

# Effects of Transcranial Magnetic Stimulation on Patients with Major Depressive Disorder: A Systematic Review and Meta-analysis

Min Huang; Quingfang Cao; Fengyi Zhang; Weiju Tang; Xiaojing Yu; Fan Xu; and Xiechuan Weng

To evaluate the symptoms improvement of major depressive disorder (MDD) by transcranial magnetic stimulation (TMS) the PubMed, Embase, Cochrane Library, China National Knowledge Infrastructure (CNKI), and SinoMed (a biomedical literature service system managed by the Institute of Medical Information [IMI] and Library of the Chinese Academy of Medical Sciences [CAMS]) library databases were searched. A total of 12 studies involving 1318 patients were included. It was found that the Hamilton Depression rating scale (HAMD) score decreased after TMS (-4.47, 95% CI: -7.10 to -1.84,  $P < 0.00001$ ). It showed a decreased grade of Montgomery-Asberg Depression Rating Scale (MADRS) after TMS treatment (-2.72, 95% CI: -4.75 to -0.70,  $P=0.0004$ ) compared to the control. The difference in the Involvement-Detachment scale (IDS) score was not statistically significant between the groups (-2.40, 95% CI: -7.42 to 2.62,  $P=0.05$ ). The Clinical Global Impression (CGI) score decreased after TMS treatment (-1.12, 95% CI: -2.43 to -0.20,  $P < 0.00001$ ) compared to the control group. The HAMD, MADRS, and CGI scores showed an improvement trend after TMS treatment, indicating TMS has a therapeutic effect on major depression.

**Keywords:** Major Depressive Disorder; Meta-analysis; Therapeutic evaluation; Transcranial Magnetic Stimulation

Major depression disorder (MDD) is a common illness that severely limits psychosocial functioning and reduces the quality of life. In 2008, the World Health Organization (WHO) ranked MDD as the third leading cause of the global burden of disease and projected it would rank first by 2030.<sup>1</sup> Detection, diagnosis, and management of MDD often pose challenges to clinicians due to its diverse manifestations, unpredictable course, prognosis, and variable treatment response.<sup>2</sup>

The first-line treatment of MDD is a single therapy using appropriate antidepressants, such as selective serotonin re-uptake inhibitor (SSRI), serotonin-norepinephrine re-uptake inhibitor (SNRI), or norepinephrine and specific

serotonin antidepressants (NaSSA). However, when depression is resistant to first-line treatment, two types of drugs can be used in combination to strengthen the antidepressant effect. If it is still resistant, electroconvulsive therapy (ECT) can be used.<sup>3</sup> However, some patients do not achieve sufficient remission with the existing first-line treatment, or they have difficulty tolerating the side effects of antidepressants and seizures caused by ECT.<sup>4</sup> Thus, identifying alternative treatments for MDD is a research hotspot worldwide.

Transcranial magnetic stimulation (TMS) has emerged in the last few decades as one such potential option for treatment-resistant depression.<sup>5</sup> Unlike ECT, TMS does not require anesthesia and generally does not cause seizures. If correct

**Corresponding Author:** Prof. Xiechuan Weng, Department of Neuroscience, Beijing Institute of Basic Medical Sciences, Beijing, 100850, China, Email: wengxc2000@163.com

**Funding:** This work was supported by the National Natural Science Foundation of China under Grant number 8216050478; and the Chengdu Science and Technology Bureau Focuses on Research and Development Support Plan under Grant number 2019-YF09-00097-SN.

**Disclosures:** The authors report there are no competing interests to declare. All data will be made available upon reasonable request.

Received: February 6, 2025  
Revised: July 6, 2025  
Accepted: August 12, 2025

doi: 10.3121/cmr.2025.2011

procedures and safety guidelines are followed, TMS can be conducted relatively painlessly to conscious patients and can be used for outpatients or inpatients.<sup>6</sup> The aim of TMS treatment is to stimulate brain regions involved in emotional regulation. TMS works by passing a high-intensity current through a copper coil, creating a magnetic field that can be used to generate an electric field when in contact with the surface of the brain. This electric field is sufficient to generate action potentials and alter network activation within the cortex.<sup>7</sup> It is unclear how TMS exerts an antidepressant effect. The possible mechanism is that TMS exerts an antidepressant effect by altering neurochemical levels, blood flow, and other brain activity.<sup>7</sup> The anti-depressant effects of TMS were studied using single pulses, paired pulses, repeated pulses, and low-frequency or high-frequency pulses of repeated pulses.

