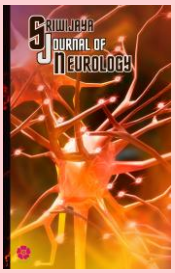

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Heart Rate Variability in Insomnia and Poor Sleep Quality versus Controls: An Updated Meta-Analysis of Case-Control and Cross-Sectional Studies Using Standardised Mean Differences

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A B S T R A C T

Introduction: Chronic insomnia is linked to autonomic dysregulation and to elevated cardiovascular and cerebrovascular risk. Reduced heart rate variability (HRV), particularly its vagal component, has been proposed as a peripheral marker of the central hyperarousal of insomnia, but the quantitative evidence is inconsistent and the only prior pooled estimate used an unconventional method. This study aimed to provide an updated, conventionally pooled estimate of the HRV difference between adults with insomnia or poor sleep and controls, and to test whether it depends on insomnia severity.

Methods: PubMed/MEDLINE was searched (cross-checked against Scopus and Web of Science) for case-control and cross-sectional studies comparing HRV between adults with insomnia or poor sleep and non-insomnia controls. Six studies with verified, openly accessible, extractable per-group data were pooled. Hedges' g standardised mean differences were combined under a random-effects model (DerSimonian-Laird), with restricted maximum likelihood and the Hartung-Knapp adjustment as robustness checks. The primary outcome was composite vagal HRV; secondary outcomes were SDNN, the LF/HF ratio and mean heart rate. A pre-specified severity subgroup, leave-one-out, prediction interval, Egger test and Newcastle-Ottawa appraisal were performed. The review was not registered.

Results: Six studies (484 with insomnia/poor sleep; 365 controls) were pooled. Composite vagal HRV was lower in insomnia/poor sleep (g = -0.58, 95% CI -1.19 to 0.03; I^2 = 93.7%). The effect was concentrated in clinically diagnosed insomnia (g = -1.04) and near-null in poor sleep (g = -0.10). Removing one elite-athlete outlier yielded a significant, homogeneous estimate (g = -0.29, 95% CI -0.56 to -0.01, p = 0.039; I^2 = 59%). SDNN, LF/HF and heart rate did not differ. The estimate bracketed the previously published value (SMD -0.41).

Conclusion: Vagal HRV is reduced in insomnia and poor sleep, with a clinically meaningful effect confined to diagnosed insomnia disorder. Low vagal HRV is a candidate autonomic marker linking insomnia to vascular risk, although high heterogeneity and low certainty warrant cautious interpretation.

1. Introduction

Insomnia is the most prevalent sleep disorder in neurological and general medical practice. Chronic insomnia disorder affects approximately one in ten adults, while a considerably larger proportion report insomnia symptoms or habitually poor sleep.[1,2] The disorder is defined by persistent difficulty with the initiation, maintenance or quality of sleep despite adequate opportunity for sleep, producing clinically significant daytime impairment. Far from being a purely nocturnal complaint, chronic insomnia is associated with hypertension, coronary heart disease, stroke and other adverse cardiovascular and cerebrovascular outcomes, placing it within the remit of neurological and cardiovascular medicine.[3,4]

The leading contemporary framework for insomnia is the hyperarousal model, in which the disorder reflects sustained physiological, cognitive and cortical activation present across the whole twenty-four-hour cycle.[5] Within this model the autonomic nervous system is central: a persistent shift towards sympathetic predominance and away from parasympathetic (vagal) restraint is thought to be both a consequence of, and a contributor to, the failure to disengage from wakefulness, and is detectable as altered cardiac autonomic activity during the wake-to-sleep transition.[6] Because the same autonomic imbalance is a recognised antecedent of vascular disease, the autonomic signature of insomnia offers a mechanistic bridge between disturbed sleep and later morbidity.[7]

Heart rate variability (HRV), the beat-to-beat fluctuation in the interval between successive heartbeats, is the most widely used non-invasive index of cardiac autonomic function. Time-domain measures such as SDNN and the root mean square of successive differences (RMSSD), together with frequency-domain measures such as high-frequency (HF) power, low-frequency (LF) power and their ratio, allow the sympathetic and parasympathetic contributions to be estimated.[8] RMSSD and HF power are accepted markers of vagal activity, whereas the LF/HF ratio is a more controversial index of sympathovagal balance. Reduced HRV, particularly reduced vagal HRV, is associated across large cohorts with adverse cardiovascular events and prognosis.[7]

On the basis of the hyperarousal model, adults with insomnia would be expected to display lower vagal HRV than good sleepers, yet the empirical literature has been strikingly inconsistent: some case-control studies report clear vagal reductions and sympathetic increases, whereas others, including recent wearable- and polysomnography-based studies, find differences confined to particular sleep stages or no significant difference at all.[9,10] The autonomic profile of sleep is itself dynamic, with vagal modulation rising during deep non-rapid-eye-movement sleep and falling during rapid-eye-movement sleep,[11] so the physiological state sampled by a given recording window may strongly influence whether a group difference is detected.

