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Transcranial Magnetic Stimulation (TMS)

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Objectives

- Participants will be able to:

1. Describe the basic principles of Transcranial Magnetic Stimulation (TMS)
2. Identify the clinical indications of TMS.

TMS Consult Case

- 21 year old college student referred for TMS consult
- Dx: Major depressive disorder since high school; recently diagnosed to have obsessive-compulsive disorder.
- Medications tried: 3 antidepressants
- Therapy received: cognitive behavior therapy; started Exposure and Response Prevention (ERP therapy)
- Patient: “Should I do TMS for my depression or for my OCD?”

Neuropathology of Depression

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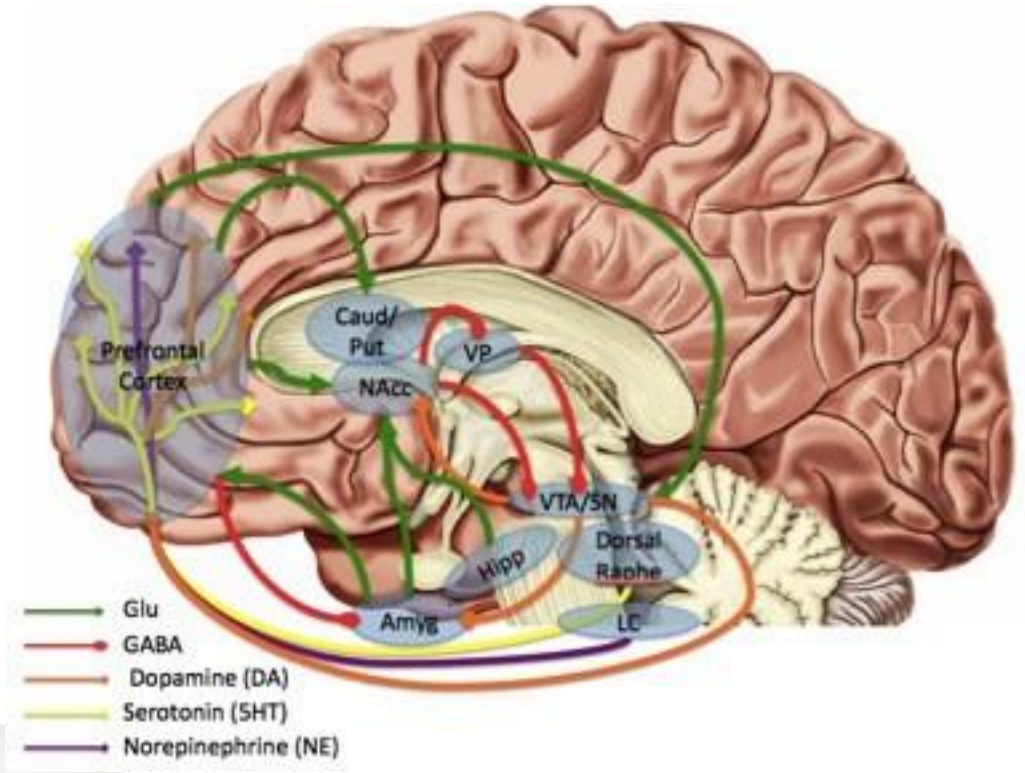
Regions, Transmitters and Circuits in Major Depressive Disorder



Regions, transmitters and circuits implicated in the pathology of major depressive disorder (MDD) by human neuroimaging studies. Past studies have identified alterations in monoamine levels and receptor availability as well as alterations in glutamate and GABA. These neurotransmitter systems participate in larger circuits involved in the experience and regulation of emotion, responses to stress, and processing of rewards. Note: placement of structure labels is approximate. Amyg = amygdala; Caud = Caudate; GABA = GABAergic projections; Glu = glutamatergic projections; Hipp = hippocampus; NAcc = nucleus accumbens; Put = Putamen; SN = substantia nigra; VP = ventral pallidum; VTA = ventral tegmental area. Republished with permission from Treadway and Zald [49].

Treadway, Michael & Pizzagalli, Diego. (2014). Imaging the pathophysiology of major depressive disorder - from localist models to circuit-based analysis. Biology of mood & anxiety disorders. 4. 5. 10.1186/2045-5380-4-5.

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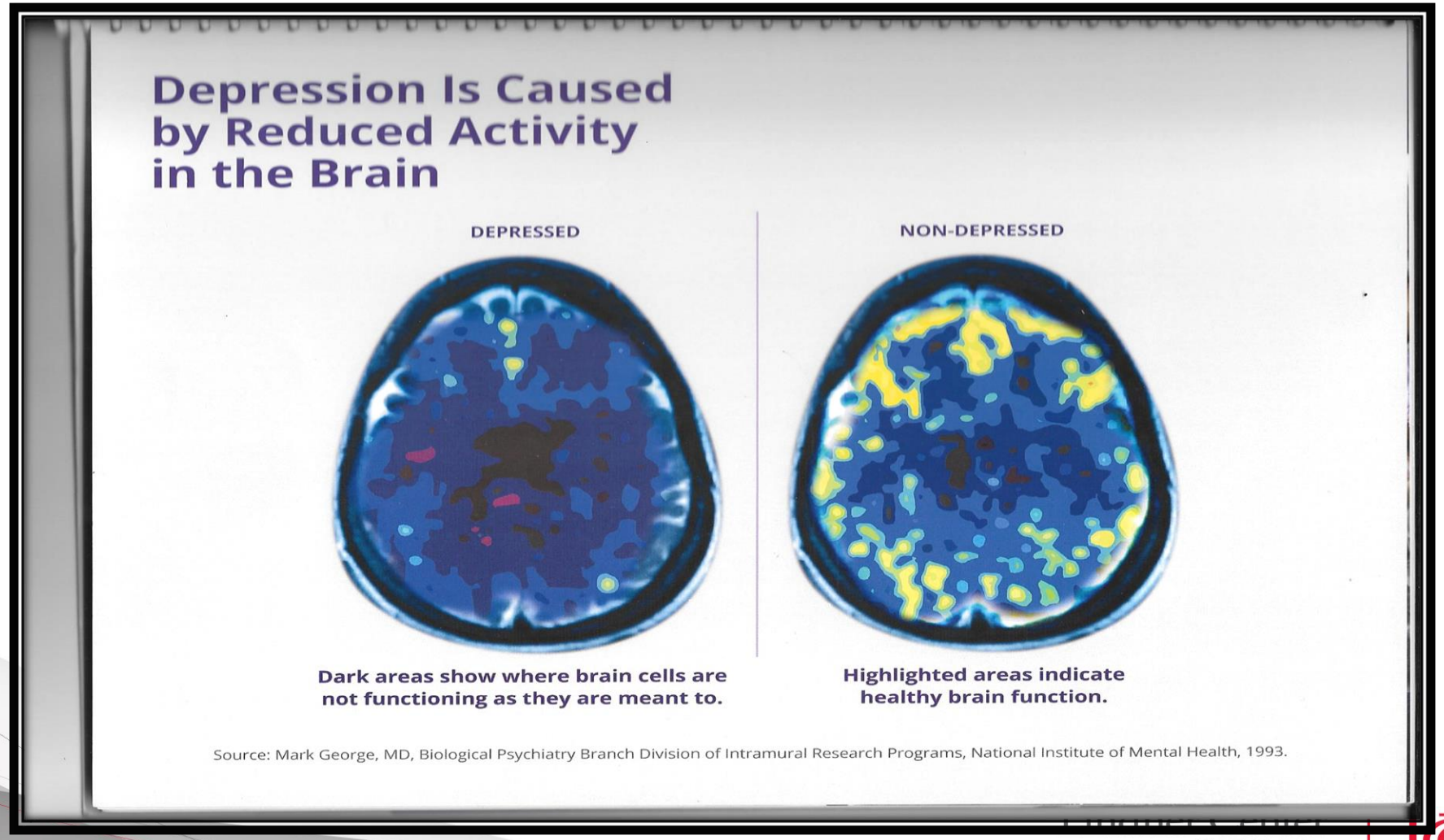
Neuropathology of Depression



