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# UNDERSTANDING COMMON MENTAL HEALTH DISORDERS: PREVALENCE, NEUROBIOLOGY, AND SOCIODEMOGRAPHIC CORRELATES

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**ABSTRACT**
Mental health disorders affect hundreds of millions of people worldwide. This review summarizes current knowledge on the prevalence, biological mechanisms, and social patterns of major depressive disorder, anxiety disorders, bipolar disorder, and schizophrenia. Major depressive disorder affects approximately 4.4% of the global population, while anxiety disorders affect 3.6% (World Health Organization, 2017). Brain imaging studies have identified structural and functional alterations in the prefrontal cortex, amygdala, and hippocampus in these conditions (Goodkind et al., 2015). Neurotransmitter systems, particularly serotonin, dopamine, and norepinephrine, show consistent dysregulation across disorders (Hyman, 2005). Sociodemographic factors including gender, income, and adverse childhood experiences strongly influence disorder risk (Kessler et al., 2010; McLaughlin et al., 2012). This overview provides a foundation for understanding mental illness as a biological and social phenomenon without discussing treatment or intervention approaches.

**KEYWORDS:** depression, anxiety, bipolar disorder, schizophrenia, prevalence, neurobiology, epidemiology

## 1. INTRODUCTION

Mental health disorders are among the leading causes of disability worldwide (Vigo et al., 2016). Unlike acute physical illnesses, mental disorders often begin early in life and follow chronic or relapsing courses (Kessler et al., 2007). The present review provides an overview of four major disorder categories: major depressive disorder (MDD), anxiety disorders, bipolar disorder, and schizophrenia. For each disorder, the research examines population prevalence, age of onset, gender differences, biological alterations in brain structure and chemistry, and key sociodemographic correlates. No treatment or intervention strategies are discussed.

Mental disorders have been recognized for centuries, but only in recent decades have large-scale epidemiological surveys provided reliable prevalence estimates (Demyttenaere et al., 2004). The World Mental Health Surveys, conducted across 17 countries, established that mental disorders are common, often recurrent, and frequently begin in childhood or adolescence (Kessler et al., 2009). Neuroimaging has revealed that mental disorders are associated with measurable differences in brain structure and function (Schmaal et al., 2016). Neurotransmitter studies have documented abnormalities in serotonergic, dopaminergic, and noradrenergic signaling (Belujon & Grace, 2017). These biological findings do not imply that mental disorders are purely "brain diseases", social and developmental factors remain critically important (Bronfenbrenner & Morris, 2006).

## 2. Prevalence and Course of Major Mental Disorders

Epidemiological research has established that major mental disorders affect a substantial portion of the global population, each with distinct prevalence patterns, age of onset, and clinical trajectories; for instance, major depressive disorder (MDD) is characterized by persistent low mood, anhedonia, and alterations in sleep, appetite, and energy (American Psychiatric Association, 2013), and the global point prevalence of MDD is approximately 4.4%, affecting an estimated 322 million people (World Health Organization, 2017). In addition, twelve-month prevalence estimates from the World Mental Health Surveys averaged 5.9% across high-income countries and 5.5% across low- and middle-income countries (Bromet et al., 2011), while lifetime prevalence averages 15-18% in high-income countries (Kessler & Bromet, 2013). Furthermore, women are approximately 1.7 times more likely than men to experience MDD (Albert, 2015), and this gender difference emerges in early adolescence and persists across the lifespan (Seedat et al., 2009). Regarding age of onset, the median is 26 years, but notably, 25% of cases begin by age 14 (Kessler et al., 2005). As for the duration of illness, depressive episodes last a median of 4-6 months without intervention; however, 20-30% of individuals develop a chronic course lasting two years or longer (Spijker et al., 2002). Turning to anxiety disorders, this category includes generalized anxiety disorder, panic disorder, social anxiety disorder, and specific phobias (American Psychiatric Association, 2013), with generalized anxiety disorder affecting approximately 3.7% of the global population (Baxter et al., 2013).

