

Spec. Dr. Ali TORABI, Assoc. Prof. Dr. Nadir KOÇAK, Assoc. Prof. Dr. Rukiye TEKDEMİR  
Asst. Prof. Dr. Özkan BAĞCI, Lecturer Dr. Süleyman NERGİZ, Prof. Dr. Tülün ÇORA  
Assoc. Prof. Dr. İbrahim Halil YILDIRIM, Sara RAFI EI GAVGANI

# BIPOLAR DISORDER

Biology, Genetics and  
Clinical Perspectives



SELCUK  
UNIVERSITY  
PRESS

---

# BIPOLAR DISORDER

## BIOLOGY, GENETICS AND CLINICAL PERSPECTIVES

---

AUTHORS

SPEC. DR. ALİ TORABI

ASSOC. PROF. DR. NADİR KOÇAK

ASSOC. PROF. DR. RUKİYE TEKDEMİR

ASST. PROF. DR. ÖZKAN BAĞCI

LECTURER DR. SÜLEYMAN NERGİZ

PROF. DR. TÜLÜN ÇORA

ASSOC. PROF. DR. İBRAHİM HALİL YILDIRIM

SARA RAFIEI GAVGANI



SELCUK  
UNIVERSITY  
PRESS

## BIPOLAR DISORDER: BIOLOGY, GENETICS AND CLINICAL PERSPECTIVES

### Authors:

Spec. Dr. Ali TORABI (ORCID: 0000-0002-1843-1592)

Assoc. Prof. Dr. Nadir KOÇAK (ORCID: 0000-0002-1104-1292)

Assoc. Prof. Dr. Rukiye TEKDEMİR (ORCID: 0000-0001-7912-5727)

Asst. Prof. Dr. Özkan BAĞCI (ORCID: 0000-0002-9896-6764)

Lecturer Dr. Süleyman NERGİZ (ORCID: 0000-0001-6979-3713)

Prof. Dr. Tülün ÇORA (ORCID: 0000-0001-9787-7519)

Assoc. Prof. Dr. İbrahim Halil YILDIRIM (ORCID: 0000-0001-5518-5004)

Sara RAFİEİ GAVGANI (ORCID: 0009-0001-8258-4817)

**Cover Design:** Tuğçe DELEN

**Selcuk University Press:** June, 2026

**ISBN:** 978-975-448-264-5

**DOI:** <https://doi.org/10.59726/SUPress/9789754482645>

**Keywords:** 1. Bipolar disorder, 2. Molecular genetics, 3. Neurobiology, 4. Clinical phenomenology, 5. Biomarkers.

**Cite This:** Torabi, A., Koçak, N., Tekdemir, R., Bağcı, Ö., Nergiz, S., Çora, T., Yıldırım, İ. H., Gavgani, S. R., (2026), Bipolar Disorder: Biology, Genetics and Clinical Perspectives, Selcuk University Press.



Selcuk University Press in under the body of Scientific Publications Coordinatorship.

**Publisher:** Selcuk University Press

**Publisher Certification Number:** 43463

**Scientific Publications Coordinator:** Prof. Dr. Tuncer ACAR

**Adress:** Selçuk Üniversitesi Yayınları, Alaeddin Keykubat Yerleşkesi, Akademi mah. Yeni İstanbul Cad. No:369

Posta Kodu: 42130, Selçuklu-Konya/Türkiye

**Web:** [yayinevi.selcuk.edu.tr](http://yayinevi.selcuk.edu.tr)

**E-posta:** [yayinevi@selcuk.edu.tr](mailto:yayinevi@selcuk.edu.tr)

**Phone:** 0 (332) 241 00 41



This work is licensed under the Creative Commons Attribution-NonCommercial 4.0 International (CC BY-NC 4.0). To view a copy of this license, visit <https://creativecommons.org/licenses/by-nc/4.0/>



This license allows for copying any part of the work for personal use, not commercial use, providing author attribution is clearly stated.