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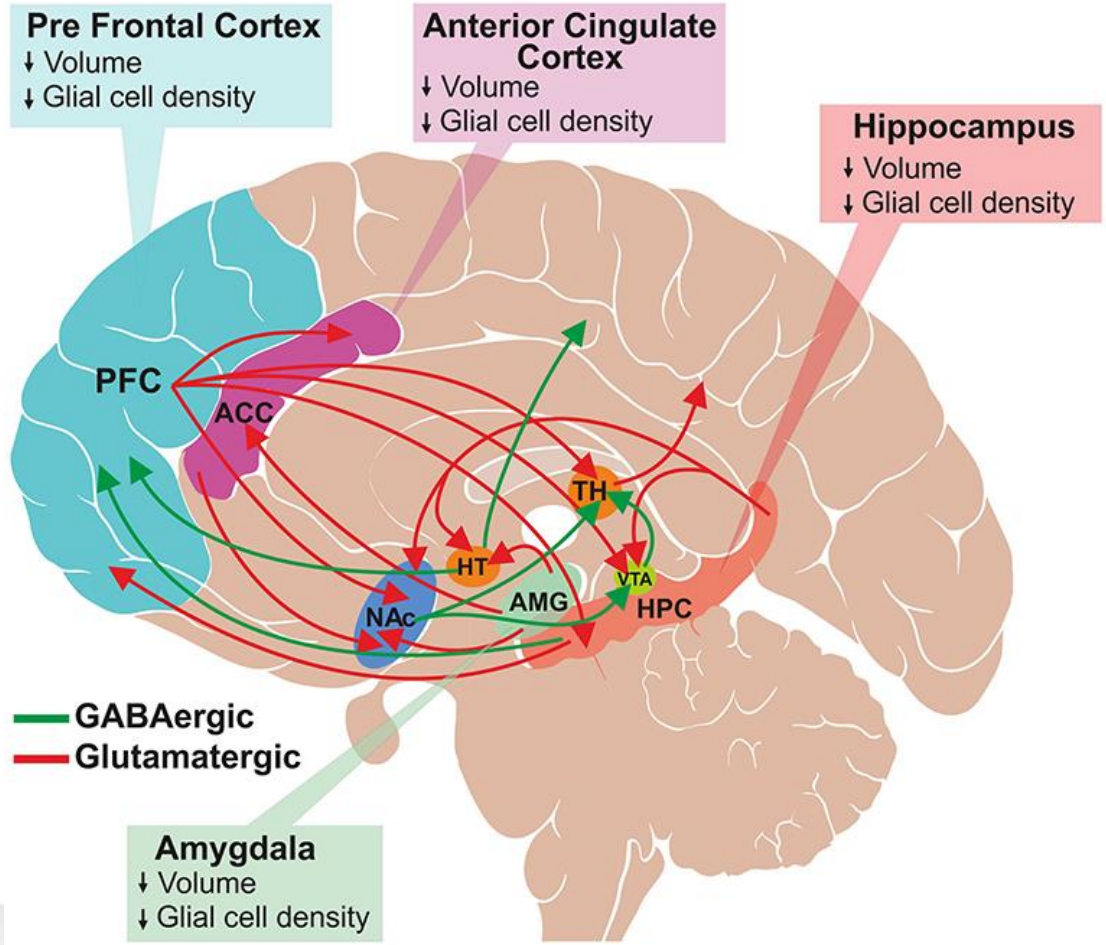
Glutamate and GABA Homeostasis and Neurometabolism in Major Depressive Disorder (Frontier)



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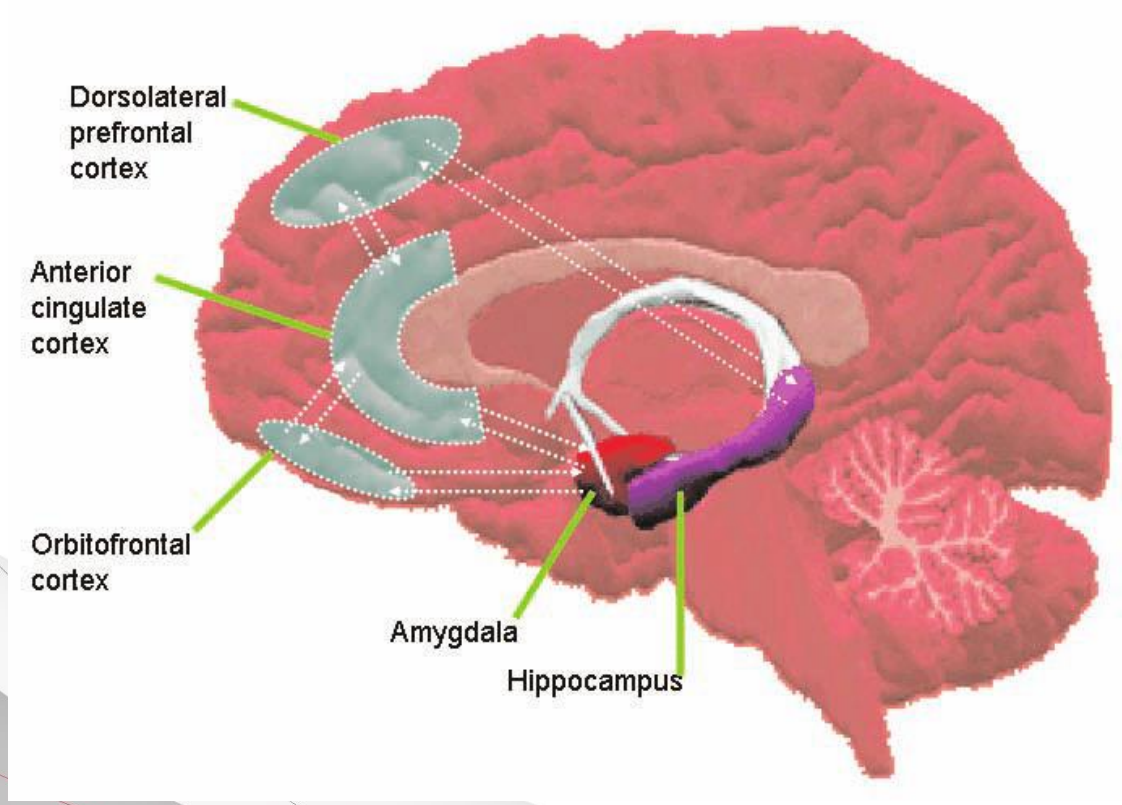
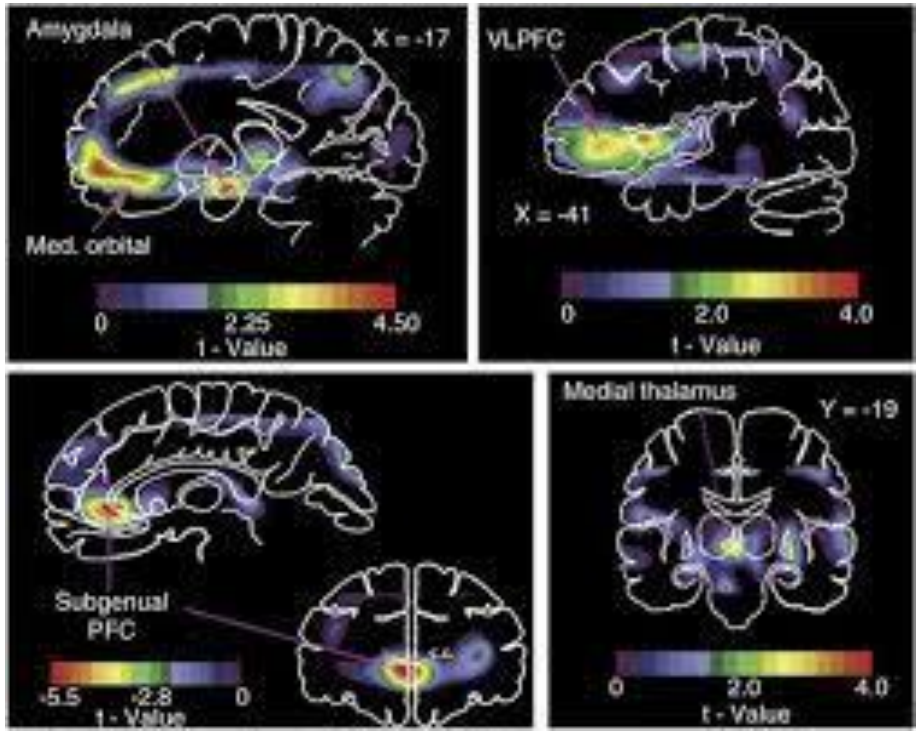
Neurocircuitry of Mood Disorders

(Source: Nature and ResearchGate)





Price, J., Drevets, W. Neurocircuitry of Mood Disorders. *Neuropsychopharmacol* **35**, 192–216 (2010). https://doi.org/10.1038/npp.2009.104

