Harvard TMS Course

# Pharmacology and TMS

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President-Elect | Clinical TMS Society
Editor-in-Chief | *Transcranial Magnetic Stimulation*

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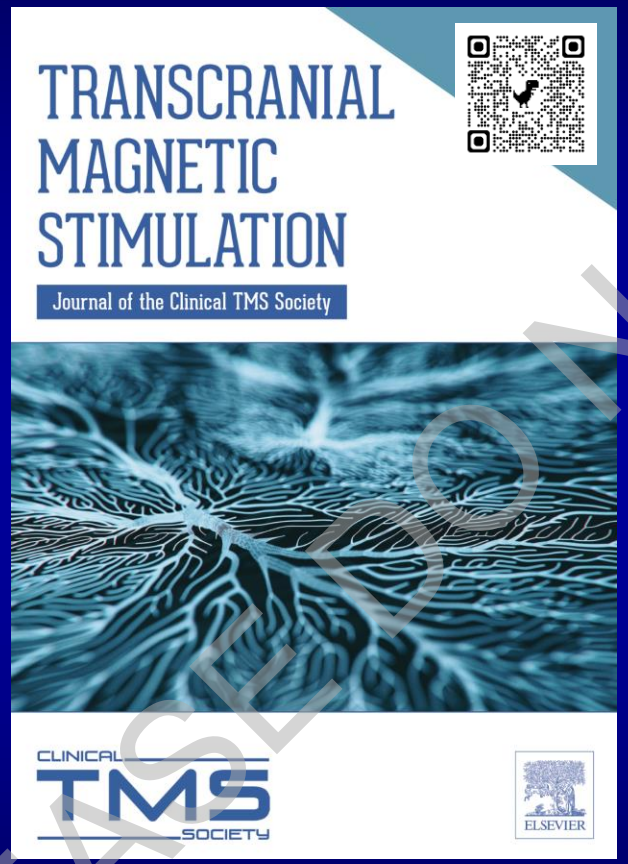
# Disclosures

- Editorial Stipend
- Speaking Honoraria
- Equity and/or payment for Consulting: Zeta surgical, NRX pharmaceuticals (CMIO).

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NIH National Institute of Mental Health

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DARPA DEFENSE ADVANCED RESEARCH PROJECTS AGENCY

BRAIN & BEHAVIOR RESEARCH FOUNDATION
Awarding NARSAD Grants

NIH National Institute on Alcohol Abuse and Alcoholism

NIH National Institute on Drug Abuse

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# Learning Objectives

1. The brain is an Electro+Chemical organ (~TMS+Pharm)
2. TMS induces brain changes – know rate limiting mechanism
3. Pharmacology can enhance or attenuate TMS effects
4. Chronic (daily medications) vs. Acute effects: homeostatic plasticity

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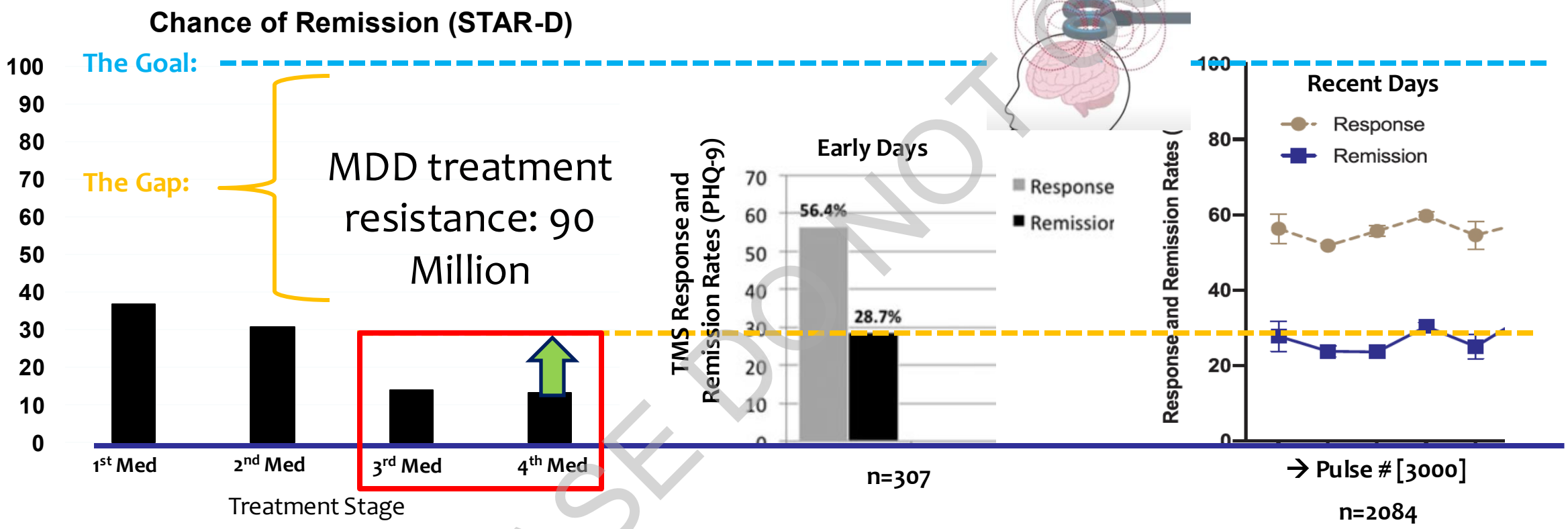
# TMS is effective… But Can it be better?




Adapted from Rush et al., *AJP*, 2006 (STAR-D)

Carpenter et al., *Depress Anxiety*, 2012

Sackeim et al., *J Affect Dis*, 2020

# HOW do we make it better?

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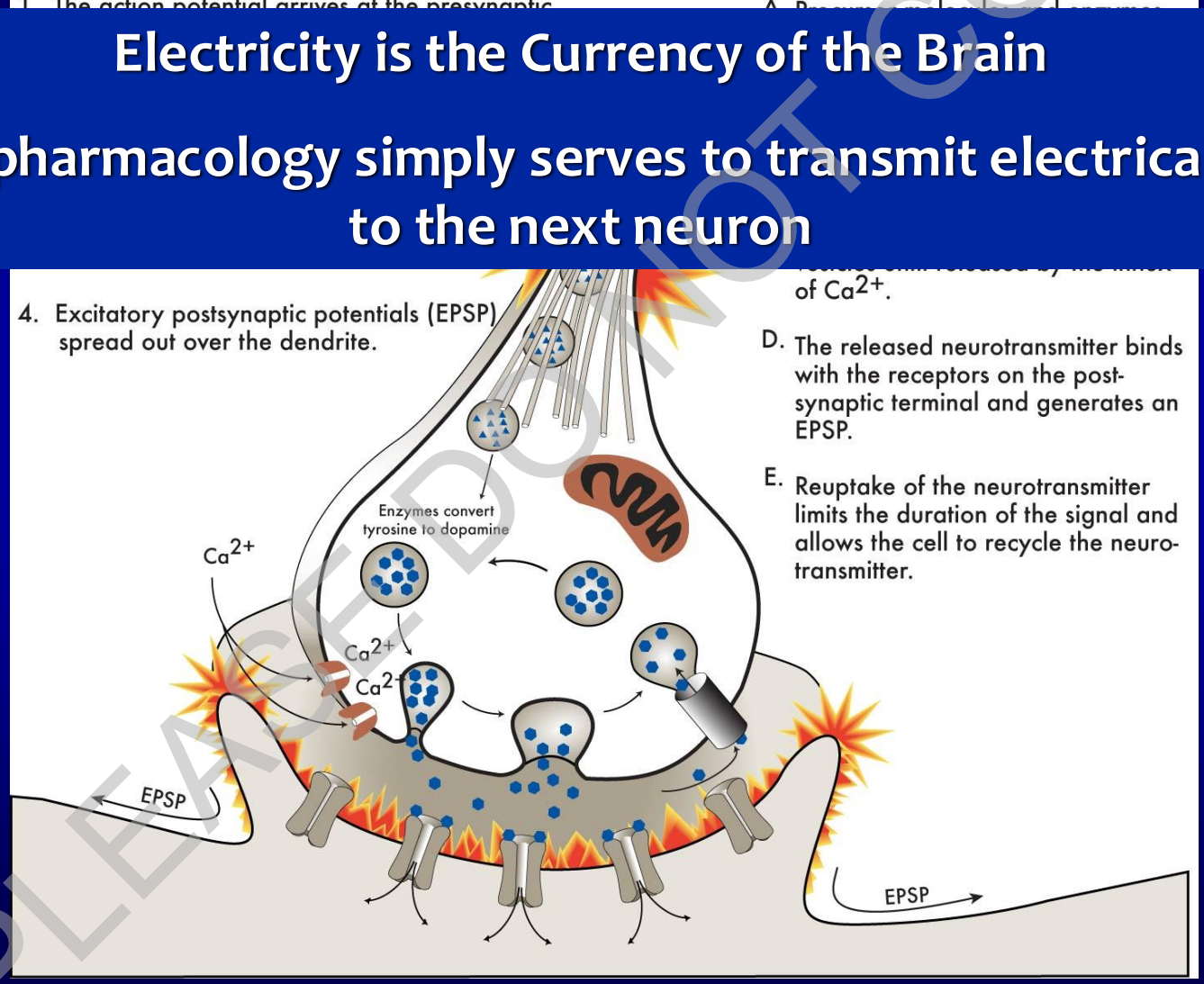
# The Brain is an Electrochemical Organ

**Electricity is the Currency of the Brain**

**Synaptic pharmacology simply serves to transmit electrical signals to the next neuron**

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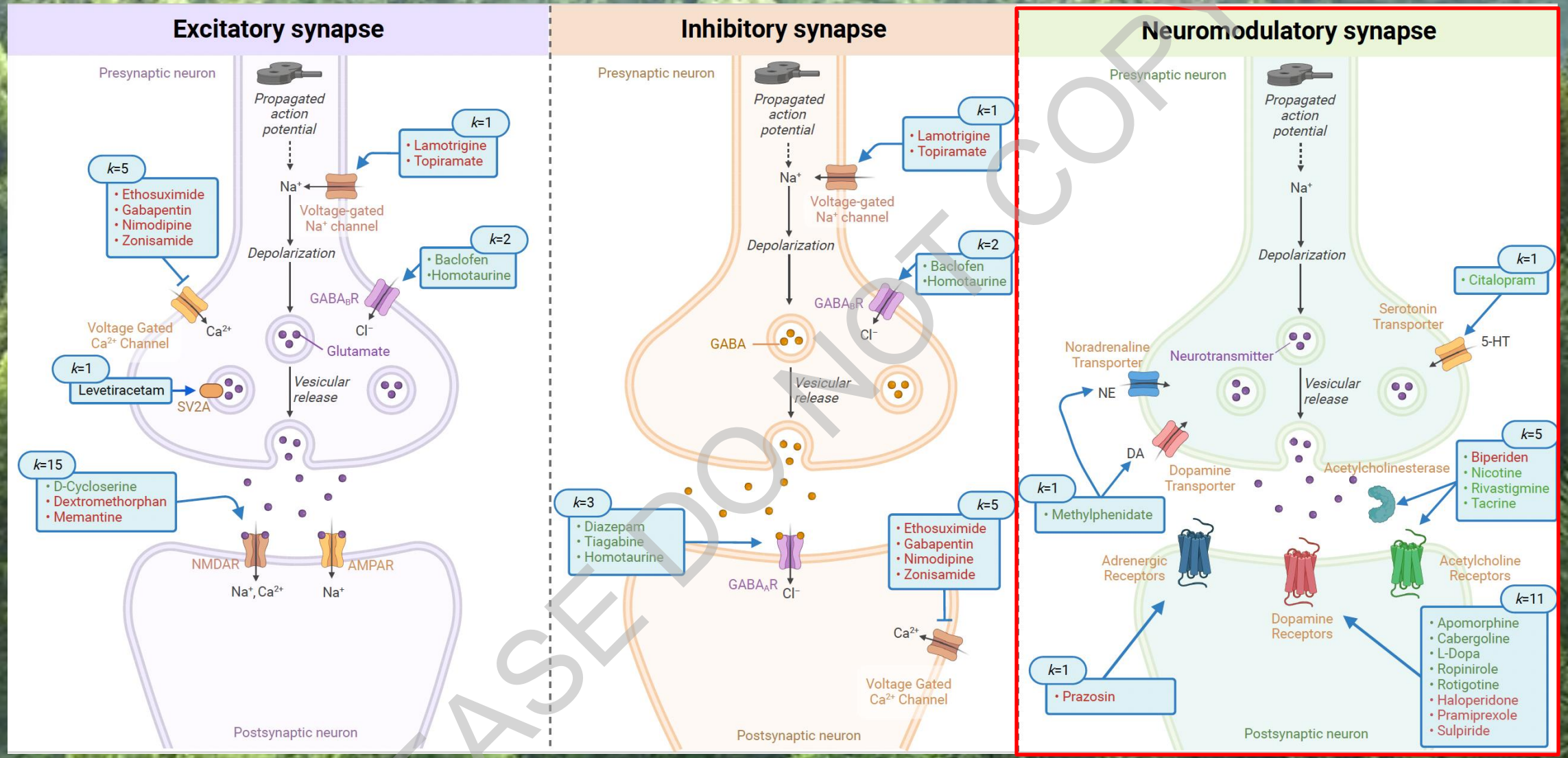
Slide adapted from Mark George and Higgins & George, *Brain Stimulation Therapies for Clinicians*, 2019




Sohn et al., *J Psychiatry Neurosci*, 2024

# Survey of Pharmacologic Enhancement

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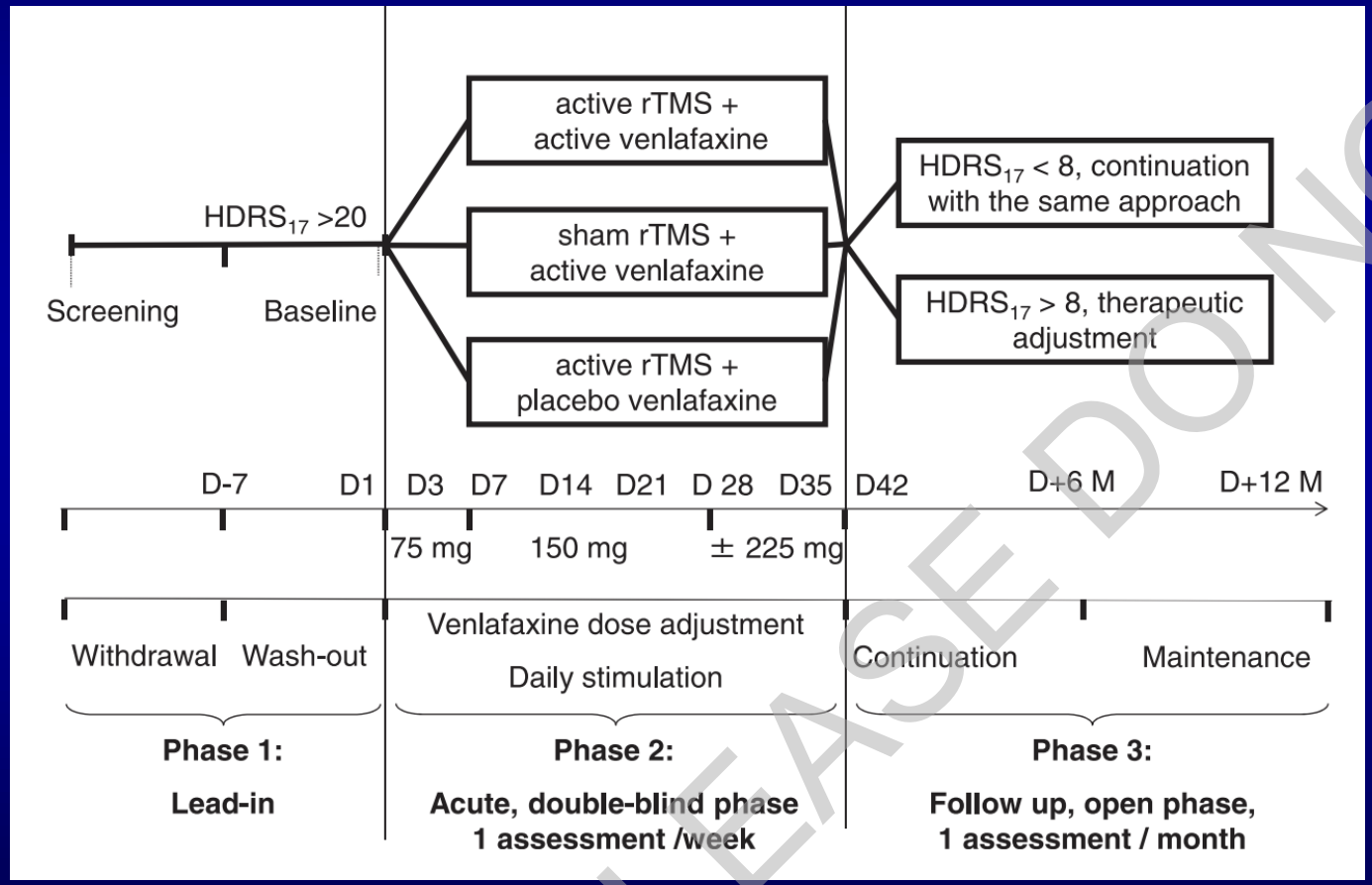
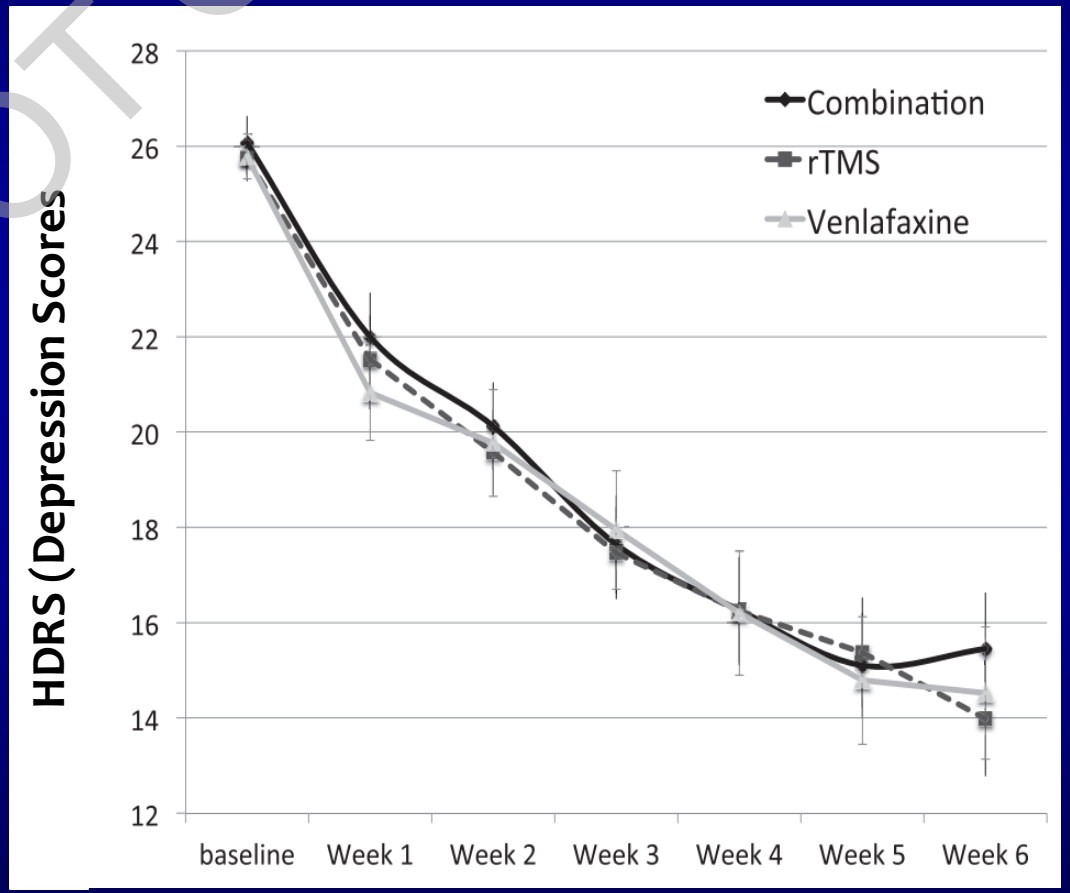
# Only other Pharm+TMS clinical trial: SNRI