# Bipolar Disorder

Biology, Genetics, and Clinical Perspectives

---

*A Comprehensive Psychogenetic Review*

**NeuroPsyBiT Research Group**

RTSGD NeuroPsychogenetic Bipolar Türkiye

*in collaboration with*

Research and Treatment Society of Genetic Disorders (RTSGD)

---



*Dedicated to all individuals and families living with bipolar disorder,  
and to the researchers and clinicians who devote their careers  
to understanding and treating this complex condition.*

*"In the middle of difficulty lies opportunity."*

*— Albert Einstein*



# Table of Contents

Table of Contents .....	6
Preface .....	13
Historical Perspectives and the Evolution of the Bipolar Concept .....	16
1.1 Ancient Descriptions of Mania and Melancholia .....	16
1.2 The Nineteenth Century: Falret, Baillarger, and the Birth of Circular Insanity .....	16
1.3 Emil Kraepelin and the Manic-Depressive Concept .....	17
1.4 The Twentieth Century: From Manic-Depressive Illness to Bipolar Disorder .....	18
1.5 Contemporary Nosology and Future Directions .....	19
1.6 The Bipolar Spectrum: Expanding Boundaries and Controversies .....	20
1.7 Cultural and Cross-Cultural Perspectives on Bipolar Disorder .....	21
References for Chapter 1 .....	22
1.8 Epidemiology of Bipolar Disorder: Global Prevalence and Incidence .....	22
1.9 The Burden of Illness: Disability, Quality of Life, and Mortality .....	24
1.10 Bipolar Disorder Across the Lifespan: Childhood to Late Life .....	25
1.11 The Concept of Temperament and Its Relationship to Bipolar Disorder .....	26
1.12 Bipolar Disorder and the Concept of Recovery .....	28
1.13 Neuropsychological Endophenotypes and Premorbid Functioning .....	29
1.14 Health Economics and the Cost of Bipolar Disorder .....	30
Diagnostic Criteria and Classification Systems .....	32
2.1 DSM-5 Criteria for Bipolar I Disorder .....	32
2.2 DSM-5 Criteria for Bipolar II Disorder .....	33
2.3 Cyclothymic Disorder and Other Specified Bipolar Disorders .....	34
2.4 ICD-11 Classification of Bipolar Disorder .....	35
2.5 Screening Instruments and Rating Scales .....	35
2.6 The Diagnostic Delay Problem and Its Consequences .....	37
References for Chapter 2 .....	38
2.7 Dimensional Approaches and the RDoC Framework .....	38
2.8 Pediatric Bipolar Disorder: Diagnostic Controversies and Considerations .....	39
2.9 The Role of Biomarkers in Bipolar Disorder Diagnosis and Monitoring .....	40
2.10 The Assessment of Functioning and Quality of Life in Bipolar Disorder .....	42

