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MESSAGE



Dr. Sanjay Kumar Saha

Honorary Secretary
Indian Association of Private Psychiatry,
West Bengal State Branch

To,

Prof. Ranjan Bhattacharyya,
Honorary Editor
Bengal Journal of Private Psychiatry
Indian Association of Private Psychiatry West Bengal State Branch

Dear Prof Bhattacharyya,

On behalf of Indian Association of Private Psychiatry West Bengal State Branch, as the Honorary Secretary, let me take this opportunity to congratulate you on your tenure as the Honorary Editor of Bengal Journal of Private Psychiatry of Indian Association of Private Psychiatry West Bengal State Branch.

Since you have taken over, the journal has reached new heights, and we have had consistent publications along with relevant and noteworthy articles required to a private practitioner.

As the secretary of Indian Association of Private Psychiatry West Bengal State Branch, with support from our President, **Dr. Arabinda Brahma**, it has always been our endeavor that our association strives to enhance the knowledge and skills of a private practitioner of psychiatry in West Bengal. Accordingly, we have always curated our educational programs to this need with involvement of maximum members with a mix of dynamic youngsters and experienced seniors.

Your dedication and hard work has helped the Bengal Journal of Private Psychiatry to flourish and as I bid adieu from this post of Honorary Secretary, I wish you and the journal more successes in future.

Yours sincerely,



Dr. Sanjay Kumar Saha
Honorary Secretary
Indian Association of Private Psychiatry
West Bengal State Branch



Dr. Ranjan Bhattacharyya

Hony. Editor

Bengal Journal of Private Psychiatry

To

The President, Vice President, Hony Secretary, Hony Treasurer, Esteemed EC members, Life fellows and members of IAPP, West Bengal State Branch.

Dear Sir/Madam,

Bengal Journal of Private Psychiatry (BJPP) is the official publication and journal of Indian Association of Private Psychiatry, West Bengal State Branch (IAPP WBSB). It's a unique and challenging opportunity for me when I was assigned the task of being the Hony Editor of BJPP, the official mouthpiece of IAPP WBSB. I love this back-office staff as I was familiar while doing the work as editorial board members of IJPP, JIMA, Your Health & BJPP. But there is no state journal of IAPP in the country which sustained so long despite many challenges.

At the outset, my challenge was to make the journal regularized and viable. I am extremely thankful to all the contributors who have sent original research articles, pictorial CME, case reports, case series, meta-analysis or systemic reviews in three consecutive years. I am indebted to all contributors to share their intellectual property to get published in BJPP.

I am especially grateful to all the EC members of IAPP West Bengal State Branch who have shown trust on me. This time the job is more challenging being the organizing secretary of PSYCON 2026.

Two unique things have happened this year.

1. Firstly, the BJPP crossed 100 pages this year.
2. Secondly, we've created website of Bengal Journal of Private Psychiatry with archiving of past issues. You may click to download past issues.www.bjppjournal.com

I hope, I will be able to live up to your expectation in future as well. Kindly submit your research articles, experience and viewpoints in BJPP, because learning is a continuous and bidirectional process which gives us immense dividend when you share.

Enjoy reading the current edition of BJPP published in PSYCON 2026 and give your valuable feedback.

Long live BJPP.

Long live IAPP, West Bengal State Branch.



Prof (Dr) Ranjan Bhattacharyya

Hony Editor, Bengal Journal of Private Psychiatry



Dr. Sanjay Garg

Imm. Past Secretary

Indian Association of Private Psychiatry, West Bengal State Branch

Dear Dr. Ranjan Bhattacharyya,

It gives me immense pleasure to convey my warm congratulations and sincere appreciation for your valuable contribution as the **Editor of the Bengal Journal of Private Psychiatry**.

Academic journals play a vital role in nurturing professional excellence, encouraging scientific enquiry, and creating a meaningful platform for clinicians to share their experiences and perspectives. Your efforts as Editor have contributed significantly to advancing these objectives and to giving greater visibility to the academic and professional work of psychiatrists in private practice.

Through your commitment, editorial vision, and consistent efforts, the journal has served not merely as a repository of articles, but as an important forum for **clinical knowledge, research, professional reflection, and exchange of ideas**. Your ability to bring together diverse perspectives while maintaining academic standards is particularly commendable.

The work of an editor often happens quietly behind the scenes, requiring considerable time, patience, judgement, and dedication. Your contribution to strengthening the journal and promoting a culture of academic engagement among members of the **Indian Association of Private Psychiatry** is therefore especially worthy of recognition.

I am confident that, under your continued guidance, the journal will achieve even greater reach and distinction and will further enrich the academic landscape of Indian psychiatry.

Please accept my heartfelt congratulations and best wishes for your continued success. I look forward to seeing the journal scale new heights in the years ahead.

With warm regards and best wishes,

Long live IAPP.

Dr. Sanjay Garg

Imm. Past Secretary

Indian Association of Private Psychiatry

West Bengal State Branch

Memorabilia

Dr AB Dutta, MBBS(Cal), DPM, FRCPsych(Lond)
Consultant Psychiatrist, Duttanagar Mental Health Centre, Calcutta

I thank the organizing committee of PSYCON 2023 for inviting me to the inaugural session of the conference in Darjeeling District, where the West Bengal State Branch of the Indian Psychiatric Society was born on 31st October, 1985 in my presence.

I am taking this opportunity to recount the journey to the formation of Zonal and State branches of the IPS, many of the present generation of psychiatrists in the country may not know, was my brainchild.

Indian Psychiatric Society (IPS), originally established on January 23, 1939 in Lahore as the Indian Division of the Royal Medico-psychological Association (RMPA), the forerunner of the current Royal College of Psychiatrists, London with Dr. J. N. Dhunibhoy elected as its first President, was formed as an independent society on 7th January, 1947 in New Delhi with Dr. R. B. Davis as its President. The first annual conference of IPS was held in Patna in 1948. Dr. Nagendra Nath De, served the IPS as Editor of the Journal for the first five years and as President in 1948. Dr. J. Roy and Dr. B. L. Bhattacharyee were Presidents of IPS in 1949 and 1951 respectively. No other Fellow from West Bengal served the Society as its office bearer until 1962 when Dr. C. C. Saha became its President.

I came to know the Indian Psychiatric Society at a very young age, 73 years ago when I had the opportunity to attend the 3rd Annual Conference of the IPS in January 1950 as an accompanying person. I accompanied my father. It was held in a small hall of the Fergusson College in Pune at the venue of the Indian Science Congress. There were hardly 40 people, including most of the founder members of the IPS, at the conference which lasted only for a day. IPS was not connected with the Indian Science Congress Association in any manner whatsoever.

Ten years later, shortly after I had entered into the field of psychiatry, I was asked to serve the Organising Committee of the 13th Annual Conference of the IPS at Calcutta in 1960 as a junior office bearer but with a bigger responsibility. The conference was held at the first unaided private psychiatric hospital in India in Dum Dum, founded by my father Kaviraj Atul Behari Dutt. Among those who participated at that conference, no one except myself and Dr. O. Somasundaram who became my batch-mate at the Institute of Psychiatry, London at the Maudsley few months later, is alive to-day. Dr. Somasundaram went on to become President of the IPS. The Society had 56 Fellows, 61 Members and 28 Associate Members in 1959. Closing balance with the Hon. General Secretary on 31.12.1959 was Rs.135-83.

2

I joined the IPS as a Fellow in 1965 and closely looked into the activities of the Society including its very poor financial condition which necessitated increase in annual membership subscription rate and request for donation from members for publication of the journal at short intervals. I also noticed that in spite of being a national body of psychiatrists, IPS was being controlled by a small group of members working in some specific government hospitals or institutes.

The Executive Council of the society formed five “zones” of the society in 1971 to hold annual zonal conference in each zone and nominated Dr. L. P. Shah as Chairman of the Regional Committee to coordinate with the zones and to submit a report on the activities of the zones at the annual meeting of the Council. Formation of zones by the Executive Council, however, had no constitutional validity.

The Executive Council was formed at the annual general meeting of the Society and only 3 members constituted a quorum for a meeting. All office bearers had to bear their own travel & accommodation expenses. Only the General Secretary would get railway fare to attend the annual meeting of the Executive Council and President and General

Secretary were exempted from paying the registration fee for the annual conference.

The annual conference of the Society could be convened only by Fellows & Members of the Society who usually organized it either at a psychiatric hospital or under the banner of his department in a medical college or institute or by a newly formed State or local "Psychiatric Society" or a "Reception Committee" of the annual conference of the IPS. The IPS was not concerned with the assets and liabilities of the organizers. The organising committee could donate a part of their surplus amount to the IPS. The organising "society", usually paid Rs. 5/- for affiliated membership of the IPS and sometimes donated Rs. 5000/- from their surplus fund to the IPS to institute an award or even 2 awards for Rs. 10,000/- in the name of that Society. The office bearers of the organizing committee, however, worked extremely hard for a year to make the conference a memorable one. The Editor of the Journal of the Society was elected by the Executive Council. A registered medical practitioner engaged in whole time practice of psychiatry for more than 5 years or 2 years after obtaining a recognized post-graduate qualification in psychiatry could enroll himself as a Fellow of the IPS. In 1973-74 IPS had five affiliated societies -

1. Kerala Psychiatric Association
2. Poona Psychiatric Association
3. Indian Psychiatric Society, Mysore State Branch
4. Indian Psychiatric Society- Assam Branch, renamed as Assam Psychiatric Society
5. Institute of Community Psychiatry and Mental Health, Bombay

It may be mentioned that there was no provision for formation of a state branch of IPS in its constitution (Memorandum of Association and Rules & Regulations including Bye-laws) before 1984.

It must be understood that the IPS membership status of any category is not an academic qualification and should not be used as such by members along with his or her recognised academic qualifications.

3

At the AGB meeting of the IPS in 1975 in Trivandrum, I suggested, as Chairman of the Regional Committee, formation of zonal and local branches and representation from the zonal branches to the Executive Council of the IPS. AGB accepted my suggestion and asked me to submit proposal for constitutional amendment to incorporate my suggestions at the next AGB. I was nominated Regional Coordinator by the Executive Council. My proposals for constitutional amendments with provisions for 2 Zonal Representatives from each zonal branch to the Executive Council and 4 Direct Members elected by the Fellows of the Society were approved by the Executive Council in 1975 in Bangalore and by the AGB in 1976 in Nagpur. However, the task was not easy.

Apprehending lack of adequate support from zones in the election of office bearers and Executive Council members of the parent body, some office bearers and members working in transferable central government services did not want formation of zonal branches and election of zonal representatives. The minutes of the proceedings of the AGB meeting in 1975 were altered and constitutional amendments proposed by the Zonal Coordinator and accepted by an ordinary meeting of the Executive Council was not presented either at the annual meeting of the Council or at the AGB, presided over by the first lady President of the IPS Dr. Ajita Chakraborty, which was adjourned till next day even without confirming the minutes of the last AGB meeting or accepting the Secretary's report and the AGB asked the Executive Council to consider the proposed constitutional amendments immediately along with correct minutes & report of the General Secretary. The constitution of IPS was amended as proposed by me next day by the AGB and formation of zonal branches was approved.

A model zonal branch constitution for all the zonal branches was framed by me in 1975 in consultation with the former Zonal Secretaries, especially with Dr. L.P.Shah and Dr. G.C.Boral. This was readily accepted and adopted by members of all the zones and then approved by the Executive Council and implemented by the members of the zonal branches which elected their office bearers and representatives to the Executive Council of the IPS. The annual conference of the IPS came to be known as Annual National Conference of the IPS or famously as ANCIPS.

The zonal branches started functioning normally from 1978, two years after overcoming another major resistance from the same group of members who did not want formation of the zonal branches. Just after condoling the death of members during the previous year, the new President of the IPS made a very serious allegation against me based on a nicely cooked-up story at the AGB meeting in 1977 in Calcutta which made the members attending meeting very

angry and hostile and even prompted a very senior member like Dr. R. B. Davis to propose, without any previous notice at the fag end of the very noisy meeting, that “all constitutional amendments passed during last 2 to 3 years be treated as cancelled”. It was supported by much less than 2/3rd members attending the meeting without a quorum. Constitution was amended only a year ago to form the zonal branches.

Under the restrain order passed in Title Suit No.10 of 1978 in the Court of 2nd Munsif at Alipore (Dr.A.B.Dutt vs. Dr.J.S.Neki & others) the decisions taken at the most disgraceful AGB meeting of IPS held in 1977 could not be given effect to and the circulated minutes of the proceedings, except declaration of results of the election, could not be placed for confirmation at the next AGB meeting in 1978 in New Delhi.

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The case was, however, mutually settled out of the Court after five years on January 8, 1983 in Bombay as desired by the AGB and recorded in the following Terms of Settlement signed by Dr. Jaya Nagaraja, President & Dr. L.P.Shah Hony. General Secretary of the IPS and the petitioner –

“The Executive Council acting on behalf of Annual General Body of the Indian Psychiatric Society after reviewing all the relevant documents relating to the issues involved in the above case with a view to settle the issue out of the court have reached mutual agreement on the following terms :-

- 1) As directed by the A.G.B. of the Society held in Bombay, on 7.1.1983, the Executive Council carefully considered the complaints filed by Dr. A. B. Dutt against the former office-bearers of the Society relating to the alleged incident during Calcutta conference in 1977 and the minutes of the meeting as circulated previously.
- 2) The Council on enquiry found that the allegations made against Dr. A. B. Dutt were untrue. The Council, while acting on behalf of the A.G.B. is of opinion that the Calcutta A.G.B. in 1977 was not conducted in a proper manner.
- 3) In order to settle the matter out of the court, as directed by the A.G.B. the Council acting on behalf of the I.P.S. expresses deep regret to Dr. A. B. Dutt for any hurt caused to him at the meeting at Calcutta and by the circulation of the minutes of that meeting in their original form, and recalls these minutes except Item No. 5 from the entire membership of the Society.
- 4) In view of the above, Dr. A. B. Dutt has consented to withdraw the Court Case No. 10 of 1978 filed by him in the Court of 2nd Munsif, Alipore.5). The Honorary General Secretary shall circulate the terms of settlement to all Fellows/Members.
- 6) Dr. A. B. Dutt shall apply for the withdrawal of the case within 4 weeks of the receipt of the copy of the circular”

At its Post-AGB meeting in 1983 the IPS Executive Council decided to have a new constitution by repealing the old one and requested me to frame it as Chairman of the Constitution Committee. I accepted the assignment on being assured that the next AGB meeting would not be adjourned even after midnight without adopting the new constitution. It was a herculean job and I kept my words and the AGB also honored its own assurance by adopting the proposed new constitution within half an hour in January, 1984.

Along this eventful journey I earned the support of various esteemed colleagues. I could not accomplish this very hard task so quickly and smoothly without very active and whole hearted support of Dr. L. P. Shah who served the IPS as General Secretary & President and was recipient of the D. L. N. Murty Rao Oration award. Over the years Dr. & Mrs Shah became my close family friends and I had to make the sad journey to Mumbai to spend few hours at his bedside at the K.E.M Hospital 2 days before my good friend Dr. Shah left for his eternal abode.

5

Among many changes in the new constitution of the IPS, expansion of aims and objectives of the society, changes in minimum qualification and experience for enrollment as a Fellow, provisions for zonal representatives and direct members of the executive council, election of editor of the Journal of the IPS by the fellows of the society along with other office bearers instead of few members of the executive council, provisions of state branches under the zonal branches of the IPS, affiliation and restriction on affiliation with other societies and formation of Tribunal were made. A new clause relating to the fund, accumulated in the hand of the organizing committee of the ANCIPS, completely changed the financial conditions of the Society at all levels. To-day the national body of the IPS along with its zonal & state branches stand on a very strong financial base and is able to fulfill all its aim and objectives mentioned in its constitution.

On 31st October, 1985 members of the West Bengal Psychiatric Society, established in 1962 by IPS President Dr. C. C. Saha but not connected with IPS, met at Darjeeling and dissolved the said society and thereafter the members of the IPS in West Bengal formed the first state branch of the IPS, adopted the model IPS State Branch constitution which was drafted by me and made ready for presentation by Dr. Dinen Banerjee, elected its office bearers and applied to the national body of the IPS for approval through the IPS Eastern Zonal Branch which met at the same venue for its annual conference next day. The Executive Council of the IPS approved the formation of its West Bengal State Branch on October 31, 1985.

IPS West Bengal State Branch received the surplus amount of Rs. 1,00,000/- which was lying with Dr. Gobinda Sen, Treasurer of the Organising Committee of the ACIPS, 1977 held at Calcutta. This is first state branch of the IPS which organized the ANCIPS in 1987 and contributed 50% of the surplus amount of the conference fund to the headquarters' fund at the AGB meeting of IPS in 1988, as provided in the new constitution.

Since its formation, the IPS West Bengal State Branch has been under the control of very devoted members, initially under the guidance of senior fellows like Dr. Gobinda Sen, Dr. Subir Bal, Dr. G. C. Boral, Dr. M. M. Majumdar, Dr. A. B. Patra, who are no more with us, and subsequently under subsequently under Dr. A. B. Ghosh, Dr. R. R. Ghosh Roy. Dr. Ashim Mallick, Dr. Dinen Banerjee, , Dr. Amar Nath Mallick, Dr. Prabir Paul, and later on under the magnifying glass of the very bright stars of the Society Dr. Gautam Saha, who along with his very talented and devoted team comprising of Drs. Uday Choudhury, Abir Mukherjee, Malay Ghosal, Arabinda Bramha, Ranjan Bhattacharya, Rajarshi Ray, Sarmistha Chakraborty, Kaustav Chakraborty, Sabyasachi Mitra and many others have elevated the status of this branch to a very respectable level in both national & international fields. A small plant has now become a big banyan tree. I am proud of your achievements & delighted to see your contributions in different fields of mental health and also to national and international professional bodies at present through distinguished leaders from IPS Eastern Zonal Branch, e.g. Dr. Gautam Saha (WB), President of SAARC Psychiatric Federation and Dr. Vinay Kumar (Bihar), President of the IPS.

The Role of Leadership in Psychiatry: From Clinical Excellence to Systems Leadership

Dr Rajashree Ray, Professor and HOD, GIMSH, Durgapur

Introduction

Leadership has become increasingly important within medicine as healthcare systems have become more complex, multidisciplinary and interconnected. Psychiatry is especially dependent on good leadership as mental healthcare is delivered in clinical, social, organizational and policy contexts.

Psychiatrists work regularly with colleagues in medicine, nursing, psychology, social work, occupational therapy, primary care, public health and the voluntary sector. They also interact with patients, families, communities, administrators and policy makers. Therefore the traditional model of the psychiatrist as an autonomous clinical expert is inadequate to describe contemporary psychiatric practice. The modern psychiatrist can be a clinician, educator, researcher, service developer, advocate, mentor and organizational leader all at the same time.

The World Health Organization has put leadership and governance at the heart of efforts to strengthen mental health systems. Leadership is therefore not just an organizational issue; it is central to the ability of mental health systems to translate evidence and policy into effective care. Bhugra and colleagues were among the first psychiatric writers to make a clear distinction between leadership and management and to make a case explicitly for leadership development within psychiatry. Their book *Leadership in Psychiatry* was written specifically to address leadership, not management skills, in psychiatric and mental healthcare and covers clinical leadership, decision-making, team development, mentoring, gender and leadership, burnout, ethics and diversity.

This article looks at why leadership matters in psychiatry, what makes for effective psychiatric leadership and how future leaders can be developed.

What is Leadership in Psychiatry?

People tend to confuse leadership with authority, seniority or management. These concepts are overlapping but not synonymous. Management is mainly about planning, organizing, coordinating resources and ensuring processes are functioning well. Purpose, direction, influence, relationships and change are at the heart of leadership. A psychiatrist need not hold a formal leadership position to demonstrate leadership. Leadership is demonstrated by the trainee who recognizes a patient-safety issue and takes the lead on a quality-improvement project. A consultant demonstrates leadership by creating a psychologically safe multidisciplinary team. A psychiatrist who advocates for improved mental-health policy or challenges stigma is also exercising leadership.

Leadership can therefore be conceptualised at multiple levels: (Figure 1)

Clinical leadership – influencing clinical decisions and standards of care.

Team leadership – creating effective multidisciplinary teams.

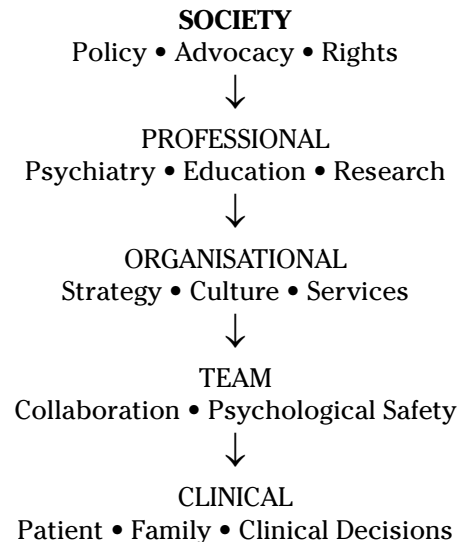
Organisational leadership – influencing services, structures and culture.

Professional leadership – contributing to psychiatric organisations and professional standards.

Societal leadership – advocating for mental health, human rights and public policy.

The Healthcare Leadership Model similarly conceptualises leadership as a set of behaviours including inspiring shared purpose, leading with care, engaging teams, developing capability, connecting services and influencing for results.

Figure 1. The Multilevel Model of Psychiatric Leadership



Leadership operates across all levels simultaneously

Figure legend: Psychiatric leadership is a multilevel process. Leadership at one level influences and is influenced by leadership at other levels. The model illustrates the movement from direct clinical influence to broader organisational, professional and societal impact.

Why Leadership Matters in Psychiatry

The impact of leadership can be considered across three interconnected domains: patients, professionals and systems.

Patients

Leadership influences the organisation and quality of clinical care. Psychiatric leaders have a role in the development of clinical pathways, quality-improvement initiatives, risk-management systems, multidisciplinary working and patient-centred services.

Mental health systems often require coordination across organizational boundaries. Effective leadership is therefore necessary to integrate hospital, community, primary-care and social-care services.

Professionals

Leadership has important implications for the psychiatric workforce. Poor organizational cultures are linked to disengagement, dissatisfaction and burnout, whilst supportive leadership can foster professional well-being.

A large study of physicians found a significant association between leadership quality and physician satisfaction and burnout. Leadership is therefore a workforce as well as an organizational issue. Psychiatry also involves considerable emotional labor. Professionals are regularly confronted with trauma, suicidality, chronic disability, social disadvantage and complex ethical dilemmas. Psychiatric leaders who understand these realities and create enabling environments can improve team resilience.

Systems

At a systems level, psychiatric leaders are increasingly called upon to influence policy, allocate funds, create new models of care and respond to technological and societal change. The rapid development of digital psychiatry,

artificial intelligence, neuroscience, precision medicine and community-based models of care means that psychiatric leadership must become increasingly adaptive. Leaders need to be able to deal with uncertainty, interpret evidence and mobilize people around change.

Psychological Safety and the Psychiatric Team

Psychiatry is a team-based activity. Hence, leadership cannot be understood in isolation from team culture. Psychological safety is a shared belief that the team is safe for interpersonal risk-taking. Edmondson showed a link between psychological safety and team learning behaviour.

For psychiatric teams, this has direct clinical relevance. Team members must feel able to say:

“I am concerned about this patient.” “I do not agree with this decision.”
 “I may have made an error.” “I do not understand the rationale.”
 “Can we reconsider this?”

A hierarchy in which junior staff are afraid to challenge senior clinicians can undermine both learning and patient safety. Effective psychiatric leaders therefore create an environment where challenge is not seen as disloyalty and uncertainty is not viewed as incompetence.

Compassionate and Inclusive Leadership

Traditionally healthcare leadership has been associated with authority, decisiveness and individual achievement. Contemporary approaches increasingly focus on relational, compassionate and inclusive leadership. Compassionate leadership does not mean avoiding accountability or difficult conversations. Rather, it combines high standards with sensitivity to the experiences and needs of colleagues. Psychiatry is particularly well placed to develop this model. At the core of psychiatric practice is an understanding of relationships, context, suffering, and meaning.

These skills can be transferred to organizational leadership. Inclusive leadership is equally important. Mental health services are increasingly delivering care to culturally, socially and economically diverse populations. Diverse leadership experiences may challenge institutional blind spots and foster greater responsiveness. Gender is a key dimension of diversity in leadership. In many countries, women are a large part of the psychiatric workforce, but representation may decrease progressively at senior organizational, educational, and professional levels. This cannot be solved simply by urging women to be more confident or ambitious. Structural issues of access to sponsorship, visibility, networks, committee opportunities and transparent promotion processes also need to be addressed.

The Competencies of the Contemporary Psychiatric Leader

Leadership competency should be viewed as multidimensional. (Figure 2)

Figure 2. The Psychiatric Leadership Competency Wheel



Figure legend: Effective psychiatric leadership requires integration of clinical expertise with interpersonal, organisational and strategic competencies. No single characteristic is sufficient.

Key competencies include:

Clinical credibility - Leadership in medicine cannot be divorced from professional credibility. Leaders need to understand the realities of clinical practice and stay connected to patients and teams.

Communication - Leaders need to communicate purpose, listen actively, manage difficult conversations and adapt communication for different audiences.

Emotional intelligence - Self-awareness, emotional regulation, empathy, and comprehension of interpersonal dynamics are particularly important in psychiatry.

Systems thinking - Psychiatric leaders need to understand how clinical decisions interact with organizational structures, financing, policy, workforce and wider social determinants.

Negotiation and influence - Influence increasingly occurs without direct authority. Leaders must build coalitions and negotiate across professional and organizational boundaries.

Change management - Change creates uncertainty and resistance. Leaders need to explain why change is necessary, involve stakeholders and maintain momentum.

Mentoring and Developing Others - Leadership is ultimately a developmental activity. A leader who does not develop future leaders will have little impact on the organisation.

Developing Future Leaders

Leadership development should not start when a person becomes a consultant, clinical director or head of department. It should begin in the postgraduate training years. A comprehensive leadership curriculum can include five elements. (Figure 2)

1. Self-awareness – awareness of your values, strengths, limitations and leadership style.
2. Experiential learning – involvement in quality improvement, teaching, research, service development and professional organisations.
3. Mentoring: offering guidance, perspective and career support.
4. Sponsorship: Proactively making opportunities and championing emerging leaders.
5. Feedback and reflection: structured feedback, including 360-degree assessment where appropriate, and reflective practice.

Leadership development should therefore be longitudinal rather than episodic

Figure 3. The Leadership Development Continuum

Figure legend: Leadership development is a continuum rather than a single transition into a senior post. Each career stage should provide opportunities for increasing responsibility, reflection, mentorship and influence.

Leadership, Women and the Future of Psychiatry

Women psychiatrists are increasingly assuming influential clinical, academic, and professional roles. Nevertheless, leadership pathways may remain affected by unequal access to informal networks, sponsorship, recognition and opportunities. A modern approach should move beyond the concept of “fixing the woman”—for example, by focusing exclusively on confidence or assertiveness—and examine the systems in which leadership occurs.

Organisations can contribute by

- making leadership pathways transparent
- ensuring equitable access to committees and high-visibility projects
- developing formal mentorship and sponsorship programmes
- supporting flexible career pathways
- monitoring gender representation in leadership
- recognising different leadership styles
- ensuring that leadership development begins early in career progression.

Women should not simply be encouraged to enter existing leadership structures; they should also be supported to reshape those structures.

From Individual Leaders to Leadership Cultures

The most important transition for psychiatry may be from an individual-leader model to a leadership-culture model. An organisation that depends on one charismatic leader is vulnerable. An organisation that develops leadership capacity across its workforce is more resilient. This is particularly relevant to psychiatric services, where effective care depends on multidisciplinary collaboration rather than individual authority. The ultimate question should therefore change from: “Who is the leader?” to: “How does this organisation enable leadership to emerge?” This shift transforms leadership from a positional concept into an organisational capability.

Leadership as Legacy

The most enduring contribution of a psychiatric leader may not be a title, award or position. It may be the trainee who was encouraged to lead. The colleague who was given an opportunity. The service that became more humane. The patient whose voice was heard. The policy that changed. Or the culture in which the next generation feels able to challenge, innovate and contribute. Leadership is therefore ultimately a question of legacy. A successful psychiatric leader does not merely occupy a position of influence. They create the environment for others to exert influence.

Conclusion

Leadership is no longer an optional extra to psychiatric practice. It is a core professional competency with implications for patient care, workforce well-being, organizational culture and mental health systems. Contemporary psychiatric leadership must move beyond hierarchy and authority to collaboration, influence, psychological safety, compassion and systems thinking. It also needs to be more inclusive, valuing diverse perspectives and addressing structural barriers to engagement. Leadership education must start during psychiatric training and continue throughout a professional career. Mentorship, sponsorship, experiential learning, feedback and reflection should be incorporated into professional development. The future of psychiatry will depend not only on the treatments we develop or the evidence we generate, but on how well we lead people, organizations and systems through change. The best leaders will be those who leave behind not only successful careers, but stronger teams, better systems and more confident future leaders.

Key Messages

- Leadership is a core component of psychiatric practice, not merely a function of senior administrative positions
- Effective psychiatric leadership influences patient care, service quality, workforce well-being, organisational culture and mental health policy.

- Leadership differs from management: management coordinates resources and processes, whereas leadership provides direction, purpose, influence and change.
- Contemporary psychiatric leaders require competencies in clinical credibility, communication, emotional intelligence, systems thinking, negotiation, ethical reasoning and change management.
- Distributed leadership is particularly relevant to psychiatry because effective mental healthcare depends on multidisciplinary and cross-sector collaboration.
- Leadership development should begin during psychiatric training and continue throughout professional life.
- Mentorship, sponsorship, reflective practice, experiential learning and structured feedback are essential components of leadership development. Inclusive leadership requires organisations to address structural barriers to leadership, including inequitable access to opportunities, visibility, sponsorship and decision-making.
- The most sustainable measure of leadership is not the position achieved by an individual but the capacity created in others.

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Challenges in Management of Bipolar Disorder

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Overview

- Diagnostic Difficulties
- Suicidality
- Treatment Complexity
- Management in Pregnancy and Lactation
- Comorbidities
- Nonadherence
- Gender Differences in BD Management

Diagnostic Difficulties

- Early diagnosis is often missed: Up to 70% of patients first present with depression, leading to misdiagnosis as unipolar depression.
- Delayed recognition of mania/hypomania: Manic symptoms may appear years after initial depressive episodes.
- Mixed states: complicate diagnosis, as patients may show both depressive and manic symptoms simultaneously.
- Bipolar II is much more missed than Bipolar I [Low reliability of diagnosis of hypomania from history].