Brunelin et al., *Brain Stim*, 2014

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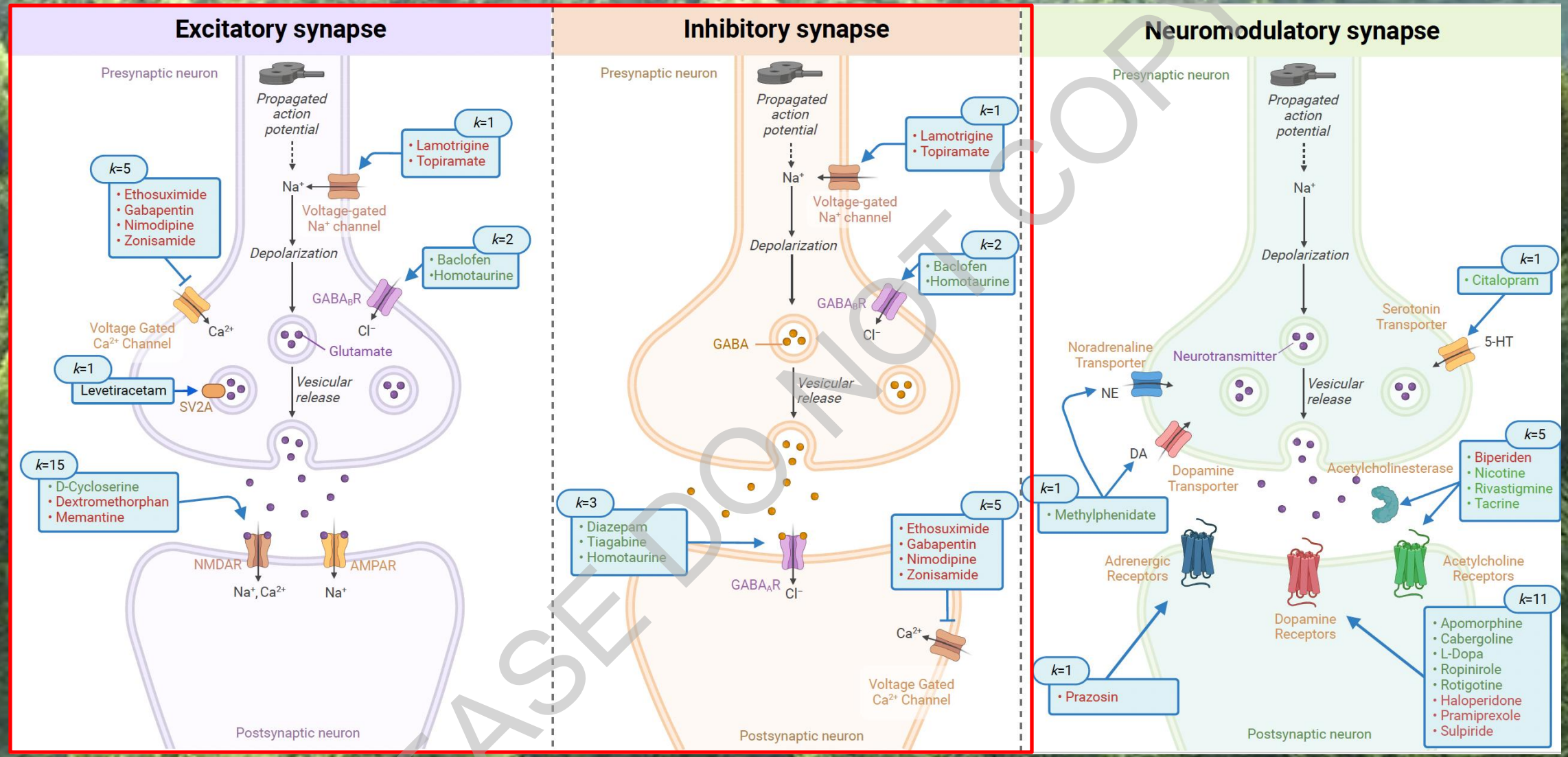


Sohn et al., *J Psychiatry Neurosci*, 2024

# Survey of Pharmacologic Enhancement

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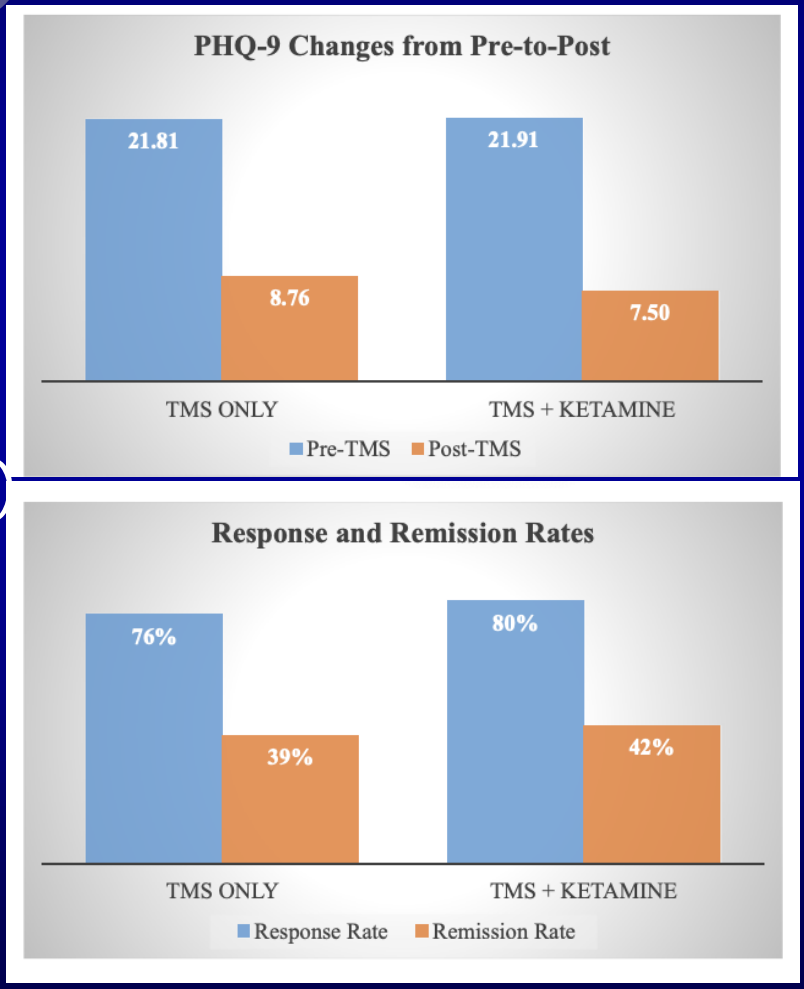
# Does Ketamine Enhance TMS Effects?

- Systematic Review (Debowska, *Front Neurosci*, 2023):
  - No Prospective Studies!
  - 11 studies reported
    - *n* of 1 Case studies: 7
    - 4 retrospective studies: total *n* of 53
      - 1-Hz x2 studies (short-term and 2-year follow up)
      - 10-Hz x1 study
    - All report improvement
    - 1 comparative study

36 TMS (H-coil) +/- 6 IV ketamine treatments




Shanok et al., *Psychopharm*, 2024

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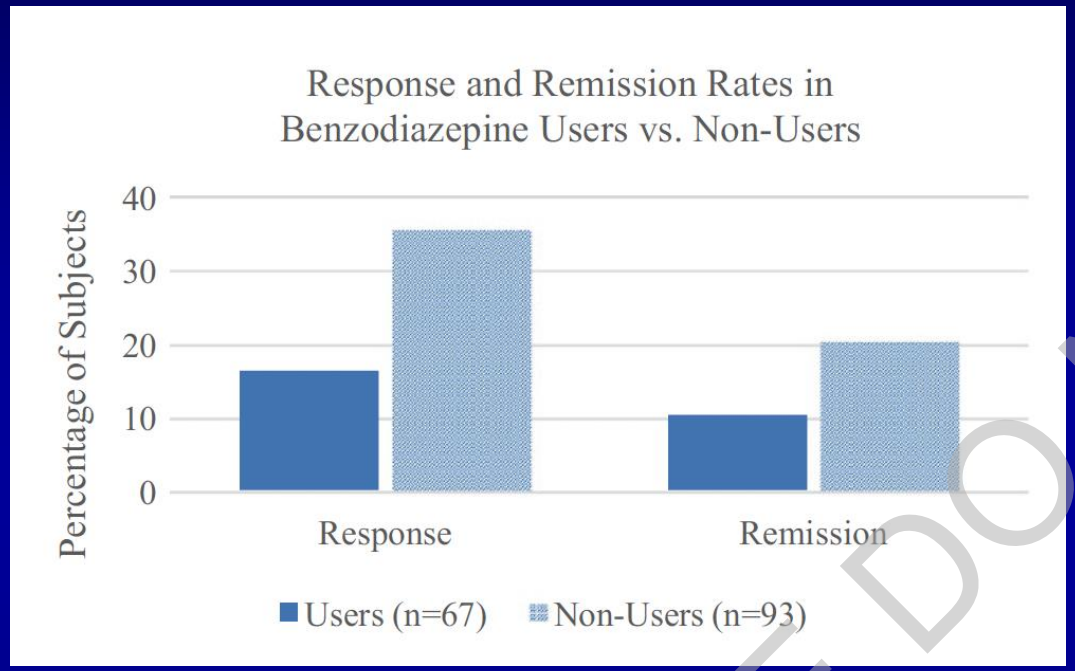
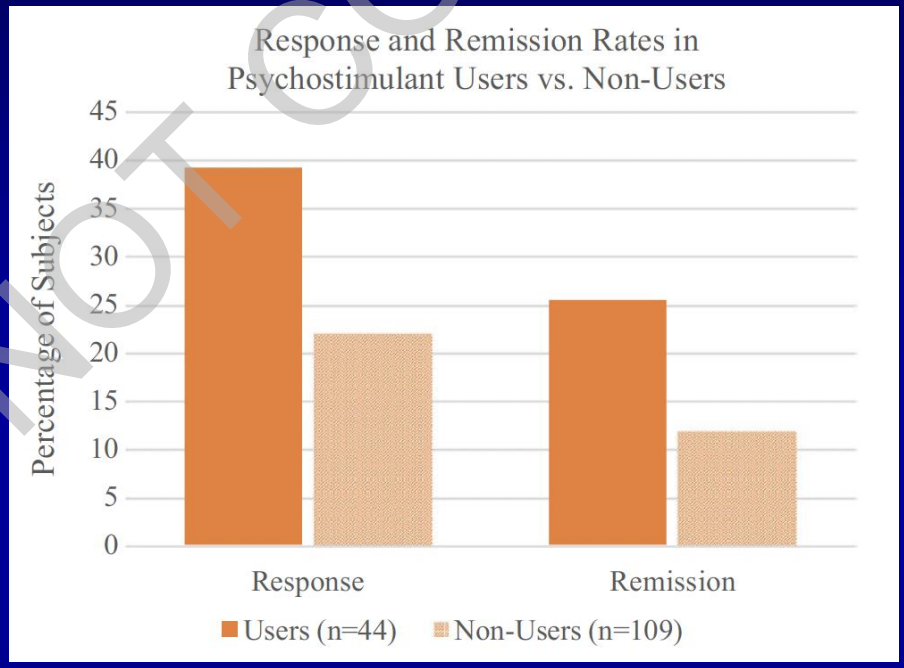
# Benzo's & Stimulants




Hunter et al., *Brain Behav*, 2019

Supported by: THREE-D study sub-analysis: 123/388 patients. (Kaster, AJP, 2019)

- BDZ users more likely NON-responders
- BDZ users more likely slower trajectory

BDZ Not Supported by: Two clinical trials: 64/121 patients. (Fitzgerald, Brain Stim, 2020)

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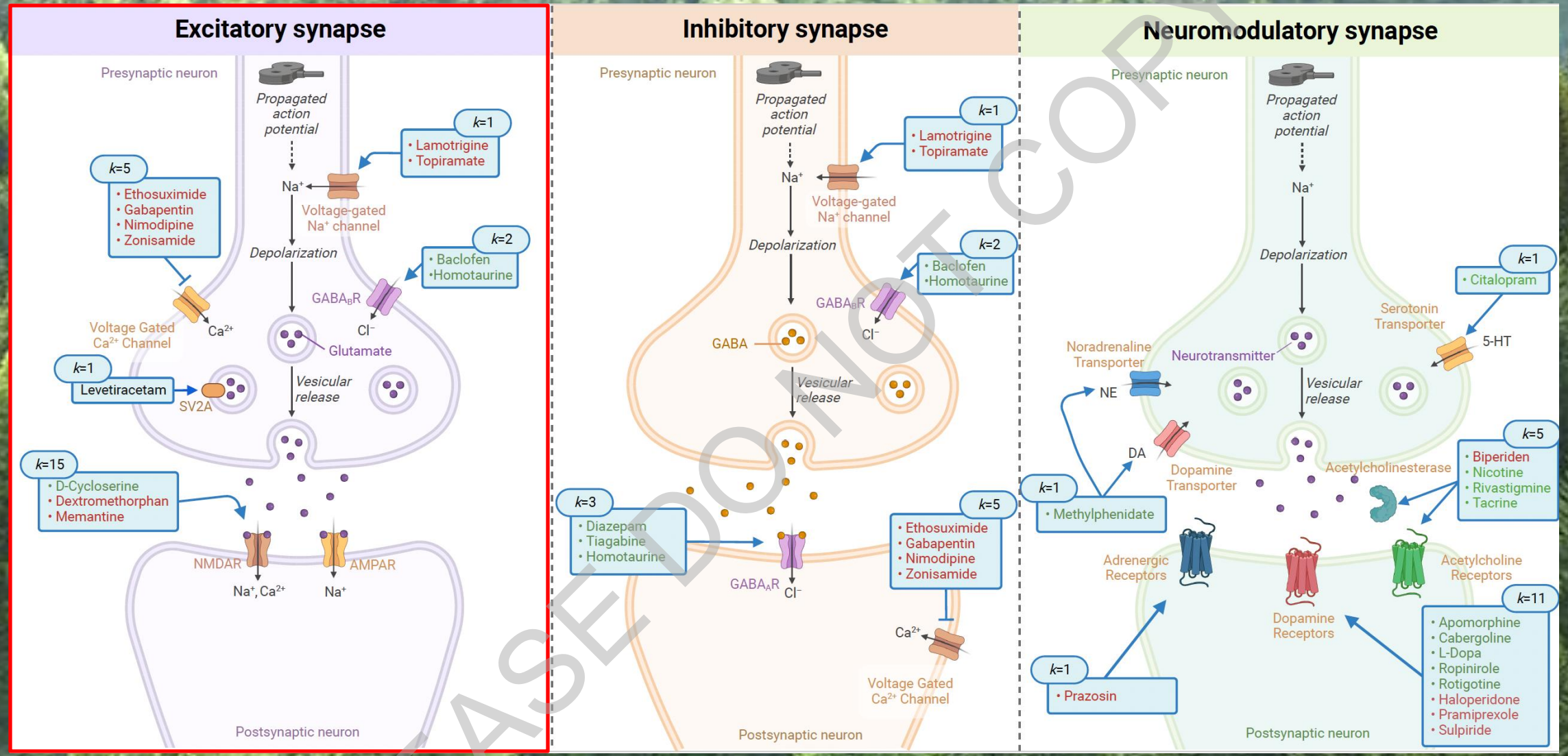


Sohn et al., *J Psychiatry Neurosci*, 2024

# Survey of Pharmacologic Enhancement

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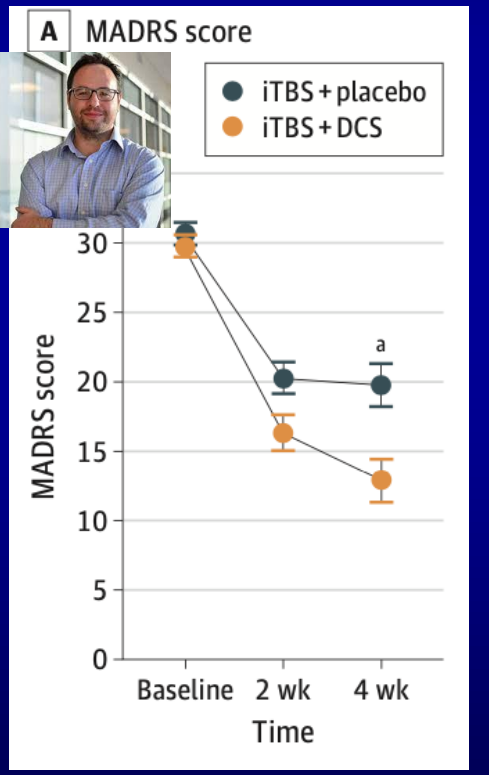
# What does Synaptic Plasticity have to do with Clinical TMS? (Naturalistic)

