

Effectiveness of transcranial magnetic stimulation for posttraumatic stress disorder: A multisite, propensity-matched cohort study of treatment parameters

Yosef A. Berlow^{a,b}, Samantha L. Cilli^a, F. Andrew Kozel^c, Amin Zandvakili^{a,b}, Noelle Marcotullio^a, Camila Cosmo^{a,b}, Miriam A. Goldberg^{a,b}, Bo Dehm Wicklund^d, Michelle R. Madore^{d,e}, Noah S. Philip^{a,b,*}

^a Center for Neurorestoration and Neurotechnology, VA Providence Healthcare System, Providence, RI, USA

^b Department of Psychiatry and Human Behavior, Alpert Medical School of Brown University, Providence, RI, USA

^c Department of Behavioral Sciences and Social Medicine, Florida State University College of Medicine, Florida State University, Tallahassee, FL, 32306, USA

^d Mental Illness Research, Education and Clinical Center, VA Palo Alto Health Care System, Palo Alto, CA, 94304, USA

^e Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Stanford, CA, 94305, USA

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ABSTRACT

Background: Transcranial magnetic stimulation (TMS) is increasingly used off-label for posttraumatic stress disorder (PTSD), often applying protocols developed for depression. While prior studies suggest high-frequency TMS can improve PTSD symptoms, few have been adequately powered to compare protocols. We examined whether three common TMS protocols yield equivalent outcomes for PTSD in a large, multisite cohort of veterans.

Methods: Clinical outcomes were analyzed from 756 veterans with comorbid PTSD and depression treated with antidepressant TMS across multiple VA sites. Protocols included left prefrontal 10 Hz TMS (n = 526), left prefrontal intermittent theta burst TMS (iTBS; n = 71), and deep TMS (dTMS; n = 61). PTSD symptoms were measured with the PTSD symptom checklist for DSM-5 (PCL-5). Primary outcomes included PTSD response (>10-point reduction) and remission (PCL-5 <33). Propensity score matching adjusted for baseline differences. Equivalence was set at 10 % for categorical outcomes and Cohen's d = 0.25 for continuous outcomes. Depression outcomes were also analyzed.

Results: All three TMS protocols produced substantial PTSD symptom reductions (18–22 points). PTSD response rates were 63 % (10 Hz), 65 % (iTBS), and 78 % (dTMS); remission rates were 47 %, 48 %, and 49 %, respectively. Both iTBS and dTMS were noninferior to 10 Hz (all ps < 0.05). Depression outcomes demonstrated similar patterns.

Discussion: In this large multisite cohort study of veterans with PTSD and depression, 10 Hz, iTBS, and dTMS protocols demonstrated comparable clinical effectiveness. Limitations are those inherent to cohort studies of veterans. These findings support the effectiveness of TMS for PTSD, and protocol selection based on patient-specific or logistical considerations rather than efficacy differences.

1. Introduction

Posttraumatic stress disorder (PTSD) is a prevalent psychiatric disorder characterized by intrusive thoughts and recollections, avoidance of trauma-related stimuli, hyperarousal, and disturbances in mood and cognition [1,2]. This disabling disorder is often comorbid with medical and psychiatric disorders, substance use disorders, and suicide risk

[3–5]. Evidence-based treatments for PTSD include psychotherapy and pharmacology; newer options, such as amygdala-driven neurofeedback based on data from a multicenter open-label study, are also emerging [6]. However, treatments are often insufficiently efficacious, particularly in military veterans [7].

Noninvasive brain stimulation has emerged as a potentially promising treatment option for patients with PTSD [8]. Among

* Corresponding author. Center for Neurorestoration and Neurotechnology, 830 Chalkstone Ave Providence, RI, 02908, USA.

E-mail address: Noah_Philip@Brown.edu (N.S. Philip).

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neuromodulatory approaches, therapeutic transcranial magnetic stimulation (TMS) is the most common modality. TMS operates on the principle of Faraday's law, whereby rapidly fluctuating magnetic fields induce electrical currents in cortical tissue, leading to local neuronal depolarization. These effects propagate through polysynaptic pathways to ultimately modulate brain networks associated with symptom improvement [9]. TMS is most often applied to the dorsolateral prefrontal cortex (DLPFC; except in obsessive compulsive disorder) and calibrated using an individual's cortical excitability (i.e., the motor threshold). TMS is now widely available and cleared by the US Food and Drug Administration for pharmacoresistant major depressive disorder, obsessive compulsive disorder, and nicotine cessation, utilizing various devices and protocols (for description and review of TMS, see [10–12]).

