

MicroRNA-137 in Schizophrenia: A Bioinformatics-Based Review of a Genetic Locus and Regulatory Candidate

Ansh Yadawad¹, Rachel Gunraj²

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Schizophrenia is a serious mental health disorder that affects thinking, emotions, and behavior, but it is often diagnosed only after symptoms appear. Bioinformatics offers a way to study large genetic datasets and identify genetic markers that may help researchers better understand schizophrenia risk. In this research paper, the National Human Genome Research Institute–European Bioinformatics Institute Genome-Wide Association Studies Catalog (NHGRI–EBI GWAS Catalog) was searched using the term “schizophrenia”. The catalog collates the results of genome-wide association studies, which identify genetic variants contributing to diseases by comparing the DNA of those affected with specific conditions against the DNA of the general population. From this search, three frequently reported genes or loci were identified: *CACNA1C*, *GRIN2A*, and *MIR137HG*. These genes were chosen because they appeared in schizophrenia related GWAS records and because they are connected to brain processes that may matter in schizophrenia. *CACNA1C* is involved in calcium signaling, *GRIN2A* is involved in NMDA/glutamate receptor signaling, and *MIR137HG* is connected to *miR-137*, which helps regulate genes during brain development. *MIR137HG* is a GWAS-associated genomic locus linked to *miR-137* biology, but it is not the same as mature *miR-137*, the processed *microRNA* molecule. These findings highlight the *MIR137HG/miR-137* pathway as an important area for future research. However, this study only summarizes reporting frequency and biological relevance from public databases and published literature. It does not test whether *miR-137* can diagnose, predict, or detect schizophrenia in patients.

Keywords: schizophrenia; *miR-137*; bioinformatics; genome-wide association studies; GWAS; *CACNA1C*; *GRIN2A*; genetic loci; reporting frequency.

Introduction

Schizophrenia is a severe mental health disorder that affects approximately 23 million people worldwide¹, or about 1 in 345 people globally and 1 in 233 adults, according to the World Health Organization¹. Although it is less common than many other mental disorders, schizophrenia can cause major disruptions in thinking, emotions, behavior, education, work, and daily functioning.

Common symptoms include hallucinations, delusions, memory difficulties, and impaired concentration². The disorder typically begins in late adolescence or early adulthood, a critical period when the brain is still maturing³. Although early treatment can improve long term outcomes, identifying schizophrenia before clear symptoms appear remains difficult^{4–6}. Early warning signs, such as sleep changes or academic decline, are often dismissed as normal teenage stress.

Schizophrenia has a strong genetic component, with studies estimating that genetics may explain about 70 to 80% of the risk⁷. However, schizophrenia is not caused by one sin-

gle gene or mutation. Instead, many different genetic variants, each with a small effect, can add together and increase a person’s overall risk⁸. Since these risk factors are spread across the genome, researchers need large genetic datasets and computational tools to identify patterns that may be linked to schizophrenia⁸. Because schizophrenia has a strong genetic component and involves many small genetic risk factors, researchers use bioinformatics to study large genetic datasets and identify patterns that may be linked to disease risk. This field makes it possible to analyze huge amounts of genetic data and find patterns that would be impossible to see by hand. Because schizophrenia involves many genetic risk factors, researchers use GWAS and bioinformatics to analyze large datasets and identify recurring schizophrenia associated loci^{9–13}. These tools are useful for schizophrenia research because they can help identify repeated genetic signals across large datasets. Bioinformatics can scan thousands of genetic variations at once and reveal subtle patterns associated with disease^{9–13}.

Scientists are focusing on using bioinformatics to identify biomarkers, which are measurable signals in the body that can reveal the presence or likelihood of disease. Biomark-

¹ Westford Academy, Westford, MA, 01886

² University of Florida, USA

ers can appear in many forms, such as molecular signals, protein patterns, or imaging features in the brain¹⁴. Biomarkers are measurable biological signals that can help researchers study disease processes. However, biomarkers for schizophrenia are still much less established than biomarkers for some other neurological diseases. One recent study identified possible blood biomarkers for schizophrenia, but more research is needed to confirm these findings¹⁵. Therefore, this study uses bioinformatics only to explore genetic signals that may be useful for future research, not to identify a validated clinical test.

