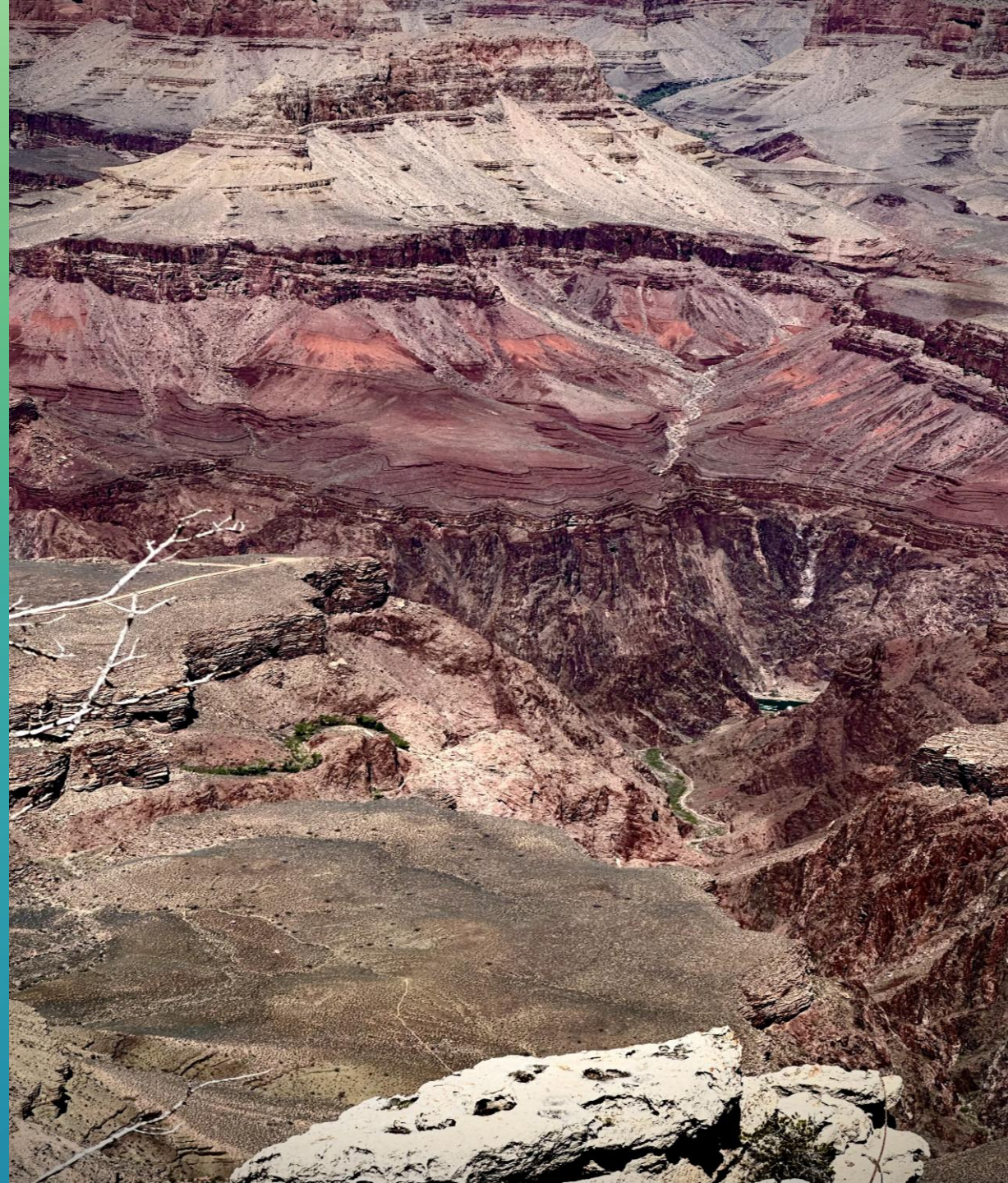
BEYOND MEDICATION CYCLING

IDENTIFYING CANDIDATES FOR TRANSCRANIAL MAGNETIC STIMULATION IN TREATMENT-RESISTENT DEPRESSION

R. Wallace DO IFMCP



DISCLOSURE

OWNER BRAIN TREATMENT CENTER MICHIGAN

Did you know that…

Depression in the United States…

Affects over 18 million adults **(one in ten)** in any given year.
Is the leading cause of disability for ages 15-44.
Is the primary reason why **someone dies of suicide about every 12 minutes**. – over 41,000 people a year.
In comparison: homicide claims less than 16,000 lives each year, according to 2013 CDC statistics.