Over the last 20 years, numerous studies have supported the notion that TMS can be efficacious for PTSD, although there has been considerable variability in examining this question. Paralleling research of TMS for depression, early trials used lower cumulative exposure to TMS, which increased over time as the field became more comfortable with use. The first randomized controlled trial (RCT) included $n = 29$ participants who received 10 Hz (4000 pulses over 10 sessions), 1 Hz (1000 pulses over 10 s), or sham stimulation to the right DLPFC, and found that 10 Hz yielded superior improvements [13]. These findings were supported by work from Nam et al., [14], who found that 1 Hz right-sided TMS (18,000 pulses over 15 sessions) was superior to sham for PTSD. Later, Ahmadizadeh and Rezaei investigated the efficacy of 20 Hz bilateral stimulation to the DLPFC versus 20 Hz right-sided TMS or sham stimulation (24,000 pulses over 10 sessions). While there was no significant difference in PTSD symptom reduction between the active groups, the unilateral right-sided 20 Hz group showed greater improvement versus sham [15].

Whether different protocols might yield varied clinical outcomes in PTSD remains an open question. The few trials that directly compared different protocols have yielded inconsistent findings. Boggio et al. [16] compared left and right-sided TMS and found that higher frequency stimulation on the left yielded superior mood outcomes, whereas right-sided stimulation appeared more beneficial for anxiety. However, the simple heuristic that lower frequency stimulation on the right was superior for PTSD symptoms was challenged by a study by Kozel et al. [17], who compared right-sided DLPFC 1 Hz TMS to 10 Hz right-sided DLPFC TMS (36 sessions, 86,400 total pulses) in 44 participants. Both groups had significant improvements in PTSD, depression, and functional gains. Contrary to this study, Leong et al. [18] later reported that 1 Hz right-sided TMS improved PTSD symptoms versus 10 Hz and sham stimulation with two weeks of treatment.

Protocols other than standard TMS have also been investigated for efficacy in treating PTSD. Intermittent theta burst stimulation (iTBS), a more recent iteration of TMS that delivers a larger number of pulses in a short period of time [19], has also recently emerged as a promising protocol for PTSD. Philip et al., [20] demonstrated iTBS to the right DLPFC ($n = 50$) could yield improvements in PTSD and social and occupational functioning compared to sham, with positive outcomes retained at one year [20,21]. Similarly, Yuan et al. compared 10 Hz right DLPFC TMS to right DLPFC iTBS versus sham in 75 participants. Each group received 27,000 cumulative pulses, and while both active groups showed significantly improved PCL-5 scores versus sham, there were no significant differences between the active groups [22].

TMS can also be combined with adjunctive interventions, such as psychotherapy or trauma-focused exposure. Kozel et al. [23] combined once-weekly 1 Hz TMS with cognitive processing therapy, and showed active stimulation was superior to sham. Isserles et al. [24] combined bilateral deep TMS (H1 dTMS) at the bilateral prefrontal cortex with script-driven trauma exposure immediately before stimulation and compared its effects to dTMS with exposure to a non-traumatic event and sham dTMS with trauma exposure. While the active dTMS + trauma exposure group showed significant improvement in intrusive PTSD symptoms and reduced heart rate during trauma script exposure, these

results were contradicted by its follow-up study in 2021; using a different coil (H7 dTMS), dTMS with trauma script exposure was found to be significantly inferior to sham dTMS + trauma script exposure [25]. Currently, meta-analyses of TMS for PTSD outnumber novel RCT publications and indicate that TMS is generally effective at PTSD symptom reduction over time, with the caveat that results are inconsistent across stimulation parameters as reviewed above [26,27]. Yet, with the extant data, another interpretation is that there remains a need for more definitive evidence that active TMS is superior to sham for PTSD.

Another source of evidence on treatment parameters comes from large-scale cohort studies. Since FDA clearance of TMS for major depressive disorder (MDD) in 2008, clinicians have used TMS to treat MDD and comorbid conditions. The most extensive data supporting TMS use for PTSD to date comes from a naturalistic TMS registry study from the US Veterans Affairs (VA) Healthcare system. In the VA Clinical TMS Program, Veterans with depression can receive TMS if they meet standard FDA-clearance eligibility. As a part of this program, clinics systematically evaluate PTSD symptoms during TMS using the gold-standard PTSD Checklist for DSM-5 (PCL-5) [28]. In the initial cohort examined in this program ($n = 770$, enrolled between October 2017 and March 2020), over two-thirds of Veterans ($n = 521$; 68.4 %) had comorbid PTSD with MDD. TMS was associated with statistically significant and clinically meaningful PTSD improvements (~ 19 -point reduction on the PCL-5; Cohen's $d = 1.2$ (95 % Confidence interval [CI] 1.10–1.38); all $ps < 0.001$). Furthermore, 65.3 % achieved a clinically meaningful reduction in PTSD (defined as a >10 -point improvement on the PCL-5, the operational definition of response) and 46.1 % no longer met PTSD severity criteria after TMS (defined as a PCL-5 < 33) [29]. Follow-up studies also found that the presence of mild traumatic brain injury did not meaningfully impact outcomes [30]. As such, this registry study provides a robust source of real-world data that can be evaluated to answer questions that are otherwise challenging to address with adequate sample sizes in clinical trials.