However, different studies have shown inconsistent results on the efficacy of TMS in MDD. Previous reviews have highlighted key limitations of TMS for MDD, namely, the heterogeneity in methodology and the variable quality of included studies.<sup>8-10</sup> These issues remain unresolved and have been emphasized in recent studies, which point out concerns about trial design, stimulation parameters, and adjunctive treatments. Given the persistence of these challenges and the evolving nature of TMS research (such as the emergence of accelerated TMS and combined therapeutic strategies), a new meta-analysis is warranted to synthesize the latest evidence and provide updated insights into the efficacy of TMS for MDD. Hence, this study systematically reviewed existing studies on TMS for the treatment of MDD, analyzed the study design, treatment options, efficacy, and tolerability, and performed a meta-analysis to determine the level of effectiveness of TMS in treating MDD.

## Methods

### Retrieval Strategy

Two reviewers (TWJ and YXJ) independently searched the China Journal Full-Text Database, Wanfang Data Knowledge Service Platform, Wipro Chinese Journal Full-text Database, PubMed, Embase, and Cochrane Library databases from 2000-01-01 to 2024-01-01. The search terms used were as follows: #1 TS= (“Major Depressive Disorder” OR “MDD” OR “Depressive”); #2 TS= (“Transcranial Magnetic Stimulation” OR “TMS”); #3 #1 AND #2 AND.

### Inclusion and Exclusion Criteria

Inclusion criteria were as follows: (1) randomized controlled trials (RCTs) and self-control trials for TMS for depression; (2) patients with primary depression who met the diagnostic criteria for depression, without

restrictions in age, gender, course and severity; (3) the experimental group (TMS) and the control group (sham stimulation); and (4) primary outcome measures included the Hamilton depression rating scale (HAMD), secondary outcome measures included the Montgomery-Asberg Depression Rating Scale (MADRS), the Involvement-Detachment Scale (IDS), the Montgomery-Asberg Depression Rating Scale (MADRS), or Clinical Global Impression (CGI). Exclusion criteria were as follows: (1) secondary depression patients, such as postpartum depression and other related conditions; (2) duplicate publications; (3) literature where the original study data were lost or unsuitable for meta-analysis; (4) the treatment group was given treatment other than TMS, or the design of the group was not reasonable.

### Literature Screening and Data Extraction

Two researchers independently read the titles and abstracts of all the studies. Thereafter, duplicate and unqualified literature (such as reviews, animal experiments, etc.) were excluded. The full-text of the remaining studies were read, and studies with unclear outcomes, unclear diagnoses, and unreasonable experimental design were excluded. Finally, the two researchers cross-checked the included studies with each other, and if it was difficult to determine whether to include any study, a third researcher was consulted to decide. If the data provided by the study were not comprehensive, it was not included. The outcome measures included the HAMD, the MADRS, the IDS, or the CGI scale. Remission was defined as