[3]Kessler RC et al. Prevalence, Severity, and Comorbidity of Twelve-Month DSM-IV Disorders in the National Comorbidity Survey Replication (NCS-R). Archives of General Psychiatry, 2005 Jun; 62:617-627.
[4]Ibid
[5]Centers for Disease Control and Prevention (CDC) Web -based Injury Statistics Query and Reporting System (WISQARS) [Online].(2013,2011)National Center for Injury Prevention and Control, CDC (producer).
Available from http://www.cdc.gov/injury/wisqars/index.html.

Depression in the Workplace…

Causes 490 million disability days from work each year in the U.S.
Accounts for $23 billion in lost workdays each year.
Takes an economic toll over $100 billion each year from U.S. business.

[6]Merikangas KR et al. The Impact of Comorbidity of Mental and Physical Conditions on Role Disability in the US Adult Household Population. Arch Gen Psychiatry/vol 64 (no.10), Oct 2007.
[7]Greenberg P et al. The Economic Burden of Adults with Major Depressive Disorder in the United States (2005 and 2010). The Journal of Clinical Psychiatry/vol 76 (no. 2), Feb 2015.

Burden of Depression

Major Depressive Disorder (MDD) = leading cause of disability

High recurrence, morbidity, mortality

Often Treatment Resistance

~30–40% patients fail ≥2 antidepressants

Defined as Treatment-Resistant Depression (TRD)

McIntyre RS, Alsuwaidan M, Baune BT, et al. Treatment-resistant depression: definition, prevalence, detection, management, and investigational interventions. *World Psychiatry*. 2023;22(3):394-412. doi:10.1002/wps.21120

Limitations of Current Treatments

1. Medications: delayed onset, side effects
2. Psychotherapy: variable access
3. ECT: effective but cognitive risks

Options for treatment resistant depression

Electroconvulsive therapy (ECT)

ECT has the **strongest overall antidepressant effect**, especially for severe, psychotic, catatonic, or highly treatment-resistant depression.

A 2024 meta-analysis reported pooled **response around 73%** and **remission around 51%**, and other meta-analytic work has similarly found very high response but low remission rate

1. Tokutsu Y, Umene-Nakano W, Shinkai T, et al. Follow-up Study on Electroconvulsive Therapy in Treatment-resistant Depressed Patients after Remission: A Chart Review. *Clin Psychopharmacol Neurosci*. 2013;11(1):34-38. doi:10.9758/cpn.2013.11.1.34 I

Ketamine / intranasal esketamine

For **rapid symptom reduction**, ketamine-class treatments are among the most effective options, especially in **treatment-resistant depression (TRD).**,

The main findings of the meta-analyses were as follows: there was **no statistically significant difference between ketamine and ECT groups regarding the improvement of depressive symptoms** (primary outcome). Ketamine has less muscle pain

1. de A Simoes Moreira D, Gauer LE, Teixeira G, Fonseca da Silva AC, Cavalcanti S, Quevedo J. **Efficacy and adverse effects of ketamine versus electroconvulsive therapy for major depressive disorder**: A systematic review and meta-analysis. *J Affect Disord*. 2023;330:227-238. doi:10.1016/j.jad.2023.02.152

Antidepressant treatment options

- **Combined treatment: antidepressant medication + psychotherapy**
  For many adults with nonpsychotic major depression, **combined treatment** tends to outperform either medication alone or psychotherapy alone.
- **Antidepressant medication (standard pharmacotherapy)**
  For acute adult major depression, **antidepressants are clearly more effective than placebo** overall.
- **Repetitive transcranial magnetic stimulation (rTMS)**
  rTMS is an evidence-based option for depression, especially after inadequate medication response.
- **Psychotherapy alone**
  Psychotherapies such as CBT, interpersonal therapy, and behavioral activation are effective for adult depression. **Combined treatment usually does better than either one by itself.**
- **Vagus nerve stimulation (VNS)**
  VNS has evidence for **markedly treatment-resistant depression**, especially for longer-term benefit, but it is not usually ranked above ECT, ketamine/esketamine, standard antidepressants, or rTMS for typical acute antidepressant effect.
- Łysik A, Logoń K, Szczygieł A, Wołoszczak J, Wrześniewska M, Leszek J. Innovative approaches in the treatment-resistant depression: exploring different therapeutic pathways. *Geroscience*. 2025;47(4):5543-5558. doi:10.1007/s11357-025-01615-8

What is transcranial magnetic stimulation

Definition
Non-invasive neuromodulation using magnetic fields

Mechanism (Basic)
Magnetic pulse → electric current → neuronal depolarization

Target
Left dorsolateral prefrontal cortex (DLPFC)
FPz Pz
MRI guided Neuronavigation

