The Relationship between Cortical GABA Levels and Short-Interval Intracortical Inhibition: A Meta-analysis of Magnetic Resonance Spectroscopy and Transcranial Magnetic Stimulation Studies

Sharma P,[1] Grunberg K,[1] Sharma P,[2] Kunwar D[3]

[1]Department of Psychiatry
UC Davis School of Medicine,
Sacramento, California.

[2]Department of Biostatistics,
Yale School of Medicine,
New Haven, CT, USA.

[3]Department of Psychiatry
Kathmandu University School of Medicine Sciences,
Dhulikhel, Kavre, Nepal.

Corresponding Author
Pragyan Sharma
Department of Psychiatry
UC Davis School of Medicine,
Sacramento, California.
E-mail: ppsharma@ucdavis.edu

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ABSTRACT

Cortical gamma-aminobutyric acid (GABA) is the brain's principal inhibitory neurotransmitter and plays a key role in regulating motor cortical excitability. Short interval intracortical inhibition (SICI), measured via paired-pulse transcranial magnetic stimulation (TMS), is a widely used physiological index of GABAergic function. However, the strength and consistency of the association between GABA levels, quantified in vivo using magnetic resonance spectroscopy (MRS), and SICI remain unclear across studies.

We conducted a random-effects meta-analysis of studies reporting correlations between MRS-derived cortical GABA concentrations and TMS-measured SICI in healthy human participants. Effect sizes were transformed to Fisher's Z scores and pooled using the restricted maximum likelihood (REML) method. Heterogeneity was assessed using the I^2 statistic and Cochran's Q test. Potential publication bias was evaluated via visual funnel plot inspection and Egger's regression test. Results: A total of 23 effect sizes were included in the analysis. The pooled Fisher's Z was –0.14 (95% CI, –0.26 to –0.02; P = .03), corresponding to a small but statistically significant negative correlation ($r \approx -0.14$). This finding suggests that higher cortical GABA levels are modestly associated with stronger intracortical inhibition. Heterogeneity was low to moderate ($I^2 = 35.5\%$; Q = 34.73; P = .04). Funnel plot inspection revealed mild asymmetry, but Egger's test showed no significant small-study effects ($\beta_1 = -0.46$; P = .58), indicating minimal publication bias.

This meta-analysis provides quantitative evidence supporting an inverse association between cortical GABA concentrations and SICI, reinforcing GABA's role in modulating intracortical inhibition. While the effect size is modest, the findings appear robust and consistent. Future research should employ standardized acquisition and reporting protocols and explore the clinical relevance of this relationship in populations with disrupted GABAergic signaling.

KEY WORDS

Cortical GABA levels, Magnetic resonance spectroscopy, Restricted maximum likelihood, Short interval intracortical inhibition

INTRODUCTION

GABA (gamma-aminobutyric acid) is the brain's primary inhibitory neurotransmitter and plays a fundamental role in maintaining cortical excitability and regulating neural network activity. Alterations in GABAergic signaling have been implicated in a range of neuropsychiatric conditions, including schizophrenia, autism spectrum disorder, and mood disorders.[1-3] While several lines of evidence have linked GABA levels to functional inhibitory mechanisms, the relationship between neurochemical markers of GABA and physiological indices of cortical inhibition remains incompletely understood.

Two widely used non-invasive tools for assessing GABAergic function in humans are magnetic resonance spectroscopy (MRS) and transcranial magnetic stimulation (TMS). MRS allows for in vivo quantification of cortical GABA concentrations, while TMS-based paradigms such as short-interval intracortical inhibition (SICI) probe fast-acting inhibitory circuits within the motor cortex, thought to be mediated in part by GABA_A receptor activity.[4,5] Despite conceptual overlap, the correspondence between these two modalities has been variable across studies, with some reporting positive, negative or null associations between GABA levels and SICI strength.[6-8]

This variability may reflect methodological heterogeneity across studies, including differences in SICI protocols e.g., interstimulus intervals, voxel localization for MRS,

participant demographics, or GABA quantification approaches.[9,10] Moreover, MRS and TMS capture distinct aspects of GABAergic function, tonic vs. phasic inhibition which may further complicate interpretation.[11] To date, no comprehensive meta-analysis has quantitatively synthesized these findings to clarify the nature and magnitude of the GABA–SICI relationship.

