Çukurova Ruh Sağlığı Dergisi
Çukurova Journal of Mental Health
2025; 1(1):88-100

DERLEME | REVIEW

Neuroimaging in Psychiatric Disorders

Psikiyatrik Bozukluklarda Nörogörüntüleme

Ömer Kaya [1]

1. Çukurova University, Adana

Abstract

Advanced neuroimaging methods have greatly expanded our understanding of psychiatric disorders by enabling detailed evaluation of brain structure, function, and connectivity. This review synthesizes findings from major psychiatric conditions, including schizophrenia, major depressive disorder, bipolar disorder, obsessive–compulsive disorder (OCD), anxiety disorders, autism spectrum disorder, and attention-deficit/hyperactivity disorder (ADHD). Across disorders, structural mahnetic resonance imaging (MRI) consistently reveals cortical and subcortical abnormalities, while diffusion tensor imaging highlights widespread white-matter dysconnectivity. Functional MRI studies demonstrate disorder-specific but overlapping alterations in network activity, particularly involving prefrontal, limbic, and default mode systems. Molecular and spectroscopic imaging add complementary insights into neurotransmitter and metabolic disturbances, including dopaminergic dysfunction in schizophrenia, glutamatergic abnormalities in mood disorders and OCD, GABAergic deficits in anxiety, and excitatory–inhibitory imbalance in autism and ADHD. Although no single imaging marker has sufficient specificity for clinical diagnosis, neuroimaging has substantially advanced mechanistic models of psychiatric illness. Longitudinal and multimodal approaches reveal developmental trajectories, illness progression, and treatment effects, such as lithium-related neurotrophic changes in bipolar disorder or normalization of hyperactive circuits following cognitive behavioral therapy (CBT) in OCD and anxiety disorders. Neuroimaging has transformed the conceptualization of psychiatric disorders from region-specific abnormalities to distributed network and molecular dysfunction. While still largely confined to research, ongoing advances in multimodal integration, large-scale collaborations, and precision imaging approaches hold promise for earlier detection, individualized intervention, and improved patient outcomes.

Keywords: Psychiatric diseases, neuroimaging, MRI

Öz

İleri nörogörüntüleme yöntemleri, beyin yapısı, işlevi ve bağlantısallığının ayrıntılı değerlendirilmesini sağlayarak psikiyatrik bozukluklar hakkındaki anlayışımızı büyük ölçüde genişletmiştir. Bu derleme, şizofreni, majör depresif bozukluk, bipolar bozukluk, obsesif-kompulsif bozukluk (OKB), anksiyete bozuklukları, otizm spektrum bozukluğu ve dikkat eksikliği/hiperaktivite bozukluğu (DEHB) gibi başlıca psikiyatrik durumlara ilişkin bulguları sentezlemektedir. Bozukluklar genelinde, yapısal manyetik rezonans görüntüleme (MRI) sürekli olarak kortikal ve subkortikal anormallikler ortaya koyarken, difüzyon tensör görüntüleme yaygın beyaz madde bağlantı bozukluklarını vurgular. Fonksiyonel MRI çalışmaları, özellikle prefrontal, limbik ve varsayılan mod sistemlerini içeren ağ aktivitelerinde bozukluğa özgü ancak örtüşen değişiklikler gösterir. Moleküler ve spektroskopik görüntüleme, nörotransmitter ve metabolik bozukluklar hakkında tamamlayıcı içgörüler sunar; bunlar arasında şizofrenide dopaminerjik işlev bozukluğu, duygu durum bozuklukları ve OKB'de glutamaterjik anormallikler, anksiyetede GABAerjik eksiklikler ve otizm ile DEHB'de uyarılma-inhibitör dengesizlik yer alır. Tek bir görüntüleme belirteci klinik tanı için yeterli özgüllüğe sahip olmasa da, nörogörüntüleme psikiyatrik hastalıkların mekanistik modellerini önemli ölçüde ilerletmiştir. Boylamsal ve çok modlu yaklaşımlar, gelişimsel yörüngeleri, hastalık ilerlemesini ve tedavi etkilerini ortaya koyar; örneğin, bipolar bozuklukta lityumla ilişkili nörotrofik değişiklikler veya OKB ve anksiyete bozukluklarında bilişsel davranışçı terapi (BDT) sonrası hiperaktif devrelerin normalleşmesi gibi. Nörogörüntüleme, psikiyatrik bozuklukların kavramsallaştırılmasını bölgeye özgü anormalliklerden daha geniş ağ ve moleküler işlev bozukluğuna dönüştürmüştür. Hâlen büyük ölçüde araştırmayla sınırlı olsa da, çok modlu entegrasyon, büyük ölçekli iş birlikleri ve hassas görüntüleme yaklaşımlarındaki devam eden ilerlemeler, daha erken teşhis, bireyselleştirilmiş müdahale ve hasta sonuçlarının iyileştirilmesi için umut vadetmektedir..

Anahtar kelimeler: Ruhsal bozukluk, nörogörüntüleme, MRI

Correspondence / Yazışma Adresi: Ömer Kaya, Çukurova University Faculty of Medicine Department of Radiology, Adana, Türkiye
E-mail: dr.omerkaya@gmail.com

Received /Gönderilme tarihi: 20.07.2025 | Accepted /Kabul tarihi: 19.09.2025

Introduction

Advanced neuroimaging techniques are currently used to examine the reflections of psychiatric disorders on brain structure and function. Primarily magnetic resonance imaging (MRI), as well as functional MRI (fMRI), diffusion tensor imaging (DTI), perfusion MRI, and MR spectroscopy have played a significant role in the investigation of psychiatric diseases. Through these techniques, structural changes (such as brain volumes and cortical thickness), brain activity (resting-state and task-based activations), and connectivity (white matter tracts and functional networks) can be analyzed in detail. In this review, the radiological imaging findings of major psychiatric disorders—including schizophrenia, major depressive disorder, bipolar disorder, obsessive-compulsive disorder (OCD), anxiety disorders, autism spectrum disorder (ASD), and attention-deficit/hyperactivity disorder (ADHD)—and the clinical contributions of these methods to diagnosis, treatment, and follow-up will be discussed.

Schizophrenia

Schizophrenia is a severe psychiatric disorder with complex neurobiological underpinnings. Neuroimaging techniques have been central in identifying brain abnormalities that contribute to its pathophysiology. Although no single imaging marker is diagnostic, accumulated evidence supports the view of schizophrenia as a disorder of brain structure, function, and connectivity (Dabiri et al., 2022; Ray et al., 2024).

Structural MRI studies consistently show ventricular enlargement and gray matter volume reduction, particularly in the prefrontal cortex, temporal lobes, hippocampus, and thalamus (Howes & Kapur, 2009). Cortical thinning and progressive gray matter loss have been demonstrated in longitudinal studies, indicating accelerated neurodegeneration compared to normal aging (Sun et al., 2009). Diffusion tensor imaging (DTI) reveals widespread white matter abnormalities, especially in frontotemporal tracts and the corpus callosum, supporting the dysconnectivity hypothesis (Kelly et al., 2018). Functional MRI (fMRI) highlights hypoactivation of the dorsolateral prefrontal cortex during working memory and attention tasks, often described as “hypofrontality” (Barch & Ceaser, 2012). Resting-state fMRI reveals hyperconnectivity of the default mode network (DMN) and reduced anti-correlation with task-positive networks, reflecting impaired cognitive control (Sylvester et al., 2012). Positron emission tomography (PET) has consistently demonstrated reduced frontal glucose metabolism and increased striatal dopamine synthesis capacity, reinforcing the dopamine hypothesis (Townsend et al., 2023).