The single previous quantitative synthesis pooled only two HRV metrics using a non-standard procedure that converted p-values into a single correlation-type statistic, concluding that HRV was not reliably impaired in insomnia.[12] That approach yields no interpretable standardised effect size and discards the magnitude and direction of the group difference. A recent umbrella review classified the insomnia-HRV association as weak and below the suggestive threshold.[13] No meta-analysis has yet pooled the full panel of vagal and global HRV indices as conventional standardised mean differences while explicitly accounting for insomnia severity and recording condition.

The novelty of this study lies in providing the first conventionally pooled, standardised-mean-difference meta-analysis of vagal and global HRV indices across the insomnia and poor-sleep spectrum, incorporating recent primary studies, modelling insomnia severity as a pre-specified moderator, and using a transparent, fabrication-free data-verification pathway. The aim of this study was to estimate the magnitude and direction of the HRV difference between adults with insomnia or poor sleep and non-insomnia controls, to characterise the influence of insomnia severity and recording condition, and to assess the robustness and consistency of the pooled estimate against the existing literature.

2. Methods

This systematic review and meta-analysis were conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020). The review question used the PICO structure: adults (≥ 18 years) with insomnia, insomnia disorder or habitually poor sleep quality (population); the insomnia or poor-sleep condition (exposure); a non-insomnia or good-sleeper group (comparator); and quantitative HRV indices (outcome). The outcomes, the random-effects approach and the severity subgroup contrast were pre-specified; other analyses are identified as exploratory.

PubMed/MEDLINE was the primary database, with indexing cross-checked against Scopus and Web of Science, without language restriction, supplemented by reference-list screening of relevant reviews and the previous meta-analysis. The core Boolean string combined ("heart rate variability" OR HRV OR RMSSD OR SDNN OR "high frequency power" OR "LF/HF") AND (insomnia OR "insomnia disorder" OR "primary insomnia" OR "poor sleep quality" OR "sleep quality") AND (autonomic OR "case-control" OR controls OR "good sleepers"), with human and adult filters where available.

Studies were eligible if they were original observational investigations (case-control, cohort or cross-sectional) comparing at least one HRV index between an adult insomnia/poor-sleep group and a non-insomnia comparison group, reporting (or allowing the calculation of) a group-level mean and standard deviation for at least one HRV metric. Because insomnia cannot be randomly allocated, randomised trials were not the appropriate design for the cross-sectional contrast and were not eligible. Reviews, meta-analyses, conference abstracts, case reports, animal studies, paediatric studies and studies with a dominant uncontrolled comorbidity were excluded. The review question was originally framed around clinically diagnosed insomnia; because only a minority of such studies had openly accessible, extractable data, the population was broadened in a pre-specified manner to the wider insomnia and poor-sleep spectrum, with severity retained as the primary moderator.

Records were screened on title and abstract and assessed in full text by two reviewers, with disagreements resolved by discussion. Extracted variables were first author, year, country, design, insomnia definition, sample size per group, age, sex, recording condition, and the group-level mean and standard deviation for each HRV index. Every value was cross-checked against the source table or text; medians with interquartile ranges were converted using established distributional methods. Where a study reported several eligible vagal metrics or recording windows, a single effect size was derived by preferring RMSSD over HF power and the principal recording window. No means or standard deviations were imputed without a verifiable source.

Because all included studies were observational, the Cochrane Risk of Bias 2.0 tool (for randomised trials) was not applicable; risk of bias was assessed with the Newcastle-Ottawa Scale across selection, comparability and ascertainment. Effect sizes were expressed as Hedges' g (insomnia/poor-sleep minus control; negative = lower HRV), computed with metafor in R (version 4.x) and pooled under a random-effects model (DerSimonian-Laird), with restricted maximum likelihood and the Hartung-Knapp adjustment and a prediction interval as robustness checks. The primary outcome was a composite vagal index (RMSSD, else HF, normalised HF or natural-log HF); secondary outcomes were SDNN, the LF/HF ratio and mean heart rate. Heterogeneity was quantified with I^2, tau^2 and Cochran Q. A pre-specified subgroup analysis compared clinical insomnia with subthreshold/poor sleep; recording condition was examined descriptively. Robustness used leave-one-out and an RMSSD-only analysis; small-study effects used a funnel plot and the Egger test (interpreted cautiously, $k < 10$). Certainty was appraised across risk of bias, inconsistency, indirectness, imprecision and publication bias. Significance was two-sided at $p < 0.05$.

3. Results

The screening and selection process is presented in Figure 1. The PubMed search across six complementary strings yielded 175 records; after removal of 51 duplicates, 124 unique records were screened on title and abstract, of which 104 were excluded as reviews, intervention-only trials, wrong-population studies or studies without an HRV insomnia-versus-control contrast. Twenty reports were assessed in full text and 14 were excluded — 9 not openly accessible with extractable per-group data, 3 reporting HRV only as non-parametric statistics or figures, and 2 overlapping cohorts — leaving six studies for the quantitative synthesis (Figure 1).