The present meta-analysis aims to address this gap by systematically reviewing and statistically integrating existing studies that report associations between cortical GABA levels (via MRS) and SICI metrics (via TMS) in healthy populations. By pooling effect sizes across diverse methodologies and participant samples, we seek to determine whether a consistent relationship emerges, estimate its strength, and evaluate sources of heterogeneity and potential bias. Understanding this relationship is crucial for establishing cross-modal biomarkers of inhibitory tone and informing future studies of GABAergic dysfunction in clinical populations.

METHODS

We conducted a systematic search of PubMed, Embase, and APA PsycINFO from database inception through March 18, 2025, without geographic restrictions. The search was limited to articles published in English. Search terms were applied to the Title and Abstract fields and combined using Boolean operators as follows:

• GABA: ("GABA" OR "4-aminobutyric acid" OR "gamma-aminobutyric acid")

• MRS: ("Magnetic Resonance Spectroscopy" OR "MRS")

• TMS: ("Transcranial Magnetic Stimulation" OR "TMS")

• Cortical Inhibition: ("cortical inhibition" OR "intra-cortical inhibition" OR "intracortical inhibition" OR "cortical silent period")

Studies were eligible if they met all the following criteria:

• Peer-reviewed primary research articles.

• Reported measurement of cortical GABA concentrations using magnetic resonance spectroscopy (MRS) in any brain region (e.g., motor cortex, prefrontal cortex, somatosensory cortex).

• Assessed short interval intracortical inhibition (SICI) using transcranial magnetic stimulation (TMS) targeting the primary motor cortex (M1).

• Included a healthy control group or provided baseline data from healthy participants.

Exclusion criteria:

Studies were excluded if they met any of the following criteria:

• Non-English language publications.

• Studies that did not report both MRS-measured GABA and TMS-measured SICI outcomes.

• Review articles, case reports, conference abstracts, or other non-primary research formats.

• Studies in which GABA was measured exclusively in non-cortical regions (e.g., cerebellum, subcortical structures).

The study selection process was done according to PRISMA flow diagram.[12] Final selected studies are depicted in the table 1.

Table 1. Study Characteristics:

Study	N (Total)	Population	Age (Mean ± SD)	SICI Interval(s)	Notes
Cuypers et al. (2021)	30	15 healthy young adults 15 healthy older adults	23.4 ± 2.2 70.7 ± 4.1	3 ms	Age-stratified groups
Dyke et al. (2017)	27	Healthy adults	23.1 ± 2.4	1 ms, 3 ms	–
Ferland et al. (2019)	11	Healthy adults	Not reported (18–40 range)	2 ms, 3 ms	–
Ferland et al. (2021)	17	Healthy adults	26.0 ± 5.4	2 ms, 3 ms	–
Harris et al. (2021)	81	36 children with ADHD 45 typically developing	10.6 ± 1.4 10.5 ± 1.5	1–4 ms	Pediatric sample
Herman et al. (2018)	46	24 healthy young adults 22 healthy older adults	31.4 ± 12.1 66.3 ± 37.2	1 ms, 3 ms	Wide age variance in older group
Mooney et al. (2017)	30	15 healthy young adults 15 healthy older adults	24.6 ± 1.1 70.3 ± 1.7	1 ms, 3 ms	–
Stagg et al. (2011)	24	Healthy adults (Exp 1 & 2)	Mean 25 (range 19–46)	1 ms, 2.5 ms	Two experiments with similar design
Tremblay et al. (2012)	24	Healthy adults	24.7 ± 4.1	3 ms	–

Two reviewers independently extracted the following from each eligible study: author, year, sample size, population characteristics, SICI protocol (e.g., delay interval), brain region for MRS voxel, correlation coefficient (r), and p-values. If a study reported multiple effect sizes for different SICI intervals (e.g., 1 ms, 2.5 ms, 3 ms), each was treated as an independent observation, as these reflect distinct inhibitory mechanisms. Studies using different

age groups or cortical regions were similarly treated as separate entries.

When SICI was defined such that higher values represented reduced inhibition (e.g., MEP ratio format), the sign of the correlation coefficient was inverted to ensure consistent directional interpretation, namely, that positive correlations indicate greater GABA associated with more inhibition.

The primary effect size for this meta-analysis was the correlation coefficient (r) quantifying the relationship between magnetic resonance spectroscopy (MRS)-derived cortical GABA levels and transcranial magnetic stimulation (TMS)-measured short-interval intracortical inhibition (SICI).