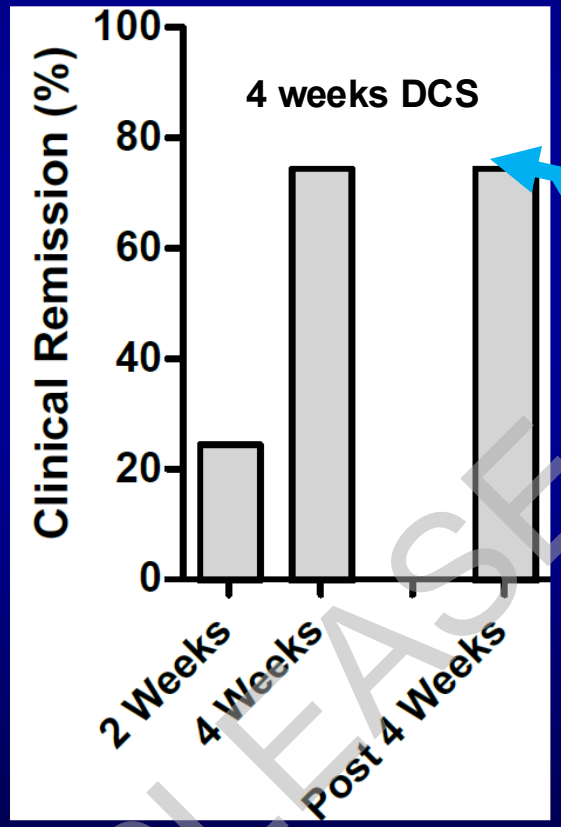
4 weeks:
75% Remission

6+ weeks:
29% Remission

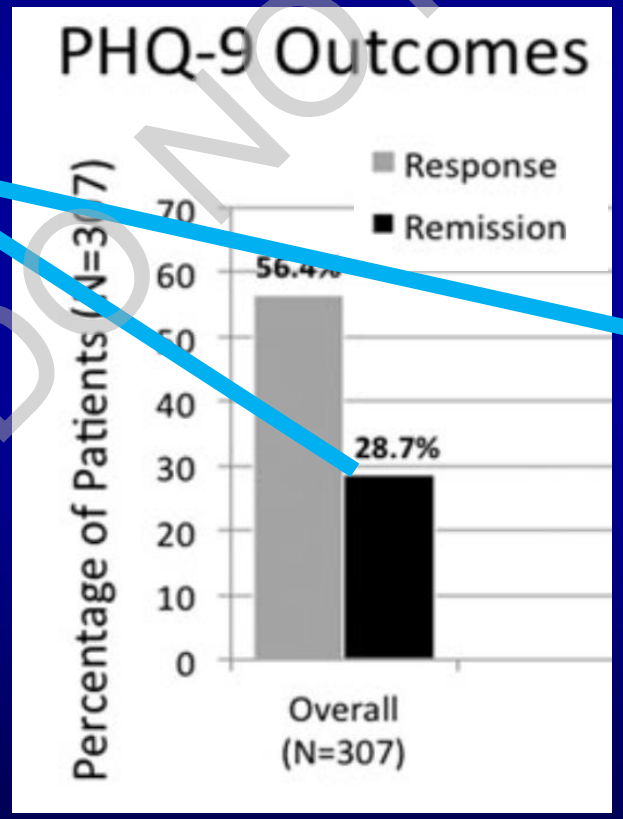
6+ weeks:
~30% Remission



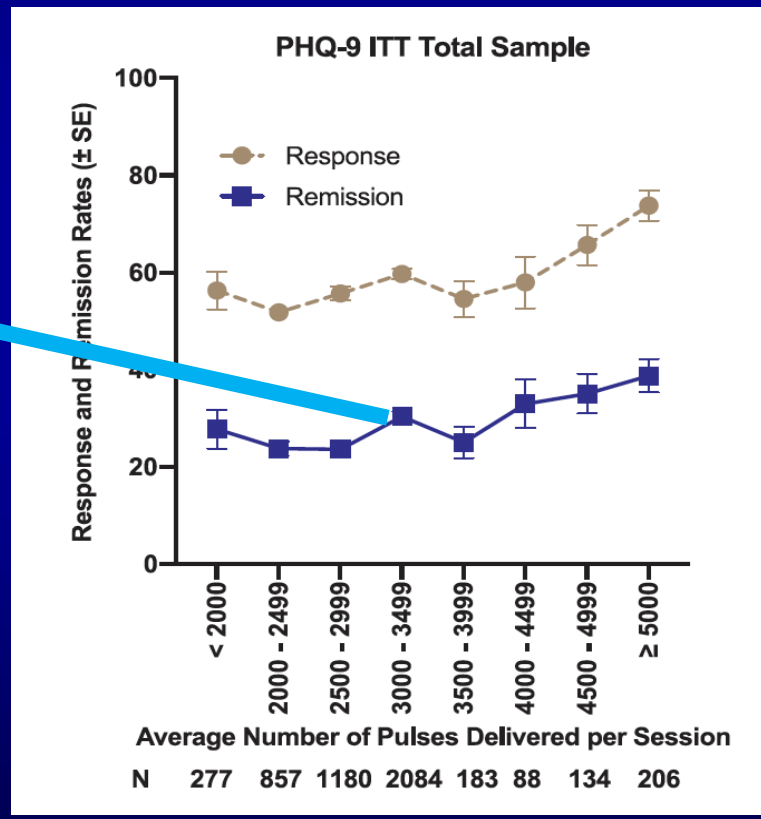

Cole et al., *JAMA Psych*, 2022




DeMayo et al., *J Affect Dis*, 2025




Carpenter et al., *Depress Anxiety*, 2012




Sackeim et al., *J Affect Dis*, 2020

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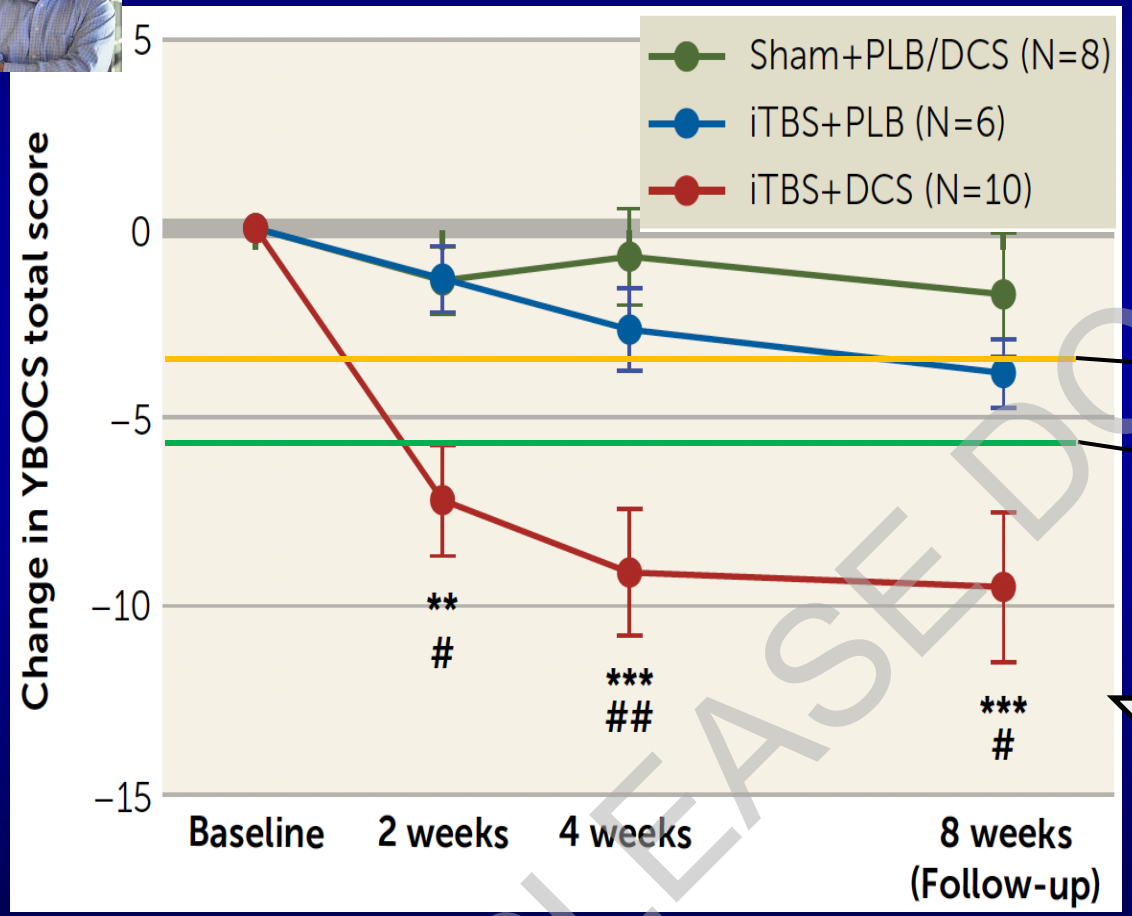
# What does Synaptic Plasticity have to do with Clinical TMS? For OCD



YBOCS Δ: 8

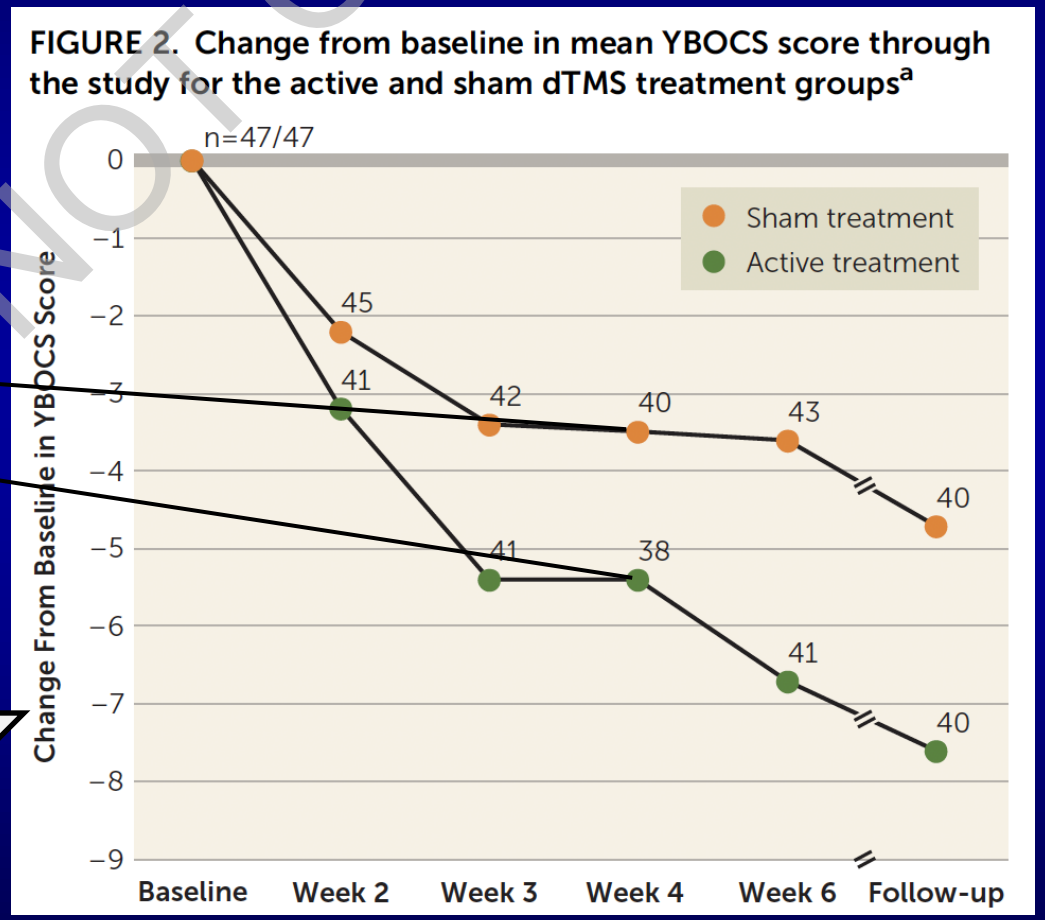
DCS v Active Δ: 6

YBOCS Δ: 2




McGirr et al., *AJP*, 2025

Improvement




Carmi et al., *AJP*, 2018

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Why would some meds improve TMS and not Others?

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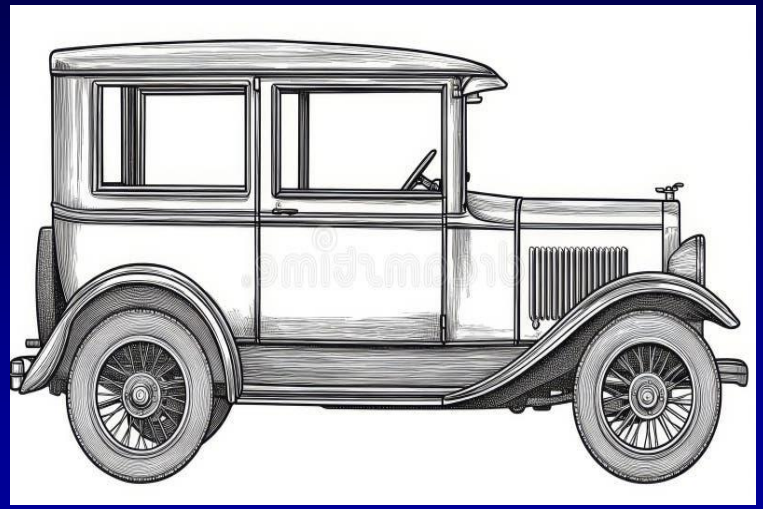
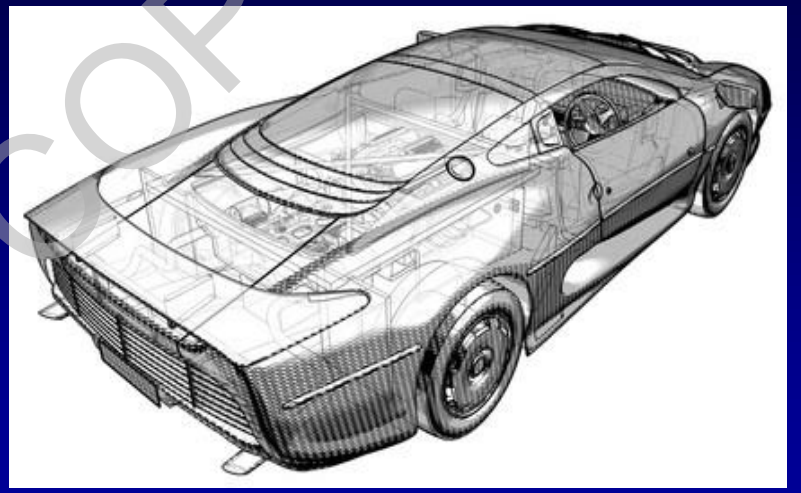
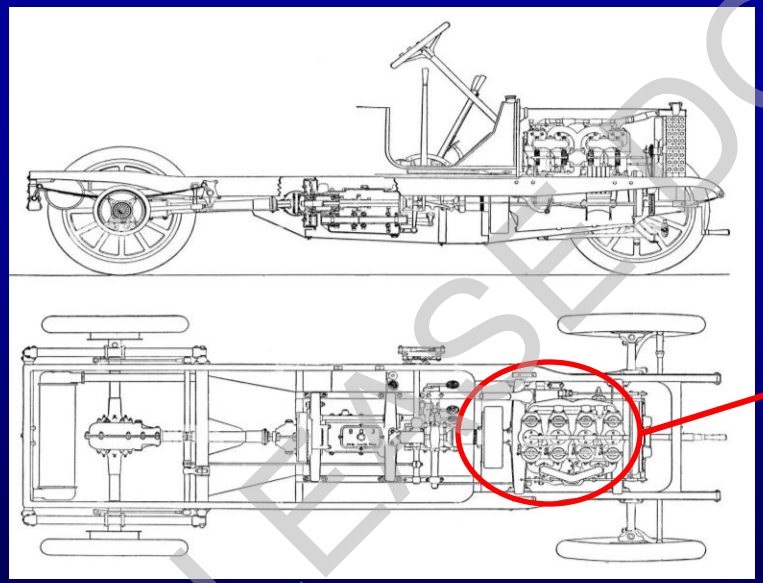
Traditional TMS

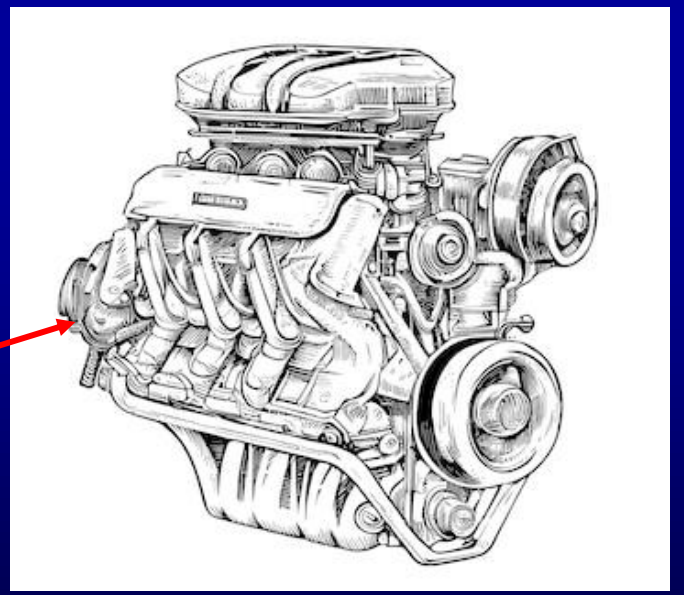


Mechanism-Guided Advancements

Potential TMS







What is the rate-limited “engine” of TMS modulation of the brain?

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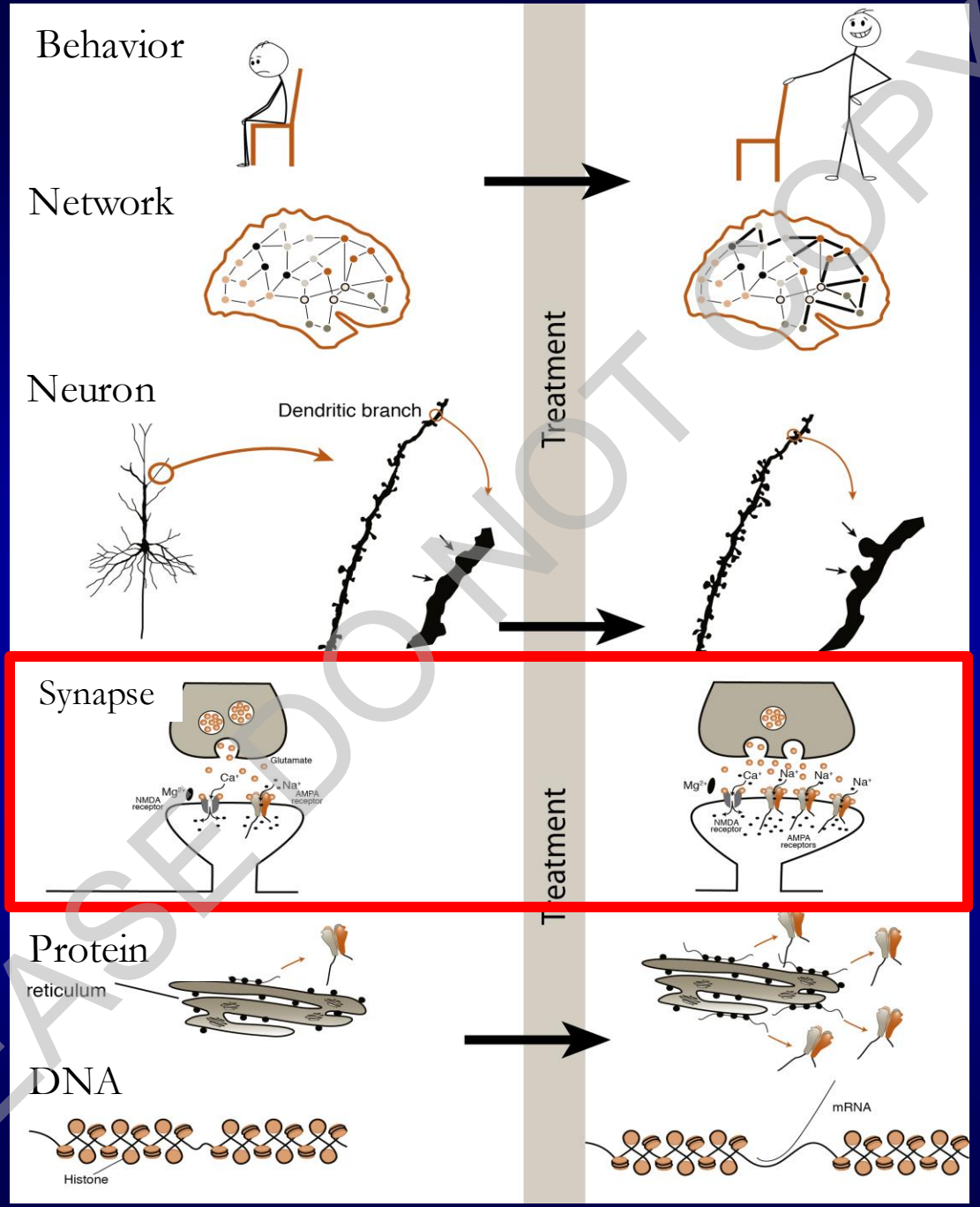
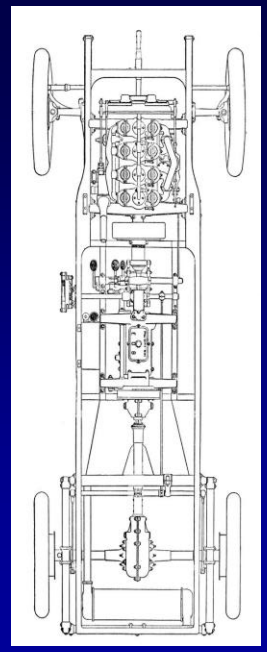
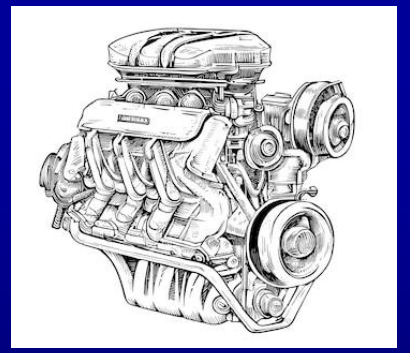
# What Underlies ("Causes") Network and Behavioral Effects?





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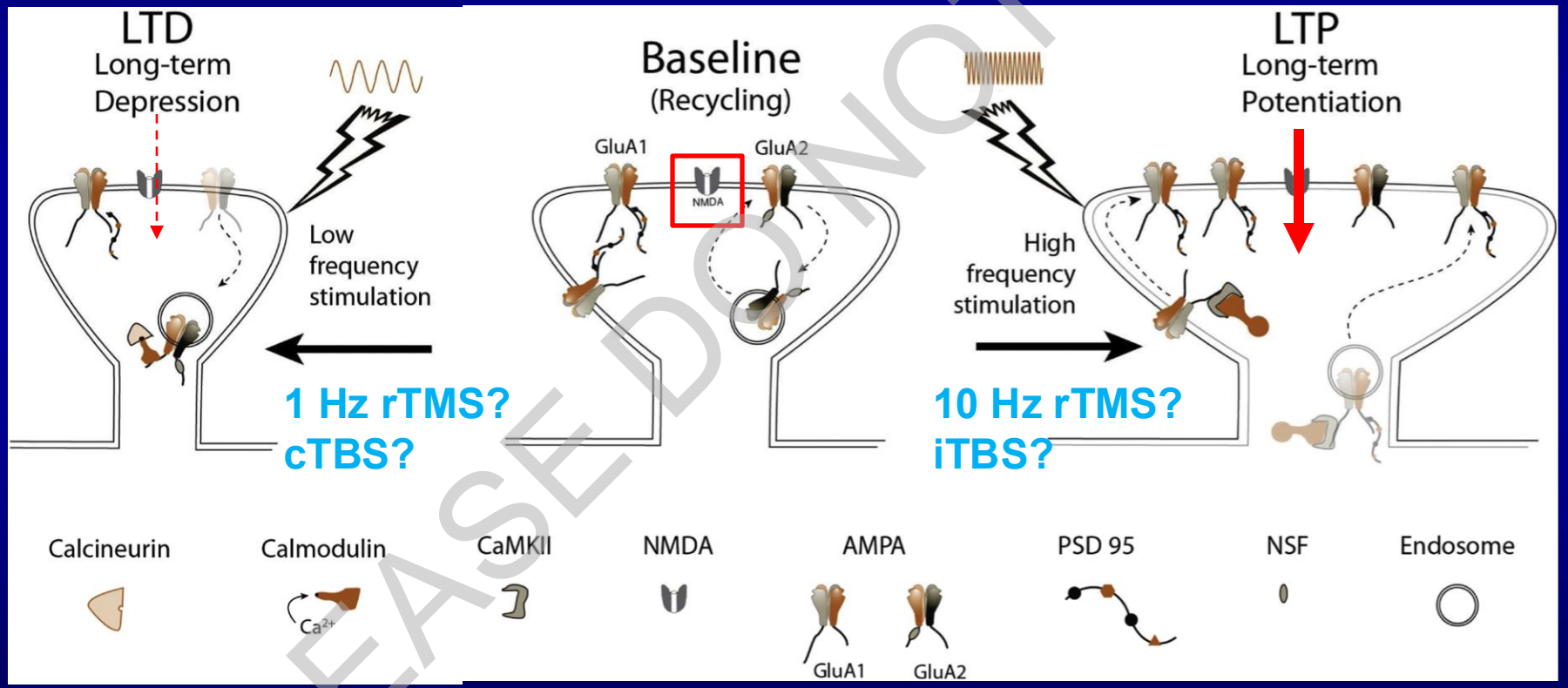
Brown et al., *Neuromodulation*, 2022