History of TMS

- ~ Origins
- 1985: First human TMS by Anthony Barker
- Initially diagnostic (motor cortex mapping)
- ~ Evolution
- 1990s: Psychiatric applications
- 2008: FDA approval for depression
- ~Present day
- rTMS, iTBS, Deep TMS
- ~Personalized targeting
- (EEG/fMRI emerging)
- Levit A. History and future directions of rTMS for treatment of depressive disorders. *Am J Psychiatry Residents J.* 2023;18(3). doi:10.1176/appi.ajp-rj.2023.180304

PATHOPHYSIOLOGY OF DEPRESSION

Network Disorder Theory

Depression = network dysfunction (not just serotonin)

Borsboom D. A network theory of mental disorders. *World Psychiatry*. 2017;16(1):5-13. doi:10.1002/wps.20375

Hypoactivity Theory

Reduced DLPFC activity

Increased limbic activity

Trifu SC, Trifu AC, Aluaş E, Tătaru MA, Costea RV. Brain changes in depression. *Rom J Morphol Embryol*. 2020;61(2):361-370. doi:10.47162/RJME.61.2.06

Functional Connectivity Theory

Dysregulated fronto-limbic circuits

Gallo, S., El-Gazzar, A., Zhutovsky, P. *et al.* Functional connectivity signatures of major depressive disorder: machine learning analysis of two multicenter neuroimaging studies. *Mol Psychiatry* **28**, 3013–3022 (2023). https://doi.org/10.1038/s41380-023-01977-5

PHYSIOLOGY OF TMS (WHY IT WORKS)

- **Cortical Excitability**
- High-frequency TMS → excitatory
- Low-frequency → inhibitory

- **Network Rebalancing**
- Restores prefrontal control over limbic system

- **Neuroplasticity**
- Long-term potentiation (LTP)-like effects
- Long-term depression (LTD)-like modulation

- Bliss TV, Cooke SF. Long-term potentiation and long-term depression: a clinical perspective. *Clinics (Sao Paulo)*. 2011;66 Suppl 1(Suppl 1):3-17. doi:10.1590/s1807-59322011001300002

Neurophysiology

1. **Synaptic Plasticity**
2. Changes glutamate signaling
3. Modulates GABA balance

4. **Dopamine Effects**
5. Increased dopamine release in striatum

6. **Neurotrophic Effects**
7. ↑ Brain-Derived Neurotrophic Factor (BDNF)

8. **EEG Changes**
9. ↑ Alpha coherence
10. ↓ slow wave burden

11. **Default Mode Network**
12. Reduces hyperconnectivity (rumination network)

Synaptic plasticity

- What is synaptic plasticity?
- Synaptic plasticity is the brain's ability to **strengthen or weaken communication between neurons** over time.
- **Synapse** = connection point between two neurons
- **Plasticity** = ability to change, adapt, and reorganize
- Two major forms
- 1. Long-Term Potentiation (LTP)
- Synaptic connection becomes **stronger** -Signals pass more efficiently
- Supports learning, memory, and adaptive recovery

- 2. Long-Term Depression (LTD)
- Synaptic connection becomes **weaker** - Helps refine circuits and reduce maladaptive signaling

- Gallo, S., El-Gazzar, A., Zhutovsky, P. *et al.* Functional connectivity signatures of major depressive disorder: machine learning analysis of two multicenter neuroimaging studies. *Mol Psychiatry* **28**, 3013–3022 (2023). https://doi.org/10.1038/s41380-023-01977-5

Dopamine effect

- “TMS may influence dopamine indirectly by modulating prefrontal–striatal circuits, with imaging studies showing stimulation-related dopamine release in connected striatal regions. This dopaminergic effect is likely one component of a broader network and plasticity mechanism rather than the sole explanation for antidepressant response.”
- Stimulate prefrontal cortex
- Modulate fronto-striatal circuits
- Trigger downstream dopamine release
- Potentially improve motivation, reward processing, and anhedonia
- Strafella AP, Paus T, Fraraccio M, Dagher A. Striatal dopamine release induced by repetitive transcranial magnetic stimulation of the human motor cortex. *Brain*. 2003;126(Pt 12):2609-2615. doi:10.1093/brain/awg268

Why it matters in depression

Depression is associated with impaired function in networks involving:
prefrontal cortex -limbic system -reward pathways -stress-response circuits

This may reflect reduced ability of circuits to adapt appropriately.

Why it matters in TMS is thought to help by promoting **activity-dependent plasticity**:
strengthening underactive regulatory networks -weakening maladaptive patterns –

improving network efficiency and connectivity

Clinical concept:
TMS may help the brain “retrain” dysfunctional circuits.

Take-Home Message
Synaptic plasticity is the plausible biologic basis for how repeated TMS sessions can produce lasting changes in mood-related brain networks.