The correlation coefficient (r) was extracted directly from each included study. To ensure a consistent directional interpretation across all studies, the sign of r was inverted in instances where the SICI metric was defined such that a higher value indicated less inhibition (e.g., a higher motor-evoked potential ratio). After this adjustment, a positive correlation consistently indicated that higher GABA concentrations were associated with stronger intracortical inhibition, while a negative correlation indicated that higher GABA was associated with weaker inhibition.

Prior to statistical synthesis, all correlation coefficients were transformed into Fisher's Z scores to normalize their sampling distributions and stabilize their variances, as the raw *r* scale is bounded and can be biased, especially for values near -1 or +1 [1, 2]. The transformation was performed using the standard formula:

Fisher's Z = 0.5 * ln ((1 + r) / (1 - r))

where ln is the natural logarithm. The standard error (SE) for each Fisher's Z score was calculated as:

SE = 1 / √ (N - 3)

where N is the study's total sample size. All meta-analytic procedures, including pooling and the calculation of confidence intervals, were performed using these transformed Fisher's Z scores.

For final interpretation and reporting, the pooled Fisher's Z estimate and its 95% confidence interval were back-transformed to the correlation metric (r) using the inverse transformation:

r = (e^(2Z) - 1) / (e^(2Z) + 1)

Meta-Analysis

All meta-analyses were conducted using Stata/BE version 18. A random-effects model was employed, utilizing the restricted maximum likelihood (REML) estimator to account for both within-study variance and between-study heterogeneity. The primary effect size measure was the correlation coefficient (r). Individual correlation coefficients were first transformed into Fisher's Z values to normalize their sampling distributions and stabilize their variances for analysis. The pooled estimate was then calculated as a weighted average of these Fisher's Z scores. For interpretability, the resulting pooled Fisher's Z and its confidence interval were back transformed to a correlation coefficient (r). The statistical significance of the pooled effect was evaluated using a z-test, with a two-tailed p-value < .05 deemed significant.

Assessment of Heterogeneity

Between-study heterogeneity was quantitatively assessed using three complementary measures: Cochran's Q statistic, the I^2 index, and the estimated variance component τ^2 (tau-squared). The I^2 index, which describes the percentage of total variation across studies that is attributable to heterogeneity rather than chance, was interpreted as follows: 25%, 50%, and 75% represented low, moderate, and high heterogeneity, respectively.

Statistical Analysis of Bias

To assess potential publication bias, we generated a funnel plot of standard error against Fisher's Z and conducted Egger's linear regression test (Sterne & Egger, 2005). The intercept (β_1) in Egger's test quantifies small-study effects, with H_o indicating no bias ($\beta_1 = 0$)

Risk of Bias

Risk of bias was assessed using the ROBINS-I tool covering seven domains including confounding, participant selection, exposure/outcome measurement, missing data, and reporting bias.[13] Two independent reviewers rated each study; disagreements were resolved by consensus. Summary ratings are presented in Table 2.

RESULTS

A total of 23 effect sizes were extracted from published studies examining the relationship between cortical GABA levels (measured via MRS) and short interval intracortical inhibition (SICI; measured via TMS).

The forest plot (Fig. 1) illustrates the individual study-level effect sizes (Fisher's Z) with corresponding 95% confidence intervals (CIs). The pooled effect size, calculated using a random-effects model, was Z = −0.14 (95% CI: −0.26 to −0.02), indicating a small but statistically significant negative association between GABA concentrations and SICI (z = −2.23, p = .03). This suggests that higher cortical GABA levels are modestly associated with stronger motor cortical inhibition.

Effect sizes varied across studies, with several small studies reporting stronger associations (e.g., Stagg et al. Experiment 2: Z = −1.07), while others reported null or slightly positive effects.[19]

Heterogeneity and Subgroup Analyses

Heterogeneity was low to moderate across studies (I^2 = 35.5%), with a τ^2 of 0.03 and Q-statistic of 34.73 (df = 22, p

Table 2. Study Characteristics: ROBINS-I Risk of Bias Assessment Summary:

Study (Year)	Confounding	Selection	Exposure (MRS)	Outcome (TMS)	Missing Data	Reporting	Overall Risk	Rationale
CUYP2021-Young	Moderate	Low	Moderate	Low	Low	Low	Moderate	Age-controlled but MRS voxel details unclear.
CUYP2021-Older	Serious	Low	Moderate	Low	Low	Low	Serious	Age-related GABA changes may confound SICS without stratification.
DYKE2017 (1ms/3ms)	Low	Low	Low	Low	Low	Low	Low	Clear protocols, homogeneous sample.
FERL2019 (A-D)	Moderate	Low	Serious	Moderate	Low	Low	Serious	Small sample (n=11), varied SICI intervals without justification.
FERL2021 (A-B)	Moderate	Low	Moderate	Moderate	Low	Low	Moderate	Inconsistent GABA-SICI directions in same cohort.
HARR2021	Serious	Moderate	Moderate	Moderate	Low	Low	Serious	ADHD vs. controls mixed; no separate analysis.
HERM2018 (1ms/3ms)	Moderate	Low	Low	Low	Low	Low	Moderate	Wide age range but controlled in analysis.
Moon2017 (1ms/3ms)	Moderate	Low	Moderate	Moderate	Low	Low	Moderate	Age-stratified but small samples per group.
Stag2011 (Exp 1-2)	Low	Low	Low	Low	Low	Low	Low	Rigorous design, clear protocols.
TREMB2012	Low	Low	Low	Low	Low	Low	Low	Standardized methods, complete reporting.

= .04), indicating some between-study variability. Despite this, the overall directionality of effect was consistent in showing a weak inverse relationship.

Subgroup or moderator analyses (e.g., by age, SICI interval, or voxel location) were not conducted due to insufficient stratified data across studies. Several included studies did not report demographic or methodological breakdowns in a format that allowed for consistent coding of potential moderators.

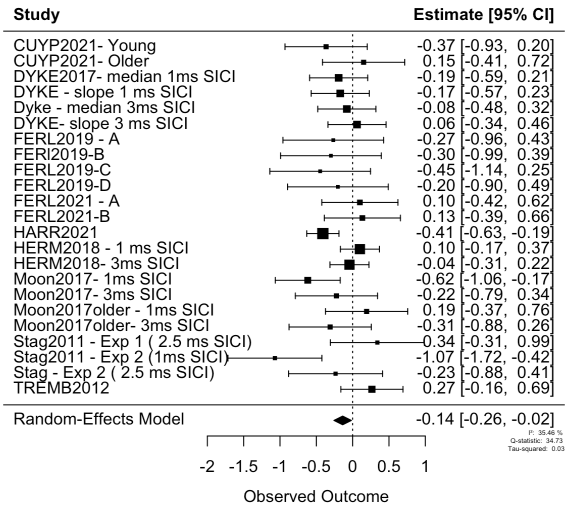

Figure 1. Forest Plot of the Association Between MRS-Measured Cortical GABA Levels and Short-Interval Intracortical Inhibition (SICI)

Note. Each square represents an individual effect size (Fisher's Z-transformed correlation coefficient) between cortical GABA levels and SICI, with the size of the square reflecting the study's inverse-variance weight. Horizontal lines indicate 95% confidence intervals. The diamond represents the pooled effect size under a random-effects model. A negative effect size indicates that higher GABA concentrations are associated with stronger intracortical inhibition.

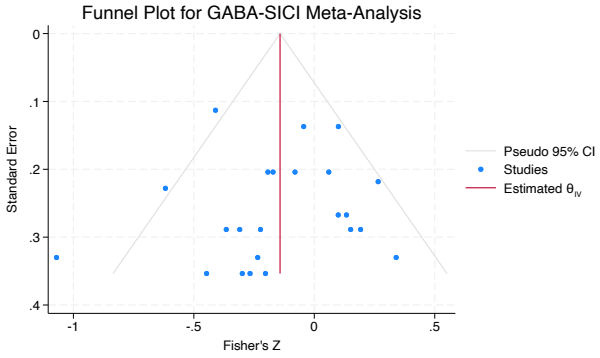

Figure 2. Funnel Plot of Publication Bias Assessment

Note. Funnel plot of standard error versus Fisher's Z for studies examining the GABA-SICI relationship. Dashed lines represent pseudo 95% confidence intervals. The asymmetric distribution of smaller studies (top-left) was not statistically significant per Egger's test ($\beta_1 = -0.46$, SE = 0.83, *z* = −0.55, *p* = .58).

Publication Bias

Visual inspection of the funnel plot (Fig. 2) revealed mild asymmetry, with a cluster of smaller studies reporting stronger negative effects. However, Egger's test did not reach significance ($\beta_1 = -0.46$, SE = 0.83, z = −0.55, p = .58), suggesting no statistically detectable small-study effects. This implies that the observed heterogeneity ($I^2 = 35.5\%$) is more likely attributable to methodological variability (e.g., SICI intervals, MRS protocols) than to publication bias.

