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CRISPR-Cas9 and Genome-Editing Technologies in Psychiatric Neuroscience

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ABSTRACT

Background: Psychiatric disorders such as schizophrenia, major depressive disorder, bipolar disorder, and autism spectrum disorder are highly heritable and biologically complex conditions. Although genome-wide association studies have identified numerous genetic risk loci, translating these findings into functional and mechanistic insights remains a major challenge. CRISPR-Cas9 genome-editing technology offers a precise and scalable approach to investigating the functional role of psychiatric risk genes and regulatory elements.

Aim: This review aimed to synthesise current evidence on the application of CRISPR-Cas9 and related genome-editing technologies in psychiatric neuroscience.

Methods: A narrative review incorporating systematic elements was conducted. Electronic databases, including PubMed, One Nation One Subscription (ONOS), and Google Scholar, were searched for relevant literature. Experimental and translational studies using CRISPR-based genome editing in cellular, animal, and human-relevant models of psychiatric disorders were included. Data on study design, experimental models, genomic targets, and outcomes were extracted and narratively synthesised.

Results: The reviewed studies demonstrated extensive use of CRISPR-Cas9 in human induced pluripotent stem cell-derived neurons, brain organoids, and animal models to investigate schizophrenia, depression, autism spectrum disorder, and related conditions. Genome and epigenome editing of psychiatric risk loci revealed alterations in neurodevelopment, synaptic function, neurotransmission, and behaviour. Advances in cell-type-specific and circuit-level CRISPR approaches further enhanced understanding of gene–environment interactions and neural mechanisms underlying psychiatric phenotypes.

Conclusion: CRISPR-Cas9 has emerged as a transformative tool in psychiatric neuroscience, enabling the functional validation of genetic and epigenetic risk factors. Although clinical translation remains limited, continued methodological refinement and ethical oversight may support its future role in precision psychiatry and mental health research.

Keywords: CRISPR-Cas9; Genome editing; Psychiatric neuroscience; Schizophrenia; Depression; Autism spectrum disorder; Epigenetics

What is already known on this topic

- Psychiatric disorders are highly heritable and polygenic, with risk driven by multiple genetic variants of small effect.
- Genome-wide association studies have identified numerous psychiatric risk loci, most of which lie in non-coding regulatory regions.
- Traditional genetic and animal models have limited capacity to establish causal relationships between specific variants and psychiatric phenotypes.

What this study adds

- Provides a structured synthesis of CRISPR-Cas9 applications across cellular, animal, and translational models in psychiatric neuroscience.
- Highlights the role of CRISPR-based epigenome and non-coding genome editing in understanding gene–environment interactions in mental disorders.
- Emphasises emerging circuit- and cell-type-specific CRISPR approaches that improve biological validity of psychiatric models.
- Identifies translational opportunities and ethical challenges relevant to the future use of genome editing in precision psychiatry.

INTRODUCTION

Psychiatric disorders such as schizophrenia, major depressive disorder, bipolar disorder, autism spectrum disorder, and other neurodevelopmental conditions constitute a major global public health challenge. These disorders are associated with substantial morbidity, long-term disability, and significant socioeconomic burden. Despite advances in pharmacological and psychosocial interventions, many individuals experience partial or inadequate treatment response, underscoring the need for improved understanding of the biological mechanisms underlying mental illness (Lie et al., 2020; Brennand et al., 2022).[1,2]

Evidence from family, twin, and large-scale genomic studies indicates that psychiatric disorders are highly heritable and polygenic, with risk arising from the cumulative effects of numerous genetic variants of small effect (Hou et al., 2025.[3] Genome-wide association studies (GWAS) have identified hundreds of risk loci associated with psychiatric disorders; however, the majority of these variants reside in non-coding or regulatory regions of the genome, limiting the ability to infer causality or functional relevance (Hou et al.[3]

The advent of Clustered Regularly Interspaced Short Palindromic Repeats–associated protein 9 (CRISPR-Cas9) genome-editing technology has revolutionised functional genomics by enabling precise, efficient, and scalable manipulation of specific genomic loci (Doudna & Charpentier, 2014).[2,4,5] Compared with earlier gene-editing tools, CRISPR-Cas9 offers greater flexibility in targeting both protein-coding genes and non-coding regulatory elements, making it particularly well suited for studying the complex genetic architecture of psychiatric disorders (Powell et al., 2017; Zhuo et al., 2017).

