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Association Between Sleep Quality and Heart Rate Variability in Adults: A Narrative Review

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Abstract: ***Background**: Poor sleep quality is increasingly recognised as a determinant of cardiac autonomic function, commonly indexed by heart rate variability (HRV). **Objective**: To synthesise evidence on the association between sleep quality and HRV in adults. **Methods**: A narrative review was conducted using a structured PECO-based search strategy targeting PubMed/PMC, and CINAHL. Eligible studies examined sleep quality (subjective and/or objective) and HRV in adults, published within the last 10 years. **Results**: Twenty-six studies were included: six in healthy/general adults, three in occupational (nursing/shift-work) populations, ten in clinical populations (obstructive sleep apnea [OSA], insomnia, comorbid insomnia and sleep apnea [COMISA], mild cognitive impairment, heart failure, and elderly patients with anxiety/depression), and seven systematic reviews, meta-analyses, or genetically informed (Mendelian randomization) studies. Thirteen of the 26 studies reported reduced or impaired HRV- lower time-domain (SDNN, RMSSD) and altered frequency-domain (elevated LF/HF) parameters- associated with poorer sleep quality, most consistently in clinical populations with OSA, insomnia, and comorbid sleep disorders. Findings in healthy adults and occupational populations were more heterogeneous, with several studies reporting null or parameter-dependent associations. **Conclusion**: Sleep quality and HRV are associated, particularly in clinical sleep-disordered populations, but the relationship is inconsistent in healthy populations and is moderated by how sleep quality and HRV are measured. Standardisation of measurement protocols and larger longitudinal studies are needed.*

Keywords: sleep quality; heart rate variability; autonomic nervous system; polysomnography; Pittsburgh Sleep Quality Index; rapid review

1. Introduction

Sleep quality is a multidimensional construct encompassing sleep continuity, depth, subjective restfulness, and daytime consequences, and is most commonly assessed using the Pittsburgh Sleep Quality Index (PSQI) or objective measures such as actigraphy and polysomnography (PSG) [1]. Heart rate variability (HRV), the beat-to-beat variation in the interval between heartbeats, is a widely used non-invasive marker of autonomic nervous system (ANS) function, reflecting the dynamic balance between sympathetic and parasympathetic (vagal) modulation of the heart [2,3]. Reduced HRV has been consistently linked to adverse cardiovascular outcomes, including hypertension, myocardial infarction, and all-cause mortality [4].

Sleep and cardiac autonomic regulation are physiologically intertwined: parasympathetic activity typically rises during non-rapid eye movement sleep, and sleep fragmentation, short sleep duration, and subjective poor sleep quality have each been proposed to blunt this restorative autonomic pattern [5]. This relationship has been studied across a spectrum of populations- from healthy adults and shift-working nurses to patients with obstructive sleep apnea (OSA), insomnia, and comorbid sleep disorders- using both subjective and objective measures of sleep quality.

Despite growing interest, the literature is heterogeneous in methodology, population, and reported direction of effect, with some studies reporting robust inverse associations between poor sleep quality and HRV, and others reporting null or inconsistent findings. A rapid, structured synthesis of this evidence is therefore timely to clarify the current state of knowledge, identify where the evidence is strongest, and highlight gaps that would benefit from more rigorous primary research or a full systematic review.

The objective of this narrative review was to synthesise the available evidence on the association between sleep quality (assessed subjectively and/or objectively) and HRV in adult populations, encompassing both healthy and clinical groups, and to characterise the consistency of this association across study designs and populations.

2. Methods

2.1 Review Design

This narrative review followed a condensed but structured approach informed by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) framework [6,7,8].

2.2 Eligibility Criteria

Eligibility was defined a priori using a Population–Exposure–Comparator–Outcome (PECO) framework (Table 1).