# Synaptic Plasticity

## *critically depends on NMDA receptors*




Brown et al, *Neuromodulation*, 2022

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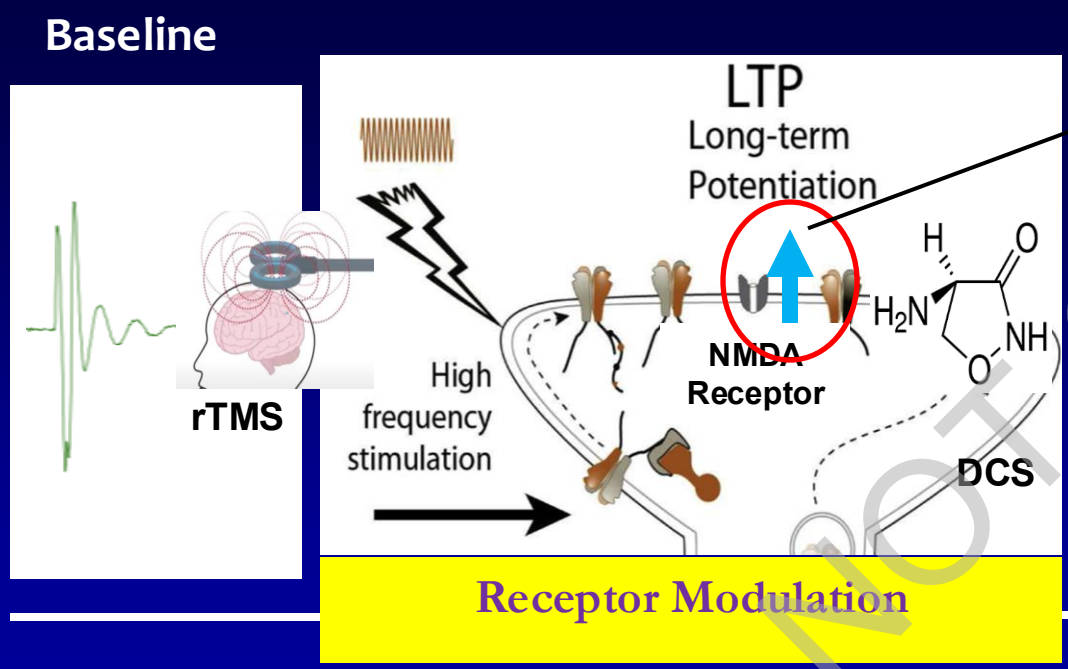
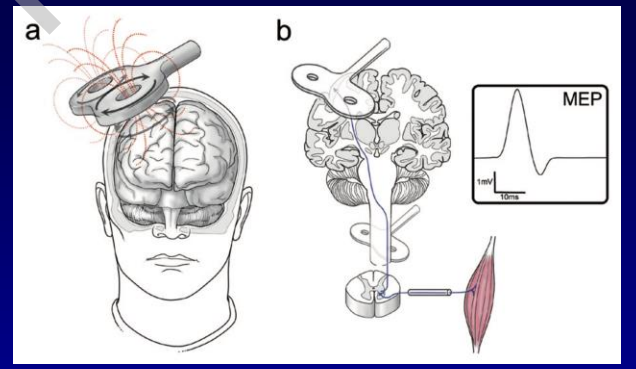
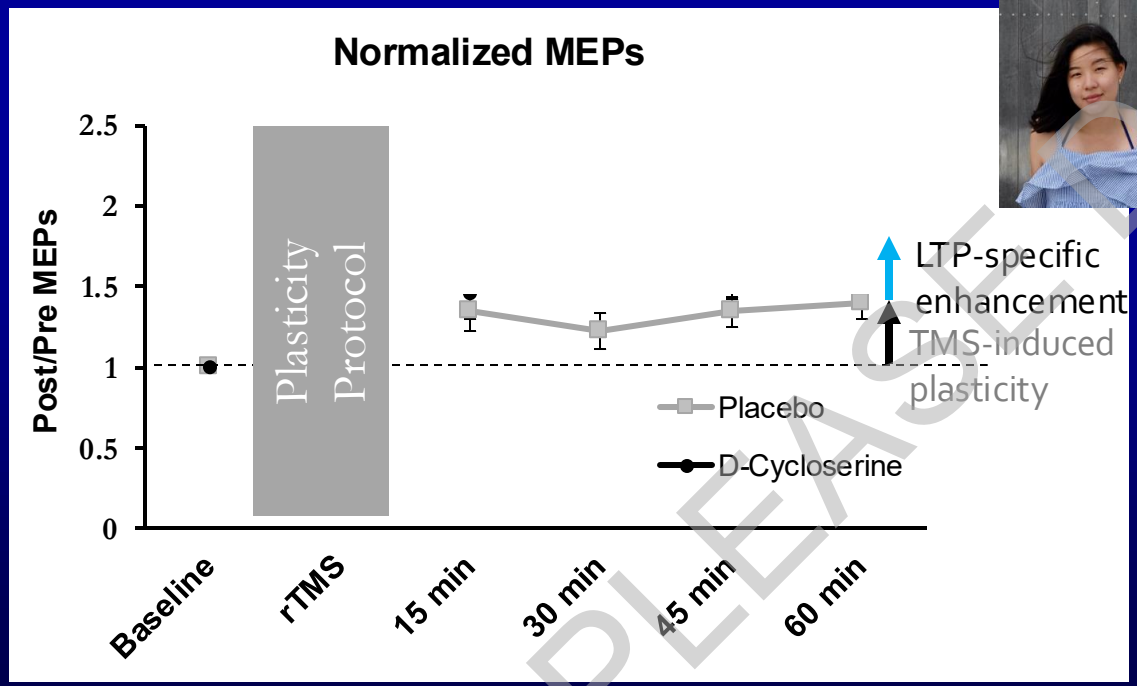
# Does TMS work through LTP?



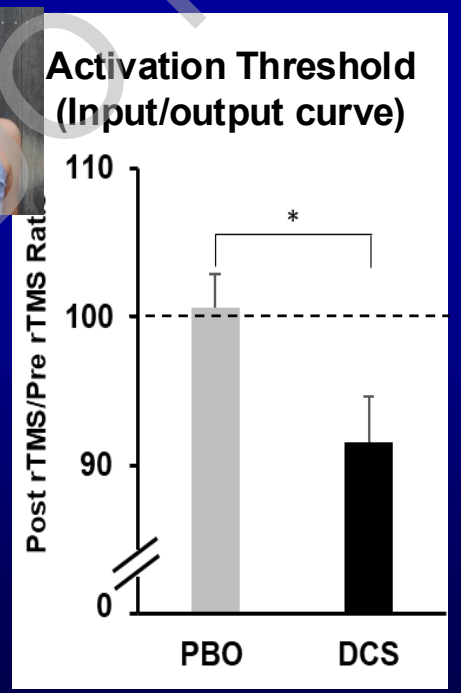




Vlachos et al., *Neuroforum*, 2017

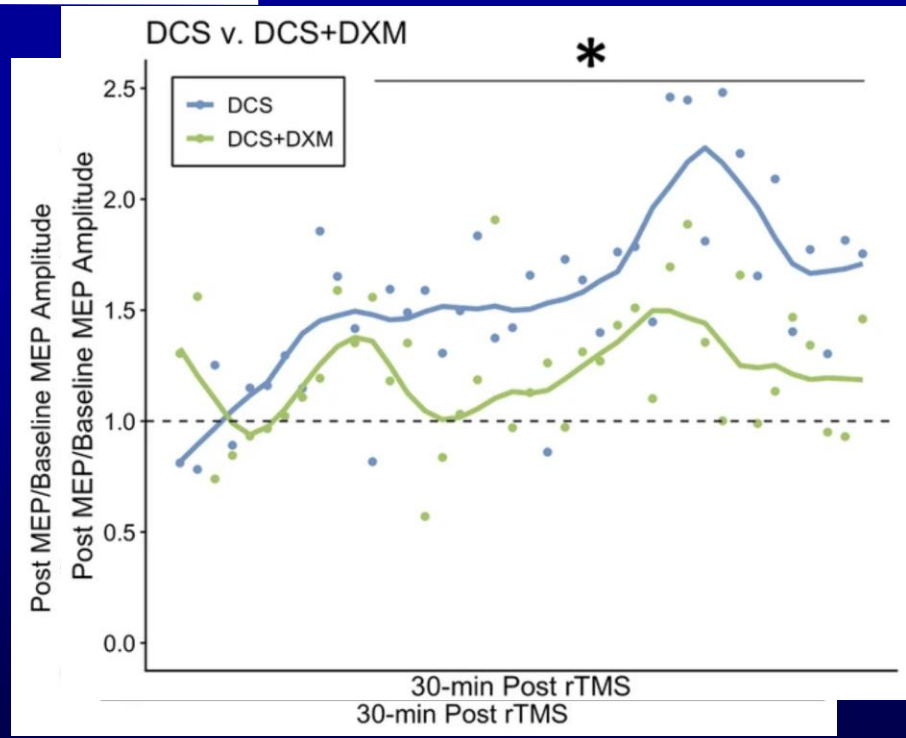



Brown et al., *Brain Stim*, 2020






Kweon, *Brain Stim*, 2022




Kweon, *Transl Psych*, 2026

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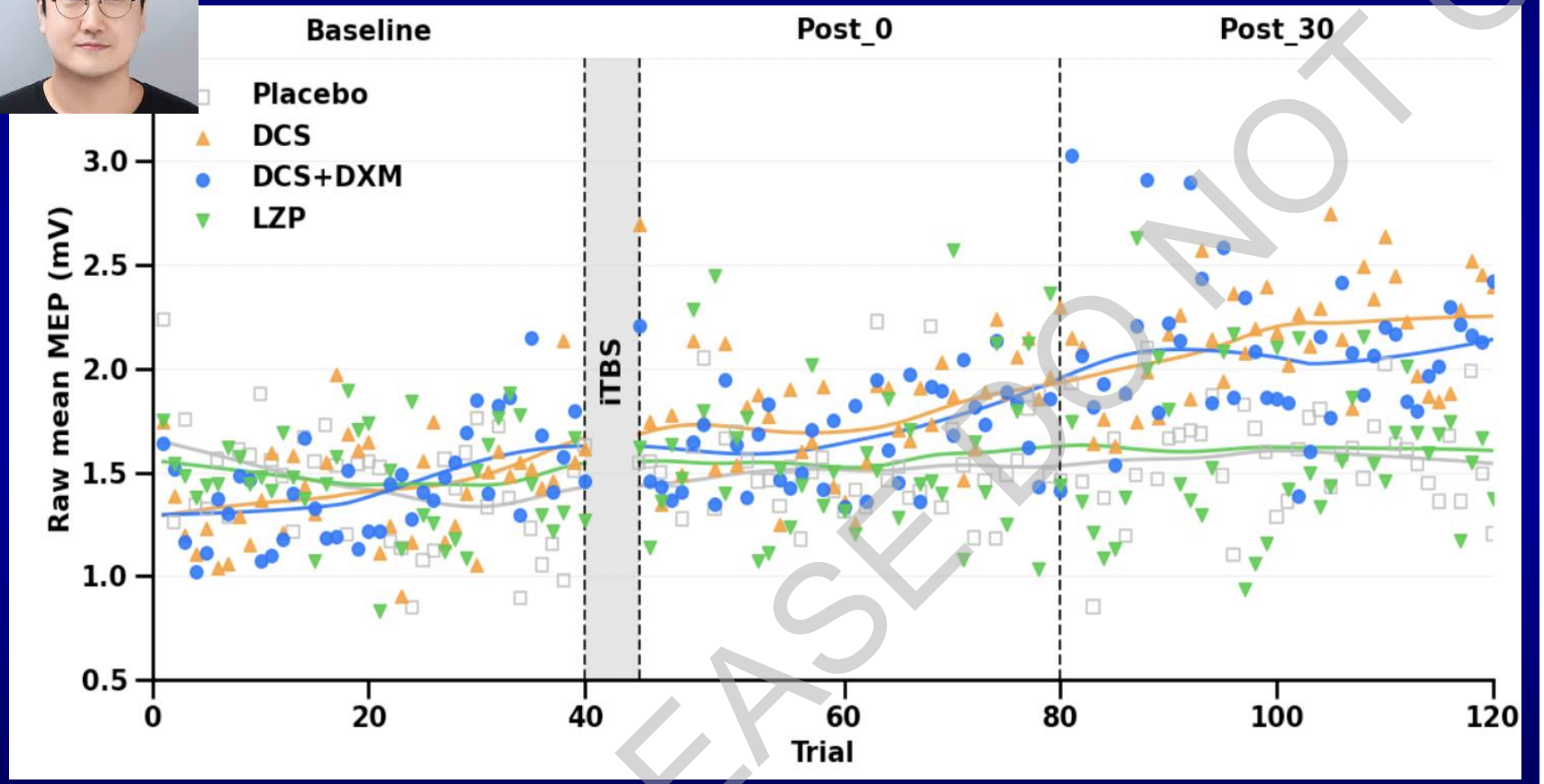
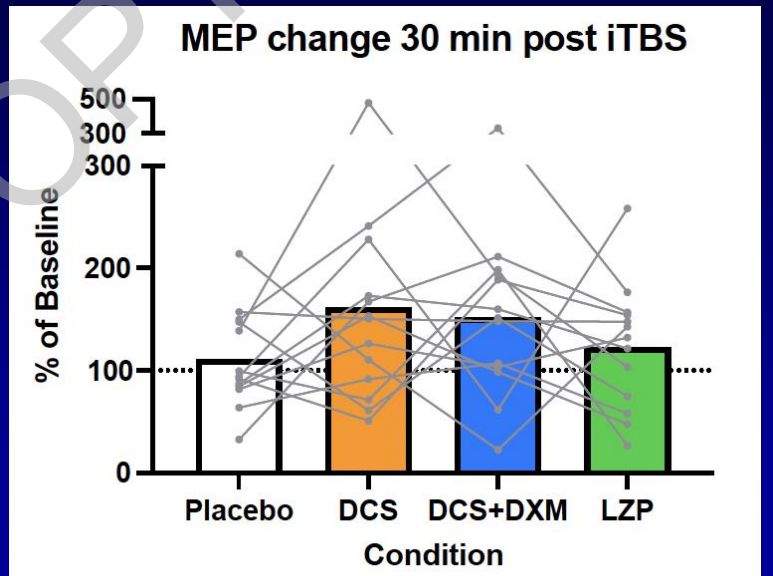
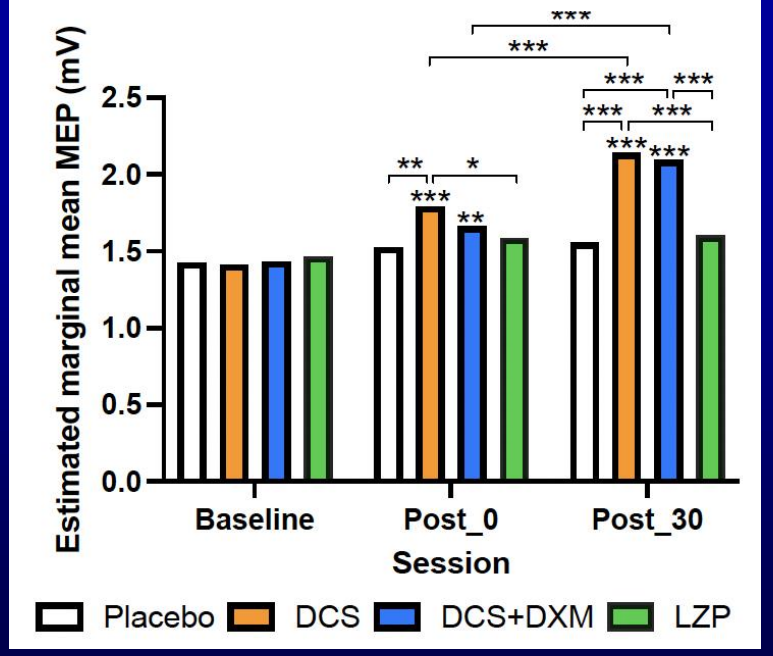
# DCS enhances iTBS-induced plasticity






Kim, *in revision*

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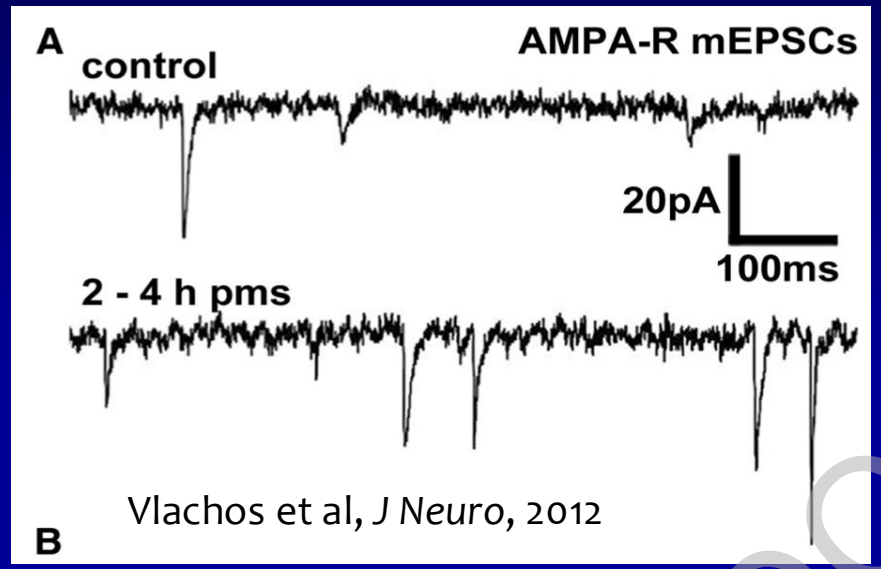
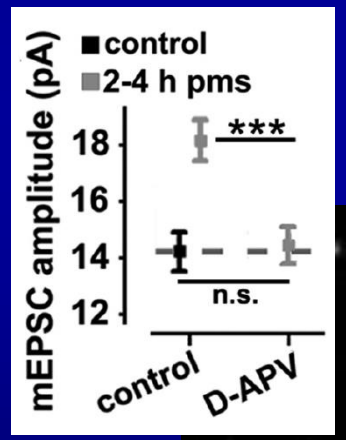
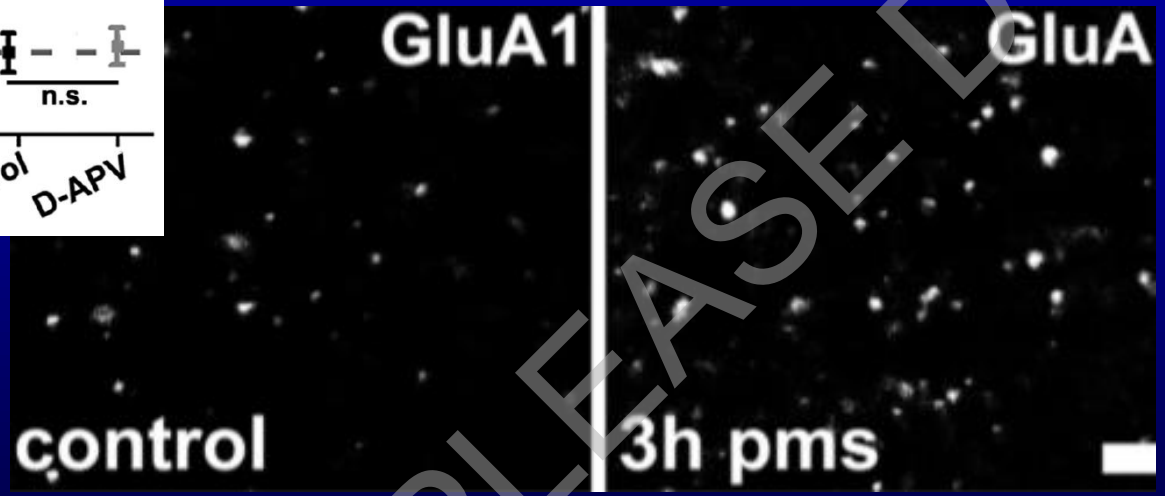
# Evidence for LTP