Taken together, the extant data across studies and meta-analyses support the effectiveness of TMS for PTSD. However, it remains unclear whether any specific TMS protocol offers a therapeutic advantage for PTSD symptoms. Many comparative studies have failed to detect significant differences in efficacy across protocols, suggesting that multiple TMS approaches might be similarly effective. If this hypothesis is supported, it could broaden access to TMS for PTSD by allowing providers to confidently use the protocols already available on their devices and in their clinics. To this end, we examined data from the VA Clinical TMS program to evaluate the effectiveness of TMS for PTSD. We hypothesized that the three FDA-cleared TMS protocols for depression would yield equivalent clinical outcomes in PTSD.

2. Methods

2.1. Selection and description of participants

We conducted a retrospective comparative effectiveness study using data from Veterans with PTSD and comorbid MDD who received TMS through the VA Clinical TMS Program. This large, multisite quality improvement initiative was approved by the VA Palo Alto/Stanford Institutional Review Board. Outcomes from over 30 VA sites are represented in this analysis. Deidentified clinical, demographic, and symptom data were collected and managed using VA Research Electronic Data Capture (REDCap) systems. As described above, prior outcomes from a portion of this cohort have been published (e.g., [28], [30], [31]); all analyses described here are unique. Most devices used in the program are manufactured by Magstim (Wales, UK) with modest representation from other devices (Magventure, Denmark; Neuronetics, USA) and dTMS systems from Brainsway (Israel), with training done via manufacturers and through centralized training at VA Palo Alto.

In brief, Veterans were referred for TMS by their primary mental health provider and assessed for eligibility based on standard clinical

criteria, including MDD diagnosis. PTSD symptom monitoring is conducted routinely as part of clinical care. Concurrent psychiatric treatments, including pharmacotherapy and psychotherapy, were permitted if stable for approximately six weeks prior to TMS and maintained throughout treatment unless clinically indicated. All demographic variables are self-reported.

For the present analysis, to be included in the PTSD group, patients were required to have clinically relevant PTSD symptoms at pretreatment baseline (operationally defined as a score of at least 33 on the PCL-5 [28], and have at least one rating scale assessment during the course of TMS treatment (i.e., the equivalent of a modified intent-to-treat sample).

2.2. TMS protocols

The three FDA-cleared TMS protocols for depression were examined (Table 1). In brief, the 10 Hz protocol involved high-frequency stimulation targeting the left DLPFC. iTBS also targeted the left DLPFC but with a patterned stimulation sequence (triplets of 50 Hz, 5 per second). Deep TMS (dTMS) was administered using an H1-coil designed to stimulate medial prefrontal regions at a greater depth and breadth than standard figure-of-8 coils, with pulse numbers following manufacturer guidelines. For simplicity, these are each referred to as “10 Hz”, “iTBS”, and “dTMS” hereafter. The approach used for each patient was determined by the treating clinician based on clinical judgment and device availability. TMS was typically delivered at 120 % of the patient’s resting motor threshold, with the left DLPFC as the target site. For figure-of-8 coils, the DLPFC was localized using the modified Beam/F3 method [32]; the rigor of this technique in the VA system has been previously described [33]; dTMS targeting used the manufacturer’s helmet position. Most patients received 20–30 treatment sessions over a six-week course, delivered five business days per week, often followed by a taper of 3–6 additional sessions. Parameters were extracted from the VA REDcap, and protocol assignments were verified for each participant prior to inclusion in the analysis. Participants with multiple TMS protocols over time were not included.

2.3. Outcome measures

Rating scales were measured at pretreatment (including the Life Events Checklist component of the PCL-5) and then weekly as part of standard clinical care. The primary outcome measure for this analysis were: 1) achieving a PCL-5 score of less than 33 (operational definition of “remission”, which corresponds to no longer likely meeting PTSD criteria [27]), and 2) a clinically meaningful reduction in PCL-5 score of at least 10 points (operational definition of “response”; [27]), with the standard 10 Hz Protocol (described below) as the main comparator. We examined raw point and percentage change in PCL-5 scores as continuous measures. Depression scores were explored as this is the current FDA-cleared use for these protocols; response was defined as a 50 % or greater reduction on the 9-item Patient Health Questionnaire (PHQ-9; [34]), and remission as a PHQ-9 score less than 5. Final outcomes were taken from the last PCL-5 or PHQ-9 scores within the six-week treatment period, gathered within a week after treatment completion.

