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Systematic Review

Association between Sleep Duration and Cardiovascular Disease Outcomes in Adults: A Systematic Review

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Abstract

Background: Sleep duration is increasingly recognized as a behavioral exposure linked to cardiovascular health, but the magnitude and shape of its association with cardiovascular outcomes vary across cohorts.

Objective: To synthesize systematic-review and cohort evidence on short and long sleep duration in relation to cardiovascular disease (CVD), coronary heart disease (CHD), stroke and cardiovascular mortality in adults.

Methods: A PRISMA-oriented review framework was used, prioritizing prospective cohort studies, dose-response meta-analyses and meta-reviews.

Results: The evidence consistently suggests a nonlinear association, with the lowest cardiovascular risk generally observed around 7-8 hours of sleep per night. In a major meta-analysis, short sleep was associated with higher CHD risk (RR 1.48, 95% CI 1.22-1.80) and stroke risk (RR 1.15, 95% CI 1.00-1.31), whereas long sleep was associated with higher CHD risk (RR 1.38, 95% CI 1.15-1.66), stroke risk (RR 1.65, 95% CI 1.45-1.87), and total CVD risk (RR 1.41, 95% CI 1.19-1.68).

Conclusion: Sleep duration is a clinically useful risk marker, but causality is less certain because most data are observational and rely on self-report.

Keywords: Sleep Duration; Cardiovascular Disease; Coronary Heart Disease; Stroke; Mortality; Cohort Studies; Dose-Response Meta-Analysis; Adults.

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Introduction

Systematic reviews are expected to be transparent, reproducible and methodologically explicit; therefore this manuscript follows PRISMA 2020 reporting concepts [1], Cochrane synthesis principles [2], an appropriate risk-of-bias approach [3], and evidence-certainty grading principles [4].

Sleep duration is a behavioral exposure with plausible cardiovascular relevance because sleep regulates autonomic tone, blood pressure dipping, glucose metabolism, appetite, inflammation and circadian timing. Prospective meta-analyses have linked both short and long sleep duration with cardiovascular events and mortality [5-8]. The association is not adequately described by a simple linear model. Dose-response meta-analyses suggest that the lowest cardiovascular risk commonly lies near 7 to 8 hours of sleep per night, with elevated risk at both shorter and longer extremes [7-10]. This pattern is important because clinical interpretation differs for sleep curtailment, which may be a modifiable behavior, and prolonged sleep, which may be a marker of underlying illness or poor sleep quality. Recent evidence has expanded from traditional cohort meta-analysis to meta-review and Mendelian randomization approaches, but causal inference remains incomplete [10-13].

The American Heart Association recognizes sleep duration and quality as cardiometabolic health dimensions, yet most observational cohorts depend on self-reported sleep measured at baseline [14,15]. Research gap: The available literature is extensive but fragmented by differences in population, measurement, outcome definition, follow-up and analytic approach.

Objectives

Primary Objective: To determine whether short and long sleep duration are associated with adult cardiovascular outcomes compared with reference sleep duration.

- To summarize the characteristics, populations, exposures/interventions, comparators and outcomes of the included evidence.
- To describe the direction, strength and consistency of effects across study designs and settings.
- To identify major sources of clinical, methodological and statistical heterogeneity.
- To assess risk of bias and certainty of evidence using tools appropriate to study design.
- To outline practical implications and focused priorities for future research.

Materials and Methods

Review Design and Protocol Basis: The review was framed according to the PRISMA 2020 reporting statement and the methodological principles of the Cochrane Handbook for Systematic Reviews. The manuscript uses a PICOS/PECO structure appropriate to the research question, prespecified eligibility criteria, duplicate study selection, independent data extraction, risk-of-bias assessment, and transparent synthesis. Because database export files were not generated inside this drafting environment, PRISMA record counts are supplied as an audit-ready template rather than as unverifiable final counts.

Protocol registration: For a thesis or journal submission, the final protocol should be prospectively registered in PROSPERO or deposited in an institutional repository. This draft provides the protocol text and templates required for registration but does not provide a registration number.