More common in bipolar than in unipolar depression

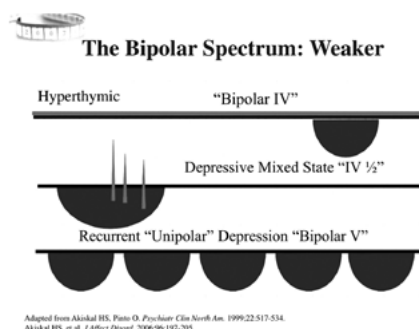
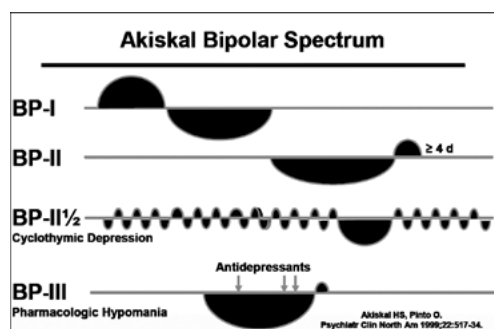
- Atypical symptoms [increased sleep, increased appetite, rejection sensitivity, leaden paralysis and mood reactivity]
- Early age at onset
- Postpartum
- Baseline hyperthymic personality
- Family history of bipolarity
- Psychosis
- Depressive mixed state
- Anxious/agitated depression
- Anergic depression
- Irritability/anger attacks
- Recurrence
- Brief duration of depressive episodes

“The Rule of 3” hinting at soft bipolarity in a clinically depressed individual

- >3 Major depressive episodes
- 3 Failed marriages
- 3 Failed antidepressants
- 3 First degree relatives with affective illness
- 3 Generation family history for affective illness
- Eminence in three fields in the family
- 3 Simultaneous jobs
- Proficiency in three languages (for US-born citizens)
- 3 Distinct professions (exercised simultaneously)

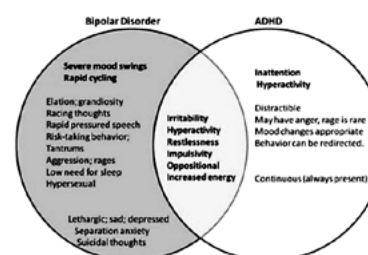


- 3 Comorbid anxiety diagnoses
- Triad of past histrionic, psychopathic, or borderline diagnoses • Triad of trait “mood lability,” “energy activity,” and “daydreaming” Flamboyance expressed in a triad of bright colors
- 3 Substances of abuse
- 3 Impulse control behaviors
- Simultaneous dating of three individuals



ADHD VS BPAD

ADHD and bipolar disorder have some overlapping symptoms, which can make diagnosis difficult.



ADHD VS Bipolar Disorder

	ADHD	Bipolar Disorder
Age of Onset	Childhood	Early Adulthood
Course	Stable	Episodic
Symptoms	• Labile Dysphoric Mood • Reduced Self Esteem • Distractibility perceived as thought wandering, without objective acceleration • Restlessness, fidgetiness, the inability to stop and relax	• Persistently euphoric, elevated or irritable mood • Inflated self-esteem or grandiosity • Distractibility due to acceleration of thought • Restlessness or hyperactivity with Increased goal-directed activity
Sleep	If circadian rhythm dysfunction is present, accompanied by discomfort and fatigue	Decreased need for sleep
Sexuality	Not affected	Increased in hypomania and mania
Psychosis	Absent	Can be present

Comorbidities

- Psychiatric comorbidities are common: Over half of patients have at least one additional disorder (e.g., anxiety, substance use).
- Impact on outcomes: Comorbidities worsen prognosis, increase suicidality, and reduce treatment adherence.

Anxiety Disorders in BD

- One-third to half of BD patients have comorbid anxiety, worsening illness course and outcomes.
- CBT is a safe first-line treatment.

- Mood stabilization should be achieved before adding specific anxiety agents.
- Quetiapine has strong evidence for efficacy in GAD and panic disorder. Limited
- Evidence suggests anxiolytic effects of olanzapine in lithium-treated patients, while risperidone showed no efficacy in a controlled trial.
- Divalproex sodium has preliminary evidence from open label studies for panic disorder or non-specific anxiety symptoms in BD.
- Gabapentin may help; pregabalin lacks specific trials.
- Use of antidepressants requires caution, especially in BD-I, and should be under mood stabilizer cover.
- Benzodiazepines are for short-term relief, at the lowest dose and duration.

OCD in Bipolar Disorder

- Prevalence of OCD in BD-I is about 21.7%, with symptoms fluctuating with mood states.
- Mood stabilization is primary; OCD may remit with stabilization alone.
- SSRIs are preferred with mood stabilizers; avoid clomipramine due to switch risk.
- SGAs like aripiprazole and risperidone may help treat OCD symptoms.
- Memantine and transcranial direct current stimulation (tDCS) show promise as adjuncts.

Attention-Deficit/Hyperactivity Disorder in BD

- Recent Western data suggest ADHD is comorbid in 10%–20% of patients with BD and is associated with adverse illness course, suicidality, and functional impairments.
- For milder ADHD symptoms, non-pharmacological interventions such as psychoeducation and behavioral strategies remain first-line treatments.
- Adjunctive bupropion may be preferred, particularly for those with frequent depressive episodes.
- Stimulant medication, such as methylphenidate, may be considered after sustained remission and long-term mood stability has been achieved, under the cover of maintenance agents, along with monitoring for mood switch.

Borderline personality disorder (BPD) in BD

- Comorbidity of BPD in BD is common (15%–20%).
- Patients with BD-BPD have longer hospital stays, higher ECT utilization, and an elevated suicide risk compared to BD alone.
- Current approaches combine treatments established for BD and BPD separately. Mood stabilizers and SGAs remain the treatment mainstay for BD, while structured psychotherapies for BD, such as dialectical behavior therapy (DBT) can be added.

Substance Use Disorders (SUD) in BD

- Substance use interferes with treatment adherence, treatment response, and worsens illness outcomes in BD.
- Bipolar spectrum disorders are common among SUDs, and often go unrecognized.
- Management for BD and SUD may be initiated simultaneously, especially when withdrawal from substance use is deemed to contribute to mood instability.
- For alcohol use disorder comorbid with BD, a randomized trial found that the combination of lithium and divalproex was more effective than lithium alone in reducing drinking frequency.
- Lamotrigine and divalproex monotherapies (if hepatic function is intact) may offer modest benefit.
- Adjunctive gabapentin and topiramate show some efficacy.
- Naltrexone has limited and inconsistent efficacy data.

- Disulfiram is not recommended due to its potential to exacerbate mania and the risks associated with impulsivity.
- For comorbid cannabis use disorder, evidence is again sparse.
- Lithium and divalproex may offer some benefit.
- Olanzapine add-on therapy was reported to reduce manic symptoms and substance-related cravings among hospitalized patients.
- Aripiprazole may reduce both alcohol and cannabis cravings.

Metabolic syndrome in BD

- Metabolic abnormalities are highly prevalent in BD, with population studies reporting 2-3 times higher rates of obesity, metabolic syndrome, type 2 diabetes, dyslipidemia, and hypertension in BD compared to the general population.
- Indian studies report metabolic syndrome in around one third of BD patients.
- Routine, periodic screening of metabolic health should be part of usual care for BD.
- Lifestyle risk factors (e.g., sedentary lifestyle, irregular sleep, substance use, etc.) should be systematically assessed.
- Agents with lower metabolic adverse effects, such as lamotrigine, lurasidone, or aripiprazole, may be preferred for first-line treatment of BD.
- Agents such as olanzapine, with a more adverse metabolic risk profile, may be considered second-line agents.
- Evidence supports the use of metformin, GLP-1 agonists, such as semaglutide, are emerging agents with a growing evidence base for antipsychotic-induced weight gain.

Suicidality

- High suicide risk: Nearly 79% of patients experience suicidal thoughts, and bipolar disorder carries one of the highest risks of suicide among psychiatric conditions.
- Warning signs:
 - Talking about wanting to die or feeling like a burden
 - Sudden withdrawal or isolation
 - Increased substance use
 - Extreme mood swings
 - Giving away possessions or saying goodbye
 - A sudden “calm” after severe distress (can indicate a decision)
- What reduces the risk
 - Consistent treatment
 - Mood stabilizers (e.g., Lithium is known to reduce suicide risk)—ECT
 - Therapy (CBT, psychoeducation, family-focused therapy)
 - Strong social support
 - Regular sleep and routine
 - Avoiding alcohol/drugs
 - Early recognition of mood changes

Nonadherence

- Nonadherence is a significant determinant of poor outcomes in BD, affecting 40%–60% of BD patients.

- Poor adherence is linked to more severe illness trajectories, including more relapses, hospitalizations, and poorer functional outcomes.
- objective methods to check adherence include prescription renewal, pill counts, medicine procurement bills and, where applicable, pharmacy refill records.
- The gold standard measure of adherence is serum drug levels. For certain drugs such as lithium and valproate, serum drug levels can offer valuable information about adherence

Evidence-based strategies and considerations to improve adherence in BD

- Establishing a good therapeutic alliance.
- Psychoeducation about illness and the need for continued treatment.
- Active involvement of family caregivers
- Collaborative or shared decision-making encourages a sense of responsibility for treatment choices.
- Simplified medication regimens (e.g., once daily).
- Adverse effects should be mitigated.
- Enquire and be vigilant about affordability issues compromising adherence.
- LAI antipsychotics may be considered for patients with repeated non-adherence and high relapse risk.
- Tele-consultations could potentially improve adherence among patients, who are geographically remote and face cost and access issues.
- telephone-based short message/service reminders
- Smartphone app-based tools

Treatment Complexity

- Multiple approved treatments (lithium, anticonvulsants, atypical antipsychotics, psychotherapy), but predicting which will work best for an individual remains difficult.
- Side effects and tolerability issues: Long-term safety of newer antipsychotics is uncertain.
- Combination therapy often required, increasing risk of drug interactions and adherence problems.
- Treatment resistance in BD is typically defined as non response to adequate trials of two or more evidence based agents.
- For mania-
 - Using combinations of mood stabilizers or mood stabilizers with antipsychotics that have lower evidence for efficacy.
 - Typical antipsychotic agents, such as haloperidol, may be used in combination with mood stabilizers, if not yet tried.
 - Clozapine for treatment-resistant acute mania.
 - Among mood stabilizers, carbamazepine/oxcarbazepine may be used alongside lithium (more commonly) or valproate.
 - ECT is an important option in managing resistant manic episodes.
- For bipolar depression-
 - Adjunctive treatments such as olanzapine, modafinil, SNRI, monoamine oxidase inhibitors, and rTMS may be considered
 - ECT should also be considered,
 - Non-invasive brain stimulation, such as accelerated intermittent theta-burst stimulation, has also been effectively tried for resistant bipolar depression.

- Options such as ketamine, pramipexole, levothyroxine, N-acetyl cysteine, light therapy with or without sleep deprivation, among others, have limited evidence for efficacy.
- Short term efficacy of adjunctive pramipexole, modafinil, and racemic intravenous ketamine.
- Celecoxib augmentation of escitalopram and treatment with metformin in BD patients with insulin resistance showed some promising results.
- Contextual indications for modified electroconvulsive therapy in bipolar disorder As a first-line treatment -
- Severe depression
- Suicidality
- Severe psychosis where rapid response is desired
- Catatonic presentations
- Depression with compromised oral intake worsening general medical status
- Extreme, delirious mania

Anti-epileptic mood stabilizers such as valproate or high dose benzodiazepines may increase seizure threshold and interfere with ECT efficacy. Co-prescribed lithium may increase the risk of prolonged seizures and neurotoxicity

Gender Differences in BD Management

- Women tend to have more depressive episodes, rapid cycling, and BD-II; men have earlier onset and more SUD.
- Women have higher suicide attempts; men have higher suicide mortality.
- Hormonal fluctuations can worsen symptoms in women.
- Women are more susceptible to lithium-induced hypothyroidism and adverse effects like hyperprolactinemia.
- Contraceptive and pregnancy considerations influence treatment choices.

Management in Pregnancy and Lactation

- Treatment during pregnancy requires risk-benefit analysis; planning should start 4–6 months pre-conception.
- Continue medications at minimum effective doses or consider substitution.
- Discontinue or switch to safer agents if possible.

Table 14: Reproductive safety of psychotropic medications for bipolar disorder^[260-269]

Mood stabilizers	Safety data	Clinical considerations
Lithium	Small absolute risk of Ebstein's anomaly (0.05%–0.1% of live births) with first-trimester exposure (twenty times higher than the general population rate). Slightly increased risks of preterm birth and low birth weight. Risks of cardiac malformation could be dose-dependent. ^[265]	Fetal echocardiography is advised at 16–20 weeks. Frequent serum lithium level monitoring may be necessary due to hemodynamic changes. (monthly till 36 weeks, weekly from 36–40 weeks; twice each week in the first 2 weeks postpartum)
Valproic acid/ Divalproex	High risk of major congenital malformations (8%–10%), including neural tube defects. Linked to later neurodevelopmental disorders. ^[266]	Contraindicated in pregnancy.
Carbamazepine	Risk of major congenital malformations (3%–6%). Risk of neonatal bleeding diathesis from vitamin K deficiency.	Avoid to the extent possible. Supplement with high-dose folic acid before conception (though evidence remains unclear). Provide maternal vitamin K late in pregnancy and neonatal vitamin K supplementation at birth.
Oxcarbazepine	The risk of major congenital malformations appears comparable to the general population rate ($\approx 3\%$). ^[267]	It can be used during pregnancy, although safety data are limited.
Lamotrigine	The malformation risk ($\approx 3\%$) is similar to the baseline population risk. ^[267] No definite evidence of neurobehavioral sequelae.	Preferred agent if clinically indicated (e.g., bipolar depression or depression-predominant course). Dose adjustment is often required due to increased clearance during pregnancy. Monitor the infant for skin rash.

Second-generation antipsychotics (SGAs)

Olanzapine,
Quetiapine,
Aripiprazole,
Risperidone

The major malformation risk is approximately 4.5% in exposed individuals versus 3.5% among unexposed individuals; no significant increase was observed after adjustment, except for a small increase associated with risperidone.^[267] Another large recent study did not replicate the risperidone signal.^[268] Maternal risks: weight gain, gestational diabetes, large-for-gestational-age infant.

These SGAs appear to be relatively safe. Monitor maternal weight and glucose (with quetiapine/olanzapine). Consider using ultrasound to assess fetal size in late pregnancy.^[269] Monitor maternal weight and glucose (with quetiapine/olanzapine).

Clozapine,
Ziprasidone,
Lurasidone

Available safety evidence is limited.

If clozapine is used, monitoring of the neonatal blood count is crucial.

Antidepressants and sedative-hypnotic agents

SSRIs

(Fluoxetine,
Escitalopram,
Sertraline, Paroxetine,
Fluvoxamine)

Slight increased risk of spontaneous abortion, preterm birth, and low birth weight. Small increased absolute risks of cardiac defects with paroxetine (2/1000). Conflicting evidence on persistent pulmonary hypertension (3rd trimester). Neonatal adaptation difficulties may occur in one-third of late-exposed infants.

Generally safe, if indicated. Avoid paroxetine.

Non-SSRI antidepressants

Limited safety data are available, but malformation risks appear to be similar to those of SSRIs.

Use if SSRIs are not suitable.

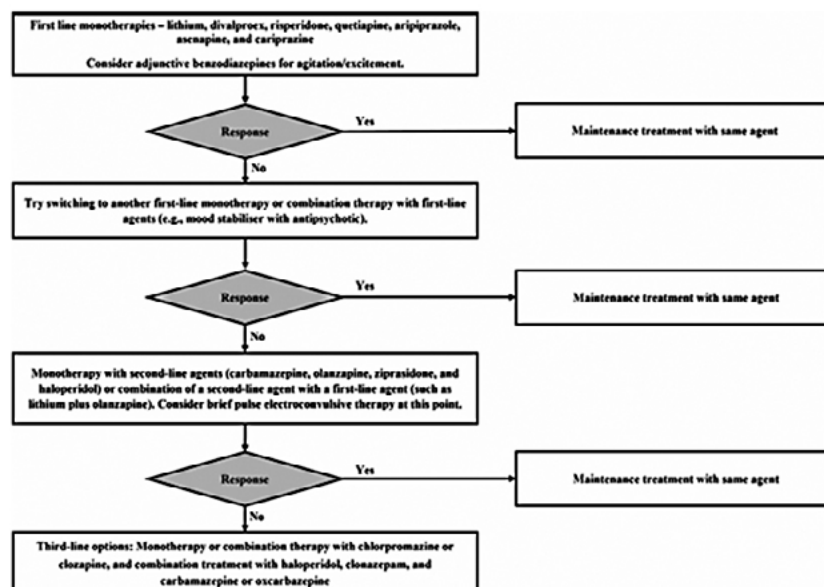
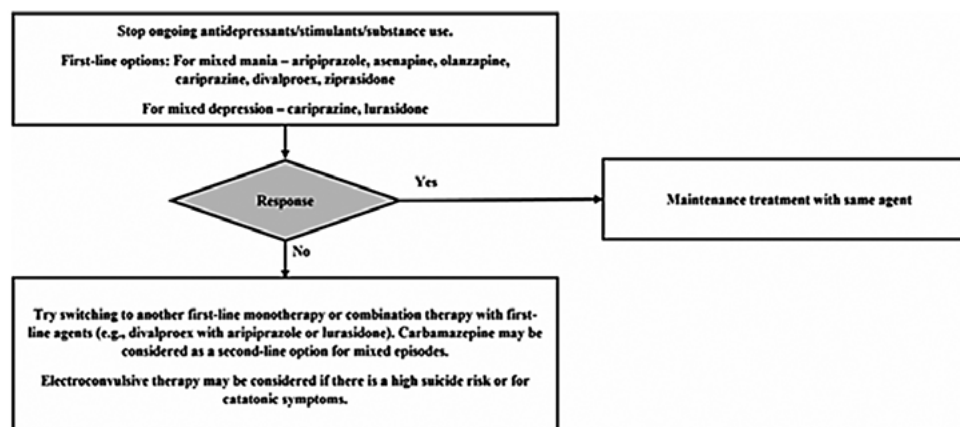
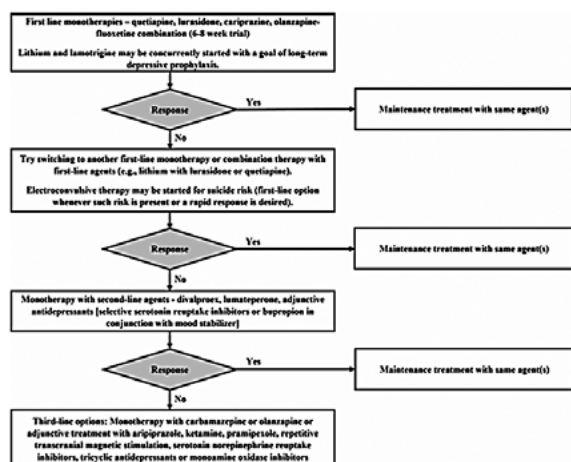


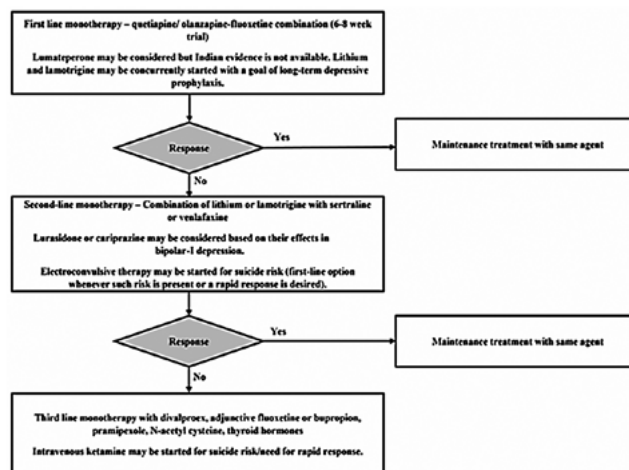
Figure 1: Algorithm for acute pharmacological management of mania



2: Algorithm for acute pharmacological management of mixed episodes in bipolar type I disorder



Algorithm for acute pharmacological management of depressive episodes in bipolar type I disorder



Algorithm for acute pharmacological management of depressive episodes in bipolar type II disorder

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Integrating Lumateperone in Real World Clinical Practice

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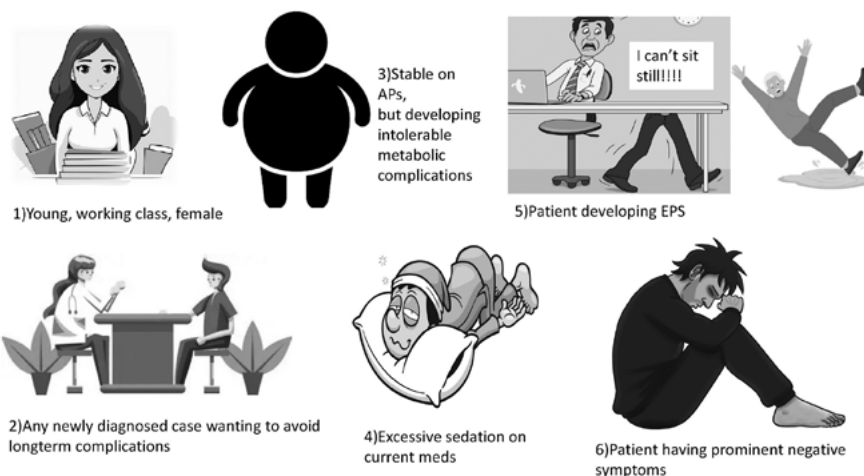
WHAT MAKES LUMATEPERONE DIFFERENT?

Most antipsychotics are discovered through dopaminergic screening, Lumateperone was developed through advanced Phenotypic Drug Discovery approaches

<u>Traditional (target-based) discovery</u>	<u>Phenotypic discovery</u>
Receptor target → Compound → Biological effects	Compound → Desired biological effects → Mechanism discovered later
Does this compound block D ₂ receptors?	Does this compound produce the overall brain changes we want?

WHO IS AN IDEAL CANDIDATE FOR LUMATEPERONE?

5HT _{2A} receptor antagonism Antipsychotic, Antiaggressive, Antidepressant, Anxiolytic	Low antagonist activity at H ₁ , 5HT _{2c} , M ₁ , α ₁ Low sedative, metabolic, anticholinergic, orthostatic hypotension	SERT inhibition Antidepressant, Anxiolytic effects
D ₂ Partial agonist (pre synaptic) D ₂ Antagonist (Post synaptic) Antipsychotic efficacy achieved at low D ₂ receptor occupancy (40%) compared to 65-80% typically required for other agents. Minimal impact on motor function and prolactin Regioselective at mesolimbic & mesocortical pathways	Half life: 13 to 21 hours (OD dosing, better compliance) No dose titration needed	Enhances NMDAR & AMPAR signalling via D ₁ receptor mediated action in PFC Pro cognitive, Antidepressant, Negative symptoms



MAJOR REASON OF TREATMENT DISCONTINUATION

- TOLERABILITY>>>EFFICACY
- The ideal antipsychotic should provide
 - ✓ efficacy
 - ✓ metabolic neutrality
 - ✓ minimal EPS
 - ✓ antidepressant /pro-cognitive effects
 - ✓ simple dosing

LUMATEPERONE ticks all the boxes

RECEPTOR PROFILE OF SGAs

Receptor	Olanzapine	Risperidone	Quetiapine	Lurasidone	Cariprazine	Aripiprazole
D2	++++	++++	++	++++	+++++ (D3>D2)	+++++ (Partial agonist)
D3	+	+	+	++	+++++	+++
5-HT _{2A}	+++++	+++++	+++++	+++++	++++	++++
5-HT _{1A}	+	+	++	+++ (Partial agonist)	++++ (Partial agonist)	++++ (Partial agonist)
5-HT ₇	++	+	+	++++	++	+
H ₁	+++++	++	+++++	+	+	+
M ₁ (Muscarinic)	+++++	-	+	-	-	-
α ₁	+++	++	++++	+	+	++

Characteristic	Olanzapine	Risperidone	Quetiapine	Lurasidone	Cariprazine	Aripiprazole
Weight gain	★★★★★ Highest	★★★ Moderate	★★★ Moderate	★ Lowest	★ Lowest	★ Lowest
Metabolic syndrome	Very High	Moderate	Moderate	Minimal	Minimal	Minimal
EPS	Low	High (dose dependent)	Very Low	Low–Moderate	Moderate	Moderate (Akathisia)
Akathisia	Rare	Moderate	Rare	Moderate	High	High
Hyperprolactinemia	Minimal	★★★★★ Highest	Minimal	Minimal ✓	None ✓	Decreases prolactin ✓
Sedation	★★★★★ Highest	Moderate	★★★★★ Highest	Minimal	Minimal	Minimal

Characteristic	Olanzapine	Risperidone	Quetiapine	Lurasidone	Cariprazine	Aripiprazole
Orthostatic hypotension	Moderate	Moderate	High	Low	Low	Mild
Anticholinergic effects	High	None	Mild	None	None	None
QT prolongation	Mild	Moderate	Mild	Minimal	Minimal	Minimal
Cognitive impairment	Moderate	Mild	Moderate	Minimal	Minimal	Minimal
Negative symptom efficacy	Moderate	Moderate	Moderate	Good	Excellent	Good
Bipolar depression	Good	Limited	Excellent	Excellent	Excellent	Moderate
Sleep benefit	Excellent	Fair	Excellent	Poor	Poor	Poor
Long-term metabolic safety	Poor	Fair	Fair	Excellent	Excellent	Excellent

FDA APPROVED INDICATIONS

- 2019 Schizophrenia → • 2021 Bipolar depression → • 2025 Adjunctive for MDD

Table 1. Preliminary list of potential indications for treatment with lumateperone.

- Schizophrenia and other primary psychiatric disorders
- Bipolar depression (either monotherapy or combination treatment)
- Acute, "clean" mania or mania with mixed features
- Maintenance treatment of BD
- Treatment-resistant MDD
- Behavioral disorders in the course of dementia
- Behavioral disorders in children and adolescents
- Impulse-control disorders
- PTSD

BD—bipolar disorder, MDD—major depressive disorder, PTSD—post-traumatic stress disorder. Adapted from Stahl [9].

PRACTICAL PRESCRIBING PEARLS

- 42 mg once daily
- No titration needed
- Taken with food
- Preferably at bedtime(though evening dose maybe considered to avoid daytime sedation next morning)
- Drug-drug interactions +(metabolized by CYP3A4 and UGT enzymes) —→ Avoid
- CYP3A4 inhibitors(ketoconazole, fluvoxamine)
- CYP3A4 inducers(carbamazepine)
- UGT inhibitors(valproic acid)
- Alcohol, sedatives
- In acutely agitated patients, combining Lumateperone with a benzodiazepine or another antipsychotic is recommended.
- In patients with schizophrenia who exhibit only a partial response to lumateperone, adding a mood-stabilizing agent like lamotrigine or topiramate may be beneficial

- A decrease in the absolute neutrophil count to below 1000/mm³ necessitates the urgent discontinuation

HOW LONG IT TAKES TO WORK?

- Approximately **1 week** to observe meaningful improvement in psychotic symptoms, the full range of therapeutic effects encompassing affective, cognitive, and behavioral dimensions: develop over **several weeks**.
- The decision on whether to continue with lumateperone or switch to another D2 antagonist in patients with schizophrenia is typically made after **4–6 weeks of treatment**.
- Notably, if the initial effects are promising, it may take **4–5 months** to observe comprehensive improvement in the patient's mental status, particularly concerning cognitive and negative symptoms.

SWITCHING TO LUMATEPERONE

Current medication → Evaluate stability → Gradual cross taper → Monitor

ADVERSE EFFECTS

• Common	• Less common	• Rare	Montaser J, Tumu S, Maram Reddy VY, Jayasuriya N, Ali M, Munir A, Elmontaser M. Lumateperone Safety and Tolerability in Schizophrenia: A Narrative Review. Cureus. 2025 Nov 5;17(11):e96165. doi: 10.7759/cureus.96165. PMID: 41356959; PMCID: PMC12680432.
Sedation	Dizziness	Neutropenia	
Somnolence	Orthostasis	Suicidal ideation reports	
Dry mouth		Mania/hypomania signals	
Nausea			

FEW NOTABLE TRIALS ON LUMATEPERONE

Table 2. Summary of key clinical trials on lumateperone.

Study	Patient Population	Study Design	Dose(s) of Lumateperone	Efficacy Outcomes	Safety Profile
Correll et al., 2020 [19]	N = 450 patients with schizophrenia	Phase 3, randomized, double-blind, placebo-controlled	42 mg/day	Significant reduction in PANSS total score vs. placebo	Similar incidence of adverse events as placebo; low rates of EPS
Calabrese et al., 2021 [36]	N = 381 patients with bipolar depression	Phase 3, randomized, double-blind, placebo-controlled	42 mg/day (monotherapy)	Significant improvement in MADRS score vs. placebo	Well tolerated; minimal weight gain
Suppes et al., 2022 [38]	N = 554 patients with bipolar depression	Phase 3, adjunctive therapy with lithium or valproate	42 mg/day	Greater reduction in depressive symptoms vs. adjunctive placebo	Favorable safety profile; low risk of metabolic side effects

MADRS—Montgomery–Åsberg Depression Rating Scale; PANSS—Positive and Negative Syndrome Scale.

BUT IS IT TOO GOOD TO BE TRUE: HYPE OR HOPE?

- Most studies are done for a short duration(4–6 weeks)
- Few head-to-head comparisons(against a range of established SGA)
- Conflict of interest issues
- Focus on surrogate endpoints rather than direct measures of functional improvement
- Scant evidence regarding the effectiveness and safety profile in special populations such as the elderly, pregnant women, and individuals with renal, cardiac, hepatic, or other medical issues
- Consequently, while early findings are encouraging, the generalizability of these results to long-term, real-world treatment remains uncertain.

LUMATEPERONE: HYPE OR HOPE?

- The open-label switch study by Correll et al. showed short-term improvements when patients transitioned to lumateperone, but these benefits reversed after switching back to other antipsychotics, showing the need for sustained treatment data.

- While emerging meta-analytic data have begun to evaluate lumateperone's efficacy and safety across psychiatric disorders, comprehensive network meta-analyses directly comparing it to a wider range of antipsychotics and studies focusing specifically on cognitive outcomes remain limited.

Safety and tolerability of lumateperone 42 mg: an open-label antipsychotic switch study in outpatients with stable schizophrenia. Correll CU, Vanover KE, Davis RE, Chen R, Satlin A, Mates S. Schizophr Res. 2021;228:198–205

OTHER CHALLENGES

- Most psychiatrists have years of experience with

Quetiapine/Olanzapine/Aripiprazole

- Questions that arise with any new molecule

Will it be effective?

When should I switch?

What if the patient worsens?

- How many of us will really switch to Lumateperone for a patient experiencing weight gain on Olanzapine and not to another SGA?
- Experience builds CONFIDENCE

PATIENT FACTORS

- In many settings, affordability influences adherence more than efficacy.
- Doctor, why should I change when my current medicine is cheaper?

Drug	Typical strength	Approx. price for 10 tablets
Olanzapine	10 mg	₹70–100
Aripiprazole	10 mg	₹100–250
Risperidone	2 mg	₹60–120
Quetiapine	100 mg	₹120–180
Cariprazine	1.5 mg	₹90–180
Lurasidone	40 mg	₹140–250
Lumateperone	42 mg	₹360–380

DIFFICULTY DURING SWITCHING

- Rebound insomnia
- Rebound anxiety
- Rebound cholinergic symptoms

PRACTICAL TIPS FOR SUCCESSFUL INITIATION

- ✓ Set realistic expectations.
- ✓ Warn about mild transient side effects
- ✓ Avoid judging efficacy too early
- ✓ Explain that improvement may take several weeks.
- ✓ Follow up early
- In everyday clinical practice, the success of lumateperone depends less on its pharmacology which is already well established and more on –

Selecting the right patient, counselling appropriately, and switching thoughtfully.

Good initiation strategies translate trial efficacy into real-world effectiveness.

TAKE HOME MESSAGES

- ✓ A multimodal antipsychotic
- ✓ Excellent metabolic profile
- ✓ Best viewed as a personalized treatment option rather than a universal replacement.
- ✓ Effective in schizophrenia and bipolar depression
- ✓ Minimal EPS and prolactin elevation

Appears most promising for patients who prioritize metabolic and motor safety or who have experienced intolerance to other agents, rather than as a general first-line antipsychotic.