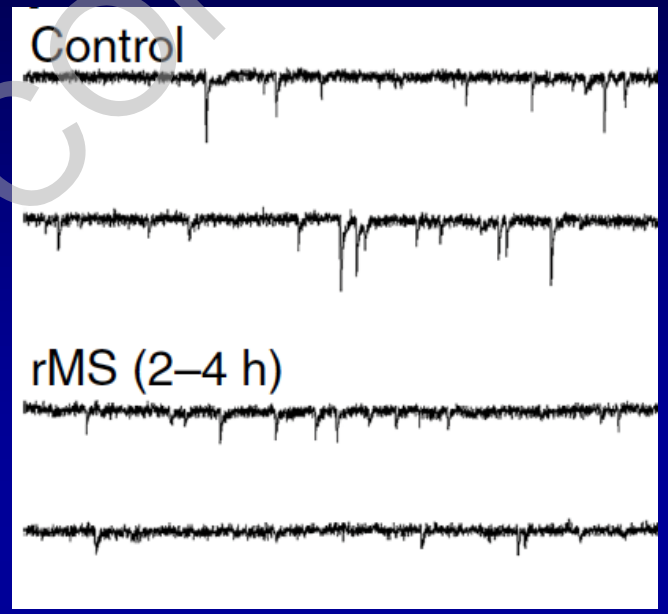

Vlachos et al, *J Neuro*, 2012

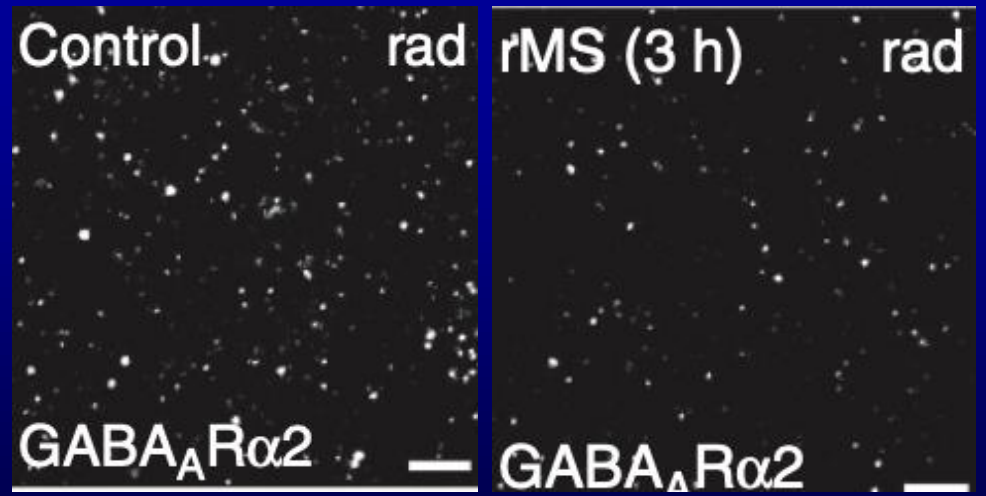



Vlachos et al, *J Neuro*, 2012

# And ↓GABA!




Lenz et al, *Nat Comm*, 2016

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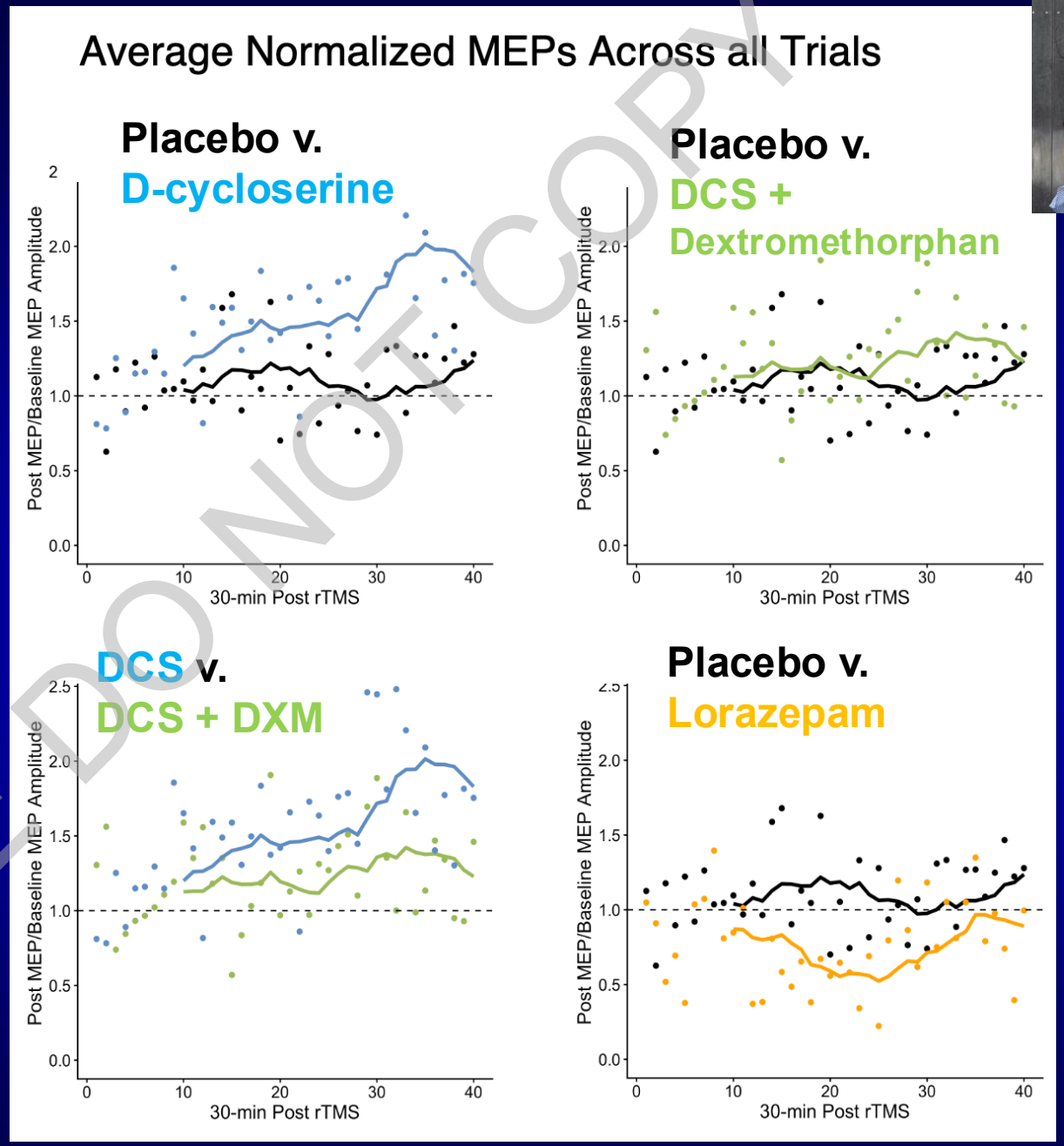
# Does 10-Hz rTMS ↑ LTP or ↓GABA?

- NMDAR agonism (DCS) → enhances plasticity
- NMDAR antagonist → (DXM) partially "knocks down"
- GABAR agonism → NOT increased after TMS






Kweon et al., *Transl Psych*, in press

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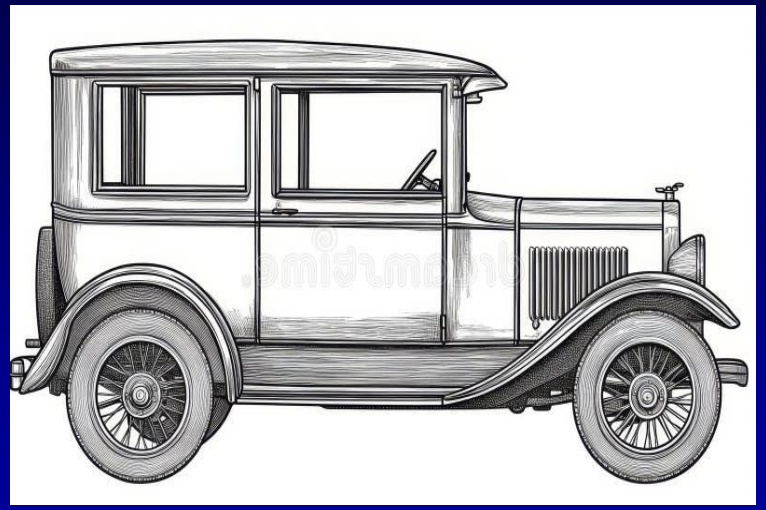
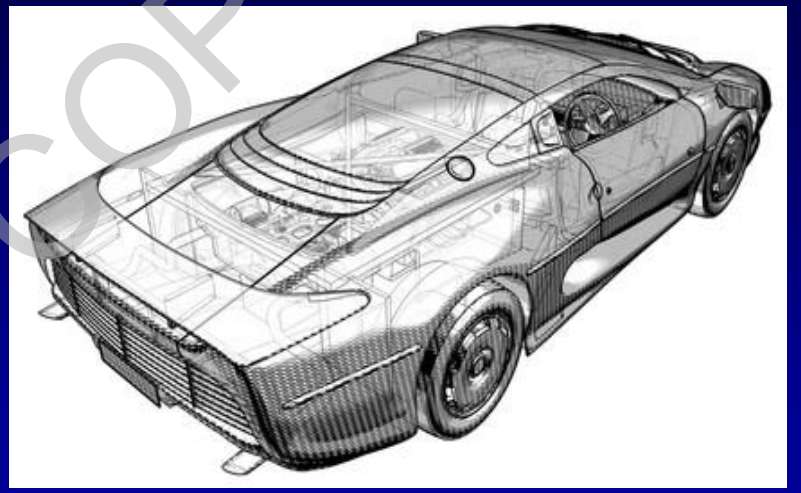
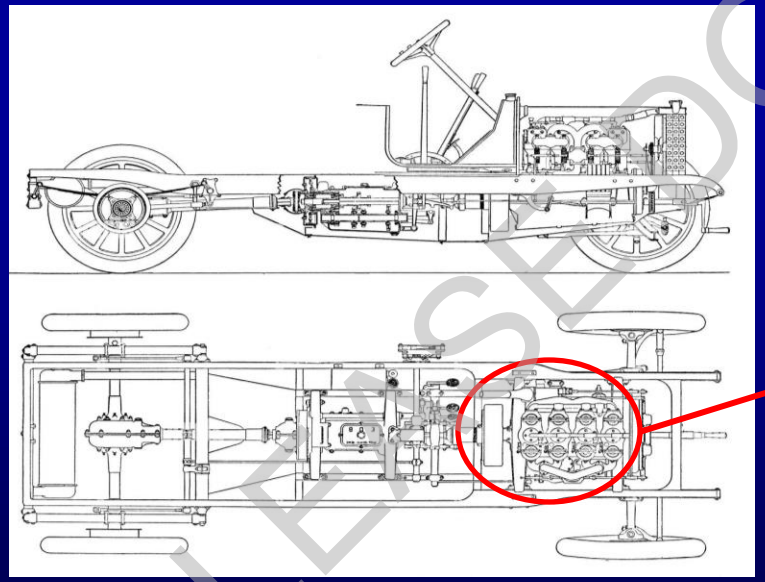
Traditional TMS

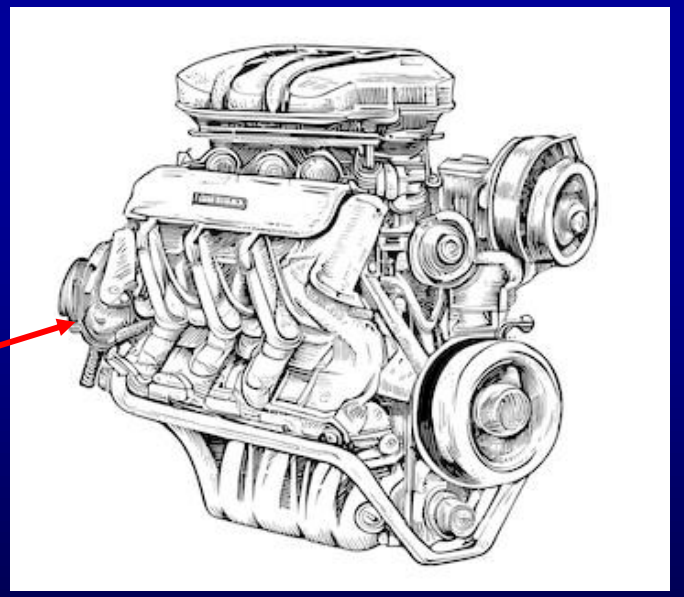


Mechanism-Guided Advancements

Potential TMS







Can leveraging rate-limited “engine” TMS improve efficie?

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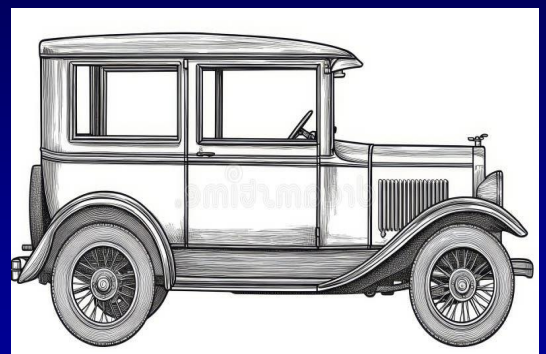
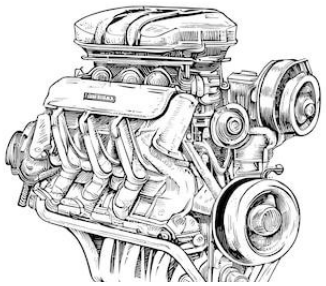
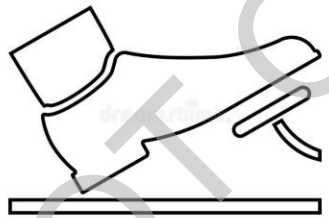
# What if we Combine the 1) Enhanced Efficiency of Mechanism-guided Augmentation with the 2) Rapid action of Accelerated TMS?



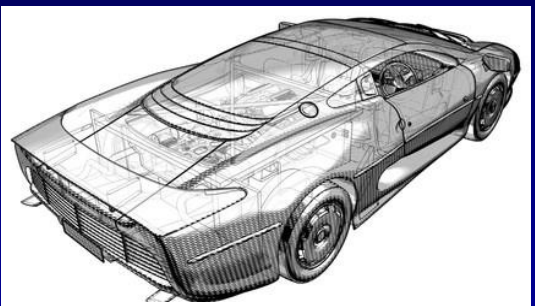
TMS



TMS mechanisms



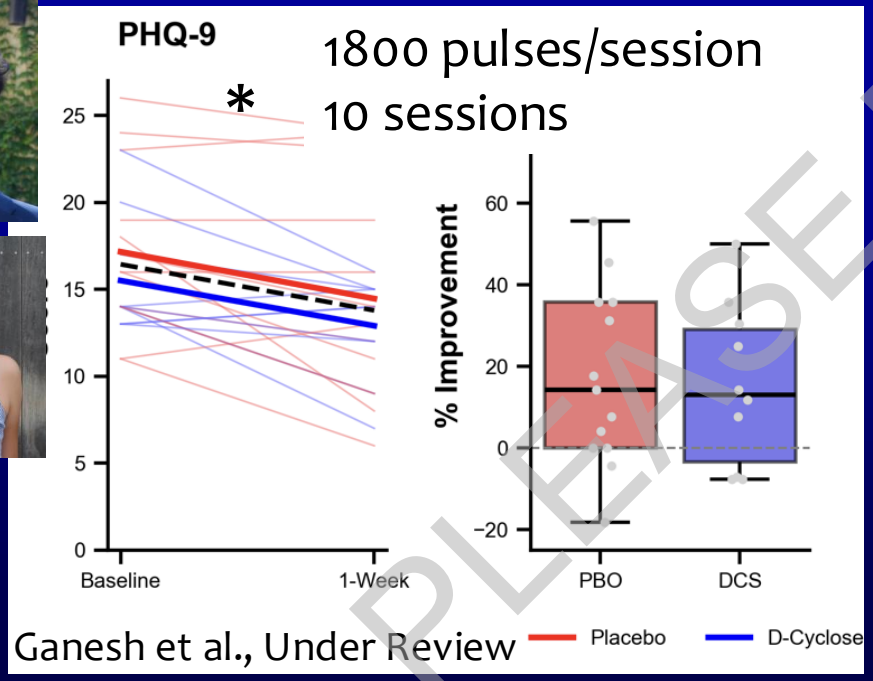
aTMS



Next Gen TMS??





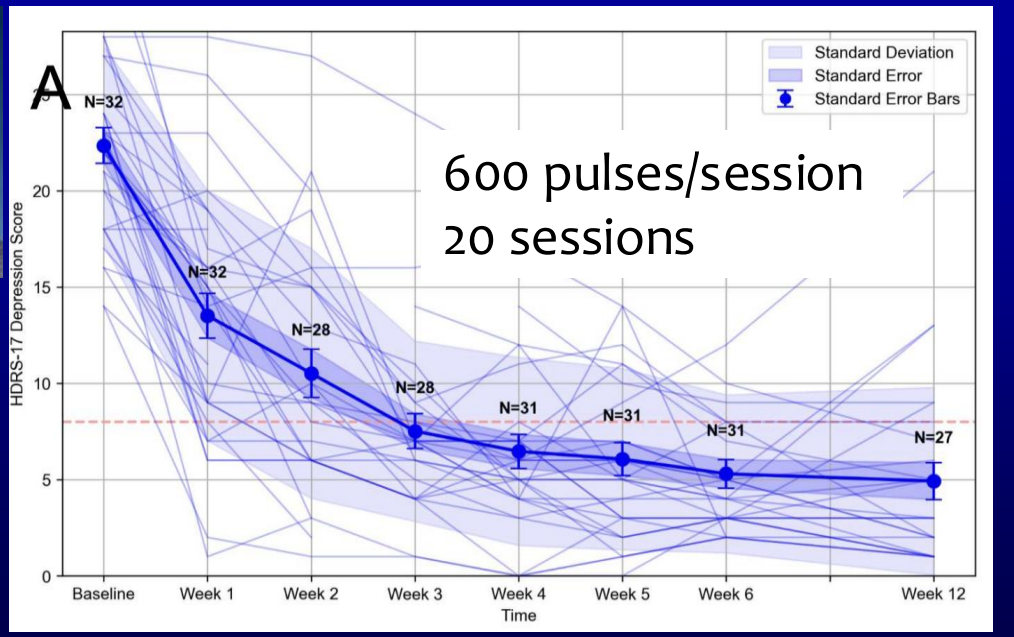



1800 pulses/session
10 sessions

Ganesh et al., Under Review



?




600 pulses/session
20 sessions

Vaughn et al., *TMS*, 2025

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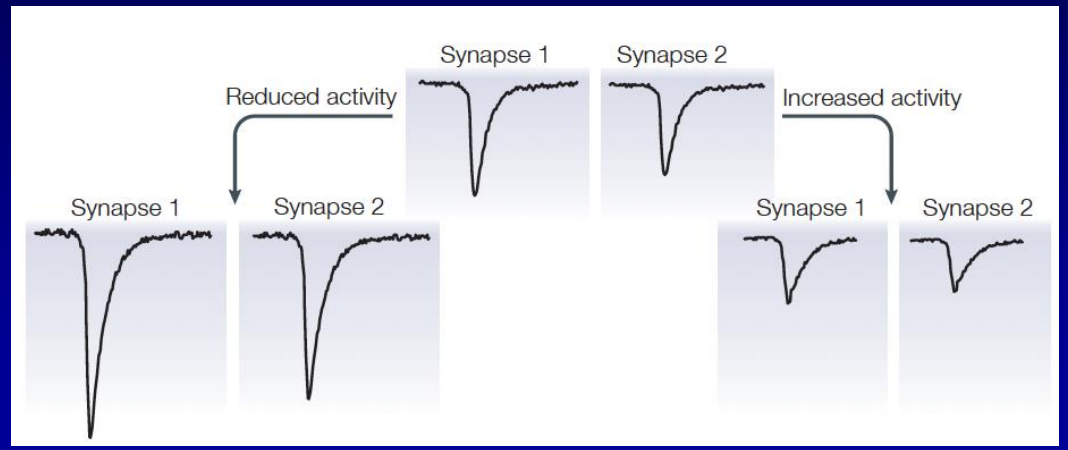
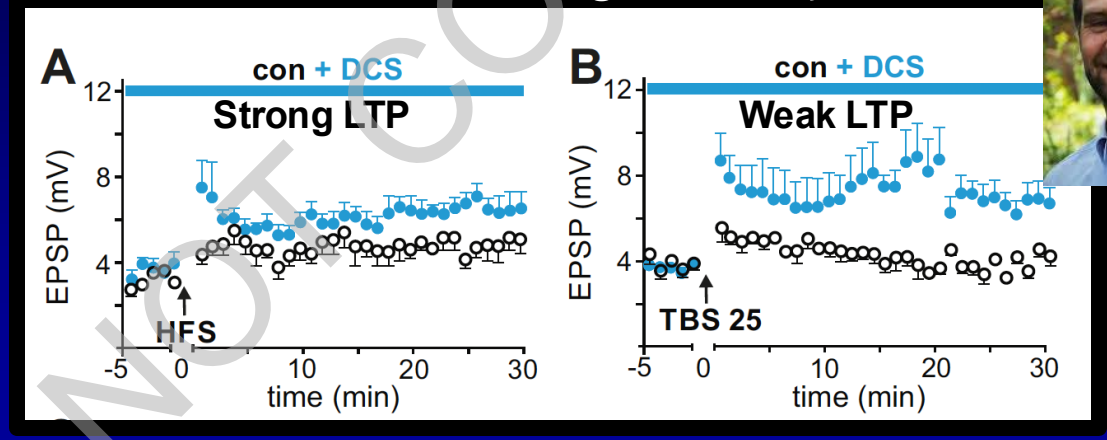
# Like Cake:
# There is too much of a good thing