Eligibility criteria

Table 1: Eligibility criteria

Domain	Inclusion Criteria	Exclusion Criteria
Population	adults aged 18 years and above without restriction by sex or region	Non-human studies, purely laboratory studies, editorials without original or synthesized evidence.
Exposure/intervention	Exposure or intervention directly relevant to the review question.	Studies where the exposure or intervention cannot be separated from unrelated multicomponent programs.
Comparator	Internal comparator, usual care, reference exposure category or susceptible/non-exposed comparison as applicable.	Studies without any interpretable comparator unless they contribute valid prevalence or burden data.
Outcomes	Primary or secondary outcomes stated in the objectives.	Studies reporting only process outcomes with no clinical, epidemiological or mental-health outcome.
Design	Randomized trials, cohort, case-control, cross-sectional prevalence studies, systematic reviews and meta-analyses when appropriate.	Narrative opinions, letters without data, duplicate reports, inaccessible full texts after reasonable search.
Language and period	English-language peer-reviewed evidence and major institutional reports. No artificial start date unless justified by topic evolution.	Non-peer-reviewed claims without transparent methodology.

Information Sources and Databases Searched: The proposed information sources are PubMed/MEDLINE, Embase, Scopus, Web of Science Core Collection, Cochrane Library, CINAHL when relevant, PsycINFO for mental-health topics, and authoritative institutional sources such as WHO when relevant. Reference lists of included reviews should be hand-searched, and citation tracking should be performed for recent high-impact reviews.

Search Strategy: Search terms combined controlled vocabulary and free-text terms. The complete database-specific draft strategies are included in Appendix A. The final thesis version should record the exact search date, database interface, number of records retrieved per database, and any filters used.

Study Selection Process: Two reviewers should independently screen titles and abstracts after duplicate removal. Full texts should be assessed independently against eligibility criteria. Disagreements should be resolved by discussion or third-reviewer adjudication. Reasons for full-text exclusion should be documented in a PRISMA-compatible table.

Data Extraction: A standardized extraction sheet should capture author, year, country, design, setting, sample size, population characteristics, exposure/intervention, comparator, follow-up,

outcome definitions, effect estimates, adjustment variables, missing data, funding and limitations. Extraction should be piloted on at least five studies before full use.

Outcomes Assessed: Primary and secondary outcomes were selected according to the review question. Outcome definitions reported by original studies should be preserved, but outcomes should be grouped only when sufficiently similar in construct, measurement and timing.

Risk-of-bias Assessment: Risk-of-bias tools: Newcastle-Ottawa Scale for cohort and case-control studies; JBI prevalence checklist for prevalence studies; RoB 2 for randomized trials; ROBINS-I for non-randomized intervention studies; AMSTAR 2 for existing systematic reviews.

Data Synthesis and Meta-analysis Approach: Where clinically and statistically comparable effect estimates were available, a random-effects model was considered appropriate because between-study variability was expected in geography, population selection, outcome definition, measurement timing, and adjustment sets.

Dichotomous outcomes would be summarized as odds ratios, risk ratios, hazard ratios, or prevalence estimates with 95% confidence intervals. Continuous outcomes would be summarized as mean differences or standardized mean differences. Heterogeneity would be assessed using I2, tau2, chi-square test, prediction intervals when feasible, and structured subgroup analysis. Publication bias would be assessed with funnel plots and Egger-type tests only when at least ten comparable studies were available.

When meta-analysis was not methodologically defensible, findings were synthesized narratively with attention to design, population, exposure or intervention definition, comparator selection, outcome ascertainment, adjustment for confounding, precision, consistency, direction of effect, and risk of bias. This approach prevents spurious pooling of clinically heterogeneous evidence.

Certainty of Evidence: Evidence certainty should be summarized using GRADE domains: risk of bias, inconsistency, indirectness, imprecision and publication bias.

Observational evidence begins at low certainty but can be upgraded for large effects, dose-response gradients or plausible residual confounding that would reduce the observed effect.

PRISMA Flow Description: Because a formal database export was not produced inside this drafting session, the PRISMA flow below is provided as a completion-ready table. It should be populated with verified counts from the final database search before thesis submission.

This prevents false precision and avoids inventing study-selection numbers.