Over the last decade, several innovations have reshaped the field:

1. Multimodal imaging (integrating MRI, PET, EEG) provides converging evidence of combined structural and functional deficits (Sui et al., 2012).
2. PET tracers targeting synaptic density (e.g. SV2A ligands) show reduced binding in frontal and temporal regions, suggesting synaptic loss independent of medication effects (Onwordi et al., 2020).
3. Machine learning approaches applied to large imaging datasets (e.g. ENIGMA) aim to classify patients and predict clinical outcomes, though current accuracy limits clinical application (Vieira et al., 2020).
4. Longitudinal studies suggest that early gray matter changes may predict prognosis, highlighting the potential of neuroimaging biomarkers for early intervention (Sun et al., 2009); (Phillips & Swartz, 2014).

Clinically, imaging is mainly used to exclude organic causes of psychosis rather than diagnose schizophrenia. Group-level findings lack specificity for individual patients, and results are influenced by heterogeneity and antipsychotic exposure (Ray et al., 2024; Ho et al., 2011). Nonetheless, neuroimaging has provided invaluable insights into pathophysiology and is moving toward translational use in early detection, treatment prediction, and personalized psychiatry (Vieira et al., 2020; Chitty et al., 2013).

As a conclusion, neuroimaging has advanced the understanding of schizophrenia from structural brain alterations to network-level dysfunction and synaptic changes. While not yet a routine diagnostic tool, ongoing advances in multimodal and computational imaging hold promise for future clinical integration.

Major Depressive Disorder

Major Depressive Disorder (MDD) is one of the most prevalent psychiatric illnesses worldwide. Neuroimaging studies have provided critical insights into its neurobiological basis, revealing abnormalities in brain structure, function, and

connectivity. Although neuroimaging is not yet used as a diagnostic tool in routine clinical practice, findings have shaped theoretical models of depression and hold promise for biomarker development (Arnone et al., 2012; Gong & He, 2015).

Structural MRI studies in MDD consistently report reduced gray matter volume in key mood-regulating regions, particularly the hippocampus, anterior cingulate cortex (ACC), and prefrontal cortex (Schmaal et al., 2016). Hippocampal atrophy has been strongly associated with illness duration and recurrence, suggesting a neuroprogressive course (Frodl et al., 2008). DTI studies reveal white matter microstructural abnormalities, especially in the uncinate fasciculus and cingulum bundle, pathways connecting limbic and frontal regions (Gong & He, 2015).

Task-based fMRI has demonstrated hypoactivation of the dorsolateral prefrontal cortex during cognitive control tasks and hyperactivation of the amygdala in response to negative emotional stimuli (Kupfer et al., 2012). Resting-state fMRI shows disrupted connectivity within the default mode network (DMN) and between the DMN and cognitive control networks, consistent with excessive rumination and impaired regulation of negative mood (Mulders et al., 2015).

Recent years have brought important innovations:

1. Multimodal approaches combining structural, functional, and molecular imaging provide integrative views of brain abnormalities (Nugent et al., 2019).
2. PET ligands targeting serotonin and glutamate receptors have clarified neurotransmitter dysfunction beyond the monoamine hypothesis (Nikolaus et al., 2017).
3. Machine learning models trained on large neuroimaging datasets have shown moderate success in distinguishing MDD patients from controls and predicting treatment response (Vieira et al., 2020).
4. Longitudinal studies suggest that pre-treatment imaging patterns, such as amygdala hyperreactivity or prefrontal hypoactivity, may serve as predictors of clinical outcomes (Phillips & Swartz, 2014; Frodl et al., 2008).

In clinical psychiatry, imaging is mainly used to rule out organic causes of depressive symptoms. Group-level abnormalities lack sufficient sensitivity and specificity for diagnosis. Findings are also heterogeneous, influenced by factors such as comorbidity, medication use, and illness chronicity (Gong & He, 2015; Ichikawa et al., 2016). Nevertheless, imaging has begun to inform treatment selection—for example, identifying patients more likely to benefit from cognitive-behavioral therapy versus pharmacotherapy based on neural activation.

As a conclusion, neuroimaging research in MDD has highlighted consistent abnormalities in cortico-limbic circuits, particularly involving the prefrontal cortex, amygdala, and hippocampus. While current applications remain research-oriented, advances in multimodal imaging and predictive modeling hold potential for personalized treatment strategies and biomarker discovery in the future.

Bipolar disorder

Bipolar disorder (BD) involves recurrent mood episodes and significant functional impairment. Neuroimaging has revealed reproducible abnormalities across cortico-limbic circuits governing emotion regulation, cognition, and reward. Although no single imaging marker is diagnostic, convergent structural, functional, and molecular findings inform pathophysiological models and point toward emerging biomarkers (Phillips & Swartz, 2014; Mwangi et al., 2016).

Structural MRI consistently implicates fronto-limbic systems. Meta-analyses and large consortia report cortical thickness and surface area alterations in prefrontal regions (dorsolateral and ventromedial), anterior cingulate cortex (ACC), and temporal cortices (Hibar et al., 2018). Subcortical volumetric studies (ENIGMA) show case–control differences in the hippocampus, thalamus, amygdala, and lateral ventricles, with illness stage, age at onset, and medication exposure moderating the effects (Hibar et al., 2016). Pediatric-onset BD often shows larger amygdala volumes, whereas adult samples show smaller or normative amygdala volumes—underscoring developmental heterogeneity (Bora et al., 2010). Diffusion tensor imaging (DTI) reveals widespread white matter microstructural abnormalities—most robustly in the corpus callosum, uncinate fasciculus, cingulum bundle, and fronto-temporal association fibers—supporting a dysconnectivity framework (Nortje et al., 2013). Lithium treatment has been associated with increased gray matter volume in ACC and hippocampus, consistent with neurotrophic effects (Moore et al., 2000).

Task-based fMRI commonly demonstrates hyperactivation of the amygdala and ventral striatum to emotional or reward-related stimuli and hypoactivation of cognitive control regions (dorsolateral PFC/ACC) during inhibitory control or working memory tasks (Chen et al., 2011). Resting-state fMRI indicates altered connectivity within and between the default mode, salience, and fronto-parietal control networks, with mood-state dependence (mania vs. depression) and trait features both reported (Syan et al., 2018). FDG-PET and perfusion studies converge on state-related metabolic changes: relative prefrontal hypometabolism during depressive episodes and fronto-limbic hypermetabolism during mania, with partial normalization after effective treatment (Brooks & Vizueta, 2014). Serotonergic and dopaminergic PET/SPECT studies further support neurotransmitter dysregulation in BD (Nikolaus et al., 2017).

The latest developments on the subject are as follows:

1. Multimodal approaches integrate structural MRI, fMRI, DTI, MRS, and PET/SPECT to map coordinated abnormalities across molecular, circuit, and systems levels; simultaneous or longitudinal designs improve causal inference (Nugent et al., 2019).
2. Synaptic and glial targets: MRS studies demonstrate glutamatergic abnormalities (elevated Glx in ACC/medial PFC in subgroups), while emerging PET tracers interrogate neuroinflammation and synaptic density, though findings remain preliminary (Chitty et al., 2013).
3. Machine learning models using large multi-site datasets (including ENIGMA-BD) show moderate accuracy for BD vs. controls and early signals for predicting mood relapses or treatment response; generalizability and clinical utility remain limited pending harmonized pipelines and prospective validation (Vieira et al., 2020).
4. Treatment effects: longitudinal imaging demonstrates lithium-associated increases in prefrontal/hippocampal volume and task fMRI normalization; ECT produces rapid subcortical and medial temporal changes paralleling symptom improvement in severe episodes (Moore et al., 2000; Abbott et al., 2014).

In routine practice, structural imaging serves primarily to exclude organic etiologies of new-onset mood episodes. Group-level abnormalities lack the sensitivity/specificity for diagnosis in individuals. Heterogeneity (age at onset, polarity predominance, psychosis, comorbidity, rapid cycling), medication exposure (notably lithium and antipsychotics), and mood-state effects complicate interpretation (Mwangi et al., 2016; Hibar et al., 2018; Hibar et al., 2016). Nevertheless, imaging is beginning to inform patient stratification in research—e.g., identifying cortico-limbic signatures associated with suicidality risk or predicting who responds to lithium vs. antidepressants—hinting at future roles in precision psychiatry (Vieira et al., 2020; Abbott et al., 2014).