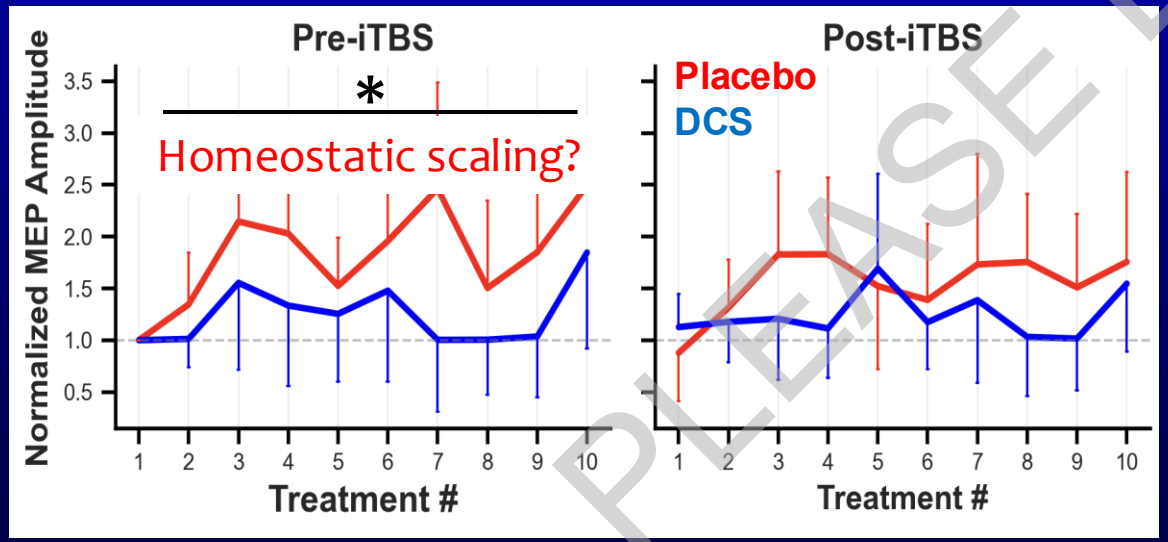

Turrigiano et al, *Nature Reviews Neuroscience*, 2004

Vestring, *Transl Psych*, 2024



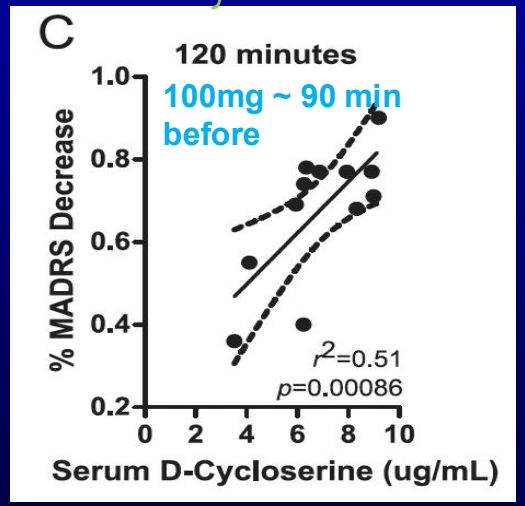



# Cortical Excitability+/- DCS



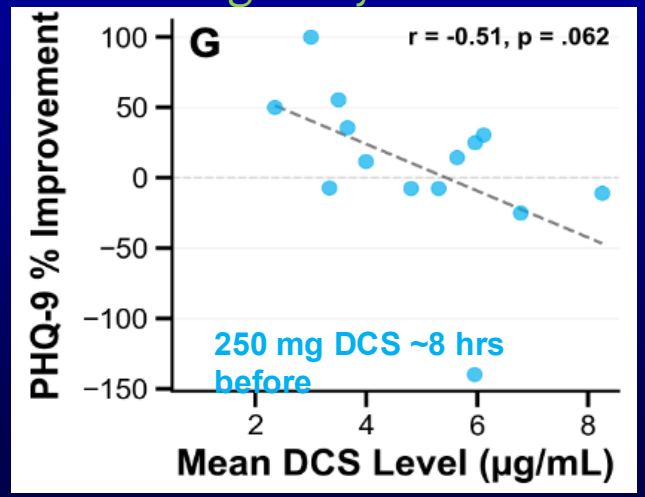

# DCS serum concentrations

Daily 600-iTBS




DeMayo et al., *J Affect Dis*, 2025

Single Day 1800-iTBS




Ganesh et al., Under Review

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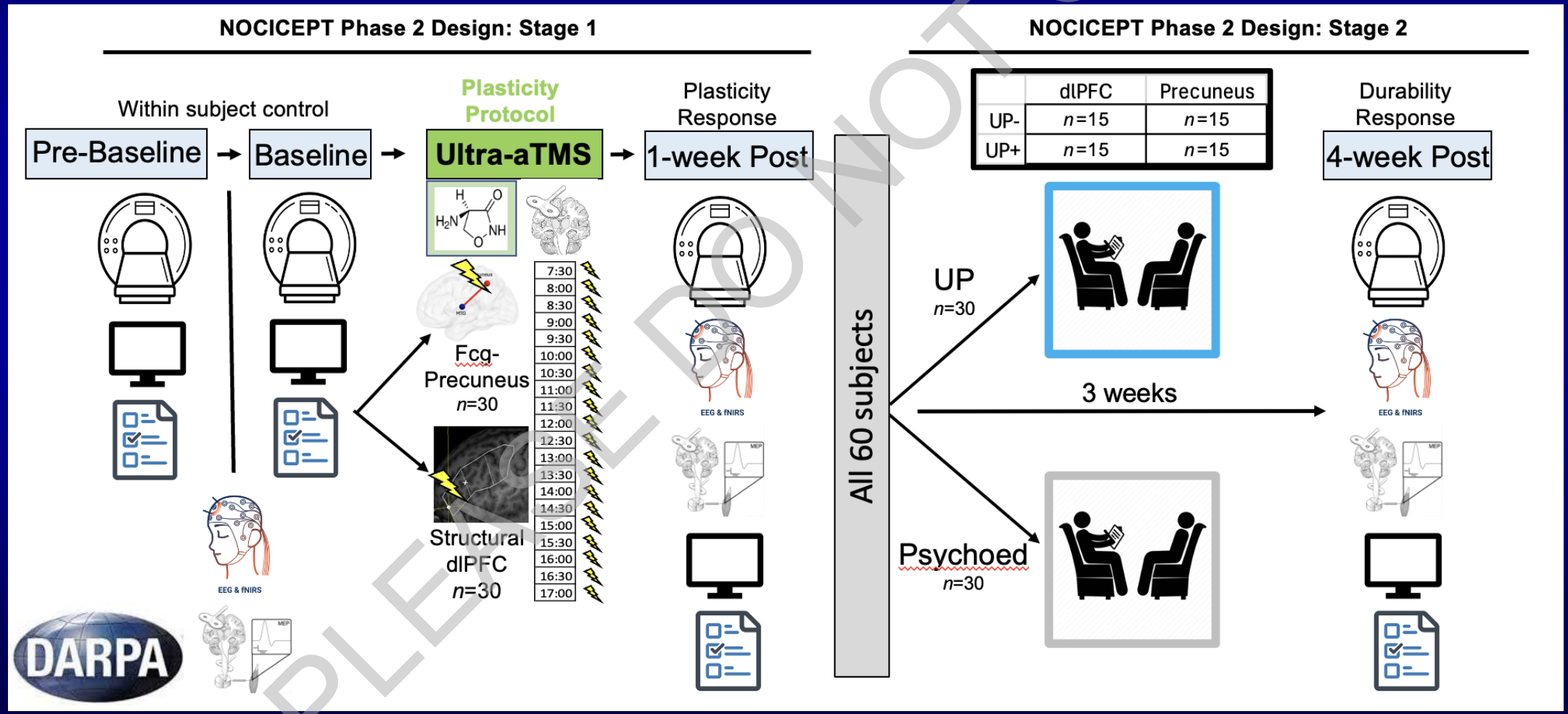
# Single day ultra-accelerated TMS course: DLPFC v. Precuneus RCT →

## Synaptic Plasticity-Augmented Rapid Circuit Stimulation (SPARC-TMS)

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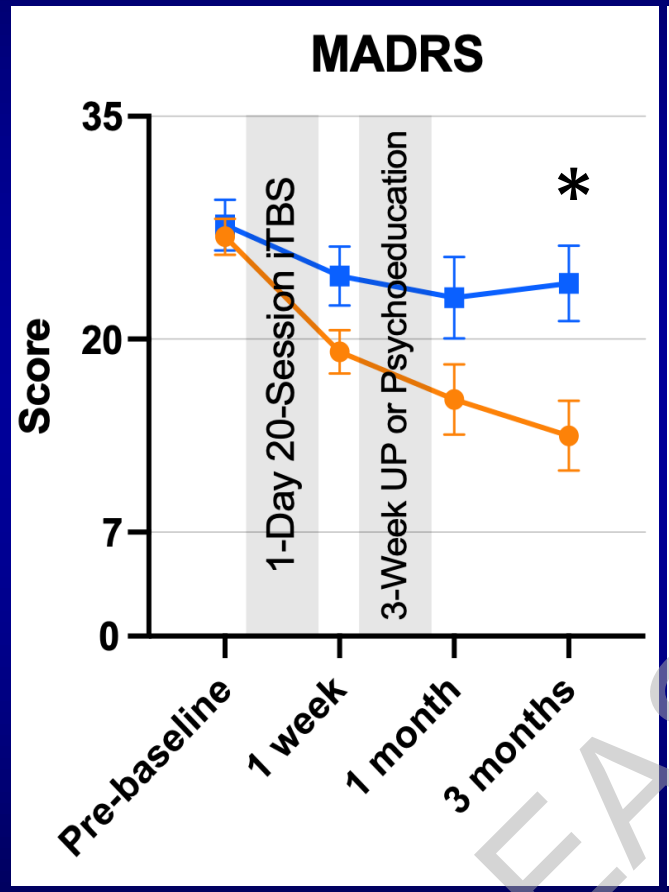
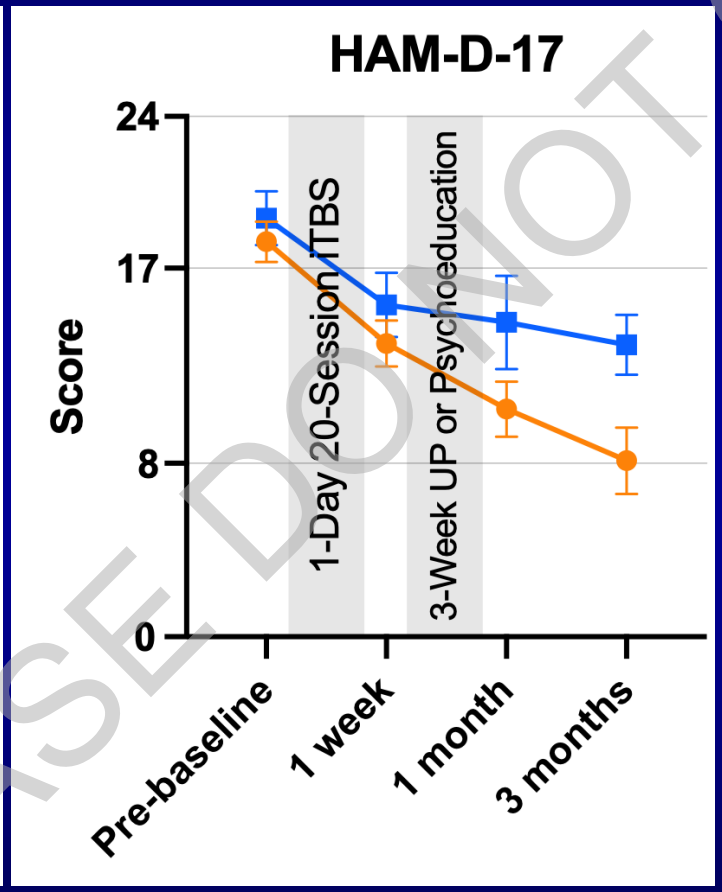
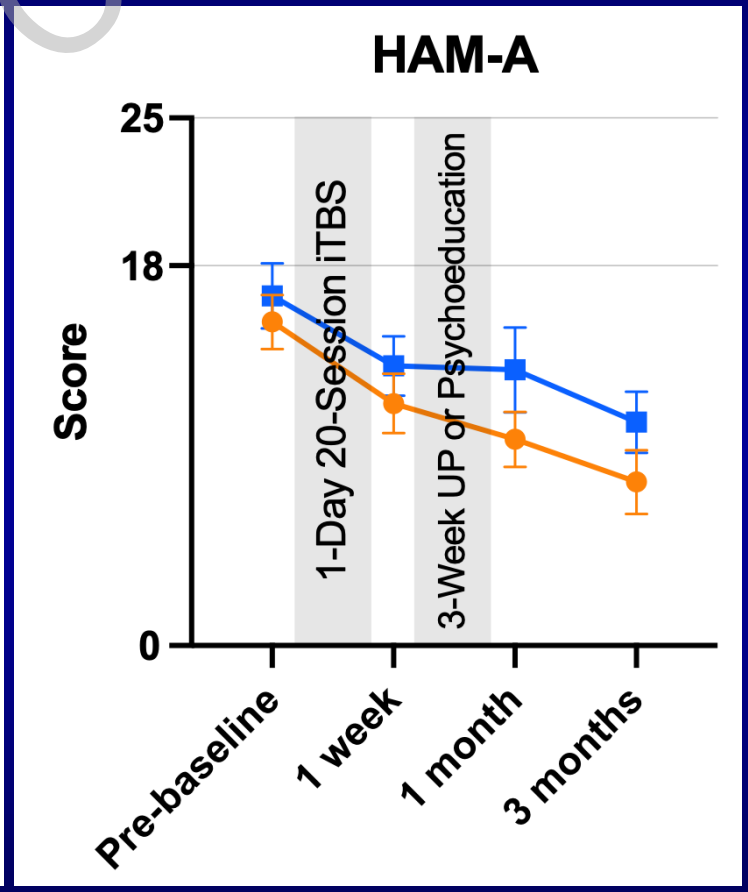
# Dorsolateral Prefrontal Cortex vs. Parietal (with functional connectivity to precuneus)




DLPFC (Structural)
IPL (FCG-Precuneus)

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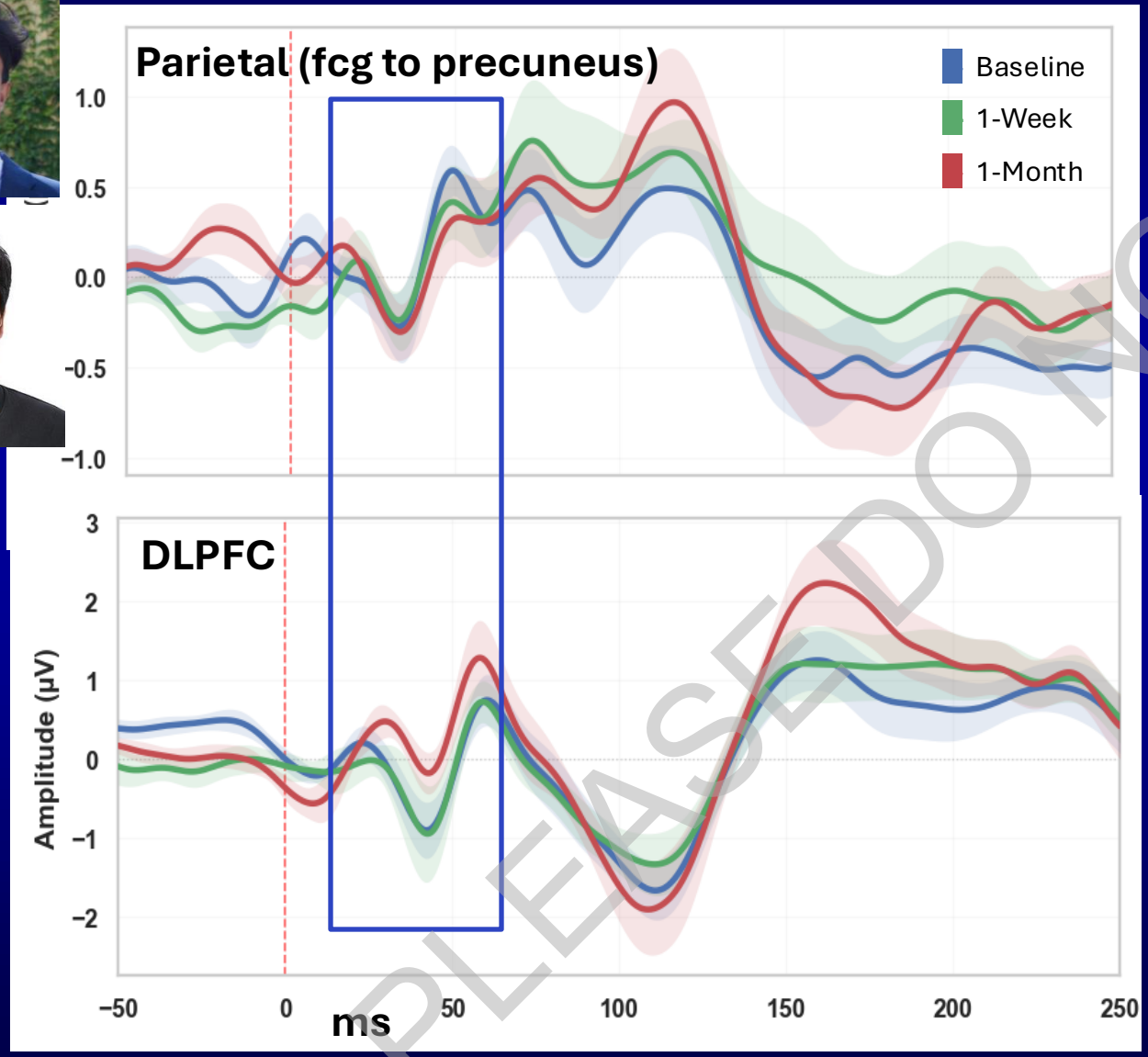
# Plasticity induced by 1-day of TMS over 1 month:

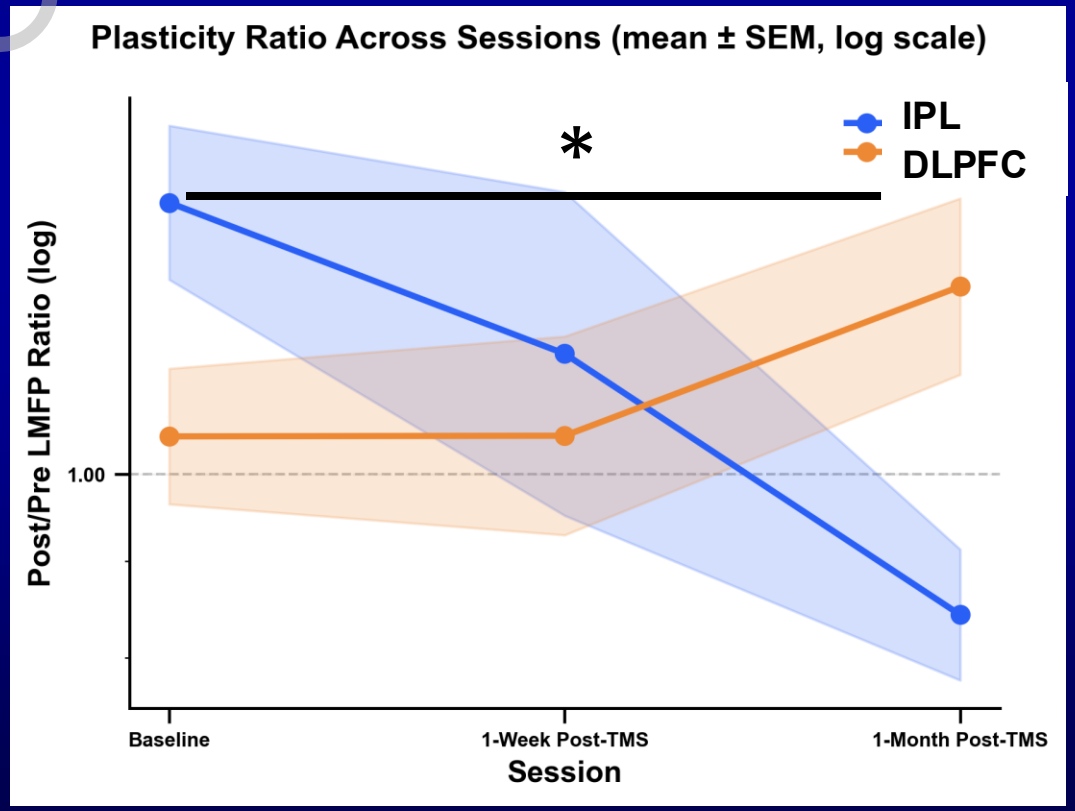




Local Mean Field Potential




## DLPFC Potentiates
## IPL Depresses

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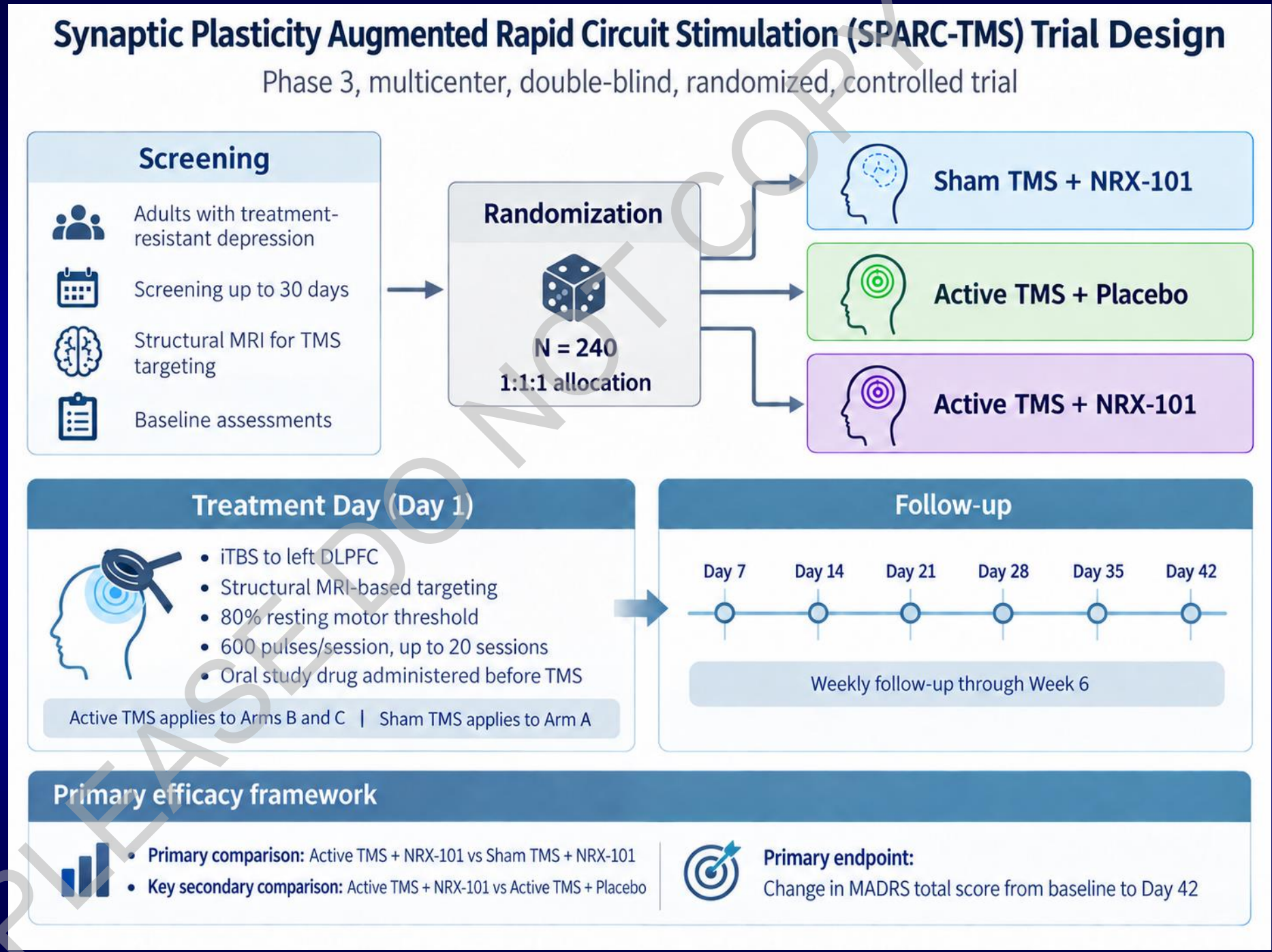
# What's next?

