential value in predicting treatment response in patients with MDD-SI (Cao et al., 2018).

Compared with ECT, structural MRI evidence related to pharmacological treatment (antidepressants) remains relatively limited and inconsistent (Schmaal et al., 2016). A small number of longitudinal MRI studies suggest that sustained antidepressant treatment may be associated with stabilization or even modest increases in hippocampal volume. For example, in a 3-year follow-up study, patients who maintained antidepressant treatment exhibited increased left hippocampal volume (Frodl et al., 2008), whereas a longer duration of untreated depressive episodes was associated with hippocampal volume reduction (Sheline, Gado, & Kraemer, 2003). However, high-quality studies specifically focusing on patients with MDD-SI and using changes in suicidal ideation as the primary outcome are still lacking (Schmaal et al., 2020). Therefore, from the perspective of structural neuroimaging, ECT remains the intervention with the most consistent supporting evidence (Gryglewski et al., 2021).

Current structural MRI evidence indicates that, following effective treatment—particularly ECT—patients with MDD-SI may exhibit reversible structural changes in key brain regions such as the hippocampus, anterior cingulate cortex, and prefrontal cortex (Pirnia et al., 2016; Wilkinson et al., 2017). These treatment-related structural plasticity changes may constitute an important neural basis underlying the alleviation of suicidal ideation and provide support for the use of structural imaging markers as tools for predicting treatment response and investigating underlying mechanisms (Joshi et al., 2016; Pirnia et al., 2016).

3.2 Treatment-Related Functional Brain Changes

Existing longitudinal fMRI studies indicate that, following effective treatment, the alleviation of suicidal ideation in patients with MDD-SI is accompanied by functional reorganization in specific brain networks (Su et al., 2025; Wang et al., 2023). These changes are not uniformly distributed across the whole brain (X. Li et al., 2021) but are selectively concentrated in the prefrontal–cingulate–limbic system (Wang et al., 2023), the default mode network (DMN) (Verdijk et al., 2024), and reward- and emotion regulation–related networks. These findings suggest that treatment may exert its effects through two major mechanisms: restoring top-down regulatory control and suppressing excessive self-referential processing.

Following ECT, patients with MDD-SI commonly exhibit increased activity in prefrontal regions, including the dorsolateral prefrontal cortex (DLPFC) and the ventromedial/orbitofrontal prefrontal cortex (vmPFC/OFC) (Wang et al., 2023), along with a reduction in the amygdala's hyperreactivity to negative stimuli (Redlich et al., 2017). More importantly, enhanced functional connectivity between the prefrontal cortex and the amygdala has been observed, suggesting a restoration of top-down cognitive control over emotional responses (Redlich et al., 2017).

The anterior cingulate cortex (ACC) is one of the key nodes with the most consistent evidence of treatment-related functional changes (Pirnia et al., 2016). Longitudinal studies have indicated that, following ECT, patients with depression accompanied by suicidal ideation exhibit significant treatment-related reorganization in both resting-state ACC activity (Wang et al., 2023) and its functional connectivity with the prefrontal–limbic system (Cano et al., 2016). Given the central role of the ACC in processes such as rumination, self-monitoring, and conflict monitoring (Hamilton et al., 2012), this functional reorganization is thought to contribute to the rapid alleviation of suicidal ideation (Schmaal et

al., 2020).

Following ECT, abnormal internal connectivity within the default mode network (DMN) shows partial normalization or reorganization (Verdijk et al., 2024), with some studies reporting increased connectivity (Pang et al., 2022) and others showing reduced connectivity in specific pathways (Denier et al., 2023). These changes particularly involve the medial prefrontal cortex (mPFC), posterior cingulate cortex (PCC), and their coupling with memory-related structures such as the hippocampus. In addition, the anticorrelation between the DMN and task-positive networks, such as the prefrontal control network, is enhanced (Moreno-Ortega et al., 2019). Given that patients with MDD-SI commonly exhibit DMN hyperactivity (S. Zhang et al., 2016) and increased rumination (Hamilton et al., 2015), these changes are thought to correspond to a reduction in repetitive self-referential thoughts related to suicide.

In task-based fMRI studies examining rapid-acting treatments, increased responses in reward-related brain regions, including the striatum, have been observed during reward feedback processing (Kotoula et al., 2022). In contrast, evidence related to ECT has been derived primarily from resting-state studies, which indicate increased intrinsic activity or connectivity within orbitofrontal/prefrontal reward-related circuits, and these changes have been associated with improvements in anhedonia (T. Zhang et al., 2021).