Traditional TMS vs SAINT/SNT vs MeRT For Adult Depression / TRD

	Traditional TMS	SAINT / SNT	MeRT
What it is	Standard evidence-based rTMS or iTBS delivered once daily over several weeks	Accelerated, high-dose **iTBS** delivered multiple times per day over a few days, usually with **fcMRI-guided targeting**	Personalized, usually **qEEG-guided** TMS framework that adjusts frequency/targeting based on electrophysiology
Typical target	Usually **left DLPFC**, **right DLPFC**, or bilateral prefrontal targets	Individualized left prefrontal target selected by **functional connectivity** methods	Individualized target and frequency based on **qEEG-derived findings**
Protocol style	Once-daily weekday sessions for **4–6 weeks**	Multiple sessions/day over about **5 days**	6 weeks 5 days a week for 15 minutes
Personalization level	Low to moderate	High	High
Evidence strength	**Strongest and most mature** for routine depression care; guideline and consensus supported	**Promising and growing**, with strong early TRD results, but less mature than standard TMS	**Emerging**; biologically plausible, but less standardized and less validated than traditional TMS
Main advantage	Best-established safety, efficacy, payer familiarity, and clinical standardization	Faster response potential; high remission rates reported in specialized TRD studies	Attempts to match treatment to individual brain physiology
Main limitation	Longer course; some patients respond slowly or incompletely	Resource-intensive; early results may not generalize fully outside specialty centers	Fewer large sham-controlled depression trials; protocol heterogeneity
Best use case	Standard outpatient **MDD / TRD** treatment	**TRD** when rapid response and specialty-center resources are available	Precision-medicine / investigational-leaning settings where individualized EEG-based care is part of the model
	Established standard of care	**Next-generation accelerated**	**Personalized, emerging neuromodulation**

Bottom-line teaching point

- **Traditional TMS** = most established and most defensible as routine standard care.
- **SAINT/SNT** = accelerated, high-dose, connectivity-guided iTBS with promising rapid-response data in TRD.
- **MeRT** = individualized qEEG-guided TMS approach with biologic plausibility, but a less standardized evidence base.

“No single protocol is universally superior for all patients. Traditional TMS remains the best-established depression protocol family;

SAINT/SNT is a promising accelerated precision approach;
MeRT is personalized and emerging rather than definitive.”

“SAINT/SNT is still TMS, but it differs from traditional TMS by using more sessions, more pulses, and more advanced targeting over a compressed time frame.

MeRT differs by emphasizing EEG-based personalization, but its research base is more heterogeneous and should be framed carefully.”

Neurotropic effect

TMS stimulates motor activity

Repeated stimulation engages plasticity pathways

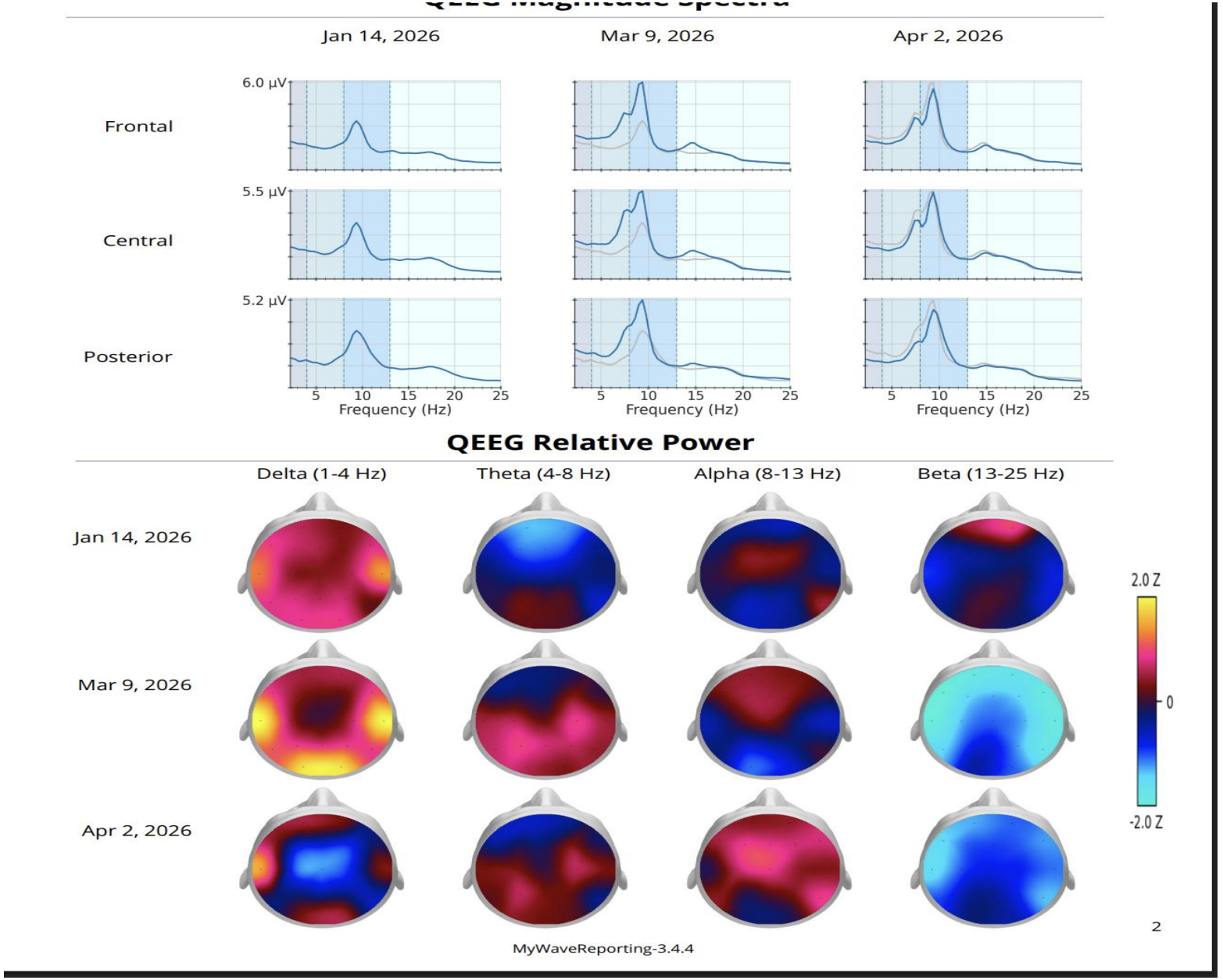
BDNF and related trophic signals may increase