Table 1
TMS parameters.

TMS Protocol	Frequency	Percent of Resting MT	Intertrain Interval	Pulses per Session
10 Hz	10 Hz	120 %	11–26s	3000
iTBS	50 Hz triplets at 5Hz	120 %	8s	600
dTMS	18 Hz	120 %	20s	1980

Abbreviations: TMS, transcranial magnetic stimulation, MT, motor threshold, iTBS, intermittent theta burst stimulation; dTMS, deep TMS.

2.4. Propensity score matching

To address baseline differences between treatment groups, we used propensity score matching based on a comprehensive set of covariates. Propensity score matching is a statistical approach to create matched samples in retrospective studies to infer causal effects [35]. These included demographic characteristics (age, sex, race, marital status, education level, and employment status) and pretreatment symptom severity measured by total scores on PCL-5 and PHQ-9. Patients with missing values on any covariates were excluded from the matched analyses.

We performed 2:1 nearest-neighbor matching without replacement using the MatchIt package in R. Separate matching procedures were conducted for comparisons between 10 Hz and iTBS and between 10 Hz and dTMS; smaller groups were used as the reference for matching. Propensity scores were estimated via logistic regression including all selected covariates. Covariate balance was evaluated using standardized mean differences and visualized using Love plots and histograms of propensity scores generated with the cobalt package. All subsequent comparisons were conducted using the matched samples. The iTBS and dTMS groups were not directly compared due to insufficient sample size for propensity matching.

2.5. Statistical testing

To evaluate whether the different TMS protocols yielded comparable clinical outcomes, we conducted both noninferiority and equivalence testing using the two one-sided test (TOST) procedure implemented in the TOSTER package in R. For continuous outcomes, the equivalence and noninferiority margins were prespecified at $d = 0.25$, corresponding to approximately 4.6 points on the PCL-5. This threshold follows the rationale and approaches of previous studies assessing TMS protocols equivalence for depression [36,37] and represents a conservative difference that is roughly one half of the 10-point reduction used for meaningful change on this scale. For categorical outcomes, the equivalence and noninferiority margin was set at an absolute risk difference of 10 %, also consistent with previous noninferiority studies of TMS for depression [37]. For each comparison, we report the 90 % confidence interval for the mean difference, the TOST p-values for equivalence and noninferiority, and the p-value from a conventional two-sided null hypothesis significance test (NHT) set at $p < 0.05$. Note that equivalence testing requires both confidence interval bounds fall within the pre-specified margins, whereas noninferiority requires only the lower bound exceeds the margin.

3. Results

Demographics and pretreatment symptom burden for the sample are described in Table 2. Self-reported comorbidities were expected of this patient population and reported in Supplemental Table 1. Of the available 756 patients, $n = 658$ received FDA-cleared TMS protocols and were used in the analysis. The majority ($n = 526$) received standard TMS, and smaller groups received iTBS and dTMS ($n = 71$, and 61 , respectively). Average PTSD intensity was in the moderate to severe range across groups. Compared to the two other groups, the dTMS group was significantly younger (mean $\pm$ standard deviation [SD] of 45.61 ± 10.74 years), had a higher percentage of women (41 %), and were more employed (27.9 %). This group also had statistically significant but not clinically meaningful higher pretreatment PCL-5 scores (57.80 ± 11.6 in the dTMS group, compared to 53.54 ± 11.87 and 56.21 ± 11.01 for the 10 Hz and iTBS groups, respectively). Propensity matching yielded balanced comparison groups (Supplemental Fig. 1A and B), with corresponding sample sizes of 140 and 70 patients in the 10 Hz vs. the iTBS comparisons, and 118 and 59 in the 10 Hz vs. dTMS groups. Regarding treatment duration, average length of 10Hz treatment was 5.7 weeks corresponding to 28.5 sessions; for iTBS, it was 4.7 weeks corresponding

Table 2
Cohort demographics.