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Triple Network Hypothesis of Psychopathology

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Functional Network Modulation in TRD

(Idlett-Ali, S, Salazar C, Bell M, Short EB, Rowland N; Frontiers in Human Neuroscience, 2023)

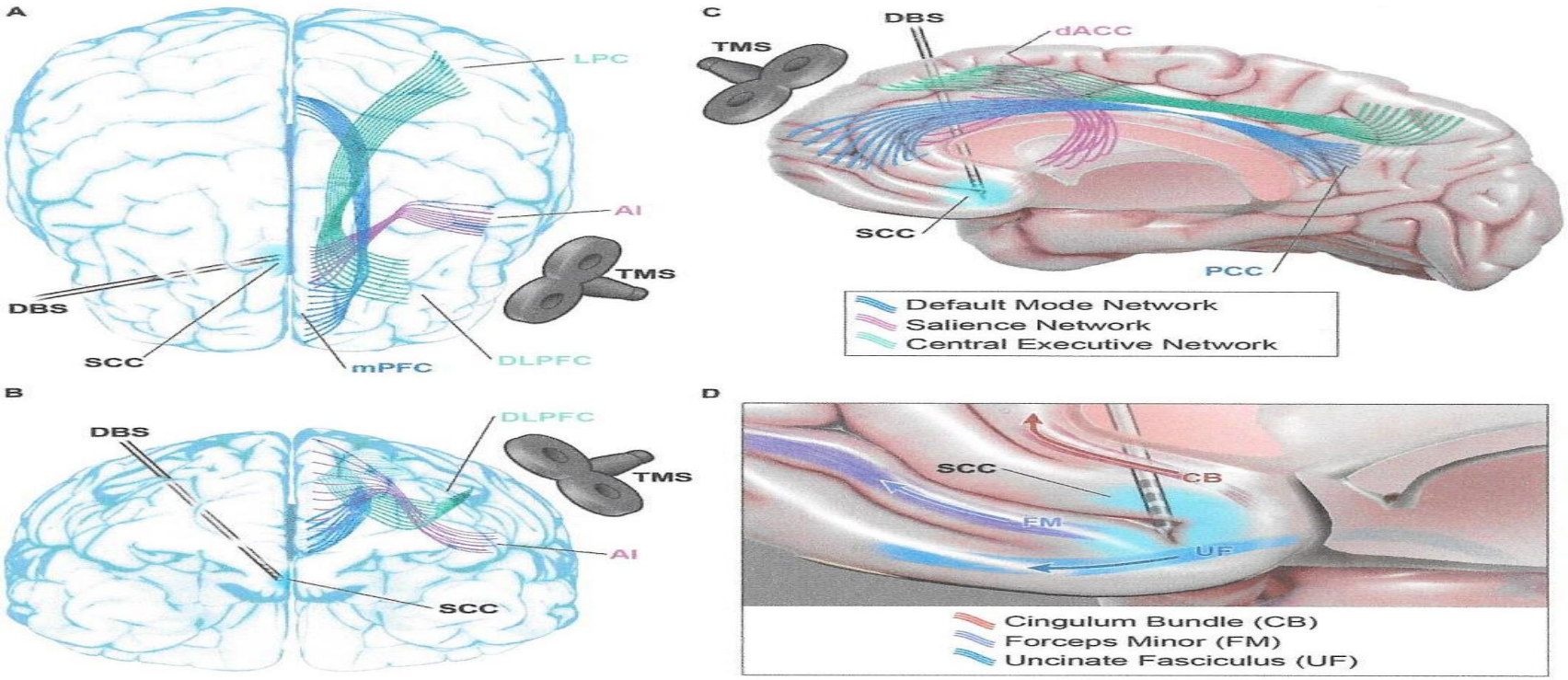

FIGURE 2
Functional network modulation in TRD. Default mode network (DMN – blue), salience network (SN – purple), and central executive network (CEN – green) are accessible for modulation with transcranial magnetic stimulation (TMS) and deep brain stimulation (DBS). Dorsal lateral prefrontal cortex (DLPFC) TMS may modulate CEN via direct modulation of DLPFC and its projections to lateral parietal cortex (LPC) **(A–C)**. DLPFC TMS could indirectly modulate SN via functional connections with anterior cingulate cortex (ACC), anterior insula (AI), or DMN via functional connections with medial prefrontal cortex (mPFC) and posterior cingulate cortex (PCC). DBS at the intersection of subcallosal cingulate cortex (SCC), the cingulum bundle (CB), uncinate fasciculus (UF), and forceps minor (FM) may modulate SN via projections to dorsal anterior cingulate cortex (dACC) and AI **(C,D)**. Functional connections to DLPFC, mPFC, and PCC also present avenues for modulation of CEN and DMN. Please see **Table 1** for full listing of network structures.

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Historical Background

- 1985: Dr. Anthony T. Barker, PhD – pioneered in developing transcranial magnetic stimulation (TMS).
- Barker AT, Jalinous R, Freeston IL. *Non-invasive stimulation of the human motor cortex.* Lancet 1985:1:1106-7.
- Dr Barker wins First International Brain Stimulation Award, Elsevier, 2016.

Historical Background

- 1997: TMS was approved by Health Canada;
  - then in 2002, it was approved for treatment resistant depression.
- 2008: US FDA approved TMS for major depressive disorder with the Neurostar TMS device.
  - Dr Mark S. George, M.D. , Medical University of South Carolina, pioneered use of TMS in the US, as early as 2000.
  - **2010: LCOH started using Neurostar TMS (Neuronetics)**
  - 2012: European Union approved TMS for MDD
- 2013: Brainsway was given FDA approval for its novel, patented H-coil deep TMS device.

Historical Background

- 2013: US-FDA expanded use of TMS for treating pain associated with certain migraine headaches.
- 2018: US-FDA approved use of TMS for treatment of Obsessive Compulsive Disorder (OCD)
- 2018- US-FDA approved intermittent theta-burst stimulation (iTBS) as a new variant of TMS for the treatment of TRD (Mendlewitz, et al, 2019)
- 2022: FDA cleared Accelerated TMS protocol for depression. **S**tanford **A**ccelerated **I**ntelligent **N**euromodulation **T**herapy (**SAINT Depression Protocol**).
  - Dr. Nolan Williams, M.D at Stanford University- pioneer

TMS for Adolescents

- March 27, 2024:
  - *Neuronetics (Neurostar TMS) received 510 (K) clearance to use its Transcranial Magnetic Stimulation (TMS) device as an adjunct to treat adolescents (ages 15-21 years) with major depressive disorder (MDD).*
- 510 (K) FDA Clearance
  - *510 (K) refers to the section of the Food, Drug and Cosmetic Act*
  - *Purpose of 510(K) submission is to prove that your device is safe and effective by comparing it to a similar, legally marketed device ("predicate device").*
- Efficacy
  - *Among **1169 adolescents** analyzed through NeuroStar's TrakStar platform, **78%** showed clinically meaningful improvement in depression severity.*
    - *Accessed online, Psychiatric Times, and FDA website, 04/18/2024*

Transcranial Magnetic Stimulation

Magnetic Coils

- Circular
- Figure of 8
- Double Cone (Neurostar)
- H - shaped (Brainsway)