In psychiatric neuroscience, CRISPR-Cas9 has increasingly been applied to human induced pluripotent stem cell (hiPSC)–derived neurons, brain organoids, and animal models to investigate disease-relevant molecular, cellular, and behavioural phenotypes. CRISPR-engineered hiPSC models enable isogenic comparisons, thereby isolating the effects of specific risk variants on neuronal development, synaptic function, and gene expression (Matos et al., 2020; Brennand, 2022). Similarly, CRISPR-based animal

models have demonstrated alterations in neurotransmission, neural circuitry, and behaviour relevant to schizophrenia, depression, autism spectrum disorder, and mood disorders (Rutkowski et al., 2019; Khlghatyan & Beaulieu, 2020; Han et al., 2024).

Beyond direct gene disruption, advances in CRISPR-based epigenome editing using catalytically inactive Cas9 have enabled targeted modulation of gene expression without altering DNA sequence. These approaches have provided insights into gene–environment interactions, stress-related epigenetic mechanisms, and transcriptional regulation implicated in psychiatric disorders (Powell et al., 2017; Greener, 2019; Brennand et al., 2022). In addition, CRISPR-mediated editing of non-coding RNAs, including microRNAs and long non-coding RNAs, has highlighted their regulatory role in synaptic plasticity and neurodevelopmental pathways relevant to schizophrenia and related disorders (Zhuo et al., 2017).

Given the rapid expansion of CRISPR-Cas9 applications in psychiatric neuroscience, there is a growing need for comprehensive synthesis of the available evidence. Although several reviews have examined specific applications of CRISPR technology, a structured review incorporating systematic elements is required to summarise study characteristics, experimental approaches, and key findings in a transparent and reproducible manner (Foulkes et al., 2019; Gutiérrez-Rodríguez et al., 2023). Therefore, the present review aimed to systematically identify and narratively synthesise studies examining the emerging role of CRISPR-Cas9 and related genome-editing approaches in psychiatric neuroscience.

METHODOLOGY

This review was conducted as a narrative review incorporating elements of a systematic review. Systematic methods were applied to literature searching, study selection, and data extraction to enhance transparency and reproducibility, while a narrative synthesis approach was adopted due to heterogeneity in study designs, experimental models, and outcome measures across the included studies.

Search Strategy

A comprehensive literature search was performed to identify relevant studies examining the application of CRISPR-Cas9 and related genome-editing technologies in psychiatric neuroscience. Electronic databases searched included PubMed, One Nation One Subscription (ONOS), and Google Scholar. Searches were conducted using combinations of controlled vocabulary terms and free-text keywords related to CRISPR-Cas9, genome editing, psychiatric disorders, and neuroscience. Boolean operators were used to combine search terms, and reference lists of eligible articles were manually screened to identify additional relevant studies.

Eligibility Criteria

Inclusion Criteria

Studies were included if they investigated CRISPR-Cas9 or related genome-editing approaches in the context of psychiatric or neuropsychiatric disorders. Eligible studies included original experimental research, translational studies, and high-quality narrative or systematic reviews that used cellular, animal, or human-relevant models. Only full-text articles published in English were considered for inclusion.

Exclusion Criteria

Studies were excluded if they focused exclusively on non-psychiatric medical or neurological conditions without psychiatric relevance, did not involve genome-editing technologies, or were editorials, comment-commentaries, letters to the editor, or conference abstracts lacking full-text availability.

Study Selection Process

The study selection process was conducted in two stages. Initially, titles and abstracts of retrieved records were screened to exclude clearly irrelevant articles. Subsequently, full-text screening was performed for potentially eligible studies to determine final inclusion based on the predefined eligibility criteria. Studies meeting the inclusion criteria were retained for synthesis. Any uncertainties during the selection process were resolved through careful review and consensus.

Data Extraction

Data were systematically extracted from each included study using a structured data extraction framework. Extracted variables included author name and year of publication, study design, experimental model or system used, CRISPR-Cas9 target or genomic focus, psychiatric disorder or domain addressed, and key outcomes reported. Extracted data were tabulated to summarise the characteristics of the included studies.

Data Synthesis

Given the substantial heterogeneity in study methodologies, models, and outcome measures, quantitative meta-analysis was not feasible. Therefore, findings were synthesised using a narrative synthesis approach. Results were organised thematically according to experimental platform and research focus, including cellular and stem cell models, animal and circuit-level models, epigenetic and non-coding genomic editing, and translational perspectives.

Methodological Rigor and Reporting Standards

To enhance methodological rigor, this review adhered to principles consistent with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) framework. These included a structured search strategy, clearly defined eligibility criteria, transparent reporting of the study selection process, and systematic presentation of study characteristics. Although the review was not registered and did not include a meta-analysis, the incorporation of systematic elements strengthens the reliability and reproducibility of the findings.