	10 Hz rTMS		iTBS		dTMS		p value
n	526		71		61		
Age (mean (SD))	49.94	(12.97)	48.44	(14.34)	45.61	(10.74)	0.039
Sex = Females (%)	116	(22.30)	18	(25.70)	25	(41.00)	0.006
marital (%)							0.55
Never Married	110	(21.20)	23	(32.90)	11	(18.00)	
Separated	19	(3.70)	2	(2.90)	3	(4.90)	
Divorced	152	(29.30)	18	(25.70)	20	(32.80)	
Married	229	(44.20)	26	(37.10)	27	(44.30)	
Widow(er)	8	(1.50)	1	(1.40)	0	0.00	
Race (%)							0.112
White	377	(73.80)	47	(67.10)	38	(63.30)	
Black	68	(13.30)	10	(14.30)	16	(26.70)	
Asian	10	(2.00)	3	(4.30)	0	0.00	
Asian Indian	2	(0.40)	0	0.00	0	0.00	
Native American/Alaskan	4	(0.80)	3	(4.30)	0	0.00	
Pacific Islander	5	(1.00)	1	(1.40)	0	0.00	
Middle Eastern	1	(0.20)	0	0.00	0	0.00	
Other	31	(6.10)	2	(2.90)	4	(6.70)	
Multiracial	13	(2.50)	4	(5.70)	2	(3.30)	
Education (%)							0.173
Elementary School	3	(0.60)	0	0.00	0	0.00	
High School/GED	55	(10.70)	9	(12.90)	1	(1.70)	
Some college	149	(29.00)	16	(22.90)	22	(36.70)	
Associate/Vocational	99	(19.30)	13	(18.60)	13	(21.70)	
Bachelor's degree	129	(25.10)	15	(21.40)	12	(20.00)	
Master's degree	71	(13.80)	13	(18.60)	9	(15.00)	
Doctorate degree	8	(1.60)	4	(5.70)	3	(5.00)	
Employment (%)							0.087
Unemployed, Not looking	136	(26.60)	19	(27.10)	17	(27.90)	
Unemployed/Looking for work	72	(14.10)	17	(24.30)	12	(19.70)	
Employed (Part-time)	39	(7.60)	4	(5.70)	3	(4.90)	
Employed (Full-time)	94	(18.40)	15	(21.40)	17	(27.90)	
Retired	171	(33.40)	15	(21.40)	12	(19.70)	
Baseline PHQ9 (mean(SD))	19.91	(4.32)	20.41	(4.51)	20.39	(3.96)	0.507
Baseline PCL-5 (mean(SD))	53.54	(11.87)	56.21	(11.01)	57.28	(11.60)	0.019

Demographics of the patient cohort. Those significantly, or near-significantly differing, across groups are highlighted in bold.

Abbreviations: SD, Standard deviation; PHQ9, 9-item Patient Health Questionnaire; PCL-5, PTSD Checklist for DSM-5.

to 23.5 treatments; and for dTMS it was 6.4, corresponding to 32 sessions. There were significant correlations between weeks of treatment and total point improvements: PCL-5: $r = 0.17$, $p < 0.001$, PHQ-9: $r = 0.21$, $p < 0.001$. Thus, length of treatment accounted for 2.8 % of the variance in PTSD improvement and 4.4 % of the variance of depression improvement with longer treatment associated with larger symptom reduction.

3.1. PTSD results

Reduction in PTSD symptoms over time in the whole sample are depicted in Fig. 1. All three TMS protocols resulted in similar and clinically significant reductions in PCL-5 scores. Unmatched analyses found comparable results and are included in the supplement.

3.2. Matched 10 Hz vs. iTBS

In the matched analysis of 10 Hz vs. iTBS, PTSD remission rates were 43.6 % and 48.6 %, respectively. Rates of PTSD response (score reduction >10 points) occurred in 58.6 % and 65.7 %, respectively, and mean reduction in PCL-5 scores were 17.8 ± 19 and 20.8 ± 19 , respectively. Both groups had more than 30 % reduction in PTSD symptoms. While iTBS outcomes were nominally superior to 10 Hz, there were no significant differences between these protocols on any PTSD outcome measures (NHT, all $p > 0.10$). Noninferiority of iTBS vs. 10 Hz was supported across all outcome measures (all $p < 0.05$). Equivalence was not supported between the two protocols (Table 3).