Pulse

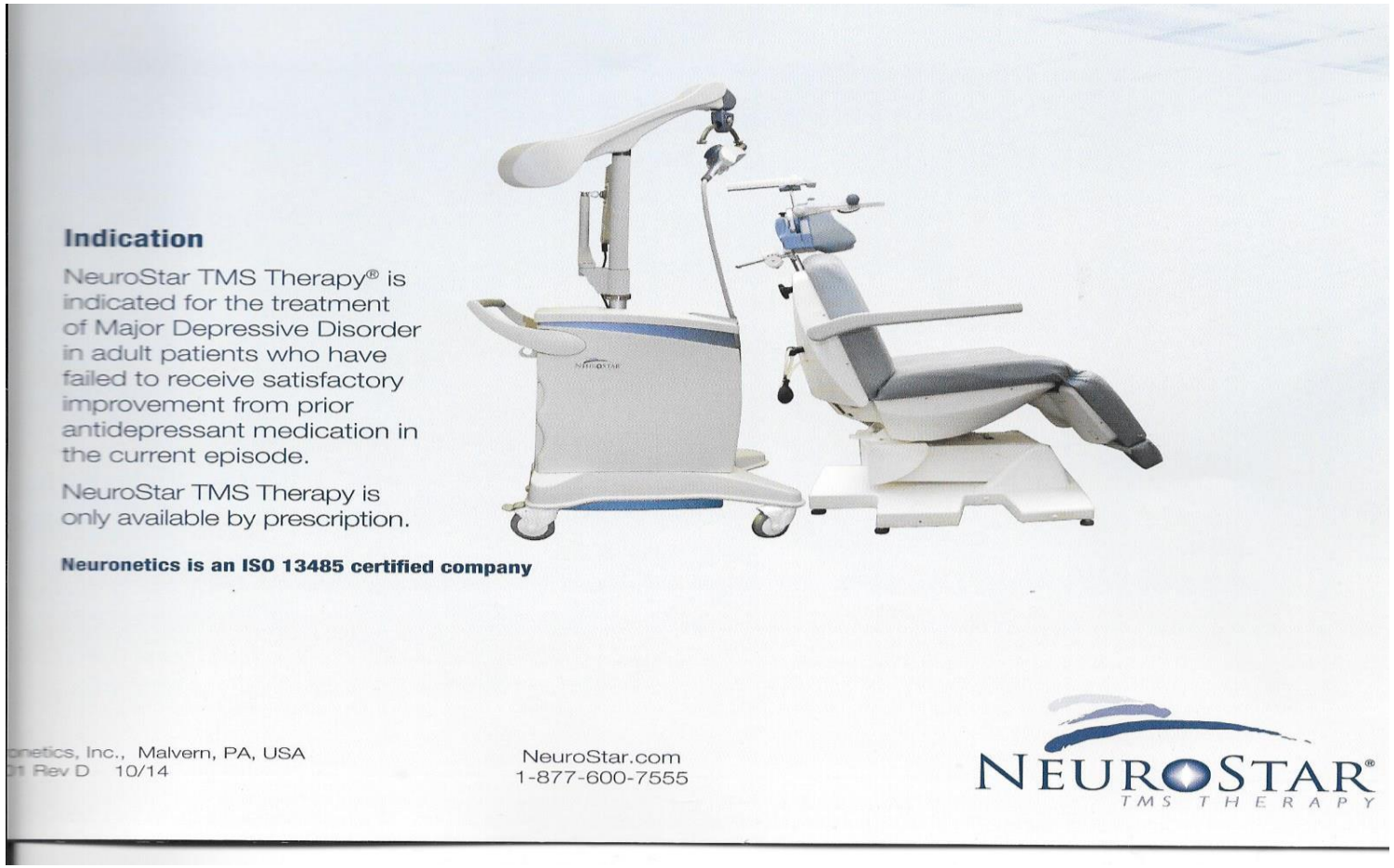
- Single-pulse TMS
- Paired-pulse TMS
- **Repetitive-pulse TMS (rTMS)**

Types of TMS

- **Low Frequency rTMS**
  - Frequency </= 1 Hz (decreases cortical excitability)(Chen, Pascual-Leone)
- **High Frequency rTMS**
  - Frequency >/= 5 (5-20)Hz (increase cortical excitability)
- **Theta-Burst Stimulation (TBS) TMS**
  - Intermittent Theta Burst (excitatory effects)
    - Bursts of three pulses at a frequency of 50 Hz every 200 ms; at 5 Hz, delivery is over 2 sec and repeated every 10 sec, 20 times in succession)
  - Continuous Theta Burst (inhibitory effects)

Transcranial Magnetic Stimulation for Depression

- Procedure
- Safety
- Efficacy



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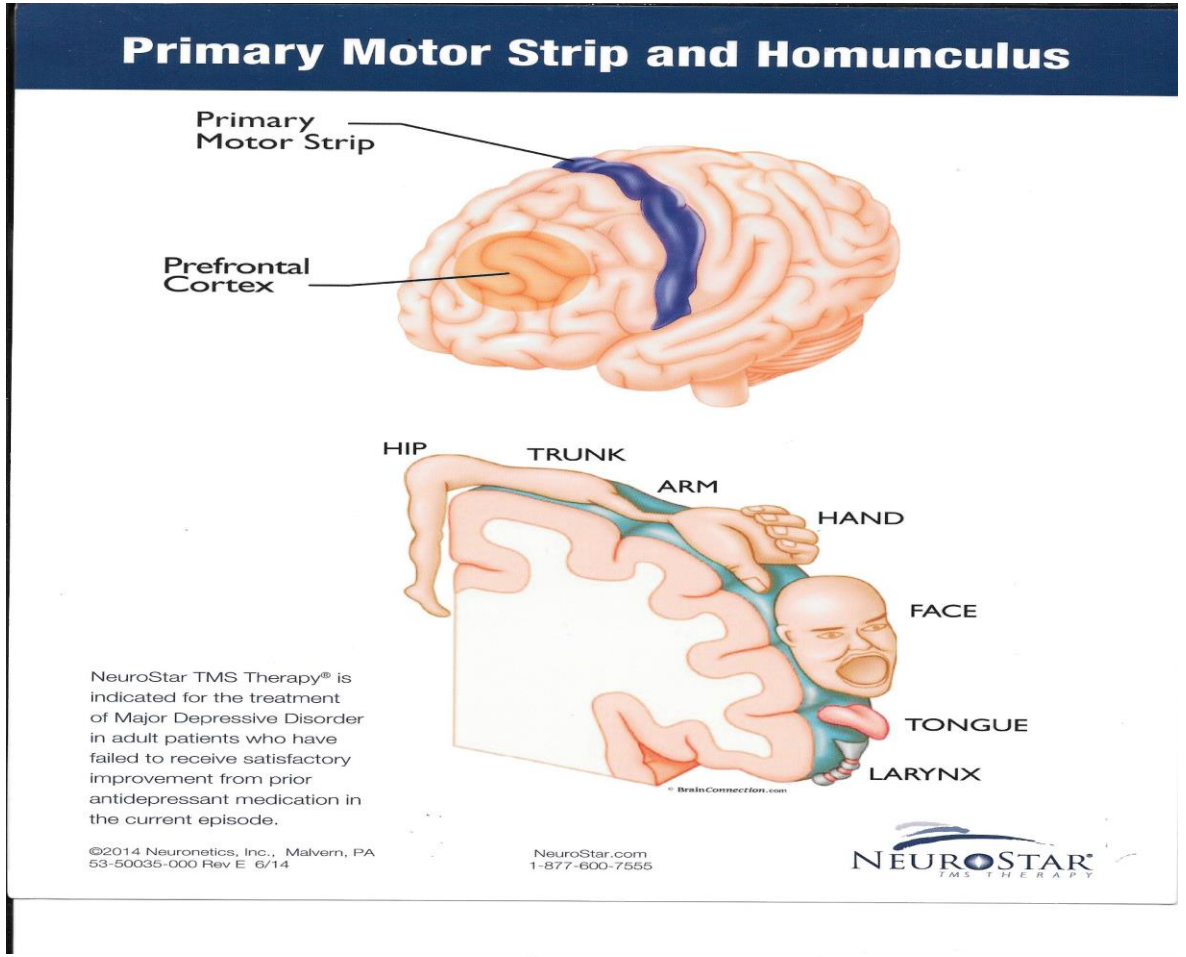
TMS Procedure

- **1. First Session:**

Initial TMS session:

Motor Threshold (MT) ***Mapping*** and ***Determination***



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TMS Procedure

- **TMS Treatment Sessions**
  - High-Frequency, Repetitive
  - Duration: 18-20 minutes
  - Daily treatments: 5 days a week
  - Total Treatments: 30-36 sessions

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Basic Principles of Transcranial Magnetic Stimulation

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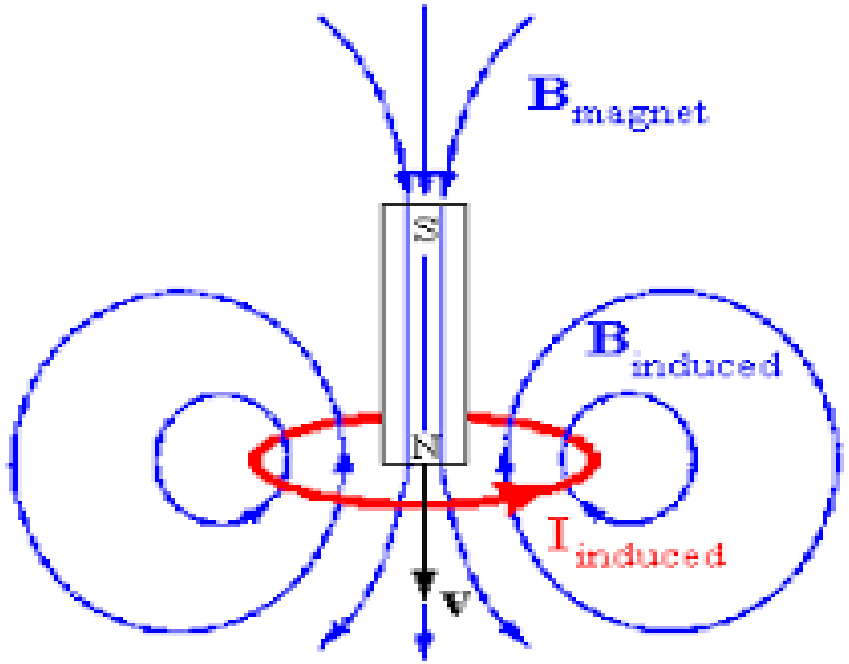
Faraday's Law of Electromagnetic Induction

Faraday's Law

Faraday's law of induction is a basic **law** of electromagnetism predicting how a magnetic field will interact with an electric circuit to produce an electromotive force (EMF)—a phenomenon called electromagnetic induction.