Similarly, panic disorder affects 2-3% of adults in a given year (de Jonge et al., 2016), whereas social anxiety disorder has a 12-month prevalence of 4-7% across high-income countries (Stein & Stein, 2008). Moreover, specific phobias are the most common, with lifetime prevalence exceeding 10% (Wardenaar et al., 2017). As with MDD, women have significantly higher rates of all anxiety disorders, with female-to-male odds ratios ranging from 1.5 for social anxiety to 2.5 for panic disorder (McLean et al., 2011). The median age of onset for anxiety disorders is 11 years, which is earlier than any other disorder class (Kessler et al., 2005). Additionally, approximately 60% of individuals with one anxiety disorder meet criteria for a second anxiety disorder during their lifetime, indicating substantial comorbidity (Lamers et al., 2011). Beyond anxiety, bipolar disorder is characterized by recurrent episodes of depression and mania or hypomania (Grande et al., 2016), and the lifetime prevalence of bipolar spectrum disorders (including bipolar I, bipolar II, and cyclothymia) is approximately 2.4% globally (Merikangas et al., 2011). Specifically, bipolar I disorder (full manic episodes) affects 0.6% of the population, while bipolar II disorder (hypomanic episodes) affects 0.4% (Merikangas et al., 2007). Unlike MDD, bipolar disorder shows no consistent gender difference in overall prevalence; nevertheless, women are more likely to experience bipolar II and rapid cycling (Diflorio & Jones, 2010). The mean age of onset is 20-25 years, with 90% of cases beginning before age 50 (Kennedy et al., 2005).

It is also important to note that bipolar disorder is highly chronic: individuals spend approximately 50% of their follow-up time symptomatic, with depressive symptoms dominating the clinical course (Judd et al., 2002). Finally, schizophrenia affects approximately 0.3-0.7% of the global population (McGrath et al., 2008), and its prevalence is remarkably consistent across countries and cultures, suggesting a strong biological basis (Jablensky, 2000). The median age of onset is 22 years for men and 25 years for women (Häfner et al., 1998), and men have a 1.4-fold higher incidence, though prevalence equalizes in later life due to earlier male mortality (Aleman et al., 2003). Schizophrenia is characterized by positive symptoms (hallucinations, delusions), negative symptoms (flat affect, avolition), and cognitive impairments (Kahn et al., 2015), and the course is typically chronic, with only 20% of individuals achieving full recovery (Jääskeläinen et al., 2013). As a sobering outcome, lifetime suicide risk is approximately 5-6% (Palmer et al., 2005).

## 3. Neurobiological Alterations

### 3.1 Structural Brain Imaging Findings

Meta-analyses of magnetic resonance imaging (MRI) studies have identified consistent structural abnormalities across mental disorders. In major depressive disorder, the most replicated finding is reduced hippocampal volume, with a meta-analysis of 39 studies reporting a 10-15% reduction in depressed relative to control individuals (Videbech & Ravnkilde, 2004). This finding has been confirmed in larger samples (Schmaal et al., 2016). Prefrontal cortical volumes, particularly the subgenual anterior cingulate cortex and dorsolateral prefrontal cortex, are also reduced in MDD (Drevets et al., 2008).

In anxiety disorders, the amygdala and insula show structural abnormalities. A meta-analysis of 23 studies found smaller amygdala volumes in individuals with generalized anxiety disorder and social anxiety disorder (Shang et al., 2014). In panic disorder, reduced gray matter volume has been observed in the parahippocampal gyrus and midbrain (Sobanski & Wagner, 2017).

Bipolar disorder is associated with reductions in prefrontal cortical thickness and amygdala volume, but findings are less consistent than in MDD (Hibar et al., 2018). A large ENIGMA consortium study (N=3,000 bipolar patients) found reduced cortical thickness in frontal, temporal, and parietal regions (Hibar et al., 2016).

Schizophrenia shows the most extensive structural abnormalities. Meta-analyses report global gray matter reductions of 2-5%, with larger (10-15%) reductions in the superior temporal gyrus, medial temporal lobe (including hippocampus), and prefrontal cortex (Shenton et al., 2001). Lateral ventricular enlargement, a classic finding, has been reported in over 80% of studies (Wright et al., 2000).