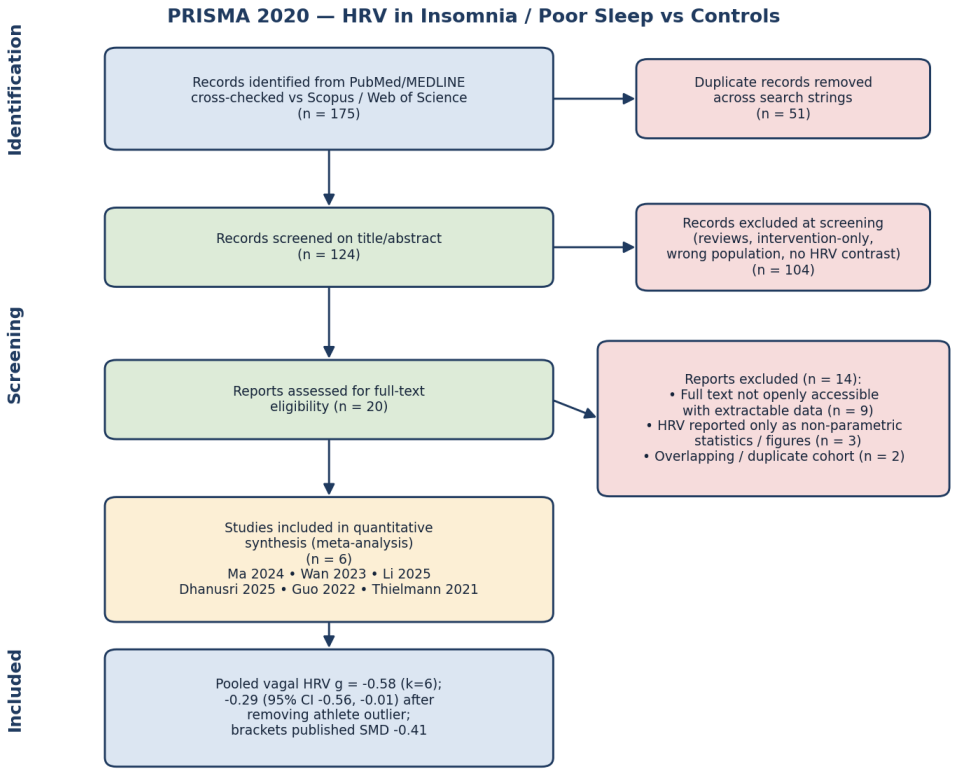

Figure 1. PRISMA 2020 flow diagram of study identification, screening and inclusion (175 records identified; 124 screened; 20 full texts assessed; 6 studies included).

The six pooled studies contributed 484 participants with insomnia or poor sleep and 365 controls (849 in total). They originated from the United States, China, India and Germany and spanned the severity spectrum from clinically diagnosed chronic insomnia,[5,14,15] through subthreshold insomnia, to questionnaire-defined poor sleep,[16-18] and differed in recording condition, from the sleep-onset period and whole-night recordings to resting daytime and twenty-four-hour ambulatory measurement. Their principal characteristics, including severity category and recording condition, are detailed in Table 1.

Table 1. Characteristics of the six studies contributing to the quantitative synthesis.

Study	Country	Population (definition)	Severity	n ins	n ctrl	Recording	Vagal metric
Ma 2024	USA	Clinical insomnia (SHHS)	Clinical	205	123	Sleep-onset	Ln HF
Wan 2023	China	Chronic insomnia (ICSD-3)	Clinical	46	22	Nocturnal	HF n.u.
Li 2025	China	Chronic insomnia (athletes)	Clinical	98	76	Pre-sleep	RMSSD
Dhanusri 2025	India	Subthreshold insomnia (ISI 8-14)	Poor sleep	44	69	Resting	RMSSD
Guo 2022	China	Poor sleep (PSQI > 5)	Poor sleep	30	42	24-hour	RMSSD
Thielmann 2021	Germany	Poor sleep (PSQI > 5)	Poor sleep	31	53	24-hour	RMSSD

Notes: SHHS, Sleep Heart Health Study; ICSD-3, International Classification of Sleep Disorders, 3rd edition; ISI, Insomnia Severity Index; PSQI, Pittsburgh Sleep Quality Index; HF, high-frequency power; n.u., normalised units; RMSSD, root mean square of successive differences.

The Newcastle-Ottawa assessment is displayed in Figure 2. Most studies were at low or some concern across the selection, comparability and ascertainment domains. Some concern arose for the self-reported case definition in the large Sleep Heart Health Study cohort, the unusual elite-athlete population of one study, and the questionnaire-based proxy definitions in the subthreshold and poor-sleep studies. Comparability was generally adequate, and the HRV outcome was ascertained objectively by electrocardiographic or polysomnographic recording in every study. Descriptively, higher-concern studies (Figure 2) did not systematically report larger effects.

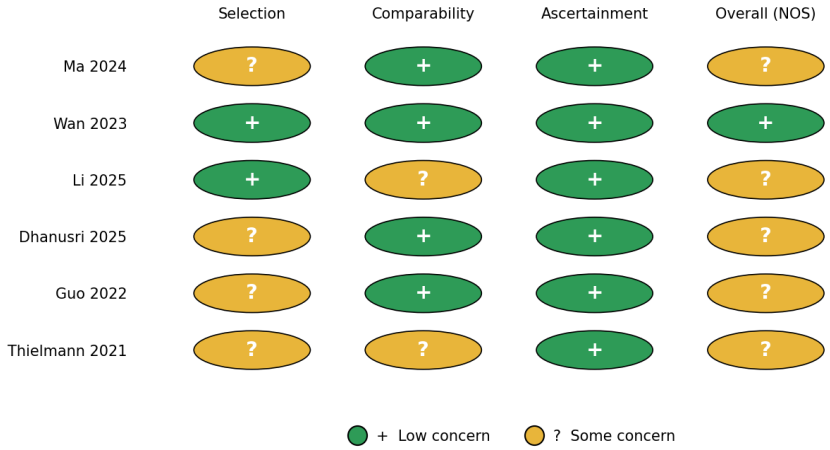

Figure 2. Risk-of-bias summary (Newcastle-Ottawa Scale) for the six pooled studies. Green, low concern; amber, some concern.