2.11 Neuropsychological Assessment in Clinical Practice.....	43
2.12 Differential Diagnosis in Depth: Bipolar II vs Borderline Personality Disorder ...	44
<b>Phenomenology of Manic and Hypomanic Episodes .....</b>	<b>46</b>
3.1 The Core Features of Mania.....	46
3.2 Cognitive Symptoms of Mania: Grandiosity, Racing Thoughts, and Distractibility	46
3.3 Behavioral Manifestations and Psychosocial Consequences.....	47
3.4 Hypomania: Phenomenology and Diagnostic Challenges .....	48
3.5 Psychotic Features in Mania.....	49
3.6 Neurocognitive Profile During Manic Episodes.....	50
<b>References for Chapter 3 .....</b>	<b>50</b>
3.7 Creativity and Bipolar Disorder: Myth and Reality.....	51
3.8 Manic Episodes in the Digital Age: Social Media, Technology, and Online Behavior .....	52
3.9 The Impact of Mania on Family Systems and Caregivers.....	53
3.10 Emergency Management of Acute Mania.....	54
<b>Phenomenology of Bipolar Depressive Episodes.....</b>	<b>56</b>
4.1 Clinical Features of Bipolar Depression.....	56
4.2 Mixed Depressive Features and Agitated Depression .....	57
4.3 Suicidality in Bipolar Depression.....	58
4.4 Atypical Depression and Bipolar Depression: Differential Features.....	58
<b>References for Chapter 4 .....</b>	<b>59</b>
4.5 Cognitive Dysfunction in Bipolar Depression.....	60
4.6 Treatment-Resistant Bipolar Depression .....	61
4.7 The Neurobiological Basis of Anhedonia in Bipolar Depression .....	62
4.8 Distinguishing Bipolar from Unipolar Depression: Clinical Markers .....	63
4.9 Sleep Disturbances in Bipolar Depression: Clinical Assessment and Management .....	64
<b>Mixed States, Rapid Cycling, and Course Specifiers .....</b>	<b>66</b>
5.1 Mixed States: Historical and Contemporary Perspectives .....	66
5.2 Rapid Cycling: Definition, Epidemiology, and Clinical Significance.....	66
5.3 Course and Prognosis of Bipolar Disorder.....	68
<b>References for Chapter 5 .....</b>	<b>68</b>
5.4 Seasonal Patterns and Bipolar Disorder .....	69
5.5 The Kindling and Sensitization Model in Detail.....	70
5.6 Subsyndromal Symptoms and Interepisode Functioning .....	71

5.7 Pharmacological Treatment of Bipolar Disorder: An Overview .....	71
5.8 Psychoeducation and Self-Management Strategies .....	73
5.9 Maintenance Treatment Strategies and Long-Term Prognosis .....	74
<b>Comorbidities and Differential Diagnosis.....</b>	<b>77</b>
6.1 Psychiatric Comorbidities .....	77
6.2 Medical Comorbidities .....	78
6.3 Differential Diagnosis.....	79
References for Chapter 6 .....	80
6.4 Bipolar Disorder and Trauma: Complex Intersections .....	80
6.5 Bipolar Disorder and Substance Use: A Complex Bidirectional Relationship .....	81
6.6 The Stigma of Bipolar Disorder: Impact and Interventions.....	81
6.7 Physical Health Monitoring and Intervention: A Comprehensive Approach .....	83
6.8 Special Populations: Women, Elderly, and Cultural Considerations in Treatment.....	84
<b>Neuroanatomy and Neural Circuits in Bipolar Disorder.....</b>	<b>87</b>
7.1 The Prefrontal-Limbic Circuit Model.....	87
7.2 White Matter Integrity and Connectivity .....	88
7.3 Neuroimaging During Mood States .....	89
7.4 Structural Brain Abnormalities: Meta-Analytic Evidence .....	90
References for Chapter 7 .....	90
7.5 The Default Mode Network and Self-Referential Processing.....	91
7.6 Neuroimaging Advances: From Structure to Dynamic Networks .....	92
7.7 Neuroplasticity Mechanisms in Bipolar Disorder and Treatment Response .....	93
7.8 Emerging Neuroimaging Technologies and Their Potential for Bipolar Disorder .....	94
<b>Neurotransmitter Systems in Bipolar Disorder .....</b>	<b>96</b>
8.1 The Monoamine Systems: Serotonin, Norepinephrine, and Dopamine .....	96
8.2 Glutamate and GABA: The Excitatory-Inhibitory Balance .....	97
8.3 Second Messenger Systems and Intracellular Signaling .....	98
References for Chapter 8 .....	99
8.4 The Cholinergic-Adrenergic Balance Hypothesis.....	99
8.5 Neuropeptides and Neuromodulators .....	100
8.6 The Purinergic System and Uric Acid in Bipolar Disorder.....	101
8.7 The Endocannabinoid System in Bipolar Disorder.....	102
<b>Neuroendocrine Axes and Circadian Biology .....</b>	<b>104</b>
9.1 The Hypothalamic-Pituitary-Adrenal (HPA) Axis.....	104