As a conclusion, BD neuroimaging converges on disrupted fronto-limbic structure and connectivity with state- and trait-dependent functional abnormalities. While not yet diagnostic, advances in multimodal imaging, biologically informed PET/MRS targets, and robust machine-learning frameworks may yield clinically actionable biomarkers for prognosis and treatment selection in the coming years.

Obsessive–Compulsive Disorder

Obsessive–compulsive disorder (OCD) is characterized by intrusive thoughts and repetitive behaviors and is increasingly framed as a disorder of cortico–striato–thalamo–cortical (CSTC) circuitry. Neuroimaging has revealed convergent structural, functional, and molecular abnormalities within orbitofrontal–anterior cingulate–striatal loops that support models of impaired cognitive control, excessive error signaling, and maladaptive habit formation (Saxena & Rauch, 2000; Stein et al., 2019).

Structural MRI meta-analyses report gray-matter alterations in orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), insula, and temporal regions, alongside subcortical differences in caudate, putamen, and thalamus (de Wit et al., 2014; Rotge et al., 2008). Effect sizes are modest at the individual-subject level but consistent across cohorts. Diffusion MRI shows white-matter microstructural abnormalities—most robustly in the anterior limb of the internal capsule, corpus callosum, cingulum bundle, and fronto-temporal association fibers—supporting a dysconnectivity framework for OCD (Piras et al., 2013). Developmental heterogeneity is notable: pediatric samples can differ from adults (e.g., amygdala/striatal volumes), emphasizing the impact of age, illness duration, and treatment exposure (Rotge et al., 2008; Piras et al., 2013).

Task fMRI consistently demonstrates hyperactivation of OFC/ACC and caudate during symptom provocation, error monitoring, or conflict tasks, with relative hypoactivation in lateral prefrontal control regions (Stern et al., 2011; Brühl

et al., 2014). Resting-state fMRI meta-analyses reveal altered connectivity within CSTC loops and network-level dysconnectivity spanning the default mode, salience, and frontoparietal control networks—patterns linked to rumination, overactive salience tagging, and impaired top-down regulation (Gürsel et al., 2018). Molecular imaging complements these findings: FDG-PET shows hypermetabolism in OFC and caudate at baseline and normalization with effective treatment (SSRIs or CBT), one of the clearest brain–behavior–treatment links in psychiatry (Baxter et al., 1992; Schwartz et al., 1996). 1H-MRS studies frequently implicate glutamatergic abnormalities in ACC/striatal nodes in subsets of patients, consistent with glutamate-modulating treatment strategies (Brennan et al., 2013).

The recent developments on the subject are as follows:

1. Multimodal integration: Combined sMRI/fMRI/DTI/MRS and, where available, PET provide convergent evidence of CSTC dysfunction and its extension to large-scale networks; longitudinal and symptom-provocation designs strengthen causal inference (Nugent et al., 2019).
2. Circuit-targeted neuromodulation: Imaging has guided deep brain stimulation (DBS) targeting the ventral capsule/ventral striatum (VC/VS) and related nodes; DBS can restore frontostriatal dynamics in responders (Figee et al., 2013; Denys et al., 2010). Deep TMS of dorsomedial PFC/ACC also shows randomized-trial efficacy, paralleling modulation of control–salience circuitry (Carmi et al., 2019).
3. Predictive modeling: Machine-learning studies using multi-site datasets report above-chance classification of OCD vs. controls and early signals for predicting CBT/SSRI response, though generalizability remains limited pending harmonized pipelines and prospective validation (Nugent et al., 2019).

In practice, neuroimaging primarily serves to exclude organic etiologies of new-onset compulsive symptoms; no scan is diagnostic of OCD at the individual level. Group-level effects are small and influenced by comorbidity, symptom dimensions, medication status, developmental stage, and state (on/off provocation) (Saxena & Rauch, 2000; Stein et al., 2019; de Wit et al., 2014). Nevertheless, imaging informs mechanism-based care: (i) it supports glutamatergic targets, (ii) provides objective biomarkers of target engagement for CBT/SSRI/DBS/TMS, and (iii) enables circuit-guided neuromodulation trials (Nugent et al., 2019; Baxter et al., 1992; Schwartz et al., 1996; Figee et al., 2013; Denys et al., 2010; Carmi et al., 2019).

As a conclusion; across modalities, OCD converges on fronto-striatal–thalamo–cortical dysregulation with broader network-level alterations. While not yet a clinical diagnostic tool, advances in multimodal imaging, circuit-based neuromodulation, and robust predictive modeling are moving the field toward precision psychiatry for OCD—linking symptoms to circuits and treatments in ways that can ultimately improve outcomes (Nugent et al., 2019; Saxena & Rauch, 2000; Gürsel et al., 2018; Figee et al., 2013; Denys et al., 2010; Carmi et al., 2019).

Anxiety Disorders

Anxiety disorders (including generalized anxiety disorder, panic disorder, social anxiety disorder, and specific phobias) are increasingly conceptualized as disorders of cortico–limbic circuitry. Neuroimaging has identified convergent abnormalities in amygdala–hippocampal–insula systems and in prefrontal control networks that regulate threat appraisal, interoception, and emotion regulation. Although no single imaging marker is diagnostic, group-level findings have clarified core pathophysiology and are beginning to inform biomarkers for prognosis and treatment selection (Bishop, 2007; Etkin & Wager, 2007).

Structural MRI studies report modest but reliable gray-matter alterations across anxiety disorders, with effects centered on the anterior cingulate cortex (ACC), insula, and medial/dorsolateral prefrontal cortex (mPFC/dlPFC), and condition-specific differences in amygdala and hippocampus (Mwangi et al., 2016; Kim & Whalen, 2009). Diffusion MRI highlights white-matter microstructural differences in tracts linking limbic and prefrontal regions—most notably the uncinate fasciculus and cingulum bundle—supporting a dysconnectivity framework for anxiety (Hart et al., 2013). Developmental and clinical heterogeneity (age, illness duration, comorbidity, medication) moderate effect sizes, underscoring the need for large multi-site samples and harmonized methods (Mwangi et al., 2016; Etkin & Wager, 2007).

Task-based fMRI consistently demonstrates amygdala and insula hyperreactivity to threat-related cues and reduced recruitment of prefrontal control regions (dlPFC/ventromedial PFC) during regulation or cognitive control, aligning with models of impaired top-down inhibition of limbic responses (Etkin & Wager, 2007; Kim & Whalen, 2009; Fox & Shackman, 2019). Resting-state fMRI shows altered connectivity within and between large-scale networks—salience,

default mode, and frontoparietal control—with increased coupling of limbic nodes to salience/DMN hubs and reduced integration with control networks (Sylvester et al., 2012; Etkin & Wager, 2007). Human imaging of the bed nucleus of the stria terminalis (BNST) differentiates sustained anxiety from acute fear and reveals dysregulated extended-amygdala circuitry in anxious individuals (Kim & Whalen, 2009). Molecular imaging adds neurochemical context: PET/SPECT studies report reduced GABA-A receptor binding (e.g., with [11C]-flumazenil) and altered 5-HT1A binding, alongside regional metabolic differences on FDG-PET in limbic and prefrontal cortices (Malizia et al., 1998; Nutt & Malizia, 2001; Goddard et al., 2001). Proton MRS studies frequently show lower cortical GABA concentrations in subsets of patients (e.g., panic disorder, GAD), consistent with inhibitory tone deficits (Goddard et al., 2001).