Table 1: PECO eligibility framework

Element	Definition applied in this review
Population	Adults (≥18 years); both healthy/general and clinical populations (e.g. obstructive sleep apnea, insomnia, comorbid insomnia and sleep apnea, mild cognitive impairment, heart failure, elderly with anxiety/depression) were eligible
Exposure	Sleep quality- assessed subjectively (e.g. Pittsburgh Sleep Quality Index, self-report) and/or objectively (actigraphy, polysomnography)
Comparator	Good vs. poor sleep quality; higher vs. lower sleep-quality scores; sleep-disordered vs. non-disordered groups
Outcome	Heart rate variability- time-domain (SDNN, RMSSD, pNN50), frequency-domain (LF, HF, LF/HF), or non-linear HRV parameters
Study designs	Cross-sectional, case-control, cohort, and secondary/registry analyses; systematic reviews and meta-analyses included for evidence synthesis; intervention studies included only where baseline/observational sleep–HRV data were reported
Timeframe	Published within the last 10 years (2016–2026)
Language	English

2.3 Information Sources and Search Strategy

A structured Boolean search strategy combining controlled vocabulary (MeSH/CINAHL headings) and free-text terms for "sleep quality" and "heart rate variability," limited to adult populations and English-language records published within the last 10 years, was developed for both PubMed and CINAHL. Given the narrative-review timeframe, PubMed, PubMed Central, and adjacent openly accessible biomedical sources (journal publisher platforms) were searched directly.

2.4 Study Selection

Records were screened against the PECO eligibility criteria in a single-reviewer rapid-screening process, consistent with accepted narrative review conventions [6,8].

2.5 Data Extraction

For each included study, the following were extracted: author(s) and year, country, study design, population and sample size, sleep-quality measure (subjective/objective), HRV parameters reported, and the direction of the reported association. Given the heterogeneity of populations, designs, and outcome metrics, findings were synthesised narratively rather than pooled quantitatively.

3. Results

3.1 Study Selection

The structured search process identified 34 candidate records. After consolidating duplicate/overlapping entries (n=2) and screening the remaining 32 unique records against the PECO eligibility criteria, 26 studies were included in the narrative synthesis. Six excluded records did not meet eligibility criteria: one measured an outcome other than HRV, two were trial registry entries without reported results, one examined an exposure other than sleep quality, one fell outside the 10-year publication window, and one was a non-peer-reviewed secondary summary rather than a primary source.

3.2 Study Characteristics

Table 2 summarises the characteristics of the 26 included studies, ordered as: healthy/general adult populations (references 9–14), occupational populations of nurses and shift workers (15–17), clinical populations including OSA, insomnia, COMISA, mild cognitive impairment, heart failure, and elderly patients with anxiety/depression (18, 20–22, 25–30), and systematic reviews, meta-analyses, or genetically informed studies (19, 23, 24, 31–34- note that references 19 and 23 are also listed within their respective clinical population sections, as they provide both primary synthesis and higher-level evidence).