Synapses and networks may reorganize more adaptively

“The neurotrophic effect of TMS refers to its potential to enhance neuroplasticity-related processes, including BDNF-associated signaling, thereby supporting synaptic remodeling and adaptive reorganization of mood-related brain circuits. Evidence is promising but heterogeneous, and BDNF should not yet be treated as a definitive clinical biomarker.”

Asadizeidabadi A, Hosseini S, Pyatkov A. Effects of Repetitive Transcranial Magnetic Stimulation on Tumor Necrosis Factor Alpha in Neuropsychological Disorders: A Systematic Review and Meta-Analysis. *Brain Behav*. 2025;15(2):e70329. doi:10.1002/brb3.70329

qEEG changes



Default Mode Network

network of brain regions active during restful introspection and mind-wandering, involved in self-referential thought.

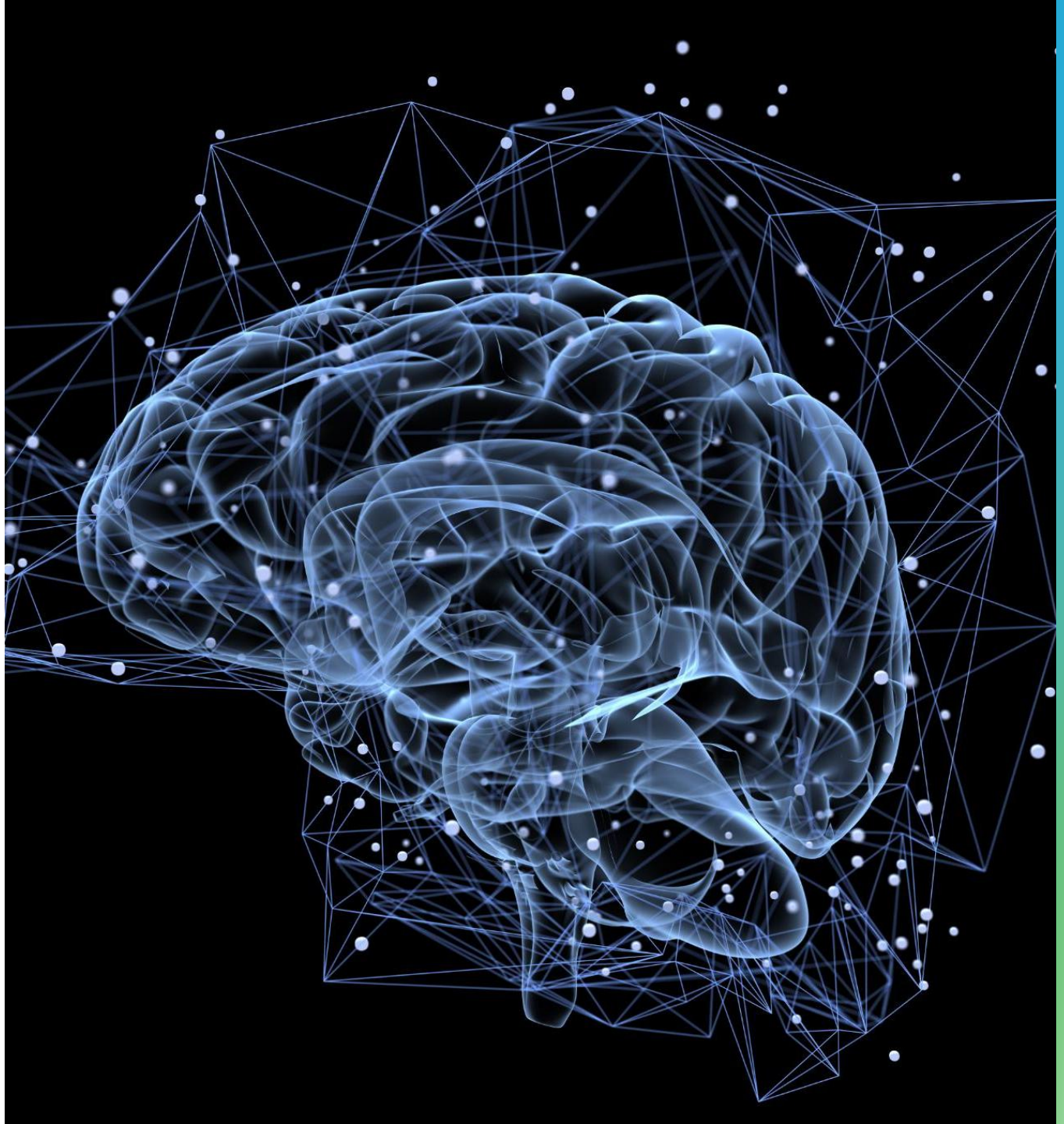
Functions

It **supports internal thoughts,** daydreaming, recalling memories, and envisioning the future, playing a key role in mental health and cognition.

Implications

Understanding the DMN helps in studying mental health conditions like depression and anxiety, and in exploring how the brain transitions between different states.

Azarias FR, Almeida GHDR, de Melo LF, Rici REG, Maria DA. The Journey of the Default Mode Network: Development, Function, and Impact on Mental Health. *Biology (Basel)*. 2025;14(4):395. Published 2025 Apr 10. doi:10.3390/biology14040395



Default Mode Network (DMN) and Why TMS Helps

- **DMN = brain's internal narrative network**
- Active during **self-reflection, memory, mind-wandering**
- Core areas: **mPFC, PCC/precuneus, angular gyri**
- In depression, DMN can become **overactive or poorly regulated**
- This contributes to **rumination, negative self-focus, emotional stuckness**
- TMS stimulates **prefrontal regulatory circuits**
- TMS can shift connectivity between **DLPFC/cognitive control network** and **subgenual cingulate/DMN-related mood circuits**