Experience is medicine's greatest textbook, and every patient adds a new page.

Neuromodulation

Nishant Goyal, Professor, Dept. of Psychiatry, VMMC & SJH, New Delhi-110029

FOUNDATIONS

What Is Neuromodulation?

The targeted alteration of nervous system activity — electrically, magnetically, chemically, or mechanically — to modulate neural function for research or therapeutic benefit

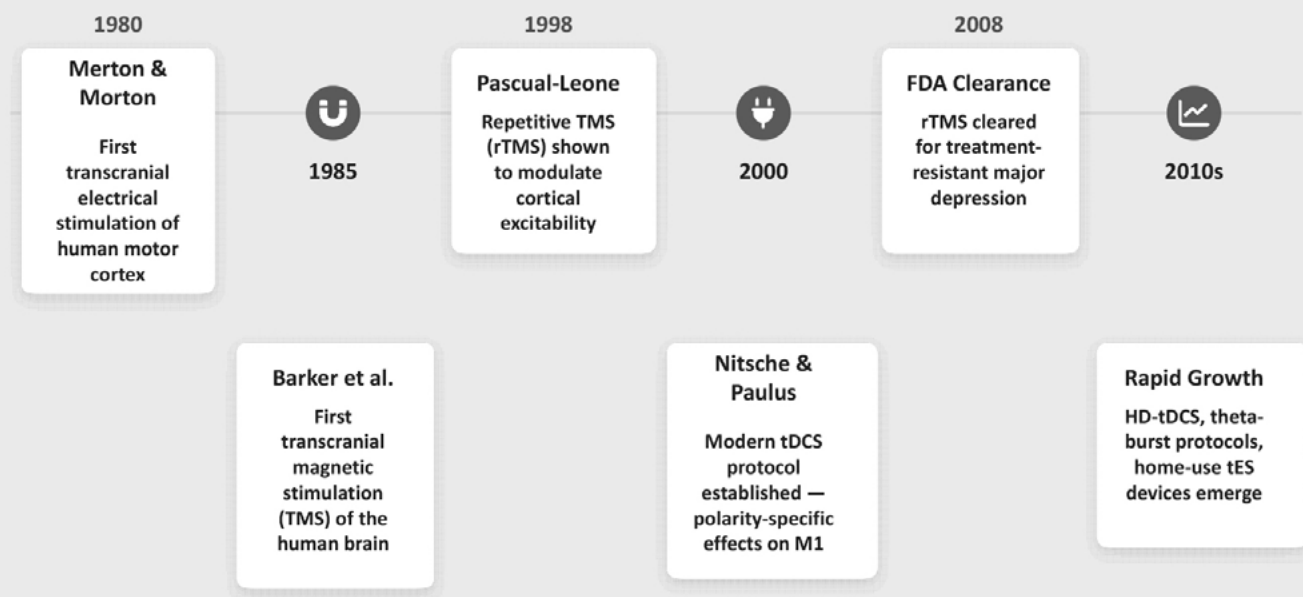
-  **Reversible**
Effects are typically transient and titratable, unlike ablative procedures
-  **Network-level**
Acts on distributed cortical circuits, not single neurons
-  **Dose-dependent**
Direction & magnitude of effect depend on stimulation parameters

THE NEUROMODULATION SPECTRUM

-  **Invasive**
DBS, VNS, spinal cord stimulators — implanted electrodes/generators
-  **Non-Invasive (this workshop)**
rTMS, tDCS, tACS, tRNS — delivered through the scalp, no surgery
-  **Pharmacological**
Neurotransmitter systems modulated by drugs

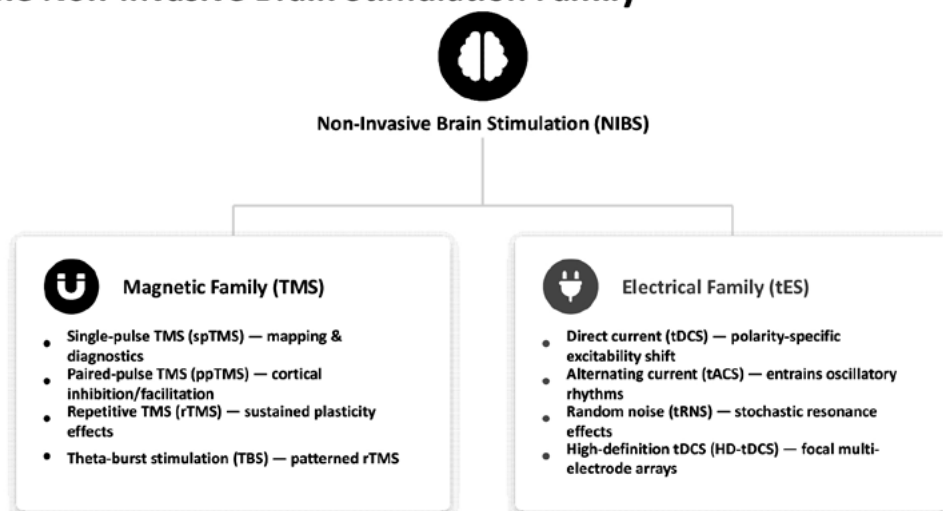
FOUNDATIONS

A Brief History of Brain Stimulation

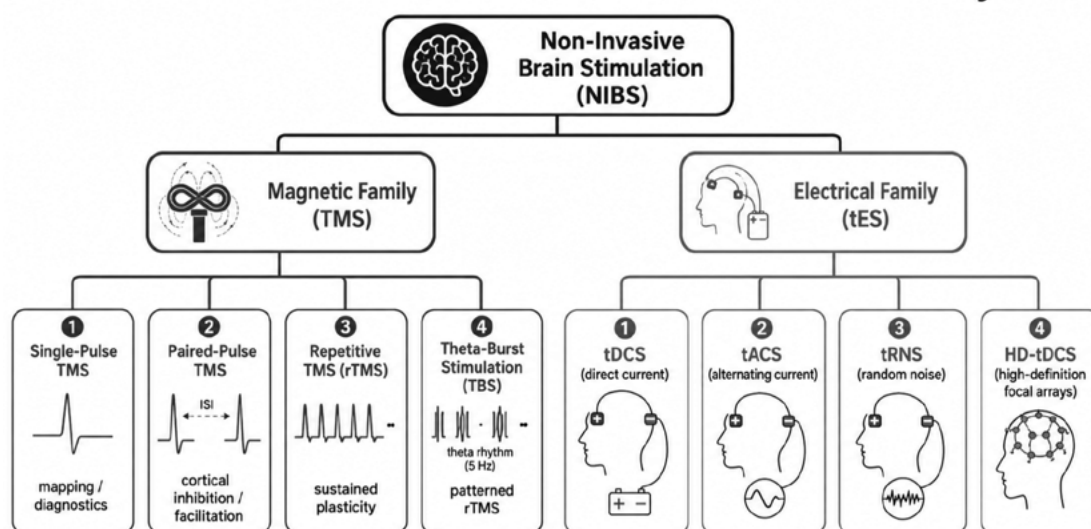


FOUNDATIONS

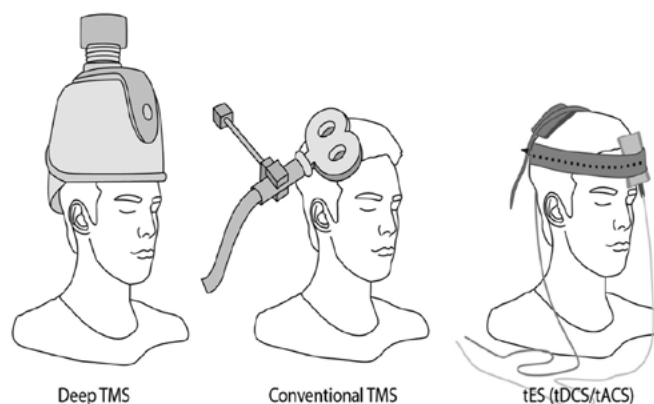
The Non-Invasive Brain Stimulation Family



The Non-Invasive Brain Stimulation Family



Major modalities...



FOUNDATIONS

Why Non-Invasive Brain Stimulation?

**Research Tool**

Establishes causal brain-behavior links by transiently perturbing defined circuits

**Diagnostic Tool**

Probes corticospinal integrity, inhibitory tone, and interhemispheric balance

**Therapeutic Tool**

Drives lasting plasticity to treat depression, pain, stroke deficits & more

**Plasticity Induction**

Mimics LTP/LTD-like mechanisms to reshape maladaptive network activity

PART ONE



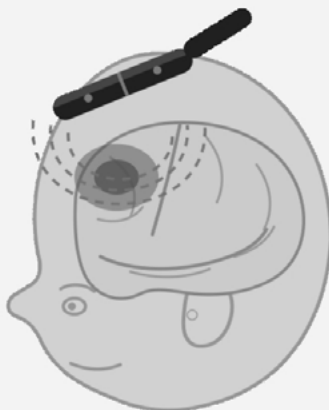
Transcranial Magnetic Stimulation

Electromagnetic principles, parameters, protocols, and clinical applications of rTMS

rTMS · BASIC PRINCIPLE

Electromagnetic Induction — Faraday's Law

A brief, high-current pulse through the coil generates a rapidly changing magnetic field, which passes unimpeded through scalp and skull to induce a secondary electric current in the underlying cortex



- 1 Pulsed current in the coil**
Capacitor bank discharges through the windings in ~100–300 microseconds
- 2 Rapidly changing magnetic field**
Reaches ~1.5–2.5 tesla at the coil face and crosses skull unattenuated
- 3 Induced eddy current in cortex**
A secondary current is induced parallel to the plane of the coil
- 4 Neuronal depolarization**
Axonal membranes reach threshold and fire — the biological signal

Effective depth = 2–3 cm from the coil face — the field falls off steeply, confining rTMS to superficial cortex

HARDWARE

Anatomy of a TMS System

**Main Unit / Stimulator**

Houses high-voltage capacitors and control electronics that generate the current pulse

**Capacitor Bank**

Charges to high voltage, then discharges through the coil in microseconds

**Stimulating Coil**

Circular, figure-8, or H-coil geometry — determines focality and depth

**Neuronavigation (optional)**

MRI-guided targeting for reproducible, individualized coil placement

**Control Interface**

Sets intensity (%MT), frequency, train duration, inter-train interval

**Safety Monitoring**

EMG feedback, emergency stop, and real-time parameter limits

Coil Geometries: Focality vs. Depth Trade-off

**Circular Coil**

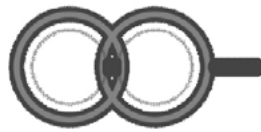
FOCALITY

Low

DEPTH

Moderate

Broad, non-focal stimulation; historical and early research use

**Figure-8 (Butterfly)**

FOCALITY

High

DEPTH

Superficial (~1.5–2 cm)

The clinical workhorse — motor mapping and DLPFC targeting

**H-Coil (Deep TMS)**

FOCALITY

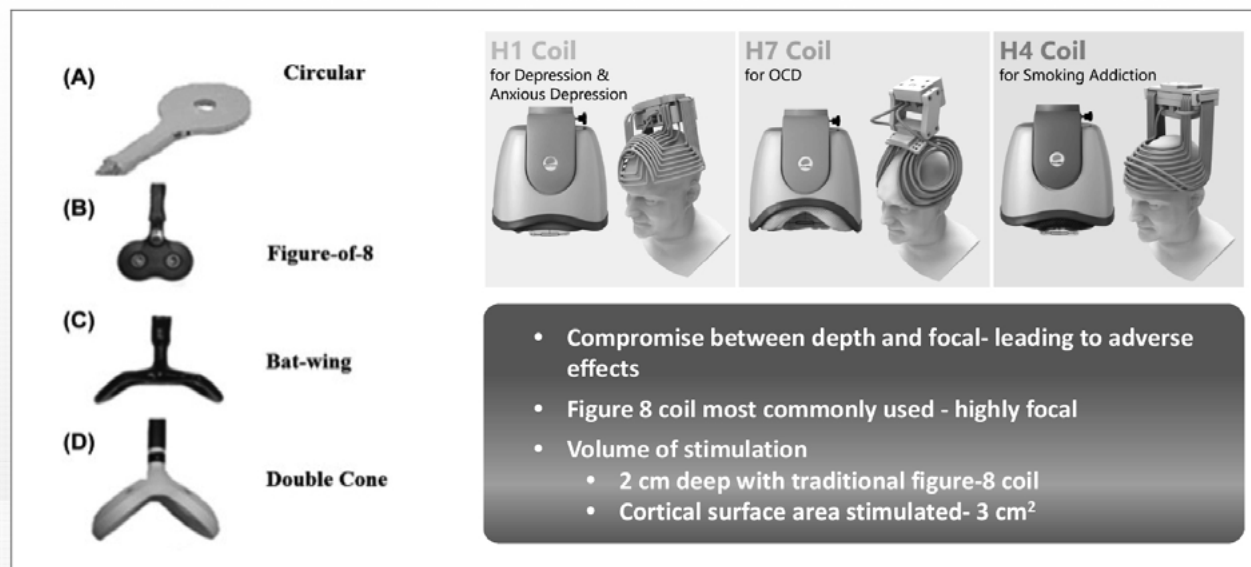
Moderate

DEPTH

Deep (~3–4 cm)

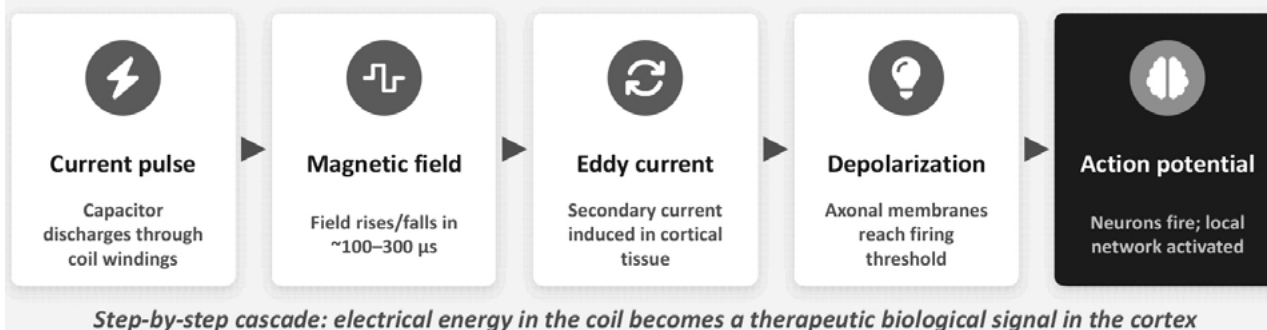
Reaches deeper structures — OCD and addiction circuits

Types of Coils: Quest for Depth

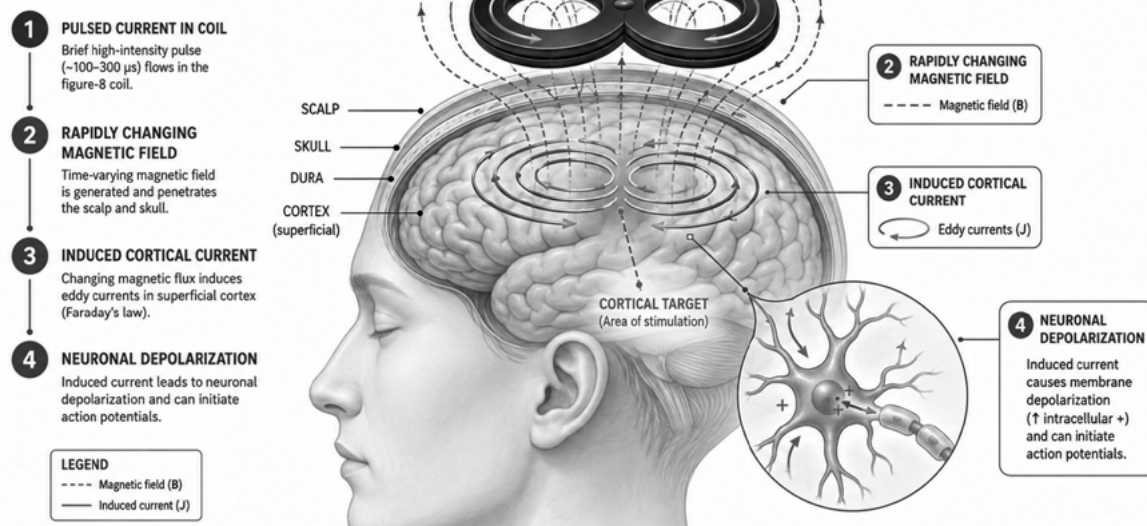


MECHANISM

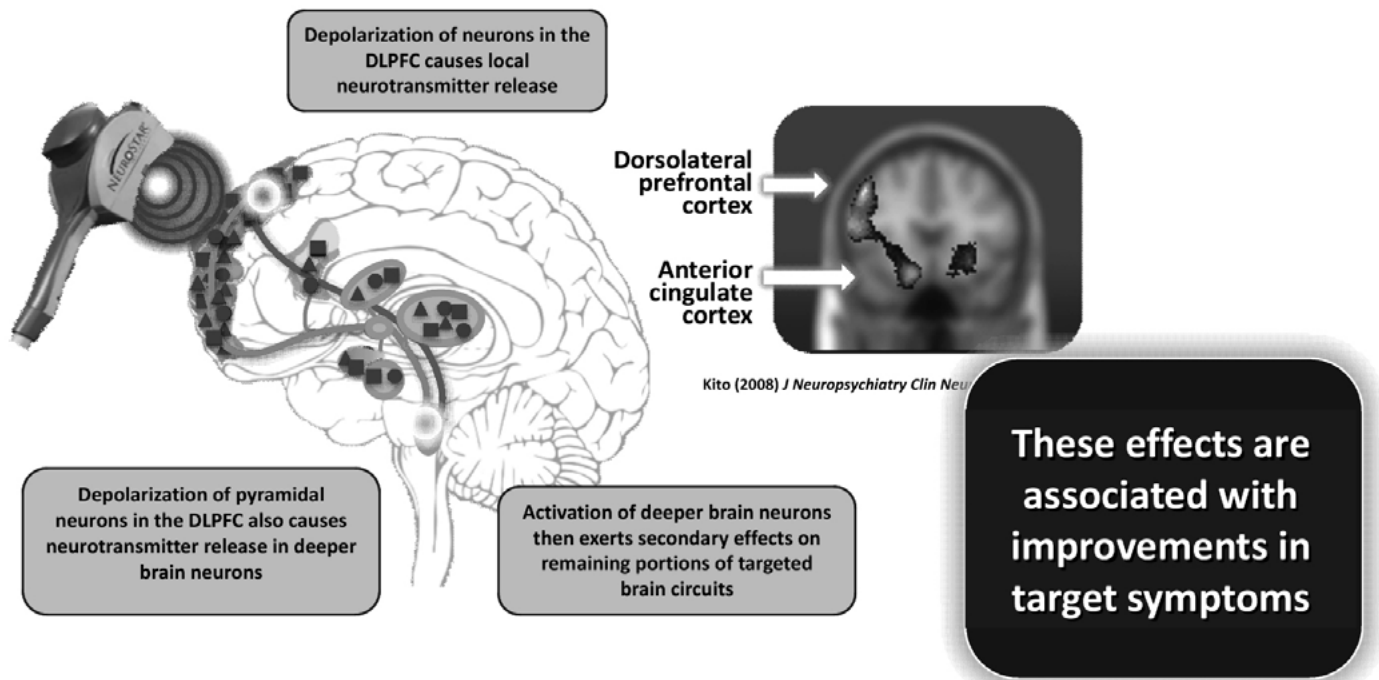
From Pulse to Neuronal Effect



rTMS ELECTROMAGNETIC INDUCTION



Mechanism of Action - TMS



PROTOCOLS

Stimulation Paradigms: sp / pp / rTMS



spTMS
Single-Pulse TMS



One discrete pulse delivered at a time; pulses widely spaced

Motor mapping, motor-evoked potentials (MEPs), corticospinal excitability



ppTMS
Paired-Pulse TMS



Two pulses (conditioning + test) at set inter-stimulus intervals

Intracortical inhibition/facilitation (SICI, ICF)



rTMS
Repetitive TMS



Trains of regularly spaced pulses at a fixed frequency

Sustained plasticity — the therapeutic workhorse

PEDAGOGY

rTMS Parameters That Matter



Intensity

% of resting/active motor threshold (%RMT) — typically 80–120%



Frequency

Pulses per second (Hz) — determines direction of effect



Train duration

Length of a continuous pulse burst, in seconds



Inter-train interval

Rest period between trains, allows safety compliance



Total pulse count

Sum of pulses per session — links to overall dose



Session number

Daily sessions over 2–6 weeks for therapeutic protocols

MECHANISM

Frequency Sets the Direction of Effect



≤ 1 Hz

Low-Frequency rTMS

INHIBITORY EFFECT

LTD-like suppression of cortical excitability

CLINICAL LOGIC

Hyperactive/hyperexcitable cortex — e.g. contralateral homologue in stroke, right DLPFC in depression



≥ 5 Hz

High-Frequency rTMS

EXCITATORY EFFECT

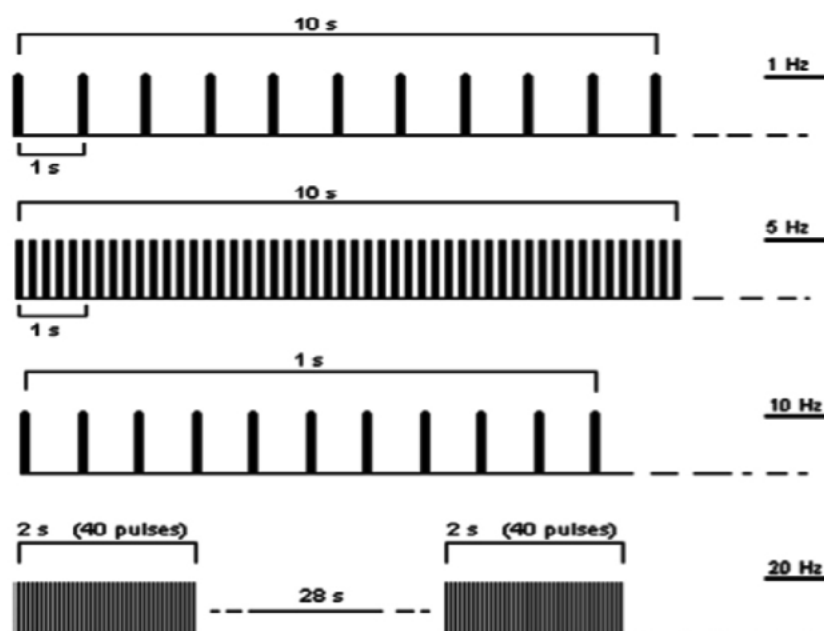
LTP-like facilitation of cortical excitability

CLINICAL LOGIC

Hypoactive cortex — e.g. left DLPFC in depression, ipsilesional M1 in stroke

LF-rTMS

HF-rTMS



Patterned Stimulation: Theta-Burst (TBS)

TBS mimics endogenous hippocampal theta-gamma coupling: short bursts of 3 pulses at 50 Hz, repeated at 5 Hz (theta rhythm) — producing robust effects in a fraction of standard rTMS time

iTBS

Intermittent TBS



Excitatory (LTP-like)

Session duration: ≈ 3 minutes

Pattern: 2s ON burst train / 8s OFF, repeated

Fast excitatory protocol — e.g. accelerated depression regimens

cTBS

Continuous TBS



Inhibitory (LTD-like)

Session duration: ≈ 40 seconds

Pattern: Uninterrupted 40s burst train

Fast inhibitory protocol — e.g. research on cortical suppression

Theta Burst Stimulation

Table 1: Comparison of different conventional patterns of repetitive magnetic stimulation

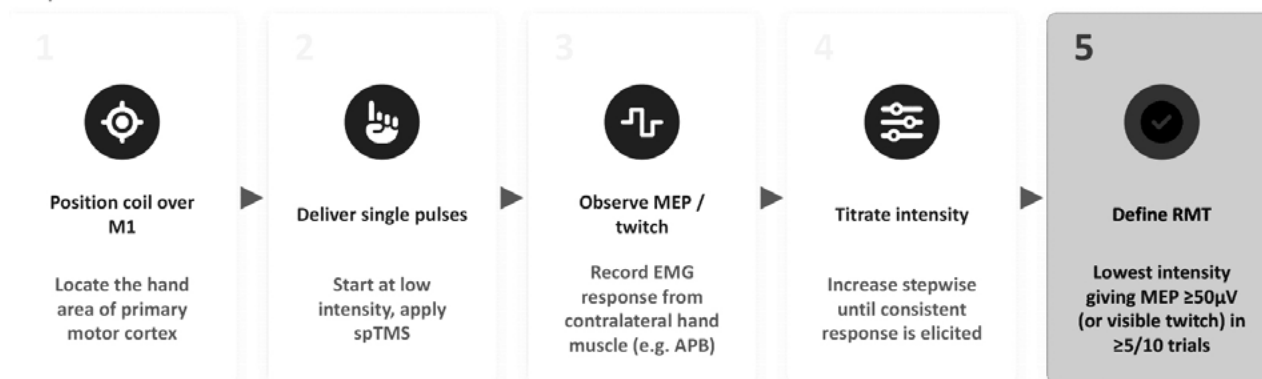
Type of effect	Conventional rTMS		TBS	
	Excitatory ^[1] (5 - 20 Hz)	Inhibitory ^[2,3] (0.9 - 1 Hz)	Excitatory ^[4] (intermittent)	Inhibitory ^[5] (continuous)
Number of pulses	20 - 50	180 - 810	600	300 or 600
Intensity	110% - 200% RMT	115% RMT	80% AMT	80% AMT
Duration of effects	3 - 4 min	15 min	15 min	20 - 60 min

Note: AMT= active motor threshold; Hz= Herz; min= minutes; RMT=resting motor threshold; rTMS= repetitive transcranial magnetic stimulation; TBS= theta-burst stimulation; ^[1] (Pascual-Leone et al. 1994); ^[2] (Chen et al. 1997); ^[3] (Wassermann et al. 1996); ^[4] and ^[5] (Huang et al. 2005).

DOSING

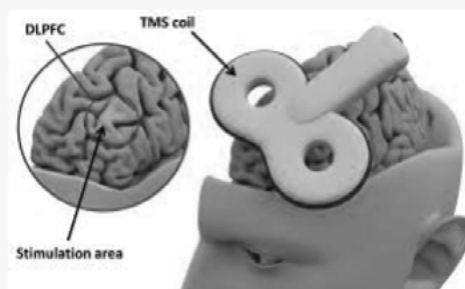
Determining the Resting Motor Threshold (RMT)

Every rTMS protocol is individually dosed as a percentage of the RMT — the minimum intensity producing a reliable motor response



Targeting DLPFC: from approximate to individualized

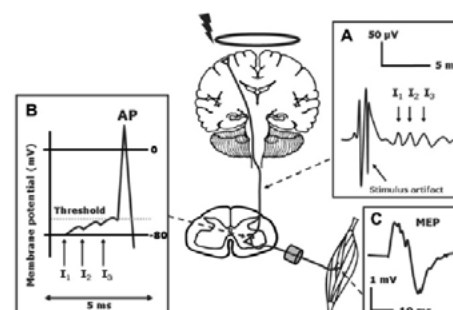
- DLPFC is typically defined by areas 9 and 46 in the Brodmann classification system
- How to identify?
 - standard '5-cm method' for coil localization (crude method)
 - F3 EEG point (International 10-20 system) is known to relate to DLPFC and is likely to be more anterior than a 5-cm localized treatment
 - **Beam method**
 - MRI based neuronavigation



Trapp et al., 2025; Dannhauer et al., 2023

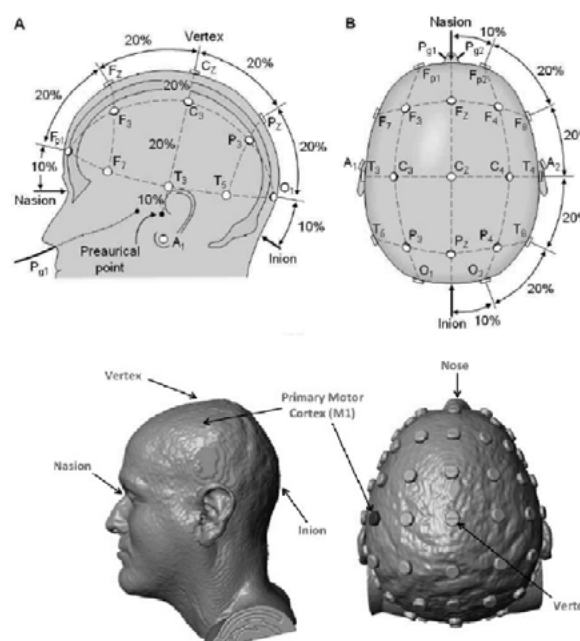
Motor threshold

- The motor threshold (MT) is defined as *"the lowest level of stimulation capable of causing a twitch in the contralateral thumb, specifically, the abductor pollicis brevis or index finger (FDI)"*
- MT is also defined as *"the magnetic stimulus intensity at which a motor evoked potential (MEP) above an a priori amplitude threshold (usually > 50µV in a resting muscle) is observed with a probability $p_i = 0.5$ (i being the intensity of the magnetic stimulus)"*
- Major determinant of TMS dosage defined as *"a percentage of the MT in order to ensure efficacy, while minimizing the risk of adverse reactions"*



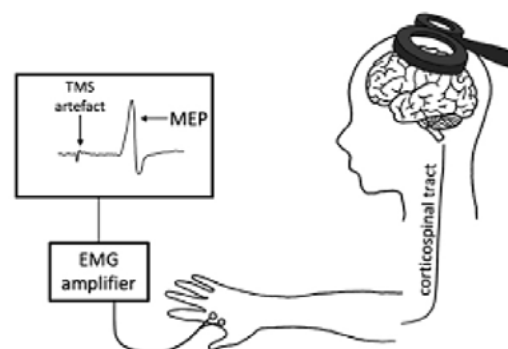
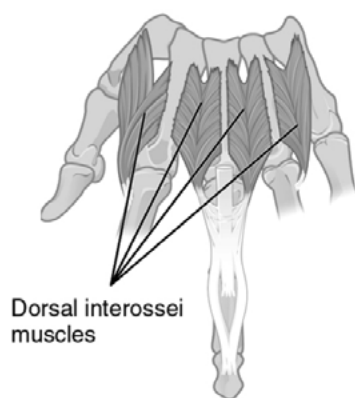
Resting Motor Threshold

- Preparation
 - Participant is seated in a comfortable chair and asked to relax the contralateral arm by placing it on the arm rest, and EMG electrodes, when applicable, are attached.
 - Surface marking of vertex (Cz, International 10-20 system)
 - 5cms lateral and 0.5-1 cm anterior to Cz is M1



EMG based RMT determination

- Electrode on Muscle Belly
- Reference on Tendon
- Ground on a bone



RMT: Min. 50 mV response in minimum 5/10 stimuli

Locating the Left DLPFC: The Beam F3 Method

The conventional "5 cm rule" misses the DLPFC in a substantial minority of patients. Beam F3 uses three simple tape-measure values to approximate the EEG F3 position — no neuronavigation required

STEP 1 — TAKE THREE SCALP MEASUREMENTS



Nasion → inion

Over the vertex, front to back



Tragus → tragus

Over the vertex, ear to ear



Head circumference

Around the widest contour

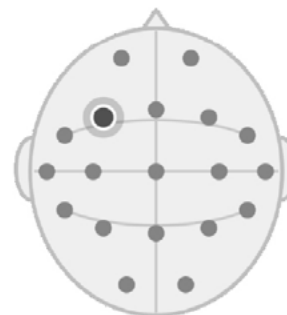
STEP 2 Enter the three values into the Beam F3 calculator

The freely available application returns two outputs — distances X and Y — as a polar-coordinate approximation of F3 relative to the vertex.

