Mini-Review

Psychiatry

Are There Any Shared Genes for Psychiatric Disorders?

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Psychiatric disorders have traditionally been classified as distinct diagnostic entities based on clinical symptoms, yet growing genetic evidence challenges the notion that these conditions are biologically independent. Advances in genomics, particularly genome-wide association studies and large-scale consortia efforts, have revealed substantial genetic overlap across major psychiatric disorders such as schizophrenia, bipolar disorder, major depressive disorder, autism spectrum disorder, and attention-deficit/hyperactivity disorder. Rather than single disorder-specific genes, shared polygenic risk architectures appear to influence multiple psychiatric phenotypes, affecting neurodevelopment, synaptic function, neurotransmission, and brain plasticity. This review examines the evidence for shared genetic factors across psychiatric disorders, explores the biological pathways they implicate, and discusses how pleiotropy reshapes our understanding of psychiatric classification, diagnosis, and treatment. While shared genes do not erase meaningful clinical differences, they highlight common biological vulnerabilities that transcend traditional diagnostic boundaries and suggest a need for more integrative, dimensional models of mental illness.

Keywords: Psychiatric Genetics; Pleiotropy; Polygenic Risk; Neurodevelopment; Mental Disorders

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PSYCHIATRIC disorders have long been approached as separate clinical categories, each defined by characteristic symptom clusters, age of onset, and treatment responses. Schizophrenia, depression, bipolar disorder, autism, and attention-deficit/hyperactivity disorder have historically occupied distinct conceptual spaces within psychiatry. This categorical framework has shaped diagnostic manuals, research agendas, and therapeutic strategies for decades. However, as genetic tools have advanced, a different picture has emerged—one in which psychiatric disorders share substantial genetic underpinnings, blurring diagnostic boundaries (American Psychiatric Association, 2013; Cross-Disorder Group of the

Psychiatric Genomics Consortium, 2013).

Early genetic studies already hinted at this overlap. Family and twin studies consistently demonstrated that psychiatric disorders are heritable, but they also revealed cross-disorder familial risk. For example, relatives of individuals with schizophrenia show increased rates of bipolar disorder and major depression (Kenneth S. Kendler et al., 2003). These findings suggested that genetic liability does not map neatly onto single diagnoses but instead reflects shared inherited vulnerabilities.

The advent of genome-wide association studies (GWAS) marked a turning point. These studies demonstrated that psychiatric disorders are highly polygenic, influenced by thousands of variants of small effect (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013; Benjamin M. Neale et al., 2019). Importantly, many variants confer risk across multiple disorders, providing molecular evidence of shared genetic architecture.

Large cross-disorder analyses further confirmed this overlap. Genetic correlation studies show strong shared risk between schizophrenia and bipolar disorder, with moderate overlap with major depressive disorder (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013; Brainstorm Consortium, 2018). These findings indicate that overlapping sets of genes influence psychosis, mood regulation, and cognition.

Autism spectrum disorder and attention-deficit/hyperactivity disorder provide another example. Both are neurodevelopmental conditions with shared genetic influences affecting synaptic development and neuronal connectivity (Angelica Ronald et al., 2008; Benjamin M. Neale et al., 2010). This overlap helps explain their frequent co-occurrence and familial clustering.

The concept of pleiotropy is central to understanding shared genes. Pleiotropic variants influence multiple traits, often through fundamental biological processes such as synaptic plasticity and calcium signaling (Patrick F. Sullivan et al., 2012). Because these processes are essential to brain function, their disruption can produce diverse psychiatric outcomes.

Polygenic risk scores further illustrate shared liability. These scores aggregate genetic variants to estimate risk and have been shown to predict multiple psychiatric traits across diagnostic boundaries (Peter M. Visscher et al., 2017). For example, schizophrenia polygenic risk is associated with cognitive and mood-related traits in the general population.

At the biological level, shared genes converge on neural development and synaptic function. Genes involved in neurotransmission, synapse formation, and receptor signaling are implicated across multiple disorders (Psychiatric Genomics Consortium, 2020). This suggests that disrupted neuronal communication is a common pathway in psychiatric illness.

Gene regulation also plays a major role. Many risk variants lie in non-coding regions that regulate gene expression, particularly during brain development (ENCODE Project Consortium, 2012). These regulatory effects are context-dependent, contributing to variability in clinical presentation.

Rare variants further contribute to shared risk. Copy number variations (CNVs) have been associated with autism, schizophrenia, and other neurodevelopmental disorders (Jonathan Sebat et al., 2007). These variants often disrupt genes critical for early brain development, leading to broad phenotypic effects.

Environmental factors interact with genetic risk. Stress, trauma, and other exposures influence whether genetic vulnerability manifests as illness (Avshalom Caspi et al., 2003). This gene–environment interplay highlights the complexity of psychiatric disorders.

The recognition of shared genetic architecture has implications for classification. Traditional diagnostic systems may not reflect underlying biology, prompting dimensional approaches such as the National Institute of Mental Health Research Domain Criteria (RDoC) framework (Insel et al., 2010).

Shared genetics also influences treatment strategies. Therapies targeting common biological pathways—such as synaptic plasticity or neurotransmitter systems—may have cross-disorder benefits (Thomas R. Insel, 2014). This perspective encourages broader, mechanism-based approaches to treatment.

Clinically, shared genetic risk helps explain comorbidity. Many patients meet criteria for multiple disorders, and symptoms evolve over time. Genetic overlap provides a biological basis for this complexity and supports integrated care approaches (Sullivan et al., 2012).

Ethical considerations are also important. While shared genes indicate vulnerability, they do not determine outcomes. Responsible communication is essential to avoid stigma and deterministic interpretations.

Despite progress, limitations remain. Many studies focus on European populations, and effect sizes of individual variants are small. Understanding how genes interact within biological systems remains a major challenge (Visscher et al., 2017).

Future research will integrate genomics with neuroimaging, transcriptomics, and longitudinal data to better understand shared risk. Advances in single-cell biology and brain models may clarify how pleiotropic genes affect specific circuits.

In conclusion, psychiatric disorders share substantial genetic architecture. Rather than being driven by disorder-specific genes, they arise from overlapping polygenic risk affecting core neurobiological processes. This shared foundation explains comorbidity and challenges traditional diagnostic boundaries, offering a path toward more integrated and biologically informed psychiatry.■

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