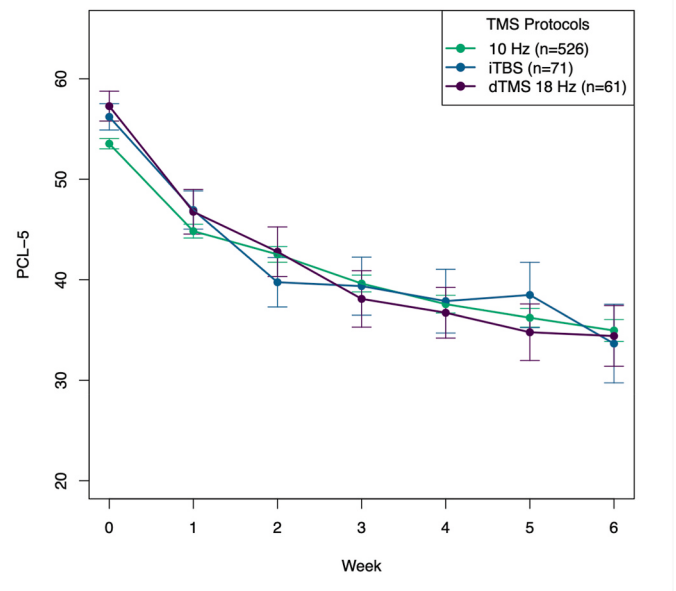


Fig. 1. PTSD Symptoms over time Trajectory of PTSD symptom change over time with TMS treatment. Error bars represent standard error. No significant differences (all $ps > 1$) were observed comparing protocols. Abbreviations: PCL-5, PTSD checklist for DSM5; TMS, transcranial magnetic stimulation; iTBS, intermittent theta burst stimulation; dTMS, deep TMS.

Table 3

Clinical outcomes.

10 Hz vs iTBS Matched							
	10 Hz	iTBS	Difference	90 % CI	NHT p	TOST p	NI p
n	140	70					
PCL-5 < 33 (%)	43.6	48.6	5.0	[-7.0, 17.0]	0.49	0.25	0.020
10 pt PCL-5 Improvement (%)	58.6	65.7	7.1	[-4.4, 18.7]	0.31	0.34	0.0074
PCL-5 Improvement (mean (SD))	17.8(19)	20.8(19)	3.0	[-1.7, 7.65]	0.29	0.26	0.0032
PCL-5 Percent Improvement (mean(SD))	32.0(34)	37.1(37)	5.1	[-3.7, 13.8]	0.34	0.23	0.0045
PHQ-9 Response (%)	37.9	45.7	7.8	[-4.0, 19.7]	0.28	0.38	0.0068
PHQ-9 Remission (%)	12.1	21.4	9.3	[0.02, 18.5]	0.10	0.45	0.0003
10 Hz vs dTMS Matched							
	10 Hz	dTMS	Difference	90 % CI	NHT p	TOST p	NI p
n	118	59					
PCL-5 < 33 (%)	43.2	49.2	5.9	[-7.1, 19.0]	0.46	0.30	0.022
10 pt PCL-5 Improvement (%)	67.8	78	10.2	[-1.2,21.5]	0.14	0.51	0.0017
PCL-5 Improvement (mean (SD))	19.3(18)	22.1(16)	2.8	[-1.6, 7.3]	0.29	0.30	0.0043
PCL-5 Percent Improvement (mean(SD))	35(32)	41(29)	5.9	[-2.1, 13.9]	0.22	0.36	0.0028
PHQ-9 Response (%)	34.7	44.1	9.3	[-6.0, 24.6]	0.23	0.47	0.0067
PHQ-9 Remission (%)	12.7	15.2	2.5	[-6.7, 11.7]	0.65	0.091	0.013

Clinical outcomes comparing 10 Hz vs. iTBS, and 10 Hz vs. dTMS after propensity matching. Significant p values are highlighted in bold. Equivalence margins were set at $d = 0.25$ for continuous variables and 10 % for categorical variables. The two one-sided test (TOST) evaluates equivalence using both bounds of the 90 % confidence interval (CI), whereas noninferiority (NI) evaluates only the lower bound.

Abbreviations: TMS, transcranial magnetic stimulation; iTBS, intermittent theta burst stimulation; dTMS, deep TMS; NHT, null hypothesis testing; TOST, two one-sided test; NI, noninferiority.

3.3. Matched 10 Hz vs. dTMS

PTSD remission occurred in 43.2 % vs. 49.2 % in the matched 10 Hz and dTMS groups, respectively, and response rates were 67.8 % and 78 %, respectively. Mean reduction was 19 ± 18 and 22.1 ± 16 points on the PCL-5, and percent improvement was 35 ± 32 and 41 ± 29 . While the dTMS PTSD outcomes were nominally superior to 10 Hz, we were unable to detect any significant differences in any PTSD outcomes between these protocols (all NHT, all $p > 0.10$). Noninferiority of dTMS vs. 10 Hz was supported across all PTSD outcome measures ($p < 0.05$). Equivalence was again not supported (all TOST p values > 0.05) (Table 3).

3.4. Depression outcomes

Reductions in PHQ-9 scores over time for the whole sample are depicted in Fig. 2, reflecting similar and clinically relevant reduction in depressive symptoms in this cohort with comorbid PTSD and MDD. Response and remission rates on the PHQ-9 were thematically similar to those found in PTSD. In propensity matched comparisons, both iTBS and dTMS yielded nominally superior depression outcomes in most tests compared to 10 Hz, however, there were no significant differences (NHT, all $p > 0.10$). iTBS was noninferior to 10 Hz on PHQ-9 response and remission (all p values < 0.05). dTMS was noninferior to 10 Hz for depression response and remission ($p < 0.02$). However, equivalence was not supported (TOST, $p > 0.05$) (Table 3).