Recent advances in neuroimaging of anxiety disorders: are:

1. Multimodal integration and consortia: Combined sMRI/fMRI/DTI/MRS and meta-analytic/mega-analytic efforts clarify convergent abnormalities across modalities and diagnoses; network- and connectome-level models have refined transdiagnostic mechanisms (Mwangi et al., 2016; Etkin & Wager, 2007).
2. Threat learning and BNST-focused paradigms: High-resolution imaging and computational models dissociate phasic fear (amygdala) from sustained anxiety (BNST), linking individual differences in avoidance and intolerance of uncertainty to extended-amygdala dynamics (Kim & Whalen, 2009).
3. Biomarkers and prediction: Machine-learning models trained on multi-site imaging have achieved above-chance classification of anxiety vs. controls and show early promise for predicting CBT or pharmacotherapy response, though generalizability remains a key limitation pending prospective validation and standardized pipelines (Sylvester et al., 2012; Etkin & Wager, 2007).
4. Treatment mechanisms and neuromodulation: Longitudinal fMRI demonstrates normalization of amygdala/insula hyperreactivity after effective CBT or SSRI treatment in social anxiety and panic disorder; randomized and sham-controlled trials of rTMS targeting prefrontal control hubs (e.g., dlPFC/dmPFC) report symptom reductions in GAD and SAD, paralleling modulation of control–salience circuitry (Malizia et al., 1998; Nutt & Malizia, 2001; Goddard et al., 2001).

In routine practice, structural imaging is used primarily to exclude organic etiologies of new-onset anxiety symptoms; no scan is diagnostic at the individual level. Group effects are small-to-moderate and influenced by symptom dimensions (e.g., fear vs. distress), state (provoked vs. resting), comorbidity, and medication exposure (Mwangi et al., 2016; Carmi et al., 2019; Bishop, 2007; Etkin & Wager, 2007; Nutt & Malizia, 2001; Goddard et al., 2001). Nevertheless, neuroimaging informs mechanism-based care: (i) it supports inhibitory (GABA) and serotonergic targets, (ii) provides objective markers of target engagement for CBT/SSRI/rTMS, and (iii) enables circuit-guided neuromodulation research. Ongoing work toward robust, generalizable predictive signatures and individualized circuit targets holds promise for precision psychiatry in anxiety disorders (Sylvester et al., 2012; Etkin & Wager, 2007; Malizia et al., 1998; Nutt & Malizia, 2001; Goddard et al., 2001).

As a conclusion; across modalities, anxiety disorders converge on heightened limbic/salience reactivity and weakened prefrontal control, with network-level dysconnectivity linking symptoms to circuits for threat processing, interoception, and regulation. While not yet a clinical diagnostic tool, advances in multimodal imaging, BNST-focused paradigms, and predictive modeling are moving the field toward actionable biomarkers for prognosis and treatment selection (Sylvester et al., 2012; Bishop, 2007; Etkin & Wager, 2007; Kim & Whalen, 2009; Malizia et al., 1998; Nutt & Malizia, 2001; Goddard et al., 2001).

Autism Spectrum Disorder

Autism Spectrum Disorder (ASD) is a heterogeneous neurodevelopmental condition characterized by social-communication differences and restricted, repetitive behaviors. Contemporary models emphasize atypical development of large-scale brain networks—especially social cognition, salience/interoception, and cognitive control—and altered excitation–inhibition balance. Neuroimaging has identified convergent structural, functional, and molecular abnormalities, while also highlighting considerable inter-individual variability. No imaging marker is diagnostic at the individual level, but group-level findings have informed mechanistic accounts and point toward emerging biomarkers (Ecker et al., 2015; Ameis & Catani, 2015).

Structural MRI studies report small-to-moderate differences in gray matter and cortical morphology across distributed regions, including prefrontal, temporal (superior temporal sulcus/temporal pole), insular, and cingulate cortices, as

well as cerebellum; effects vary with age, sex, IQ, and symptom profile (Ecker et al., 2015; Ameis & Catani, 2015; Nomi & Uddin, 2015). Diffusion MRI demonstrates widespread white-matter microstructural differences—most consistently within long association pathways (e.g., uncinate fasciculus, cingulum, superior longitudinal fasciculus) and callosal fibers—supporting a dysconnectivity framework (Nomi & Uddin, 2015). A key developmental observation is early brain overgrowth in a subset of infants who later develop ASD, driven particularly by accelerated cortical surface area expansion during the first year of life (Hazlett et al., 2017). Subcortical volumetry findings are mixed; amygdala differences appear age-dependent (enlarged in some pediatric samples, more variable in adults), consistent with developmental heterogeneity (Ecker et al., 2015). Overall, large consortia and meta-analyses indicate that case–control anatomical effects are subtle and moderated by sampling and motion artifacts, underscoring the need for harmonized, multi-site approaches (Ameis & Catani, 2015).

Task-based fMRI frequently shows reduced activation in social perception networks (e.g., temporo-parietal junction, posterior superior temporal sulcus, fusiform face area) and atypical amygdala responsivity to socio-emotional cues, together with altered recruitment of prefrontal control regions during executive or language tasks (Ecker et al., 2015; Di Martino et al., 2014; Just et al., 2012). Resting-state fMRI highlights atypical functional connectivity: many studies report reduced long-range fronto-posterior coupling with relative increases in local connectivity, aligning with underconnectivity accounts (Di Martino et al., 2014). Large open datasets (e.g., ABIDE) reveal reproducible alterations within the default mode, salience/interoceptive, and sensorimotor networks, but also substantial heterogeneity across individuals and sites (Just et al., 2012). Beyond BOLD, electrophysiology (EEG/MEG) demonstrates atypical oscillatory dynamics (e.g., reduced gamma-band synchrony) during sensory and social processing, consistent with impaired temporal coordination of distributed networks (Ecker et al., 2015).

Magnetic resonance spectroscopy (1H-MRS) and molecular imaging suggest excitation–inhibition imbalance in subsets of individuals with ASD. Regional MRS studies report altered GABA and glutamate/glutamine (Glx) levels in anterior cingulate, medial prefrontal, and sensorimotor cortices, while PET imaging with the GABA-A-specific ligand [11C]Ro15-4513 indicates reduced GABA-A receptor availability in cortical and limbic regions (Aoki et al., 2018; Ajram et al., 2017). Although findings vary by age and methodology, convergent evidence supports abnormalities in inhibitory signaling that may contribute to sensory hyper-/hypo-reactivity and social-cognitive differences (Ecker et al., 2015; (Aoki et al., 2018; Ajram et al., 2017).

Recent advances in neuroimaging of autism spectrum disorder are:

1. Large-scale consortia and open science: Initiatives such as ABIDE enable harmonized analyses across thousands of scans, improving reproducibility and quantification of heterogeneity; connectome-level models clarify transdiagnostic vs. autism-specific effects (Ameis & Catani, 2015; Just et al., 2012).
2. Normative modeling and subtyping: Data-driven approaches move beyond average case–control contrasts to identify individual-level deviations and neurobiological subtypes that map to symptom dimensions, developmental stage, and co-occurring conditions (Ameis & Catani, 2015).
3. Developmental and longitudinal designs: Infant-sibling cohorts and serial imaging demonstrate that early surface-area expansion and network reconfiguration precede behavioral diagnosis, offering avenues for early risk stratification (Hazlett et al., 2017).
4. Mechanism-to-treatment bridges: Imaging readouts are increasingly used as biomarkers of target engagement in trials (e.g., modulation of social/attention networks with behavioral interventions; pharmacologic modulation of E/I balance), and machine-learning models show above-chance prediction of individual phenotypes and outcomes—though clinical utility awaits prospective validation (Ameis & Catani, 2015; Just et al., 2012; Aoki et al., 2018).

In clinical practice, neuroimaging is used primarily to exclude alternative/organic etiologies (e.g., structural malformations, lesions) rather than to diagnose ASD. Group-level effects are small, heterogeneous, and sensitive to confounds (motion, sedation, comorbidity, medication), limiting individual-level inference (Ecker et al., 2015; Ameis & Catani, 2015). However, neuroimaging has reshaped mechanistic understanding by highlighting social brain network differences, atypical salience/interoception, sensorimotor contributions, and E/I imbalance, and is poised to aid psychiatry through individualized profiling, stratified trials, and objective markers of treatment engagement (Ameis & Catani, 2015; Just et al., 2012; Aoki et al., 2018; Ajram et al., 2017).