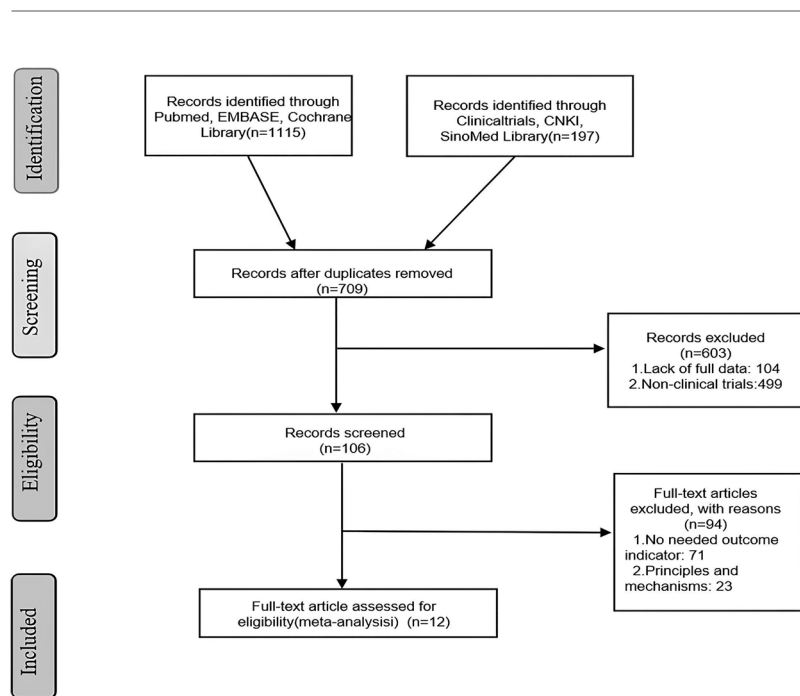


Figure 1. Flowchart of study selection process.

**Table 1.** Characteristics of the studies included in the present meta-analysis

Year	Group	Sample	Subjects (N)	Age (mean (SD))	Gender M/F	Intervention	Outcomes
2020	I	153	153	I=42.8(17.57)	I=42/111	I=Post-TMS	HAMD
	C	153		C=42.8(17.57)	C=42/111	C=Pre-TMS	
2018	I	10	18	I=30.13 (5.78)	-	I=active TMS	HAMD, CGI
	C	8		C=42.8(17.57)	-	C=sham TMS	
2020	I	15	31	I=47.3 (11.5)	I=6/9	I=active TMS	HAMD
	C	16		C=49.7 (11.0)	C=3/13	C=sham TMS	
2007	I	155	301	I=47.9±11.0	I=14%	I=active TMS	MADRS
	C	146		C=48.7±10.6	C=26%	C=sham TMS	
2018	I	81	164	I=55.6 (12.2)	I=67%	I=active TMS	MADRS, HAMD
	C	83		C=54.8 (12.6)	C=65%	C=sham TMS	
2009	I	15	30	52.3 ± 11.9	18/12	I=Real-TMS	MADRS, HAMD
	C	15				C=Sham-TMS	
2015	I	59	120	46.2(11.9)	66/54	I=active TMS	HAMD, MADRS, IDS
	C	61				C=sham TMS	
2010	I	92	190	47.1(11.5)	82/108	I=active TMS	HAMD, MADRS, IDS, CGI
	C	98				C=sham TMS	
2013	I	12	22	53.05(8.75)	14/8	I=active TMS	HAMD, MADRS
	C	10				C=sham TMS	
2012	I	28	56	32.06(7.35)	17/39	I=active TMS	HAMD, MADRS
	C	28				C=sham TMS	
2020	I	50	50	43.0±15.4	35/15	I=TMS	HAMD, CGI
2011	I	15	15	I=47 (8.51)	7/8	I=TMS	HAMD, IDS, CGI
	C	15		C=47 (8.51)	7/8	C=sham	

Abbreviations: I, Experimental group; C, Control group; TMS, transcranial magnetic stimulation; HAMD, Hamilton Depression rating scale; CGI, Clinical Global Impression; MADRS, Montgomery-Asberg Depression Rating Scale; IDS, Involvement-Detachment Scale

the HAMD score  $\leq 7$ , the MADRS score  $\leq 10$ ,<sup>11</sup> the IDS score  $< 14$ ,<sup>12</sup> and the CGI scale  $< 1$ .<sup>13</sup>

#### Quality Assessment

The methods in Cochrane Handbook 5.1.0 were used for quality evaluation and risk assessment, mainly from the aspects of random number generation, scheme allocation concealment, blinding implementation, completeness of outcome data, selective reporting of outcomes and other biases, and low-risk bias, unclear and high-risk bias evaluations.

#### Statistical Methods

Meta-analysis was performed using Review Manager 5.4 software from the International Collaborative Network for Evidence-Based Medicine. The mean and standard deviation (SD) was used as the effect quantities, and the estimated values of each point and the 95% confidence interval (CI) were given. Heterogeneity analysis was performed using the  $\chi^2$  test. If the  $P \geq 0.10$  and  $I^2 \leq 50\%$ , the heterogeneity was considered to be small, and the fixed-effects model was used for meta-analysis; If the  $P < 0.10$  and the  $I^2 > 50\%$ , heterogeneity was considered as large and analyzed using a random-effects

model. Finally, the forest map was used for descriptive analysis, and the funnel diagram was used to determine publication bias.