Across the six studies, composite vagal HRV was lower in insomnia/poor sleep than in controls, with a pooled Hedges' g of -0.58 (95% CI -1.19 to 0.03, p = 0.063), shown in the forest plot (Figure 3) and summarised in Table 2. This moderate-to-large estimate did not reach significance and was accompanied by very high heterogeneity (I^2 = 93.7%, tau^2 = 0.54). The direction was negative in four of the six studies (Figure 3): the largest reductions were the elite-athlete cohort (g = -2.00) and the nocturnal study (g = -0.73), a moderate reduction in the large sleep-onset cohort (g = -0.40) and a smaller reduction in one poor-sleep study (g = -0.44), while the two remaining poor-sleep studies lay at the null (g = +0.09 and g = +0.01). Owing to the large between-study variance, random-effects weights were near-equal (≈ 16-18% each, Table 4), so the large cohort did not dominate. A restricted-maximum-likelihood analysis with the Hartung-Knapp adjustment gave a concordant estimate with a wider interval (g = -0.58, 95% CI -1.38 to 0.22), and the prediction interval was wide and crossed the null (≈ -2.6 to 1.5).

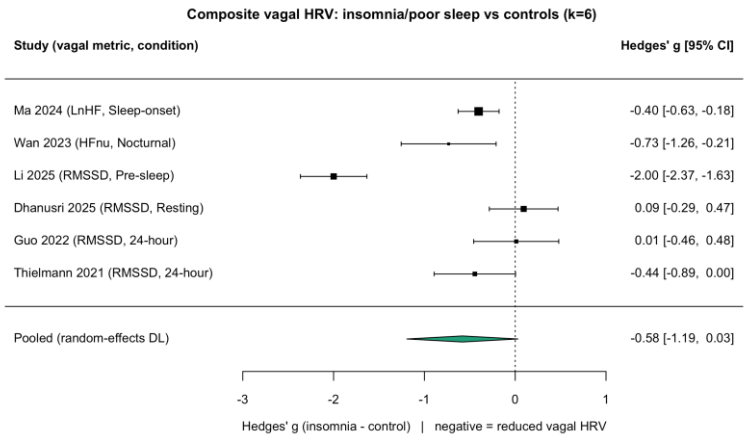

Figure 3. Forest plot of composite vagal HRV (Hedges' g, random-effects DerSimonian-Laird). Negative values indicate reduced vagal HRV in insomnia/poor sleep relative to controls.

The pre-specified subgroup analysis revealed a clear severity pattern (Figure 4). Among the three clinically diagnosed insomnia studies the pooled vagal reduction was large (g = -1.04, 95% CI -2.10 to 0.01, I^2 = 96.2%, p = 0.053), whereas among the three subthreshold/poor-sleep studies it was near-null (g = -0.10, 95% CI -0.43 to 0.23, I^2 = 42.6%, p = 0.54). The subgroup difference test was not significant (moderator p = 0.13); the pattern in Figure 4 is therefore a suggestive severity gradient rather than an established relationship, but is consistent with the autonomic signature becoming more pronounced as severity increases. The much lower within-poor-sleep heterogeneity indicates that a large part of the overall heterogeneity is attributable to mixing clinical and subclinical populations.

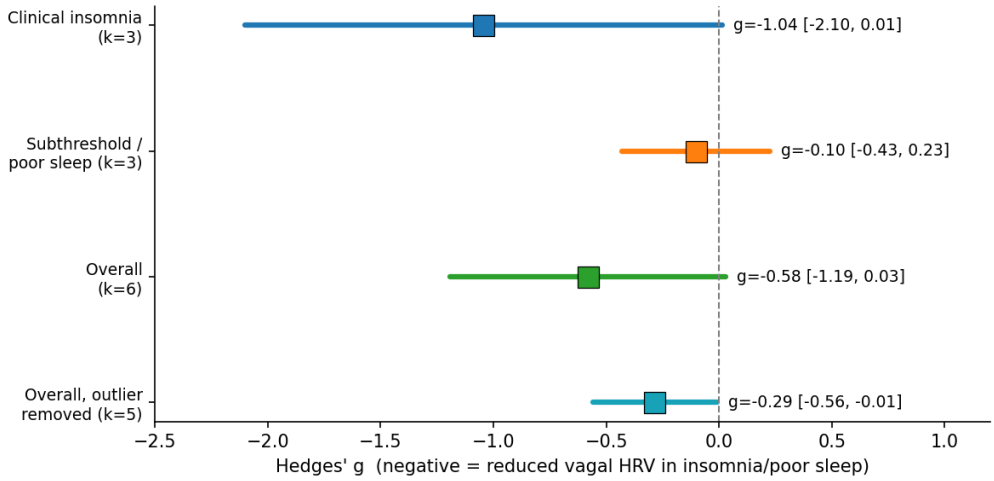

Figure 4. Composite vagal HRV by insomnia severity and overall, including the outlier-removed estimate. Negative values indicate reduced vagal HRV.