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# Recap

- NMDAR agonist, d-cycloserine, enhances TMS effectiveness
  - …Through NMDA receptor activation
  - …Which is central to LTP
  - …suggests TMS works through LTP.
  - May be Transdiagnostic!
  - May allow for *single day* protocols! → Sham and Placebo controlled pivotal RCT funded, to start Sept 1.

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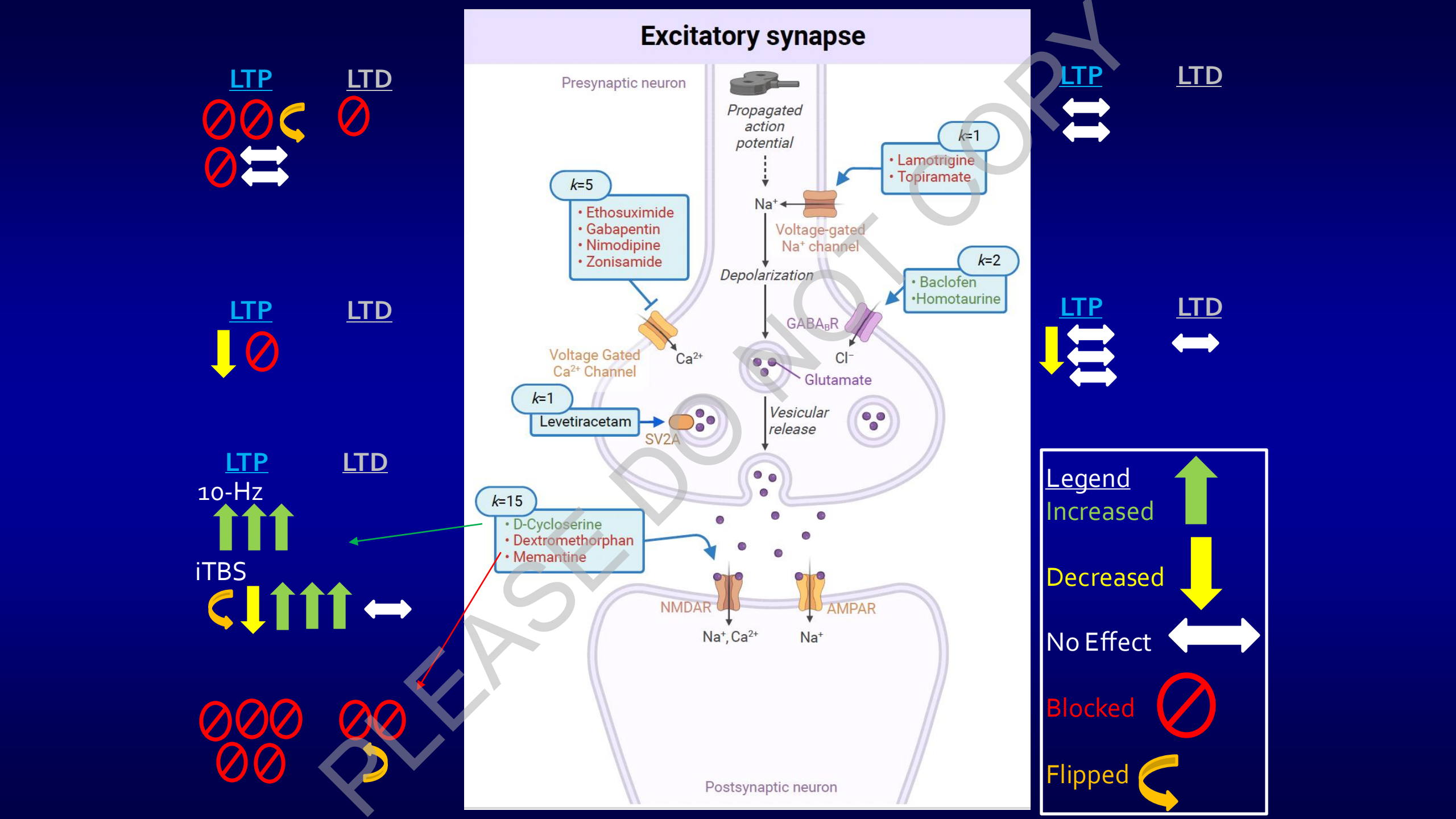

Excitatory synapse
Presynaptic neuron
Propagated action potential
k=1
• Lamotrigine
• Topiramate
Na+
Voltage-gated Na+ channel
k=5
• Ethosuximide
• Gabapentin
• Nimodipine
• Zonisamide
Depolarization
k=2
• Baclofen
•Homotaurine
GABA$_B$R
Cl−
Voltage Gated Ca2+ Channel
Ca2+
Glutamate
k=1
Levetiracetam
SV2A
Vesicular release
k=15
• D-Cycloserine
• Dextromethorphan
• Memantine
NMDAR
AMPAR
Na+, Ca2+
Na+
Postsynaptic neuron
LTP
LTD
10-Hz
iTBS
Legend
Increased
Decreased
No Effect
Blocked
Flipped
PLEASE DO NOT COPY

Legend

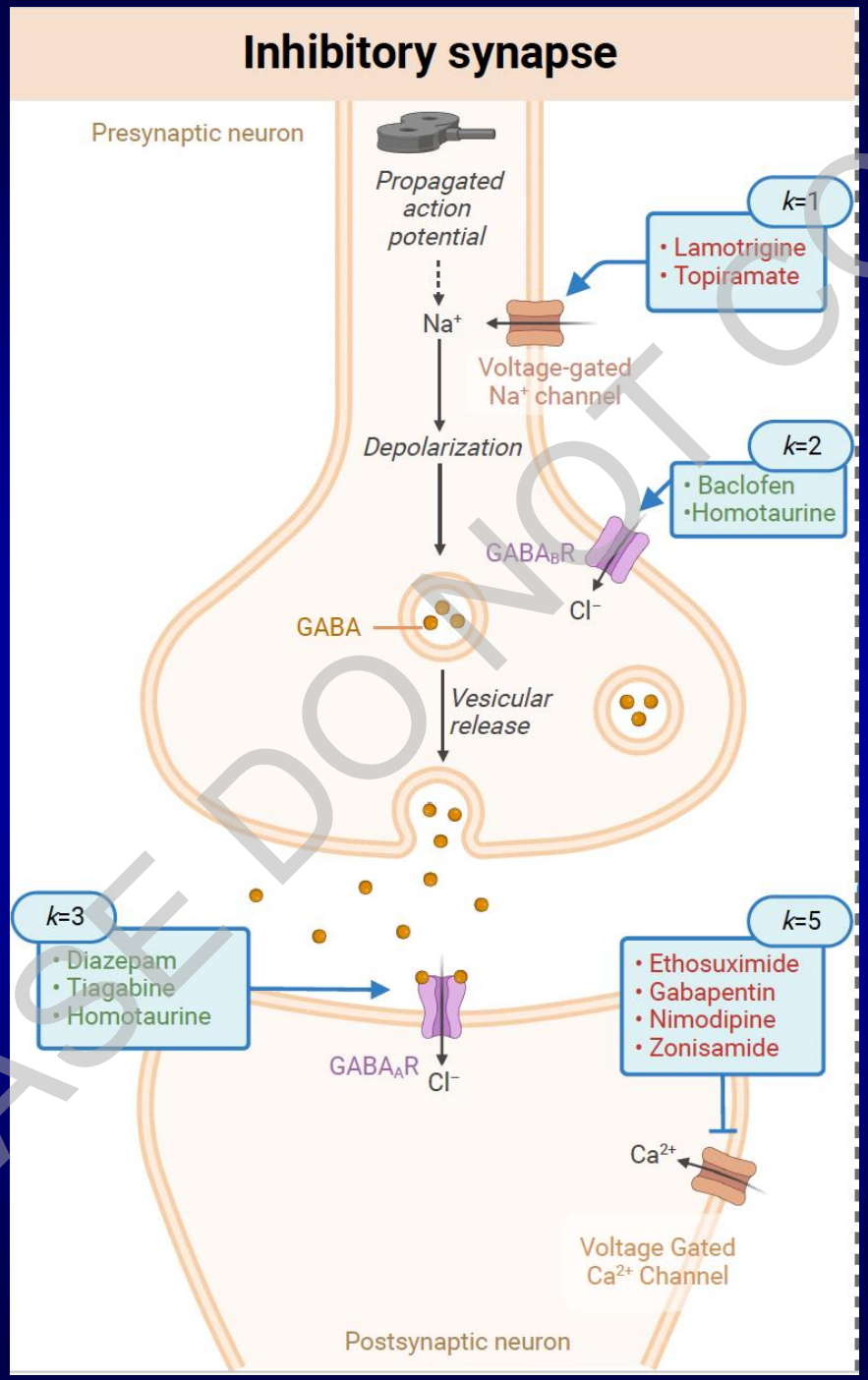
Increased

Decreased

No Effect

Blocked

Flipped




LTP LTD

LTP LTD

LTP LTD

LTP LTD

Clinical:

Prelim Data:

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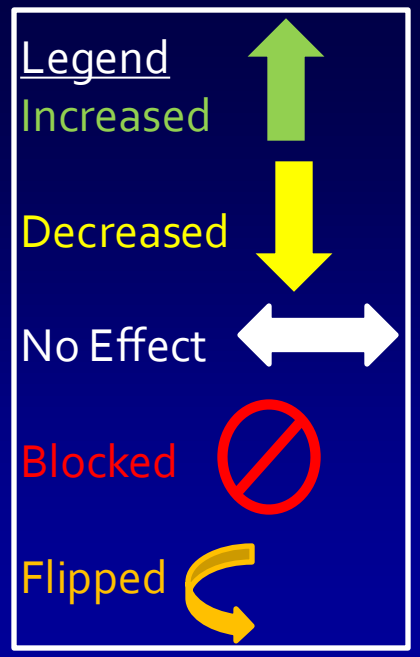


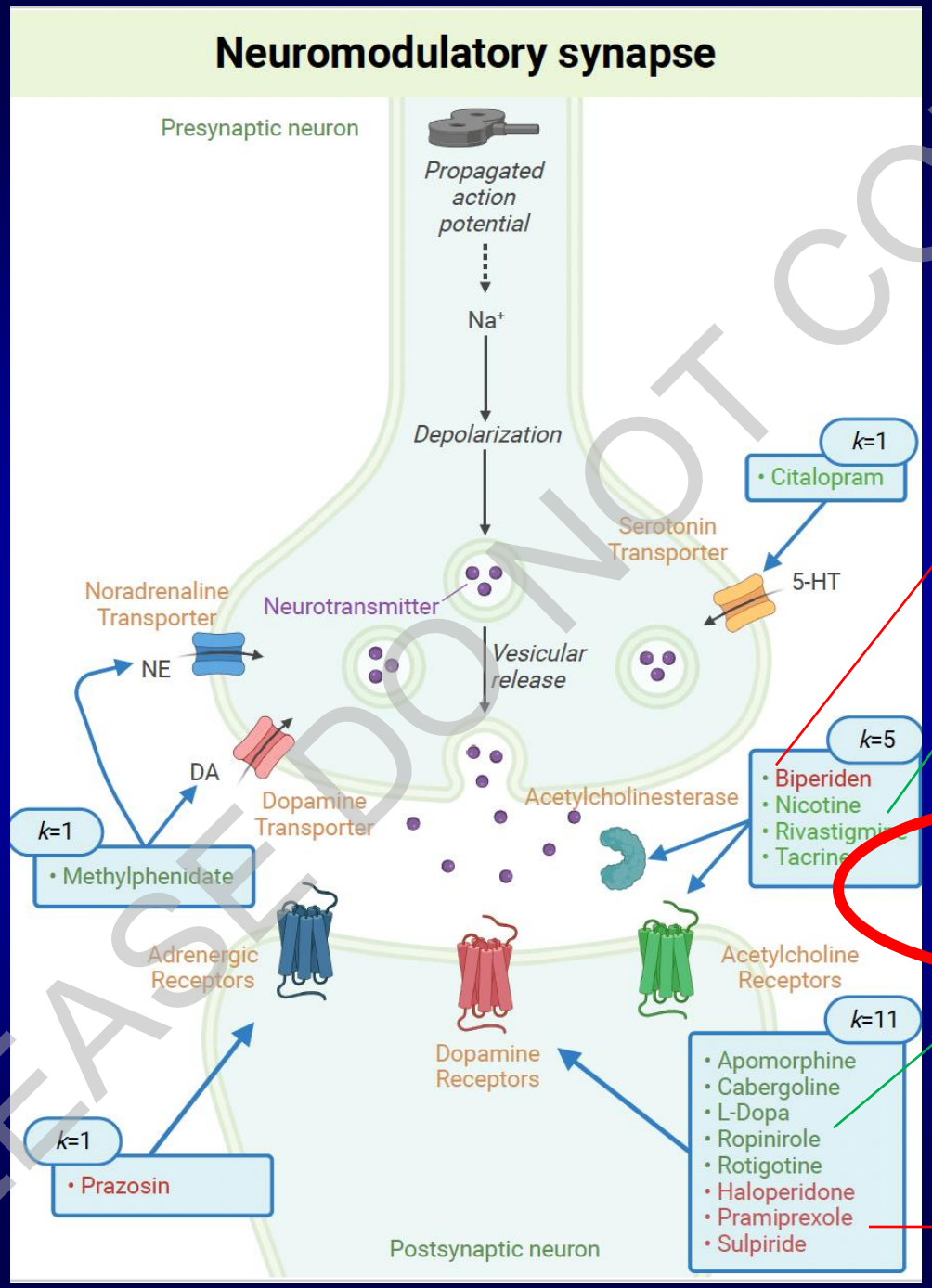


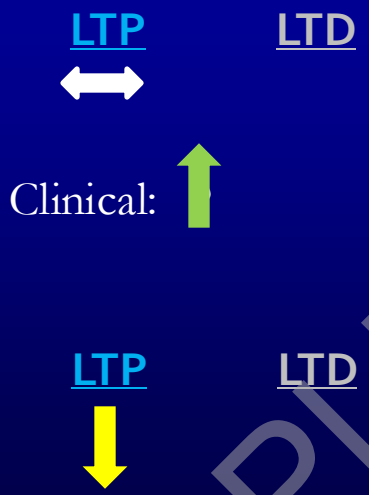


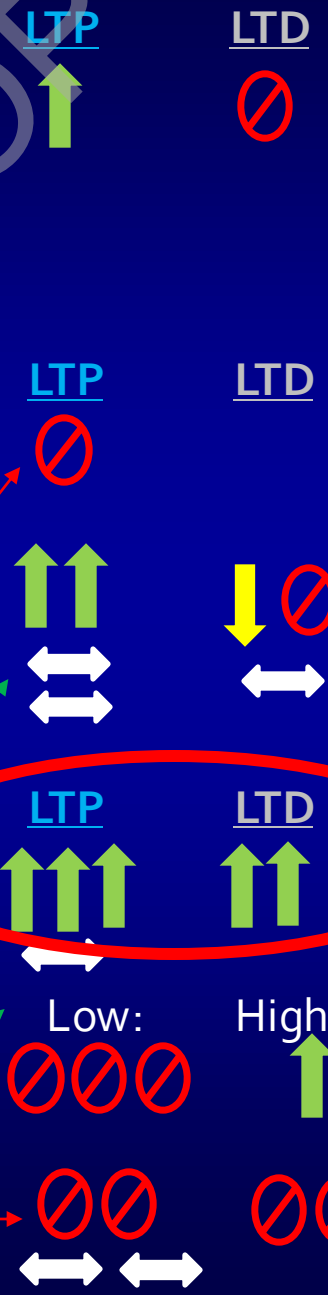


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What role do daily medications have in TMS response?

What recommendations should we make for our patients?

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# Is TMS better with Meds?