Some genes and genomic loci linked to schizophrenia are important because they influence how brain cells develop and communicate. *CACNA1C* is involved in neuronal calcium channel signaling^{16,17}. *GRIN2A* encodes a subunit of the NMDA glutamate receptor and contributes to neuronal signaling and synaptic function¹⁸. *MIR137HG* is a genomic locus connected to *miR-137* expression¹⁹. Mature *miR-137* is a regulatory *microRNA*²⁰. Studies have linked it to neuronal maturation and synaptic processes^{21–23}. These genes and loci were selected because they appeared in schizophrenia related GWAS records and because published studies connect them to biological processes relevant to schizophrenia.

Bioinformatics offers new ways to identify genetic markers of schizophrenia. Large GWAS can analyze thousands of genetic variants and identify schizophrenia associated loci across large populations^{9–13}. *miR-137* is of interest because experimental studies have linked it to neuronal maturation, presynaptic plasticity, and glutamatergic signaling^{21–23}. Recognizing these repeated genetic signals will provide a good basis on which to explore how bioinformatics may study genes that increase the risk of schizophrenia^{8,9,11–13}. The objective of this study is to use publicly available genome wide association data to identify genes that are frequently reported in schizophrenia related studies and to examine their possible biological importance using published scientific literature. This study does not prove that these genes can diagnose or predict schizophrenia, but it may help point to genetic signals that could be useful for future research on schizophrenia risk and biological pathways involved in the disorder^{9,21}.

Materials and Methods

This study used publicly available genetic data and published research articles to examine genes and genetic regions connected to schizophrenia. No patient samples, private medical records, or personal information were used.

GWAS Catalog Data Extraction and Ranking Procedure

The NHGRI-EBI GWAS Catalog file used in this study was downloaded on May 26, 2026. The search was for schizophrenia, identified by MONDO:0005090, and included

child traits. The downloaded file was named “gwas-association-downloaded_2026-05-26-MONDO_0005090-withChildTraits.tsv.” No additional disease filters were applied after the file was downloaded.

The file included information such as disease or trait, mapped gene, reported gene, PubMed ID, study title, p-value, and study accession number.

Data Cleaning and Gene Mapping Rules

The original file contained 6,509 association records. Twenty rows that were completely identical were removed, leaving 6,489 records. Records with a blank MAPPED.GENE field were also removed. After this step, 6,409 association records remained for the analysis.

Repeated PubMed IDs and study accession numbers were kept when the records represented different genetic associations. This was done because the study counted association records rather than only counting unique research papers.

The MAPPED.GENE column was used for the frequency count. When a cell contained several entries separated by commas, each comma-separated entry was placed in its own row. If the same gene name appeared more than once in one association record, it was counted only once for that record.

Hyphenated entries, such as *TMF1P1–ERCC4* and *DRD2–TMPRSS5*, were kept together as single mapped locus entries. They were not separated into individual genes. After the data were cleaned and organized, there were 6,862 mapped gene or locus occurrences representing 2,834 unique entries.

Frequency and Ranking

The mapped gene and locus entries were counted using a Microsoft Excel PivotTable. The entries were then ranked from the highest to the lowest reporting frequency.

Reporting frequency means the number of GWAS Catalog association records in which a gene or locus appeared. A higher count did not necessarily mean that a gene had a stronger biological effect or a stronger connection to schizophrenia. The ranking was not used to measure effect size, statistical significance, causation, independent replication, or diagnostic value.

Selection of Genes and Loci

The frequency ranking was used as a starting point for choosing genes and loci for closer discussion. *CACNA1C* was selected because it had the highest reporting frequency in this analysis. *MIR137HG* and *GRIN2A* were also selected because they appeared in the GWAS Catalog records and because published studies connect them to brain processes related to schizophrenia.