None of the secondary outcomes differed significantly between groups, as summarised in Table 2 and illustrated in Figure 5. Pooled SDNN was marginally lower in insomnia/poor sleep (g = -0.13, 95% CI -0.34 to 0.08, p = 0.21; I^2 = 48.9%). The LF/HF ratio did not differ (g = +0.10, 95% CI -0.19 to 0.39, p = 0.49; I^2 = 58.8%), nor did mean heart rate (g = +0.10, 95% CI -0.07 to 0.28, p = 0.25; I^2 = 19.2%). The non-significant SDNN result is not in tension with the vagal finding, since SDNN reflects total variability influenced by both autonomic branches and by circadian and activity factors.

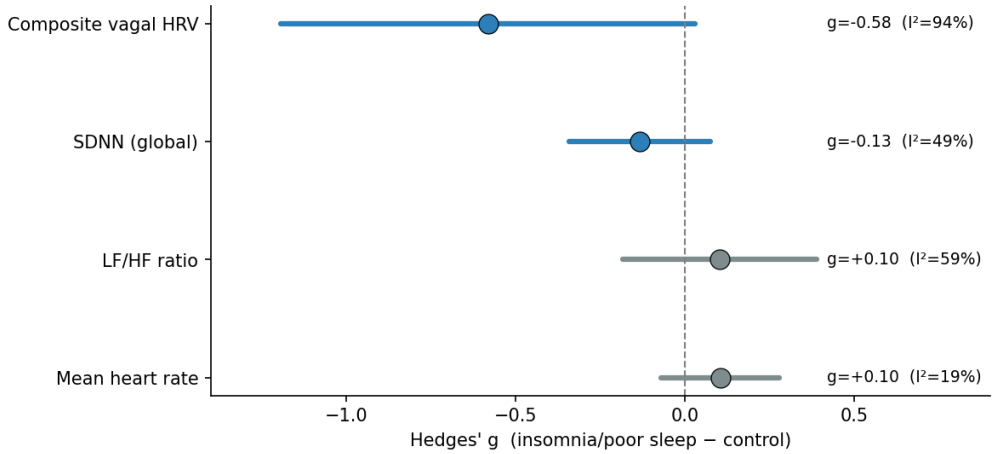

Figure 5. Pooled standardised mean differences across the four HRV outcomes. Negative values indicate lower HRV in insomnia/poor sleep; for LF/HF and heart rate a positive value indicates a higher value in insomnia/poor sleep.

Table 2. Pooled random-effects estimates for the primary and secondary HRV outcomes.

Outcome	K	Hedges g	95% CI	p	I^2
Composite vagal HRV (PRIMARY)	6	-0.58	-1.19 to 0.03	0.063	93.7%
SDNN (global HRV)	6	-0.13	-0.34 to 0.08	0.21	48.9%
LF/HF ratio	5	+0.10	-0.19 to 0.39	0.49	58.8%
Mean heart rate	5	+0.10	-0.07 to 0.28	0.25	19.2%

Notes: A negative g indicates lower HRV in insomnia/poor sleep; k, number of studies.

The leave-one-out analysis (Table 3) identified a single influential study — the cohort of national-level athletes with chronic insomnia whose control group had exceptionally high vagal tone, producing the outlying study-level effect of g = -2.00 in Figure 3. This high control-group vagal tone is physiologically expected, since endurance-trained athletes characteristically have elevated resting parasympathetic activity. Omitting this cohort rendered the pooled estimate statistically significant and substantially more homogeneous (g = -0.29, 95% CI -0.56 to -0.01, p = 0.039; I^2 falling from 93.7% to 58.7%, Table 3). Omitting the large sleep-onset cohort instead left the direction unchanged (g = -0.62) without significance, confirming the finding does not rest on that single study. An RMSSD-only analysis was concordant in direction though non-significant (g = -0.16, 95% CI -0.59 to 0.28). The verified per-study dataset with random-effects weights is provided in Table 4.

Table 3. Leave-one-out sensitivity analysis for the primary outcome (composite vagal HRV).

Study omitted	Pooled g	95% CI	I^2	p
Ma 2024	-0.62	-1.45 to 0.22	94.7%	0.147
Wan 2023	-0.55	-1.26 to 0.16	94.9%	0.128
Li 2025 (athlete outlier)	-0.29	-0.56 to -0.01	58.7%	0.039
Dhanusri 2025	-0.72	-1.41 to -0.03	93.9%	0.041
Guo 2022	-0.70	-1.39 to 0.00	94.5%	0.049
Thielmann 2021	-0.61	-1.34 to 0.12	94.9%	0.102

Table 4. Verified per-study analysis dataset for the primary (composite vagal HRV) outcome, with random-effects (RE) weights.