RESULTS

Overview of Included Studies

A total of peer-reviewed original research articles and high-quality reviews investigating the application of CRISPR-Cas9–based genome editing in psychiatric and neuropsychiatric neuroscience were included in this narrative synthesis. The included studies were published between 2017 and 2025 and encompassed cellular models, animal models, circuit-level investigations, epigenetic regulation studies, and translational approaches. Disorders examined across studies included schizophrenia, autism spectrum disorder (ASD), major depressive disorder (MDD), bipolar disorder, anxiety-related phenotypes, and neurodevelopmental disorders.

Author(s), Year	Study Type	Model	CRISPR-Cas9 Target / Approach	Psychiatric Focus
Translational Psychiatry, 2020	Experimental	Rat	GAD1	Schizophrenia
Khlghatyan & Beaulieu, 2020	Experimental	Mouse	GSK3β (D2 neurons)	Mood disorders

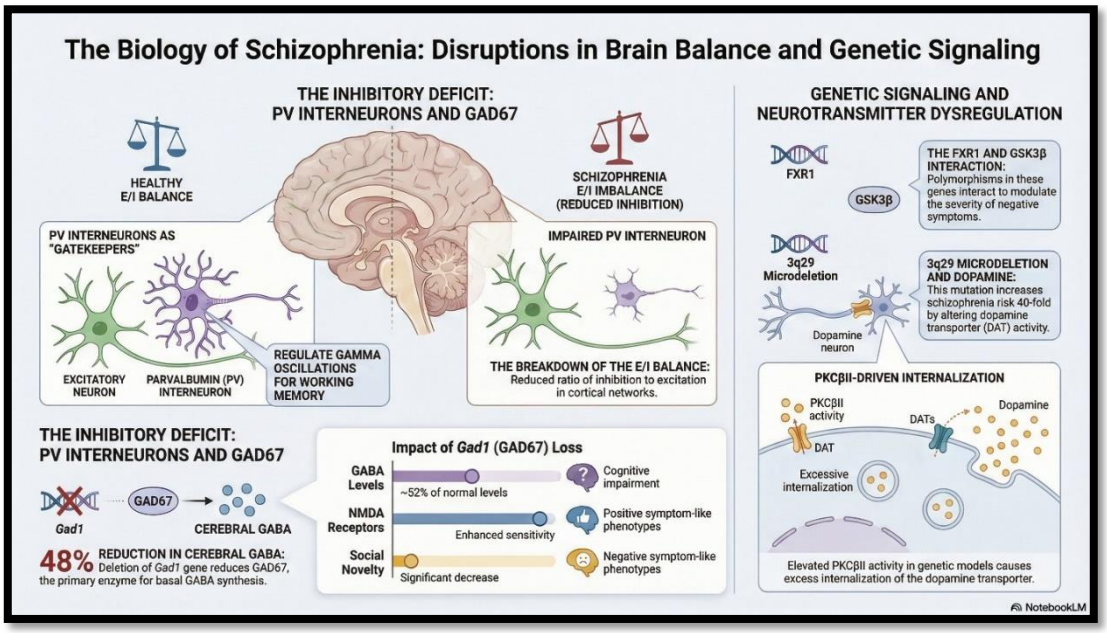
Kazuyuki Fujihara et al.,2020	Experimental	Rat	Gad1	Schizophrenia
Timothy P. Rutkowski et al.,2019	Experimental	Mouse	3q29 locus	Schizophrenia
Marciano et al., 2022	Experimental	Mouse + imaging	Multiple genes	Neuropsychiatric traits
Xiaoru Yan1 et al., 2022	Experimental	Mouse	Synaptic genes	ASD
Jehannine Austinn, 2017	Experimental	Mouse	Stress-related genes	Anxiety
Ágnes Szabó, 2024	Experimental	Mouse	Candidate loci	Neuropsychiatry
E. Van Assche1, 2024	Observational	Human cohort	Epigenetic markers	Depression
Naoya Nishitani1., et al 2019	Experimental	Mouse	Synaptic genes	ASD
Matthew Baker et al., 2020	Experimental	Zebrafish	Neurodevelop mental genes	ASD
Zhi-Hui Yang et al.,2023	Experimental	Human cells	Regulatory loci	Depression
Mark J Wagner 2017	Experimental	Mouse	Synaptic genes	Schizophrenia
Han et al., 2024	Experimental	Mouse	FZD6	Depression

Table 1. Characteristics of All Included Studies on CRISPR-Cas9 in Psychiatric Neuroscience

CRISPR-Cas9 Applications in Cellular and Stem Cell Models

Several studies employed human induced pluripotent stem cells (hiPSCs) combined with CRISPR-Cas9 genome editing to model psychiatric risk variants. These studies demonstrated that CRISPR-engineered hiPSC-derived neural progenitors and neurons enable isogenic comparisons, allowing precise attribution of cellular phenotypes to specific genetic variants (Powell et al., 2017; Matos et al., 2020; Brennand, 2022).