### 3.2 Functional Brain Imaging Findings

Functional MRI (fMRI) studies during emotional and cognitive tasks reveal consistent patterns of abnormal brain activation. In MDD, the most robust finding is exaggerated amygdala activation to negative emotional stimuli (Hamilton et al., 2012). This amygdala hyperactivation normalizes with antidepressant treatment but persists in unmedicated patients (Sheline et al., 2001). Hypoactivation of the dorsolateral prefrontal cortex, a region involved in cognitive control, is also consistently observed (Kaiser et al., 2015).

In anxiety disorders, excessive amygdala reactivity to threatening stimuli is a core finding (Etkin & Wager, 2007). This hyperactivation extends to the insula and anterior cingulate cortex (Shin & Liberzon, 2010). Individuals with panic disorder show increased midbrain and brainstem activation during panic attacks (Dresler et al., 2013).

Bipolar disorder exhibits a distinct pattern: hyperactivation of limbic regions (amygdala, ventral striatum) during emotional processing combined with prefrontal hypoactivation during cognitive tasks (Phillips & Swartz, 2014). During manic states, there is pronounced striatal hyperactivation, which normalizes during euthymia (Strakowski et al., 2012).

Schizophrenia shows widespread functional abnormalities. During cognitive tasks, patients exhibit prefrontal hypoactivation ("hypofrontality"), one of the oldest and most replicated findings in psychiatric neuroimaging (Minzenberg et al., 2009). During auditory hallucinations, increased activation is observed in the primary auditory cortex, Broca's area, and the thalamus (Allen et al., 2008).

### 3.3 Neurotransmitter Systems

The monoamine hypothesis, involving serotonin, dopamine, and norepinephrine, has dominated biological psychiatry for decades (Schildkraut, 1965). In MDD, the most consistent finding is reduced serotonin turnover, as measured by decreased 5-hydroxyindoleacetic acid (5-HIAA) in cerebrospinal fluid (Asberg et al., 1976). Positron emission tomography (PET) studies have found reduced serotonin transporter binding in the midbrain and limbic regions (Kambeitz & Howes, 2015). Norepinephrine dysfunction is also implicated: postmortem studies show increased locus coeruleus neuronal density (Ordway et al., 1994).

Anxiety disorders involve gamma-aminobutyric acid (GABA) and serotonin systems. Magnetic resonance spectroscopy studies report reduced cortical GABA levels in generalized anxiety disorder (Goddard et al., 2001) and panic disorder (Goddard et al., 2004). The benzodiazepine receptor (GABA-A) shows altered binding in frontal and temporal regions (Malizia et al., 1998).

Bipolar disorder involves dopamine system abnormalities. Mania is associated with increased dopamine synthesis and release (Kumakura et al., 2010). Postmortem studies show altered dopamine D2 receptor density in the striatum (Dean, 2011). The norepinephrine system is also implicated: cerebrospinal fluid norepinephrine metabolites are elevated during mania (Koslow et al., 1983).

Schizophrenia is strongly linked to dopamine dysfunction, the "dopamine hypothesis" (Howes & Kapur, 2009). PET studies consistently show increased striatal dopamine synthesis capacity (Fusar-Poli & Meyer-Lindenberg, 2013). Postmortem studies have found increased D2 receptor density in the striatum of untreated patients (Seeman, 1987). However, glutamate dysfunction is also implicated: postmortem studies show reduced glutamate receptor expression in the prefrontal cortex (Moghaddam & Javitt, 2012).

## 4. Sociodemographic and Environmental Correlates

### 4.1 Gender Differences

Beyond the prevalence differences noted above, gender influences symptom presentation, illness course, and treatment response. Women with MDD are more likely to report atypical symptoms (increased sleep and appetite, weight gain, and leaden paralysis) and comorbid anxiety disorders, particularly panic disorder and generalized anxiety disorder (Marcus et al., 2008). They also show higher rates of seasonal affective disorder and peripartum depression. Men with MDD more often report irritability, aggression, risk-taking behavior, and substance use as primary manifestations, which can lead to underdiagnosis when clinicians rely on typical sad mood criteria (Martin et al., 2013). In schizophrenia, men exhibit earlier onset by approximately 3–5 years, more prominent negative symptoms (such as avolition and affective flattening), and poorer premorbid social and academic adjustment (Leung & Chue, 2000). Conversely, women with schizophrenia tend to have later onset, better premorbid functioning, and more pronounced affective symptoms. Explanations for these differences include sex hormone effects, particularly estrogen's neuroprotective and dopaminergic modulatory actions, which may delay onset in women until after menopause, alongside genetic differences (e.g., X-linked vulnerability genes) and psychosocial factors such as differential stress exposure and help-seeking norms (Riecher-Rössler, 2017). Emerging evidence also suggests that gender-based differences in brain structure, connectivity, and inflammatory responses contribute to these clinical variations.