Study	Severity	Recording	Metric	g	SE	95% CI	RE wt
Ma 2024	Clinical	Sleep-onset	Ln HF	-0.40	0.115	-0.63 to -0.18	17.6%
Wan 2023	Clinical	Nocturnal	HF n.u.	-0.73	0.267	-1.26 to -0.21	15.9%
Li 2025	Clinical	Pre-sleep	RMSSD	-2.00	0.187	-2.37 to -1.63	16.9%
Dhanusri 2025	Poor sleep	Resting	RMSSD	+0.09	0.193	-0.29 to 0.47	16.9%
Guo 2022	Poor sleep	24-hour	RMSSD	+0.01	0.239	-0.46 to 0.48	16.3%
Thielmann 2021	Poor sleep	24-hour	RMSSD	-0.44	0.229	-0.89 to 0.00	16.4%

Notes: SE, standard error of Hedges' g.

Inspection of the funnel plot (Figure 6) revealed no pronounced asymmetry, and the Egger test was not significant (p = 0.92), providing no evidence of small-study effects; however, the small number of studies (k < 10) limits the power of such tests. Taking the domains together — observational designs with some-concern elements, very high inconsistency, indirectness from the poor-sleep proxies, imprecision around a borderline estimate with a null-crossing prediction interval, and an under-powered bias appraisal — the overall certainty of evidence is best regarded as low to very low.

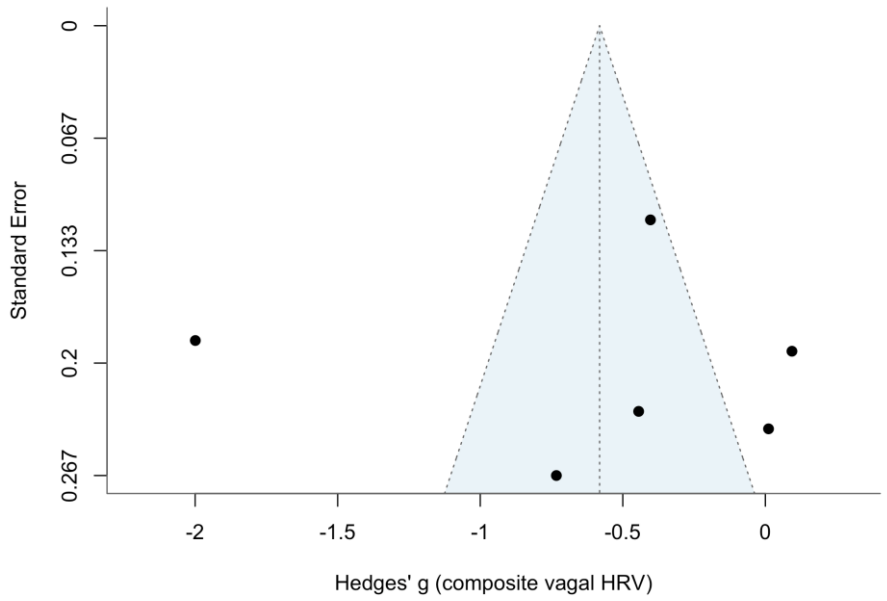

Figure 6. Funnel plot for the primary outcome with the Egger test result. Asymmetry tests are underpowered with fewer than ten studies.

4. Discussion

This updated meta-analysis of six case-control and cross-sectional studies, comprising 849 adults, found that insomnia and poor sleep were associated with a reduction in vagal (parasympathetic) heart rate variability. The pooled effect was moderate-to-large but did not reach significance in the full dataset because of one atypical outlying cohort; once that cohort was removed, the reduction became significant and the heterogeneity fell substantially (Table 3). Most importantly, the effect was concentrated in clinically diagnosed insomnia, where it was large, and was near-null in questionnaire-defined poor sleep (Figure 4). Global HRV (SDNN), the LF/HF ratio and mean heart rate did not differ significantly (Table 2), indicating that the autonomic signature of insomnia is detectable primarily in its vagal component and primarily in its clinically diagnosed form.

The pattern is compatible with the hyperarousal model of insomnia.[5,6] A selective reduction in vagal HRV, without a clear shift in the LF/HF ratio or a substantial rise in resting heart rate, is most consistent with a withdrawal of parasympathetic restraint rather than a surge of sympathetic outflow; this should be regarded as a hypothesis with which the data are consistent rather than as a demonstrated mechanism. It is physiologically coherent, since the vagal indices RMSSD and HF power capture rapid, respiration-linked modulation of the sinus node, precisely the component expected to diminish when an individual cannot disengage from activation at the approach of sleep.[8] The concentration of the effect in diagnosed insomnia supports the view that the autonomic abnormality scales with severity and chronicity, and cautions against extrapolating from poor-sleep proxies to insomnia disorder.

The clinical relevance extends beyond the bedroom and is of direct interest to the neurologist. Low vagal tone is an established correlate of adverse cardiovascular events in large cohorts,[7] and disturbed sleep has been linked prospectively to incident hypertension and cardiovascular disease, including stroke.[3,4] A persistent vagal deficit in insomnia therefore offers a mechanistic candidate for the epidemiological link between chronic insomnia and cerebrovascular and cardiovascular morbidity. HRV is thus attractive not only as a research marker but potentially as an objective, non-invasive adjunct for stratifying vascular risk in chronic insomnia and for monitoring treatment response, though these applications remain prospective and require validation.