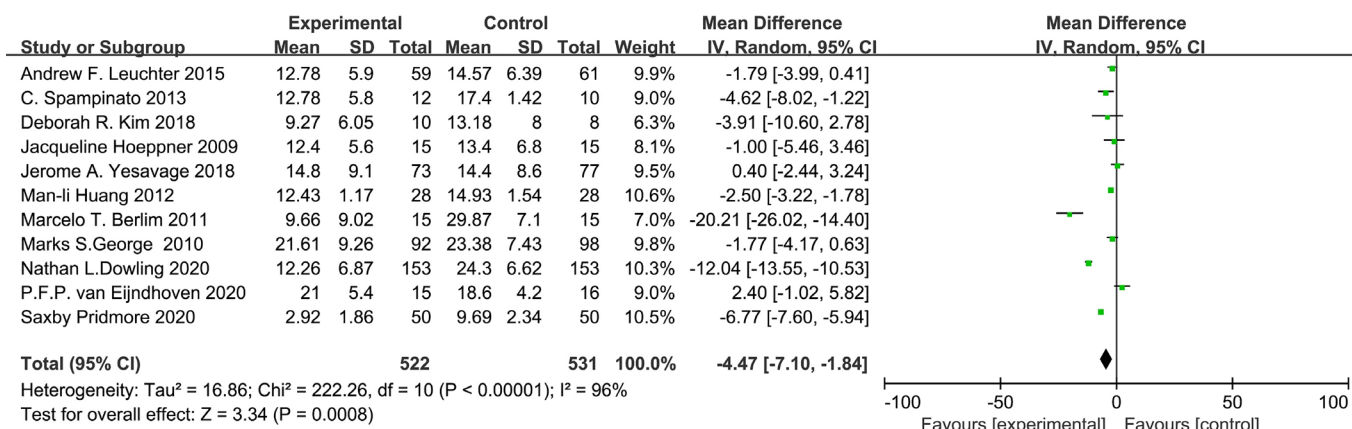
#### Results

##### Study Selection and Characteristics

A total of 12 studies were included from 1,312 retrieved studies. The detailed study selection process is shown in Figure 1. We summarized the demographic data of 685 patients in the experimental group and 633 patients in the control group. Descriptive statistics were used to identify the demographic information, including intervention, age, gender, subject, and outcomes (Table 1). Additionally, we used the Cochrane handbook, published research, and registration trials to assess the risk-of-bias (Supplementary Figure S1 and Figure S2, available online).

##### HAMD

The evaluation indicators of 11 studies<sup>13-23</sup> included HAMD. The heterogeneity test results showed  $P < 0.1$ ,  $I^2 > 50\%$ , so the random-effects model was used. Comparison of the HAMD score of the two groups showed the score decreased after TMS (-4.47, 95% CI: -7.10 to -1.84,  $P < 0.00001$ ) (Figure 2).



**Figure 2.** Forest plot showing mean difference and 95% CI of HAMD score between the two groups. MADRS

Seven studies<sup>14,16-19,22,24</sup> were included. We found that  $P < 0.1$ ,  $I^2 > 50\%$ , so we evaluated MADRS using a random-effects model. Importantly, the MADRS grade decreased after the TMS treatment (-2.72, 95% CI: -4.75 to -0.70,  $P=0.0004$ ) compared to the control (Figure 3).

#### IDS

Three studies<sup>16,19,23</sup> were included. Based on  $P < 0.1$ ,  $I^2 > 50\%$ , we chose to use a random-effects model. There was no statistical difference in the IDS score between the two groups (-2.40, 95% CI: -7.42 to 2.62,  $P=0.05$ ) (Figure 4).

#### CGI

CGI effect changed after treatment. Three studies<sup>13,15,19,23</sup> were included. The heterogeneity test showed that  $P < 0.1$ ,  $I^2 > 50\%$ , so the random-effects model was selected. Compared with the control group, the CGI grade decreased after TMS treatment (-1.12, 95% CI: -2.43 to 0.20,  $P<0.00001$ ) (Figure 5).