The three genes or loci were therefore selected based on both reporting frequency and biological relevance. Their selection does not mean that they are the main causes of schizophrenia or that they can be used as diagnostic biomarkers.

Review of Published Studies

Peer reviewed studies were reviewed to better understand the biological roles of *CACNA1C*, *GRIN2A*, *MIR137HG*, and mature *miR-137*. The review focused on processes related to schizophrenia, including calcium signaling, glutamate-receptor function, synaptic plasticity, brain development, and gene regulation.

The published studies were used to explain the biological importance of the selected genes and loci. They were not used to calculate the frequency counts or to test diagnostic accuracy.

Results

To look at genetic markers related to schizophrenia, the NHGRI-EBI GWAS Catalog results were reviewed and the mapped genes were counted based on how often they appeared. This was done because schizophrenia is not usually caused by one single gene, but instead involves many genetic factors^{8,9,11–13}. Table 1 shows the top 10 mapped genes from the GWAS Catalog search.

Table 1 Top 10 mapped genes by reporting frequency in schizophrenia-related GWAS Catalog records

Rank	Mapped Gene	Reporting Frequency
1	<i>CACNA1C</i>	44
2	<i>LINC01470</i>	34
3	<i>MAD1L1</i>	32
4	<i>TCF4</i>	31
5	<i>TSNARE1</i>	26
6	<i>CACNB2</i>	26
7	<i>TMF1P1 - ERCC4</i>	24
8	<i>IMMP2L</i>	23
9	<i>MPHOSPH9</i>	22
10	<i>DRD2 - TMPRSS5</i>	22

Some genes had the same reporting frequency. When there was a tie, the first 10 entries from the sorted worksheet were shown. *ZNF804A* and *CSMD1* also appeared 22 times.

Table 1 helps show how the selected genes compared with other genes in the same search results. *CACNA1C* was selected because it had the highest reporting frequency in this analysis. *MIR137HG* and *GRIN2A* were also selected for

closer discussion because they appeared in the GWAS Catalog records and are connected to schizophrenia-related brain pathways discussed in published research^{3,9,21}. Therefore, the three genes were not chosen only because of frequency. Reporting frequency was used as a starting point, but biological relevance was also considered.

These genes were chosen because they appeared in schizophrenia-related GWAS records and because they are connected to brain processes that may matter in schizophrenia^{3,9}. *CACNA1C* is involved in neuronal calcium-channel signaling^{16,17}, *GRIN2A* is involved in NMDA/glutamate-receptor signaling¹⁸, and *MIR137HG* is connected to *miR-137* biology and gene regulation^{19,21–23}. This means the genes were not selected only because they appeared often in the database.

Table 2 Genes and genomic locus selected for focused discussion

Mapped Gene	Reporting Frequency	Reason included
<i>CACNA1C</i>	44	Highest reporting frequency in this analysis and involved in neuronal calcium-channel signaling
<i>MIR137HG</i>	19	GWAS-associated locus connected to <i>miR-137</i> biology and gene regulation
<i>GRIN2A</i>	17	Encodes an NMDA receptor subunit involved in glutamate signaling, synaptic plasticity, learning, and memory

Table 2 shows the three genes selected for closer discussion in this paper. These numbers only show how often each gene appeared in the GWAS Catalog records. They do not prove that one gene is more important than another or that any of these genes can be used for diagnosis.

The frequency ranking was used only to help choose genes for further discussion. A higher frequency does not always mean that a gene has a stronger connection to schizophrenia. Some genes may appear more often because they have been studied more, because the same genetic region appears in multiple records, or because of how the GWAS Catalog maps genes. For this reason, the results should be understood as a summary of reporting frequency, not as proof that these genes can be used as diagnostic biomarkers. *MIR137HG* was included for closer discussion because previous research con-

nects this genomic locus and *miR-137* biology to gene regulation and schizophrenia related biological pathways^{19,21–23}.