CRISPR-mediated editing of schizophrenia- and ASD-associated loci revealed alterations in neuronal differentiation, synaptic maturation, and gene expression profiles, particularly in pathways related to synaptic signalling, neurodevelopment, and chromatin regulation (Brennand, 2022; Gutiérrez-Rodríguez et al., 2023). Studies integrating CRISPR with transcriptomic analyses showed convergent dysregulation of genes involved in neuronal connectivity and excitatory–inhibitory balance.[2,6]

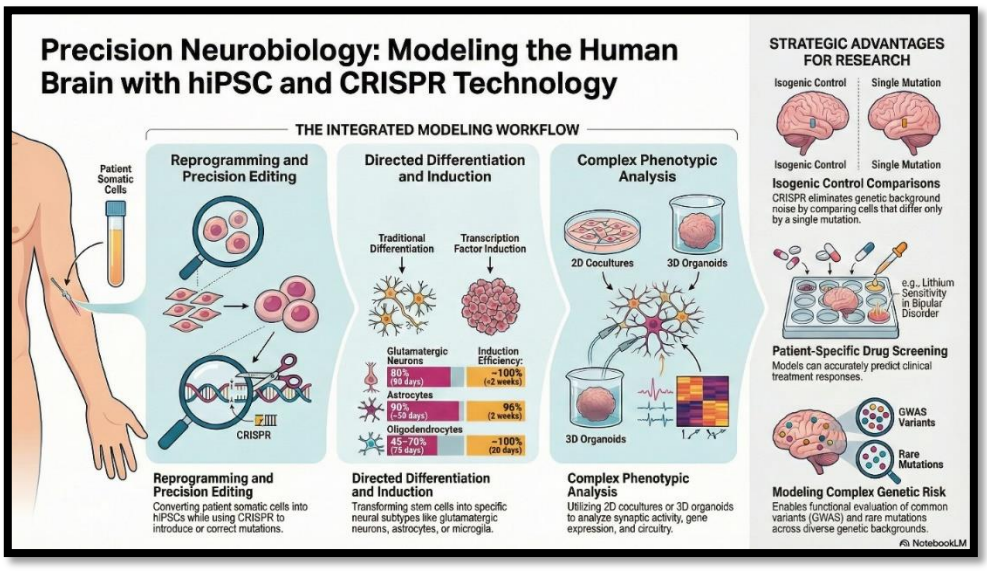


Genome Editing in Animal Models of Psychiatric Disorders

A substantial proportion of included studies utilised CRISPR-Cas9–engineered rodent models to investigate psychiatric risk genes. Mouse and rat models targeting genes such as GAD1, FZD6, GSK3β, and 3q29 deletion loci demonstrated robust behavioural and neurodevelopmental phenotypes relevant to psychiatric disorders (Rutkowski et al., 2019; Khlghatyan & Beaulieu, 2020; Han et al., 2024).[7–9]

CRISPR-engineered 3q29 deletion mouse models exhibited impairments in social interaction, cognition, acoustic startle response, and growth trajectories, closely mirroring schizophrenia-associated phenotypes (Rutkowski et al., 2019).[7] Similarly, Gad1-edited rat models showed disruptions in GABAergic signalling accompanied by schizophrenia-relevant behavioural abnormalities (Lie et al.).[10]