STEP 3 Mark the site on the scalp

Measure X along the head-circumference plane from the vertex, then trace Y along the scalp from the vertex to that point. Mark, then confirm coil position and angle before treating.

WHERE F3 SITS



F3 in the 10–20 system approximates the left DLPFC (viewed from above, nose at top).

Beam F3 lands roughly 2.5–3 cm anterolateral to the 5.5 cm rule and shows better inter- and intra-rater reliability, though it remains an approximation of true F3.

BEAM F3 METHOD: LOCATING THE LEFT DLPFC

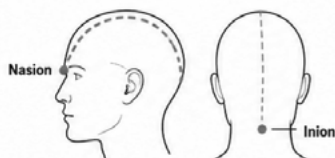
A 3-MEASUREMENT APPROACH + 10-20 EEG MAP

STEP 1: THREE SCALP MEASUREMENTS

Use a flexible measuring tape and record all three measurements (cm).

1 NASION-TO-INION

Measure from the nasion (bridge of nose) over the top of the head to the inion (most prominent point on the back of the head).



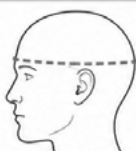
2 TRAGUS-TO-TRAGUS

Measure straight across the head from the center of the left tragus to the center of the right tragus.



3 HEAD CIRCUMFERENCE

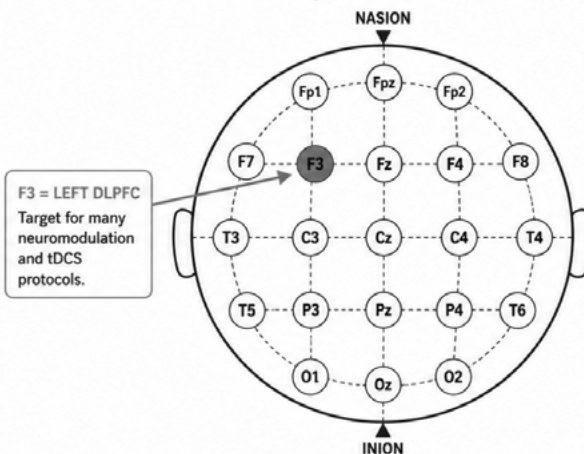
Measure around the head at the largest circumference, passing above the eyebrows and over the most prominent part of the occiput.



i Record all three measurements. These values inform proportional placement based on the Beam F3 method.

STEP 2: 10-20 EEG MAP – LOCATE F3

Using the 10-20 system, F3 represents the approximate location of the left dorsolateral prefrontal cortex (DLPFC).



● F3 approximates the left DLPFC (Brodmann area 46/9)

○ Individual anatomy varies; use the Beam F3 measurements for precision.

Beam, W., Borckardt, J. J., Reeves, S. T., & George, M. S. (2009). An efficient and accurate new method for locating the F3 position for prefrontal TMS applications. *Brain Stimulation*, 2(1), 50–54.

Neurophysiological Basis: LTP/LTD-Like Plasticity

rTMS effects are believed to share molecular machinery with activity-dependent synaptic plasticity — the same NMDA-receptor-dependent processes that underlie learning and memory

● Calcium influx:

Repeated depolarization opens NMDA receptors, admitting Ca^{2+}

● Synaptic tagging:

High-frequency trains favor LTP-like strengthening; low-frequency favors LTD-like weakening

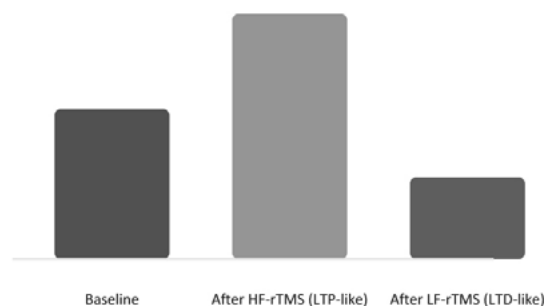
● Receptor trafficking:

AMPA receptor density at the synapse increases (LTP) or decreases (LTD)

● Duration:

Effects can outlast the stimulation session — the basis for cumulative, multi-day protocols

SYNAPTIC STRENGTH SHIFT



CLINICAL USE

Functional & Clinical Applications



Depression

HF-rTMS over left DLPFC — FDA-cleared for treatment-resistant MDD



OCD

HF over dmPFC/ACC (H-coil) — FDA-cleared adjunct therapy



Stroke Rehabilitation

LF over contralesional M1 or HF over ipsilesional M1 — motor recovery



Chronic Pain

HF over M1 — modulates pain-processing networks



Tinnitus

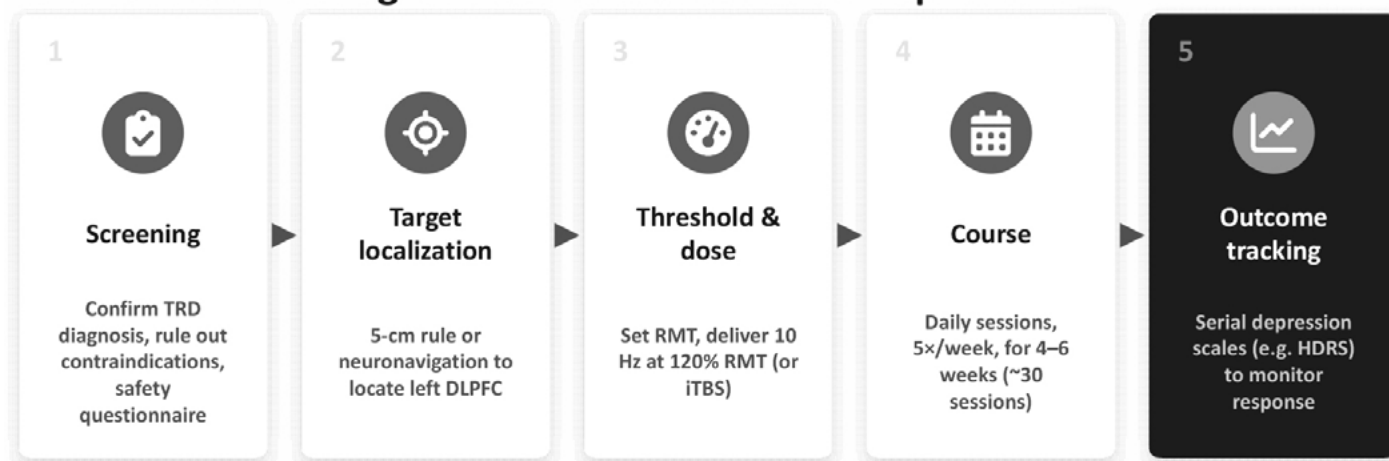
LF over temporoparietal cortex — reduces phantom auditory perception



Addiction / Smoking

HF over DLPFC or insula (H-coil) — reduces craving

Protocol Walkthrough — Treatment-Resistant Depression



Illustrative protocol for teaching purposes — actual parameters follow site-specific clinical guidelines

SAFETY

Safety Screening & Contraindications

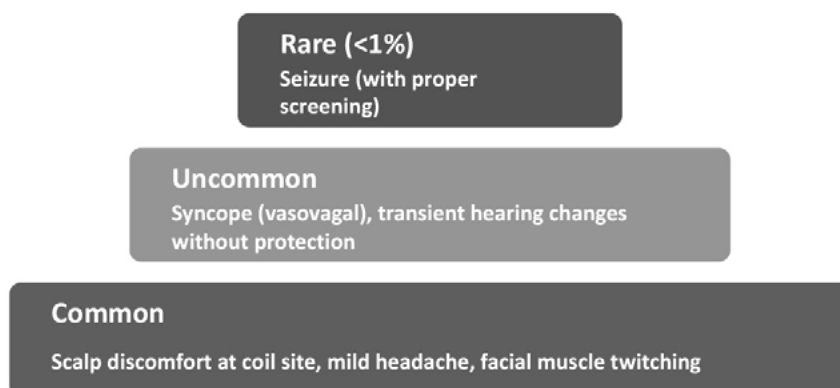
ABSOLUTE PRECAUTIONS

-  **Ferromagnetic implants**
Cochlear implants, aneurysm clips, cranial hardware near coil
-  **Implanted electronic devices**
Pacemakers, deep brain stimulators, medication pumps
-  **Seizure history**
Personal history of seizures or epileptogenic lesions (relative)

RELATIVE PRECAUTIONS

-  **Medications**
Drugs that lower seizure threshold
-  **Sleep deprivation**
Increases seizure risk — screen before session
-  **Pregnancy**
Generally considered low-risk but requires case-by-case judgment

Adverse Effects: Frequency Pyramid



With standard safety screening (per published consensus guidelines), rTMS has an excellent safety record — seizure risk is estimated at well under 1 in 1,000 sessions

PART TWO



Transcranial Direct Current Stimulation

Electrical principles, montages, parameters, and clinical applications of tDCS

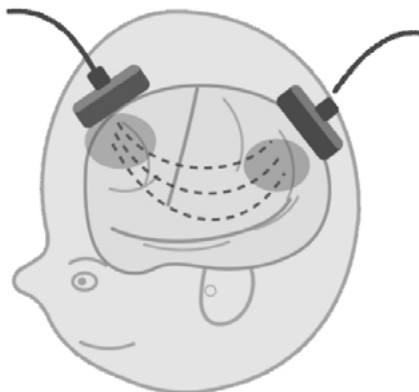


Direct Current Stimulation

BASIC PRINCIPLE

Weak Electrical Current Flow

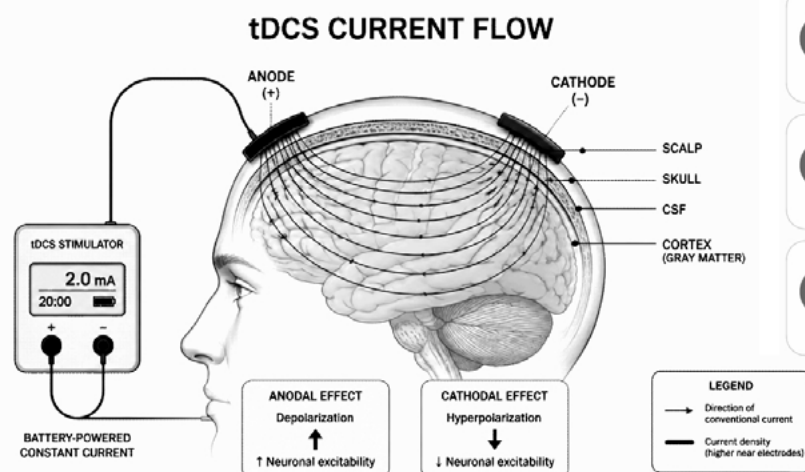
A constant, low-amplitude current (typically 1–2 mA) flows between scalp electrodes, passing through skin, skull and CSF to reach the cortex — modulating the probability of firing without directly triggering action potentials



- 1 **Constant-current stimulator**
A battery-powered source holds output steady at 1–2 mA despite changing impedance
- 2 **Anode (+) — current enters**
Beneath it the resting membrane potential shifts toward threshold
- 3 **Current spreads through tissue**
Only a fraction reaches cortex; much is shunted through scalp and CSF
- 4 **Cathode (–) — current exits**
Beneath it the membrane potential shifts away from threshold
Excitability shifts in opposite directions beneath the anode and the cathode — polarity, not frequency, sets the direction

Figure Focus: tDCS Current Flow

Visualizing current entry, spread through tissue, and polarity-dependent effects



Key Points

- 1 Current enters at the anode and exits at the cathode while traversing scalp and intracranial tissue
- 2 Anodal stimulation generally shifts membrane potential toward threshold; cathodal shifts it away
- 3 The figure clarifies why montage design and electrode placement strongly influence outcomes

HARDWARE

Anatomy of a tDCS System



Battery-Powered Stimulator

Small, portable constant-current source — typically 9V battery-driven for safety



Current Control Dial

Sets output current (mA) and session timer; ramps up/down gradually



Electrodes

Conductive rubber pads, typically 25–35 cm² surface area



Saline-Soaked Sponges / Gel

Interface layer that ensures even current distribution and reduces impedance



Head Straps / Cap

Secures electrode position and pressure throughout the session



Impedance Monitor

Continuously checks contact quality to prevent hot-spots or burns

SETUP

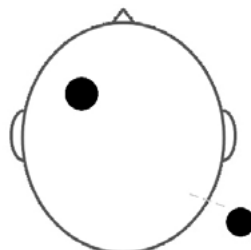
Electrode Montages



Bilateral

Anode and cathode both sit over cortical targets — e.g. anodal left DLPFC with cathodal right DLPFC

● Anode (+) ● Cathode (-)



Unilateral + Extracephalic

One electrode over the cortical target, the return electrode placed off the head (e.g. shoulder)



High-Definition (4x1)

A centre electrode ringed by four returns confines current to a much smaller cortical patch

MECHANISM

Subthreshold Membrane Polarization

Unlike TMS, tDCS does not directly trigger action potentials. Instead, the weak current shifts resting membrane potential — making neurons more or less likely to fire in response to their own natural inputs



Under the Anode (+)
Depolarization

Threshold

Resting (–70mV)

Resting membrane potential shifts toward threshold — neurons become more excitable and more likely to fire



Under the Cathode (–)
Hyperpolarization

Threshold

Resting (–70mV)

Resting membrane potential shifts away from threshold — neurons become less excitable and less likely to fire

Polarity Matters: Anodal vs. Cathodal Effects



Anodal tDCS

EXCITABILITY INCREASE

CLINICAL LOGIC

Boosting hypoactive regions — e.g. left DLPFC in depression, ipsilesional M1 after stroke, cognitive enhancement studies



Cathodal tDCS

EXCITABILITY DECREASE

CLINICAL LOGIC

Calming hyperactive regions — e.g. pain-processing cortex, epileptogenic foci (investigational)

PEDAGOGY

tDCS Parameters That Matter



Current intensity

Typically, 1–2 mA constant current, ramped up/down over ~10–30 sec



Electrode size

Larger pads spread current (less focal); smaller pads/HD arrays increase focality



Current density

Intensity + electrode area — key determinant of safety and local effect



Session duration

Commonly 20–30 minutes per session



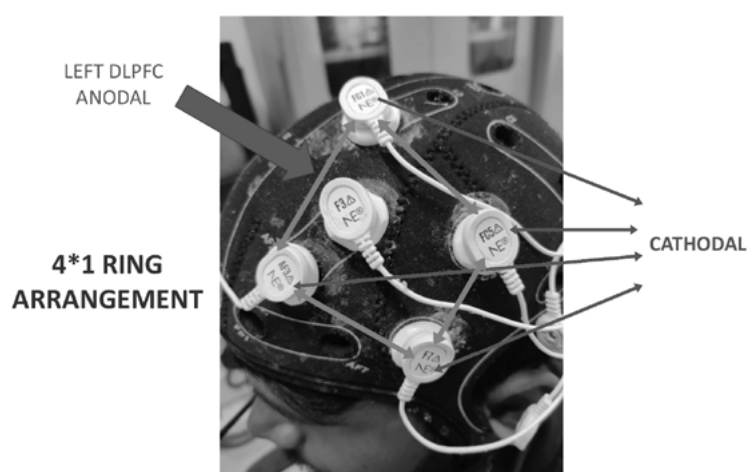
Montage / polarity

Anode & cathode placement determines which region is facilitated vs. suppressed



Session number

Often 5–10+ consecutive daily sessions for cumulative clinical effect



RELATED TECHNIQUES

Beyond tDCS: tACS and tRNS

tDCS

Direct Current

Constant unidirectional current
— polarity-specific excitability shift

tACS

Alternating Current



Sinusoidal current at a set frequency — entrains and synchronizes endogenous cortical oscillations

tRNS

Random Noise



Current amplitude/frequency varies randomly — proposed stochastic resonance effect enhances signal detection

CLINICAL USE

Functional & Clinical Applications



Depression

Anodal over left DLPFC ($\pm$ cathodal right) — adjunct or standalone trials



Chronic Pain

Anodal over M1 — widely studied for fibromyalgia, neuropathic pain



Stroke Motor Recovery

Anodal ipsilesional / cathodal contralesional M1 — adjunct to motor training



Cognitive Enhancement

Anodal over DLPFC or parietal cortex — working memory, attention research



Tinnitus

Cathodal/anodal over temporoparietal or DLPFC regions



Aphasia Rehabilitation

Anodal over perilesional language areas paired with speech therapy

COMBINED APPROACHES

Pairing tDCS with Cognitive & Motor Training

Because tDCS modulates the probability of firing rather than driving it directly, its effects are strongly activity-dependent — concurrent task engagement shapes which synapses are strengthened



SAFETY

Safety, Tolerability & Adverse Effects

COMMON & MILD (TOLERABILITY)



Tingling / itching

Under the electrodes, especially at ramp up/down



Skin redness (erythema)

Transient, resolves within minutes to hours



Mild fatigue or headache

Occurs in a minority of sessions

RARE — REQUIRES CARE



Skin burns

From inadequate sponge saturation or poor contact — preventable with proper technique



Contraindications

Broken skin at electrode site, metal implants under electrodes

tDCS is generally considered well-tolerated at conventional doses ($\leq 2\text{mA}$, $\leq 40\text{cm}^2$ electrodes) — no confirmed reports of seizure at these parameters in the literature.

PART THREE



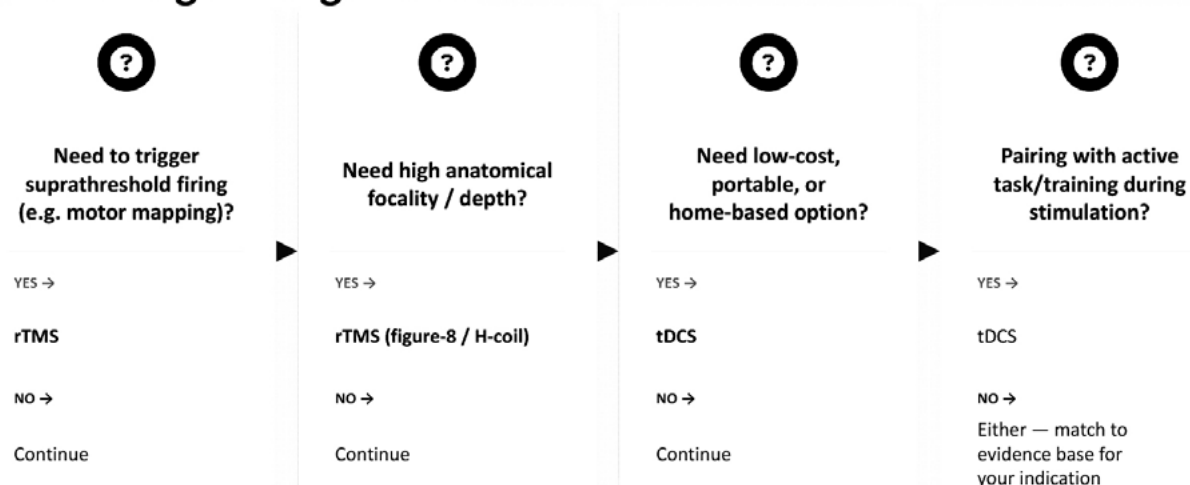
Synthesis & Future Directions

Comparing rTMS and tDCS, guiding stimulation with imaging, and where the field is heading

rTMS vs. tDCS: Side-by-Side

DIMENSION	rTMS	tDCS
Energy delivered	Magnetic field → induced current	Direct weak electrical current
Direct neuronal firing?	Yes — can trigger action potentials	No — subthreshold modulation only
Focality	High (especially figure-8 coil)	Lower (higher with HD-tDCS)
Portability & cost	Bulky, expensive equipment	Small, low-cost, portable
Blinding / sham	More difficult (clicking sound, scalp sensation)	Easier (brief tingling only at onset)
Typical session	~20–40 min, sits under coil	~20–30 min, wearable during tasks
Evidence base	FDA-cleared for MDD, OCD; large trials	Strong research base; fewer regulatory approvals

Choosing the Right Tool



Guiding Stimulation with Neuroimaging & EEG

Individual anatomy and network organization vary — imaging and electrophysiology increasingly personalize target selection and dosing rather than relying on scalp landmarks alone



MRI-Guided Neuronavigation

Individual structural MRI co-registered with coil position for precise, reproducible targeting



Resting-State fMRI

Functional connectivity maps identify optimal network nodes (e.g. DLPFC subregion connected to subgenual cingulate)



EEG Biomarkers

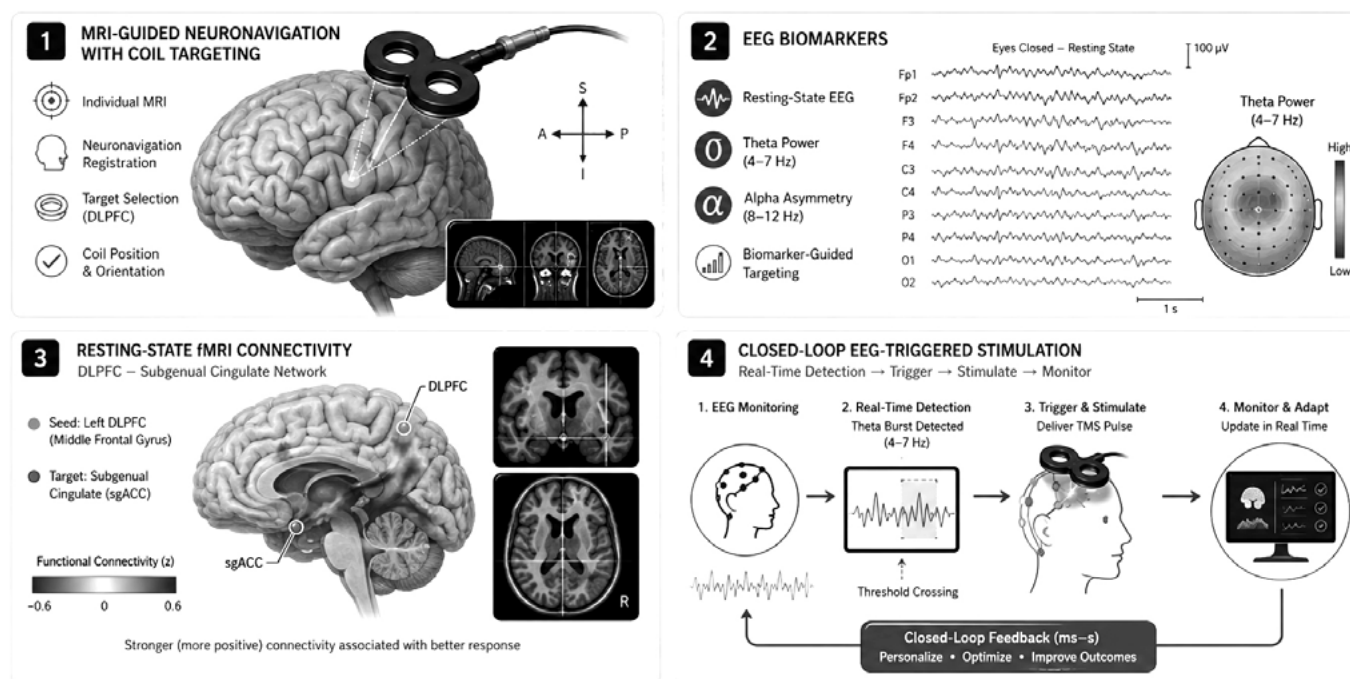
Resting-state or task-related EEG signatures help select candidates and predict response



Closed-Loop / EEG-Triggered

Real-time EEG can trigger stimulation pulses at specific oscillatory phases

Guiding Stimulation with Neuroimaging & EEG



Current Research Frontiers



Personalized Dosing

Individual anatomy & E-field modeling to standardize actual cortical dose



Biomarker-Guided Selection

EEG/fMRI signatures to predict who will respond before treatment begins



Home-Based tES

Remotely supervised tDCS/tACS expanding access beyond clinic visits



Combined Therapies

NIBS paired with pharmacotherapy or psychotherapy for synergistic effect



Network-Based Targeting

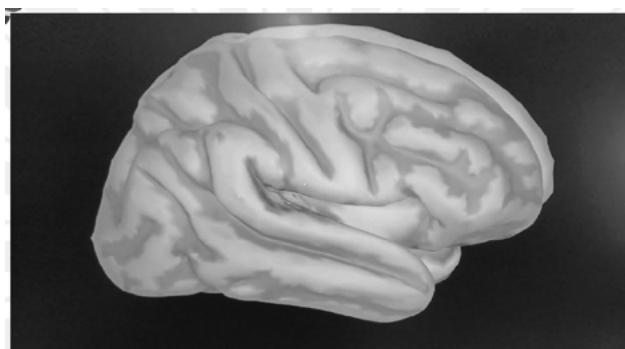
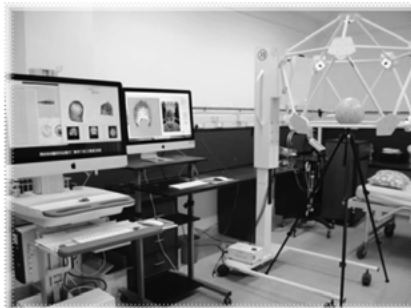
Moving from single regions to distributed circuit-level targeting



Expanding Populations

Pediatric, geriatric, and pregnancy safety/efficacy data still maturing

Precision Neuromodulation...



Region of interest to be left primary auditory cortex as localized manually by EEG using ASSR

Take-Home Messages



rTMS uses magnetic induction to drive suprathreshold neuronal firing — frequency sets the direction (inhibitory vs. excitatory) of lasting effect



tDCS uses weak direct current to shift resting membrane potential — polarity (anodal vs. cathodal) sets the direction of excitability change



Parameters are not interchangeable between techniques — dosing, focality, and depth trade-offs should guide protocol selection



Both techniques have strong safety profiles when screening and parameter guidelines are followed rigorously



Clinical evidence is most mature for depression and OCD (rTMS) and pain/motor rehabilitation (tDCS) — but the field is expanding rapidly

Efficacy of Divalproex in acute mania, bipolar depression & maintenance therapy for bipolar disorder: An Overview

Dr. Nishant Saha, MD(Psychiatry), Medical Officer, Addiction Treatment Facility
Murshidabad Medical College & Hospital

Bipolar Disorders

- Bipolar disorder is a mood disorder that is characterized by episodes of mania, hypomania, and major depression.
- Subtypes of bipolar disorder: bipolar I and bipolar II

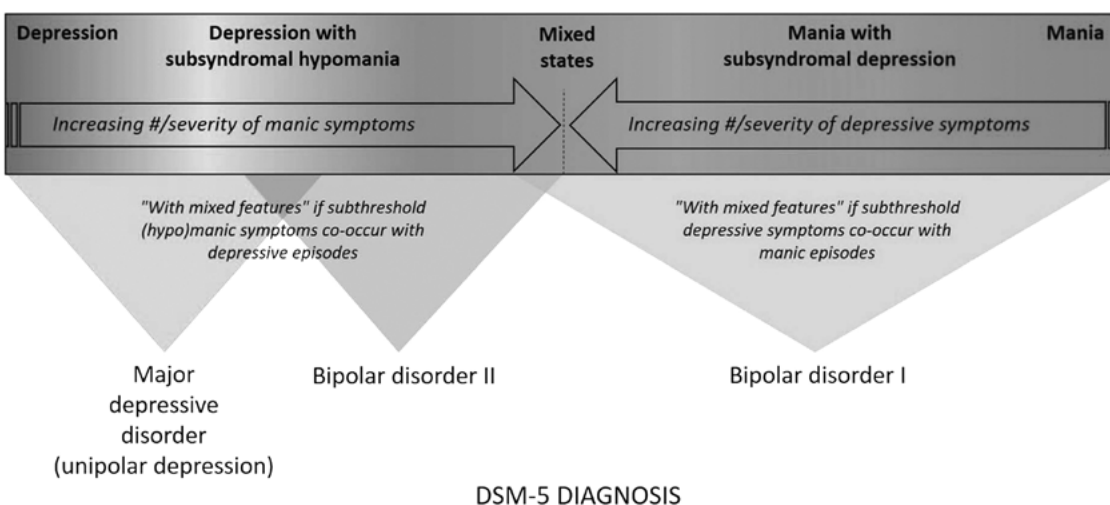
American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 5th Edition, American Psychiatric Association, Arlington, VA, 2013.

Bipolar Disorder Epidemiology

- Global
- Estimated lifetime prevalence: among adults 1-3%
- Community surveys in 14 countries: lifetime prevalence of bipolar I & bipolar II disorder: 2.8%
- Another set of community surveys in 11 countries: lifetime prevalence
 - bipolar I disorder: 0.6%
 - bipolar II disorder 0.4%
- Mean age of onset
 - Bipolar I disorder: 18 years
 - bipolar II disorder: 20 years
- The ratio of men to women who develop bipolar disorder:~1:1

Up2date 2014. Available from: <http://www.uptodate.com/contents/bipolar-disorder-in-adults-epidemiology-and-pathogenesis?topicKey=PSYCH%2F678&elapsedTimeMs=7&view=print&displayedView=full#>

Mood disorders spectrum & DSM–5 diagnosis



CNS Spectrums (2017),
22, 203–219.

DSM-5 Diagnosing Criteria**Bipolar I vs II Disorder**

Bipolar I disorder	Bipolar II disorder	
Manic episode(s)	Yes	No
Hypomanic episode(s)	Commonly occur, but not required	Yes
Major depressive episode(s)	Usually occur, but not required	Yes
Mixed features	May occur	May occur
Anxious distress	May occur	May occur
Rapid cycling	May occur	May occur
Psychotic features	May occur	May occur
Catatonia	May occur	May occur

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 5th Edition, American Psychiatric Association, Arlington, VA, 2013.

DSM-5 - Specifiers for bipolar and related disorders

Specifier	Manic episode	Depressive episode	Illness course
Anxious distress	X	X	
Mixed features	X	X	
Rapid cycling			X
Melancholic features		X	
Atypical features		X	
Psychotic features	X	X	
Catatonia	X	X	
Peripartum onset	X	X	
Seasonal pattern			X
Remission	X	X	
Current episode severity	X	X	

Divalproex - MOLECULAR PROFILE**Valproate Kinetics**

Highly protein bound to albumin and prealbumin



The free fraction goes from 10% to 18.5%



Efficacy and adverse effects are mediated by free valproate levels

Chadwick, D. W. (1985). Concentration-effect relationships of Valproic acid. Clinical Pharmacokinetics, 10(2), 155-163.

Valproic Acid (VPA) Level

- No need to separate the free fraction from the bound fraction unless:
 - Another highly protein-bound drug is being used
 - There is a low serum albumin level

Chadwick, D. W. (1985). Concentration-effect relationships of Valproic acid. Clinical Pharmacokinetics, 10(2), 155-163.

Valproic Acid (VPA) Level

	Therapeutic Range
Free Valproate Level (mcg/mL)	7–23
Total Valproate Level (mcg/mL)	60–120

Epilepsy: 50 to 100 mcg/mL of total valproate; Mania: plasma total valproate 85 and 125 mcg/mL

Chadwick, D. W. (1985). Concentration-effect relationships of Valproic acid. Clinical Pharmacokinetics, 10(2), 155-163.

Valproate Kinetics



Half-life: 9–16 hours



Hydrophilic



Low volume of distribution

Chadwick, D. W. (1985). Concentration-effect relationships of Valproic acid. *Clinical Pharmacokinetics*, 10(2), 155-163.