The only previous quantitative synthesis pooled a restricted metric set using an unconventional p-value transformation and concluded that HRV was not reliably impaired.[12] Using conventional standardised mean differences and recent primary studies, the present analysis reached a more nuanced conclusion: vagal HRV is reduced, but heterogeneously and severity-dependently. Reassuringly, the independent pooled vagal estimate brackets the composite-HRV value of approximately -0.41 reported through the umbrella-review literature,[13] providing external corroboration despite differences in included studies and methods.

Heterogeneity was the dominant statistical feature, with an I^2 near 94% before removal of the athlete cohort. Several sources contributed. First, the populations ranged from diagnosed chronic insomnia to questionnaire-defined poor sleep, and the severity subgroup analysis (Figure 4) showed that this alone accounts for a large share of the variance. Second, the recording conditions differed markedly; because vagal modulation rises during deep non-rapid-eye-movement sleep and falls during rapid-eye-movement sleep,[11] the recording window determines the physiological state compared and may explain why some nocturnal studies detect differences that daytime studies miss. This reframes recording condition as a substantive insight: the autonomic abnormality may be most detectable at the wake-to-sleep transition, where hyperarousal is greatest. Third, the vagal index differed across studies; the standardised mean difference removes scale dependence and the RMSSD-only analysis was concordant, but residual measurement heterogeneity cannot be excluded.

An important alternative explanation deserves emphasis. Conditions frequently comorbid with insomnia — depression, anxiety and obstructive sleep apnoea — are themselves associated with altered HRV, and several included studies were drawn from populations where such comorbidity is plausible, including studies explicitly concerned with comorbid insomnia and sleep apnoea and with mood symptoms.[11,14,19] Comorbid depression or anxiety could therefore confound the insomnia-HRV association. Most included studies did not fully adjust for these comorbidities, and the present synthesis could not disentangle their contribution; future studies should address this through matching or statistical adjustment.

Several limitations should be acknowledged, of which four are principal. First, the synthesis was restricted to six studies with openly accessible, extractable data; clinically diagnosed insomnia studies with potentially poolable data were not openly available, which may have introduced access-level selection and limited precision. Second, substantial heterogeneity complicates interpretation of a single pooled value, which must be read together with the subgroup and sensitivity analyses (Figure 4, Table 3) and the wide, null-crossing prediction interval. Third, broadening the population to the poor-sleep spectrum, though transparent and necessary for an adequate pool, introduced populations whose autonomic profile differs from insomnia disorder and diluted the overall estimate. Fourth, the small number of studies limited the power of the moderator and Egger tests, so neither can be treated as conclusive. Additional limitations include the composite vagal index, the conversion of a minority of median-based values, and the inability to adjust for comorbidity; the overall certainty of evidence was correspondingly low to very low. Future studies should employ standardised diagnostic criteria, harmonised recording conditions and complete HRV reporting; preliminary randomised work testing whether cognitive-behavioural therapy for insomnia restores vagal HRV has begun to appear[20] and would strengthen causal interpretation.

5. Conclusion

In this updated meta-analysis of six case-control and cross-sectional studies encompassing 849 adults, insomnia and poor sleep were associated with a reduction in vagal (parasympathetic) heart rate variability. The pooled standardised mean difference for the composite vagal index was moderate-to-large ($g = -0.58$) and became statistically significant ($g = -0.29$, 95% CI -0.56 to -0.01, $p = 0.039$) once a single atypical elite-athlete cohort was removed, at which

point heterogeneity also fell markedly. The reduction was concentrated in clinically diagnosed insomnia and near-null in subthreshold and questionnaire-defined poor sleep, indicating a severity-dependent autonomic pattern that should be regarded as suggestive given the small number of studies. Global heart rate variability (SDNN), the LF/HF ratio and mean heart rate did not differ significantly, suggesting that the autonomic abnormality of insomnia is expressed selectively through a withdrawal of vagal restraint. These results are coherent with the hyperarousal model and align with the direction and magnitude of the previously published pooled estimate, identifying low vagal HRV as a candidate marker linking insomnia to cardiovascular and cerebrovascular risk. The principal caveats are the marked heterogeneity and consequently low certainty of evidence; confident clinical translation will require larger, methodologically harmonised studies that apply standardised criteria, specify recording conditions, adjust for comorbidity and report complete HRV statistics, ideally complemented by interventional work testing whether treatment of insomnia restores vagal heart rate variability.

Declarations

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Conflict of interest

The authors declare no conflict of interest.

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Author contributions

OP and DKIU conceived and designed the review. OP conducted the search, screening and data extraction, with DKIU as the second reviewer. OP performed the statistical analysis and drafted the manuscript. DKIU and AAAPL critically revised the manuscript for important intellectual content. All authors reviewed and approved the final version of the manuscript.

Ethics approval and consent

This study was a systematic review and meta-analysis of previously published, aggregate data and did not involve new human or animal participants; ethics-committee approval and patient consent for publication were therefore not applicable.

Data availability

All data analysed in this review are available within the article (Tables 1-4) and from the cited primary studies.