- Repeated sessions promote **neuroplasticity and healthier network synchronization**
- Clinical result: **less rumination, better focus, improved emotional regulation, better mood**
- Goal is **not to shut off the DMN**, but to **restore balance and flexibility**

- Azarias FR, Almeida GHDR, de Melo LF, Rici REG, Maria DA. The Journey of the Default Mode Network: Development, Function, and Impact on Mental Health. *Biology (Basel)*. 2025;14(4):395. Published 2025 Apr 10. doi:10.3390/biology14040395

Mechanisms of TMS in Depression

Default Mode Network Effects

- Acutely, TMS elicits transient current flow and neuronal depolarization in cortical tissue directly beneath the stimulation site and interconnected circuits
- Repetitive TMS produces longer-lasting effects on neural function via long-term potentiation-like plasticity mechanisms
- These durable effects depend on NMDA receptor signaling
- TMS to the left DLPFC modulates activity in distant brain regions that function abnormally in depression

* Liston C, Chen AC, Zebley BD, et al. *Default mode network mechanisms of transcranial magnetic stimulation in depression. Biol Psychiatry*. 2014;76(7):517-5

Transcranial Magnetic Stimulation (TMS) for Major Depression

Evidence Summary

Landmark Trial

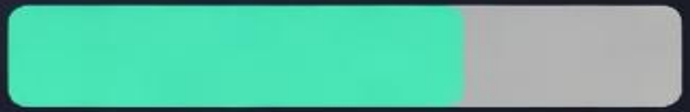
- O'Reardon et al. (2007) - Biological Psychiatry
- Large multicenter RCT
- Significant improvement vs sham

Meta-Analysis

- Wei et al. (2017)
- rTMS + antidepressants
- Effect size:
  SMD = -0.84
  (95% CI -1.19 to -0.48)

Remission Rates

Active rTMS: 36%



Sham: 8%

Important Clinical Note: Patients with mild to moderate depression showed significantly better outcomes

Bottom Line:
Effective non-drug option for treatment-resistant depression

Source rTMS, rTMS, O, TP-WH. Tremstanu Franscranial Magnetic Ssimulatio TMS.+ O, 9, MS, MS, Un, Major eper 201.5, Tramsfor Magnetic Depression, Trastod Depression, Ceyunt, At 1/CP-El Tligeir. 2019.