Table 2: Characteristics of the 26 included studies

Study	Country	Population / n	Design	Sleep quality measure	Key HRV-related finding
Sajjadieh et al., 2020	Iran	Hospital staff, n=260	Cross-sectional	PSQI	Poorer sleep quality/efficiency associated with lower SDANN; sleep duration alone not associated with HRV
Guo et al., 2022	China	Medical students, n=74	Cross-sectional	PSQI + 24-h ECG	No significant correlation between 24-h HRV indices and PSQI global score; bedtime SDNN correlated with sleep-disturbance subscale
Herhaus et al., 2022	Germany	Healthy adults, n=26	Pre–post pilot (HRV-biofeedback)	PSQI + actigraphy	4-week HRV-biofeedback improved subjective sleep quality and increased SDNN/total power/LF; no change in actigraphy metrics
Hannon et al., 2025	Ireland	Healthy adults, n=41	14-day observational	Daily self-reported sleep rating	Higher daily RMSSD associated with better self-reported sleep quality
Fein et al., 2026	USA (MIDUS)	Adults, n=391	Cross-sectional	PSQI + actigraphy	No direct main effect of sleep quality/duration on HRV; interacted with exercise — short sleep + inadequate exercise linked to lower HF-HRV/RMSSD
Fein et al., 2024 (conf. abstract)	USA (MIDUS)	Adults, n=391	Cross-sectional	Actigraphy	Sleep duration alone not associated with HRV; combined with inadequate exercise frequency, lower HF-HRV/RMSSD
Hsu et al., 2021	Taiwan	Female nurses, n=393	Cross-sectional	PSQI	Negative correlation between PSQI global score and total power, low frequency, and LF/HF ratio
Burch et al., 2019	USA	Nurses, n=18	Ambulatory pilot	Shift status (day vs. night)	Night nurses lacked the circadian rise in HRV coherence during sleep seen in day nurses
Panwar et al., 2024	India	Female nurses, n=38	Cross-sectional	PSQI	Night-shift nurses had higher PSQI and lower SDNN/total power/HF, and higher LF/HF, than morning-shift nurses
Lin et al., 2023	Taiwan	OSA referrals, n=62	Cross-sectional	Polysomnography (AHI)	RMSSD negatively correlated with BMI/neck circumference; HRV parameters associated with OSA severity markers
Sequeira et al., 2019	Brazil	OSA vs. controls (systematic review)	Systematic review	Polysomnography	Adults with OSA show diminished vagal tone and higher sympathetic responsiveness vs. controls
Statello et al., 2023	Italy	Suspected OSA referrals	Cross-sectional	Polysomnography/ECG	Nocturnal HRV parameters help predict severe OSA/sleep-disordered breathing

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Ucak et al., 2024	Australia	OSA patients (Sydney Sleep Biobank)	Cross-sectional	Polysomnography + Epworth Sleepiness Scale	HRV profile differs between "sleepy" and "non-sleepy" OSA patients, suggesting differential cardiovascular risk
McCall et al., 2023	USA	Adults with/without insomnia symptoms	Case-control (2 studies)	Insomnia symptom severity	Lower daytime HRV (higher arousal) in adults with insomnia symptoms; correlated with symptom severity
Dodds et al., 2017	Australia	Insomnia patients (22 studies reviewed)	Critical review	Mixed across studies	Inconsistent, high risk-of-bias evidence; HRV impairment in insomnia not conclusively demonstrated
Yan et al., 2025	China	Population GWAS data (FinnGen)	Mendelian randomization	Genetic HRV traits / insomnia diagnosis	Genetically predicted lower HRV (pvRSA/HF, RMSSD, SDNN) causally associated with increased insomnia risk; reverse direction not significant
Martín-Montero et al., 2025	Spain/ Germany (SHHS)	COMISA vs. OSA/insomnia/controls, n=5,335 ECGs	Cross-sectional (secondary analysis)	Polysomnography + insomnia questionnaire	COMISA shows reduced parasympathetic activity while awake and heightened sympathetic activity during sleep, distinct from isolated OSA or insomnia
Ma et al., 2024	USA	Insomnia with/without OSA, n=328	Cross-sectional	Self-reported insomnia + polysomnography	Insomnia (regardless of OSA severity) associated with higher heart rate and lower SDNN at sleep onset; comorbid disease showed lowest HRV
Muñoz-Martínez et al., 2025	Spain	Syncope/OSA patients, n=304	Multicentre cross-sectional	Respiratory polygraphy	Distinct nocturnal HRV patterns identified across syncope, OSA, and comorbid groups vs. controls
Grässler et al., 2021	Germany	Older adults with MCI, n=42	Cross-sectional	PSQI	Largely non-significant associations between resting HRV and PSQI in MCI; one significant correlation (HF-nu and sleep efficiency)
Krishnan et al., 2026	UK	Older heart failure patients, n=150	Cross-sectional (actigraphy)	Actigraphy (sleep efficiency; HRV not directly measured)	Poor actigraphic sleep efficiency associated with higher NT-proBNP and worse frailty/functional outcomes
Liu et al., 2025	China	Elderly, n=355	Case-control	Clinical sleep-disorder classification	Sleep disorders independently predicted reduced pNN50 and SDANN; anxiety and depression showed additional independent effects
Zhang et al., 2025	China	RCT participants (meta-analysis), n=549	Systematic review/meta-analysis of RCTs	Experimental sleep deprivation	Sleep deprivation associated with significantly reduced RMSSD and increased LF and LF/HF ratio; SDNN/HF changes non-significant
Zhao & Jiang, 2023	China	Insomnia disorder patients (17 studies), n=921	Systematic review/meta-analysis	SDNN / HF-norm	Small but statistically significant pooled reduction in HRV in insomnia disorder (R-equivalent = 0.19, 95% CI 0.12–0.25)
Correia et al., 2023	South Africa	Mixed populations (systematic review)	Systematic review	Mixed across studies	Sleep-related HRV associated with both sleep and depressive/anxiety symptoms, though findings mixed across studies
Mostafa et al., 2026	UK	Adults with poor sleep health (21 studies), n=1,222	Systematic review/meta-analysis/meta-regression	CBT-I, sleep hygiene, sleep-extension trials	Behavioural sleep interventions significantly reduced blood pressure and heart rate; no significant change in HRV