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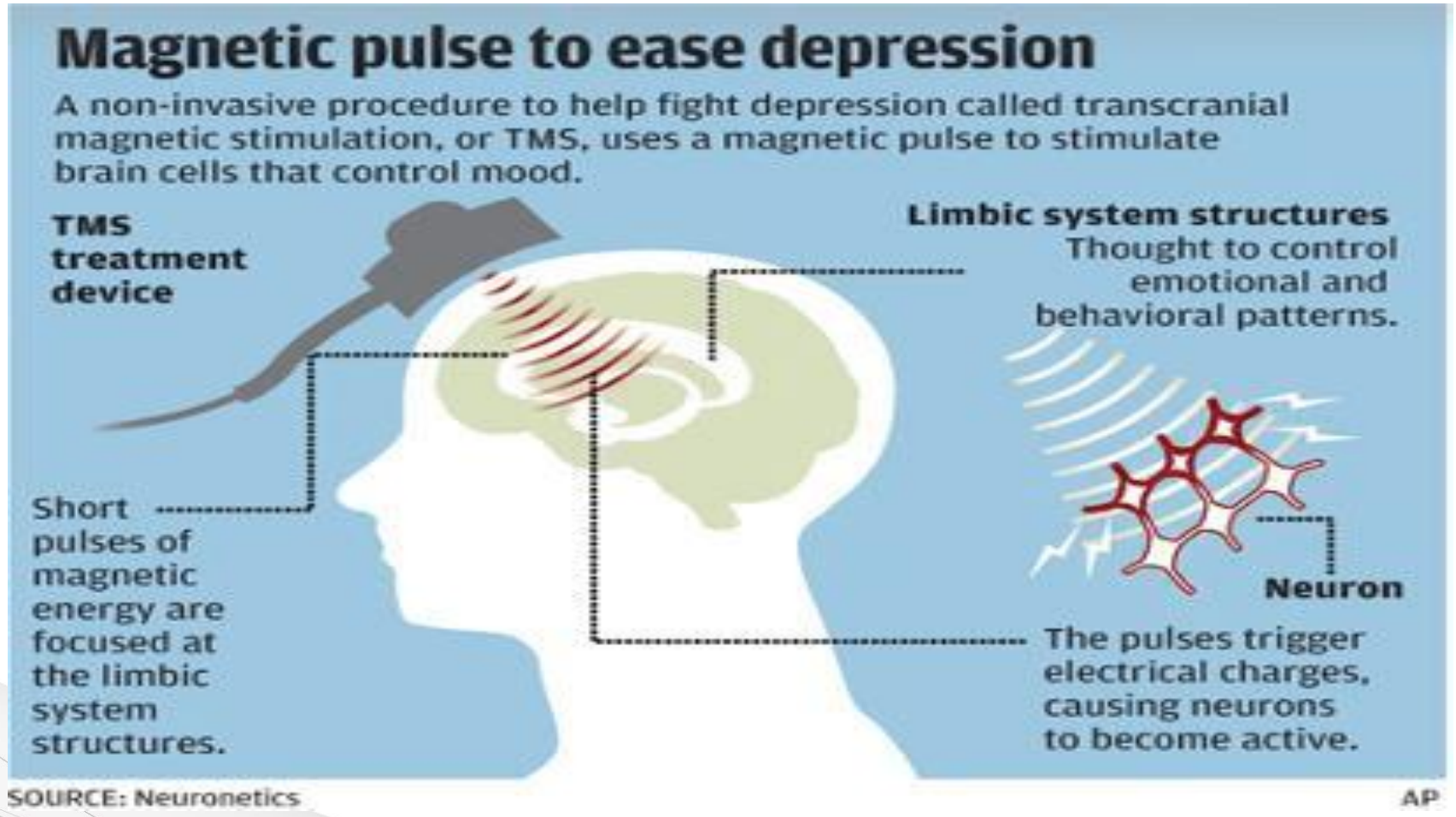
Transcranial Magnetic Stimulation



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Spatial Distribution of Current Density of rTMS

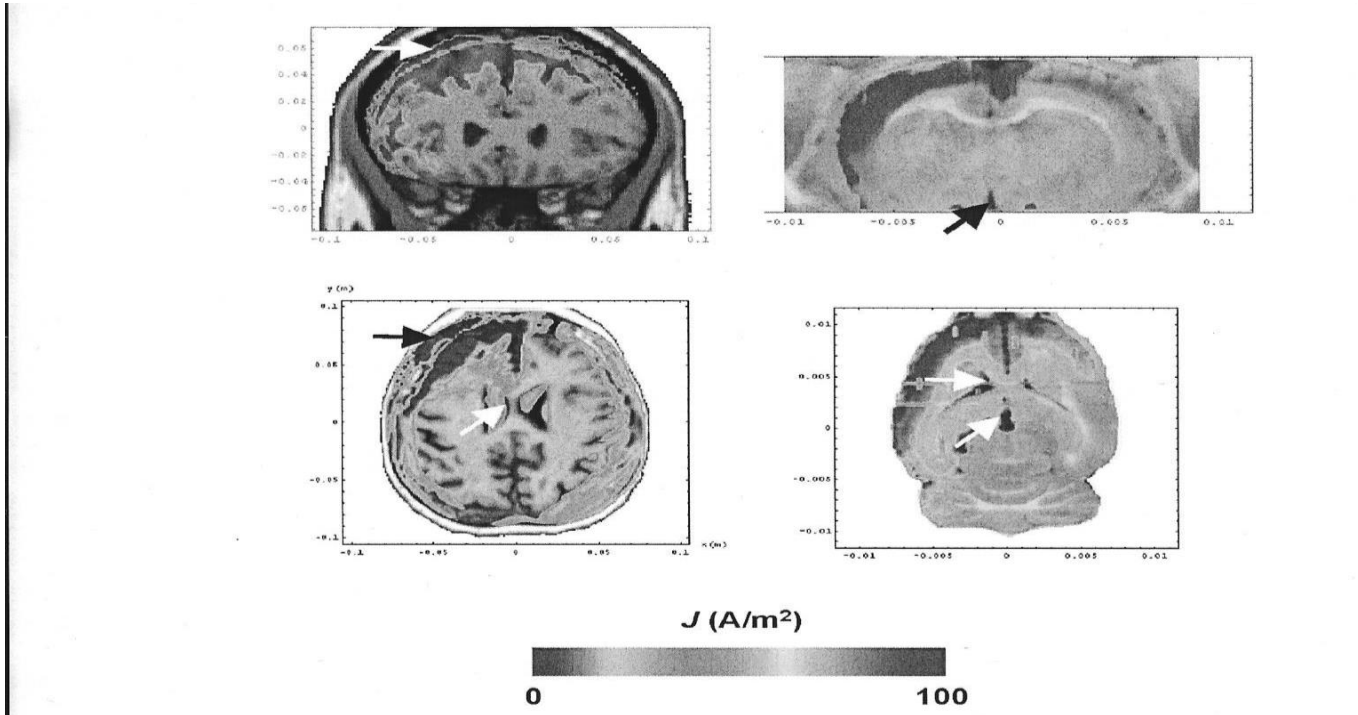

Fig. 1. Spatial distribution of current density (density plot) induced in one coronal layer (upper panel) and one transversal layer (lower panel) o human (left panel) and rat brain (right panel) by rTMS. The electrical characteristics of the brain are reconstructed from MRI images, whereby th conductivity distribution of the tissue is mapped onto the anatomical map of the brain. *Human:* Maximum coil current intensity I_{max} = 4000 A. Puls