Depression Severity Predicts rTMS Outcomes

Patients with mild or moderate depression had significantly better outcomes following rTMS

Grammer GG et al. Front Psychiatry 2015;6:114

Safety Profile

- **Overall Safety**
- Well tolerated with minimal adverse effects

- **Common Side Effects**
- Headache
- Scalp discomfort
- **Compared to Medications**
- Fewer systemic side effects
- **Compared to ECT**
- No anesthesia
- No cognitive impairment

Risks

- Seizure risk extremely low
- **Results:** There were no significant differences in seizure frequency in any subject after TMS exposure. There was no occurrence of seizures in healthy individuals, and no worsening of hemiplegic attacks in people with AHC(alternating hemiplegia of childhood) **35 patients**
- Pang S, D'Ambrosio S, Battaglia G, Jiménez-Jiménez D, Perulli M, Silvennoinen K, et al. The impact of transcranial magnetic stimulation (TMS) on seizure course **in people with and without epilepsy**. Clin Neurophysiol Pract. 2022;7:174-182. doi:10.1016/j.cnp.2022.05.005
- **Results:** Studies indicate that TMS is a safe and effective treatment for **MDD during pregnancy**, showing significant reductions in depression scores and increased response and remission rates compared to sham TMS. TMS was well tolerated with minimal side effects. **235articles**
- Angeline S, Tiyatiye B, Akosile W. Transcranial Magnetic Stimulation in Pregnancy: Efficacy, Safety, and Future Implications for Perinatal Mental Health Care. *Brain Behav*. 2025;15(2):e70304. doi:10.1002/brb3.70304

- Stultz DJ, Osburn S, Burns T, Pawlowska-Wajswol S, Walton R. Transcranial Magnetic Stimulation (TMS) **Safety with Respect to Seizures: A Literature Review.** ***Neuropsychiatr Dis Treat*****. 2020**;16:2989-3000. Published 2020 Dec 7. doi:10.2147/NDT.S276635

- "This 2020 literature review by Stultz and colleagues provides a comprehensive look at TMS seizure risk.
- The **overall risk is less than 1%**, and when seizures do occur, they are typically self-limiting and managed with supportive care.
- Importantly, the authors note that this risk is **comparable to — or lower than — many commonly prescribed psychiatric medications**.
- TMS has even been used successfully in higher-risk groups such as patients with epilepsy, traumatic brain injury, and those who had a previous TMS-related seizure.
- Bottom line: While seizure is a rare but serious potential side effect, for patients with treatment-resistant depression, the benefits of TMS often outweigh the risks."

Safety

- The risk of TMS-related seizures is <1% overall.
- TMS has successfully been used in patients with epilepsy, traumatic brain injuries, and those with a prior TMS-related seizure.
- The rate of TMS-related seizures is comparable to that of most psychotropic medications
- While having a seizure is a rare but serious adverse effect of TMS, the benefits of treating refractory depression with TMS may outweigh the risk of suicidal ideation and other significant complications of depression.
- Contraindications are uncontrolled seizures, bipolar, schizophrenia, any metal in the brain or head, cochlear implants, recent stroke or brain bleed. (Not a comprehensive list)

- Stultz DJ, Osburn S, Burns T, Pawlowska-Wajswol S, Walton R. Transcranial Magnetic Stimulation (TMS) Safety with Respect to Seizures: A Literature Review. *Neuropsychiatr Dis Treat*. 2020;16:2989-3000. Published 2020 Dec 7. doi:10.2147/NDT.S276635

NEUROPLASTICITY FOCUS

1. **Core Concept**
2. Depression = impaired neuroplasticity
3. **TMS Role**
4. Theory:Restores plasticity through:
    1. Synaptic strengthening
    2. Network reorganization
5. **Timing Matters**
6. Repeated sessions → cumulative effect

LIMITATIONS / BIAS DISCUSSION

1. **Limitations**
2. Response variability
3. Not all patients respond
4. **Controversies**
5. Some studies suggest variability in effectiveness
6. “TMS demonstrates moderate-to-strong evidence for efficacy; however, heterogeneity in response highlights the need for individualized treatment planning.”