Valproate: Sublinear Kinetics

At higher doses, the higher free fraction stays in the plasma compartment	→	- It is cleared by the liver - The valproate levels do not increase in a dose-dependent manner
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Chadwick, D. W. (1985). Concentration-effect relationships of Valproic acid. *Clinical Pharmacokinetics*, 10(2), 155-163.

Valproate: Absolute Contraindications



Mitochondrial disorders or mutations



Urea cycle disorders



Children < 2 years



Pregnancy



Allergies



Childbearing potential without effective contraception

divalproex sodium. [package insert]. U.S. Food and Drug Administration website. https://www.accessdata.fda.gov/drugsatfda_docs/label/2020/018723s063lbl.pdf

Revised December 2020. Accessed Dec, 2020.

Valproate in patient with liver impairment

- Divalproex should not be administered to patients with hepatic disease or significant hepatic dysfunction

divalproex sodium. [package insert]. U.S. Food and Drug Administration website. https://www.accessdata.fda.gov/drugsatfda_docs/label/2020/018723s063lbl.pdf.

Revised December 2020. Accessed Dec, 2020.

Divalproex ER

Advantages over Delayed-release (DR) Formulation

- Divalproex ER was associated with superior tolerability with less frequent tremor, weight gain, and gastrointestinal complaints (all $P < 0.001$), but not less hair loss.¹
- ER formulations can be administered once daily²
 - Increased compliance, convenience observed with fewer dosages per day

1. *Epilepsy & Behavior*. 2004;5:746–51.

2. *CNS Drugs* 2007;21(9):765-74.

Divalproex and Valproate



Divalproex becomes valproate



Can increase AST or ALT

Chadwick, D. W. (1985). Concentration-effect relationships of Valproic acid. *Clinical Pharmacokinetics*, 10(2), 155-163.

Divalproex

Mechanism of Action: Bipolar disorder

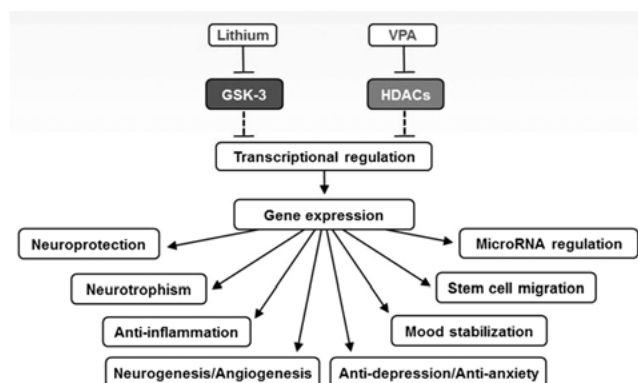
- In BPD: dysfunction may be mediated primarily by downregulation in expression of GABAergic genes (e.g., glutamic acid decarboxylase⁶⁷ [GAD67] and reelin) associated with DNA methyltransferase (DNMT)-dependent hypermethylation of their promoters

- Divalproex:
 - ↑ es GABA activity
 - Mimics action of GABA at postsynaptic receptor
 - Enhances BDNF expression
 - Inhibits histone deacetylase (HDAC)
 - ↑ es antiapoptotic protein Bcl-2
- Induce DNA-demethylation —> facilitate chromatin remodeling.

BDNF, brain-derived neurotrophic factor Neuropharmacology 2011 Jun; 60(7–8):1007-16.

Divalproex

Mechanism of Action: Bipolar disorder



HDACs, histone deacetylases; GSK-3, glycogen synthase kinase-3 Pharmacol Rev 2013 Jan;65:105–42.

Indications

- Acute treatment of manic or mixed episodes associated with bipolar disorder, with or without psychotic features
- Monotherapy and adjunctive therapy of complex partial seizures and simple and complex absence seizures; adjunctive therapy in patients with multiple seizure types that include absence seizures
- Prophylaxis of migraine headaches

DIVALPROEX IN BIPOLAR DISORDERS

Divalproex: Treatment of BPD – Acute Phase

Divalproex vs. placebo

- Randomized, placebo-controlled, trial
- Total: n=54; bipolar I: n = 20; bipolar II: n = 34 patients
- 67% (36 of 54) met DSM-IV criteria for rapid cycling.
- Divalproex: statistically significant improvement in MADRS scores compared with placebo from week 3 onward.

Parameter	Divalproex	Placebo	P- value
Proportions of patients meeting response criteria	38.50% (10 of 26)	10.70% (3 of 28)	0.017
proportions of patients meeting remission criteria	23.10% (6 of 26)	10.70% (3 of 28)	0.208

- Divalproex sodium: efficacious & reasonably well tolerated in the acute treatment of mood stabilizer-naïve patients with bipolar depression, particularly for those with rapid-cycling type I presentations

J Clin Psychiatry 2011 Jun;72(6):813-9.

Comparative Efficacy of Introducing Mood Stabilizers in Acute or Maintenance Treatment of Bipolar Disorder: Observational Prospective Study

- 55 bipolar patients aged 14-77 years were recruited through a convenience sampling technique.
- Most of the respondents were between 25-36 years (54.6%). Females constituted 58.6% of the sample.
- There was no difference in the duration of symptom resolution between given mood stabilizers within 0-3 days (Median=2, N=33) and 4- 16 days (Median=2 N=20).
- There was an association between insight and religion ($p=.005$) and black ethnicity ($p=.002$).
- Insight was associated with adherence ($p<.001$).
- Sodium valproate had a faster onset of mood stabilizing effect (less than 7 days) than carbamazepine (7-14 days) and the psychiatrist considered medication effectiveness and client history as the most important factors in the choice of a mood stabilizer for BD.

Impaired insight is a predictor for non-adherence. Mood stabilizers can be started at any time in the acute treatment of bipolar disorder. Sodium valproate must be given a priority in the acute treatment of bipolar disorder, except in women of reproductive age.

International Journal of Multidisciplinary Studies and Innovative Research. 2023;11(5):1700-1719. DOI: 10.53075/Ijmsirq/098877567556556

Divalproex ER: Acute Bipolar II depression

Divalproex vs. placebo

- 7-week, open-label trial of divalproex-ER monotherapy / adjunctive therapy
- Mean MADRS scores (LOCF): ↓ed significantly from baseline; from 30.1 to 15.2, $p<.00001$
- Overall response rate: 54%.
- Divalproex-ER: generally well tolerated, with no early discontinuations due to adverse events.

LOCF: last observation carried forward J Affect Disord. 2010 Jul;124(1-2):170-3.

Divalproex: Maintenance Treatment of BPD

- Preventing study withdrawal due to any mood episode
 - Valproate was more effective than placebo
 - No difference in efficacy was found between valproate and lithium
- Participants dropping out of treatment for any cause
 - Valproate was associated with fewer participants dropping out when compared with placebo or lithium
- Prevent relapse
 - Combination therapy with lithium plus valproate was more likely to prevent relapse than monotherapy with valproate

Cochrane Database of Systematic Reviews 2013, Issue 10. Art. No.: CD003196. DOI:10.1002/14651858.CD003196.pub2.

Suicide Attempts

Divalproex vs. Li, SGAs

- 6 year retrospective study of Bipolar disorder

Medicine	Suicide attempts /10,000 months of exposure
Li + divalproex	6.3
Divalproex alone	7.0
Lithium alone	7.7
Atypical antipsychotics alone	26.1

- Lithium & divalproex provided protection against suicide attempts

J Affect Disord 2013 Feb 15;145(1):77-82.

Anti-suicidal effectiveness of valproate

- Despite advances in psychiatric treatment, individuals with schizophrenia (SZ) and bipolar disorder (BD) continue to experience alarmingly high suicide rates.
- This study aimed to evaluate the anti-suicidal effectiveness of these medications using a nationwide health insurance database in South Korea.
- A retrospective cohort study was conducted using data from the National Health Information Database.
- This study included 102,540 patients with SZ and 96,336 patients with BD diagnosed between 2007 and 2010.
- Suicide hazard ratios (HR) were calculated using a time-dependent Cox regression analysis.
- Suicide rates per 100,000 person-years were 308.0 for SZ and 285.1 for BD.
- After adjustment for confounders, lithium and valproate prescriptions were associated with significantly lower suicide hazard ratios in both disorders
 - SZ
 - o HR of lithium: 0.58, 95 % CI: 0.46-0.72;
 - o HR of valproate: 0.61, 95 % CI: 0.52-0.71
 - BD
 - HR of lithium: 0.54, 95 % CI: 0.44-0.65;
 - HR of valproate: 0.66, 95 % CI: 0.57-0.76
- Clozapine was associated with a lower suicide hazard in patients with SZ but remained statistically non-significant. Lithium and valproate have significant anti-suicidal effects in patients with SZ and BD, underscoring the potential role of mood stabilizers in suicide prevention among them.

J Psychiatr Res. 2025 Mar 26;185:105-111. doi: 10.1016/j.jpsychires.2025.03.045. Online ahead of print.

Divalproex

Cost-effective analysis

- Divalproex sodium extended-release (ER) vs valproic acid
- 2 primary outcomes: GI side effects; treatment success

Parameter	Divalproex - ER	Valproic acid
Average probability of GI adverse event	0.10	0.36
Average probability of treatment success	0.58	0.45
Cost/patient	\$25 336.13	\$34 208.84

- Divalproex sodium ER results in lower total costs than valproic acid in the treatment of bipolar disorder
- Negative incremental cost of using Divalproex sodium ER relative to VPA in a majority (approximately 70%) of cases

Curr Med Res Opin 2007 Aug; 23(8);1997-2004.

Valproate Use During Spermatogenesis and Risk to Offspring

This nationwide cohort study included 12,35,353 singletons; 1336 children had fathers who had filled prescriptions for valproate during spermatogenesis (ie, 3 months prior to conception).

Children with major congenital malformations in the first year of life and with neurodevelopmental disorders before death or end of follow-up were identified in Danish health registers. Adjusted relative risks (ARRs) of congenital malformations, adjusted hazards ratios (AHRs) of neurodevelopmental disorders were calculated and adjusted for relevant confounders.

- Among 12,35,353 live births, 634,415 [51.4%] were boys.
- Median follow-up
 - 10.1 years (IQR, 5.1-14.8 years) for valproate-exposed children
 - 10.3 years (IQR, 5.2-15.6 years) for valproate-unexposed children.
- 43,903 children (3.6%) received a diagnosis of major congenital malformations in the first year of life, and 51,633 children (4.2%) received a diagnosis of neurodevelopmental disorders during follow-up.
- When comparing the risk among valproate-exposed children with that among unexposed children,
 - ARR of major congenital malformations - 0.89 (95% CI, 0.67-1.18)
 - AHR of neurodevelopmental disorders - 1.10 (95% CI, 0.88-1.37),
 - AHR of autism spectrum disorder - 0.92 (95% CI, 0.65-1.30).
- There still was NO increased risk of any of the included end points in analyses addressing the robustness of the findings, ie,
 - dose-response analyses, sibling analyses, analyses restricted to children of fathers with epilepsy,
 - analyses that used children with paternal lamotrigine exposure as active comparator,
 - analyses that used children with paternal exposure to valproate only before spermatogenesis as a negative control exposure

In all analyses based on this large Danish cohort study, results suggest that exposure to valproate during spermatogenesis was NOT associated with offspring risk of congenital malformations or neurodevelopmental disorders, including autism spectrum disorder.

JAMA Netw Open. 2024;7(6):e2414709. doi:10.1001/jamanetworkopen.2024.14709

Impact of mood stabilizers on creativity

While a DSM-5 criterion for both hypomania and mania is impaired functioning, the majority of those with a bipolar condition report improved functioning. When offered a mood stabilizer, many express concerns about any impact on their creativity.

- Objective: to address impact of mood stabilizer on creativity.
- Method: Reference is made to the impact of differing mood stabilizers on cognitive performance and the limited data on any specific impact on creativity, while some personal observations are offered.
- Results: There appears to be a distinctive gradient in the cognitive impacts of differing mood stabilizers, with
 - Lithium offering the highest risk
 - Carbamazepine and valproate providing a slight risk
 - Lamotrigine seemingly without cognitive side-effects.
- The question argues clinician for close observation of any cognitive side-effects when lithium is introduced.

Australasian Psychiatry. 2023;0(0):1-3. DOI: 10.1177/10398562231211127

Divalproex ER Dose in Bipolar Disorder

- Long acting preparations: prescribed once/day
 - Initial: 25 mg/kg/day given once daily
 - Dose should be adjusted as rapidly as possible to desired clinical effect
 - Maximum recommended dose: 60 mg/kg/day.

Up2date 2014. Available from: <http://www.uptodate.com/>

Divalproex

Placement according to Canadian Network for Mood and Anxiety Treatments (CANMAT) and International Society for Bipolar Disorders (ISBD) 2018 guideline

DOI: 10.1111/bdi.12609

ORIGINAL ARTICLE

WILEY BIPOLAR DISORDERS

Canadian Network for Mood and Anxiety Treatments (CANMAT) and International Society for Bipolar Disorders (ISBD) 2018 guidelines for the management of patients with bipolar disorder

Divalproex in bipolar disorders: CANMAT Guideline

Why is divalproex recommended as a first-line maintenance treatment for bipolar I disorder?

In the only large double-blind placebo controlled RCT of divalproex monotherapy (373), it was not more effective than placebo in preventing relapse of mood episodes i.e. time to any mood episode. However, in this trial, lithium which has been shown in many other studies to be effective in relapse prevention, was also found to be no more effective than placebo. Thus, these results suggest that this trial was a failed trial and not a negative trial.

Most modern studies of maintenance therapy use enriched design, meaning that those that responded in acute phase to the medication being tested are randomized to continuation of the same drug or replacement with placebo. This practice to a large extent mirrors clinical practice as clinicians are likely to continue the medication that worked in the acute phase for maintenance treatment. Interestingly, in the divalproex RCT, some but not all patients that were randomized into the double-blind phase were divalproex responders. In a post-hoc analysis of this study, in this enriched subgroup of patients that responded to divalproex during the acute phase and randomized to continuation of divalproex vs switch to placebo, divalproex was more effective in preventing relapse of mood episodes compared with placebo.

Further, divalproex was superior to placebo on a number of other secondary efficacy measures such as lower rates of discontinuation for any mood episode or a depressive episode. Surprisingly, there was also a trend for superiority of divalproex relative to lithium in time to any mood episode. Other RCTs have shown that divalproex is as effective as lithium (221) in preventing relapse of mood episodes.

As well, two meta-analysis have concluded that divalproex is effective in preventing relapse of mood episodes (370, 372), and a population based cohort study in the UK showed that there were no differences in efficacy between divalproex, quetiapine and olanzapine in the maintenance treatment of bipolar disorder (359).

Taken together, we believe these efficacy data support our rationale for a Level 1 rating. This along with clinical experience, real world cohort data, and safety, justify our recommendation of divalproex as a first-line maintenance treatment.

Bipolar Disord. 2018 Mar 14. doi: 10.1111/bdi.12609.

Hierarchical rankings of first and second-line treatments recommended for management of acute mania

	Level of evidence by phase of treatment					Considerations for treatment selection				
	Acute mania	Maintenance Prevention of any mood episode	Prevention of mania	Prevention of depression	Acute depression	Acute Safety concerns	Tolerability concerns	Maintenance Safety concerns	Tolerability concerns	Risk of depressive switch
First-line treatments: Monotherapies										
Lithium	●	●	●	●	●	+	+	++	++	+
Quetiapine	●	●	●	●	●	+	++	++	++	+
Divalproex	●	●	●	●	●	+	+	++*	+	+
Aripiprazole	●	●	●	●	n.d.	+	+	+	+	+
Asipiprazole	●	●	●	n.d.*	n.d.	+	+	+	+	+
Risperidone (4-6 mg)	●	●	●	n.d.*	n.d.	+	+	+	++	+
Risperidone	●	●	●	n.d.	n.d.	+	+	+	++	+
Cariprazine	●	n.d.	n.d.	n.d.	●	+	+	+	+	+
First-line treatments: Combination therapies										
Quetiapine + Li/DVP	●	●	●	●	●*	+	++	+++*	++	+
Aripiprazole + Li/DVP	●	●	●	n.d.*	●	+	+	+++*	++	+
Risperidone + Li/DVP	●	●	●	n.d.	●	+	++	+++*	++	+
Aripiprazole + Li/DVP	●	●	●	n.d.	●	+	+	+++*	+	+
Second-line treatments: Combination therapies										
Olanzapine	●	●	●	●	●*	+	++	+++	++	+
Carbamazepine	●	●	●	●	●	+	++	+++	++	+
Olanzapine + Li/DVP	●	●	●	●	n.d.	+	++	+++*	++	+
Lithium + DVP	●	●	●	n.d.	n.d.	+	++	++	++	+
Ziprasidone	●	●	●	n.d.	●	++	++	++	+	+
Haloperidol	●	n.d.	●	●	n.d.	+	++	+++	++	++
ECT	●	●	●	●	●	+	++	+	++	+

DVP, divalproex; ECT, electroconvulsive therapy; Li, lithium.

●, level 1 evidence; ●, level 2 evidence; ●, level 3 evidence; ●, level 4 evidence; ●, level 1 negative evidence; ●, level 2 negative evidence; ●, level 3 negative evidence; ●, level 4 negative evidence; n.d., no data.

*Although monotherapies are listed above combination therapies in the hierarchy, combination therapies may be indicated as the preferred choice in patients with previous history of partial response to monotherapy and in those with psychotic mania or in situations where rapid response is desirable.

*Did not separate from placebo in those with index mania; no studies available in index depression.

*No controlled trials; however, clinical experience suggests that it is a useful strategy.

Bipolar Disord. 2018 Mar 14. doi: 10.1111/bdi.12609.

Hierarchical rankings of first and second-line treatments recommended for management of acute bipolar I depression

	Level of evidence by phase of treatment					Considerations for treatment selection				
	Maintenance				Acute mania	Acute		Maintenance		Risk of manic/hypomanic switch
	Acute depression	Prevention of any mood episode	Prevention of depression	Prevention of mania		Safety concerns	Tolerability concerns	Safety concerns	Tolerability concerns	
First-line treatments										
Quetiapine	●	●	●	●	●	+	++	++	++	-
Lurasidone + Li/DVP	●	● ^a	● ^a	● ^a	n.d.	+	++	++ ^a	++/+	-
Lithium	●	●	●	●	●	+	+	++	++	-
Lamotrigine	●	●	●	●	■	++	=	=	=	=
Lurasidone	●	●	●	●	n.d.	=	+	=	+	=
Lamotrigine (adj)	●	●	●	●	■	++	+	++	++	=
Second-line treatments										
Divalproex	●	●	●	●	●	-	+	++ ^a	+	-
SSRIs/bupropion (adj)	●	n.d.	●	n.d.	n.d.	-	+	-	+	+
ECT	●	●	●	●	●	+	++	+	++	-
Cariprazine	●	n.d.	n.d.	n.d.	●	-	+	-	-	-
Olanzapine-fluoxetine	●	n.d.	n.d.	n.d.	n.d.	+	++	+++	+	+

adj, adjunctive; DVP, divalproex; ECT, electroconvulsive therapy; Li, lithium; SSRIs, selective serotonin reuptake inhibitors.

●, level 1 evidence; ●, level 2 evidence; ●, level 3 evidence; ●, level 4 evidence; ■, level 1 negative evidence; ■, level 2 negative evidence; ■, level 3 negative evidence; ■, level 4 negative evidence; n.d., no data; -, limited impact on treatment selection; +, minor impact on treatment selection; ++, moderate impact on treatment selection; +++, significant impact on treatment selection.

^aTrend for superiority on the primary efficacy measure, hence the lower rating.

^bEffective in those with an index episode of depression.

^cNegative data from the trial are probably due to methodological issues; rating based on expert opinion.

^dDivalproex and carbamazepine should be used with caution in women of child bearing age.

Bipolar Disord. 2018 Mar 14. doi: 10.1111/bdi.12609.

Hierarchical rankings of first-and second-line treatments recommended for maintenance treatment in bipolar disorder

	Level of evidence by phase of treatment					Considerations for treatment selection				
	Maintenance			Acute		Acute		Maintenance		
	Prevention of any mood episode	Prevention of depression	Prevention of mania	Depression	Mania	Safety concerns	Tolerability concerns	Safety concerns	Tolerability concerns	
First-line treatments										
Lithium	●	●	●	●	●	+	+	++	++	
Quetiapine	●	●	●	●	●	+	++	++	++	
Divalproex	●	●	●	●	●	-	+	++ ^c	+	
Lamotrigine	●	●	●	●	■	++	-	-	-	
Asenapine	●	●	●	n.d.	●	-	+	-	+	
Quetiapine + Li/DVP	●	●	●	●	●	+	++	+++ ^c	++	
Aripiprazole + Li/DVP	●	n.d. ^a	●	●	●	+	+	++ ^c	++	
Aripiprazole	●	n.d. ^a	●	■	●	-	+	-	+	
Aripiprazole OM	●	n.d. ^a	●	n.d.	n.d.	-	+	-	+	
Second-line treatments										
Olanzapine	●	●	●	● ^b	●	+	++	+++	++	
Risperidone LAI	●	n.d. ^a	●	n.d.	n.d.	-	+	+	++	
Risperidone LAI (adj)	●	●	●	n.d.	n.d.	+	++	+++	++	
Carbamazepine	●	●	●	●	●	++	++	++	++	
Paliperidone (>6 mg)	●	n.d. ^a	●	n.d.	●	-	+	+	++	
Lurasidone + Li/DVP	● ^c	● ^a	●	●	n.d.	+	++	++ ^c	++/-	
Ziprasidone + Li/DVP	●	n.d. ^a	●	■	■	++	++	++ ^c	+	

DVP, divalproex; LAI, long-acting injectable; Li, lithium; OM, once monthly.

●, level 1 evidence; ●, level 2 evidence; ●, level 3 evidence; ●, level 4 evidence; ■, level 1 negative evidence; ■, level 2 negative evidence; ■, level 3 negative evidence; ■, level 4 negative evidence; n.d., no data; -, limited impact on treatment selection; +, minor impact on treatment selection; ++, moderate impact on treatment selection; +++, significant impact on treatment selection.

^aDid not separate from placebo in those with index mania; no studies available in index depression.

^bDid not separate on core symptoms of depression.

^cDivalproex and carbamazepine should be used with caution in women of child bearing age.

^dTrend for superiority on the primary efficacy measure, hence the lower rating.

^eEffective in those with an index episode of depression.

Bipolar Disord. 2018 Mar 14. doi: 10.1111/bdi.12609.

Strength of evidence and treatment recommendations for acute management of bipolar II depression

Recommendation	Agent	Level of evidence	Third-line		
First-line	Quetiapine	Level 1		Agomelatine (adj)	Level 4
Second-line	Lithium	Level 2		Bupropion (adj)	Level 4
	Lamotrigine	Level 2		Divalproex	Level 4
	Bupropion (adj)	Level 2		EPA (adj)	Level 4
	ECT	(Level 3)		Fluoxetine ^a	Level 3
	Sertraline ^a	Level 2		Ketamine (IV or sublingual) (adj) ^c	Level 3
	Venlafaxine ^a	Level 2		N-acetylcysteine (adj)	Level 4
				Pramipexole (adj)	Level 3
				T3/T4 thyroid hormones (adj)	Level 4
				Tranylcypromine	Level 3
				Ziprasidone ^b	Level 3
			Not recommended	Paroxetine	2 negative

adj, adjunctive; ECT, electroconvulsive therapy; EPA, eicosapentaenoic acid.

^aFor patients with pure depression (non-mixed).

^bFor patients with depression and mixed hypomania.

Bipolar Disord. 2018 Mar 14. doi: 10.1111/bdi.12609.

Strength of evidence and treatment recommendations for maintenance treatment of bipolar II disorder

Recommendation	Agent	Evidence level
First-line	Quetiapine	Level 1
	Lithium	Level 2
	Lamotrigine	Level 2
Second-line	Venlafaxine	Level 2
Third-line	Carbamazepine	Level 3
	Divalproex	Level 3
	Escitalopram	Level 3
	Fluoxetine	Level 3
	Other antidepressants	Level 3
	Risperidone ^a	Level 4

^aPrimarily for prevention of hypomania.

Bipolar Disord. 2018 Mar 14. doi: 10.1111/bdi.12609.

Safety/tolerability concerns and risks of treatment-emergent switch with pharmacological agents indicated for use in bipolar disorder

	Safety concerns		Tolerability concerns		Risk of treatment emergent switch	
	Acute	Maintenance	Acute	Maintenance	Mania/hypomania	Depression
Lithium	+	++	+	++	-	-
Anticonvulsants						
Carbamazepine	++	++ ^a	+	++	-	-
Divalproex	-	++ ^a	+	+	-	-
Gabapentin	-	-	+	+	-	-
Oxcarbazepine	+	+	+	+	-	-
Lamotrigine	++	-	-	-	-	-
Atypical antipsychotics						
Aripiprazole	-	-	+	+	-	-
Asenapine	-	-	+	+	-	-
Cariprazine	-	-	+	-	-	-
Clozapine	++	+++	++	+++	-	-
Lurasidone	-	-	+	+	-	-
Olanzapine	+	+++	++	++	-	-
Paliperidone	-	+	+	++	-	-
Quetiapine	+	++	++	++	-	-
Risperidone	-	+	+	++	-	-
Ziprasidone	++	++	++	+	-	-

Bipolar Disord. 2018 Mar 14. doi: 10.1111/bdi.12609.

Divalproex**Evidence/Place in Bipolar Disorder Management**

- Acute Mania/Hypomania phase of Bipolar disorder: level 1 evidence; 1st line choice by CANMAT guideline
- Acute Depressive phase of Bipolar disorder: level 2 evidence; 2nd line choice by CANMAT guideline
- Maintenance therapy (prevention of any mood phase) of Bipolar disorder: 1st line (level 1) by CANMAT guideline
- US FDA approval:
 - Divalproex ER: Acute treatment of manic or mixed episodes associated with bipolar disorder, with or without psychotic features

Depakote/Depakote ER. Prescribing information. Sanofi-Aventis Limited. Revised April 2014. Bipolar Disord. 2018 Mar 14. doi: 10.1111/bdi.12609.

Key Points

- Divalproex becomes valproate.
- Valproate can be given directly, but it is highly protein bound.
- For patients with normal serum albumin, it is adequate to get the total valproate level.
- Efficacy and adverse effects are mediated by free valproate levels.
- Valproate is contraindicated in advanced cirrhosis but not necessarily in patients with hepatic inflammation.
- Divalproex is useful in the management of acute mood episode and also as maintenance therapy.
- Divalproex has an early onset of action compared to lithium, CBZ
- Recent evidence show that divalproex also has anti-suicidal effects
- Divalproex has only a slight effect on cognition
- Recent evidence show that divalproex has a little effect on spermatogenesis

World Suicide Prevention Day: from sadness to suicide, break the silence

Prof (Dr.) Ranjan Bhattacharyya, MD, DNB (Psychiatry), MNAMS
Bankura Sammilani Medical College, West Bengal

What is Suicide?

- Suicide is an attempt to solve a problem of intense emotional pain with impaired problem-solving skills (Kalafat & Underwood, 1989)

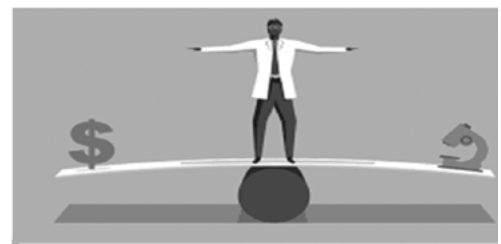
A permanent solution to a temporary problem

Outline

- Suicide
- Epidemiology
- Suicidal Risk Assessment
- Emergency Care
- Legal Duties
- Media & Ethics
- Psychological Autopsy
- Conclusion



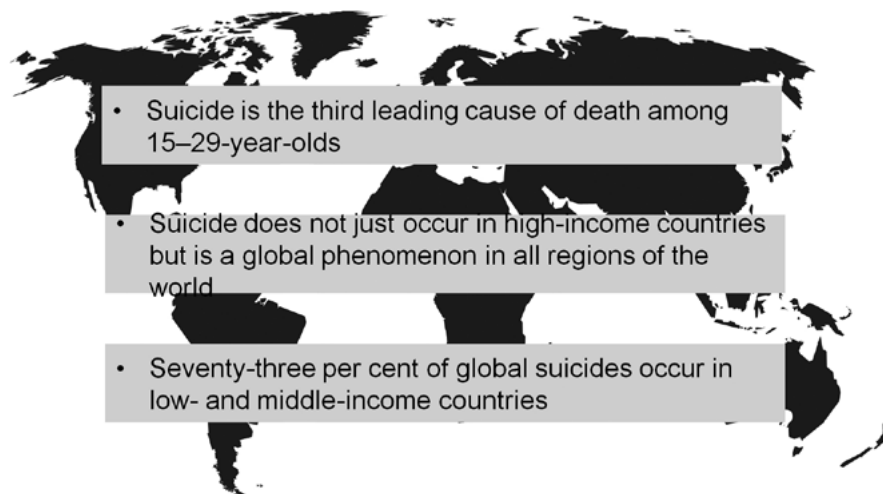
No conflicts of interest



Statistics in India & CISF reports on its Personnel

Indian Statistics - According to National Crime Records Bureau (NCRB) data, the national suicide rate stood at 12.4 per lakh, while it has been reduced to 9.87 per lakh, in 2024.

CISF Reports - The CISF claims to have taken creditable steps in combating this challenge by implementing a series of proactive measures which have led to substantial reduction in death by suicides of force members in 2024 (40% decline)

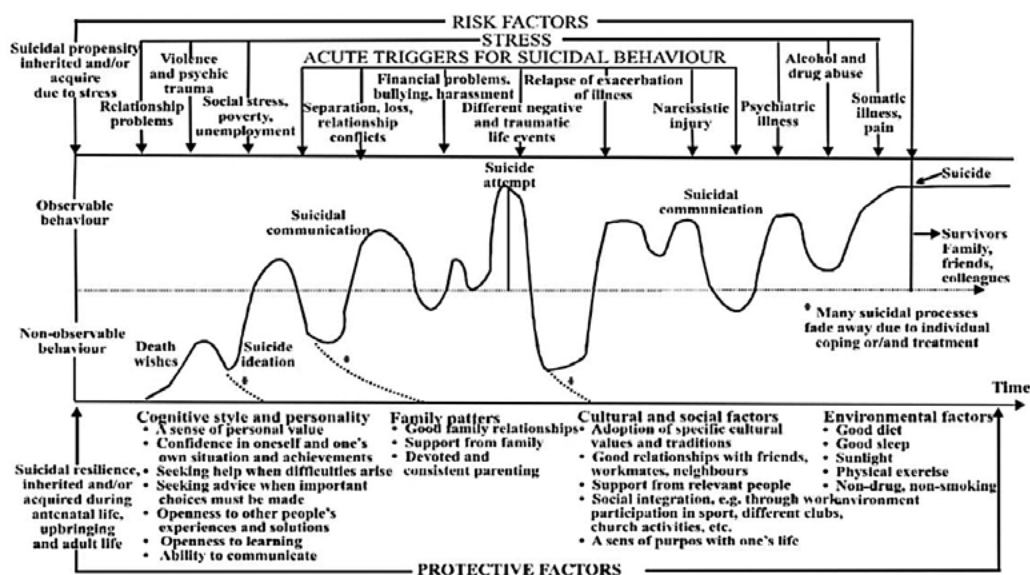


World Health Organization. Suicide Factsheet. Available at: <https://www.who.int/news-room/fact-sheets/detail/suicide>

Accidental Deaths and Suicides in India 2021



Figure 1: Stress vulnerability model and development of the suicidal process from suicidal ideation to suicide (Wasserman D, 1999).