9.2 Thyroid Axis and Bipolar Disorder .....	104
9.3 Circadian Rhythm Disruption .....	105
References for Chapter 9 .....	106
9.4 The Hypothalamic-Pituitary-Gonadal Axis and Sex Differences .....	106
9.5 Sleep Architecture and Sleep Disorders in Bipolar Disorder .....	107
9.6 Chronotherapeutics: Light Therapy, Dark Therapy, and Sleep Manipulation.....	109
<b>Neuroinflammation, Oxidative Stress, and Mitochondrial Dysfunction .....</b>	<b>111</b>
10.1 The Neuroinflammation Hypothesis.....	111
10.2 Oxidative Stress and Antioxidant Defenses.....	111
10.3 Mitochondrial Dysfunction.....	112
References for Chapter 10 .....	113
10.4 The Gut-Brain Axis and Microbiome in Bipolar Disorder.....	114
10.5 Calcium Signaling Abnormalities: A Unifying Cellular Mechanism.....	115
10.6 Neuroprogression: Evidence, Mechanisms, and Clinical Implications.....	115
10.7 The Kynurenine Pathway: Linking Inflammation, Neurotransmission, and Neurodegeneration .....	117
10.8 N-Acetylcysteine and Antioxidant Strategies: Clinical Evidence .....	118
<b>Heritability, Family Studies, Twin Studies, and Adoption Studies.....</b>	<b>121</b>
11.1 Evidence for Genetic Contribution: Family Studies.....	121
11.2 Twin Studies: Parsing Genetics from Environment.....	121
11.3 What Heritability Does and Does Not Tell Us .....	123
References for Chapter 11 .....	124
11.4 Endophenotype Strategies in Bipolar Disorder Research.....	124
11.5 Consanguinity Studies and Isolated Populations.....	125
11.6 Adoptee and High-Risk Studies: Clarifying Transmission Mechanisms.....	126
11.7 Population Genetics and Global Diversity in Bipolar Disorder .....	127
<b>Linkage Studies and Early Molecular Genetics .....</b>	<b>129</b>
12.1 The Linkage Era: Promises and Limitations .....	129
12.2 Chromosome Regions of Interest .....	129
12.3 Transition to Association Studies.....	130
References for Chapter 12 .....	131
12.4 The Amish Studies Revisited: Lessons for Modern Genetics .....	131
<b>Genome-Wide Association Studies and Common Variant Architecture .....</b>	<b>133</b>
13.1 The GWAS Revolution in Bipolar Disorder .....	133
13.2 Key GWAS Loci and Implicated Genes .....	134

13.3 Polygenic Risk Scores and Cross-Disorder Genetics .....	135
References for Chapter 13 .....	136
13.4 Whole-Exome and Whole-Genome Sequencing Studies .....	136
13.5 Transcriptomics and Gene Expression Studies .....	137
13.6 The Psychiatric Genomics Consortium and International Collaboration .....	138
13.7 Mendelian Randomization and Causal Inference in Bipolar Research .....	139
<b>Candidate Genes: From Hypothesis to Evidence</b> .....	<b>141</b>
14.1 Monoaminergic Candidate Genes .....	141
14.2 Ion Channel and Signaling Pathway Genes .....	141
14.3 Clock Genes and Circadian Candidates .....	142
References for Chapter 14 .....	143
14.4 Immune and Inflammatory Candidate Genes .....	144
14.5 Mitochondrial Genes and Energy Metabolism Candidates .....	144
14.6 Convergent Evidence and Pathway Validation .....	145
<b>Epigenetics: Beyond the DNA Sequence</b> .....	<b>147</b>
15.1 Epigenetic Mechanisms: DNA Methylation, Histone Modification, and Non-Coding RNA.....	147
15.2 Environmental Epigenetics: Stress, Trauma, and Medication Effects .....	148
15.3 Epigenetic Clocks and Biological Aging in Bipolar Disorder .....	149
References for Chapter 15 .....	150
15.4 Pharmacoepiggenetics: How Mood Stabilizers Modify the Epigenome .....	150
15.5 Single-Cell Epigenomics and Spatial Epigenetic Mapping.....	151
15.6 Transgenerational Epigenetic Inheritance and Intergenerational Trauma .....	152
<b>Gene-Environment Interactions and the Exposome</b> .....	<b>154</b>
16.1 Environmental Risk Factors for Bipolar Disorder .....	154
16.2 Gene-Environment Interaction Models .....	155
16.3 Developmental Perspectives and Sensitive Periods .....	155
References for Chapter 16 .....	156
16.4 The Microbiome as an Environmental Factor .....	156
16.5 The Developmental Origins of Bipolar Disorder: A Neurodevelopmental Perspective.....	157
16.6 Urbanization, Migration, and Social Determinants of Bipolar Disorder .....	158
<b>Rare Variants, Structural Variants, and the Missing Heritability</b> .....	<b>162</b>
17.1 Rare Variant Contributions .....	162
17.2 Copy Number Variants and Structural Genomic Variation.....	162