3.3 Distribution of included studies by population

Of the 26 included studies, six examined healthy or general adult populations [9-14], three examined occupational populations of nurses and shift workers [15-17], ten examined clinical populations [18,20,21,22,25,26,27,28,29,30], and seven were systematic reviews, meta-analyses, or genetically informed (Mendelian randomization) studies providing higher-level evidence synthesis [19, 23, 24, 31, 32, 33, 34] (Figure 3).

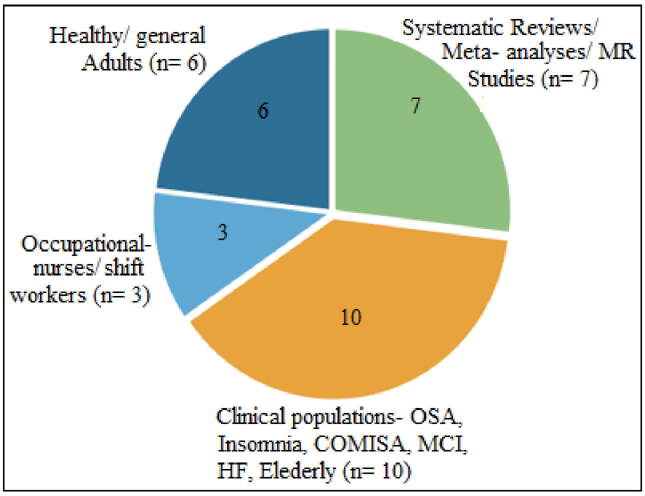

Figure 3: Distribution of the 26 included studies by population type

3.4 Sleep quality and HRV in healthy and general adult populations

Evidence in healthy and general adult populations was mixed. In a cross-sectional study of 260 Iranian hospital staff, poorer subjective sleep quality and lower sleep efficiency were associated with a lower rise in SDANN, although sleep duration itself was not associated with HRV parameters [9]. By contrast, a study of 74 Chinese medical students found no significant correlation between 24-hour HRV indices and PSQI global scores, although bedtime SDNN correlated with the sleep-disturbance subscale specifically [10]. A pilot intervention study using mobile HRV-biofeedback training reported improvements in subjective sleep quality and increases in SDNN, total power, and low-frequency power after four weeks, although objective (actigraphy) sleep metrics did not change [11]. A 14-day observational study using daily short-term HRV recordings in 41 healthy adults found that higher daily RMSSD was associated with better self-reported sleep quality, alongside lower fatigue and stress [12]. Finally, two related analyses of the Midlife in the United States (MIDUS) Biomarker cohort (n=391) found no significant main effect of sleep quality or duration alone on HRV, but observed a significant interaction whereby short sleep combined with inadequate exercise was associated with lower HF-HRV and RMSSD [13,14].