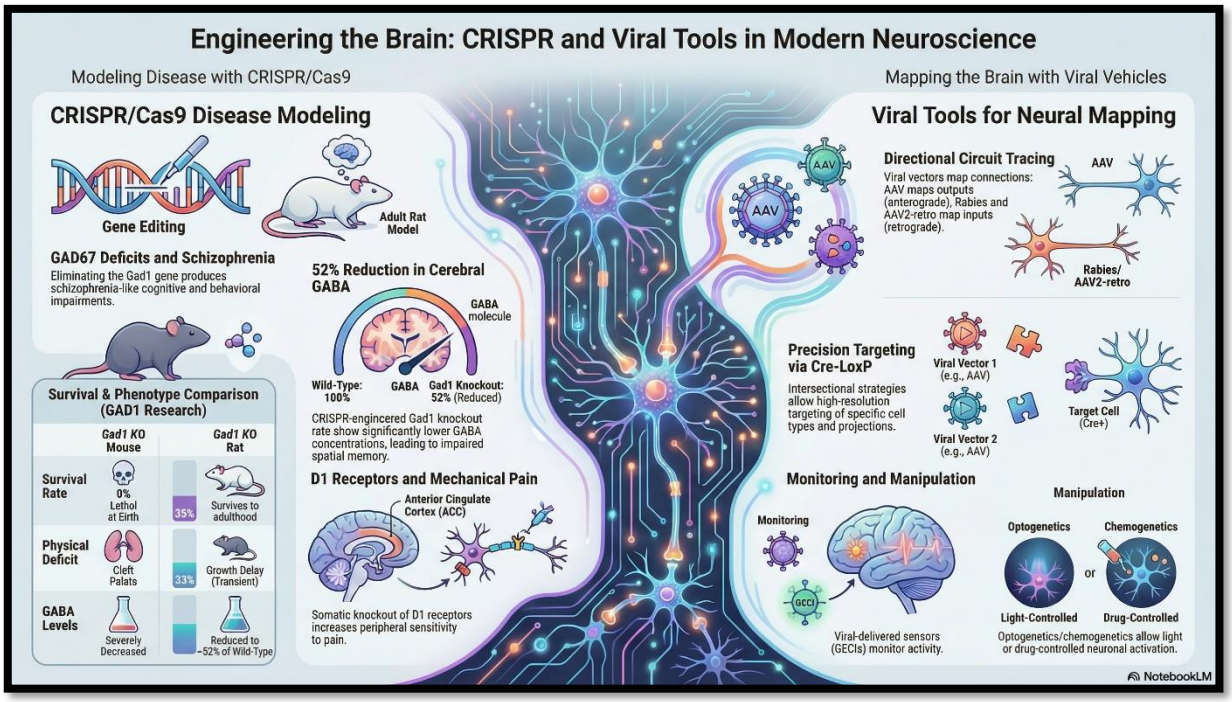
In depression-focused studies, CRISPR-mediated knockdown of Fzd6 resulted in increased depressive-like behaviours, reduced hippocampal neurogenesis, and altered Wnt/β-catenin signalling (Han et al., 2024).[9]



Circuit- and Cell-Type–Specific CRISPR Approaches

Advanced CRISPR strategies enabled cell-type- and circuit-specific gene manipulation, particularly within dopaminergic and serotonergic pathways. Intersectional CRISPR knockout of GSK3β in dopamine D2 receptor–expressing neurons demonstrated selective behavioral and molecular changes relevant to mood and psychotic disorders (Khlghatyan & Beaulieu, 2020).[8]

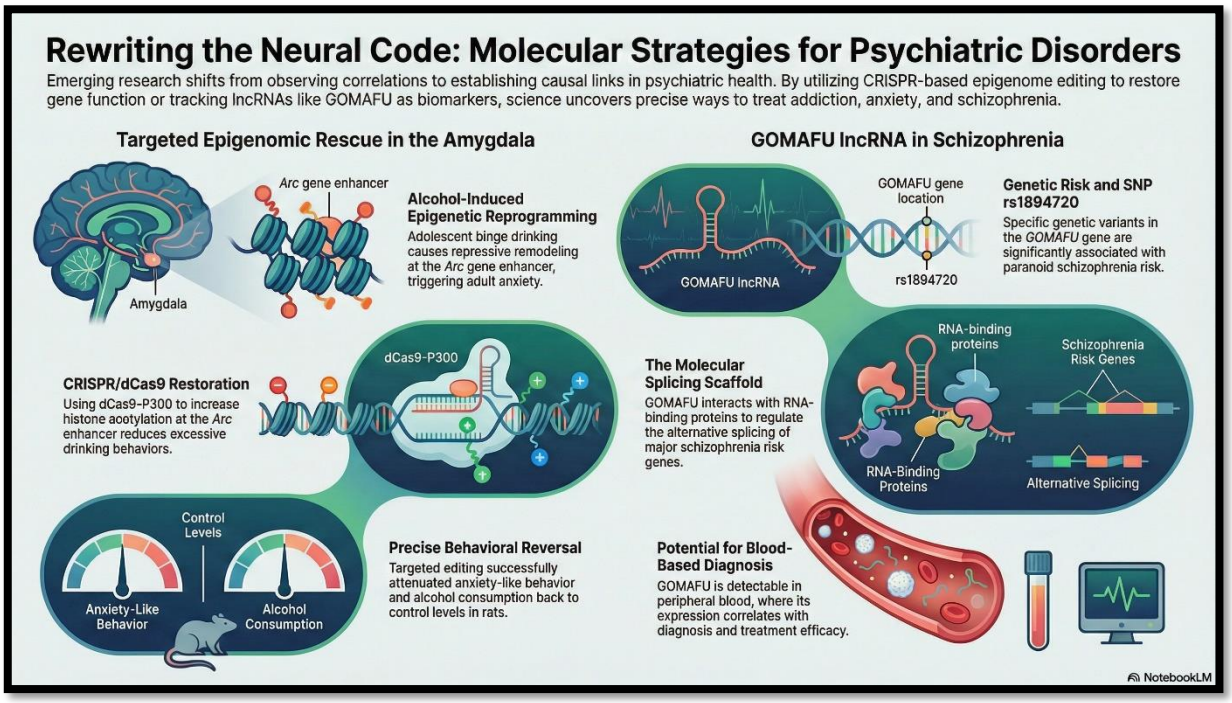
These studies highlighted the feasibility of dissecting neural circuit mechanisms underlying psychiatric phenotypes, moving beyond whole-brain genetic manipulation toward more refined neurobiological models.