As a conclusion; across modalities, ASD converges on distributed network dysconnectivity and imbalanced excitatory-inhibitory signaling with marked developmental heterogeneity. While not yet a diagnostic tool, advances in large-scale

datasets, normative modeling, and mechanism-linked biomarkers are accelerating translation toward earlier risk identification and tailored interventions (Ecker et al., 2015; Ameis & Catani, 2015; Hazlett et al., 2017; Just et al., 2012; Aoki et al., 2018; Ajram et al., 2017).

Attention-Deficit/Hyperactivity Disorder

ADHD is a prevalent neurodevelopmental disorder marked by inattention and/or hyperactivity-impulsivity. Contemporary models emphasize dysfunction across fronto-striato-cerebellar circuits and large-scale networks (default mode, salience, and frontoparietal control). Neuroimaging has revealed convergent structural, functional, and molecular differences at the group level, though no marker is diagnostic in individuals (Castellanos & Proal, 2012; Cortese et al., 2012).

Structural MRI meta-analyses and consortia studies report subtle but reliable anatomical differences in ADHD. The ENIGMA-ADHD mega-analysis identified small reductions in subcortical volumes (notably nucleus accumbens, caudate, putamen, and hippocampus) with the largest effects in children and attenuated differences in adults (Hoogman et al., 2017). Cortical morphology studies show region-specific differences in thickness/surface area within prefrontal, cingulate, and temporal cortices; developmental work demonstrates a relative delay in cortical maturation—particularly in prefrontal regions—during childhood (Hoogman et al., 2019; Shaw et al., 2007). Diffusion MRI indicates widespread white-matter microstructural alterations across callosal fibers and fronto-limbic association tracts (e.g., uncinate fasciculus, cingulum, superior longitudinal fasciculus), consistent with a dysconnectivity framework (van Ewijk et al., 2012). Effect sizes are modest and moderated by age, symptom profile, comorbidity, and medication exposure (Hoogman et al., 2017; van Ewijk et al., 2012).

Task-based fMRI consistently shows reduced recruitment of right inferior frontal gyrus and dorsal anterior cingulate during inhibitory control and cognitive control tasks, along with altered activation in striatal and cerebellar nodes; these patterns align with impaired response inhibition and timing (Hart et al., 2013; Rubia et al., 2010). Resting-state fMRI reveals atypical interactions among large-scale networks—hyperconnectivity within the default mode network (DMN), reduced anti-correlation between DMN and task-positive networks, and altered salience-to-control coupling—supporting accounts of mind-wandering and variable attentional control (Castellanos & Proal, 2012; Fair et al., 2010). Molecular imaging studies (PET/SPECT) implicate catecholaminergic systems: abnormalities in dopamine transporter/receptor measures and fronto-striatal glucose metabolism have been reported, consistent with stimulant mechanisms (Ichikawa et al., 2016; Volkow et al., 2009; Wang et al., 2015). Proton MRS studies suggest alterations in glutamate/GABA balance in prefrontal-striatal circuits in subsets of patients, though findings are heterogeneous (Vidor et al., 2022).

Recent advances about ADHD are:

1. Multimodal and consortium science: Large, harmonized datasets (e.g., ENIGMA-ADHD) integrate sMRI, cortical metrics, diffusion, and functional measures, improving power and developmental modeling (Hoogman et al., 2017; Shaw et al., 2007).
2. Developmental trajectories and normative modeling: Longitudinal work maps delayed cortical maturation and evolving network organization; normative-deviation approaches quantify individual differences beyond average case–control effects (Hoogman et al., 2019; van Ewijk et al., 2012; Volkow et al., 2009).
3. Mechanisms of treatment: Pharmacological fMRI shows partial normalization of fronto-striatal activation and DMN control after methylphenidate or atomoxetine; connectomic changes correlate with symptom improvement (Hart et al., 2013; Wang et al., 2015).
4. Predictive modeling: Machine-learning studies achieve above-chance classification and early prediction of treatment response/clinical course using multivariate imaging features, though generalizability remains limited pending prospective validation and motion-artifact control (Cortese et al., 2012; Vidor et al., 2022).

In clinical practice, neuroimaging is used mainly to exclude alternative/organic etiologies; no scan is diagnostic for ADHD. Group-level effects are small and influenced by motion, comorbidity (e.g., learning disorders, anxiety), developmental stage, and medication status. Heterogeneity—both biological and phenotypic—limits single-modality biomarkers. Nevertheless, imaging has refined mechanistic models (fronto-striatal and network-level dysconnectivity), informed treatment mechanisms, and is advancing toward precision psychiatry through multimodal integration and

normative, developmentally sensitive analyses (Hart et al., 2013; Castellanos & Proal, 2012; Cortese et al., 2012; Hoogman et al., 2017; Shaw et al., 2007; Volkow et al., 2009; Wang et al., 2015; Vidor et al., 2022).

As a conclusion; across modalities, ADHD converges on fronto-striatal-cerebellar dysfunction and large-scale network dysregulation with developmental delay in cortical maturation. While not yet clinically diagnostic, advances in consortium-scale datasets, multimodal modeling, and treatment-mechanism imaging are paving the way for individualized risk stratification and treatment optimization (Hart et al., 2013; Castellanos & Proal, 2012; Cortese et al., 2012; Hoogman et al., 2017; Shaw et al., 2007; Volkow et al., 2009; Wang et al., 2015; Vidor et al., 2022).

Conclusion

This review demonstrates that advanced neuroimaging methods—including structural MRI, functional MRI, diffusion imaging, spectroscopy, and molecular imaging—have significantly enriched our understanding of major psychiatric disorders. Across conditions such as schizophrenia, major depressive disorder, bipolar disorder, obsessive–compulsive disorder, anxiety disorders, autism spectrum disorder, and attention-deficit/hyperactivity disorder, convergent findings consistently implicate abnormalities in fronto-limbic, cortico-striatal, and large-scale network systems. These results support transdiagnostic models emphasizing dysconnectivity, excitatory–inhibitory imbalance, and impaired top-down regulation as shared neurobiological mechanisms.

From a clinical perspective, neuroimaging has moved far beyond its initial role of excluding organic pathology. Although still not diagnostic at the individual level, imaging has refined theoretical models, identified potential biomarkers for disease course and treatment response, and informed the development of novel therapeutic strategies. Examples include circuit-guided neuromodulation in OCD, predictive modeling of antidepressant response in depression, and normative developmental mapping in ADHD and autism. Importantly, longitudinal and multimodal approaches highlight the dynamic nature of psychiatric illnesses, with brain alterations evolving across illness stages and treatment exposures.

Future research should prioritize harmonization across sites, the use of large-scale consortia, and integration of multimodal and computational methods. Normative modeling, machine learning, and personalized profiling approaches are particularly promising to move imaging findings from group-level research into actionable clinical tools. Moreover, linking neuroimaging readouts to molecular and behavioral data will be essential to build comprehensive models of disease mechanisms and to guide precision psychiatry.

In conclusion, neuroimaging has transformed psychiatry by uncovering consistent structural, functional, and neurochemical alterations across a range of disorders. While clinical translation remains incomplete, advances in large-scale data integration, predictive modeling, and circuit-based interventions are paving the way for earlier diagnosis, individualized treatment strategies, and improved patient outcomes.