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Unique Mechanism of Action of rTMS

The TMS Process

1. Precise pulsed magnetic fields induce small electric currents in the prefrontal cortex of the brain

2. Local neurons depolarize, which leads to activation of deep brain structures via trans-synaptic pathways

3. Activation of these pathways in the limbic system leads to the release of neurotransmitters

4. Blood flow and glucose metabolism rise in the activated regions, which is thought to result in improved mood

Functional Connectivity Changes by rTMS

Neuroimaging studies have documented changes in functional connectivity between tissue directly stimulated by NeuroStar TMS and in deep brain regions known to be involved in mood regulation[4]

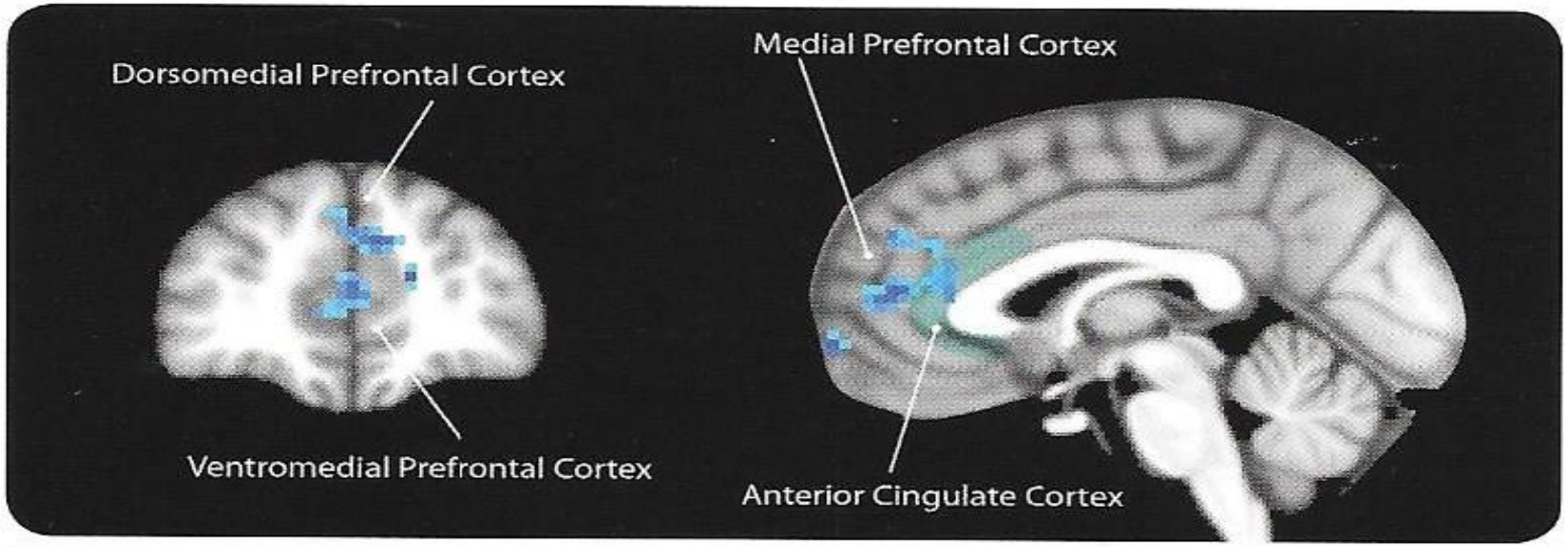

Figure reproduced with permission of MJ Dubin, MD, PhD, Weill Cornell Medical College

Neurobiological Mechanisms of rTMS

(Post A, Keck M.; J Psych Research 35 2001)

PERGAMON

Journal of Psychiatric Research 35 (2001) 193–215

JOURNAL OF PSYCHIATRIC RESEARCH

www.elsevier.com/locate/jpsychires

Transcranial magnetic stimulation as a therapeutic tool in psychiatry: what do we know about the neurobiological mechanisms?

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Max Planck Institute of Psychiatry, Kraepelinstrasse 2-10, D-80804 Munich, Germany

Abstract

Potential therapeutic properties of repetitive transcranial magnetic stimulation (rTMS) have been suggested in several psychiatric disorders such as depression, mania, obsessive-compulsive disorder, posttraumatic stress disorder and schizophrenia. By inducing electric currents in brain tissue via a time-varying strong magnetic field, rTMS has the potential to either directly or trans-synaptically modulate neuronal circuits thought to be dysfunctional in these psychiatric disorders. However, in order to optimize rTMS for therapeutic use, it is necessary to understand the neurobiological mechanisms involved, particularly the nature of the changes induced and the brain regions affected. Compared to the growing number of clinical studies on its putative therapeutic properties, the studies on the basic mechanisms of rTMS are surprisingly scarce. rTMS currently still awaits clinical routine administration although,there is compelling evidence that it causes changes in neuronal circuits as reflected by behavioural changes and decreases in the activity of the hypothalamic-pituitary-adrenocortical system. Both alterations suggest regional changes in neurotransmitter/neuromodulator release, transsynaptic efficiency, signaling pathways and in gene transcription. Together, these changes are, in part, reminiscent of those accompanying antidepressant drugs.

Keywords: Repetitive transcranial magnetic stimulation; Stress; Depression; Antidepressant; HPA system; ACTH; BDNF; Dopamine; Neuroprotection; Vasopressin

1. Introduction

1.1. Physical background and historical overview

Transcranial magnetic stimulation (TMS) was introduced in 1985 (Barker et al. 1985) as a neurological technique for non-invasively inducing motor movement by direct magnetic stimulation of the brain's motor cortex to measure connectivity and excitability (e.g. Curt et al., 1998; Keck et al., 1998; review: Rossini and Rossi, 1998; Hallett, 2000). It depends on the basic principle of mutual induction, discovered by Michael Faraday in 1831 (Faraday, 1831), whereby electrical fields E can be converted into magnetic fields B, and magnetic fields B can be converted into electrical energy. In the case of TMS, a brief surge of current flows through the stimulation coil to produce a transient magnetic field B. This field passes freely into the surrounding medium and induces an electric field E which impedes the magnetic field. If the electric field E falls in a conductor (i.e. brain tissue), then current will flow (Figs. 1 and 4). The ability of this current to painlessly excite nerve cells depends upon its time course, magnitude and direction. It is important to note that the effects obtained by use of TMS do not occur on the basis of the magnetic field applied but are achieved by the electric field induced that ultimately leeds to neuronal depolarization. Charge is moved across the excitable neuronal membranes, creating a transmembrane potential. If sufficient, this causes membrane depolarization and initiates an action potential, which then propagates along the nerve. In contrast to the direct transcranial electrical currents used in electroconvulsive therapy, magnetic fields are unaffected by the high impedance of the skull. Thus, TMS can stimulate the cerebral cortex relatively painlessly in awake patients (Barker et al., 1985).

The idea that nerve cells could be excited indirectly by magnetic fields via the principle of mutual induction is not new as already in 1896 d'Arsonval reported to the Société de Biologie in Paris that when a subject's head was placed in a strong time-varying magnetic field (110

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0022-3956/01/$ - see front matter
PII: S0022-3956(01)00023-1

- 27 studies (rats) dealing with behavioral and neurochemical effects of TMS
  - **Changes in stress coping –" higher"**
  - **Attenuation of the stress-induced activity of the HPA system**
    - Higher taurine level; lower **vasopressin** level
    - ACTH
  - **Monoamines**
    - Increase hippocampal **dopamine** and **serotonin**
  - **Neuroprotective effect of TMS**
    - Increase **secreted amyloid precursor protein** (s-APP); and **brain-derived neurotrophic factor** (BDNF)

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Neuroplasticity

- Hebbian Theory:
  - **Donald Hebb**, 1949 book *The Organization of Behavior*
  - **Hebb's Rule:** "When an axon of cell A is near enough to excite cell B and repeatedly and persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased."
- Spike-timing-dependent-plasticity (STDP)

Bienenstock-Cooper-Munro Theory (1982)

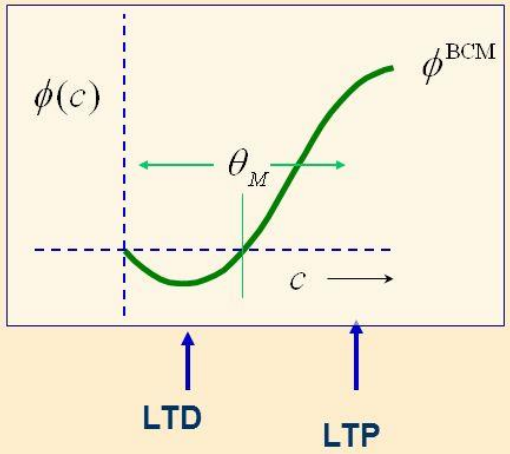
BCM Theory

(Bienenstock, Cooper, Munro 1982;
Intrator, Cooper 1992)

$$\frac{dm_j}{dt} = \eta d_j \phi(c, \theta_M)$$

$$\theta_M \propto E[c^2] =$$

$$\frac{1}{\tau} \int_{-\infty}^{t} c^2(t) e^{-(t-t')/\tau} dt$$



Requires

- Bidirectional synaptic modification LTP/LTD
- Sliding modification threshold
- The fixed points depend on the environment, and in a patterned environment only selective fixed points are stable.