4. Discussion

This is the first large-scale comparison of standard, FDA-cleared TMS protocols that demonstrates meaningful PTSD symptom reduction. Using propensity matched analyses of a large observational cohort across many VA sites, we were unable to detect any significant differences in PTSD outcomes between protocols. Both iTBS and dTMS demonstrated noninferior outcomes when compared with standard 10 Hz TMS for PTSD. TMS was associated with clinically significant improvement across a wide variety of metrics, including operationally defined PTSD outcomes of response and remission, and symptom reduction. In their totality, these outcomes broadly support the effectiveness of TMS for PTSD.

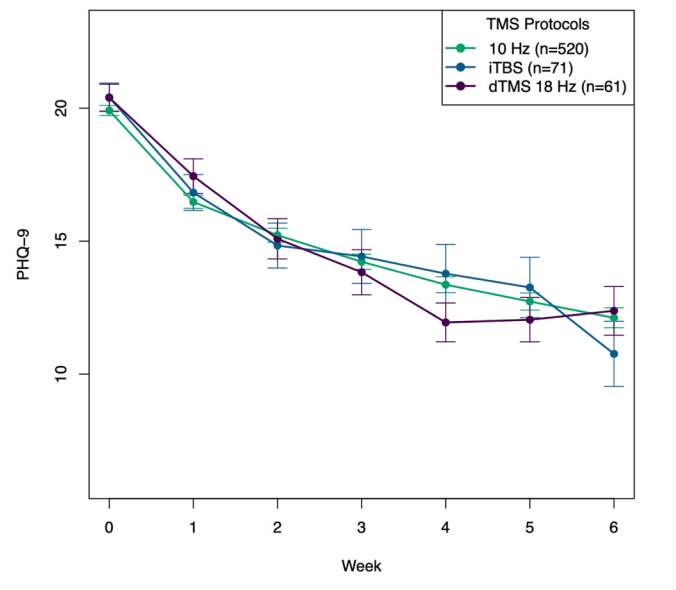


Fig. 2. MDD outcomes over time Trajectory of MDD symptom change over time with TMS treatment. Error bars represent standard error. No significant differences (all $ps > 1$) were observed comparing protocols.

Abbreviations: PHQ-9, 9-item Patient Health Questionnaire; TMS, transcranial magnetic stimulation; iTBS, intermittent theta burst stimulation; dTMS, deep TMS.

To our knowledge, this is the first demonstration of the non-inferiority of newer TMS protocols compared to 10 Hz for PTSD. Consistent with our hypothesis, comparable outcomes were observed when comparing stimulation parameters (e.g., 10 Hz vs. iTBS) and coil types (e.g., left sided figure-of-8 vs. H1 dTMS). This finding is noteworthy given that these systems target and stimulate different brain regions, as suggested by their respective electrical field models [38]; speculatively this phenomenon may be due to both systems ability to stimulate the putative PTSD “circuit” identified through lesion-based analysis [39]. These findings can thus be considered a minimum by

which to evaluate future precision TMS protocols in PTSD. While these findings indicate effectiveness of TMS for PTSD, it is important to recognize they provide sufficient room for improvement through novel methods such as imaging-guidance (e.g., [40]), accelerated TMS (e.g., [41]), use with psychotherapy [23], plasticity-enhancing pharmacological augmentation (e.g., [42]) or other future methods. That stated, it will be important that future approaches are able to demonstrate superiority to the magnitude of PTSD symptom reduction observed here.

This study is also the first to demonstrate noninferiority for categorical outcomes for MDD, with the caveat that these were patients with comorbid PTSD. Noninferiority of neuronavigated 10 Hz and iTBS in MDD has been demonstrated previously [37]; however, this is the first demonstration of noninferiority between 10 Hz and dTMS without neuronavigation. These outcomes are comparable to those from a small randomized controlled trial that did not find a separation in remission rates when comparing dTMS, 10 Hz TMS, with both superior to sham [43]. While that study found nominal differences in response rates and other metrics, it is important to note the compared groups used 20 sessions, which is fewer than the standard 30 (plus six taper) used in US settings as described in this report.

Stimulation with dTMS as a standalone treatment also demonstrated effectiveness for PTSD symptom reduction in this cohort. This determination is notable in the context of a randomized controlled trial that found active dTMS paired with brief symptom provocation was significantly inferior to sham dTMS with the same pairing, and both arms demonstrated significant improvements from baseline [25]. These findings indicate that dTMS without provocation for PTSD is an important area of use for this technology.