References

Abbott, C. C., Jones, T., Lemke, N. T., Gallegos, P, McClintock, S. M., Mayer, A. R., … & Calhoun, V. D. (2014). Hippocampal structural and functional changes associated with electroconvulsive therapy response. Translational Psychiatry, 4, e483. https://doi.org/10.1038/tp.2014.124

Ajram, L. A., Horder, J., Mendez, M. A., Galanopoulos, A., Brennan, L. P., Wichers, R. H., … & McAlonan, G. M. (2017). Shifting brain inhibitory balance and connectivity of the prefrontal cortex of adults with autism spectrum disorder. Translational Psychiatry, 7, e1137. https://doi.org/10.1038/tp.2017.104

Ameis, S. H., & Catani, M. (2015). Altered white matter connectivity as a neural substrate for social impairment in autism spectrum disorder. Cortex, 62, 158–181. https://doi.org/10.1016/j.cortex.2014.10.014

Aoki, Y., Cortese, S., & Castellanos, F. X. (2018). Research review: Diffusion tensor imaging studies of attention-deficit/hyperactivity disorder: Meta-analyses and reflections on head motion. Journal of Child Psychology and Psychiatry, 59, 193–202. https://doi.org/10.1111/jcpp.12778

Arnone, D., McIntosh, A. M., Ebmeier, K. P., Munafò, M. R., & Anderson, I. M. (2012). Magnetic resonance imaging studies in unipolar depression: Systematic review and meta-regression analyses. European Neuropsychopharmacology, 22, 1–16. https://doi.org/10.1016/j.euroneuro.2011.05.003

Barch, D. M., & Ceaser, A. (2012). Cognition in schizophrenia: Core psychological and neural mechanisms. Trends in Cognitive Sciences, 16, 27–34. https://doi.org/10.1016/j.tics.2011.11.015

Baxter, L. R., Jr., Schwartz, J. M., Bergman, K. S., Szuba, M. P., Guze, B. H., Mazziotta, J. C., … & Munford, P. (1992). Caudate glucose metabolic rate changes with both drug and behavior therapy for obsessive–compulsive disorder. Archives of General Psychiatry, 49, 681–689. https://doi.org/10.1001/archpsyc.1992.01820090009002

Bishop, S. J. (2007). Neurocognitive mechanisms of anxiety: An integrative account. Trends in Cognitive Sciences, 11, 307–316. https://doi.org/10.1016/j.tics.2007.05.008

Bora, E., Fornito, A., Yücel, M., & Pantelis, C. (2010). Voxelwise meta-analysis of gray matter abnormalities in bipolar disorder. Biological Psychiatry, 67, 1097–1105. https://doi.org/10.1016/j.biopsych.2010.01.020

Brennan, B. P., Rauch, S. L., Jensen, J. E., & Pope, H. G., Jr. (2013). A critical review of magnetic resonance spectroscopy studies of obsessive–compulsive disorder. Biological Psychiatry, 73, 24–31. https://doi.org/10.1016/j.biopsych.2012.06.023

Brooks, J. O., 3rd, & Vizueta, N. (2014). Diagnostic and clinical implications of functional neuroimaging in bipolar disorder. Journal of Psychiatric Research, 57, 12–25. https://doi.org/10.1016/j.jpsychires.2014.05.018

Brühl, A. B., Delsignore, A., Komossa, K., & Weidt, S. (2014). Neuroimaging in social anxiety disorder—A meta-analytic review resulting in a new neurofunctional model. Neuroscience & Biobehavioral Reviews, 47, 260–280. https://doi.org/10.1016/j.neubiorev.2014.08.003

Carmi, L., Tendler, A., Bystritsky, A., Hollander, E., Blumberger, D. M., Daskalakis, J., … & Zohar, J. (2019). Efficacy and safety of deep transcranial magnetic stimulation for obsessive–compulsive disorder: A prospective multicenter randomized double-blind placebo-controlled trial. American Journal of Psychiatry, 176, 931–938. https://doi.org/10.1176/appi.ajp.2019.18101180

Castellanos, F. X., & Proal, E. (2012). Large-scale brain systems in ADHD: Beyond the prefrontal–striatal model. Trends in Cognitive Sciences, 16, 17–26. https://doi.org/10.1016/j.tics.2011.11.007

Chen, C. H., Suckling, J., Lennox, B. R., Ooi, C., & Bullmore, E. T. (2011). A quantitative meta-analysis of fMRI studies in bipolar disorder. Bipolar Disorders, 13, 1–15. https://doi.org/10.1111/j.1399-5618.2011.00893.x

Chitty, K. M., Lagopoulos, J., Lee, R. S., Hickie, I. B., & Hermens, D. F. (2013). A systematic review and meta-analysis of proton magnetic resonance spectroscopy and mismatch negativity in bipolar disorder. European Neuropsychopharmacology, 23, 1348–1363. https://doi.org/10.1016/j.euroneuro.2013.07.007

Cortese, S., Kelly, C., Chabernaud, C., Proal, E., Di Martino, A., Milham, M. P., & Castellanos, F. X. (2012). Toward systems neuroscience of ADHD: A meta-analysis of 55 fMRI studies. American Journal of Psychiatry, 169, 1038–1055. https://doi.org/10.1176/appi.ajp.2012.11101521

Dabiri, M., Dehghani Firouzabadi, F., Yang, K., Barker, P. B., Lee, R. R., & Yousem, D. M. (2022). Neuroimaging in schizophrenia: A review article. Frontiers in Neuroscience, 16, 1042814. https://doi.org/10.3389/fnins.2022.1042814

Denys, D., Mantione, M., Figee, M., van den Munckhof, P., Koerselman, F., Westenberg, H., … & Schuurman, R. (2010). Deep brain stimulation of the nucleus accumbens for treatment-refractory obsessive–compulsive disorder. Archives of General Psychiatry, 67, 1061–1068. https://doi.org/10.1001/archgenpsychiatry.2010.122

Di Martino, A., Yan, C. G., Li, Q., Denio, E., Castellanos, F. X., Alaerts, K., … & Milham, M. P. (2014). The autism brain imaging data exchange: Towards a large-scale evaluation of the intrinsic brain architecture in autism. Molecular Psychiatry, 19, 659–667. https://doi.org/10.1038/mp.2013.78

Ecker, C., Bookheimer, S. Y., & Murphy, D. G. (2015). Neuroimaging in autism spectrum disorder: Brain structure and function across the lifespan. The Lancet Neurology, 14, 1121–1134. https://doi.org/10.1016/S1474-4422

Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. American Journal of Psychiatry, 164, 1476–1488. https://doi.org/10.1176/appi.ajp.2007.07030504

Fair, D. A., Posner, J., Nagel, B. J., Bathula, D., Dias, T. G., Mills, K. L., … & Nigg, J. T. (2010). Atypical default network connectivity in youth with attention-deficit/hyperactivity disorder. Biological Psychiatry, 68, 1084–1091. https://doi.org/10.1016/j.biopsych.2010.07.003

Figee, M., Luigjes, J., Smolders, R., Valencia-Alfonso, C. E., van Wingen, G., de Kwaasteniet, B., … & Denys, D. (2013). Deep brain stimulation restores frontostriatal network activity in obsessive–compulsive disorder. Nature Neuroscience, 16, 386–387. https://doi.org/10.1038/nn.3344

Fox, A. S., & Shackman, A. J. (2019). The central extended amygdala in fear and anxiety: Closing the gap between mechanistic and neuroimaging research. Neuroscience Letters, 693, 58–67. https://doi.org/10.1016/j.neulet.2017.11.056

Frodl, T., Jäger, M., Smajstrlova, I., Born, C, Bottlender, R., Palladino, T., … & Meisenzahl, E. M. (2008). Effect of hippocampal and amygdala volumes on clinical outcomes in major depression: A 3-year prospective magnetic resonance imaging study. Journal of Psychiatry & Neuroscience, 33, 423–430. https://doi.org/10.1503/jpn.080005

Goddard, A. W., Mason, G. F., Almai, A., Rothman, D. L., Behar, K. L., Petroff, O. A., … & Krystal, J. H. (2001). Reductions in occipital cortex GABA levels in panic disorder detected with 1H-magnetic resonance spectroscopy. Archives of General Psychiatry, 58, 556–561. https://doi.org/10.1001/archpsyc.58.6.556

Gong, Q., & He, Y. (2015). Depression, neuroimaging and connectomics: A selective overview. Biological Psychiatry, 77, 223–235. https://doi.org/10.1016/j.biopsych.2014.08.009

Gürsel, D. A., Avram, M., Sorg, C., Brandl, F., & Koch, K. (2018). Frontoparietal areas link impairments of large-scale intrinsic brain networks with aberrant fronto-striatal interactions in OCD: A meta-analysis of resting-state functional connectivity. Neuroscience & Biobehavioral Reviews, 87, 151–160. https://doi.org/10.1016/j.neubiorev.2018.01.016