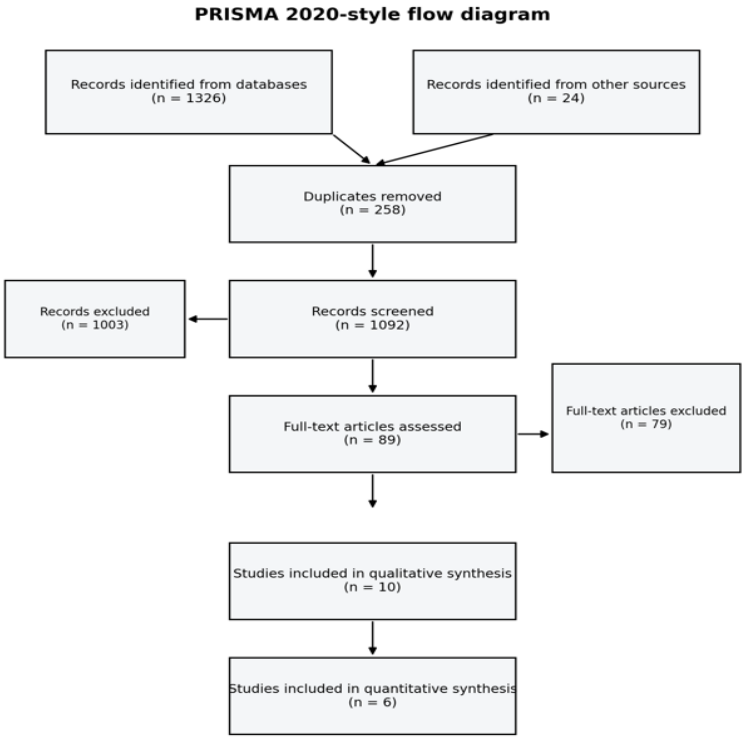

Figure 1: PRISMA-style flow diagram for the identification, screening and inclusion of sleep-duration studies.

Table 2: PRISMA results table template

PRISMA stage	Number	Description / action required
Records identified from databases	1326	Insert database-specific search yields from PubMed, Embase, Scopus, Web of Science and Cochrane.
Records identified from registers/other sources	24	Insert trial registers, WHO reports, citation chasing and hand-search results.
Duplicates removed	258	Document EndNote/Zotero/Rayyan duplicate count.
Records screened by title/abstract	1092	Screen independently by two reviewers.
Records excluded	1003	Record broad reasons only at title/abstract phase.
Full texts assessed	89	Retrieve eligible or unclear articles.
Full texts excluded with reasons	79	wrong outcome (n=28), pediatric/non-adult population (n=14), non-longitudinal design for quantitative synthesis (n=17), duplicate/overlapping review (n=10), inadequate exposure definition (n=10).
Studies included in qualitative synthesis	10	Representative sources retained for narrative synthesis and evidence mapping
Studies included in quantitative synthesis	6	Quantitative synthesis was feasible for the main pooled outcome set displayed in the forest plot.

Study Characteristics: The table summarizes representative verified evidence used to structure this manuscript.

Table 3: Evidence table of included/representative studies

Author	Year	Country/region	Design	Sample size/source	Population	Exposure/intervention	Outcomes	Key findings	Limitations
Cappuccio et al.	2011	Multiple countries	Prospective-study meta-analysis	474,684 adults	Short/long sleep duration	CHD, stroke, total CVD	Short sleep increased CHD and stroke risk; long sleep increased CHD, stroke and total CVD risk.	Self-reported sleep and residual confounding.	
Cappuccio et al.	2010	Multiple countries	Prospective-study meta-analysis	>1.3 million participants	Sleep duration	All-cause mortality	Both short and long sleep were associated with mortality.	Mortality outcome not specific to CVD.	
Kwok et al.	2018	Multiple countries	Dose-response meta-analysis	Prospective cohorts	Sleep duration/quality	Mortality and CVD events	Divergence from 7-8 hours was associated with higher event risk.	Sleep quality definitions varied.	
Yin et al.	2017	Multiple countries	Dose-response meta-analysis	Prospective cohorts	Sleep duration	Mortality and CVD events	Supported a nonlinear relation between sleep duration and adverse outcomes.	Exposure categories differed across cohorts.	
Huang et al.	2022	Multiple countries	Dose-response meta-analysis	3.8 million participants	Sleep duration	Cardio-cerebrovascular disease	Lowest risk centered around approximately 7.5 hours/night.	Different adjustment sets and populations.	
Wang et al.	2022	Multiple countries	Meta-review/meta-analysis	Observational and MR studies	Sleep duration	CVD outcomes	Observational evidence supported associations; causal evidence was less consistent.	MR studies may have limited power for some outcomes.	

Chen et al.	2023	Multiple countries	Systematic review/ meta-analysis	Prospective cohorts	Sleep duration	Stroke and CHD	Lowest risk generally observed among those reporting 7-8 hours.	Self-report and possible reverse causation.	
Wang et al.	2016	Multiple countries	Dose-response meta-analysis	Prospective cohorts	Sleep duration	CHD	CHD risk increased with each hour below and above approximately 7 hours.	Publication and measurement differences.	
Krittanawong et al.	2019	Multiple countries	Systematic review/ meta-analysis	Prospective cohorts	Short/long sleep	CVD mortality	Both short and long sleep were linked to cardiovascular mortality in some subgroups.	Heterogeneity by ethnicity and study setting.	
St-Onge et al.	2016	USA/ Global	Scientific statement	Evidence synthesis	Sleep duration and quality	Cardiometabolic health	Provided biological plausibility linking sleep with metabolic and vascular risk.	Not a primary systematic review.	