When considering MDD outcomes, these results (and others) continue to stand in contrast to the only RCT that exclusively examined TMS in Veterans with MDD [44]. That study used 10 Hz TMS using a 6 cm rule to target the DLPFC. That study found a very small reduction in PTSD outcomes using the PTSD Checklist, military version (PCL-M). While this rating scale differs, the large reduction in PTSD symptoms remains consistent in more recent examinations; while direct comparisons are challenging, a key difference is the more recent adoption of individualized scalp-based methods which are precise across a broad range of head sizes (e.g., 32–33).

This work has several important limitations, most notably those inherent to cohort studies of US military veterans, where TMS is received with concurrent care; only the three FDA-cleared once daily depression protocols were evaluated. This was not a randomized trial, yet gold-standard propensity methods were employed to ensure rigor. We also cannot disentangle nonspecific or sham effects from the observed outcomes, particularly as the sham effect in TMS is growing over time [45]. The iTBS and dTMS groups were compared to the reference 10 Hz group due to power considerations, and other protocols (e.g., right-sided stimulation, dTMS systems delivering 10Hz or iTBS) were not represented in sufficient numbers for examination. There may also have been site-specific differences related to device use (e.g., clinics with only a dTMS system), which we attempted to account for using propensity matching. We recognize that the TMS protocol is only one factor in a patient's clinical care and does not account for individual variability such as treatment resistance or Criterion A PTSD event (as assessed in the PCL-5 life event checklist) and cannot determine whether PTSD would be considered "primary" (i.e., occurring before MDD onset), and did not attempt to match on comorbidities due to power considerations. We did not examine safety, due to the very small (<5) number of known (e.g., seizure) events. We also were not always able to track protocol changes; as such, we used the first protocol as the index course. We did not examine PTSD symptom clusters or item-level responses, nor are able to examine the influence of different adjunctive pharmacological or psychotherapy treatment protocols. Whether these findings hold in patients with PTSD without MDD symptoms is unknown; because patients had comorbid PTSD and MDD by definition for inclusion we cannot disentangle effects in different domains. We also relied upon self-report

measures used in clinical care, and whether clinician-rated scales (e.g., Clinician Administered PTSD Scale) would yield different results is undetermined. We also used operational definitions of response and remission that, while consistent with prior use, mean that patients with very low PTSD pretreatment symptom burden might have a higher likelihood of achieving "remission"; however, the pretreatment PTSD symptom severity (over 50, with SD of 10) means this was uncommon at best. Finally, response biomarkers (e.g., neuroimaging, etc.) were not included and how these contribute to clinical decision making remain an important area of study (e.g., [46–47]).

In summary, this study demonstrates that the three US FDA-cleared TMS protocols for depression are effective for comorbid PTSD and that the newer TMS approaches, iTBS and dTMS, are noninferior to 10 Hz TMS. Symptom improvements were substantial and consistent across protocols and devices. This work continues to support the effectiveness of TMS for PTSD, and supports selecting TMS protocols based on patient-specific or logistical considerations rather than efficacy differences. Simply stated, clinicians should feel comfortable knowing that regardless of the device or protocol used in their clinic, TMS is likely to improve PTSD.

CRedit authorship contribution statement

Yosef A. Berlow: Writing – review & editing, Visualization, Investigation, Formal analysis. **Samantha L. Cilli:** Writing – review & editing, Visualization. **F. Andrew Kozel:** Writing – review & editing, Writing – original draft, Visualization, Investigation. **Amin Zandvakili:** Writing – review & editing, Formal analysis. **Noelle Marcotullio:** Writing – review & editing, Visualization. **Camila Cosmo:** Writing – review & editing. **Miriam A. Goldberg:** Writing – review & editing. **Bo Dehm Wicklund:** Writing – review & editing. **Michelle R. Madore:** Writing – review & editing, Writing – original draft, Visualization, Investigation. **Noah S. Philip:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Resources, Conceptualization.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: NSP reports a relationship with Motif Neurotech that includes: consulting or advisory. Noah S Philip MD reports a relationship with Grey Matter Neuroscience that includes: board membership. Noah S Philip MD reports a relationship with Pulvinar Neuro that includes: board membership. Noah S Philip MD reports a relationship with UptoDate Inc that includes: royalties. NSP is on the editorial board of Brain Stimulation.

FA Kozel reports a relationship with NIRx that includes: equipment loan. FA Kozel reports a relationship with Neuronetics that includes: equipment loan.

If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.brs.2025.11.007>.

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