Studies on rapid-acting treatments targeting suicidal ideation, such as ketamine, have shown that increased connectivity within the prefrontal–limbic system (M. H. Chen et al., 2019) and reduced activity in the default mode network (DMN) (Scheidegger et al., 2012) can emerge within a short time frame. These patterns are highly consistent with the network-level changes observed following ECT in terms of "network directionality" (Wang et al., 2023), although they occur on a shorter timescale (Stapper et al., 2025), suggesting that different treatments may alleviate suicidal ideation through similar functional pathways. However, it should be noted that neuroimaging studies of rapid-acting treatments in patients with MDD-SI are currently limited by small sample sizes and short follow-up periods. Larger longitudinal studies with suicidal ideation as the primary outcome are still needed to validate the specificity of the underlying network mechanisms (Sajid, Mann, & Grunebaum, 2025).

Functional neuroimaging evidence suggests that, following effective treatment—particularly ECT—patients with MDD-SI exhibit enhanced prefrontal–limbic regulatory control, normalization of ACC function, and suppression of excessive self-referential processing within the default mode network (DMN). These network-level functional reorganizations may provide an important neural basis for the rapid alleviation of suicidal ideation.

3.3 Potential Mechanisms Underlying Treatment-Related Neuroimaging Changes

Existing neuroimaging studies suggest that the structural and functional changes observed in patients with MDD-SI following effective treatment are unlikely to represent isolated effects in specific brain regions but rather reflect functional reorganization across multiple key brain networks. Based on current structural and functional imaging evidence, the potential mechanisms underlying treatment-related neuroimaging changes may primarily involve the restoration of prefrontal–limbic regulatory control, suppression of excessive self-referential processing within the default mode network (DMN), and improvement in the functioning of reward-processing networks (Verdijk et al., 2024; Wang et al., 2023).

Restoration of prefrontal–limbic regulatory control is considered one of the key neural mechanisms underlying the therapeutic alleviation of suicidal ideation. Numerous studies have shown that patients with MDD-SI commonly exhibit reduced regulatory control of the prefrontal cortex over the limbic system at baseline, manifested by decreased activity in the dorsolateral prefrontal cortex (DLPFC) and ventromedial prefrontal cortex (vmPFC) (Kim et al., 2017), along with exaggerated amygdala responses to negative emotional stimuli (Victor et al., 2010). Following ECT and other biological treatments, increased prefrontal activity accompanied by enhanced functional connectivity between the prefrontal cortex and the amygdala has been observed, suggesting a restoration of top-down emotional regulation (Redlich et al., 2017). The enhancement of this regulatory control may help suppress negative emotional responses and impulsive behaviors, thereby reducing the persistence of suicide-related thoughts at the neural level.

Suppression of excessive self-referential

processing within the default mode network (DMN) may represent another important mechanism underlying the alleviation of suicidal ideation. The DMN is primarily involved in self-referential processing and internally oriented cognition, and in patients with MDD-SI it often exhibits hyperactivity and increased internal connectivity, which have been closely associated with rumination, negative self-evaluation, and pessimistic expectations about the future (Hamilton et al., 2015). Longitudinal neuroimaging studies have shown that, following ECT or rapid-acting treatments, patterns of internal DMN connectivity undergo partial normalization (Verdijk et al., 2024), accompanied by enhanced functional anticorrelation between the DMN and the prefrontal control network (Moreno-Ortega et al., 2019). Such network-level functional reorganization may help reduce excessive self-focus and repetitive negative thinking, thereby attenuating the persistent cognitive activation of suicidal ideation.

Improvement in reward-processing network function may also contribute to treatment-related neuroimaging changes. Patients with MDD-SI often exhibit reduced responsiveness to reward-related stimuli in the ventral striatum and orbitofrontal cortex, indicating impairments in hedonic processing and value evaluation (Jollant et al., 2011). Following effective treatment, some studies have observed increased responsiveness in reward-related brain regions as well as improved functional connectivity within prefrontal–striatal circuits (T. Zhang et al., 2021). These changes may reflect the restoration of positive emotional processing, thereby alleviating feelings of hopelessness and negative expectations about the future to some extent.

Overall, treatment-related neuroimaging changes may reflect coordinated reorganization among the emotion regulation network, the default mode network (DMN), and reward-processing networks. In particular, enhanced prefrontal–limbic regulatory control, suppression of excessive self-referential processing within the DMN, and restoration of reward-processing function may jointly constitute key neural mechanisms underlying the alleviation of suicidal ideation. These findings further suggest that different biological treatments may exert their therapeutic effects by modulating common core brain network pathways, thereby facilitating the reduction of suicidal ideation at the neural level.