- **BCM Theory of synaptic modification** postulates that:
  - **Long term depression (LTD)** is facilitated by high levels of preceding postsynaptic activity;
  - **Long term potentiation (LTP)** is facilitated by low levels of recent postsynaptic activity.

Blais BS, Cooper L, BCM Theory (2008), Scholarpedia 3(3):1570.

doi:10.4249/scholarpedia.1570

Metaplasticity

- Metaplasticity
  - Is a broad term encompassing a series of endogenous neural processes linked to activity-dependent synaptic plasticity.
  - Also refers to changes occurring on longer time scales (hours-to-weeks) possibly via a subtle morphological synapto-dendritic changes.

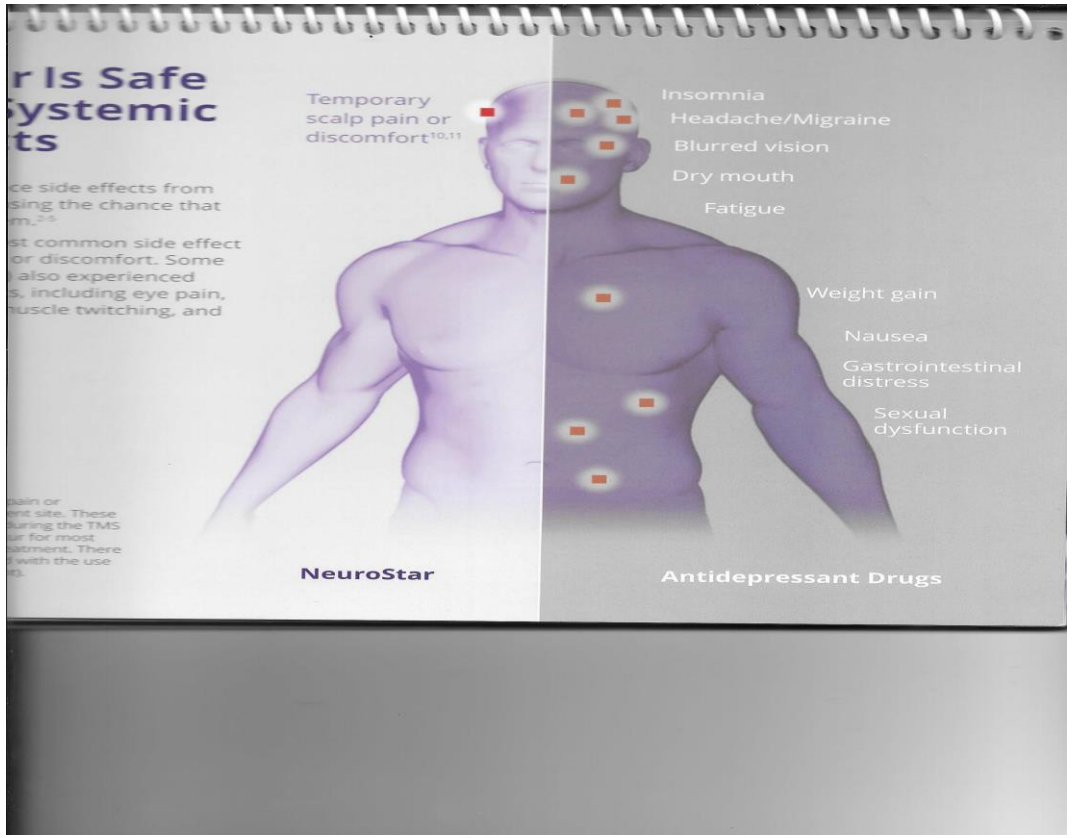
- Cocchi L, Zalesky A, et al, Transcranial magnetic stimulation in obsessive-compulsive disorder: A focus on network mechanisms and state dependence, *Neuroimage: Clinical* 2018; 19: 661-674

TMS Safety

Side-effects:

- Most Common:
  - **Local discomfort**
  - **Headache**
- Rare:
  - Seizure
- Contraindication:
  - Ferro-metallic object

TMS vs Antidepressants



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TMS Safety

2009

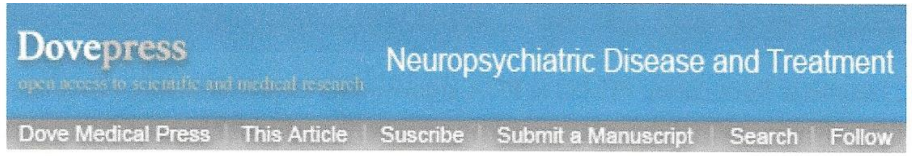
- **The Safety of TMS Consensus Group**
- Rossi, Simone, Hallett, M, Rossini, P, Pascual-Leone, A, and The Safety of TMS Consensus Group

- Safety, ethical consideration, and application guidelines for the use of transcranial magnetic stimulation in clinical practice and research.
- *Clin Neurophysiol. Dec 2009, 120(12): 2008-2039*

2021

- **Safety and recommendations for TMS use in healthy subjects and patient populations, with updates on training, ethical and regulatory issues: Expert Guidelines**
- Rossi, S, Antal, A, Bestmann S , et al
- *Clin Neurophysiol. Jan 2021: 132(1): 269-306*
- Consensus Statement from the International Federation of Clinical Neurophysiology (IFCN) Workshop on "*Present, Future of TMS: Safety, Ethical Guidelines*", Siena, Italy, Oct 17-20, 2018

TMS Safety: Risk of Seizure



Neuropsychiatr Dis Treat. 2020; 16: 2989–3000.
Published online 2020 Dec 7. doi: 10.2147/NDT.S276635
PMCID: PMC7732158
PMID: 33324060

Transcranial Magnetic Stimulation (TMS) Safety with Respect to Seizures: A Literature Review

Debra J Stultz,[1] Savanna Osburn,[1] Tyler Burns,[1] Sylvia Pawlowska-Wajswol,[1] and Robin Walton[1]

Abstract

Transcranial magnetic stimulation is an increasingly popular FDA-approved treatment for resistant depression, migraines, and OCD. Research is also underway for its use in various other psychiatric and medical disorders. Although rare, seizures are a potential adverse event of TMS treatment. In this article, we discuss TMS-related seizures with the various coils used to deliver TMS, the risk factors associated with seizures, the differential diagnosis of its presentations, the effects of sleep deprivation and alcohol use on seizures, as well as seizure risks with protocols for traditional TMS, theta-burst stimulation, and accelerated TMS. A discussion is presented

- The risk of TMS-related seizures is **<1% overall (1:60,000)**
- **Risk Factors**: sleep deprivation, TBI, multiple head concussions; epilepsy; AED discontinuation; alcohol use
- Seizures are self-limiting; and treatment recommendations are supportive in nature.
- TMS-related seizure is reportable to the FDA and TMS manufacturer