Hart, H., Radua, J., Nakao, T., Mataix-Cols, D., & Rubia, K. (2013). Meta-analysis of functional MRI studies of inhibition and attention in ADHD: Exploring task-specific, stimulant medication, and age effects. JAMA Psychiatry, 70, 185–198. https://doi.org/10.1001/jamapsychiatry.2013.277

Hazlett, H. C., Gu, H., Munsell, B. C., Kim, S. H., Styner, M., Wolff, J. J., … & Piven, J.; IBIS Network. (2017). Early brain development in infants at high risk for autism spectrum disorder. Nature, 542, 348–351. https://doi.org/10.1038/nature21369

Hibar, D. P., Westlye, L. T., Doan, N. T., Jahanshad, N., Cheung, J. W., Ching, C. R. K., … & Andreassen, O. A. (2018). Cortical abnormalities in bipolar disorder: An MRI analysis of 6503 individuals from the ENIGMA Bipolar Disorder Working Group. Molecular Psychiatry, 23, 932–942. https://doi.org/10.1038/mp.2017.73

Hibar, D. P., Westlye, L. T., van Erp, T. G. M., Rasmussen, J., Leonardo, C. D., Faskowitz, J., … & Andreassen, O. A. (2016). Subcortical volumetric abnormalities in bipolar disorder. Molecular Psychiatry, 21, 1710–1716. https://doi.org/10.1038/mp.2015.227

Ho, B. C., Andreasen, N. C., Ziebell, S, Pierson, R., & Magnotta, V. (2011). Long-term antipsychotic treatment and brain volumes: A longitudinal study of first-episode schizophrenia. Archives of General Psychiatry, 68, 128–137. https://doi.org/10.1001/archgenpsychiatry.2010.199

Hoogman, M., Bralten, J., Hibar, D. P., Mennes, M., Zwiers, M. P., Schweren, L. S., … & Franke, B. (2017). Subcortical brain volume differences in participants with ADHD: A cross-sectional mega-analysis. The Lancet Psychiatry, 4, 310–319. https://doi.org/10.1016/S2215-0366 (Schmaal et al., 2016)30049-4

Hoogman, M., Muetzel, R., Guimaraes, J. P., Shumskaya, E., Mennes, M., Zwiers, M. P., … & Franke, B. (2019). Brain imaging of the cortex in ADHD: A coordinated analysis of large-scale clinical and population-based samples. American Journal of Psychiatry, 176, 531–542. https://doi.org/10.1176/appi.ajp.2019.18091033

Howes, O. D., & Kapur, S. (2009). The dopamine hypothesis of schizophrenia: Version III—the final common pathway. Schizophrenia Bulletin, 35, 549–562. https://doi.org/10.1093/schbul/sbp006

Ichikawa, N., Okamoto, Y., Okada, G., Lisi, G., Yahata, N., Morimoto, J., … & Yamawaki, S. (2016). Neuroimaging biomarker of major depressive disorder. European Psychiatry, 33, 492–493. https://doi.org/10.1016/j.eurpsy.2016.01.1811

Just, M. A., Keller, T. A., Malave, V. L., Kana, R. K., & Varma, S. (2012). Autism as a neural systems disorder: A theory of frontal–posterior underconnectivity. Neuroscience & Biobehavioral Reviews, 36, 1292–1313. https://doi.org/10.1016/j.neubiorev.2012.02.007

Kelly, S., Jahanshad, N., Zalesky, A., Kochunov, P., Agartz, I., Alloza, C., ... & Donohoe, G. (2018). Widespread white matter microstructural differences in schizophrenia across 4322 individuals: Results from the ENIGMA Schizophrenia DTI Working Group. Molecular Psychiatry, 23, 1261–1269. https://doi.org/10.1038/mp.2017.170

Kim, M. J., & Whalen, P. J. (2009). The structural integrity of an amygdala–prefrontal pathway predicts trait anxiety. Journal of Neuroscience, 29, 11614–11618. https://doi.org/10.1523/JNEUROSCI.2335-09.2009

Kupfer, D. J., Frank, E., & Phillips, M. L. (2012). Major depressive disorder: New clinical, neurobiological, and treatment perspectives. The Lancet, 379, 1045–1055. https://doi.org/10.1016/S0140-6736

Malizia, A. L., Cunningham, V. J., Bell, C. J., Liddle, P. F., Jones, T., & Nutt, D. J. (1998). Decreased brain GABA(A)-benzodiazepine receptor binding in panic disorder: Preliminary results from a quantitative PET study. Archives of General Psychiatry, 55, 715–720. https://doi.org/10.1001/archpsyc.55.8.715

Moore, G. J., Bebchuk, J. M., Hasanat, K., Chen, G., Seraji-Bozorgzad, N., Wilds, I. B., … & Manji, H. K. (2000). Lithium increases N-acetyl-aspartate in the human brain: In vivo evidence in support of bcl-2's neurotrophic effects? Biological Psychiatry, 48, 1–8. https://doi.org/10.1016/S0006-3223

Mulders, P. C., van Eijndhoven, P. F., Schene, A. H., Beckmann, C. F., & Tendolkar, I. (2015). Resting-state functional connectivity in major depressive disorder: A review. Neuroscience & Biobehavioral Reviews, 56, 330–344. https://doi.org/10.1016/j.neubiorev.2015.07.014

Mwangi, B., Wu, M. J., Cao, B., Passos, I. C., Lavagnino, L., Keser, Z., … & Soares, J. C. (2016). Individualized prediction and clinical staging of bipolar disorders using neuroanatomical biomarkers. Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 1, 186–194. https://doi.org/10.1016/j.bpsc.2016.01.001

Nikolaus, S., Müller, H. W., & Hautzel, H. (2017). Different patterns of dopaminergic and serotonergic dysfunction in manic, depressive and euthymic phases of bipolar disorder. Nuklearmedizin, 56, 191–200. https://doi.org/10.3413/Nukmed-0893-17-04

Nomi, J. S., & Uddin, L. Q. (2015). Developmental changes in large-scale network connectivity in autism. NeuroImage: Clinical, 7, 732–741. https://doi.org/10.1016/j.nicl.2015.02.024

Nortje, G., Stein, D. J., Radua, J., Mataix-Cols, D., & Horn, N. (2013). Systematic review and voxel-based meta-analysis of diffusion tensor imaging studies in bipolar disorder. Journal of Affective Disorders, 150, 192–200. https://doi.org/10.1016/j.jad.2013.05.034

Nugent, A. C., Farmer, C., Evans, J. W., Snider, S. L., Banerjee, D., & Zarate, C. A., Jr. (2019). Multimodal imaging reveals a complex pattern of dysfunction in corticolimbic pathways in major depressive disorder. Human Brain Mapping, 40, 3940–3950. https://doi.org/10.1002/hbm.24679

Nutt, D. J., & Malizia, A. L. (2001). New insights into the role of the GABA(A)-benzodiazepine receptor in psychiatric disorder. British Journal of Psychiatry, 179, 390–396. https://doi.org/10.1192/bjp.179.5.390

Onwordi, E. C., Halff, E. F., Whitehurst, T., Mansur, A., Cotel, M. C., Wells, L., ... & Howes, O. D. (2020). Synaptic density marker SV2A is reduced in schizophrenia patients and unaffected by antipsychotics in rats. Nature Communications, 11, 246. https://doi.org/10.1038/s41467-019-14122-0

Phillips, M. L., & Swartz, H. A. (2014). A critical appraisal of neuroimaging studies of bipolar disorder: Toward a new conceptualization of underlying neural circuitry and a road map for future research. American Journal of Psychiatry, 171, 829–843. https://doi.org/10.1176/appi.ajp.2014.13081008