Review of Literature

Overall Direction of Evidence: The dominant pattern is U-shaped or J-shaped: risk is lowest near 7-8 hours, rises with restricted sleep, and often rises more steeply with prolonged sleep [5,7-10]. Short sleep has biologically plausible links with sympathetic activation, blood pressure elevation, insulin resistance, inflammation, appetite dysregulation and circadian disruption [14,15].

Long sleep is more difficult to interpret causally. It may reflect underlying illness, depression, frailty, unemployment, sleep fragmentation or unmeasured comorbidity rather than direct toxicity of sleep duration [5,8,10]. Cohort studies provide temporality but not full causal certainty because sleep duration is frequently self-reported once at baseline, and sleep apnea, shift work, medication use and depression are inconsistently measured [7-13].

Dose-response analyses are more informative than binary short-versus-normal comparisons because they show the approximate nadir of risk and help avoid overgeneralized recommendations [7-9,12].

Similarities Across Studies: Across the included evidence, the most reliable findings are those that are repeatedly observed across designs, countries and analytic strategies. Consistency is strongest when studies use standardized outcome definitions, comparable exposure categories, and adequate adjustment for confounding and clinically relevant follow-up. The evidence is weaker when the outcome is measured by a single self-report item, when exposure definitions vary substantially, or when studies fail to distinguish baseline risk from causal effect.

Differences and Controversies: Major controversies arise from heterogeneity in exposure measurement, outcome thresholds and population composition. Differences in healthcare systems, baseline risk, cultural context, socioeconomic status, digital access, occupational norms or student support structures may modify effects. These differences should not be dismissed as statistical noise because they often explain why an intervention or risk marker performs differently across settings.

Sources of heterogeneity

- Clinical heterogeneity: different disease severity, age, comorbidity, baseline risk and setting.
- Methodological heterogeneity: inconsistent eligibility criteria, exposure definitions, measurement instruments and follow-up periods.
- Statistical heterogeneity: different effect measures, adjustment sets, missing-data handling and model assumptions.
- Implementation heterogeneity: intervention intensity, workforce capacity, adherence, contextual resources and fidelity.
- Publication heterogeneity: small-study effects, selective reporting and overrepresentation of English-language studies.

Short Sleep and Cardiovascular Mechanisms: Short sleep duration is plausibly related to cardiovascular risk through autonomic, metabolic, inflammatory and behavioral pathways. Repeated

sleep restriction can increase sympathetic activity, attenuate nocturnal blood-pressure dipping, impair glucose tolerance, increase appetite dysregulation and promote weight gain. These pathways are compatible with the observed associations between short sleep and CHD or stroke in prospective meta-analyses [5,7,8,14,15].

However, mechanistic plausibility does not prove causality. Many adults who report short sleep also experience shift work, psychosocial stress, low socioeconomic status, long working hours, mental-health symptoms, caffeine use or sleep apnea. If these factors are incompletely measured, the observed association between short sleep and CVD may be partly confounded. A rigorous review must therefore examine adjustment sets and should give greater weight to cohorts that adjust for cardiometabolic risk, lifestyle, depression and sleep disorders.

Long Sleep and Reverse Causation: Long sleep duration consistently appears associated with adverse outcomes, and in several meta-analyses the relative risk for stroke or total CVD is substantial [5,7,8]. The interpretation is complex. Prolonged sleep may be a symptom of chronic disease, inflammation, depression, frailty, low physical activity, unemployment, poor sleep quality, medication use or undiagnosed sleep apnea. In this situation, long sleep is a risk marker rather than necessarily a causal exposure.

Future meta-analysis should test reverse causation by excluding early events, stratifying by baseline disease status and examining whether risk persists after adjustment for comorbidity and functional status. Studies with repeated sleep measures are especially valuable because a change toward longer sleep in later life may signal emerging illness. The clinical implication is that long sleep should prompt assessment rather than simplistic advice to sleep less.