17.3 Accounting for the Missing Heritability .....	163
References for Chapter 17 .....	164
17.4 The Genetics of Lithium Response.....	164
17.5 Somatic Mosaicism and Post-Zygotic Mutations.....	165
17.6 The Future of Bipolar Disorder Genomics: Emerging Technologies.....	166
<b>Molecular Clustering Models and Phenotype-Genotype Integration .....</b>	<b>168</b>
18.1 Pathway-Based Clustering of Genetic Findings.....	168
18.2 Mapping Clusters to Clinical Phenotypes .....	169
18.3 Toward a Precision Psychiatry Framework.....	170
References for Chapter 18 .....	171
18.4 Pharmacogenomics of Bipolar Disorder: Current Evidence.....	171
18.5 Biological Staging of Bipolar Disorder: An Integrative Framework.....	172
18.6 Toward Biomarker-Guided Treatment Selection.....	173
<b>Novel Hypotheses and Integrative Models.....</b>	<b>176</b>
19.1 The Network Dysregulation Hypothesis.....	176
19.2 The Psychogenetic Vulnerability-Threshold Model.....	176
19.3 Future Directions: From Hypotheses to Testable Predictions.....	178
References for Chapter 19 .....	179
19.4 The Role of Artificial Intelligence and Computational Approaches.....	179
19.5 Translational Priorities and the Bench-to-Bedside Gap .....	180
19.6 Ethical Considerations in Bipolar Disorder Genomics.....	181
19.7 The Promise of Induced Pluripotent Stem Cell Models.....	183
<b>Summary of Key Learning Points and Conclusions .....</b>	<b>185</b>
20.1 Clinical Foundations — What We Know .....	185
20.2 Neurobiology — What We Understand .....	185
20.3 Genetics and Molecular Biology — What We Have Discovered.....	186
20.4 Integration and Future Directions — Where We Are Going .....	186
References for Chapter 20 .....	187
20.5 Recommendations for Clinical Practice .....	188
20.6 Open Questions and Future Research Priorities .....	189
20.7 A Message to Patients and Families.....	190
20.8 Glossary of Key Terms .....	191
<b>Word Index.....</b>	<b>193</b>
<b>About NeuroPsyBIT.....</b>	<b>196</b>

## Preface

Bipolar disorder is one of the most complex and debilitating psychiatric conditions known to humankind. Affecting approximately 1–2% of the global population, it is characterized by extreme oscillations in mood, energy, and cognition that profoundly disrupt the lives of those affected and their families. Despite centuries of clinical observation and decades of intense research, the precise pathophysiology of bipolar disorder remains an open question, a testament to the staggering complexity of the human brain and genome.