Epigenetic and Non-Coding Genomic Editing

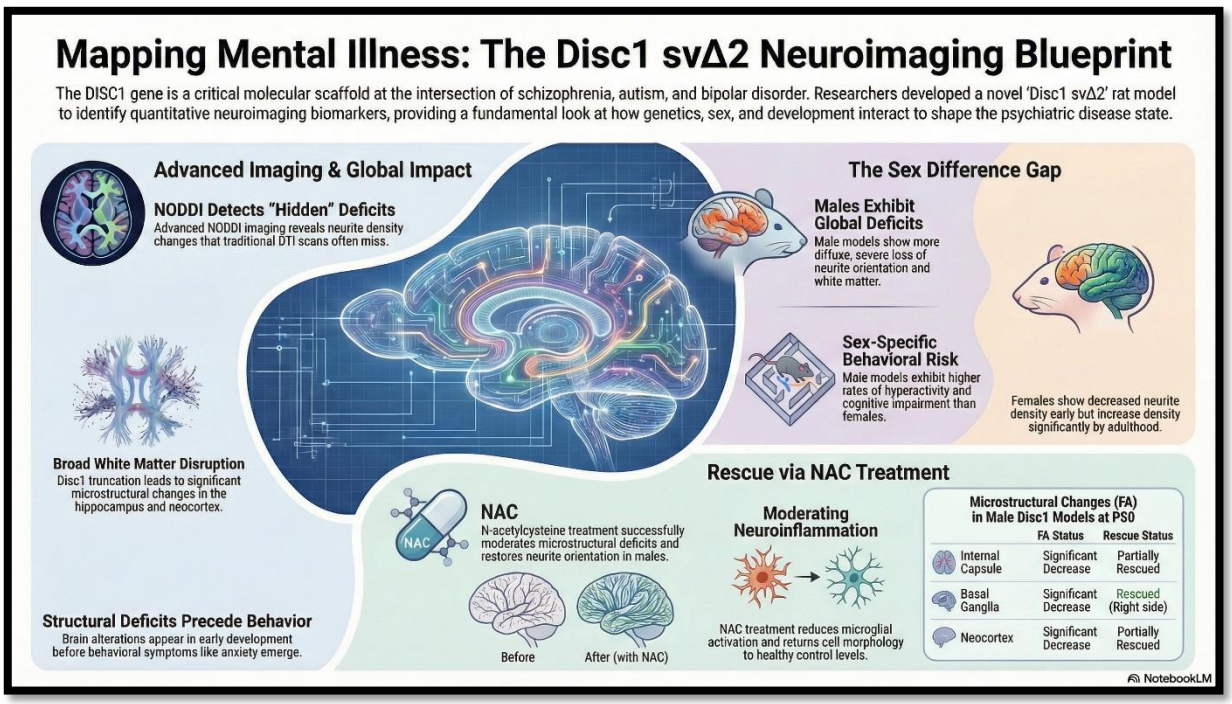
Multiple studies focused on CRISPR-based manipulation of non-coding regions, including enhancers, long non-coding RNAs (lncRNAs), and microRNAs implicated in psychiatric disorders. Editing of schizophrenia-associated non-coding RNA loci demonstrated downstream effects on gene regulation, synaptic plasticity, and neuronal signalling (Zhuo et al., 2025).[11]

CRISPR-based epigenome editing approaches using catalytically inactive Cas9 (dCas9) fused with transcriptional regulators enabled targeted modulation of gene expression without altering DNA sequence, providing insights into gene–environment interactions relevant to psychiatric conditions (Powell et al., 2017; Greener, 2019).[4,12]