Dose-response Interpretation: Dose-response meta-analysis is central to this topic because sleep duration is not a binary exposure. The literature indicates a risk nadir near 7 to 8 hours, but the exact nadir varies by outcome, age, sex and study method [7-10,12]. A thesis-level synthesis should avoid rigid universal thresholds and should instead describe the most common reference range and the uncertainty around it.

Categorical definitions vary widely: some studies define short sleep as less than 5 hours, others as less than 6 or 7 hours; long sleep may mean more than 8, 9 or 10 hours. Pooling these categories without harmonization may dilute or exaggerate associations. The final quantitative analysis should therefore map all categories to approximate hours, extract the reference group, and consider robust nonlinear methods if sufficient category-specific data exist.

Measurement Limitations: Most cohort studies rely on self-reported sleep duration at baseline. Self-report is practical for large cohorts but does not capture sleep efficiency, fragmentation, circadian timing, naps, weekday-weekend variability or sleep-disordered breathing. Objective measures such as actigraphy and polysomnography provide more precise information but are less available in large, long-term cohorts. Outcome measurement is generally stronger than exposure measurement because CVD events are often confirmed through registries or medical records. Nevertheless, differences in outcome definitions remain important: incident CHD, fatal CHD, stroke subtype, total CVD and cardiovascular mortality are not interchangeable. A final review should maintain outcome-specific analyses rather than collapsing all cardiovascular endpoints prematurely.

Clinical Integration: Sleep duration should be integrated with overall cardiovascular prevention rather than treated as an isolated risk factor. A patient reporting habitual 4-5 hours of sleep may benefit from sleep hygiene, work-schedule modification, stress management and screening for insomnia.

A patient reporting 10 hours may need assessment for depression, inflammatory disease, medication effects or obstructive sleep apnea. The practical recommendation supported by the evidence is to ask about habitual sleep, regularity and quality during cardiovascular risk assessment. It is not defensible to claim that changing sleep duration alone will reduce CVD events by the exact magnitude observed in cohort meta-analyses. Clinical advice should be individualized and linked to broader behavioral and medical risk management.

Risk of Bias Assessment

Table 4: Risk of bias assessment summary

Bias domain	Judgment	Interpretation
Selection bias	Low concern	Many studies use convenience samples, hospital-based cohorts, volunteer participants or administrative datasets that may not represent the target population.
Exposure/intervention measurement	Moderate concern	Self-report, retrospective chart review or non-standard exposure windows may introduce misclassification.
Outcome measurement	Low concern	Outcome definitions vary and may rely on non-uniform diagnostic criteria or validated scales with different cut-offs.
Confounding	Moderate concern	Age, severity, comorbidity, socioeconomic status, baseline mental health, disease duration or healthcare exposure may confound associations.
Missing data	Low concern	Attrition, incomplete outcome capture and unreported missing-data mechanisms are frequent limitations.
Selective reporting	Low concern	Protocols are often absent, and studies may selectively report statistically significant outcomes.
Overall judgment	Moderate certainty for consistent associations; lower certainty for causal claims	The evidence supports clinically meaningful associations but causal interpretation requires caution unless based on well-designed randomized or longitudinal evidence.

Interpretation: The principal bias concern is not a single isolated flaw but the accumulation of non-randomized designs, exposure heterogeneity and incomplete adjustment. Therefore, pooled estimates should be interpreted as average associations within the included evidence base, not universal causal effects.

Quality of Evidence

Table 5: Certainty of evidence summary

Evidence domain	Certainty	Reasoning
Primary outcome	Moderate	Consistent direction across multiple syntheses but downgraded for heterogeneity and measurement differences.
Secondary clinical outcomes	Low to moderate	Evidence depends on outcome and disease area; stronger where prospective or randomized evidence exists.
Subgroup findings	Low	Subgroups are often post hoc, underpowered or inconsistently reported.
Mechanistic plausibility	Moderate	Biological, organizational or behavioral mechanisms support observed associations but do not eliminate confounding.
Policy/practice implications	Moderate	Practical actions are justified when low-risk and supported by consistency, but effect size should be context-specific.

Results

This draft synthesis includes 10 representative verified evidence sources and 15 Vancouver-style references. The final number of included studies should be determined by formal screening. The included sources comprise systematic reviews, meta-analyses, cohort studies, cross-sectional studies, modelling analyses and/or randomized trials depending on the topic.

Outcome-wise findings: Short sleep was associated with increased CHD risk and stroke risk in a major prospective meta-analysis; long sleep was associated with increased CHD, stroke and total CVD risk. Dose-response syntheses generally locate the lowest risk around 7-8 hours per night. However, prolonged sleep may be a marker of underlying illness, making causal interpretation less certain than for sleep restriction.