Discussion

Interpretation of the Selected Genes and Locus

Studies show that *miR-137*, *GRIN2A*, and *CACNA1C* can each affect neuronal signaling in different ways. Smrt et al. linked *miR-137* to neuronal maturation²², while Siegert et al. found that increased *miR-137* reduced the proteins *CPLX1*, *NSF*, and *SYT1* and disrupted presynaptic plasticity²¹. In *GRIN2A*-mutant mice, researchers observed changes in glutamate and dopamine signaling¹⁸. In rats with reduced *CACNA1C* activity, researchers found reduced calcium signaling, disrupted synaptic plasticity, and learning difficulties¹⁶. Mature *miR-137* is a regulatory *microRNA*, whereas *CACNA1C* and *GRIN2A* are protein-coding genes involved in complex neuronal signaling processes. These findings support discussing them as separate schizophrenia-associated factors, but they do not establish that *miR-137* directly regulates *GRIN2A* or *CACNA1C*.

Other studies have also looked at how these genes and molecules may be connected to schizophrenia. One study found that changing *miR-137* levels in human brain cells affected genes involved in neuron development²⁴. Another study found that differences in *MIR137HG* and genes controlled by *miR-137* were linked to changes in gray matter in people with schizophrenia²⁵. In mice, lower levels of *miR-137* affected learning, social behavior, and communication between brain cells²⁶. A brain imaging study also found that a *CACNA1C* variant was linked to differences in brain activity during attention tasks²⁷. In another study, *GRIN2A*-mutant mice showed patterns of brain activity that were similar to some patterns seen in people with schizophrenia²⁸. A study of human brain tissue also found differences in *microRNA* expression in the prefrontal cortex of people with schizophrenia and schizoaffective disorder²⁹. These studies show why *miR-137*, *CACNA1C*, and *GRIN2A* are useful to study, but they do not prove that these factors directly cause schizophrenia or can be used to diagnose it.

Distinguishing *MIR137HG* from mature *miR-137*

MIR137HG and mature *miR-137* are related but not identical. *MIR137HG* refers to a genomic locus identified in GWAS records, while mature *miR-137* is the processed *microRNA* molecule that can regulate gene expression. Therefore, a GWAS association at the *MIR137HG* locus does not prove that mature *miR-137* levels are changed in blood, CSF, or other patient samples. In this study, *MIR137HG* should be interpreted as a schizophrenia-associated locus of interest, not as

evidence that mature *miR-137* is already a validated early-detection biomarker. Additional primary studies have examined the *MIR137HG/miR-137* pathway using human neural cells, postmortem brain tissue, and neuronal models. These studies reported changes in gene-expression pathways after *miR-137* manipulation, an association between a schizophrenia risk variant and *miR-137* expression in the prefrontal cortex, disrupted neurodevelopmental signaling after *miR-137* inhibition, and effects of variation within *MIR137HG* on *miR-137* transcript processing^{30–33}.

Limitations and Future Research

The findings of this study should be interpreted carefully. Reporting frequency shows how often a gene or locus appeared in the reviewed GWAS Catalog records, but it does not measure effect size, causation, diagnostic accuracy, or clinical usefulness. This study also did not examine patient samples, gene-expression levels, or clinical outcomes. Therefore, the findings cannot show whether *MIR137HG*, mature *miR-137*, *CACNA1C*, or *GRIN2A* can predict or diagnose schizophrenia.

Future research could combine GWAS findings with gene-expression studies, laboratory experiments, and clinical data to better understand how these genes and loci contribute to schizophrenia risk. Figure 1 presents a hypothetical future research workflow and does not represent the methods used in this study or a currently validated clinical process.

The “Clinical Application” stage shown in the figure is hypothetical and would require further laboratory studies, clinical testing, and validation. The present study did not analyze patient samples, test diagnostic accuracy, or evaluate treatment selection.

Clinical application would require prospective studies that directly evaluate predictive accuracy, clinical usefulness, feasibility, and ethical concerns. These questions were not examined in the present study.