#### Publication Bias

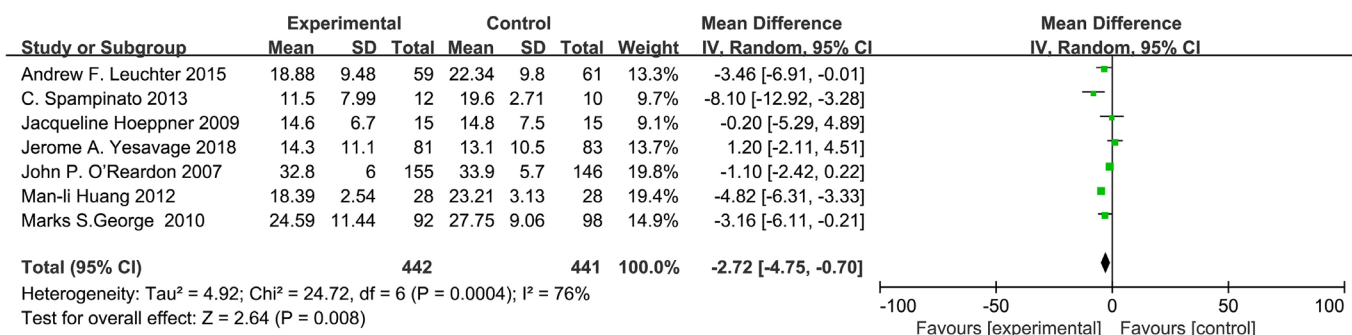
We used Review Manager 5.4 to make funnel plots for the

studies involved in the four outcome indicators (Supplementary Figures S3, available online).

#### Discussion

The core characteristics of MDD are “low mood” and “loss of interest or pleasure in almost all activities,” which may be accompanied by symptoms such as anxiety, sleep disturbances, apathy, and cognitive dysfunction.<sup>25</sup> Tricyclic antidepressants and ECT have been recommended in the past to treat patients with depression.<sup>26</sup> In addition, deep brain stimulation, although in the early stages of research, could become a therapy for treatment-resistant depression, and TMS has been approved by the FDA for the treatment of major depression.<sup>27</sup>

In 2008, Kennedy showed that HAMD-17 or MADRS was more sensitive to drug placebo separation and established optimal doses than the full scale.<sup>25</sup> Therefore, in this meta-analysis, we used HAMD as the first outcome variable, and MADRS, IDS, and CGI as secondary variables to analyze the results. The purpose was to explore the improvement effect of TMS on the scale score of MDD, to infer the efficacy of TMS on MDD.



**Figure 3.** Forest plot showing mean difference and 95% CI of MADRS score between the two groups.

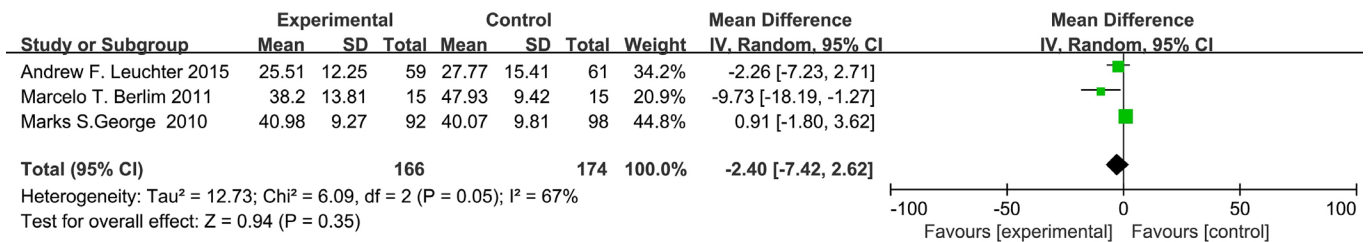


Figure 4. Forest plot showing mean difference and 95% CI of IDS score between the two groups.