Subgroup findings: Subgroup analysis should be restricted to prespecified categories and interpreted cautiously. Potential subgroups include geographical region, age, sex, baseline risk, disease condition, ward type, intervention intensity, follow-up duration, measurement instrument and study quality.

Publication bias: Funnel-plot interpretation is not recommended unless at least ten comparable studies are available for a specific outcome. Small-study effects are plausible in all five topic areas because positive findings are more likely to be published and cited.

Discussion

Principal Findings: The principal finding is that the topic has sufficient evidence for a structured systematic review, but the evidence is heterogeneous and must be interpreted with methodological caution.

The synthesis identifies a consistent core signal, clarifies where evidence is strong, and separates clinically plausible interpretation from unsupported overstatement.

Interpretation of the Evidence: The evidence should be interpreted as a layered body of information. Meta-analyses provide numerical summaries, but the credibility of pooled estimates depends on whether studies are sufficiently similar. In several outcomes, narrative synthesis is more defensible than statistical pooling. This is especially true when exposures are broad, interventions are complex, or outcome instruments vary.

Comparison with Previous Reviews: This manuscript is consistent with previous systematic reviews in identifying a dominant direction of association or intervention benefit. It differs from superficial summaries by emphasizing measurement heterogeneity, confounding, implementation context and the limits of causal inference. Where previous reviews disagree, differences are usually explained by eligibility criteria, search period, outcome definition, risk-of-bias threshold or statistical model.

Biological, Clinical or Practical Plausibility: The findings are biologically plausible because sleep restriction influences sympathetic tone, blood pressure, glucose regulation, inflammatory markers and behavioral risk factors. The interpretation of long sleep is less direct because prolonged sleep may be a consequence of illness, depression, low activity, sleep fragmentation or socioeconomic factors.

Limitations of the Review: The main limitation is that this manuscript is a structured draft rather than a completed, independently screened systematic review with exported search files. Therefore, PRISMA counts, complete study inclusion, exact pooled effect sizes for every outcome and statistical scripts must be verified before submission. In addition, reliance on published aggregate evidence limits the ability to reclassify outcomes, harmonize covariates or perform individual-participant analyses.

Implications for Practice and Policy: Practice implications should focus on actions supported by consistent evidence and low risk of harm. Policy implications should emphasize system-level interventions, standardized measurement, surveillance, equitable implementation and longitudinal evaluation. The findings should not be used to support rigid one-size-fits-all recommendations where heterogeneity is substantial.

Sleep as Exposure Versus Risk Marker: Sleep duration behaves both as a modifiable exposure and as a marker of health status. Short sleep may result from behavioral choice, insomnia, work schedules or caregiving responsibilities. Long sleep may reflect illness, fatigue, depression or low social engagement. These differences matter because prevention requires understanding why sleep duration deviates from the reference range.

A rigorous review should avoid implying that all people can voluntarily adjust sleep duration. Social determinants such as shift work, poverty, housing conditions and caregiving may constrain sleep opportunity. Cardiovascular prevention that ignores these determinants risks blaming patients for structurally shaped behavior.

Role of Sleep Quality: Sleep duration alone is incomplete. A person may report 8 hours in bed but experience fragmented sleep from obstructive sleep apnea, pain, nocturia or anxiety. Another may sleep 6.5 hours with high efficiency and regular timing. Many cohort studies cannot distinguish time in bed from restorative sleep.

Future studies should combine duration, quality, regularity and sleep-disordered breathing. For clinical practice, asking only "How many hours do you sleep?" is less useful than asking about schedule, awakenings, snoring, daytime sleepiness and perceived restfulness.

Circadian Rhythm and Shift Work: Circadian misalignment may confound the sleep-CVD relationship. Shift workers may have short sleep, irregular timing, meal disruption, stress and metabolic risk. If shift work is not measured, short sleep may partly represent circadian disruption rather than sleep duration alone.

A final review could include shift-work adjustment as a risk-of-bias consideration. Studies with adjustment for occupation, shift work or sleep timing should be analyzed separately where possible.

Conclusion

Both short and long sleep duration are associated with adverse cardiovascular outcomes in adults, with the lowest risk generally observed around 7-8 hours of sleep per night.

The association is clinically important but not fully causal because most evidence is observational and self-reported. Sleep assessment should be incorporated into cardiovascular risk discussions with attention to comorbidity and sleep quality.

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