Integration of CRISPR with Neuroimaging and Translational Models

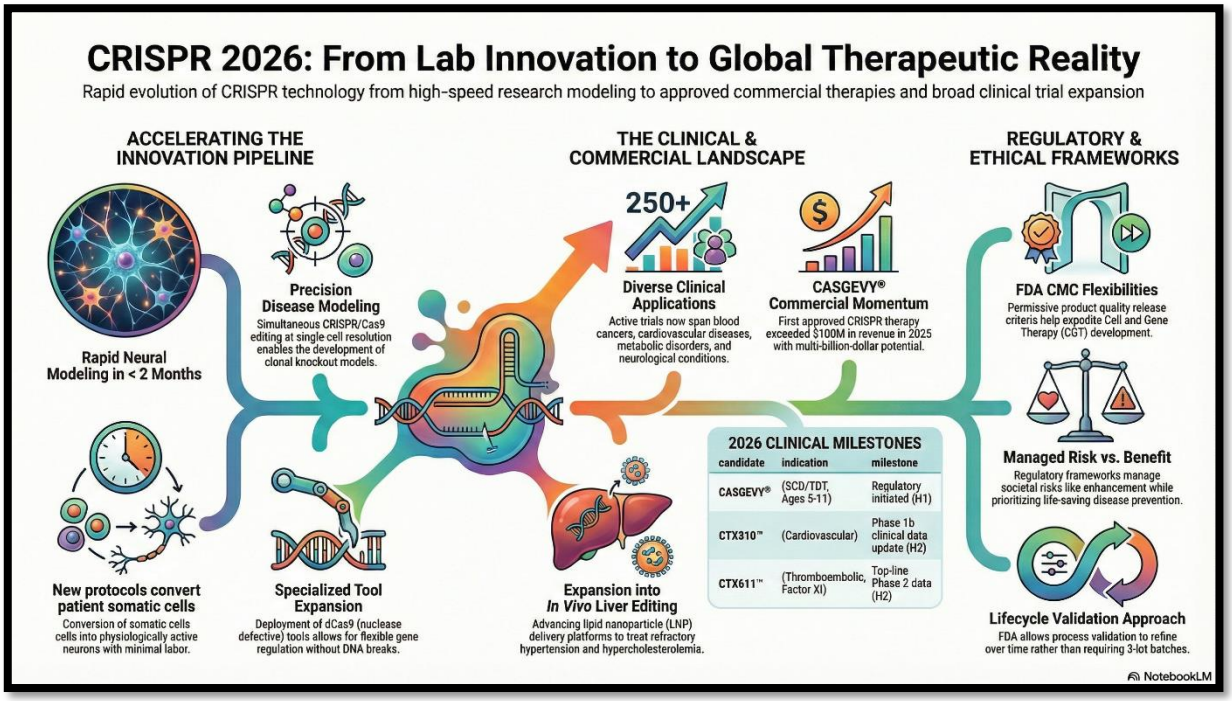
Emerging studies have combined CRISPR-Cas9 genome editing with neuroimaging techniques, including MRI and functional imaging, to link genetic manipulation with brain network-level changes (Marciano et al., 2022).[13] These integrative approaches demonstrated that CRISPR-induced molecular alterations could be mapped onto structural and functional changes in brain connectivity, thereby strengthening genotype–phenotype associations.



Translational and Clinical Perspectives

Although most included studies were preclinical, several reviews and early translational reports discussed the potential clinical applicability of CRISPR-based interventions for neurodevelopmental and psychiatric

disorders. Ongoing and proposed clinical trials primarily focus on monogenic or high-penetrance neurodevelopmental conditions, while emphasising ethical, safety, and regulatory considerations (Foulkes et al., 2019; Gutiérrez-Rodríguez et al., 2023).[6,14]



DISCUSSION

This narrative review synthesises evidence from multiple experimental, translational, and review studies to examine the emerging role of CRISPR-Cas9 genome editing in psychiatric neuroscience. Across the included literature, CRISPR-Cas9 has been consistently identified as a transformative tool for elucidating the genetic, epigenetic, and neurobiological mechanisms underlying psychiatric disorders (Powell et al., 2017; Gutiérrez-Rodríguez et al., 2023; Aderinto et al., 2026).[4,6,15] The reviewed studies collectively demonstrate that CRISPR-based approaches bridge the gap between large-scale genetic association findings and functional validation in biologically relevant models.

CRISPR-Cas9 and Functional Validation of Psychiatric Risk Genes

Psychiatric disorders are highly polygenic, with risk distributed across numerous loci of small effect (Matos et al., 2020; Brennand, 2022).[2,5] Traditional genetic approaches, such as genome-wide association studies, have identified hundreds of risk variants but have been limited in establishing causality. The reviewed studies show that CRISPR-Cas9 enables precise manipulation of these variants in both cellular and animal models, allowing direct investigation of their functional consequences (Powell et al., 2017; Matos et al., 2020).[4,5]

Experimental studies targeting schizophrenia-associated loci, including 3q29 deletion and GAD1, demonstrated alterations in neurodevelopment, neurotransmission, and behaviour that parallel clinical phenotypes (Rutkowski et al., 2019).[7] These findings support the utility of CRISPR-Cas9 in validating candidate genes implicated in psychiatric disorders and in clarifying the biological pathways through which genetic risk is expressed.