Outcome indicators in this meta-analysis were the objective outcome variable scale, which made our results more objective and avoided human emotional bias from affecting the outcome. We reviewed numerous studies on TMS for the treatment of MDD and included clinical research data from the United States, Australia, China, and other countries to analyze the efficacy of TMS objectively and comprehensively. The results showed HAMD, MADRS, and CGI scores had an improvement trend after TMS treatment, indicating TMS had a therapeutic effect on major depression. There were 17 experts representing the National Network of Centers for Depression (NNDC) and the American Psychiatric Association Research Council (APA CoR) who said multiple randomized controlled trials and published literature supported the safety and efficacy of TMS antidepressant treatment,<sup>28</sup> which was consistent with our results.

Giron et al.<sup>8</sup> reported synergistic effects when TMS was combined with cognitive behavioral therapy in treatment-resistant depression (standard mean difference [SMD] = -0.62),<sup>8</sup> whereas our focus on TMS monotherapy demonstrated significant standalone efficacy (HAMD: mean difference [MD] = -4.47). Further, Vida et al.<sup>10</sup> focused on TMS as an adjunctive therapy for MDD after two antidepressant treatment failures, with their meta-analysis of randomized sham-controlled trials showing moderate efficacy in this specific population (MADRS reduction: SMD = -0.58). In contrast, our study included a broader MDD spectrum (not limited to

treatment-failure cases) and still observed robust symptom improvement (MADRS: MD = -2.72). Sonmez et al.<sup>9</sup> specialized in accelerated TMS protocols (e.g., high-frequency, short-course stimulation), reporting faster symptom relief but with higher heterogeneity due to varied stimulation frequencies. While our study did not explicitly include accelerated TMS (focusing primarily on standard rTMS protocols), our findings of consistent efficacy across diverse standard TMS parameters (e.g., varying frequencies, durations) imply TMS benefits are not strictly dependent on “acceleration.” However, Sonmez’s work highlights a critical direction: optimizing protocols (e.g., through accelerated delivery) may enhance outcomes for time-sensitive cases—a gap future research should address to complement our study’s focus on standard rTMS efficacy.<sup>9</sup>

This meta-analysis had several limitations. First, the literature between some groups was small. More relevant literature should be included in the future. Second, there was substantial heterogeneity in studies included in this meta-analysis, which may be explained by the following reasons: (1) among the 12 clinical trials included, 3 articles did not mention the implementation of blinding method, and many articles did not report the concealment of allocation scheme; (2) although the included studies used TMS for treatment, we did not restrict the specific stimulation intensity, stimulation site, and other parameters of TMS; (3) the age range of the subjects was large, and there was difference of years included. Last, while the study adds value through the synthesis of data across

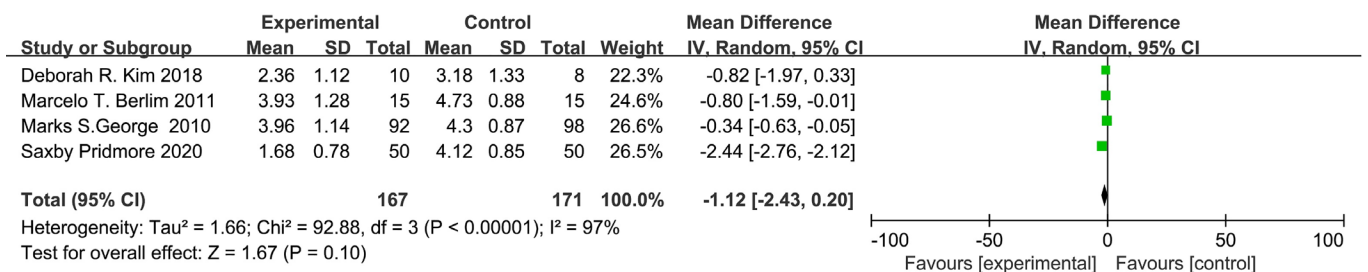


Figure 5. Forest plot showing mean difference and 95% CI of CGI scores between the two groups.

unexplained, atypical, or complex symptoms. Greater awareness of AM can reduce diagnostic delays, limit unnecessary investigations, and improve patient outcomes.