3.5 Sleep quality and HRV in occupational populations (nurses and shift workers)

Findings among nurses and shift workers were more consistently in the direction of reduced HRV with poorer sleep quality. Among 393 Taiwanese female nurses, a significant negative correlation was observed between PSQI global scores and total power, low-frequency power, and the LF/HF ratio, with shift-working nurses showing significantly lower total power and low-frequency HRV than non-shift nurses [15]. A small ambulatory pilot study of nurses found that day-shift nurses exhibited a circadian rise in HRV coherence during their sleep period that was absent among night-shift nurses, suggesting altered autonomic regulation associated with night work [16]. Similarly, a study of 38 Indian female nurses found that night-shift nurses had significantly higher PSQI scores and significantly lower SDNN, total power, and high-frequency power, alongside a higher LF/HF ratio, compared to morning-shift nurses [17].

3.6 Obstructive sleep apnea

Evidence in OSA populations was the most consistent across included studies, generally supporting an association between OSA severity and impaired HRV. In 62 adults referred for polysomnography, RMSSD was negatively correlated with body mass index and neck circumference, and HRV parameters were associated with markers of OSA severity via the apnea–hypopnea index [18]. A systematic review of cross-sectional studies concluded that adults with OSA demonstrate diminished vagal tone and heightened sympathetic responsiveness relative to controls [19]. Nocturnal HRV parameters have also been shown to help predict severe obstructive sleep-disordered breathing [20], and HRV profiles have been found to differ between "sleepy" and "non-sleepy" OSA patients drawn from the Sydney Sleep Biobank, with implications for differential cardiovascular risk stratification [21].

3.7 Insomnia

Evidence regarding insomnia was more equivocal than for OSA. Daytime HRV was found to be lower (indicating higher physiological arousal) among adults reporting insomnia symptoms, correlating with symptom severity [22]. However, a critical review of 22 studies concluded that the evidence for HRV impairment in insomnia was inconsistent and characterised by a high risk of bias, such that HRV impairment- while widely assumed- was not conclusively supported by the existing empirical literature at the time of that review [23]. More recently, a two-sample Mendelian randomization study using genome-wide association data found that genetically predicted lower HRV (across pvRSA/HF, RMSSD, and SDNN traits) was causally associated with increased insomnia risk, while the reverse pathway (insomnia causally affecting HRV) was not statistically significant- suggesting HRV changes may, in at least some cases, precede rather than follow insomnia [24]. Consistent with a bidirectional and comorbidity-sensitive relationship, a study directly comparing insomnia with and without comorbid OSA found that insomnia, regardless of OSA severity, was associated with higher heart rate and lower SDNN specifically during the sleep-onset period, with the comorbid group showing the lowest HRV of all groups studied [26].

3.8 Other clinical and comorbid populations

Several studies examined other clinical or comorbid groups. Using 5,335 electrocardiograms from the Sleep Heart Health Study, one large secondary analysis found that comorbid insomnia and sleep apnea (COMISA) was associated with a distinct autonomic signature- reduced parasympathetic activity while awake combined with heightened sympathetic activation during sleep- that differed from isolated OSA or insomnia [25]. A multicentre study of patients with unexplained syncope and/or OSA similarly identified distinct nocturnal HRV patterns across syncope, OSA, and comorbid subgroups relative to controls [27]. In older adults with mild cognitive impairment, resting HRV and PSQI scores were largely unrelated, with only one significant correlation identified (between high-frequency power and sleep efficiency), suggesting that in this population, subjective sleep-quality measures and objective HRV may not track together [28]. In a cross-sectional actigraphy study of older heart failure patients, poor sleep efficiency was associated with higher NT-proBNP and worse frailty and functional outcomes, although HRV itself was not directly measured in this study [29]. Finally, in a case-control study of 355 elderly patients, clinically defined sleep disorders independently predicted reduced pNN50 and SDANN, with anxiety and depression contributing additional, superimposed reductions in HRV [30].