Conclusion

This study used data from the NHGRI-EBI GWAS Catalog to examine genes that appeared frequently in schizophrenia-related GWAS records. Using reporting frequency as a starting point, *CACNA1C*, *GRIN2A*, and *MIR137HG* were selected for focused discussion based on both their occurrence in the GWAS Catalog records and their biological relevance to schizophrenia. The selected genes and locus are connected to biological processes relevant to schizophrenia, including calcium signaling, glutamate-receptor function, neurodevelopment, and gene regulation^{16,18,19,21,22}. However, the frequency counts used in this study should be interpreted care-

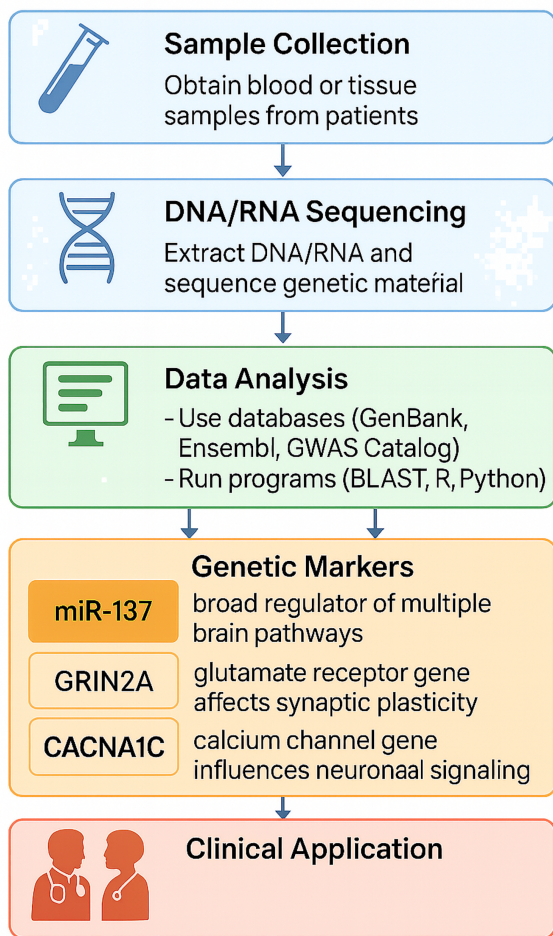


Figure. 1 Conceptual workflow showing how genetic and bioinformatics research might eventually contribute to clinical research on schizophrenia.

fully. They show how often these genes appeared in the reviewed GWAS Catalog records, but they do not show effect size, statistical strength, independent replication, or diagnostic value.

The significance of this study lies in showing how existing bioinformatics resources can be used to explore complex psychiatric disorders without generating new genetic data. *MIR137HG* remains a schizophrenia-associated genomic locus of interest^{9,11–13,19}, whereas mature *miR-137* has been linked experimentally to neuronal maturation and synaptic function^{21–23}. However, this study does not prove that *miR-137*, *CACNA1C*, or *GRIN2A* can diagnose schizophrenia or predict future illness. Instead, these findings should be viewed as a starting point for future research.

A major strength of this study is its use of a large, well-curated public database that integrates findings from many independent genetic studies, increasing the reliability of the identified associations. However, the study also has limitations. GWAS findings can show that certain genes are linked to schizophrenia, but they do not prove that those genes directly cause the disorder. Schizophrenia is influenced by many different genetic factors^{8,9,11–13}. In addition, this analysis did not examine gene expression levels or clinical outcomes, which limits how directly the findings can be applied to patient care.

Future research could build on this work by combining GWAS data with gene expression studies, functional experiments, or clinical data to better understand how *miR-137*, *CACNA1C*, and *GRIN2A* influence disease development. Further studies could also explore how these markers interact with environmental risk factors and how they may contribute to schizophrenia related biological processes. Together, these steps could help clarify how genetic loci and regulatory pathways contribute to schizophrenia risk and biology.

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