## References

1. Shah DR, Dilwali S, Friedman DI. Migraine Aura Without Headache [corrected]. *Curr Pain Headache Rep.* 2018;22(11):77. doi:10.1007/s11916-018-0725-1
2. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. *Cephalalgia.* 2018;38(1):1-211. doi:10.1177/0333102417738202
3. Thomsen AV, Ashina H, Al-Khazali HM, et al. Clinical features of migraine with aura: a REFORM study. *J Headache Pain.* 2024;25(1):22. doi:10.1186/s10194-024-01718-1
4. Viana M, Khaliq F, Zecca C, et al. Poor patient awareness and frequent misdiagnosis of migraine: findings from a large transcontinental cohort. *Eur J Neurol.* 2020;27(3):536-541. doi:10.1111/ene.14098
5. Foroozan R, Cutrer FM. Transient Neurologic Dysfunction in Migraine. *Neurol Clin.* 2019;37(4):673-694. doi:10.1016/j.ncl.2019.06.002
6. Puledra F, Sacco S, Diener HC, et al. International Headache Society global practice recommendations for the acute pharmacological treatment of migraine. *Cephalalgia.* 2024;44(8):3331024241252666. doi:10.1177/03331024241252666
7. Kurth T, Rist PM, Ridker PM, Kotler G, Bubes V, Buring JE. Association of Migraine With Aura and Other Risk Factors With Incident Cardiovascular Disease in Women. *JAMA.* 2020;323(22):2281-2289. doi:10.1001/jama.2020.7172
8. Adelborg K, Szépligeti SK, Holland-Bill L, et al. Migraine and risk of cardiovascular diseases: Danish population based matched cohort study. *BMJ.* 2018;360:k96. doi:10.1136/bmj.k96
9. Mahmoud AN, Mentias A, Elgendy AY, et al. Migraine and the risk of cardiovascular and cerebrovascular events: a meta-analysis of 16 cohort studies including 1 152 407 subjects. *BMJ Open.* 2018;8(3):e020498. doi:10.1136/bmjopen-2017-020498
10. Ng CYH, Tan BYQ, Teo YN, et al. Myocardial infarction, stroke and cardiovascular mortality among migraine patients: a systematic review and meta-analysis. *J Neurol.* 2022;269(5):2346-2358. doi:10.1007/s00415-021-10930-x
11. Frimpong-Manson K, Ortiz YT, McMahon LR, Wilkerson JL. Advances in understanding migraine pathophysiology: a bench to bedside review of research insights and therapeutics. *Front Mol Neurosci.* 2024;17:1355281. doi:10.3389/fnmol.2024.1355281
12. Scutelnic A, Bracher J, Kreis LA, et al. Symptoms and patterns of symptom propagation in incipient ischemic stroke and migraine aura. *Front Hum Neurosci.* 2023;16:1077737. doi:10.3389/fnhum.2022.1077737
13. Petrusic I, Zidverc-Trajkovic J, Podgorac A, Sternic N. Underestimated phenomena: higher cortical dysfunctions during migraine aura. *Cephalalgia.* 2013;33(10):861-867. doi:10.1177/0333102413476373
14. Doğan NÖ, Pekdemir M, Yılmaz S, et al. Intravenous metoclopramide in the treatment of acute migraines: A randomized, placebo-controlled trial. *Acta Neurol Scand.* 2019;139(4):334-339. doi:10.1111/ane.13063
15. Villalón CM, VanDenBrink AM. The Role of 5-Hydroxytryptamine in the Pathophysiology of Migraine and its Relevance to the Design of Novel Treatments. *Mini Rev Med Chem.* 2017;17(11):928-938. doi:10.2174/1389557516666160728121050
16. Huang Y, Cai X, Song X, et al. Steroids for preventing recurrence of acute severe migraine headaches: a meta-analysis. *Eur J Neurol.* 2013;20(8):1184-1190. doi:10.1111/ene.12155
17. Woldeamanuel YW, Rapoport AM, Cowan RP. The place of corticosteroids in migraine attack management: A 65-year systematic review with pooled analysis and critical appraisal. *Cephalalgia.* 2015;35(11):996-1024. doi:10.1177/0333102414566200
18. Chu CH, Liu CJ, Lin LY, Chen TJ, Wang SJ. Migraine is associated with an increased risk for benign paroxysmal positional vertigo: a nationwide population-based study. *J Headache Pain.* 2015;16:62. doi:10.1186/s10194-015-0547-z
19. Bhattacharyya N, Baugh RF, Orvidas L, et al. Clinical practice guideline: benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg.* 2008;139(5 Suppl 4):S47-S81. doi:10.1016/j.otohns.2008.08.022
20. Ferrari MD, Roon KI, Lipton RB, Goadsby PJ. Oral triptans (serotonin 5-HT<sub>1B/1D</sub> agonists) in acute migraine treatment: a meta-analysis of 53 trials. *Lancet.* 2001;358(9294):1668-1675. doi:10.1016/S0140-6736(01)06711-3
21. Webster KE, Dor A, Galbraith K, et al. Pharmacological interventions for acute attacks of vestibular migraine. *Cochrane Database Syst Rev.* 2023;4(4):CD015322. doi:10.1002/14651858.CD015322.pub2
22. De Ponti F, Tonini M. Irritable bowel syndrome: new agents targeting serotonin receptor subtypes. *Drugs.* 2001;61(3):317-332. doi:10.2165/00003495-200161030-00001
23. Bushnell C, McCullough LD, Awad IA, et al. Guidelines for the prevention of stroke in women: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2014;45(5):1545-1588. doi:10.1161/01.str.0000442009.06663.48