This book was conceived from a desire to bridge the ever-widening gap between clinical psychiatry and molecular genetics. In our experience as clinicians and researchers associated with NeuroPsyBiT (RTSGD NeuroPsychogenetic Bipolar Türkiye) and the Research and Treatment Society of Genetic Disorders (RTSGD), we have observed that while remarkable progress has been made in understanding the genetic architecture of bipolar disorder, much of this knowledge remains siloed within specialized journals and inaccessible to clinicians, trainees, and researchers in adjacent fields.

Our approach in this volume is deliberately pedagogical. We begin with the clinical foundations — the historical evolution of the bipolar concept, the modern diagnostic criteria, the rich phenomenology of manic and depressive episodes, and the nuanced sub-phenotypic features that make each patient's presentation unique. We believe that a firm grounding in clinical reality is essential before venturing into the molecular realm, for it is the clinical phenotype that ultimately defines the target of genetic investigation.

From this clinical base, we build upward through the neurobiology of bipolar disorder — examining the neuroanatomical circuits, neurotransmitter systems, neuroendocrine axes, neuroimaging findings, circadian biology, and the rapidly expanding field of neuroinflammation. Each of these domains provides critical clues about the biological substrates that genetic variants must ultimately influence.

We then turn to the genetic and molecular biology of bipolar disorder. Beginning with the foundational evidence from family, twin, and adoption studies, we trace the evolution of genetic approaches from linkage analysis through the genome-wide association study (GWAS) era and into the current landscape of whole-exome and whole-genome sequencing. We examine in detail the candidate genes that have emerged from decades of investigation, the common and rare variant architectures, and the increasingly important roles of epigenetics and gene-environment interactions.

In the final section, we attempt what we consider the most important and challenging task: integrating these disparate streams of evidence into coherent molecular clustering models. We propose hypothetical frameworks that link specific genetic variants to neurobiological pathways and, ultimately, to clinical phenotypes. These hypotheses are offered not as definitive conclusions but as starting points for future investigation, and we fully expect that many will be refined, revised, or replaced as the field advances.

Throughout this book, we have strived to present the current literature fairly and accurately, acknowledging areas of consensus and controversy with equal honesty. Each

chapter concludes with key learning points designed to consolidate the reader's understanding and facilitate review.

We hope this volume serves as a useful companion for psychiatrists, geneticists, neuroscientists, trainees, and all who seek to understand the biological foundations of bipolar disorder. The journey from bedside to bench and back again is long and winding, but it is a journey that holds the promise of transforming the lives of millions.

NeuroPsyBIT Research Group March 2026

---

**PART I**  
**CLINICAL FOUNDATIONS**

---

## CHAPTER 1

# Historical Perspectives and the Evolution of the Bipolar Concept

---

### 1.1 Ancient Descriptions of Mania and Melancholia

The recognition that extreme states of elation and profound sadness represent distinct yet related conditions extends back to the earliest written records of medical thought. Ancient Greek physicians, most notably Hippocrates (c. 460–370 BCE), described mania and melancholia as two fundamental disturbances of temperament. Hippocrates attributed these states to imbalances in the four humors — specifically, an excess of yellow bile producing mania and an excess of black bile (*melas kholé*) producing melancholia. While these humoral theories have long since been superseded, the clinical observations underlying them were remarkably astute.

Aretaeus of Cappadocia, writing in the first century CE, is widely credited as the first physician to explicitly link mania and melancholia as manifestations of a single disease process. In his treatise “On the Causes and Signs of Chronic Diseases,” Aretaeus observed that melancholia could transform into mania and that both conditions shared a common underlying etiology. This insight, revolutionary for its time, would not be fully elaborated upon for nearly two millennia.

The Roman physician Galen (129–216 CE) further refined humoral theory and distinguished several subtypes of melancholia, including forms associated with fear, despondency, and misanthropy. Throughout the medieval period, however, these observations were largely subsumed into religious and supernatural explanations of mental disturbance, with conditions we would now recognize as bipolar disorder attributed to demonic possession, moral failing, or divine punishment.