REVIEW

A Review of Transcranial Magnetic Stimulation and Transcranial Direct Current Stimulation Combined with Medication and Psychotherapy for Depression

Brian Kochanowski, MA, Karina Kageki-Bonnert, Elizabeth A. Pinkerton, BS, Darin D. Dougherty, MD,* and Tina Chou, PhD*

"Concurrent antidepressant or mood stabilizer therapy was associated with a higher rate of response." (Fitzgerald, 2006)

| TMS + MEDICATIONS | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Fitzgerald, et al. (2006)[55] | Phase 1:<br>67 received 1 Hz<br>63 received 2 Hz<br>Phase 2: (offered to nonresponders)<br>16 received 5 Hz<br>14 received 10 Hz | Randomized, controlled | Different frequencies, different target | Phase 1: 1 or 2 Hz rTMS to right PFC at 110% MT, 900-1800 pulses<br>Phase 2: 5 or 10 Hz rTMS to left PFC at 100% MT, ITI 20-25 seconds, 1500 pulses | 10 rTMS sessions per phase | Stable dose of ongoing antidepressant or mood stabilizer | HAM-D<br>BDI | 1 Hz:<br>HAM-D - 63.3% dec<br>BDI - 63.5% dec<br>2 Hz:<br>HAM-D - 66.4% dec<br>BDI - 58.8% dec<br>5 Hz:<br>HAM-D - 20.5% dec | Significant reduction in symptoms |

"The medication-free patients… had given up on medication treatment usually after multiple failed trials… they may be a different… subgroup." (Fitzgerald, 2016)

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| TMS + MEDICATIONS | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Fitzgerald, et al. (2006)[55] | Phase 1:<br>67 received 1 Hz<br>63 received 2 Hz<br>Phase 2: (offered to nonresponders)<br>16 received 5 Hz<br>14 received 10 Hz | Randomized, controlled | Different frequencies, different target | Phase 1: 1 or 2 Hz rTMS to right PFC at 110% MT, 900-1800 pulses<br>Phase 2: 5 or 10 Hz rTMS to left PFC at 100% MT, ITI 20-25 seconds, 1500 pulses | 10 rTMS sessions per phase | Stable dose of ongoing antidepressant or mood stabilizer | HAM-D<br>BDI | 1 Hz:<br>HAM-D - 63.3% dec<br>BDI - 63.5% dec<br>2 Hz:<br>HAM-D - 66.4% dec<br>BDI - 58.8% dec<br>5 Hz:<br>HAM-D - 20.5% dec<br>BDI - 22.4% dec<br>10 Hz:<br>HAM-D - 37% dec<br>BDI - 32.2% dec | Significant reduction in symptoms |
| Zhang, et al. (2019)[56] | 117 | Open-label | N/A | 10 Hz over left dlPFC at 120% MT, 80 trains, ITI 12 seconds, 2400 pulses | At least 10 rTMS sessions | Stable dose of one antidepressant | HAM-D | 51.5% dec | Significant reduction in symptoms |
| Wall, et al. (2011)[57] | 8 | Open-label | N/A | 10 Hz over left dlPFC at 120% | 30 rTMS sessions | Stable dose of ongoing SSRI and ongoing | CDRS-R | 50.5% dec | Significant reduction in symptoms |
| Hansen, et al. (2004)[50] | 6 active rTMS + medication<br>7 sham rTMS + medication (unipolar and bipolar depression) | | | 60 seconds | | | | MS + antidepressant:<br>c<br>S + antidepressant:<br>34.6% dec | Poor tolerability of rTMS and high drop-out rates |
| Wilke, et al. (2022)[47] | 37 rTMS + psychostimulants<br>Wilke, et al. (2022)<br>53 rTMS only | Retrospective | rTMS only | 10 Hz over left dlPFC at up to 120% MT, 40-pulse train, ITI 26 seconds, 3000 pulses | 30 rTMS sessions | Stable dose of ongoing psychostimulant | IDS-SR | rTMS + Psychostimulant: 43.8% dec<br>rTMS only: 29.8% dec | Combination superior |
| Berlim, et al. (2014)[58] | 17 | Open-label | N/A | 20 Hz over left dlPFC at 120% MT, 75 trains of 2 seconds, ITI 20 seconds, 3000 pulses | 20 rTMS sessions | Stable dose of ongoing medications (no benzodiazepines) | HAM-D<br>QIDS | HAM-D - 50.9% dec<br>QIDS - 27.1% dec | Significant reductions in symptoms |
| Garcia-Toro, et al. (2001)[52] | 17 active rTMS + medication<br>18 sham rTMS + medication | Randomized, controlled, double-blind | Sham TMS (coil angled differently) | 20 Hz over left dlPFC at 90% MT, 30 trains of 2 seconds, ITI 20-40 seconds | 10 rTMS sessions | Stable dose of ongoing medications | HAM-D21<br>BDI | Active rTMS + medication:<br>HAM-D21 - 26% dec<br>BDI - 17.4% dec<br>Sham rTMS + medication:<br>HAM-D21 - 6.9% dec<br>BDI - 9.7% dec | Combination superior |

**Psychostimulants: Enhances TMS response**

*Retrospective

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| TMS + MEDICATIONS | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Schüle, et al. (2003)[41] | 26 | Open-label | N/A | 10 Hz over left dlPFC at 100% MT, 15 trains of 10 seconds, ITI 30 seconds | 10-13 rTMS sessions | Mirtazapine, 45 mg/day or mirtazapine plus newly started lithium, carbamazepine or neuroleptics after full course of rTMS sessions | HAM-D | rTMS + mirtazapine (monotherapy): 38.8% dec | Combination reduced symptoms |
| Rumi, et al. (2005)[42] | 22 active rTMS + amitriptyline<br>24 sham rTMS + amitriptyline | Randomized, controlled, double-blind | Sham TMS (sham coil) | 5 Hz over left dlPFC at 120% MT, 25 trains of 10 seconds, ITI 20 seconds, 1250 pulses | 20 rTMS sessions | Amitriptyline, average dose was 110 mg/day (clonazepam allowed) | HAMD-17<br>MADRS | *Estimated from graph<br>Active rTMS + medication:<br>HAMD-17 ~ 62% dec<br>MADRS ~61% dec<br>Sham rTMS + medication:<br>HAMD-17 ~ 22% dec<br>MADRS ~23% dec | Combination superior, also accelerated symptom reduction at 1 week into treatment |
| Hu, et al. (2016)[43] | 12 left 10 Hz rTMS + quetiapine<br>13 right 1 Hz rTMS + quetiapine<br>13 sham + quetiapine<br>(bipolar II depression) | | | | | | | ted from graph<br>rTMS + medication:<br>D17 ~ 46% dec<br>S ~57% dec<br>TMS + medication:<br>D17 ~ 47% dec<br>S~59% dec<br>TMS + medication:<br>D17 ~ 41%<br>MADRS ~49% dec | Combination not superior |
| Hebel, et al. (2020)[44] | 182 rTMS + drugs for psychosis<br>117 rTMS + no drugs for psychosis | Retrospective | rTMS only | Mostly 10 Hz over left dlPFC | Different protocols | Antipsychotics | HAM-D21<br>HAM-D17 | rTMS + antipsychotics:<br>HAM-D21 - 25.2% dec<br>HAM-D17 - 25.4% dec<br>rTMS only:<br>HAM-D21 - 36.9% dec<br>HAM-D17 - 38.9% dec | Antipsychotics interfere with TMS response |
| Deppe, et al. (2020)[45] | 176 not taking benzodiazepines<br>73 taking lorazepam | Retrospective | Different protocols | Left, right, bilateral dorsolateral, or dorsomedial PFC | Different protocols | Lorazepam | HAM-D21<br>HAM-D17 | No benzodiazepines:<br>HAM-D21- 34.2% dec<br>HAM-D17 - 35.7% dec<br>Lorazepam:<br>HAM-D21 - 18.8% dec<br>HAM-D17 - 18.9% dec | Lorazepam interferes with TMS response |
| Cole, et al. (2022)[46] | 25 iTBS + placebo<br>25 iTBS + D-CS | Randomized, controlled, double-blind | Placebo capsules | Left dlPFC at 80% MT, 20 trains of triplets at 50 Hz repeated at 5 Hz, 600 pulses | 20 iTBS sessions | D-cycloserine, 100 mg at least 1 hour before iTBS | MADRS<br>QIDS | iTBS + D-CS:<br>MADRS - 56.8% dec<br>QIDS - 44.4% dec<br>iTBS + placebo:<br>MADRS - 34.7% dec<br>QIDS - 32.3% dec | Combination superior |

**Antipsychotics: interfere with TMS response**
**Lorazepam: interferes with TMS response**
*Both Retrospective

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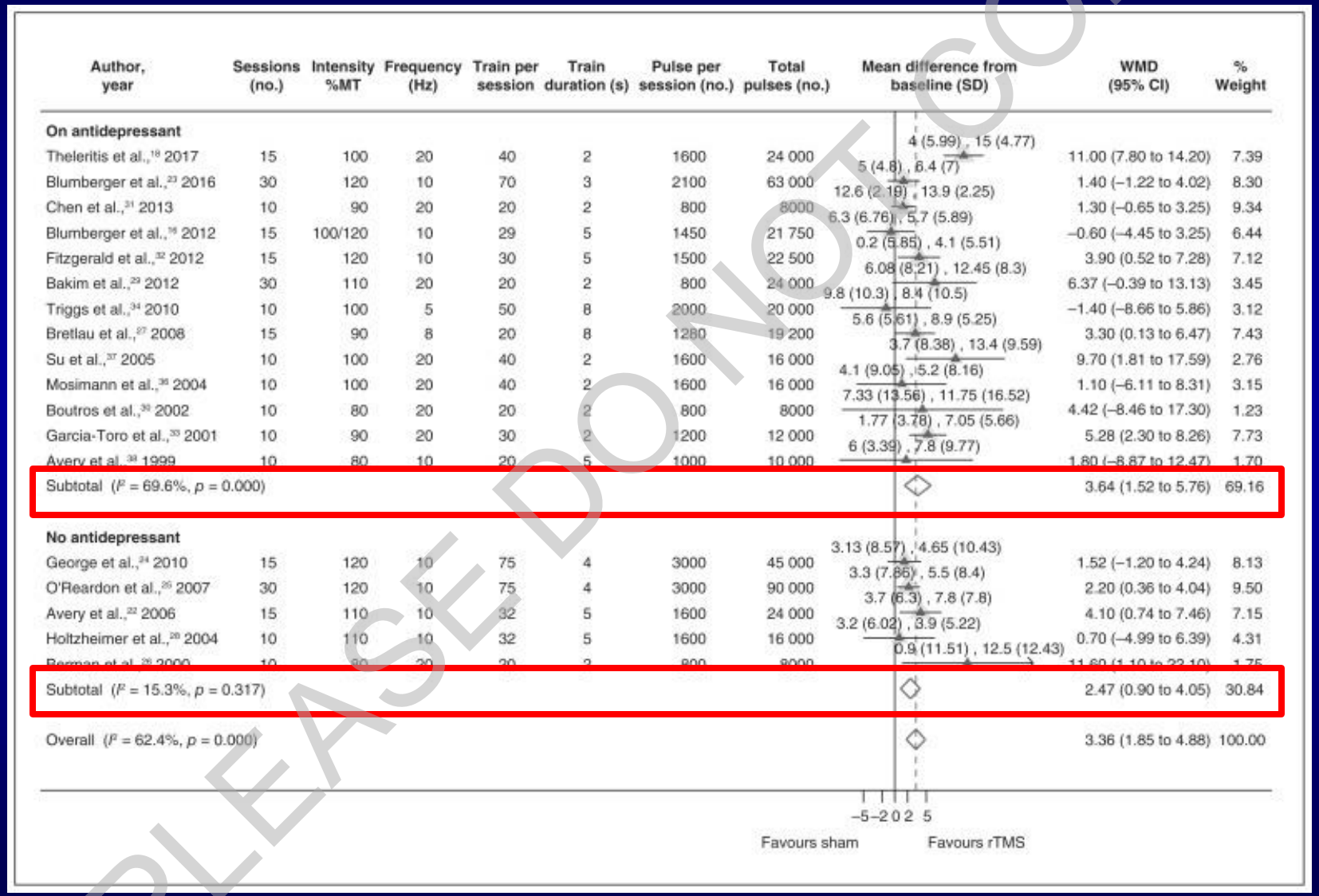
# Is TMS better with Meds?



| Author, year | Sessions (no.) | Intensity %MT | Frequency (Hz) | Train per session | Train duration (s) | Pulse per session (no.) | Total pulses (no.) | Mean difference from baseline (SD) | WMD (95% CI) | % Weight |
|---|---|---|---|---|---|---|---|---|---|---|
| **On antidepressant** | | | | | | | | | | |
| Theleritis et al.,[18] 2017 | 15 | 100 | 20 | 40 | 2 | 1600 | 24 000 | 4 (5.99) , 15 (4.77) | 11.00 (7.80 to 14.20) | 7.39 |
| Blumberger et al.,[23] 2016 | 30 | 120 | 10 | 70 | 3 | 2100 | 63 000 | 5 (4.8) , 6.4 (7) | 1.40 (–1.22 to 4.02) | 8.30 |
| Chen et al.,[31] 2013 | 10 | 90 | 20 | 20 | 2 | 800 | 8000 | 12.6 (2.19) , 13.9 (2.25) | 1.30 (–0.65 to 3.25) | 9.34 |
| Blumberger et al.,[16] 2012 | 15 | 100/120 | 10 | 29 | 5 | 1450 | 21 750 | 6.3 (6.76) , 5.7 (5.89) | –0.60 (–4.45 to 3.25) | 6.44 |
| Fitzgerald et al.,[32] 2012 | 15 | 120 | 10 | 30 | 5 | 1500 | 22 500 | 0.2 (5.85) , 4.1 (5.51) | 3.90 (0.52 to 7.28) | 7.12 |
| Bakim et al.,[29] 2012 | 30 | 110 | 20 | 20 | 2 | 800 | 24 000 | 6.08 (8.21) , 12.45 (8.3) | 6.37 (–0.39 to 13.13) | 3.45 |
| Triggs et al.,[34] 2010 | 10 | 100 | 5 | 50 | 8 | 2000 | 20 000 | 9.8 (10.3) , 8.4 (10.5) | –1.40 (–8.66 to 5.86) | 3.12 |
| Bretlau et al.,[27] 2008 | 15 | 90 | 8 | 20 | 8 | 1280 | 19 200 | 5.6 (5.61) , 8.9 (5.25) | 3.30 (0.13 to 6.47) | 7.43 |
| Su et al.,[37] 2005 | 10 | 100 | 20 | 40 | 2 | 1600 | 16 000 | 3.7 (8.38) , 13.4 (9.59) | 9.70 (1.81 to 17.59) | 2.76 |
| Mosimann et al.,[36] 2004 | 10 | 100 | 20 | 40 | 2 | 1600 | 16 000 | 4.1 (9.05) , 5.2 (8.16) | 1.10 (–6.11 to 8.31) | 3.15 |
| Boutros et al.,[30] 2002 | 10 | 80 | 20 | 20 | 2 | 800 | 8000 | 7.33 (13.56) , 11.75 (16.52) | 4.42 (–8.46 to 17.30) | 1.23 |
| Garcia-Toro et al.,[33] 2001 | 10 | 90 | 20 | 30 | 2 | 1200 | 12 000 | 1.77 (3.78) , 7.05 (5.66) | 5.28 (2.30 to 8.26) | 7.73 |
| Avery et al.,[28] 1999 | 10 | 80 | 10 | 20 | 5 | 1000 | 10 000 | 6 (3.39) , 7.8 (9.77) | 1.80 (–8.87 to 12.47) | 1.70 |
| Subtotal ($I^2$ = 69.6%, $p$ = 0.000) | | | | | | | | | 3.64 (1.52 to 5.76) | 69.16 |
| **No antidepressant** | | | | | | | | | | |
| George et al.,[24] 2010 | 15 | 120 | 10 | 75 | 4 | 3000 | 45 000 | 3.13 (8.57) , 4.65 (10.43) | 1.52 (–1.20 to 4.24) | 8.13 |
| O'Reardon et al.,[25] 2007 | 30 | 120 | 10 | 75 | 4 | 3000 | 90 000 | 3.3 (7.86) , 5.5 (8.4) | 2.20 (0.36 to 4.04) | 9.50 |
| Avery et al.,[22] 2006 | 15 | 110 | 10 | 32 | 5 | 1600 | 24 000 | 3.7 (6.3) , 7.8 (7.8) | 4.10 (0.74 to 7.46) | 7.15 |
| Holtzheimer et al.,[26] 2004 | 10 | 110 | 10 | 32 | 5 | 1600 | 16 000 | 3.2 (6.02) , 3.9 (5.22) | 0.70 (–4.99 to 6.39) | 4.31 |
| Berman et al.,[20] 2000 | 10 | 80 | 20 | 20 | 2 | 800 | 8000 | 0.9 (11.51) , 12.5 (12.43) | 11.60 (1.10 to 22.10) | 1.75 |
| Subtotal ($I^2$ = 15.3%, $p$ = 0.317) | | | | | | | | | 2.47 (0.90 to 4.05) | 30.84 |
| Overall ($I^2$ = 62.4%, $p$ = 0.000) | | | | | | | | | 3.36 (1.85 to 4.88) | 100.00 |



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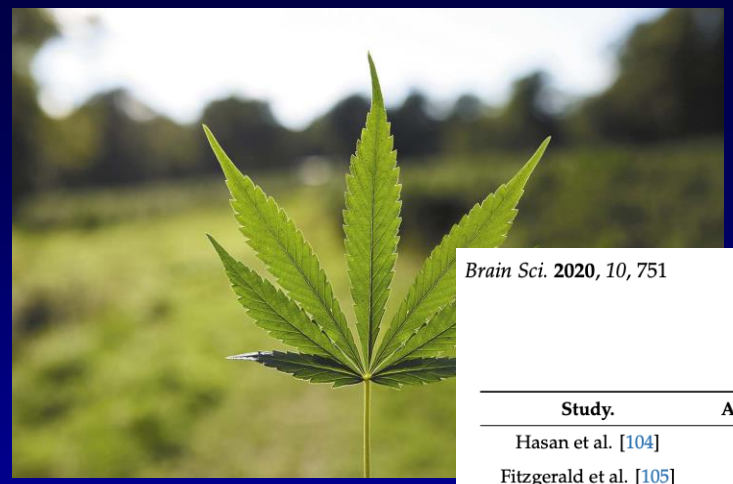
Sehatzadeh et al., *J Psychiatry Neurosci*, 2019



# What do we recommend for THC with TMS?



**Table 2.** Effects of cannabis on TMS measures.

| Study. | AMT | RMT | MEP | CSP | iSP | SAI | LAI | SICI | ICF | SICF | LICI | SIHI | LIHI | Notes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Hasan et al. [104] | – | ○ | ○ | ▲ | – | – | – | ▲ | – | – | – | – | – | Acute intake |
| Fitzgerald et al. [105] | ○ | ○ | ○ | ○ | – | – | – | ▼ | ○ | – | ○ | – | – | Heavy and light cannabis users vs. non-users |
| Martin-Rodriguez et al. [106] | ○ | ○ | ○ | – | – | – | – | ▼ | – | – | – | – | – | CUD and daily cannabis users vs. non-users |
| Wobrock et al. [107] | – | ○ | – | – | – | – | – | ▼ | ▲ | – | – | – | – | Schizophrenia cannabis users vs. non-users |
| Flavel et al. [108] | – | ○ | ○ | ○ | – | – | – | – | – | ○ | ○ | – | – | Cannabis users vs. nonusers |
| Goodman et al. [109] | – | ○ | – | ○ | – | – | – | ▲ | ○ | – | ○ | – | – | Schizophrenia cannabis users vs. non-users |
| | – | ○ | – | ○ | – | – | – | ▼ | ○ | – | ○ | – | – | Control cannabis users vs. nonusers |
| Russo et al. [110] | ○ | ○ | ○ | ○ | – | ○ | ○ | ▲ | ▼ | – | – | – | – | MS patients on 1 month of Sativex |
| Leocani et al. [111] | – | ○ | ○ | – | – | – | – | ○ | ○ | – | – | – | – | MS patients on 1 month of Sativex |
| Calabrò et al. [112] | – | – | ▲ | – | – | – | – | ▼ | ▼ | – | – | – | – | MS patients on 6 weeks of Sativex + gait training |

▲ increase; ▼decrease; ○ indicates no change; – indicates did not assess; CUD: cannabis use disorder; MS: multiple sclerosis.

Turco, *Brain Sci*, 2020

## THC–Observational Data from Butler Hospital:

(*n* of 56, 28 THC users, 28 matched)

Users: 12/28 responders, 5/28 remitters

Matched: 16/28 responders, 11/28 remitters

6 cases (Confusion, Psychosis, Sensory Changes, Panic)

-DePamphilis, *Brain Stimulation*, 2024

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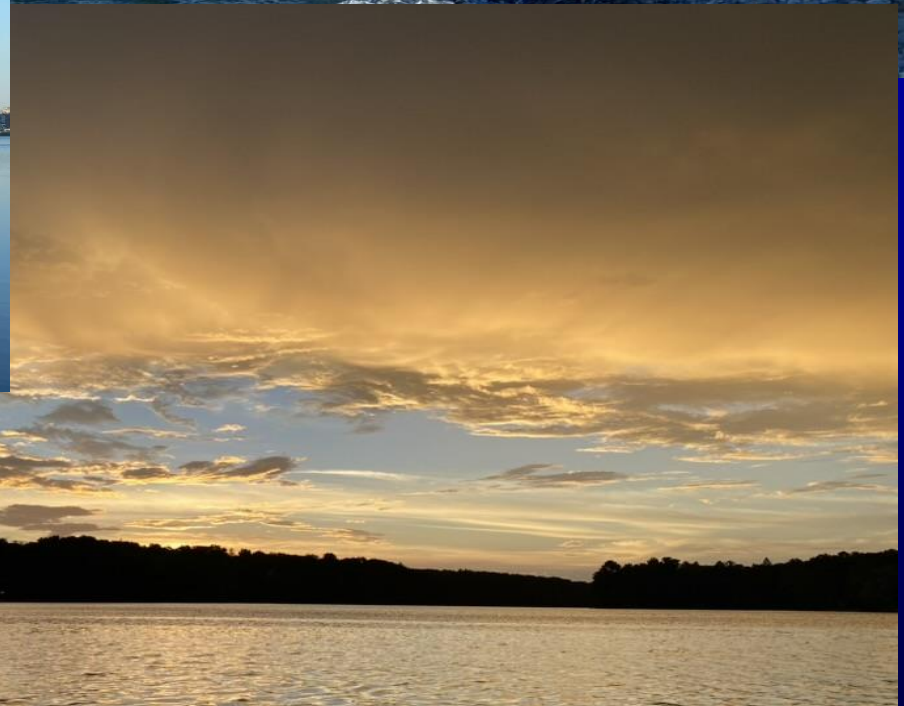
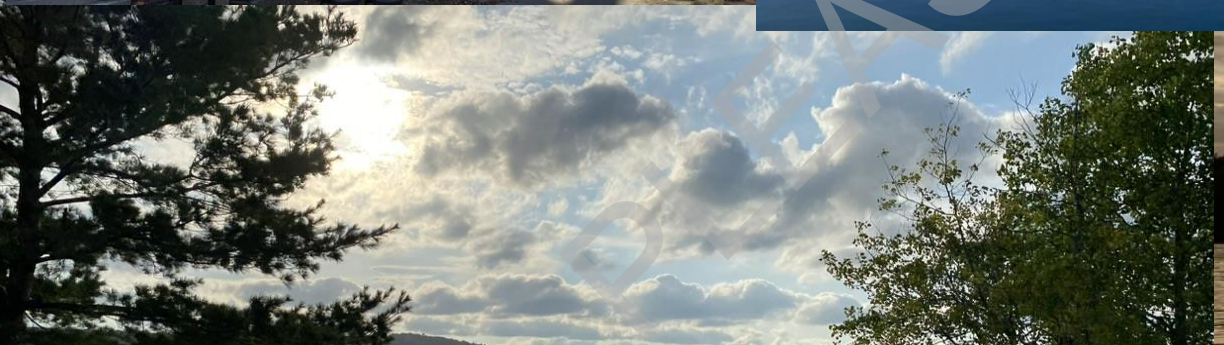
# Thank You!





## Questions?







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