Suicidal Risk Assessment - In the context of the psychiatric evaluation

Risk Factors For Suicide -

Previous suicide attempt (38)

Family history of suicidal behavior, especially death by suicide (39, 40)

Mental illness, especially a mood disorder or schizophrenia (41, 42)

Impulsivity (43-45) Agitation (46, 47)

Anxiety disorder (48)

Traumatic brain injury (49)

Recent major stressor or impending crisis such as marital loss, death of a loved one, financial loss, or legal involvement (50-52)

Abuse trauma history, especially in childhood (53)

Male and over age 60 (but the rate of suicide in women is growing) (50, 54)

Alcohol or opioid use and misuse, especially when comorbid with other disorders (55-58)

Attention-deficit hyperactivity disorder, especially in combination with disorder or other comorbidities (59)

Chronic pain (60)

Access to lethal means (e.g., guns, poisons, tricyclic antidepressants) (61)

Self-injurious behavior (51, 62)

Lesbian, gay, bisexual, or transgender (for suicidal behavior and ideation, not necessarily suicide) (63, 64)

Warning Signs

Changes in Behavior - Changes in behavior can include increased irritability, mood swings, or drastic shifts in routine that may indicate underlying issues or stressors affecting an individual.

Behavioral Indicators

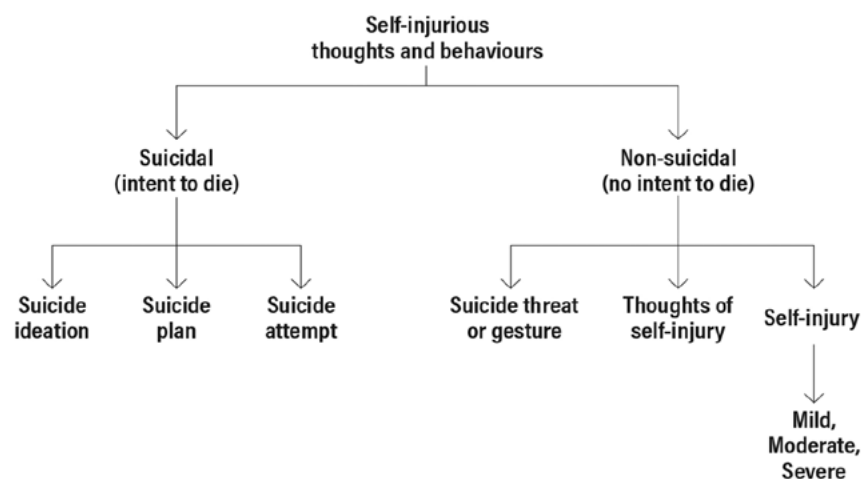
Verbal Cues - Verbal cues encompass specific language changes, such as expressions of distress or pessimism, which can be critical in identifying someone in need of support or intervention.

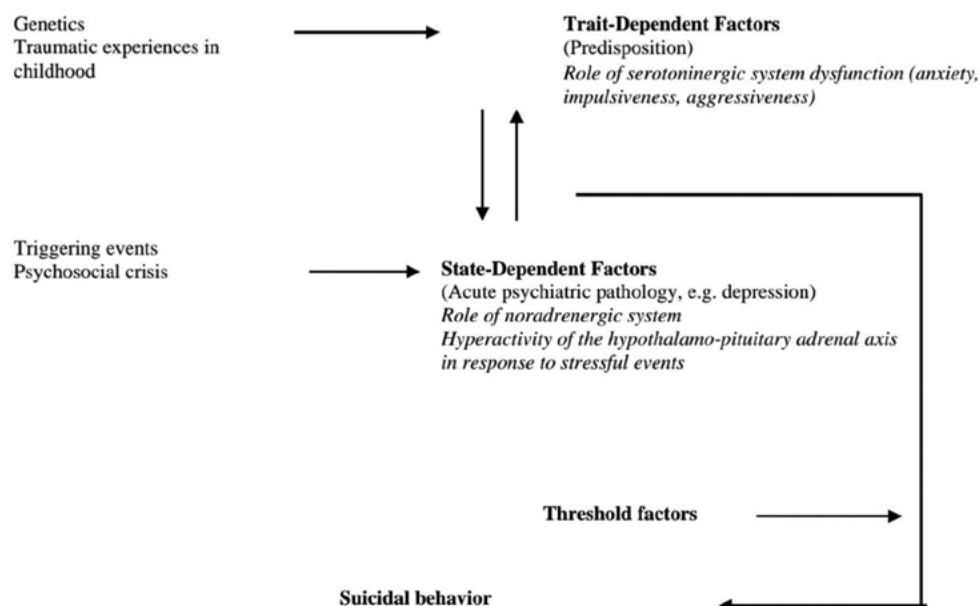
Feelings of Hopelessness - Feelings of hopelessness often manifest as a lack of motivation or enthusiasm, where individuals may express doubts about their future or capabilities

Emotional and Psychological Signs

Withdrawal from Activities - Withdrawal from activities involves a noticeable decline in participation in previously enjoyed hobbies or social interactions, which can signify emotional distress or isolation

Identifying Self injurious behavior & Suicide





Van Heeringen model (2003) of interaction between trait- and state-dependent factors in suicidal behavior and the role of neurobiological factors

Response on the Risk

3Cs Connection
Collaboration
Choice

Role of Professionals

Cognitive Behavioural Therapy (CBT): Cognitive behavioural therapy (CBT) is a popular and effective approach that empowers individuals to recognise and transform harmful thought patterns.

Dialectical Behaviour Therapy (DBT): Originally developed for individuals with borderline personality disorder, DBT is effective in reducing suicidal behaviour.

Psychodynamic Therapy: This approach explores the underlying emotional and psychological issues that contribute to mental health struggles.

Group Therapy: By promoting awareness, these campaigns can reduce stigma and motivate people to seek help.

Role of Social Media in Suicide

Hines provides mental health guidance, aiming to help people find hope. Importantly for the present study, he is also a significant content creator on Instagram (@kevinhinesstory) with more than 80,000 followers. Illustrative examples of some of his salient claims provided in his posts allow for a first impression:

“Ending your life is never a solution,”

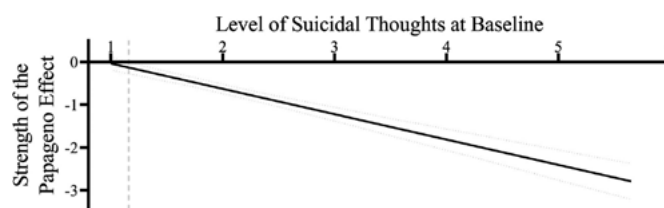
“My purpose: Sharing my story of surviving death,”

“You can always survive the pain”

“Always be there tomorrow.”



Papageno effect - The Papageno effect is the effect that mass media can have by presenting non-suicide alternatives to crises. It is named after a lovelorn character, Papageno, from the 18th-century opera *The Magic Flute*; he was contemplating suicide until other characters showed him a different way to resolve his problems.

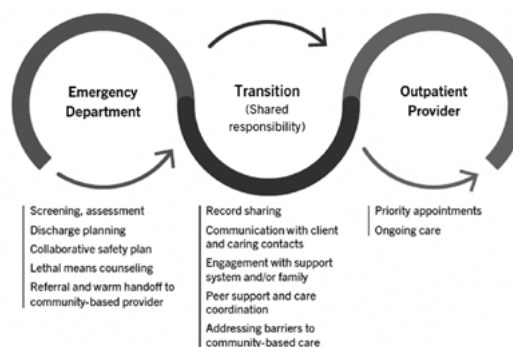


Biological markers and suicide

Biological markers	Type of analysis	Results
Serotonin (5-HT)	Post mortem	↓ 5-HT and 5-HIAA catabolite (raphe region, hypothalamus)
		↓ 5-HIAA in prefrontal cortex (inconsistent)
		Raphe region: ↑ 5 HT1A receptors; no modification 5-HT transporter sites
		Nerve terminals:
		↑ 5 HT1A and 5 HT2A post synaptic receptors in frontal cortex (controversial)
		↓ 5-HT transporter sites (ventromedian prefrontal cortex)

Emergency Care

Recommendations for Emergency Departments



Legal Duties

Sections of the Indian Penal Code (IPC) relating to suicide

Section 115 of the Mental Health Act 2017	Section 306, IPC	Section 309 of IPC
Suicidal people are presumed to be suffering from mental health problems and stress, which grants them immunity from punishment.	Abetment to suicide Persons assisting suicide may also be punished with imprisonment of up to 'up to ten years as a prison sentence.	Attempt to commit suicide Persons who attempt suicide or take steps to commit such act shall be punished with simple imprisonment for a term which may range to one year or with fine or both.

Mental Healthcare Act

2017 (MHCA), has several practical implications concerning suicide and attempted suicide in India

Decriminalization Of Attempted Suicide

- The MHCA effectively decriminalizes attempted suicide by removing Section 309 of the Indian Penal Code (IPC), — Which previously subjected individuals to punishment for attempting suicide.
- This legal change signifies a shift towards viewing suicide attempts as a mental health issue rather than a criminal act.
- As a result, individuals who attempt suicide are not liable for punishment under criminal law.

MHCA Recommendations

The MHCA emphasizes the right to access mental healthcare services for all individuals, including those at risk of suicide.



It mandates the establishment of mental health facilities and services at various levels of healthcare delivery:



Ensuring that individuals in distress have access to timely and appropriate mental health support



Envisioned the protection of rights and dignity.

Media & Ethics



Guideline for Ethically Reporting on Suicide

- AFSP advises media and others messaging about suicide to follow these ethical reporting guidelines as a matter of public health — To safeguard vulnerable people in society who are at risk for suicide.
- These guidelines are based on substantive research that has shown that — Ethical and supportive coverage about suicide can positively deepen the public's knowledge related to suicide and mitigate the occurrence of additional suicides (known as contagion,) which unethical reporting can exacerbate.
- Do not refer to a suicide attempt as “successful,” “unsuccessful” or as a “failed attempt,” and do not use the word “committed.”
- Instead, use “attempted suicide,” “made an attempt,” “died by suicide” or “took his/her life.”
- Use terminology that is not moralistic and better aligns suicide with other health conditions
- Decreases negative attitudes of mental health conditions.

Sensatizing

- Inform the audience without sensationalizing the suicide.
- This means excluding to the degree possible images or graphic depictions of a suicide death, such as details, notes, and location of death.
- Instead, focus on the lived life, any mental health or general struggles they had been public about, as well as positive aspects of the individual.
- If the story is centered on firearms or bridges, care should be taken to not describe how death occurs through these methods.

Cause

- Research shows that suicide is complex.
- Avoid reporting that a suicide death was caused by a single event.
- As research shows, no one takes their life for a single reason, but rather a combination of factors.

- Reporting one “cause” leaves the public with an overly simplistic and misleading understanding of suicide.
- Promotes the myth that suicide is the direct result of circumstances and is not preventable.

Prominence

- Do not feature suicide stories on front pages of newspapers or main landing pages of online media.
- Do not mention the word “suicide” or method in the headline or in the open in paragraphs of stories.
- This applies to broadcast.
- Prominent placement of the word suicide and method has been shown to lead to contagion (copycat suicides).

Magnitude

- Do not refer to suicide as an “epidemic,” or “skyrocketing” as this language has shown to cause contagion.
- When referencing suicide as a “leading cause of death,” include the most recent rates to ground people in facts.
- Instead, refer to suicide as a solvable and preventable public health issue.

Hope and Help

- Research tells us that showing that help is available and recovery is possible can not only lessen the risk of contagion, but also encourage people to seek help.
- Include hopeful messages for the public that support and treatment — including therapy and medications — are available for mental health conditions.
- Always provide helpline information.

Celebrity

- Report on celebrity or people of note suicides with caution.
- Glamorizing suicide deaths may inadvertently glorify suicidal behavior and present suicide as a normalized solution to pain or distress; frame suicide as preventable.
- When possible, include the celebrity’s struggles, health, or mental health experiences as a way to emphasize the fact that all humans have mental health and can struggle.
- Avoid speculation when the cause of death is unknown.

Social Media

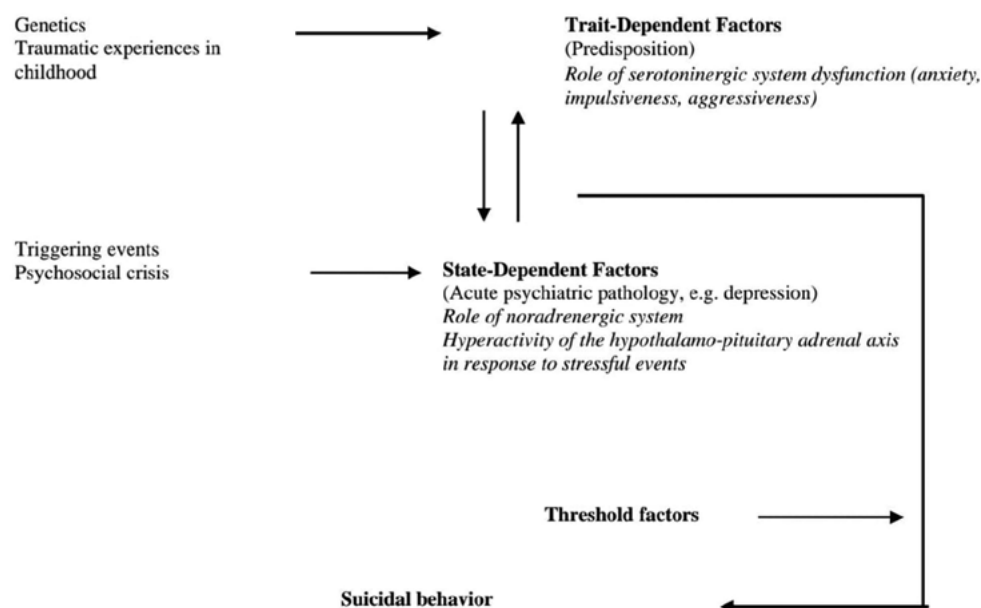
- Providing content warnings
- Not sharing news stories that do not adhere to ethical reporting guidelines
- Monitoring replies



Psychological Autopsy

Introduction

- A psychological autopsy (PA) is the reconstruction of events leading to death; ascertainment of the circumstances of the death, including suicidal intent; and an in-depth exploration of other significant risk factors for suicide.
- Psychological autopsies were originally designed to investigate, clarify, and help police enquiries into the mode of death in equivocal fatalities.
- However, in recent years, they have become more often utilized as a research tool for investigating risk factors for completed suicides.



Van Heeringen model (2003) of interaction between trait- and state-dependent factors in suicidal behavior and the role of neurobiological factors

Limitations				
Lack of standardized techniques or methods of PA	Informant bias	Lack of interviewer training	Recall bias	Issues with control selection

The role of psychological autopsy in investigating a case of atypical suicide in schizophrenia: A case report



Suicide Myths & Facts

1. Suicide happens without warning.	
TRUE	FALSE

1. Suicide happens without warning.
FALSE
Studies reveal that the suicidal person will often give many clues and warnings regarding his/her suicidal intentions.

2. Once a person decides to complete suicide, there is nothing anyone can do to stop them.	
TRUE	FALSE

2. Once a person decides to complete suicide, there is nothing anyone can do to stop them.
FALSE
Suicide is the most preventable kind of death, and almost any positive action may save a life.

3. Confronting a person about suicide will only make them angry and increase the risk of suicide.	
TRUE	FALSE

3. Confronting a person about suicide will only make them angry and increase the risk of suicide.
FALSE
Asking someone directly about suicidal intent can lower anxiety, open up communication and lower the risk of an impulsive act.

4. There is a difference in suicidal behavior between boys and girls.	
TRUE	FALSE

4. There is a difference in suicidal behavior between boys and girls.
TRUE
There is a difference, with boys making 3 times more completions than girls, and girls making 3 times more attempts than boys.

5. Talking about suicide can plant the idea in the minds of at-risk youth.	
TRUE	FALSE

5. Talking about suicide can plant the idea in the minds of at-risk youth.
FALSE
Asking about suicide does not increase the risk of an individual attempting suicide. In fact, asking the person about suicide can be a big relief and potentially save the person's life.

6. Anyone can help a suicidal student.	
TRUE	FALSE

6. Anyone can help a suicidal student.
TRUE
Anyone, not just a mental health professional can help by engaging the student in accessing the appropriate resources. Teachers can often be the first line of defense in helping an at-risk student.

Suicide prevention needs more sunlight, Candor & unashamed conversation

Neurobiology and Mental Health Benefits of Yoga

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The Role of Yoga in Stress Reduction

Physiological Effects of Yoga on Stress	Psychological Benefits of Stress Relief through Yoga		
Yoga helps reduce cortisol levels and lowers heart rate, promoting relaxation and better stress management.	Regular yoga practice enhances emotional resilience and improves overall mood by reducing feelings of tension and overwhelm.		

Yoga and Anxiety Management

Techniques to Alleviate Anxiety with Yoga	Evidence from Studies on Yoga's Impact on Anxiety
Breathing exercises, gentle stretches, and focused poses help calm the nervous system and reduce anxiety symptoms.	Research shows consistent yoga practice significantly decreases anxiety levels and enhances feelings of calmness.

Mindfulness and Meditation in Yoga Practice

Developing Mindfulness through Yoga	Meditation Practices Integrated in Yoga
Yoga encourages present-moment awareness, helping individuals become more mindful of their thoughts and emotions.	Meditative techniques within yoga foster deep relaxation and mental clarity, supporting emotional balance and stress reduction.

Designing a Yoga Routine for Mental Health

 	Key Components of a Mental Health-Focused Practice	Tips for Consistency and Motivation
	Includes mindful movement, breath awareness, gentle poses, and relaxation to promote emotional balance.	Set realistic goals, create a dedicated space, track progress, and celebrate small achievements regularly.

Yoga Breathing Techniques (Pranayama) for Calmness

Types of Breathing Exercises Used in Yoga	Benefits of Controlled Breathing on Mental Clarity
Examples include diaphragmatic breathing, alternate nostril breathing, and Ujjayi breath for relaxation.	Improves focus, reduces anxiety, and enhances overall emotional regulation through oxygen regulation.

Overcoming Barriers to Practicing Yoga

Common Challenges and Misconceptions	Strategies to Make Yoga Accessible for Everyone
Includes perceived difficulty, lack of time, and beliefs that yoga is only physical or religious.	Offer modifications, use online resources, create beginner- friendly sessions, and promote inclusivity.

Neurobiological Effects of Yoga

Impact on Brain Structure

Neuroplasticity Enhancement	Changes in Grey Matter Volume
Regular yoga supports brain plasticity, promoting the brain's ability to adapt and reorganize neural pathways.	Yoga practice is linked to increased grey matter volume in regions related to memory, emotional regulation, and body awareness.



Neurochemical Modulation



Hormonal Balance Adjustments	Regulation of Neurotransmitters
Practicing yoga helps balance stress hormones like cortisol and promotes the release of endorphins for well-being.	Yoga influences levels of serotonin, dopamine, and GABA, which improve mood and reduce anxiety.

Stress Response and Cortisol Levels

Reduction of Cortisol Secretion	Impact on Hypothalamic-Pituitary-Adrenal Axis
Yoga practice lowers cortisol, the body's primary stress hormone, leading to reduced stress and anxiety.	Yoga regulates the HPA axis, improving the body's overall stress response and resilience to stressors.

Autonomic Nervous System Regulation

Parasympathetic Nervous System Activation	Sympathetic Nervous System Inhibition
Yoga stimulates the parasympathetic system, promoting relaxation, digestion, and recovery.	Yoga inhibits the sympathetic nervous system, reducing the fight- or- flight response and calming the body.

Mental Health Benefits of Yoga

Anxiety and Depression Reduction

Alleviation of Anxiety Symptoms	Improvement in Depressive States
Yoga helps reduce physical tension and mental stress that contribute to anxiety symptoms.	Regular yoga practice can elevate mood and decrease feelings of sadness and hopelessness.

Enhancement of Emotional Regulation

Increased Mindfulness and Awareness	Better Coping Strategies Development	
Yoga encourages present-moment focus, helping individuals become more aware of their thoughts and feelings.	Through breath control and meditation, yoga teaches effective ways to manage stress and emotional challenges.	

Cognitive Function Improvement

Enhanced Attention and Concentration	Memory and Executive Function Benefits
Practicing yoga improves mental clarity and the ability to maintain focus on tasks.	Yoga supports brain functions that involve memory retention, decision-making, and problem-solving skills.

Yoga and Sleep Improvement

Techniques for Better Sleep Quality	Role of Yoga Nidra in Sleep Disorders
Gentle yoga stretches and relaxation techniques prepare the body and mind for restful and deeper sleep.	Yoga Nidra, a guided meditation practice, helps reduce insomnia and enhances sleep by promoting deep mental relaxation.

Integrating Yoga into Daily Routine

Creating a Consistent Practice Schedule	Simple Yoga Poses for Beginners
Establishing a regular time daily helps build habit and promotes steady mental health improvements.	Basic poses like Child's Pose and Cat-Cow improve relaxation and ease beginners into yoga practice.

Yoga for Depression Support

	Combining Yoga with Therapy	Specific Postures to Uplift Mood
	Using yoga alongside counseling enhances emotional healing and supports overall mental wellness.	Poses such as Warrior II and Bridge stimulate energy and can reduce symptoms of depression.

Community and Group Yoga Benefits

Social Support Through Group Sessions	Shared Motivation and Accountability
Group classes provide a sense of belonging and emotional encouragement for participants.	Practicing yoga in groups increases commitment and helps maintain a regular practice.

Promotion of Overall Well-being



Improved Sleep Quality

Yoga helps relax the nervous system, leading to more restful and restorative sleep patterns.

Increased Sense of Calm and Relaxation

The combination of movement, breath, and meditation in yoga fosters deep relaxation and inner peace.

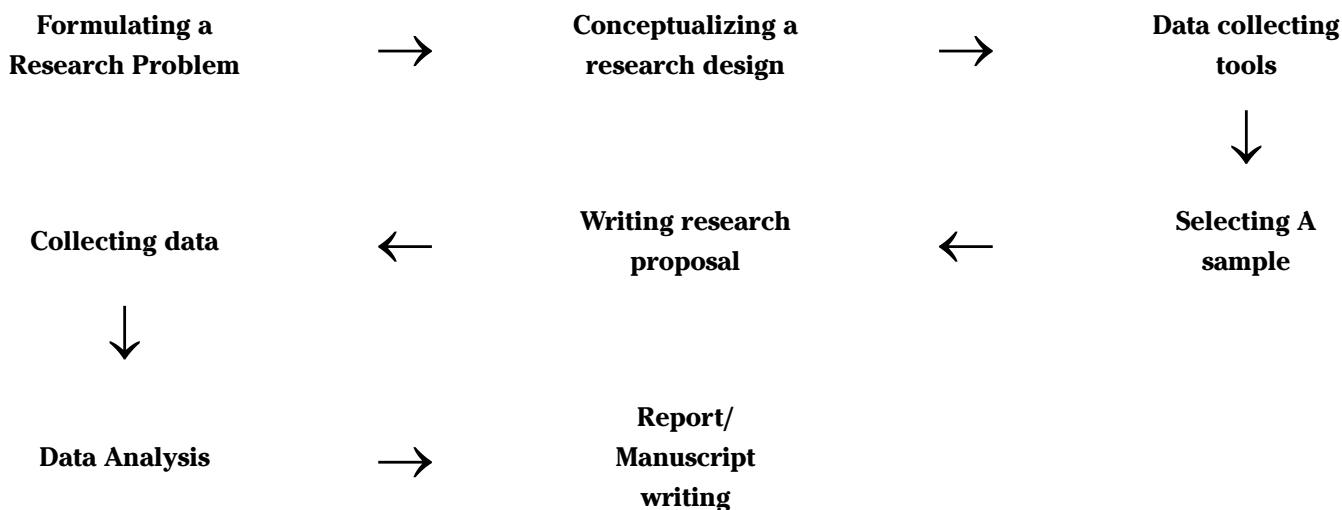
From Concept to Execution: A Practical Guide to Research Paper Writing

Dr. Samrat Singh Bhandari, Consultant Psychiatrist

Why do so many good studies fail to get published?

✓ Important Research Question → ✓ Well-designed study → ✓ Useful Data
 → × Unclear Scientific message → × Poorly organized Manuscript → Journal Rejection

Life Cycle of Research



Before You Write a Single Sentence...

Ask yourself only ONE question. • “What is the ONE message I want readers to remember?”

Not • “What all did I do?” Not • “What all statistics do I have?”

But • “What is the single take-home message of my research?”

Common Vs effective approach

Common Approach	More Effective Approach
Starts with introduction	Starts with Tables and Figures: Key findings
Writes section by section	Plan the scientific story
Edit while drafting	Complete a rough draft and revise

The Argument Matrix → Think Before You Write

Why IMRAD Alone is Not Enough?

Introduction	Review the analysed data
Why was the study needed?	↓
↓	Select 3–5 key tables and figures
Methods	↓
What was done?	Write brief statements describing the main findings
↓	↓
Results	Formulate conclusions in relation to the study objectives
What was found?	↓
↓	Identify 2–3 central messages or ideas
Discussion	↓
What do the findings mean?	Build the Argument Matrix
	↓
	Develop the high-level IMRAD outline
	↓
	Expand it into the manuscript

Building the Argument Matrix

Scientific Message	Introduction	Methods	Results	Discussion
Main finding 1	Why important?	How measured?	What found?	What does it mean?
Main finding 2	Why important?	How measured?	What found?	Interpretation
Main finding 3	Why important?	How measured?	What found?	Clinical implication

Idea	Introduction	Methods	Results	Discussion	Limitations	Conclusion and recommendations
Idea #1	→	→	→	→	→	→
Idea#2	→	→	→	→	→	→
Idea#3	→	→	→	→	→	→

*INTRODUCTION IS NOT A REVIEW OF LITERATURE

Idea	Introduction	Methods	Results	Discussion	Limitations	Conclusion and recommendations
Idea #1	↓	↓	↓	↓	↓	↓
Idea#2	↓	↓	↓	↓	↓	↓
Idea#3	↓	↓	↓	↓	↓	↓

From Key Results to Central Ideas: A Hypothetical Schizophrenia Study

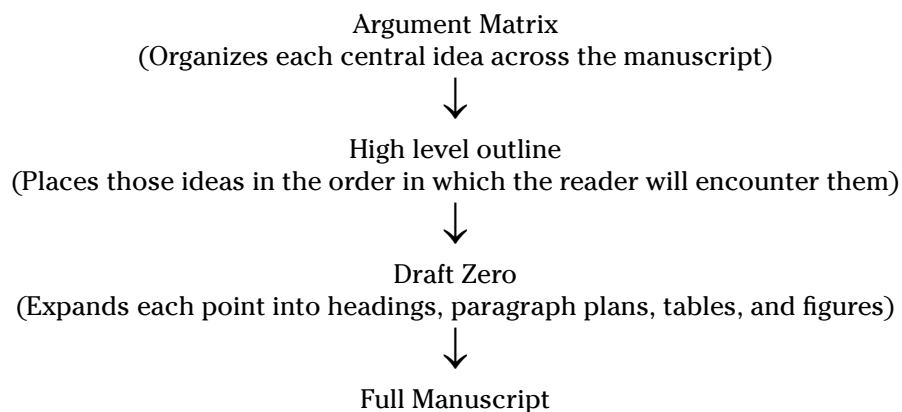
Title of the study: Medication Adherence and Its Correlates among Patients with Schizophrenia

Key table	Main finding
Table 1: Insight and adherence	Poor insight was associated with greater medication non-adherence
Table 2: Family support and adherence	Greater family support was associated with better adherence
Table 3: Substance use and adherence	Current substance use was associated with greater non-adherence

Direction of writing the high-level outline

Central Idea	Introduction	Method	Result	Conclusion
Insight & Adherence	Poor insight decrease need for treatment	Validated Scales and statistics	Poor insight associated with non-adherence	Routine assessment of insight may help identify adherence risk
Family support & Adherence	Families support treatment & follow-up	Measure perceived family support	Greater support associated with better adherence	Include families in adherence interventions
Substance use& Adherence	Comorbid substance use may reduce adherence	structured clinical questionnaire	Substance use may impair judgement, increase impulsivity and disrupt regular follow-up	Routine substance-use screening and integrated management may help improve adherence

From Argument Matrix to Full manuscript



Writing the introduction

Sections	Subsections	Content	Objective	Practical remarks
Introduction	1st Paragraph	General (Global) Background on the subject (restrict to what is relevant to RQ)	Why the research is important to study from a global perspective	Start after method and results are complete Avoid complete literature review Limit to what is necessary and relevant to RQ
	2nd Paragraph	National and Regional perspective	Zoom in to the points applies to national/regional context	
	3rd Paragraph	Emergence of the research question leading to objectives	Justify the research question	

Writing the Method Section

Sections	Subsections	Content	Objective	Practical remarks
Methods	1st Paragraph	Study design, study setting, and time period	To tell what kind of study, where and when done	keep it simple and specific
	2nd Paragraph	Participants, sampling method, inclusion and exclusion criteria	To show who all were studied and how the sample was obtained	Include only details needed to judge relevance and validity
	3rd Paragraph	Variables and tools/ instruments used for measurement	Explain what was measured and how it was measured	Mention validated scales, operational definitions, and important clinical variables
	4th Para	Data collection procedure	Describe how the information was collected	Explain but keep concise
	5th Para	Statistical analysis	Explain how the data were analyzed to answer the objectives	Match every analysis to an objective; mention tests, regression methods, confidence intervals, significance level
	Last Para	Ethical considerations	Show that the study was conducted ethically and responsibly	Mention ethics approval (give certificate no.), consent, and confidentiality in brief

Statistical Analysis: Incomplete vs Adequate Reporting

Statistical analysis: Data were analysed using SPSS version XX. Categorical variables were summarized as frequencies and percentages, while continuous variables were presented as mean $\pm$ SD or median (IQR), according to distribution. Normality was assessed using the Shapiro–Wilk test. Associations of medication adherence with insight, caregiver support, and substance use were examined using the chi-square test or Fisher’s exact test, as appropriate. Differences in continuous variables between adherent and non-adherent groups were analyzed using the independent-samples t test or Mann–Whitney U test. Variables considered clinically relevant and those associated with adherence in bivariate analysis were entered into a multivariable logistic regression model. Adjusted odds ratios with 95% confidence intervals were reported. A two-sided p value <0.05 was considered statistically significant.

Statistical Analysis: Incomplete vs Adequate Reporting

Frequency, percentage, mean, independent t-test and chi-square. Significance level $p < 0.05$.

Writing the Result section

Main objective: ✓ Describe the result of the analysis

Practical remarks

✓ Write the results after Methods ✓ Report the Estimate, not only the p Value

✗ Please avoid giving the interpretations or reasons for findings in this section

Avoid: Medication adherence was significantly associated with insight ($p = 0.01$).

Better: Patients with poor insight were more likely to be non-adherent than those with good insight, 62% versus 38%; $\chi^2 = 6.72$, $p = 0.01$. Best, when regression is used, Poor insight was independently associated with non-adherence (adjusted OR = 2.45, 95% CI 1.32–4.56, $p = 0.004$).