The Renaissance and early modern period saw a gradual return to naturalistic explanations of mental illness. Robert Burton’s monumental work “The Anatomy of Melancholy” (1621) provided an encyclopedic account of depressive states, though it did not clearly distinguish melancholia as a phase within a cyclical illness. Théophile Bonet, in the seventeenth century, made an important contribution by linking mania and melancholia under the term “manico-melancolicus,” recognizing their alternation within the same patient.

### 1.2 The Nineteenth Century: Falret, Baillarger, and the Birth of Circular Insanity

The modern conceptualization of bipolar disorder as a distinct nosological entity began to crystallize in mid-nineteenth century France. In 1851, Jean-Pierre Falret described “la folie circulaire” (circular insanity), a condition characterized by the regular alternation of mania and melancholia with intervening periods of lucidity. Falret’s contribution was

## Word Index

### A

AKAP11  
ANKK3  
Amygdala  
Anterior cingulate cortex  
Antidepressants  
Antipsychotics  
Anxiety disorders

### B

BDNF  
Bipolar I Disorder  
Bipolar II Disorder  
Bipolar spectrum

### C

CACNA1C  
CLOCK gene  
COMT  
Calcium channels  
Candidate genes  
Circadian rhythm  
Copy number variants  
Cyclothymia  
Cytokines

### D

DNA methylation  
DSM-5  
Depression  
Dopamine

### E

Endophenotype  
Epigenetics  
Excitatory-inhibitory balance

### F

FKBP5  
Functional connectivity

### G

GABA  
GSK-3  
GNAS  
Gene-environment interaction  
Genetic correlation  
Genome-wide association study

Glutamate  
Grandiosity

## H

HPA axis  
Heritability  
Hippocampus  
Histone modification  
Hypomania  
Hypothyroidism

## I

KCO-11  
IL-6  
Ion channels

## K

Ketamine  
Kindling  
Kraepelin

## L

Lamotrigine  
Linkage studies  
Lithium

## M

Mania  
Melatonin  
MicroRNA  
Mitochondrial dysfunction  
Mixed features  
Mixed states  
Monoamines  
Mood stabilizers

## N

NMDA receptor  
Neuroanatomy  
Neuroimaging  
Neuroinflammation  
Neuroprogression  
Norepinephrine