DISCUSSION

This meta-analysis synthesizes 23 effect sizes from studies employing magnetic resonance spectroscopy (MRS) and transcranial magnetic stimulation (TMS) to investigate the relationship between cortical GABA levels and short-interval intracortical inhibition (SICI). Our findings demonstrate a small but statistically significant negative association (pooled Fisher's Z = –0.14, 95% CI: –0.26 to –0.02, p = .03), suggesting that greater GABA concentrations are modestly associated with increased inhibitory tone in the motor cortex.

These results corroborate established neurophysiological models identifying GABA as a critical modulator of intracortical inhibition.[14,15] The observed negative association was consistent across diverse demographic groups, variations in SICI protocols (e.g., 1-3 ms interstimulus intervals), and differences in MRS methodologies, including voxel placement and field strength.[4,16] This convergence suggests that the relationship between cortical GABA levels and intracortical inhibitory function is robust, although modest in magnitude.

The small pooled effect size (Fisher's Z = –0.14) likely reflects the inherent complexity of bridging distinct neurobiological scales. Specifically, MRS captures tonic, regionally averaged GABA concentrations, representing a static index of inhibitory tone, whereas TMS-based SICI measures phasic, stimulus-locked inhibition mediated predominantly by GABA-A receptors.[11,17] This methodological dissociation underscores the challenges of linking neurochemical markers with dynamic neurophysiological processes and may partially account for the modest strength of association observed. While prior individual studies have reported inconsistent findings, likely due to small sample sizes, protocol variations, and analytical differences, our pooled analysis reveals a consistent negative trend when these sources of variation are harmonized. This supports the role of baseline GABAergic tone as a contributing factor to cortical inhibitory function. Moderate between-study heterogeneity (I^2 = 35.5%) suggests some variability in effect sizes, potentially due to differences in age distribution, SICI protocols, or voxel placement for GABA quantification. Nonetheless, the overall directionality of effects was consistent, and no single study unduly influenced the meta-analytic outcome, further reinforcing the reliability of the findings.

While this meta-analysis advances understanding of the GABA-SICI relationship, several limitations must be acknowledged. First, despite using a random-effects model to account for methodological variability, residual confounding from differences in SICI protocols (e.g., 1 ms vs. 3 ms intervals,MRS voxel placement, and field strength may influence the pooled estimate.[4,18] Although Egger's test showed no significant publication bias (p= 0.58), funnel plot asymmetry suggests potential underrepresentation of small studies with null effects, possibly inflating the observed association.[19] The predominance of small sample sizes (median n≈ 20) limits statistical power, while treating multiple effect sizes from single studies as independent may underestimate variance due to partial interdependence.[20] Crucially, inconsistent reporting of variables like age, sex, and MRI parameters precluded subgroup analyses to identify effect modifiers. The cross-sectional nature of included studies also prevents causal inference, highlighting the need for longitudinal or pharmaco-MRS-TMS designs to establish directionality. Finally, generalizability is restricted to healthy populations, as the GABA-SICI relationship may differ in clinical conditions with GABAergic dysfunction. Future studies should address these gaps through standardized protocols, larger samples, and clinical validation.

This meta-analysis provides the first quantitative synthesis of the relationship between cortical GABA levels and SICI in healthy human populations. The findings support the premise that baseline GABA availability contributes to individual differences in motor cortical inhibition and establish a foundation for future research into inhibitory dysfunction in neuropsychiatric disorders.

Future studies should aim for greater methodological consistency, larger and more diverse samples, and multimodal designs integrating MRS, TMS, and electrophysiological techniques (e.g., EEG or MEG). Additionally, applying these approaches to clinical populations with known GABAergic dysregulation (e.g., schizophrenia, bipolar disorder, epilepsy etc.) may reveal disorder-specific inhibitory phenotypes and inform the development of neuromodulatory biomarkers. As GABAergic circuits represent both a pathophysiological marker and a potential therapeutic target, elucidating the link between neurochemistry and intracortical inhibition may advance precision psychiatry and guide the development of individualized interventions.

CONCLUSION

This meta-analysis revealed a modest but significant negative association between cortical GABA levels and SICI strength (Fisher's Z = −0.14, p* = .03), supporting GABA's role in motor cortical inhibition. Despite methodological variability, the consistency of this relationship across studies highlights its neurobiological relevance. Future research should adopt standardized protocols and explore clinical applications in disorders with GABAergic dysfunction.

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