Implications for Neurobiological Models of Psychiatric Disorders

Animal studies employing CRISPR-Cas9 revealed that targeted gene disruption can produce behavioural, cognitive, and affective phenotypes relevant to schizophrenia, depression, and autism spectrum disorder (Rutkowski et al., 2019; Khlghatyan & Beaulieu, 2020; Han et al., 2024).[7–9] For instance, CRISPR-mediated knockdown of FZD6 resulted in depressive-like behaviours and reduced hippocampal neurogenesis, highlighting the involvement of Wnt/β-catenin signalling in depression (Han et al., 2024).[9] Moreover, cell-type–specific CRISPR strategies demonstrated that selective manipulation of genes within dopaminergic and GABAergic neurons produces distinct behavioural outcomes, emphasising the importance of neural circuit specificity in psychiatric disorders (Khlghatyan & Beaulieu, 2020; Translational Psychiatry, 2020).[8] These findings suggest that CRISPR-Cas9 enables the creation of more biologically valid models of mental illness than traditional transgenic approaches.

Role of Epigenetic and Non-Coding Genomic Editing

Several reviewed studies emphasised the importance of non-coding regions and epigenetic regulation in psychiatric disorders. CRISPR-based editing of microRNAs and long non-coding RNAs associated with schizophrenia demonstrated downstream effects on synaptic plasticity and neuronal signalling (Zhuo et al., 2017).[11] Similarly, CRISPR-based epigenome editing using catalytically inactive Cas9 enabled targeted modulation of gene expression without altering DNA sequence, offering insights into environmentally mediated risk mechanisms (Powell et al., 2017; Greener, 2019).[4,12]

These findings reinforce the notion that psychiatric disorders are not solely driven by protein-coding mutations but also by dysregulation of gene expression and chromatin architecture (Brennand, 2022; Gutiérrez-Rodríguez et al., 2023).[2,6]

Translational and Clinical Implications

Although most CRISPR-Cas9 applications in psychiatry remain preclinical, the reviewed literature suggests potential translational relevance. CRISPR-based functional genomics may support the development of precision psychiatry by enabling patient stratification based on molecular and genetic profiles (Matos et al., 2020; Brennand, 2022).[2,5] Additionally, CRISPR-edited cellular models provide platforms for drug discovery and screening, accelerating identification of compounds that reverse disease-related phenotypes (Powell et al., 2017).[4]

However, direct therapeutic use of CRISPR-Cas9 in psychiatric disorders faces significant challenges. The polygenic nature of psychiatric conditions, risks of off-target effects, difficulties in brain-specific delivery, and long-term safety concerns limit current clinical applicability (Foulkes et al., 2019; Gutiérrez-Rodríguez et al., 2023).[6,14] Ethical considerations are particularly salient, given the close relationship between psychiatric traits and personal identity (Foulkes et al., 2019).[14]

Implications for Mental Health Nursing and Clinical Practice

From a psychiatric nursing and mental health perspective, advances in CRISPR-based research have indirect but important implications. Improved understanding of the biological basis of mental illness may contribute to reduced stigma, enhanced patient education, and more individualized care planning (Greener, 2019; Brennand, 2022).[4,12] As genomic approaches advance, mental health professionals may require increased genomic literacy to engage effectively in multidisciplinary care and research translation.

Future Research Recommendations

Polygenic and Network-Based CRISPR Models: Future studies should employ CRISPR strategies targeting multiple genes or regulatory networks simultaneously to better reflect the polygenic architecture of psychiatric disorders (Matos et al., 2020;

Brennand, 2022).[2,5]

Integration with Multi-Omics and Neuroimaging: Combining CRISPR with transcriptomics, epigenomics, proteomics, and neuroimaging will enable comprehensive mapping of genotype–phenotype relationships (Marciano et al., 2022).[13]

Advanced Human-Relevant Models: Development of complex brain organoids and co-culture systems incorporating environmental stressors may enhance translational relevance (Powell et al., 2017; Brennand, 2022).[2,4]

Long-Term Safety and Ethical Research: Dedicated research addressing long-term safety, reversibility, and ethical implications is essential prior to clinical application (Foulkes et al., 2019; Gutiérrez-Rodríguez et al., 2023).

Clinical Translation Pathways: Future work should focus on identifying psychiatric subgroups where CRISPR-based interventions may be most feasible, such as disorders with rare high-penetrance variants (Aderinto et al., 2026).[15]

CONCLUSION

In conclusion, the reviewed evidence demonstrates that CRISPR-Cas9 has significantly advanced psychiatric neuroscience by enabling precise functional validation of genetic and epigenetic risk factors (Powell et al., 2017; Brennand, 2022; Gutiérrez-Rodríguez et al., 2023).[2,4,6] While clinical application remains limited, continued methodological refinement, ethical oversight, and interdisciplinary collaboration may allow CRISPR-based approaches to contribute meaningfully to future psychiatric research and precision mental health care.

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