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TMS Efficacy

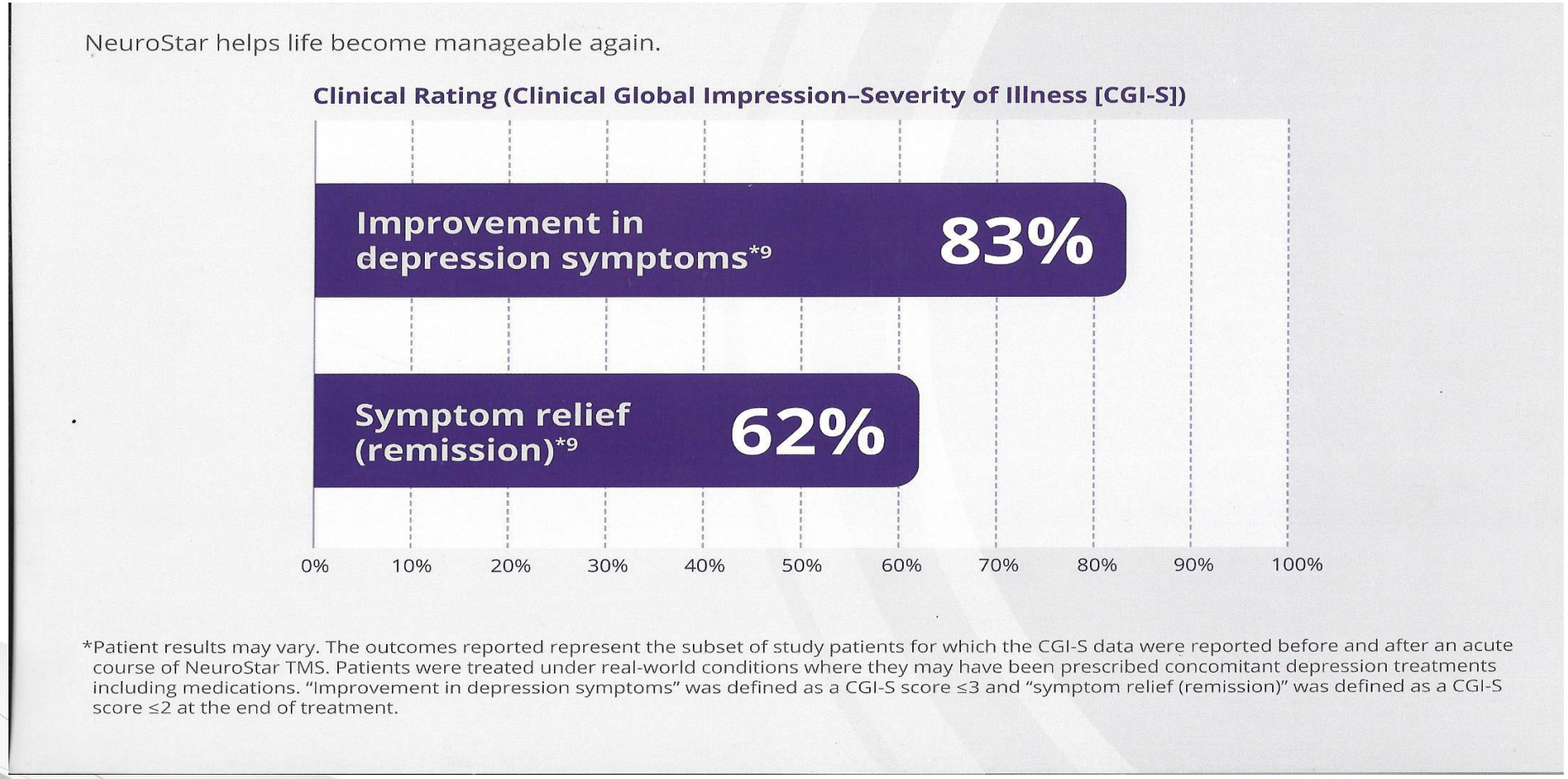
Transcranial Magnetic Stimulation (rTMS) Efficacy

- **Standardized Effect size** (Neuronetics and NIMH trials): 0.39- 0.55; **Efficacy: 40-60%**
- Deep TMS/Brainsway Trial (20 sites): At week 16: **Response rate: 38.4%; Remission rate: 31.8%**
- **Durability studies**: High (64-90%) durability for acute TMS benefits over a 3-12 month period.

- O"Reardon JP et al, 2007; George MS et al, 2010

Neurostar rTMS Efficacy

(Sackeim HA, Aaronson ST, Carpenter LL, et al, J Affect Disord, 2020;277:65-74)



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TMS Efficacy

SAINT Protocol 2020

- **Response**: >/= 50% reduction of MADRS from baseline
  - **90.48%:** One month post SAINT= **70%**
- **Remission**: MADRS score of zero (0)
  - **90.48%:** One month post-SAINT= **60%**

Cole E, Stimpson K, et al. Stanford Accelerated Intelligent Neuromodulation Therapy for Treatment-Resistant Depression. Am J Psych 2020; 177: 716-726

SNT Protocol 2022

- SAINT Protocol
- 14 Active SNT; 15 Sham SNT
- **Response:** 12/14 **(85.7%)** in active
  - 4/15 (**26.7%)** in Sham
- **Remission:** 11/14 **(78.6%)** in active
  - 2/15 (**13.3%)** in Sham

Cole E, Phillips A, et al, Stanford Neuromodulation Therapy (SNT): A Double-Blind Randomized Controlled Trial. Am J Psych 2022; 179: 132-141

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Non-Response to TMS: BDNF Single Nucleotide Polymorphism (SNP)

- **Brain-Derived Neurotrophic Factor (BDNF)**
  - **Synaptic plasticity : LTP/LTD**
  - Polymorphisms is relatively common **(65% Val66Val to 35% Val66Met)** in the Caucasian population.
- **BDNF *Val66Met***
  - ***Reduced hippocampal volume and episodic memory***
  - ***Less susceptible to the effects of TBS than the Val66Val individuals***

Cheeran B, Talelli P, et al. A common polymorphism in the brain derived neurotrophic factor gene (BDNF) modulates human cortical plasticity and the response to rTMS. J Physiol 2008 Dec 1; 586: 5717-5725

Transcranial Magnetic Stimulation (rTMS) for Obsessive –Compulsive Disorder

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Obsessive-Compulsive Disorder (OCD)

- OCD affects approximately 1.3% of the population in any given year, and up to 2.7% over the course of a lifetime.
- **Obsessions** are repetitive, stereotyped thoughts that cause anxiety or distress; experienced as intrusive or ego-dystonic and recognized as unrealistic or excessive.
- **Compulsions** are ritualized actions that are undertaken to mitigate distress, often in response to obsessions.

OCD Treatment

- **Cognitive-Behavioral Therapy** (CBT) and psychotropic medications **(SSRI**) are the first-line treatments of OCD

- **Exposure and Response Prevention (ERP)**
  - the specific CBT technique for OCD; considered the most effective treatment for OCD
- **Acceptance and Commitment Therapy (ACT)**
  - a newer therapy for anxiety and depression, and also effective for OCD

TMS for OCD

- Procedure:
  - **Provocation Planning**
    - Specific obsessions and compulsions, avoidance and symptom provocation
    - Patient distress score of 4-7 on a visual analog scale (VAS)
  - Week 1-5: 5 TMS treatments per week
  - Week 6:4 Treatments

Tendler A, Sisko E, et al; A Method to Provoke Obsessive Compulsive Symptoms for Basic Research and Clinical Interventions. *Frontiers in Psychiatry*, 2019; 10: 3389. Article 0814

TMS for OCD

TAP INTO MORE POSSIBILITIES *with NeuroStar®*

	Dash Protocol	TouchStar	OCD
Pulses	10 per second	3 pulses per burst 20 msec interpulse interval 5 bursts per second	**20 per second**
Stimulation Time	4 seconds	2 seconds	2 seconds
Inter-train Interval	11 seconds	8 seconds	20 seconds
Number of Pulses	3,000	600	2000
Intensity	120% resting Motor Threshold	120% resting Motor Threshold	100% resting Motor Threshold
Duration	18:45 minutes	3:20 minutes	18:20 minutes
Treatment Location	Left Prefrontal Cortex	Left Prefrontal Cortex	Dorsomedial Prefrontal Cortex

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TMS for OCD

- Response Rate:
  - Active treatment: **38%**
  - Sham treatment: 11%
- One-Month Follow-up:
  - Active treatment: **45%**
  - Sham treatment: 18%

Source: Neurostar.com (Neuronetics, 2022); Carmi L et al, J Am Psych , 2018

Summary

- Repetitive Transcranial Magnetic Stimulation
  - Neurocircuitry of Mood disorders
  - Neurobiological mechanisms
  - Long-term Potentiation (LTP) and Long-term depression (LTD)
  - Neuroplasticity and Metaplasticity
- Clinical indications of rTMS:
  - Major depressive disorder, severe and treatment resistant, 2008
  - Pain related to migraine headaches, 2013
  - Obsessive compulsive disorder, 2018
- Emerging trends
  - Theta Burst Stimulation (SAINT and SNT)
  - TIARA Study: for Severe, Enduring Anorexia Nervosa (SE-AN)

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Got HOPE? Thank you.

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