FUTURE DIRECTIONS

1. Future of TMS
2. EEG-guided targeting
3. fMRI guided
4. Accelerated protocols
5. Personalized neuromodulation

Take-Home Points

1. Treatment resistant depression has options
2. Do not just stop at medication
3. Do not just stop at neuromodulation
4. Add lifestyle
5. HOW…

Add Functional Medicine and Lifestyle medicine to TMS

"Goal: Improve terrain for neuroplasticity, autonomic regulation, and mood recovery"

1. REMOVE

1. Reduce factors that may impair neuroplasticity or perpetuate allostatic load
2. Ultra-processed foods, excess alcohol, added sugar
3. Sleep disruption and circadian mismatch
4. Uncontrolled inflammation, pain, toxic stress
5. Medication/substance contributors when clinically relevant
6. Food triggers or GI aggravators in selected patients
7. **Clinical tie-in:**
   Lower inflammatory and physiologic burden may improve tolerance of neuromodulation and reduce barriers to recovery.
8. Phillips C. Lifestyle Modulators of Neuroplasticity: How Physical Activity, Mental Engagement, and Diet Promote Cognitive Health during Aging. *Neural Plast*. 2017;2017:3589271. doi:10.1155/2017/3589271

Restore foundational inputs needed for optimal brain and mitochondrial function

- Protein-forward, whole-food nutrition
- Omega-3 fatty acids
- Micronutrient repletion when indicated:
  - vitamin D
  - B12/folate
  - magnesium
  - iron if deficient
- Sleep-supportive behaviors
- Morning light exposure and movement
- **Clinical tie-in:**
  Neurons require adequate substrate, cofactors, and circadian signaling for adaptive synaptic change.

- Das S, Banerjee P, Jana S, Mondal H. Unveiling the mechanistic nexus: how micronutrient enrichment shapes brain function, and cognitive health. *Front Mol Biosci*. 2025;12:1623547. Published 2025 Sep 23. doi:10.3389/fmolb.2025.1623547

Support gut-brain signaling and microbial diversity

- Fiber diversity and plant variety
- Fermented foods as tolerated
- Targeted probiotics/prebiotics when appropriate
- Constipation/diarrhea management if present
- Consider microbiome-directed interventions in selected patients
- **Clinical tie-in:**
  A healthier gut ecosystem may support vagal signaling, immune regulation, and neurotransmitter precursor availability.

- Appleton J. The Gut-Brain Axis: Influence of Microbiota on Mood and Mental Health. *Integr Med (Encinitas)*. 2018;17(4):28-32.

Promote integrity of barriers, recovery systems, and cellular resilience

1. Address gut barrier stressors where clinically relevant
2. Anti-inflammatory nutrition pattern
3. Exercise as tolerated
4. Stress reduction / mindfulness / breathwork
5. Support pain recovery and restorative sleep
6. **Clinical tie-in:**
   Repairing physiologic “terrain” may support autonomic flexibility and improve the brain’s capacity for plastic change.
7. https://my.clevelandclinic.org/health/body/the-gut-brain-connection

Rebuild adaptive rhythms and network regulation

1. TMS to modulate fronto-limbic circuitry
2. Behavioral activation
3. Psychotherapy / trauma-informed care
4. HRV-informed autonomic training where available
5. Consistent routines for sleep, meals, movement, and recovery
6. **Clinical tie-in:**
   TMS provides targeted cortical input, while lifestyle and systems interventions help stabilize the broader biologic environment that supports durable change.

7. Eldaief MC, Press DZ, Pascual-Leone A. Transcranial magnetic stimulation in neurology: A review of established and prospective applications. *Neurol Clin Pract*. 2013;3(6):519-526. doi:10.1212/01.CPJ.0000436213.11132.8e

Educational note:

This integrated model is biologically plausible and clinically appealing, but evidence for combined functional medicine + TMS protocols in depression remains emerging.

These strategies can be individualized and used as adjuncts to guideline-based care.

Caveat

- “Depression is not only a neurotransmitter disorder; it is often a systems disorder involving inflammation, circadian disruption, autonomic imbalance, pain, poor substrate delivery, and impaired neuroplasticity.
- TMS targets brain networks directly, while the 5R framework may be a useful adjunct to improve the physiologic terrain that supports adaptive recovery.”


Depression is like the Grand Canyon , it is the Grand Canyon from any direction you look at it , and it is ready to be explored!

Objectives Overview

Define Goals

IDENTIFY patients who will benefit from TMS

Set Metrics Transcranial Magnetic Stimulation (TMS) in Treatment-Resistant Depression
Mechanisms, Evidence, Safety, and Clinical Application

Align Strategies

ABSOLUTE CONTRAINDICATIONS

TMS is contraindicated if the answer to any of the below questions is YES.

1.Does the patient have a cochlear implant?

2. Does the patient have a cortical stimulator?

3 Does the patient have a deep brain stimulator?

4 Does the patient have a ventriculoperitoneal shunt?

5. Does the patient have a skull defect related to their current admission?

6. Has the patient experienced a prior TMS-related serious adverse event?

7. Has the patient had a seizure in the last 12 months while taking anti-epilepsy medication?

8. Has the patient experienced seizures related to their current admission?

safety