## O

Oxidative stress

## P

Pharmacogenomics  
Polygenic risk score  
Prefrontal cortex  
Psychosis

## R

Rapid cycling

Research Domain Criteria

**S**

SLC6A4

Schizoaffective disorder

Schizophrenia

Serotonin

Substance use disorders

Suicide

Synaptic plasticity

**T**

TNF-alpha

Thyroid

Twin studies

**V**

Valproate

**W**

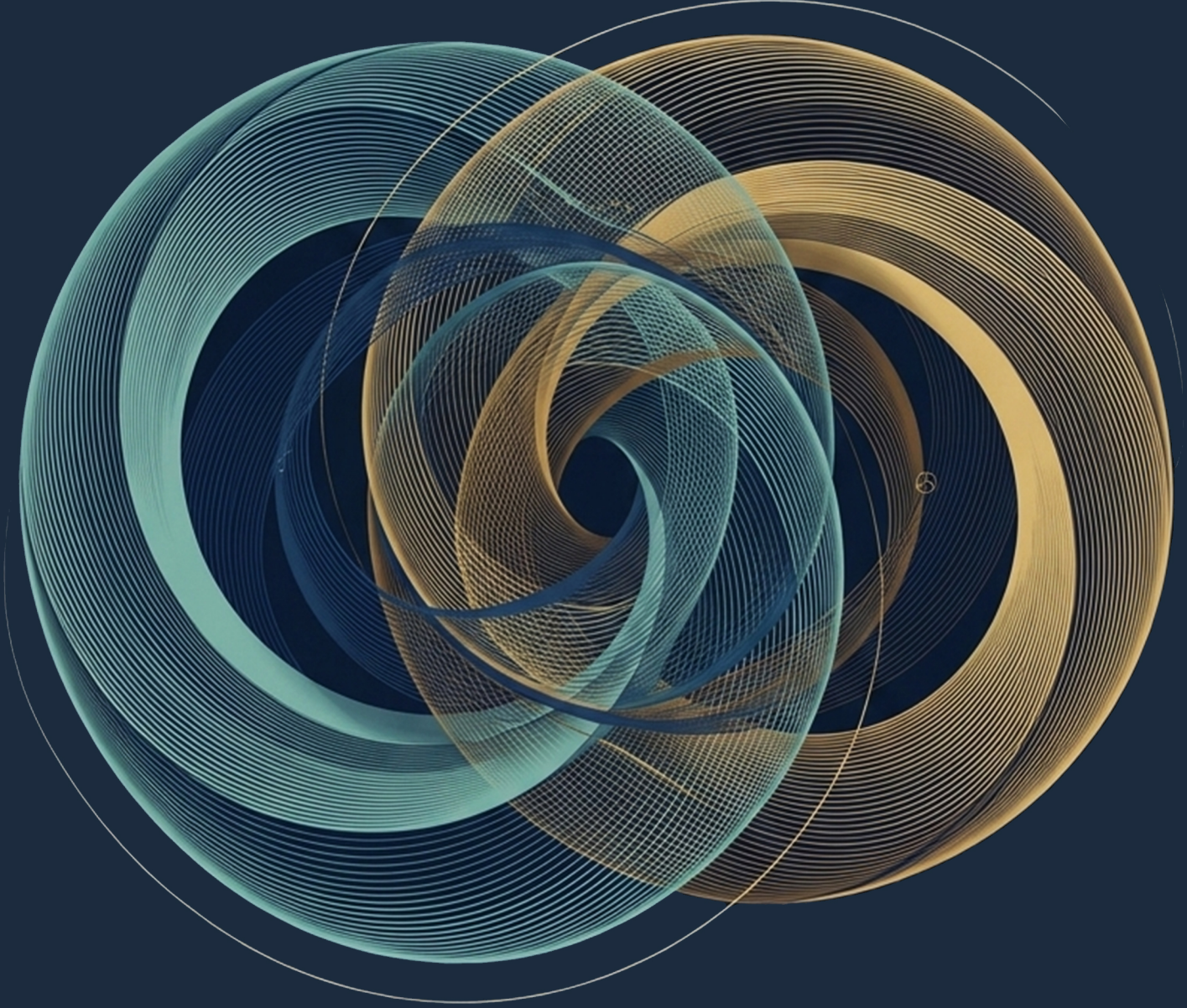
White matter

## About NeuroPsyBIT

NeuroPsyBIT (RTSGD NeuroPsychogenetic Bipolar Türkiye) is a research initiative dedicated to advancing the understanding of the genetic and neurobiological foundations of bipolar disorder. Operating in collaboration with the Research and Treatment Society of Genetic Disorders (RTSGD), NeuroPsyBIT integrates clinical psychiatry, molecular genetics, and neuroscience to develop integrative models of bipolar disorder pathophysiology and to translate genetic findings into improved clinical practice.

For more information, visit: [neuropsychit.org](http://neuropsychit.org)

---



SELÇUK  
UNIVERSITY  
PRESS

ISBN: 978-9-75448-264-5

9 789754 482645

Spec. Dr. Ali TORABI, Assoc. Prof. Dr. Nadir KOÇAK, Assoc. Prof. Dr. Rukiye TEKDEMİR  
Asst. Prof. Dr. Özkan BAĞCI, Lecturer Dr. Süleyman NERGİZ, Prof. Dr. Tülün ÇORA  
Assoc. Prof. Dr. İbrahim Halil YILDIRIM, Sara RAFIEI GAVGANI

# BIPOLAR DISORDER

## Biology, Genetics and Clinical Perspectives

BIPOLAR DISORDER

BIOLOGY, GENETICS, AND CLINICAL PERSPECTIVES



2026