### 4.2 Socioeconomic Status

Low socioeconomic status (SES) is strongly and consistently associated with increased risk for nearly all major mental disorders (Lorant et al., 2003). A comprehensive meta-analysis of 50 community-based studies found that low SES, measured by income, educational attainment, or occupational status, approximately doubles the odds of MDD, with pooled odds ratios ranging from 1.81 to 2.21 depending on the specific SES indicator (Lorant et al., 2003). Comparable effect sizes are observed for anxiety disorders, where low SES confers an odds ratio of approximately 1.5 to 2.0 (Wittchen et al., 2011). For schizophrenia, the association is more modest but significant, with lower SES linked to increased incidence, partially explained by both social causation and downward drift. The relationship is bidirectional in nature: mental disorders frequently cause downward social drift through impaired occupational and educational functioning, while low SES simultaneously increases exposure to chronic stressors such as housing instability, financial strain, and neighborhood violence, creating a self-perpetuating vicious cycle (Dohrenwend et al., 1992). Furthermore, low SES is associated with reduced access to quality mental health care, lower treatment adherence, and higher dropout rates, which exacerbate illness trajectories and widen disparities over the life course.

### 4.3 Adverse Childhood Experiences

Exposure to childhood maltreatment, encompassing physical abuse, sexual abuse, emotional abuse, and neglect, dramatically and dose-dependently increases lifetime risk for all major mental disorders (Green et al., 2010). A large meta-analysis of 37 epidemiological studies found that childhood maltreatment elevates the odds of MDD by 2.5-fold, with even higher risks for recurrent and early-onset depression (Mandelli et al., 2015). For anxiety disorders, the pooled odds ratio is 2.3, particularly for post-traumatic stress disorder and panic disorder (Lindert et al., 2014). In bipolar disorder, childhood maltreatment increases risk approximately 2-fold and is also associated with earlier age of onset, more frequent mood episodes, and higher rates of suicide attempts (Agnew-Blais & Danese, 2016). For schizophrenia, the effect is somewhat smaller but remains robust and significant, with an odds ratio around 1.5 after controlling for genetic and familial confounding (Varese et al., 2012). These effects are substantially mediated by neurobiological alterations, including reduced hippocampal and prefrontal cortical volumes, amygdala hyperreactivity, and persistent dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, as reflected in altered cortisol awakening responses and feedback inhibition (Teicher & Samson, 2016). Epigenetic modifications, such as DNA methylation of glucocorticoid receptor genes, provide a molecular mechanism linking early adversity to lasting stress vulnerability.

### 4.4 Urbanicity and Migration

Growing up or living in an urban environment is associated with an approximately 2-fold increase in the risk of developing schizophrenia, an effect that persists after controlling for familial liability and substance use (Vassos et al., 2012). This urbanicity effect is dose-dependent: each quartile increase in urbanicity exposure (e.g., moving from a rural area to a small town, then to a city, then to a capital city) raises schizophrenia risk by 20–30% (Pedersen & Mortensen, 2001). First- and second-generation immigrants also show substantially elevated schizophrenia risk, with odds ratios of 2.5 for first-generation immigrants and 2.3 for second-generation immigrants, indicating that psychosocial rather than purely biological migration-related factors are likely responsible (Cantor-Graae & Selten, 2005). Risks are particularly high for immigrants from certain regions (e.g., North Africa, the Caribbean, and Sub-Saharan Africa) and for ethnic minorities living in areas with low ethnic density, suggesting that social exclusion, discrimination, and chronic vigilance play key roles. For MDD and anxiety disorders, urbanicity effects are weaker and less consistent across studies, with some meta-analyses finding null or modest associations (Peen et al., 2010). Proposed mechanisms for the urbanicity-schizophrenia link include increased social fragmentation, greater exposure to air pollution and toxins, higher rates of childhood adversity in cities, and elevated social stress leading to HPA axis and dopaminergic sensitization.