4. Limitations and Future Directions

Although neuroimaging studies have made important progress in elucidating the neural mechanisms underlying major depressive disorder with suicidal ideation in recent years, several limitations remain in the existing literature. First, many studies are characterized by relatively small sample sizes and predominantly cross-sectional designs, which to some extent limit causal interpretations of neuroimaging findings. Although some longitudinal studies have begun to examine treatment-related structural and functional changes, research specifically focusing on changes in suicidal ideation as the primary outcome remains relatively limited.

In addition, substantial heterogeneity exists across studies in terms of suicidal ideation assessment methods, imaging acquisition parameters, and analytical strategies. For example, some studies employ dedicated suicidal ideation scales, such as the Beck Scale for Suicide Ideation (BSSI) (Beck et al., 1979) or the Columbia–Suicide Severity Rating Scale (C-SSRS) (Posner et al., 2011), whereas others rely on suicide-related items embedded within depression rating scales (Guo et al., 2024). Such methodological differences may affect the comparability of findings across studies. Furthermore, different imaging analysis approaches—such as voxel-based morphometry (VBM), cortical thickness analysis, or functional connectivity analysis—may also contribute to variability in the interpretation of results.

At present, most studies focus on a single imaging modality, while multimodal neuroimaging integration remains relatively limited. Combining multiple imaging techniques, including structural MRI, functional MRI, and diffusion tensor imaging (DTI), may provide a more comprehensive understanding of abnormal brain network patterns in MDD-SI and their treatment-related changes.

Future studies should further conduct large-sample, multicenter longitudinal investigations and integrate multimodal neuroimaging with machine learning approaches to identify imaging biomarkers capable of predicting treatment response in suicidal ideation. In addition, comparative studies examining the network-level regulatory mechanisms underlying different treatment modalities, such as

electroconvulsive therapy (ECT) and rapid-acting antidepressant interventions, may further deepen our understanding of the neural mechanisms of suicidal ideation and provide a basis for the development of personalized intervention strategies.

5. Conclusion and Future Perspectives

In summary, major depressive disorder with suicidal ideation (MDD-SI) exhibits relatively consistent and clinically meaningful abnormalities at both structural and functional brain levels. Increasing longitudinal neuroimaging evidence indicates that biological treatments—particularly electroconvulsive therapy (ECT)—not only rapidly alleviate suicidal ideation but also induce structural and functional plasticity within the prefrontal–cingulate–limbic system and related brain networks. These treatment-related neuroimaging findings provide important insights into the neural mechanisms underlying suicidal ideation and further suggest that brain network–level plasticity may represent a critical link between biological treatments and clinical symptom improvement. Moreover, these findings highlight the potential value of neuroimaging markers in predicting treatment response, guiding personalized interventions, and validating underlying neurobiological mechanisms in future research. Based on the current evidence, this review proposes an integrative conceptual framework of "baseline structural abnormalities—treatment-induced neuroplasticity—alleviation of suicidal ideation," which can be summarized in the following three aspects:

Structural Vulnerability Network Hypothesis

Patients with MDD-SI exhibit baseline structural abnormalities centered on the prefrontal–cingulate–limbic system, primarily manifested as reduced gray matter in regions such as the anterior cingulate cortex (ACC), ventromedial/orbitofrontal prefrontal cortex (vmPFC/OFC), and hippocampus. This "structural vulnerability" may provide a neuroanatomical basis for enhanced negative self-referential processing, impaired impulse control, and the development of hopelessness.

Biological Treatment–Induced Plasticity Hypothesis

Biological treatments may act on these vulnerability-related networks and induce regionally selective structural recovery—such as increased hippocampal volume and improvements in gray matter in the ACC and prefrontal cortex—while simultaneously promoting network-level functional reorganization. These changes may be characterized by enhanced top-down regulatory control within the prefrontal–limbic system and suppression of excessive self-referential processing within the default mode network (DMN).

Neuroimaging Mediation Hypothesis

The structural and functional imaging changes observed after treatment may not simply represent accompanying phenomena but may instead serve as mediators linking ECT to improvements in suicidal ideation, thereby constituting a critical bridge between neural modulation and clinical benefit.

Overall, these conclusions and hypotheses provide a testable theoretical framework for future studies focusing on suicidal ideation as a primary outcome, particularly those integrating longitudinal multimodal neuroimaging approaches to investigate underlying mechanisms and identify predictive biomarkers.

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