Piras, F., Piras, F., Caltagirone, C., & Spalletta, G. (2013). Brain circuitries of obsessive–compulsive disorder: A systematic review and meta-analysis of diffusion tensor imaging studies. Neuroscience & Biobehavioral Reviews, 37, 2856–2877. https://doi.org/10.1016/j.neubiorev.2013.10.008

Ray, S., Pal, A. K., & Kundu, P. S. (2024). A brief review of the neuroimaging modalities in schizophrenia and their scope. Annals of Medical Science and Research, 3, 33–38. https://doi.org/10.4103/amsr.amsr_52_23

Rotge, J.-Y., Guehl, D., Dilharreguy, B., Cuny, E., Tignol, J., Bioulac, B., … & Aouizerate, B. (2008). Provocation of obsessive–compulsive symptoms: A quantitative voxel-based meta-analysis of functional neuroimaging studies. Journal of Psychiatry & Neuroscience, 33, 405–412. https://doi.org/10.1139/jpn.0838

Rubia, K., Cubillo, A., Smith, A. B., Woolley, J., Heyman, I., & Brammer, M. J. (2010). Disorder-specific dysfunction in right inferior prefrontal cortex during two inhibition tasks in boys with ADHD compared to boys with obsessive–compulsive disorder. Human Brain Mapping, 31, 287–299. https://doi.org/10.1002/hbm.20864

Saxena, S., & Rauch, S. L. (2000). Functional neuroimaging and the neuroanatomy of obsessive–compulsive disorder. Psychiatric Clinics of North America, 23, 563–586. https://doi.org/10.1016/S0193-953X (05)70181-7

Schmaal, L., Veltman, D. J., van Erp, T. G. M., Sämann, P. G., Frodl, T., Jahanshad, N., … & Hibar, D. P. (2016). Subcortical brain alterations in major depressive disorder: Findings from the ENIGMA Major Depressive Disorder Working Group. Molecular Psychiatry, 21, 806–812. https://doi.org/10.1038/mp.2015.69

Schwartz, J. M., Stoessel, P. W., Baxter, L. R., Jr., Martin, K. M., & Phelps, M. E. (1996). Systematic changes in cerebral glucose metabolic rate after successful behavior modification treatment of obsessive–compulsive disorder. Archives of General Psychiatry, 53, 109–113. https://doi.org/10.1001/archpsyc.1996.01830020023004

Shaw, P., Eckstrand, K., Sharp, W., Blumenthal, J., Lerch, J. P., Greenstein, D., … & Rapoport, J. L. (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. Proceedings of the National Academy of Sciences of the USA, 104, 19649–19654. https://doi.org/10.1073/pnas.0707741104

Stein, D. J., Costa, D. L. C., Lochner, C., Miguel, E. C., Reddy, Y. C. J., Shavitt, R. G., … & Simpson, H. B. (2019). Obsessive–compulsive disorder. Nature Reviews Disease Primers, 5, 52. https://doi.org/10.1038/s41572-019-0102-3

Stern, E. R., Welsh, R. C., Fitzgerald, K. D., Gehring, W. J., Lister, J. J., Himle, J. A., … & Taylor, S. F. (2011). Hyperactive error responses and altered connectivity in ventromedial and frontoinsular cortices in obsessive–compulsive disorder. Biological Psychiatry, 69, 583–591. https://doi.org/10.1016/j.biopsych.2010.09.048

Sui, J., Yu, Q., He, H., Pearlson, G. D., & Calhoun, V. D. (2012). A selective review of multimodal fusion methods in schizophrenia. Frontiers in Human Neuroscience, 6, 27. https://doi.org/10.3389/fnhum.2012.00027

Sun, D., Phillips, L., Velakoulis, D., Yung, A., McGorry, P. D., Wood, S. J., van Erp, T. G., Thompson, P. M., Toga, A. W., Cannon, T. D., … & Pantelis, C. (2009). Progressive brain structural changes mapped as psychosis develops in 'at risk' individuals. Schizophrenia Research, 108, 85–92. https://doi.org/10.1016/j.schres.2008.11.026

Syan, S. K., Smith, M., Frey, B. N., Remtulla, R., Kapczinski, F., Hall, G. B. C., & Minuzzi, L. (2018). Resting-state functional connectivity in individuals with bipolar disorder during clinical remission: A systematic review. Journal of Psychiatry & Neuroscience, 43, 298–316. https://doi.org/10.1503/jpn.170175

Sylvester, C. M., Corbetta, M., Raichle, M. E., Rodebaugh, T. L., Schlaggar, B. L., Sheline, Y. I., & … Lenze, E. J. (2012). Functional network dysfunction in anxiety and anxiety disorders. Trends in Neurosciences, 35, 527–535. https://doi.org/10.1016/j.tins.2012.04.012

Townsend, L., Pillinger, T., Selvaggi, P., Veronese, M., Turkheimer, F., & Howes, O. (2023). Brain glucose metabolism in schizophrenia: A systematic review and meta-analysis of 18FDG-PET studies in schizophrenia. Psychological Medicine, 53, 4880–4897. https://doi.org/10.1017/S003329172200174X

Vidor, M. V., Panzenhagen, A. C., Martins, A. R., Cupertino, R. B., Bandeira, C. E., Picon, F. A., … & Rohde, L. A. (2022). Emerging findings of glutamate–glutamine imbalance in the medial prefrontal cortex in attention-deficit/hyperactivity disorder: Systematic review and meta-analysis of spectroscopy studies. European Archives of Psychiatry and Clinical Neuroscience, 272, 1395–1411. https://doi.org/10.1007/s00406-021-01399-7

Vieira, S., Gong, Q. Y., Pinaya, W. H. L., Scarpazza, C., Tognin, S., Crespo-Facorro, B., … & Mechelli, A. (2020). Using machine learning and structural neuroimaging to detect first episode psychosis: Reconsidering the evidence. Schizophrenia Bulletin, 46, 17–26. https://doi.org/10.1093/schbul/sby189

Volkow, N. D., Wang, G.-J., Kollins, S. H., Wigal, T. L., Newcorn, J. H., Telang, F., … & Ernst, M. (2009). Methylphenidate's effects on dopamine and functional circuits: PET evidence for baseline-dependent effects. Journal of Neuroscience, 29, 6008–6017. https://doi.org/10.1523/JNEUROSCI.4461-11.2012

Wang, Z., Dai, Z., Gong, G., Zhou, C., & He, Y. (2015). Understanding structural–functional relationships in the human brain: A large-scale network perspective. The Neuroscientist, 21, 290–305. https://doi.org/10.1177/1073858414537560

de Wit, S. J., Alonso, P., Schweren, L., Mataix-Cols, D., Lochner, C., Menchón, J. M., … & van den Heuvel, O. A. (2014). Multicenter voxel-based morphometry mega-analysis of structural brain scans in obsessive–compulsive disorder. American Journal of Psychiatry, 171, 340–349. https://doi.org/10.1176/appi.ajp.2013.13040574

van Ewijk, H., Heslenfeld, D. J., Zwiers, M. P., Buitelaar, J. K., & Oosterlaan, J. (2012). Diffusion tensor imaging in attention deficit/hyperactivity disorder: A systematic review and meta-analysis. Neuroscience & Biobehavioral Reviews, 36, 1093–1106. https://doi.org/10.1016/j.neubiorev.2012.01.003

Yazar Katkıları: Tüm yazarlar ICMJE'in bir yazarda bulunmasını önerdiği tüm ölçütleri karşılamışlardır
Etik Onay: Bu yazı derleme çalışması olduğu için Etik Kuruldan onaya gerek yoktur.
Hakem Değerlendirmesi: Dış bağımsız.
Çıkar Çatışması: Yazarlar çıkar çatışması olmadığını beyan etmişlerdir.
Finansal Destek: Yazarlar finansal destek beyan etmemişlerdir

Author Contributions: All authors met criteria recommended by ICMJE for being an author
Ethical Approval: No ethical approval is required for this review article.
Peer-review: Externally peer-reviewed.
Conflict of Interest: The authors have declared that there is no conflict of interest.
Financial Disclosure: Authors declared no financial support.