Writing the discussion section

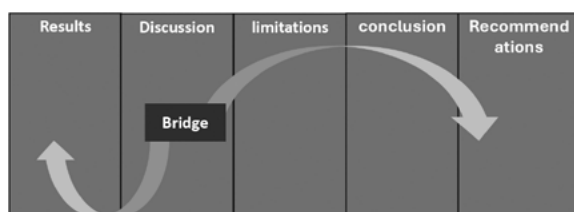
Summary of the result (without interpretation) • Summarize the information generated; Introduce key points that will be interpreted in the paragraphs of discussion)	First Paragraph
Points of discussion (1-4 different ones) in separate Paragraphs • Organize arguments around key results to place perspective and reach an interpretation • Give reasons why the findings are similar or different from previous studies • Include Limitations before conclusion/recommendations	Subsequent Paragraphs

Last two paragraphs of discussion Conclusions

- Conclusions reached on the basis of result and justified interpretation
- Gives answers to the research question(s) as much as the study can

Recommendations

- Propose what needs to be done in terms of actions and further research



Writing the conclusion section

Answer the objectives



Clinical Relevance



Future Direction

Do not overstate your findings.

Leave the reader with a clear message.

Before Finalizing the Manuscript: Use a Reporting Guideline Checklist



**Enhancing the QUALity and
Transparency Of health Research**

Study design	Reporting guideline
Observational study	STROBE
Case report	CARE
Systematic review/meta-analysis	PRISMA
Qualitative study	COREQ

Points which helps and avoids rejection

- Select the right journal and confirm scope fit.
- Follow the journal's author instructions.
- Title should be informative, searchable, interesting
- Abstract- the most important 250 words (write at the last)
- Keep the research question, methods, results, and conclusions aligned.
- Report estimates, effect sizes, confidence intervals, and exact p values.
- Check that the manuscript addresses every item in the relevant EQUATOR reporting checklist.
- Verify compliance with the relevant EQUATOR checklist.

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Conclusion:

- Begin with the analysed findings
- Select only the tables and figures that answer the objectives.
- Allow the central messages to emerge from findings and conclusions.
- Use the Argument Matrix to connect ideas across IMRAD.
- Make every conclusion and recommendation traceable to the evidence.

A Case Series showing effectivity of non directive and directive Art Therapy Sessions in reducing restlessness among an ADHD cohort

Dr. Suddhendu Chakraborty, Consultant Psychiatrist and independent researcher, Institute of Psychological and Educational Research and Monovikash Kendra, Kolkata

Keyword: Art Therapy, Attention Deficit, Hyperactivity

Introduction

Visual Art Therapy is a technique where art is used as a tool of communication and intervention to alleviate different psychological morbidities. According to the American Art Therapy Association, the creative process of art making is a form of non verbal communication of thoughts and feelings that can be used to heal. It is a specific form of Expressive Behaviour Therapy, where there is scope for growth, self understanding and emotional reparation. It is a hybrid discipline transgressing both Art and Psychology. The forerunners in the field who defines the Classical period (1940-1970) of Art Therapy are Margaret Naumberg, Edith Kramer, Hanna Kwiatkowska and Elinor Ulman. Naumberg has sometimes be referred as the 'Mother of Art Therapy'. While the initial approach were largely psychodynamic, the subsequent approaches become more person centric. A survey by Elkins and Stovall showed how Art therapy can be admixed as a tool with conventional Cognitive Behaviour Therapy (2000). This further enhances the scope of Art Therapy as an intervention. Studies have showed effectiveness of art therapy in dealing with psychopathological morbidities in children. Waller (2006) emphasises how art therapy creates a positive engagement and a safe environment between the therapist and the child. The article also shows how the technique uses the principle of sublimation in dissipating the internal stress. In this study, I have tried to investigate whether art therapy does have a quantitative and qualitative effect of children suffering from Attention Deficit Hyperkinetic Disorder (ADHD). A study conducted by Henley (1998) used a weekly two hour session addressed to children suffering from peer relations, school and family relations which explored three domains, namely free play, group discussion, and expressive art. The results showed significant changes in most of the components. Another approach by Smithemann et al (1996) used Mandala as an active centering device while addressing a cohort of Attention Deficit Disorder (ADD) and ADHD. The results were assessed using the Formal Elements Art Therapy Scale (FEATS) which showed a creative growth and increased attentional activities in the addressed children. Even attempts were made to club Visual Art Therapy with other forms of Expressive Behaviour Therapy like Music and Dance Movement while addressing issues of ADHD. Martinez et al (2024) successfully established effectivity of Art Therapy and Music Therapy in dealing with cases of Autism Spectrum Disorder (ASD) and ADHD while establishing a metaanalysis among existing article evidences in this field. Nevertheless, studies are limited and scarce while dealing approaches in Eastern India. In India, there is a typical indigenous pattern of symptomatology in children suffering from ADHD and little documented evidence as to exploring the scope of Art Therapy in dealing with them. This significant research gap prompted the present study.

Materials and methods

Children suffering from ADHD were screened for this study. The children with age not exceeding 9 years of either gender were chosen. Most of the child enrolled in the study came from socially backward class with limited economic stability, limited access to basic mental health care and a poor family support. The children were screened by ADHD with Conners Teacher Rating Scale (CTRS) investigating the domains of Hyperactivity-Impulsivity, Perfectionism, Inattention and cognitive problems, Social problems, oppositionality and anxiety. (1998) The cohort was chosen by snow ball technique of sampling. 16 children who fulfilled the inclusion criteria were chosen for the study.

Children having moderate to severe intellectual impairment were screened out using Wechsler abbreviated scale of Intelligence (WASI II). Also children having other concurrent psychological illness, medical illness and those who failed to come for a follow up visit were excluded. The children selected were schoolgoing and were mostly residing in an urban locality in South Kolkata, Bengal. The cohort of 16 children initially showed elevated scores in the domain of Hyperactivity-impulsivity (M90), inattentiveness (M88), Learning problems (M 76), Peer Relations (M 67). The children were given both directive and nondirective Art Therapy. Instructions were not given in 'non directive' and vice versa. The sessions were for 90 minutes once weekly. The assessors were two trained Psychologists having a clinical experience in the field for at least 3 years. The respondent teachers were the school teachers of the respective schools. An informed consent and ethical committee clearance were obtained. CTRS rating was done on Day 01, Day 30, Day 90 and Day 180. The results were statistically compared. Logistics included drawing sheets, crayons, pencils, water colour and erasers.

Results

22 Art Therapy sessions included both directive and nondirective art therapy that included Scribbling, messmaking, Self portrayal of events, Five point grounding and Mandala. The age group of the cohort ranged from 6 to 9 years. There were 7 girls, and 9 boys in the cohort. In the initial First recording of the repeat assessment, 10 out of 16 children showed significant changes in the indicators constituting Factor I of CTRS namely in the domains of restlessness, difficulty in waiting, excitability. The girls outnumbered the boys in 6 to 4 in the improvement. The other change was noticed in the Factor 4 domain dealing with social interactions. There were significant changes noticed in the social skills and making of friends. 9 out of 16 showed significant change, where the gender ratio of boy to girl came out to be 3 to 6. The changes continued in the second assessment (Day 90) where 12 out of 16 showed significant change in the Factor 1 (Hyperactivity Impulsivity domain) and Factor 4 (Social Problem domain). Values were decreased in Factor 6 domain with reduced anxiety, shyness, sensitivity in all 16 children. In the final follow up on Day 180, after completion of 22 sessions, the results showed a significant change in Hyperactivity impulsivity domain (M54), Inattentiveness (M50), Learning domain (M70) and Peer relation (M51).

Discussion

A case study by Habib et al (2015) showed 25 sessions of art therapy in a 6 year old child suffering from ADHD, the indicators and clinical symptoms significantly decreased. In this study, being a group cohort had an advantage of additional group therapeutic potential and the results after 22 sessions went at par with the evidence. A similar finding was recorded by Ayaz (2016) in a 6 year 5 month old girl suffering from ADHD where after 25 sessions, the outcome was noticeable. The present study also discovered an emergence of leadership ability in one boy aged 9 years in the cohort. 90 percent of the art techniques used were drawing forms, 20 percent collage, 85 percent Mandala and 77 percent scribbling. Bartoe (2014) showed the potential of Art therapy in treating ADHD as an adjunct. The present study acts in unison with the findings. A small sample size and restriction of the cohort to south Calcutta region remains a limitation of the study and draws scope for further research. Zhang et al (2024) showed the potential of using AI techniques in Art Therapy in treatment of ADHD in children. These present further insights into future research.

Conclusion

The study showed the potential of different forms of Art Therapy and its application in the treatment of ADHD. However involvement of biomarkers in the study could have made the study more sound and interesting. This warrants our future direction of study.

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Transcranial Magnetic Stimulation

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HISTORY OF ELECTRICAL STIMULATION

- The ancient Greeks and Romans knew of the shocking powers of the Nile catfish and electric ray (Finger 2000). Galen and Scribonius Largus in Rome used electric rays to treat headaches and various other disorders.
- By the early eighteenth century the leading scientists still did not know what substance was flowing through nerves (Finger 2000). Serious thinkers speculated about spirits, special fluids, and even vibrations.
- It was Luigi Galvani who, in a series of experiments published in 1791, established that electricity flows through nerves. Using rudimentary batteries, he showed that an exposed nerve could be activated with electricity and produce a seemingly natural muscle contraction.
- Aldini, Galvani's devoted nephew, conducted some of the most unusual research and showed that human muscles also moved when electrically stimulated. He applied electricity to decapitated heads at the base of a guillotine and was able to induce jaw movements, grimaces, and eye openings. ^[1]

NEUROMODULATION/ BRAIN STIMULATION TECHNIQUES

- Electrical currents or magnetic fields used to alter neuronal firing
- Tools apply either electrical or magnetic fields transcranially or involve surgical implantation of electrodes.
- **Transcranial techniques:** Cranial electrical stimulation, electroconvulsive therapy, transcranial direct current stimulation, transcranial magnetic stimulation, magnetic seizure therapy.
- **Surgical techniques:** Cortical brain stimulation, deep brain stimulation, vagus nerve stimulation^[2].

TRANSCRANIAL MAGNETIC STIMULATION

- The first known examples of anything close to modern TMS occurred in the years 1910 and 1911 (George and Belmaker 2007).
- At that time, several researchers used large electromagnets the size of suitcases (or even trunks) to induce phosphenes: the sensation of seeing light without actual light passing into the eye.
- It is unclear from that work whether the magnets stimulated the retina or the occipital cortex. These early experiments were fascinating, but of little practical value. After that, interest in the field waned for many decades.



- However, it was not until 1985 that the modern era of TMS started. That year, Anthony Baker in Sheffield, England, described the use of a noninvasive magnetic device resembling modern TMS instruments (Barker et al. 1985). The device was slow to recharge and quick to overheat, but it was a start.^[2,3]

MAGNETIC FIELD

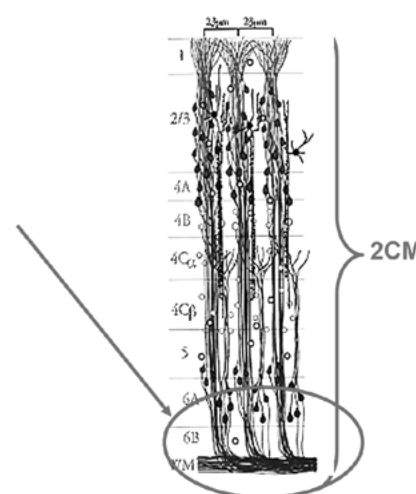
The common unit of magnetic field is the tesla, denoted “T,” equal to N/(A·m) (Force/Current-Distance); one T is about 20,000 times the earth’s magnetic field. Technically, TMS devices produce a fairly powerful magnetic field, but only very briefly. Early TMS devices emitted only a single, brief pulse. Modern devices can generate a rapid succession of pulses called repetitive TMS, or rTMS. These devices are used for behavioral treatments and can discharge on and off for several minutes.

TABLE 6-1. Examples of common magnetic force in tesla

Example	Force in tesla
Earth’s magnetic field	0.00005
Refrigerator magnet	0.1
12 V battery nail electromagnet	0.5
MRI	3.0
TMS	1.5–3.0
World’s strongest magnet (Florida State University)	45.0

DEFINITION OF TMS

- Application of a rapidly changing magnetic field to the superficial layers of the cerebral cortex, which locally induces small electric currents; an electric stimulation without an electrode.
- Also called “electrodeless electrical stimulation”.
- Current flows through coil -generates magnetic field (AMPERES LAW)
- Alternating magnetic field-generates an electric current in the brain(FARADAYS LAW)
- Electric current causes depolarization of cortical neurons specially in underlying regions
- Electrical energy is converted to magnetic fields ,which are then converted back into electrical currents in the brain.
- The electromagnet offers an alternative way to create a changing magnetic field and thus induce an electrical pulse. In this situation the electricity is used to turn the magnet on and off . This produces a pulsating magnetic field. It is this changing field that can induce an electrical current.
- Flow of induced current depolarizes nerve membranes, especially at the junction of gray and white matter, where axons bend
- Strictly speaking, the stimulation is not ‘magnetic’ but electric. Magnetic field is used only to pass through the resistance from the skull



MECHANISM OF ACTION

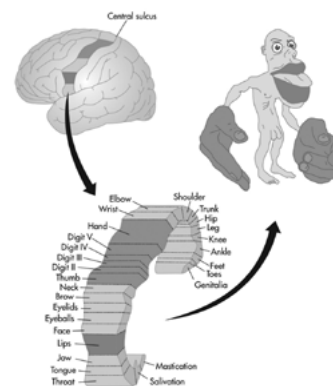
- Changes in cortical excitability
- Changes in cortical blood flow
- Changes in neurotransmitters
- Metabolic and neuroendocrine effects
- Neuroplasticity and neurogenesis

DIFFERENT COILS

- Single coil- diffuse magnetic field
- Double coil or figure of eight coil- focal high intensity
- Motor threshold-intensity at which there is motor response of APB/FBI in 5 out of 10 stimuli
- 80-110% of MT is used for various indications.
- The impulse may penetrate only 2–3 centimeters below the device. Deeper focused, noninvasive penetration is the Holy Grail of TMS research.^[4]

MOTOR CORTEX

- The discovery of the motor cortex was the next great example of the importance of electricity to central nervous system activity (Finger 2000). Gustav Fritsch noticed during the Danish-Prussian War of 1864 that accidentally irritating exposed brains of head injured soldiers often resulted in a twitch to the opposite side of the body.
- In the early 1900s, Charles Sherrington continued mapping out the details of the motor cortex. Using lightly anesthetized apes and monkeys, he recognized the contiguous nature of the motor control along the cortex. It was the great Canadian neuro- surgeon Wilder Penfield who, along with others, extended the work of Sherrington and delineated the odd-shaped little man hidden inside the cortex: the motor homunculus.^[5]



DIFFERENT TYPES OF TMS

- **Single-pulse TMS:** One pulse at a time, without regular rhythm and low rates of delivery
- **rTMS:** Pulses are repeated at regular rhythmic intervals (1-25 Hz)
- **Low frequency rTMS:** Frequency is 1 Hz or less.
- **High frequency rTMS:** frequency 5-20 Hz.

High-frequency rTMS is thought to have a stimulating effect on the cerebral cortex, whereas low-frequency rTMS is thought to have an inhibitory effect.

Theta burst stimulation (TBS): High frequency bursts (3 pulses at 50 Hz) applied at regular frequency (5 bursts per sec)

- cTBS: TBS applied continuously; inhibitory effects
- iTBS: TBS applied intermittently; excitatory effects

A standard rTMS session takes around 30 min whereas TBS session takes only 3 min and the latest research finds this modality to be non-inferior to high-frequency rTMS when used in patients with treatment-resistant depression.^[4]

TMS STIMULUS PARAMETERS

- **Train:** Several pulses administered continuously in a given period of time
- **Inter-train duration:** The time gap between two trains
- **Session:** A time period in a day when rTMS is administered; typically in one session several trains are administered
- **Frequency:** The number of pulses per second^[6]

Typical rTMS parameters

rTMS method	Pattern	Pulse mode	Pulses per burst	Frequency (Hz)	Total trains	Pulses per train	Inter-train intervals (seconds)	Pulses per session	Total time per session (minutes)
HF		Single pulse	NA	≥ 10	60	50	25	3,000	30
LF		Single pulse	NA	≤ 1	1	1,200	0	1,200	20
iTBS		Burst	3 (at 50 Hz)	5	20-30	30	8	600-900	4-7
cTBS		Burst	3 (at 50 Hz)	5	1	600-900	0	600-900	2-3

cTBS = continuous theta-burst stimulation; HF = high frequency; iTBS = intermittent theta-burst stimulation; LF = low frequency; NA = not applicable; rTMS = repetitive transcranial magnetic stimulation.

USES OF RTMS

- Study of normal brain function
- Therapeutic application:
 - rTMS applied to DLPFC in affective disorders
 - Low frequency rTMS applied to temporoparietal areas to reduce hallucinations
 - Some positive results in anxiety disorders, OCD, panic and Tourette's disorders^[6]
- Effective treatment of depression is available in the form of drugs, psychotherapy, electroconvulsive therapy (ECT) and other non-invasive brain stimulation methods.
- However, prevalence of treatment resistance in depression is close to 20% and research scholars are looking for options to tackle this challenge.
- Transcranial magnetic stimulation (TMS) is one of the newer options to treat depression.
- Imaging studies demonstrated that MDD may involve lower activity in the left DLPFC and higher activity in the right DLPFC.
- Research findings suggest that there is asymmetry in the functioning of the frontal lobe in patients with depression. Based on this concept, researchers have tried to treat patients with inhibitory (low frequency) stimulation to the right dorsolateral prefrontal cortex (DLPFC) or excitatory (high frequency) stimulation to the left DLPFC.
- rTMS treatment in MDD has often been shown to be associated with a normalization of hypo-excitability over the left prefrontal cortex and normalization of hyper-excitability over the right hemisphere.
- Kedzior et al have done a systematic review and meta-analysis of 16 double-blind, sham-controlled randomised controlled trials (RCT) using high-frequency rTMS over the left DLPFC. The antidepressant effect of rTMS was studied in follow-up and it was found that patients with unipolar depression, less severe depressive episodes, treatment-resistant depression, non-psychotic depression and those who were on concomitant antidepressant therapy had a better antidepressant effect with rTMS.^[7]
- Gaynes et al focused their meta-analysis on the role of rTMS in **treatment-resistant depression**. They searched the literature and evaluated 18 studies that rTMS was compared with sham-controlled treatment in patients who had failed to respond to two or more antidepressants. They could conclude with a high strength of evidence that rTMS produced a meaningful decrease in Hamilton Depression Rating Scale (HDRS) and likelihood to response was more than three times when compared with patients on sham control. Furthermore, patients on rTMS were five times more likely to achieve remission.^[8]
- Researchers have compared and analysed if there is any **difference** in the efficacy of rTMS applied to left versus right DLPFC. In their analysis, Chen et al pooled the data from eight RCTs and found that both high and low-frequency rTMS applied to left and right DLPFC, respectively, were equally effective. However, incidence of side effects, including seizures, was lesser with low-frequency right-sided rTMS.
- TMS is also found to be effective in **bipolar depression**. Deep TMS (dTMS) has been used in the treatment of bipolar depression and the early results suggest that the depressive features improve with TMS.
- In their open-label study on patients with unipolar and bipolar depression, Schulze et al found an **accelerated protocol** (two sessions per day) to be equally effective as the conventional TMS session that is once a day.^[9]

USES OF rTMS OTHER THAN MDD

- Auditory hallucinations in schizophrenia – 1 Hz TMS over superior temporal gyrus
- PTSD – 10 Hz over right prefrontal cortex
- ADHD – to target the right medial frontal gyrus
- Other areas being studied include Stroke rehabilitation, Migraine, Tourette's syndrome, Parkinson's disease.

SPECIAL POPULATION AND rTMS

- **Adolescence:**
 - Treatment of ADHD, tic disorders and medication-resistant epilepsy
 - Major concern is risk of seizure
 - Safe and moderately effective in depressed adolescents
 - most frequent adverse effect – mild headache
- **Pregnancy:**
 - Promising alternative to treat MDD in pregnancy

METALLIC OBJECTS, IMPLANTED PHYSIOLOGICALLY CONTROLLED ELECTRODES, AND RELATED DEVICES

- Any ferromagnetic material, internal pulse generators, or medication pumps within 30 cm of the coil placement constitute contraindications to TMS, as do cochlear implants. These objects present potential risks during TMS primarily due to potential heating effects from the magnetic field and damage to the internal circuitry of nearby implanted devices.
- Dental implants made with magnetically sensitive materials and located on the side of stimulation are also contraindications. Exceptions include dental hardware and titanium metals that have low conductivity and therefore pose little risk for damage to surrounding tissues (Rossi et al. 2009). Before a treatment session, anything worn on the head or neck that has conductive potential (e.g., jewelry, glasses) should be removed.
- Cardiac pacemakers, vagus nerve stimulation systems, spinal cord stimulators, and brain implants (e.g., aneurysm clips) pose a relative contraindication for TMS due to potential tissue injury related to mechanical movement induced by the rapidly alternating magnetic fields with TMS. Conventional TMS output, however, appears to remain below that necessary to cause tissue damage, and most device components are usually beyond the TMS-generated electromagnetic field.

ADVERSE EFFECTS OF RTMS

- Seizure induction • Headache • Scalp facial muscle twitching • Mild tinnitus, hearing loss

SEIZURE RISK WITH TMS

- Although seizure risk is the most serious potential TMS-related AE, seizures are uncommon. The most recent data indicate an estimated risk of less than 0.1% over an entire treatment course (typically 20–30 sessions) (Dobek et al. 2015).
- Of note, all documented TMS-induced seizures have occurred during treatment sessions.
- The risk of seizures during TMS compares favorably with risks from various psychotropic medications (e.g., antipsychotics, tricyclic antidepressants, bupropion) used to treat depression.
- Paradoxically, a recent systematic review indicates that TMS may suppress episodes in patients with epilepsy (Pereira et al. 2016).
- History of seizures or a seizure disorder does not pose an absolute contraindication to TMS, this issue should be carefully explored with the patient and appropriately addressed. Safeguards may include discontinuation of medications, which can lower the seizure threshold; adjustment in anticonvulsant medications to ensure adequate control; and consultation with a neurologist.
- Minimizing the risk of seizures begins with a careful assessment of factors that can increase seizure potential. These include a history of prior head injuries, stroke, brain lesions (e.g., vascular, traumatic, infectious), and/or seizure events; recent sleep deprivation; and the use of medications, alcohol, or other substances that may lower the seizure threshold.

- Administering TMS treatments within the recommended parameters (e.g., frequency, intensity, coil location, train duration, intertrain interval) is a crucial step in minimizing seizure risk.
- Because adhering to the suggested parameters may not always be possible, when parameters go beyond standard settings, patients should be informed of a potentially higher risk for a seizure.
- After the initial assessment and appropriate modification of risk factors, it is important that staff frequently re-inquire about any changes in the patient's status during the course of treatment. A corollary to such monitoring is the need for ongoing If confronted with a possible event, the treatment staff should clarify whether it is a vasovagal syncope, which may be more common than and can mimic some aspects of a seizure. Because of the possibilities of seizures and syncopal events, it is important that a licensed provider always be available on site for consultation when treatments are being provided.^[10]

SUMMARY

- High spatial and temporal resolution
- Can be used repeatedly in humans
- Provides information about the functional significance of a brain region ("virtual lesion technique")
- Restricted to brain regions close to the skull
- Difficult to interfere with higher order cognitive processes

	TRADITIONAL rTMS	DEEP rTMS or DEEP TMS		
COIL DESIGN	Figure-8 coil	H1-coil Bilateral PFC & DLPFC	H7-coil Medial PFC & ACC	H4-coil Bilateral Insula and PFC
				
DEPTH	0.7cm subdural	1.8cm subdural 2.5x deeper	3cm subdural 4x deeper	1.5cm subdural 2.1x deeper
BREADTH	3cm ³ volume	18cm ³ volume millions of more neurons	40.3cm ³ volume millions of more neurons	15.2cm ³ volume millions of more neurons
INDICATION	MDD	MDD	OCD	Smoking Cessation

Table 3-3. Issues to address prior to transcranial magnetic stimulation (TMS)

Patient's history
Epilepsy or seizure
Fainting spell(s) or syncope
Head trauma, concussion, or loss of consciousness
Hearing problems, ringing in the ears
Prior TMS treatment and any associated problems
Prior magnetic resonance imaging and any associated problems
Patient's current conditions
Presence and type of metal substance in the brain, skull, or elsewhere in the body
Presence of implanted device (e.g., cochlear implant, direct brain stimulation device, vagus nerve stimulation device)
Presence of cardiac pacemaker or intracardiac lines
Presence of medication infusion device
Current medications
Pregnancy

Source. Adapted from Rossi et al. 2011.

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Lumateperone in Bipolar Depression: A New Mechanism, New Possibilities?

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Why Lumateperone Matters

The Unmet Need in Bipolar Depression



Prevalence

>2%

Lifetime prevalence worldwide



Illness Burden

Depressive episodes
dominate the illness course



Suicide Risk

10–20×

Higher mortality; 25–50%
lifetime suicide attempts



Current Therapies

Trade-offs between efficacy,
sedation, metabolic effects
& EPS



The need for effective, better-tolerated therapies led to lumateperone.

20-08-2026

CANMAT/ISBD Bipolar Disorder Guidelines (2023)



Current Treatment Landscape in Bipolar Depression

FDA-approved pharmacological options and key limitations



Drug	Monotherapy	Adjunctive	Weight Gain	Sedation	EPS/Akathisia	Metabolic Risk	Mechanism
Quetiapine	✓	—	High	High	Low	High	D ₂ antagonist
Olanzapine + Fluoxetine	✓	—	Very High	Moderate	Low	Very High	D ₂ antagonist
Lurasidone	✓	✓	Low	Low	Moderate	Low	D ₂ antagonist
Cariprazine	✓	—	Low	Low	Moderate-High	Low	D ₃ -preferring partial agonist
Lumateperone	✓	✓	Low	Mild	Low	Low	Multimodal (5-HT _{2A} , D ₂ , glutamate)

Low Moderate High



KEY CLINICAL TAKEAWAYS



No single therapy is
ideal for every patient



Current options involve
trade-offs between
efficacy and tolerability



Metabolic burden varies
substantially across
agents



Lumateperone combines
multimodal pharmacology
with a favorable metabolic
and EPS profile.



Treatment selection should balance efficacy, tolerability, metabolic risk, and patient-specific factors.



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CANMAT/ISBD Bipolar Disorder Guidelines (2023)

Lumateperone: Drug Profile

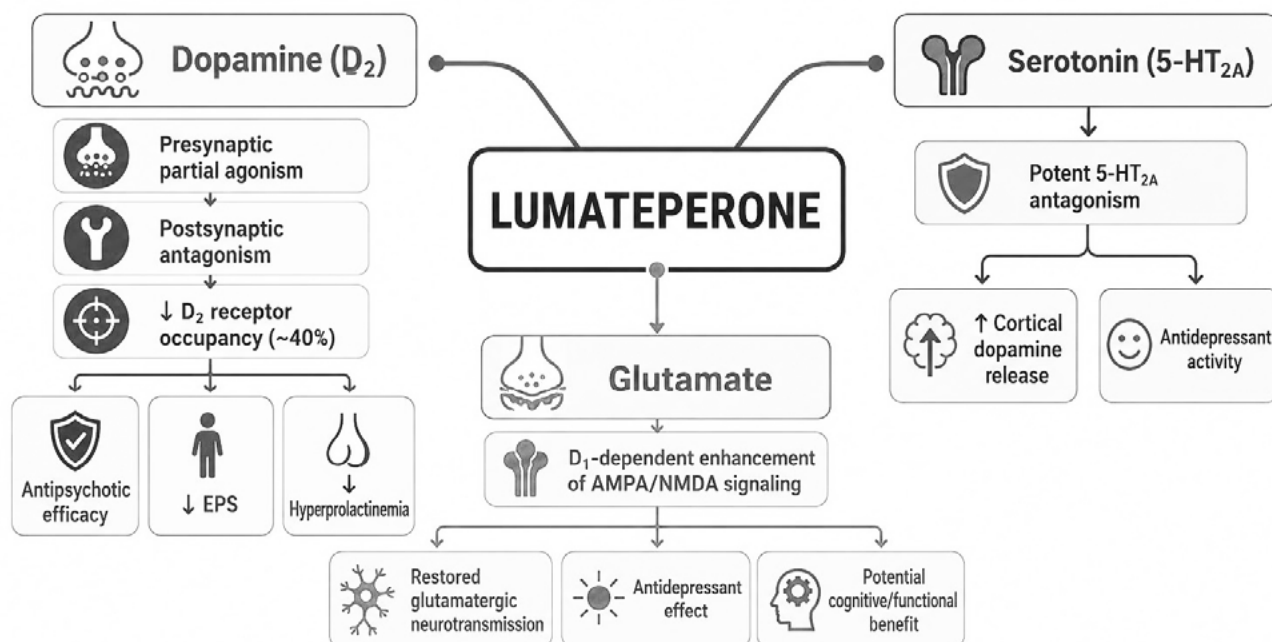
1 DRUG IDENTITY	2 PRACTICAL PRESCRIBING	3 PHARMACOKINETICS & SAFETY
 Drug class: Atypical antipsychotic	 Recommended dose: 42 mg PO once daily	 Metabolism: Primarily CYP3A4 (minor CYP2C8/CYP1A2)
 Mechanism: First-in-class; multimodal modulation of dopamine, serotonin & glutamate neurotransmission	 Dose titration: Not required	 Half-life: ~18 hours
 FDA approvals: Schizophrenia (2019); Bipolar depression (2021)	 Administration: With or without food	 Protein binding: ~97%
 Approved indication: Depressive episodes associated with Bipolar I & II disorder (monotherapy or adjunctive to lithium/valproate) in adults		 Hepatic impairment: Avoid in moderate or severe hepatic impairment (Child-Pugh B/C)
		 Renal impairment: No dosage adjustment required

One capsule • One dose • No titration

20-08-2026

CAPLYTA® Prescribing Information US FDA. 2025

Lumateperone: A Novel Mechanism of Action



Multimodal modulation of serotonin, dopamine, and glutamate distinguishes lumateperone from conventional second-generation antipsychotics.

20-08-2026

CAPLYTA® Prescribing Information. US FDA. 2025

Receptor Pharmacology: How Does Lumateperone Differ?

Relative receptor pharmacology underlying the distinct clinical profile of lumateperone.

Drug	D2	D3	5-HT _{2A}	SERT	H1	M1	Clinical Signature
★ Lumateperone	+	-	+++	+	+	0	Low EPS • Low metabolic burden • Multimodal
Olanzapine	+++	-	++	0	+++	++	High metabolic burden
Quetiapine	++	-	++	0	+++	+	Sedation • Weight gain
Lurasidone	++	-	++	0	+	0	Metabolically favorable
Cariprazine	PA	+++	+	0	+	0	D3-preferring partial agonist
Aripiprazole	PA	++	+	0	+	0	Activating • Akathisia risk
Brexpiprazole	PA	++	++	0	+	0	Lower intrinsic D2 activity

+++ High affinity | ++ Moderate | + Low | 0 Negligible | PA Partial agonist

Relative receptor affinities are qualitative summaries from published pharmacologic data and should not be interpreted as direct predictors of clinical efficacy.

Why is Lumateperone Different?