24. A Al-Khazali HM, Ashina H, Wiggers A, et al. Calcitonin gene-related peptide causes migraine aura. *J Headache Pain.* 2023;24(1):124. doi:10.1186/s10194-023-01656-4
25. Ashina M, Goadsby PJ, Dodick DW, et al. Assessment of Erenumab Safety and Efficacy in Patients With Migraine With and Without Aura: A Secondary Analysis of Randomized Clinical Trials. *JAMA Neurol.* 2022;79(2):159-168. doi:10.1001/jamaneurol.2021.4678
26. Shibata Y. Anti-Calcitonin Gene-Related Peptide Monoclonal Antibody Is Effective for Preventing Migraine Aura Without Headache. *Neurol Int.* 2024;16(6):1279-1284. doi:10.3390/neurolint16060097
27. Braca S, Miele A, Stornaiuolo A, Cretella G, De Simone R, Russo CV. Are anti-calcitonin gene-related peptide monoclonal antibodies effective in treating migraine aura? A pilot prospective observational cohort study. *Neurol Sci.* 2024;45(4):1655-1660. doi:10.1007/s10072-023-07241-6
28. Cresta E, Bellotti A, Rinaldi G, Corbelli I, Sarchielli P. Effect of anti-CGRP-targeted therapy on migraine aura: Results of an observational case series study. *CNS Neurosci Ther.* 2024;30(2):e14595. doi:10.1111/cns.14595

### Author Affiliations

Min Huang<sup>\*</sup>; Quingfang Cao<sup>†</sup>; Fengyi Zhang<sup>‡</sup>; Weiju Tang<sup>§</sup>; Xiaojing Yu<sup>¶</sup>; and Xiechuan Weng<sup>\*\*</sup>

<sup>\*</sup>Department of Physiology, School of Basic Medicine, Chengdu Medical College, Sichuan, 610500, China

<sup>†</sup>Department of Anesthesiology, West China Hospital, 610041, China

<sup>‡</sup>Department of Clinic Medicine, Chengdu Medical College, Sichuan, 610500, China

<sup>§</sup>Department of Geriatrics, the First People's Hospital of Longquanyi District, Chengdu, 610100, China

<sup>¶</sup>Department of Evidence-based Medicine and Social Medicine, School of Public Health, Chengdu Medical College, Sichuan, 610500, China.

<sup>\*\*</sup>Department of Neuroscience, Beijing Institute of Basic Medical Sciences, Beijing, 100850, China

*Author Contributions: HM, CQF and ZFY wrote the manuscript. TWJ and YXJ screened the studies and extracted the data from the studies. ZFY and CQF analyzed and interpreted the data. HM and WXC evaluated the quality of the included studies. XF and HM designed and proofread the manuscript. HM, CQF and ZFY contributed equally to the article.*