3.9 Evidence from systematic reviews, meta-analyses, and genetically informed studies

Higher-level evidence syntheses generally supported a modest but real association between poorer sleep and reduced

HRV, while highlighting substantial heterogeneity. A meta-analysis of 11 randomised controlled trials of experimental sleep deprivation (549 participants) found a significant reduction in RMSSD and increases in low-frequency power and the LF/HF ratio following sleep deprivation, although changes in SDNN and high-frequency power did not reach statistical significance [31]. A meta-analysis of 17 studies (921 participants) comparing insomnia disorder patients with controls found a small but statistically significant pooled reduction in HRV (R-equivalent = 0.19, 95% CI 0.12–0.25) with no significant heterogeneity across studies [32]. A systematic review of sleep-related HRV in the context of depression and anxiety symptoms found associations between HRV and both sleep and mood symptoms, although findings were mixed across the reviewed studies [33]. By contrast, a meta-analysis and meta-regression of 21 studies (1,222 participants) evaluating behavioural sleep interventions (cognitive behavioural therapy for insomnia, sleep hygiene, and sleep extension) found that while these interventions significantly reduced blood pressure and heart rate, they did not produce a significant change in HRV [34] — suggesting that improving sleep behaviourally may not be sufficient, on its own and over the timeframes studied, to reverse HRV alterations.

3.10 Overall direction of association across included studies

Across all 26 included studies, 13 reported reduced or impaired HRV associated with poorer sleep quality, 9 reported mixed or parameter-dependent findings, 2 reported no significant association, and 2 used a different design or outcome that was not directly comparable to a simple association (Figure 2). The strongest and most consistent evidence for reduced HRV with poorer sleep was observed in clinical populations with OSA, insomnia, and comorbid sleep disorders; evidence in healthy adults, occupational populations, and via behavioural intervention trials was more heterogeneous.

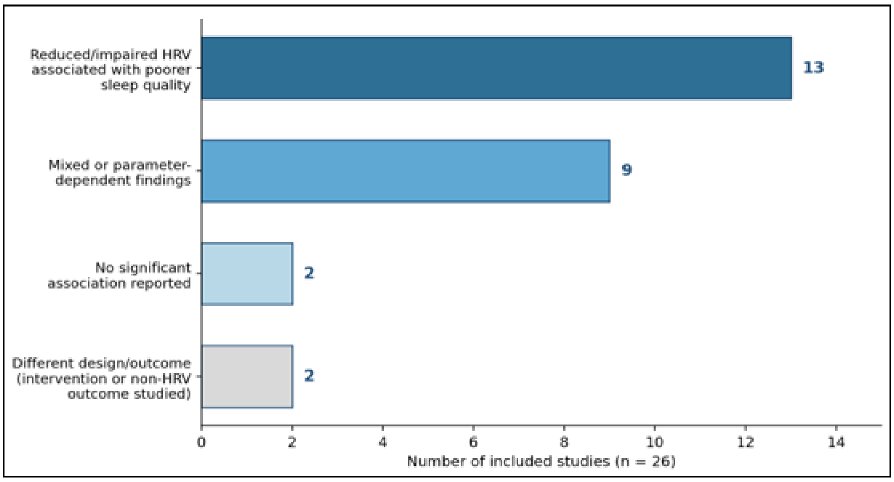

Figure 2: Direction of the reported sleep quality–HRV association across the 26 included studies

4. Discussion

This narative review synthesised evidence from 26 studies examining the association between sleep quality and HRV in adults. The overall pattern suggests a real, but population-dependent, association: reduced parasympathetic (vagal) activity and altered sympathovagal balance were most consistently observed in clinical populations with diagnosed sleep disorders- particularly OSA, insomnia, and their comorbid presentation (COMISA) — while evidence in healthy adults and occupational populations was considerably more mixed.

Several plausible mechanisms may explain the stronger clinical-population signal. In OSA, recurrent apnoeic events produce intermittent hypoxia and repeated arousals that directly perturb autonomic control during sleep itself, providing a clear physiological pathway to altered HRV [18-21]. In insomnia, the proposed mechanism is hyperarousal- a sustained elevation of sympathetic tone that could plausibly suppress HRV both during the day and at sleep onset [22,26]; however, the critical review by Dodds et al. found this evidence to be inconsistent and prone to bias [23], and the more recent Mendelian randomization analysis suggests the causal arrow may run from HRV to insomnia rather than the reverse [24], which would have implications for how this relationship should be interpreted and targeted therapeutically.