Lower functional D2 occupancy (~39%)



Potent 5-HT_{2A} antagonism



Moderate SERT inhibition



Minimal H₁/M₁ affinity

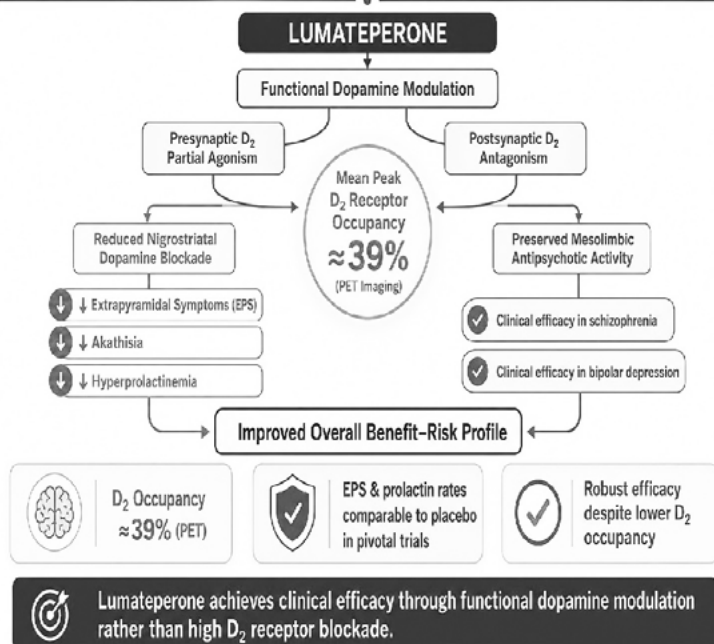


Mechanistically linked to efficacy with improved tolerability.

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Stahl's Essential Psychopharmacology, 5th Ed. 2021 | Greenwood J, et al. Ann Pharmacother. 2021

Low D₂ Occupancy: The Science Behind Better Tolerability



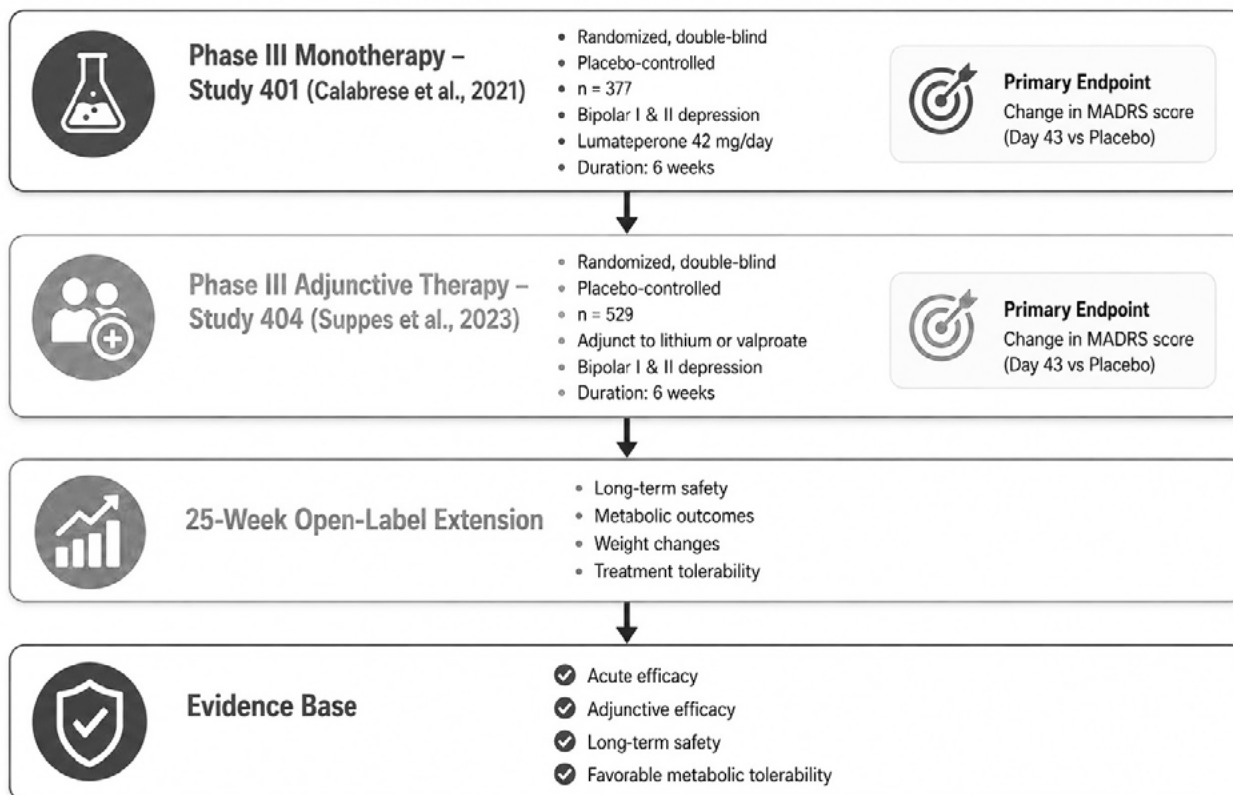
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Vanover KE, et al. Neuropsychopharmacology. 2019.

Comparison of Mean D₂ Receptor Occupancy Across Antipsychotics

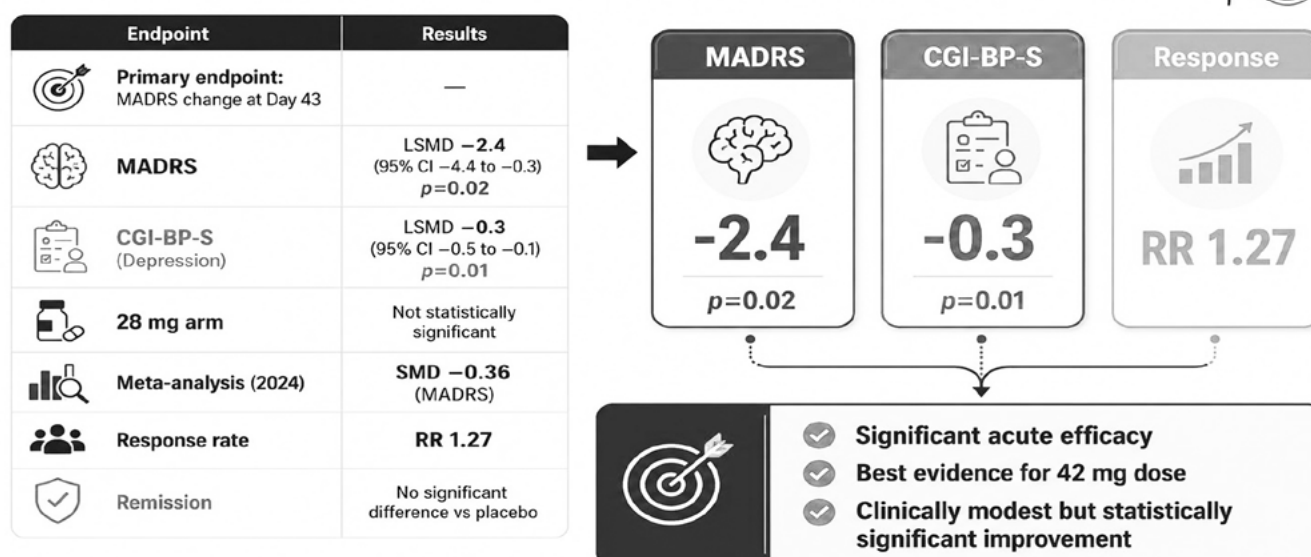
Drug	Dose range	Mean D ₂ RO in Caudate and Putamen
ITI-007	60 mg/day	~ 40%
Clozapine	75–900 mg/day	48–61%
Quetiapine	150–750 mg/day	30–62%
Ziprasidone	40–160 mg/day	56 to >59%
Risperidone	4 mg/day	72–81%
Olanzapine	5–60 mg/day	61–80%
Lurasidone	40–80 mg	>65%
Cariprazine	1.5–3 mg/day	69 to >90%
Aripiprazole	10–30 mg	88–90%

Clinical Development of Lumateperone in Bipolar Depression



20-08-2026
Calabrese JR, et al. *Am J Psychiatry* 2021 | Suppes T, et al. *Bipolar Disord.* 2023 | Toben M, et al. *Int Clin Psychopharmacol.* 2026

Acute Efficacy – What Did the Trials Show?



Lumateperone demonstrates statistically significant, clinically modest improvement in acute bipolar depression

20-08-2026

Suppes T, et al. *Bipolar Disorders.* 2023 | Peng H, et al. *Int J Neuropsychopharmacol.* 2024

Efficacy Across Bipolar Subtypes

- 1  Monotherapy analyses suggested efficacy in both Bipolar I and Bipolar II.
- 2  Significant MADRS item improvement across both subtypes.
- 3  Anhedonia factor effect size: -0.36 in Bipolar I; -0.90 in Bipolar II.
- 4  Bipolar II subgroup sizes were small, limiting precision.
- 5  CANMAT/ISBD still places lumateperone as second-line in Bipolar II depression.

Consistency of Treatment Effect

Lumateperone 42 mg



Bipolar I Depression

- ✓ Significant MADRS improvement
- ✓ Improved CGI-BP-S



Comparable treatment effect



Bipolar II Depression

- ✓ Similar efficacy trend
- ✓ No meaningful heterogeneity

Overall Interpretation

- ✓ Consistent efficacy
- ✓ Broad applicability
- ✓ Supports use across bipolar spectrum

20-08-2026
Calabrese JR, et al. *CNS Spectr*. 2021 | Suppes T, et al. *Bipolar Disorders*. 2023 | Peng H, et al. *Int J Neuropsychopharmacol*. 2024

Safety & Tolerability

-  **Common AEs in monotherapy trials:** headache 14%, somnolence/sedation 13%, dizziness 8%, nausea 8%, dry mouth 5%.
-  **In adjunctive trial:** somnolence/sedation 13%, dizziness 11%, nausea 9%, dry mouth 5%.
-  **No single AE caused discontinuation >2%.**
-  **Monotherapy EPS-related events:** 1.3% vs 1.1% placebo.
-  **Adjunctive EPS-related events:** 4.0% vs 2.3% placebo.

Clinical Safety Profile of LUMATEPERONE

1 NEUROLOGICAL SAFETY

- ✓ EPS comparable to placebo
- ✓ Minimal akathisia
- ✓ No clinically meaningful prolactin elevations

2 COMMON TEAEs

- | | |
|--|--|
|  Somnolence |  Dizziness |
|  Dry mouth |  Fatigue |
|  Nausea |  Low discontinuation due to adverse effects |

3 CARDIOVASCULAR SAFETY

- ✓ No clinically meaningful ECG changes
- ✓ No increase in cardiac profile risk markers

OVERALL BENEFIT-RISK:
LUMATEPERONE has a favorable tolerability and overall safety profile.

20-08-2026

CAPLYTA® (Lumateperone) Prescribing Information. U.S. FDA. 2025

Weight & Metabolic Profile – A Key Clinical Differentiator



Metabolic Outcomes

Parameter	Finding
Weight gain (6 weeks)	Comparable to placebo
Fasting glucose	Similar to placebo
Insulin	Similar to placebo
Total cholesterol	Similar to placebo
Triglycerides	Similar to placebo
6-month OLE mean weight change	-0.01 kg (Day 175)



Long-term Lipid Shifts (OLE)

Parameter	Shift from Normal → High
Total cholesterol	10%
Triglycerides	5%
LDL cholesterol	2%



Minimal short-term metabolic impact with essentially weight-neutral long-term outcomes.

20-08-2026

CAPLYTA® (Lumateperone) Prescribing Information. U.S. FDA. 2025.



Metabolic Burden Across Approved Bipolar Depression Treatments

Drug	Weight Gain	Metabolic Risk
Lumateperone	● Low	● Low
Lurasidone	● Low	● Low
Cariprazine	● Low	● Low
Quetiapine	● Moderate	● Moderate
Olanzapine/Fluoxetine	● High	● High

Suitable for patients with:



Obesity



Type 2 diabetes



Metabolic syndrome



High cardiovascular risk



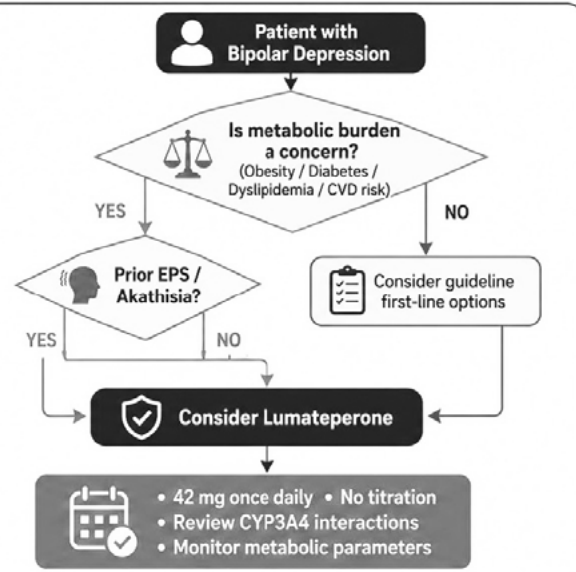
Practical Positioning in Clinical Practice

Consider Lumateperone When:

	Obesity/Metabolic syndrome	Minimal weight & metabolic effects
	Type 2 diabetes/Dyslipidemia	Favorable cardiometabolic profile
	Prior EPS or akathisia	Low D ₂ occupancy, low EPS burden
	Need for rapid initiation	42 mg once daily, no titration
	Polypharmacy	Simple dosing, review CYP3A4 interactions

Clinical Monitoring

	Review CYP3A4 inhibitors/inducers		Assess hepatic function		Monitor weight, glucose & lipids		Monitor sedation, dizziness & falls risk		Routine assessment for EPS
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Best suited for patients where tolerability—not efficacy alone—drives treatment selection.

20-08-2026

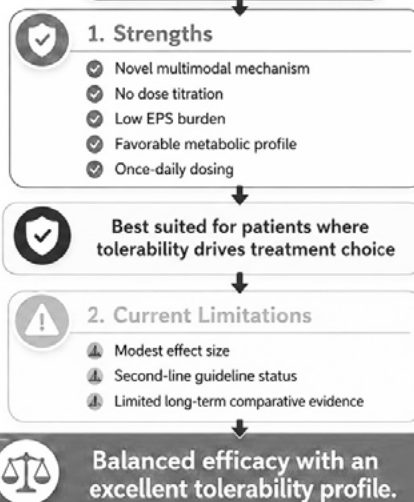
CAPLYTA® Prescribing Information. U.S. FDA. 2025 | CANMAT/ISBD Bipolar Disorder Guidelines (2023)

Key Take-Home Messages

Domain	Key Message
 Mechanism	Multimodal modulation of serotonin, dopamine & glutamate
 Efficacy	Significant but modest improvement in acute bipolar depression
 Safety	Low EPS, minimal prolactin effects, favorable tolerability
 Metabolic profile	Weight and metabolic changes comparable to placebo
 Clinical role	Second-line (CANMAT/ISBD 2023); ideal when tolerability is a priority

Benefit-Risk Summary: Lumateperone in Bipolar Depression

LUMATEPERONE



Lumateperone offers a unique balance of efficacy and tolerability, making it a valuable option for carefully selected patients with bipolar depression.

20-08-2026

CANMAT/ISBD Bipolar Disorder Guidelines (2023)

Few Clinical Questions

Question	Evidence-Based Answer
1  Is lumateperone first-line for bipolar depression?	> No. CANMAT/ISBD 2023 recommends lumateperone as a second-line option for acute bipolar I and II depression.
2  What is the recommended dose?	> 42 mg once daily, with or without food. No dose titration is required.
3  How effective is it?	> Adjunctive Phase III trial: MADRS LS mean difference -2.4 vs placebo (p=0.02). Pooled meta-analysis: SMD -0.36 , indicating a modest but significant antidepressant effect.
4  Does it work in Bipolar II disorder?	> Available data suggest similar efficacy in Bipolar II; however, subgroup analyses are smaller and less precise than those for Bipolar I.
5  Which adverse effects should I counsel patients about?	> Most common TEAEs include somnolence, dizziness, nausea, headache, and dry mouth . These are generally mild to moderate.
6  What about EPS and akathisia?	> Low EPS incidence, with rates close to placebo in acute trials; minimal prolactin elevation.
7  Does it cause weight gain?	> Short-term trials demonstrated weight and metabolic changes comparable to placebo, with minimal long-term weight change.
8  Which patients are most likely to benefit?	> Patients with metabolic syndrome, obesity, diabetes, previous EPS , or intolerance to sedating/weight-promoting antipsychotics.
9  Are there important drug interactions?	> Avoid CYP3A4 inducers . Reduce the dose with moderate or strong CYP3A4 inhibitors according to prescribing information.
10  Is there maintenance efficacy evidence?	> Not yet from randomized relapse-prevention trials . Current long-term evidence is mainly from open-label extension studies demonstrating safety and tolerability.



Key Take-home: Lumateperone offers evidence-based efficacy with a favorable tolerability profile for appropriately selected patients.

20-08-2026

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20-08-2026

Gender Incongruence- A Brief Overview

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History of Transgenders In India

Mythological Foundations:

- Concept of gender fluidity is deeply rooted in Hindu texts.
- Ardhanarishvara: synthesis of masculine (Shiva) & feminine (Shakti).
- Shikhandi (Mahabharata): transgender warrior.
- Brihannala (Arjuna's transgender role during exile).

Early Social Roles:

- Recognised as a distinct social category in ancient Indian society.
- Roles in royal courts, temples, and cultural performances

Religious & Cultural Recognition:

- Presence in epics: Ramayana & Mahabharata.
- Associated with blessings in childbirth & marriage rituals.
- Mention of "Tritiya Prakriti" (third nature) in ancient texts.

Philosophical Perspective:

- Indian thought accepted non-binary gender identities.
- Fluidity is seen as part of cosmic balance rather than pathology.

Definition

- Gender incongruence refers to a marked and persistent mismatch between an individual's experienced gender and their assigned sex at birth."
- Previously named as gender identity disorder/ gender dysphoria.
- Not a "disorder" in modern classification.
- Focus is on distress, not identity itself.
- Shift from pathology → affirmative care model.

Core Terminologies

- Sex Assigned at Birth:
 - Classification (male/female/intersex) based on external genitalia at birth.
 - It is a biological assignment, not a psychological identity.
- Gender Identity:
 - One's deeply felt internal sense of being male, female, both, or neither.
 - It may or may not align with sex assigned at birth.
- Gender Expression:
 - The external manifestation of gender through behaviour, clothing, voice, or mannerisms.
 - It is socially influenced and culturally variable.
- Sexual Orientation:
 - Refers to enduring patterns of emotional, romantic, or sexual attraction.
 - It is independent of gender identity.

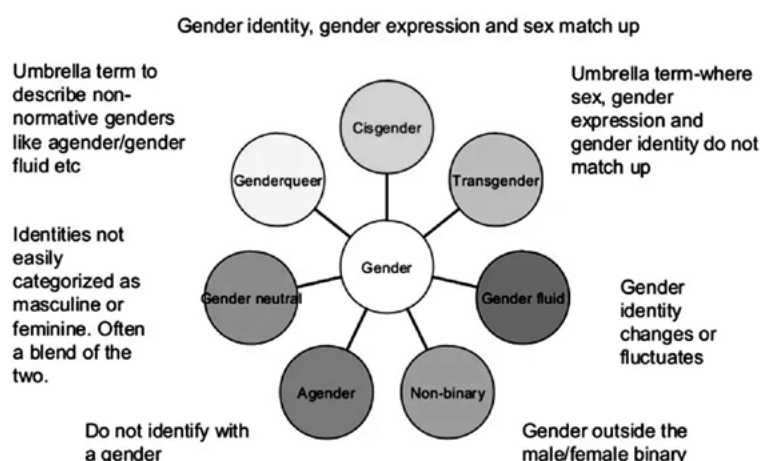
Important Identities

- Transgender:
 - Individuals whose gender identity differs from their sex assigned at birth.
 - It is an umbrella term, not a diagnosis.
- Cisgender:
 - Individuals whose gender identity aligns with their sex assigned at birth.
 - Represents the majority population baseline.
- Non-binary:
 - Individuals whose gender identity does not fit strictly into male or female categories.
 - May identify as both, neither, or somewhere in between.

Gender Fluid

- A gender identity characterised by dynamic shifts over time between different gender experiences.
- Individuals may feel more masculine, feminine, both, or neither at different points.
- Variability—the identity is not fixed but changes across time or context.
- Unlike non-binary (which may be stable), gender fluidity emphasises temporal fluctuation.
- Not pathological.
- Requires an affirmative, flexible understanding.
- Important to respect current identity.

Gender Terms



Nosological Evolution

Classification manual and edition	Gender-related taxonomy	Category
ICD-9 (1975)	Transvestism and transsexualism	Sexual deviations
DSM-III (1980)	Transsexualism	Psychosexual disorders
DSM-III-R (1987)	Transsexualism	Disorders usually first evident in infancy, childhood or adolescence
ICD-10 (1990)	Transsexualism	Gender identity disorders
DSM-IV (1994) and DSM-IV-TR (2000)	Gender identity disorder in adolescents or adults	Sexual and gender identity disorders
DSM-5 (2015)	Gender dysphoria in adolescents or adults	Gender dysphoria
ICD-11 (2021)	Gender incongruence of adolescents and adults; Gender incongruence of childhood	Conditions related to sexual health (moved out of Chapter 5 'Mental disorders')

ICD-11 criteria of gender incongruence

- Marked and Persistent Incongruence: A long-lasting inconsistency between the experienced gender and the assigned sex.
- Desire for Modification: A strong desire to be rid of some or all primary and/or secondary sex characteristics (e.g., breasts, genitalia) due to the incongruence.
- Desire for Affirmation: A strong desire to possess the primary and/or secondary sex characteristics of the experienced gender.
- Desire for Treatment: A strong desire to be treated and accepted as a person of the experienced gender (or another gender).

Sub-Categories

- Gender Incongruence of Adolescence or Adulthood (HA60): As described above.
- Gender Incongruence of Childhood (HA61): Applied to prepubertal children, characterised by a persistent incongruence, often including a strong desire to be the other gender and intense rejection of their assigned gender's characteristics.
- Gender Incongruence, Unspecified (HA6Z)

Novelty in Classification

- Removed from mental health: moved from “mental and behavioural disorders” to “conditions related to sexual health,” reducing stigma.
- No distress requirement: unlike DSM-5, GI does not require significant distress or functional impairment for diagnosis.
- Variant behavior not sufficient: gender variant behavior and preferences alone, particularly in children, do not justify a diagnosis.
- Focus on incongruence: the diagnosis centres on the discrepancy rather than psychiatric illness

Comparative Nosology

Domain	DSM-5-Tr: Gender Dysphoria	ICD-11: Gender Incongruence
Conceptual Focus	Distress Due To Incongruence	Incongruence Itself (Identity-Based)
Classification	Mental Disorder Chapter	Sexual Health Chapter (Non-Psychiatric)
Core Requirement	Clinically Significant Distress/ Impairment Mandatory	Distress Not Required For Diagnosis
Terminology Shift	Retains “Dysphoria” (Pathology-Linked)	Uses Neutral Term → Reduces Stigma
Clinical Role	Emphasis On Diagnosis & Gatekeeping	Emphasis On Access & Affirmation
Philosophical Model	Residual Medical Model	Depathologization Model

Epidemiology

- Global Scenario:
 - Estimated prevalence: ~0.3–0.6% of adult population (varies by methodology).
 - Increasing trends, especially among adolescents and young adults.
 - Rise likely reflects greater awareness, social acceptance, and access to care rather than true incidence change.
- Indian Scenario:
 - Census 2011: ~4.9 lakh individuals identified as transgender (likely underestimation).
 - Clinic-based data show rising help-seeking, particularly in urban tertiary centers.
 - Significant underreporting due to stigma, discrimination, and limited access to services.

Biological

1. Conceptual Basis:
 - Gender identity likely reflects brain sexual differentiation during early neurodevelopment.
 - Incongruence may arise when brain and body differentiation diverge.
2. Brain Sexual Differentiation:
 - Prenatal hormones (especially androgens) organise brain structures in a sex-dimorphic way.
 - These effects are permanent (organisational) and later modulated by hormones.
3. Structural Brain Findings:
 - Regions like BSTc and the hypothalamus may resemble the individual’s experienced gender.
 - However, findings are inconsistent and not diagnostic.
4. Functional Neurobiology:
 - fMRI studies show brain activation patterns aligning with identified gender, especially in the hypothalamus and insula.
 - Differences are noted in self-perception and body image networks.
5. Genetic Factors:
 - No single gene identified; studies suggest minor associations with sex hormone receptor genes.
 - Overall genetic contribution is suggestive but weak.

INFLUENCING FACTORS



6. Prenatal Hormonal Influence:

- Conditions like CAH indicate that early androgen exposure may influence gender identity.
- Hormonal effects are probabilistic, not deterministic.

7. Neurodevelopmental Model:

- Gender identity emerges from the interaction of genes, hormones, and brain development.
- It is best viewed as a spectrum, not a binary outcome.

8. Limitations:

- Most studies are small, cross-sectional, and confounded by hormone therapy.
- Causality remains uncertain (cause vs effect vs adaptation).

9. Current Consensus:

- Evidence supports a biological contribution, but no definitive marker exists.
- Gender incongruence is best understood as a biopsychosocial phenomenon.

Psychosocial Factors

- Cognitive and Attachment Theory: Some theoretical frameworks suggest that gender incongruence can be related to the development of self-representation, particularly in cases where early childhood development involves severe stress or disruption in attachment.
- Family System Factors: Some, though not all, theories propose that gender incongruence might develop as a way for a child to resolve or manage complex emotional conflicts within their family system.
- ASD and Cognitive Rigidity: Research has indicated an overrepresentation of Autism Spectrum Disorder (ASD) traits among individuals with gender incongruence. Some hypotheses suggest that cognitive traits associated with ASD, such as fixated interests or difficulties in tolerating ambiguity, may play a role.
- Socialization Factors: The internalization of societal expectations, rigid gender norms, and social interactions can lead to the psychological development of distress about one's assigned gender.

Psychiatric Comorbidities

- Common Comorbidities
 - Depressive disorders: High prevalence; strongly linked to minority stress and stigma.
 - Anxiety disorders: Social anxiety and generalised anxiety are frequent.
 - Substance use disorders: Often related to coping with distress and discrimination.
 - Suicidality & self-harm: Significantly elevated risk compared to the general population.
- Other Associations
 - Trauma-related disorders (PTSD): Due to higher exposure to violence and victimisation.
 - Autism spectrum traits: Reported increased co-occurrence (especially in adolescents).

Treatment

Approach & Assessment

1. Guiding Principles:
 - Affirmative, non-pathologising care
 - Respect autonomy, identity, and chosen pronouns
 - Avoid any form of conversion/reparative therapy
2. Comprehensive Assessment:
 - Gender history: onset, persistence, degree of incongruence
 - Gender dysphoria and level of distress
 - Psychiatric comorbidities: depression, suicidality, substance use
 - Psychosocial context: family support, stigma, functioning
3. Capacity & Readiness:
 - Assess decision-making capacity for interventions
 - Evaluate expectations and understanding of outcomes
4. Multidisciplinary Model:
 - Psychiatrist, psychologist, endocrinologist, surgeon, social worker

Management

- Psychosocial Interventions:
 - Psychoeducation & supportive psychotherapy
 - Family counselling and social support enhancement
 - Facilitation of social transition (name, pronouns, role)
- Medical Interventions:
 - Hormone therapy (gender-affirming)
 - Puberty blockers (in adolescents, case-specific)
- Surgical Interventions:
 - Gender-affirming surgeries (chest, genital, facial)
 - Requires careful assessment and informed consent

Management is not about changing identity — but enabling individuals to live in alignment with it, safely and authentically.”

Take Home Points

- Represents a shift from pathology → identity → affirmation.
- Modern nosology emphasises depathologization while preserving access to care.
- Neurobiological evidence suggests developmental underpinnings, but remains non-deterministic.
- Psychiatric comorbidities largely reflect minority stress, not intrinsic pathology.
- Management requires a multidisciplinary, affirmative, and individualised approach.
- Psychiatry’s role has evolved from gatekeeping → facilitation and support.

“Gender incongruence challenges psychiatry to balance science, ethics, and human dignity —moving beyond diagnosis toward holistic care.”

“Our task is not to define identity, but to reduce suffering and enable authentic living.”

Creativity and Negativity in Bipolar Disorder

Dr. Tamoghna Bandyopadhyay, RMO Cum Clinical Tutor
Department of Psychiatry, Murshidabad Medical College & Hospital

Opening Reflection

The same fire that enlightens can also burn: the pathology is not the presence of intensity—but the loss of modulation.	Bipolarity as a dialectic of intensity: bipolarity is not simply oscillation—it is a dialogue between extremes, where intensity itself becomes both creator and disruptor.”
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Historical–Cultural Imagination

Melancholia and genius [aristotle]: emotional depth and sensitivity may enrich perception—but this does not mean illness causes genius.	→	Aestheticization of suffering: suffering gets romanticized as art, rather than recognized as distress.	→	Caution against romanticization: appreciate creativity — but do not glorify the illness behind it.
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Noso logical Grounding

Mania, Hypomania, Depression	→	Episodicity Vs Temperament: Episodes Are The Peaks And Troughs—But Temperament Is The Underlying Terrain.”	→	Trait Vs State: Not All Creativity Seen In Bipolar Disorder Is Trait-Based — Some Is State-Driven And Unstable
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Creativity: Psychological Lens

Divergent Thinking: Divergent Thinking Expands The Field Of Possibilities—Without Guaranteeing Coherence.	Associative Fluency: Associative Fluency Is The Bridge Between Ideas—Too Tight, And Thought Is Rigid; Too Loose, And It Fragments.	Cognitive Disinhibition: Cognitive Disinhibition Opens The Gates Of The Mind—What Enters May Be Insight, Or It May Be Chaos.
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Neurocognitive Correlates

- Dopaminergic surge: dopamine does not create ideas—it assigns importance to them, sometimes excessively.”
- Reduced latent inhibition.
- Network shifts: creativity is a conversation between brain networks—bipolarity alters both the volume and the coordination of that dialogue.

Hypomania — Liminal Zone

Heightened energy: not restlessness—it is directed momentum.	→	Expansive Associations.	→	Productive edge: it is where creativity thrives—precisely because it stands so close to disintegration.
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Mania — Collapse of Form

Flight Of Ideas	Grandiosity.	Entropy of thought: abundance of ideas loses form—when creativity dissolves into cognitive chaos.”
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Depression — Silence

Psychomotor Inhibition.	→	Anhedonia.	→	Loss of narrative: is the breakdown of the self’s storyline—where experiences no longer cohere into a meaningful past, present, or future.
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Phenomenology of Negativity

Ontological heaviness: when existence itself acquires weight—making even being feel effortful.	→	Future Foreclosure:	→	Diminished Self.
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Creativity & Suffering

Transformative vs incapacitating.	Post-episode reconstruction: process of reassembling experience into a coherent narrative after an episode has resolved.
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Temporal Paradox

Episodic Creativity.	→	Fragmentation vs integration: fragmentation generates material; integration gives it meaning—creativity depends on their balance.
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Cost of Intensity

Cognitive Fragmentation.	Functional Erosion.	Identity Disruption.
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Cultural Reflections

Van Gogh	→	Virginia Woolf.	→	Sylvia Plath.	→	These individuals were not creative because they were ill—they were creative individuals who, despite illness, were able at times to transform their experiences into art.
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Closing Synthesis

Disorder of rhythm and intensity: where the music of the mind becomes either overwhelming or silent.	Creativity at the edge, not extremes: creativity does not lie in the peaks or the depths—but in the narrow, unstable space where intensity is still held within form.
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“The world breaks everyone, and afterward, some are strong at the broken places.”



“Pain and suffering are always inevitable for a large intelligence and a deep heart.”

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