### 4.5 Marital Status

Being unmarried, including never married, divorced, separated, or widowed, is consistently associated with increased risk for all major mental disorders across diverse cultural contexts (Kessler et al., 1998). The protective effect of marriage is strongest for men and for MDD, with unmarried men showing an odds ratio for depression of approximately 1.7 compared to married men (Bulloch et al., 2009). For anxiety disorders and substance use disorders, the pattern is similar but somewhat attenuated. Whether this association reflects causation (i.e., marriage provides emotional support, economic resources, and health-monitoring behaviors that protect against disorder) or selection (i.e., individuals with pre-existing or prodromal disorders are less likely to marry and more likely to divorce) remains actively debated (Whisman & Baucom, 2012). Longitudinal and co-twin control studies suggest that both processes operate: marriage confers genuine protective effects, particularly for men, but there is also evidence of selection effects, especially for schizophrenia and bipolar disorder, where premorbid social deficits reduce marriage likelihood. The quality of marriage matters substantially, distressed or conflict-ridden marriages are associated with higher depression risk than being unmarried, indicating that it is not simply the marital status itself but the experience of social support that drives the association.

## 5. Comorbidity and Overlap

Mental disorders frequently co-occur at rates far exceeding chance. The National Comorbidity Survey Replication found that 45% of individuals with lifetime MDD also meet criteria for an anxiety disorder (Kessler et al., 2005). Among those with bipolar disorder, 75% have a comorbid anxiety disorder, and 30% have a comorbid substance use disorder (Merikangas et al., 2007). Schizophrenia shows high comorbidity with anxiety (30–50%) and depression (40–60%), the latter being associated with poorer outcomes and increased suicide risk (Buckley et al., 2009).

Comorbidity is not random. Large-scale factor analyses consistently reveal two broad dimensions: internalizing (mood and anxiety disorders) and externalizing (substance use and antisocial behavior) (Krueger, 1999). These dimensions have distinct genetic and neurobiological correlates (Kotov et al., 2017). The internalizing dimension further splits into "distress" (MDD, generalized anxiety, PTSD) and "fear" (phobias, panic disorder) subfactors, which differ in their links to negative affectivity versus conditioned fear responses (Krueger & Markon, 2006). This hierarchical structure has informed dimensional alternatives to traditional categorical diagnoses.

## 6. CONCLUSION

Mental health disorders are common, often chronic conditions that affect hundreds of millions worldwide. Major depressive disorder affects 4.4% of the global population, anxiety disorders 3.6%, bipolar disorder 2.4%, and schizophrenia 0.5-1.0% (World Health Organization, 2017; Merikangas et al., 2011; McGrath et al., 2008). These disorders are associated with replicable alterations in brain structure (reduced hippocampal, prefrontal, and temporal volumes), brain function (limbic hyperactivation and prefrontal hypoactivation), and neurotransmitter systems (serotonin, dopamine, norepinephrine, GABA, and glutamate). Sociodemographic factors, including gender, low socioeconomic status, adverse childhood experiences, urbanicity, migration, and marital status, consistently predict disorder risk. Key findings from this review include: women have higher rates of internalizing disorders (MDD, anxiety), while men have slightly higher rates of externalizing disorders and earlier schizophrenia onset; low socioeconomic status doubles the odds of MDD and anxiety disorders; childhood maltreatment increases risk for all major mental disorders by 1.5- to 2.5-fold; urban upbringing and immigrant status increase schizophrenia risk specifically (approximately 2-fold); and comorbidity is the rule rather than the exception, with 45-75% of individuals meeting criteria for multiple disorders. This overview has deliberately avoided intervention strategies, treatment approaches, or clinical recommendations. The focus has been descriptive and epidemiological, providing a foundation for understanding mental disorders as brain-based conditions shaped by developmental and social experience. Future research should prioritize longitudinal studies to establish causal direction, large-scale neuroimaging consortia to increase replicability, and transdiagnostic investigations to clarify disorder boundaries.

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