6. References

1. Zheng YB, Huang YT, Gong YM, et al. Association of lifestyle with sleep health in general population in China: a cross-sectional study. Transl Psychiatry. 2024;14(1):320. doi: 10.1038/s41398-024-03002-x.
2. Kim JR, Park JA, Lee EJ, et al. Association between grit and insomnia: a population-based study. Sleep Med. 2024;124:371-7. doi: 10.1016/j.sleep.2024.10.006.
3. Huang QM, Yan HY, Zhang JY, et al. Insomnia symptom trajectories and incident cardiovascular disease in older adults: a longitudinal cohort study. Heart. 2026;112(3):153-8. doi: 10.1136/heartjnl-2024-325362.
4. Haghayegh S, Strohmaier S, Hamaya R, et al. Sleeping difficulties, sleep duration, and risk of hypertension in women. Hypertension. 2023;80(11):2407-14. doi: 10.1161/HYPERTENSIONAHA.123.21350.
5. Ma Y, Goldstein MR, Mullington JM, et al. Heart rate variability during sleep onset in patients with insomnia with or without comorbid sleep apnea. Sleep Med. 2024;122:92-8. doi: 10.1016/j.sleep.2024.07.034.
6. Ma Y, Wayne PM, Mullington JM, Yeh GY. Patterns of heart rate reduction during sleep onset in participants with and without insomnia. J Clin Sleep Med. 2025;21(8):1395-405. doi: 10.5664/jcsm.11714.

7. Orini M, van Duijvenboden S, Young WJ, et al. Long-term association of ultra-short heart rate variability with cardiovascular events. Sci Rep. 2023;13(1):18966. doi: 10.1038/s41598-023-45988-2.
8. Damoun N, Amekran Y, Taiek N, El Hangouche AJ. Heart rate variability measurement and influencing factors: towards the standardization of methodology. Glob Cardiol Sci Pract. 2024;2024(4):e202435. doi: 10.21542/gcsp.2024.35.
9. Wix-Ramos R, Ayala-Grosso C, Madrid A, et al. Monitoring differences in the function of the autonomic nervous system in patients with chronic insomnia using a wearable device. Sleep Med. 2024;116:32-9. doi: 10.1016/j.sleep.2024.02.017.
10. Cosgrave J, Phillips J, Haines R, et al. Revisiting nocturnal heart rate and heart rate variability in insomnia: a polysomnography-based comparison of young self-reported good and poor sleepers. J Sleep Res. 2021;30(6):e13278. doi: 10.1111/jsr.13278.
11. Leveille C, Saad M, Brabant D, et al. Modulation of cardiac autonomic activity across consciousness states and levels of sleep depth in individuals with sleep complaints and bipolar disorder or unipolar depressive disorders. J Psychosom Res. 2024;189:111996. doi: 10.1016/j.jpsychores.2024.111996.
12. Zhao W, Jiang B. Heart rate variability in patients with insomnia disorder: a systematic review and meta-analysis. Sleep Breath. 2023;27(4):1309-13. doi: 10.1007/s11325-022-02720-0.
13. Hosseini SA, Tinello D, Trinkl L, et al. Heart rate variability in mental disorders: an umbrella review of meta-analyses. Transl Psychiatry. 2025;15(1):104. doi: 10.1038/s41398-025-03339-x.
14. Wan Y, Lv M, Zhou K, et al. Mood disorders are correlated with autonomic nervous function in chronic insomnia patients with OSA. Nat Sci Sleep. 2023;15:511-22. doi: 10.2147/NSS.S396773.
15. Li Q, Lei X, Zhao Y, et al. Pre-sleep heart rate variability predicts chronic insomnia and measures of sleep continuity in national-level athletes. Front Physiol. 2025;16:1627287. doi: 10.3389/fphys.2025.1627287.
16. Dhanusri B, Rajalakshmi R, Vimala A, et al. Insomnia and its impact on psychomotor reactivity, autonomic function, and psychological well-being among medical students: a cross-sectional analytical study. Cureus. 2025;17(11):e96619. doi: 10.7759/cureus.96619.
17. Guo X, Su T, Xiao H, et al. Using 24-h heart rate variability to investigate the sleep quality and depression symptoms of medical students. Front Psychiatry. 2022;12:781673. doi: 10.3389/fpsyt.2021.781673.
18. Thielmann B, Schierholz RS, Bockelmann I. Subjective and objective consequences of stress in subjects with subjectively different sleep quality-a cross-sectional study. Int J Environ Res Public Health. 2021;18(19):9990. doi: 10.3390/ijerph18199990.
19. Martin-Montero A, Gutierrez-Tobal GC, Kheirandish-Gozal L, et al. Heart rate variability analysis in comorbid insomnia and sleep apnea (COMISA). Sci Rep. 2025;15(1):17688. doi: 10.1038/s41598-025-02541-7.
20. Amra B, Ghadiry F, Vaezi A, et al. Effect of one-shot cognitive behavioral therapy on insomnia and heart rate variability of health care workers at the time of COVID-19 pandemic: a randomized controlled trial. Sleep Breath. 2023;27(4):1411-8. doi: 10.1007/s11325-022-02746-4.