In healthy and occupational populations, associations were less consistent, and several studies found that sleep quality alone was not directly associated with HRV, with effects instead emerging only in interaction with other factors such as physical activity [13,14] or in the context of subjective sleep sub-domains rather than global scores [10]. This may reflect genuinely smaller effect sizes in non-clinical populations, but could also reflect measurement issues: studies using subjective PSQI scores versus objective actigraphy or polysomnography did not always agree, and the sole study with both subjective and objective sleep measures alongside HRV-biofeedback found subjective but not objective improvement following the intervention [11]. This dissociation between subjective and objective sleep-quality measures echoes the broader sleep literature and suggests that reviews and future primary studies should report both types of measure where possible rather than relying on a single modality.

The systematic reviews and meta-analyses included in this rapid review provide a useful sense-check on the primary-study findings. The insomnia meta-analysis found a real but small pooled effect [32], the sleep-deprivation meta-analysis found effects specific to certain HRV parameters (RMSSD, LF, LF/HF) rather than a uniform HRV reduction [31], and- notably- the largest identified meta-analysis of behavioural sleep interventions found that despite significant improvements in blood pressure and heart rate, HRV itself did not significantly improve [34]. Taken together, this pattern suggests that HRV may be a relatively insensitive or slow-changing outcome for evaluating sleep interventions, at least over the timeframes studied, even where more classical cardiovascular markers do improve.

From a clinical perspective, these findings support continued use of HRV as a marker of autonomic dysfunction in patients with diagnosed sleep-disordered breathing and comorbid insomnia-OSA presentations, where the signal is most consistent and the underlying mechanism best understood. In contrast, using HRV as a general marker of “poor sleep” in otherwise healthy populations, or as a primary outcome for evaluating behavioural sleep interventions, appears less well supported by the current evidence and should be interpreted cautiously.

5. Strengths and Limitations of This Rapid Review

This review has several strengths: it applied an explicit, pre-specified PECO framework; it deliberately included both subjective and objective sleep-quality measures and both healthy and clinical populations to provide a broad initial map of the evidence; and it synthesised evidence at multiple levels, from primary cross-sectional studies through to meta-analyses and a genetically informed causal analysis.

Several important limitations, characteristic of the narrative review format, should be considered. Study selection and data extraction were conducted through a structured but single-reviewer process rather than independent dual-reviewer screening with consensus, which is standard in a full systematic review; this increases the risk of selection or extraction error. Third, no formal risk-of-bias or GRADE certainty assessment was conducted for individual studies, and the heterogeneity of populations, sleep-quality measures, and HRV metrics precluded meta-analytic pooling within this review- findings are therefore synthesised narratively and should be interpreted as an initial evidence map rather than a definitive, pooled estimate of effect. Fourth, several included records are conference abstracts or secondary analyses of existing cohorts (e.g., MIDUS, Sleep Heart Health Study, Sydney Sleep Biobank) rather than independent primary studies, which may inflate the apparent number of independent data points. Finally, publication bias cannot be excluded, and grey literature was not systematically searched given the narrative review timeframe.

Given these limitations, this narrative review should be regarded as a timely, structured evidence map suitable for informing next steps- including protocol registration and full systematic review with dual-reviewer screening, formal risk-of-bias assessment, and, where feasible, meta-analysis of comparable HRV parameters- rather than as a substitute for a full systematic review.

6. Conclusion

Across 26 studies, poorer sleep quality was most consistently associated with reduced or altered HRV in clinical populations with obstructive sleep apnea, insomnia, and comorbid sleep disorders, supporting a plausible autonomic mechanism linking disordered sleep to cardiovascular risk in these groups. In healthy adults and occupational populations, the association was weaker and less consistent, and appeared to depend on how sleep quality and HRV were each measured. Behavioural sleep interventions improved blood pressure and heart rate but not HRV itself in the largest available meta-analysis, raising questions about HRV's sensitivity as an intervention outcome. Future research should prioritise standardised, combined subjective–objective sleep assessment alongside validated HRV protocols, larger longitudinal and interventional designs.

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