



Association between Psychological Stress and Short-Term Heart Rate Variability in Healthy Indian Adults: A Systematic Review

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(Received: 25 November 2025 Revised: 27 December 2025 Accepted: 11 January 2026)

KEYWORDS

Stress, Heart Rate Variability, Autonomic Nervous System, Acute Stress, Adults, India

ABSTRACT:

Background: Psychological stress alters autonomic balance, and heart rate variability (HRV) offers a non-invasive index of this regulation; however, India-specific evidence linking stress with HRV in healthy adults remains fragmented.

Objective: To synthesize India-based evidence (2015–2025) on the association between psychological stress – psychometric and task-evoked – and short-term heart rate variability (HRV) in healthy adults.

Methods: We conducted a PRISMA-guided systematic review of PubMed/MEDLINE, Scopus, and Embase (2015–2025; English; India-only) for observational or acute-stress studies in healthy adults reporting associations between stress (psychometric or task-evoked) and short-term HRV indices. Two reviewers independently screened, extracted data, and appraised risk of bias (NIH tool); findings were synthesized narratively without meta-analysis due to methodological heterogeneity.

Results: Among 462 records (PubMed 124, Embase 156, Scopus 182), 300 unique citations were screened; 56 full texts were assessed and 6 studies (total N = 550) met inclusion. Four student-laboratory cohorts, one young-adult acute-stress study, and one community cohort uniformly used short-term, supine HRV with standard indices. Resting psychometric stress–HRV findings were heterogeneous: one dataset showed strong inverse correlations with vagal HRV (e.g., SDNN ρ -0.715; RMSSD ρ -0.662; HF ρ -0.681; total power ρ -0.733) and parallel haemodynamic elevations, whereas others reported non-significant between-group differences despite directional shifts toward higher LF/LFnu and LF/HF and lower HFnu. Acute stress consistently evoked autonomic reactivity: RMSSD during stress was lower in males than females (21.0±10.9 vs 33.7±15.3 ms; $p=0.01$), and heart rate rose (80→100 bpm; $p<0.001$) with QT shortening (353→325 ms; $p<0.001$) and QTc prolongation (397→418 ms; $p<0.001$). Overall stress prevalence ranged from 56% (community women) to 97.2% (students). Risk of bias was mostly fair; methodological heterogeneity precluded meta-analysis.

Conclusion: HRV reliably captures acute stress-related vagal withdrawal in healthy Indian adults, but cross-sectional links between perceived stress and resting HRV are heterogeneous and method-sensitive, underscoring the need for standardized protocols and broader, better-controlled cohorts.

Introduction

Heart rate variability (HRV) – the beat-to-beat variation in sinus rhythm – indexes dynamic autonomic regulation of the cardiovascular system and has become a widely used non-invasive biomarker in psychophysiology and

clinical research.(1) The landmark Task Force statement codified HRV concepts, measurement standards, and frequency bands, establishing the foundation for contemporary short-term and long-term analyses.(2) HRV reflects the integrated output of central autonomic networks; higher vagally mediated indices (e.g.,



RMSSD, HF power) are generally interpreted as greater parasympathetic flexibility, whereas relative shifts toward LF and higher LF/HF are often taken to reflect sympathetic predominance or reduced vagal restraint.(3, 4) Neurovisceral integration models link prefrontal- limbic circuits to cardiac vagal control, providing a mechanistic account of how psychological states – including stress – modulate HRV.(5)

A substantial literature indicates that acute psychological stress reduces vagally mediated HRV, consistent with parasympathetic withdrawal and sympathetic activation. Meta-analytic and review evidence shows stress-related decreases in HF power and time-domain vagal indices, supporting HRV as an objective marker of stress reactivity.(6, 7) Beyond laboratory paradigms, lower HRV has prognostic value; reduced HRV predicts incident cardiovascular events in populations without known cardiovascular disease and is associated with higher all-cause and cardiac mortality in pooled cohort analyses, underscoring its clinical relevance.(8, 9) At the same time, HRV is sensitive to numerous biological and behavioural influences – including age, sex, physical activity, sleep, respiration, stimulants, and comorbid conditions – necessitating careful control or reporting to ensure valid inference.(10)

The Indian context provides a pertinent setting to examine stress–HRV associations in healthy adults. Emerging syntheses document a high burden of psychological distress in student populations – particularly medical undergraduates – where pooled estimates suggest that roughly half experience significant stress, with marked heterogeneity by sex and institution.(11) This epidemiologic backdrop, together with India’s demographic diversity and varying cardiometabolic risk profiles, motivates an appraisal of whether and how perceived or experimentally evoked stress maps onto short-term HRV in ostensibly healthy cohorts. Establishing the direction and consistency of these associations is important for two reasons. First, it can clarify whether HRV offers actionable insight into stress burden outside clinical populations, potentially informing screening or wellness programmes in educational and occupational settings.(6) Second, it can help bridge psychophysiological markers with longer-term cardiovascular risk, given the established links between lower HRV and adverse outcomes, and thereby

inform population-level prevention strategies in LMIC contexts where resource-efficient risk stratification is valuable.(9)

Against this background, we synthesise Indian studies published since 2015 that evaluate associations between stress and short-term HRV in healthy adults, spanning psychometric and acute-stress paradigms, while attending to methodological features known to influence HRV measurement and interpretation.

Materials and Methods

This review was conducted and reported in accordance with PRISMA 2020 guidance for systematic reviews. A protocol was developed a priori and followed throughout; it was not registered with PROSPERO. Institutional Ethics Committee (IEC) approval (Reference number VMKVMC&H/IEC/24/185 dated 10/07/2024) was obtained prior to the conduct of this work. As the study synthesized published, de-identified data, individual informed consent was not required.

Review question and eligibility criteria: We examined whether, among healthy Indian adults, psychological or physiological stress is associated with HRV indices.

- a. **Population:** Adults (≥ 18 years) residing in India and described as healthy community-dwelling participants or student volunteers, without acute illness or chronic cardiometabolic/psychiatric conditions unless analyzed as a clearly separable healthy subgroup.
- b. **Exposure:** Stress assessed via validated psychometric instruments (e.g., Perceived Stress Scale [PSS], Medical Student Stressor Questionnaire [MSSQ], other validated stress/anxiety scales) or via acute/real-life stress paradigms (e.g., mental arithmetic, Stroop, examination stress) with HRV recorded at rest and/or during/after the stressor. Interventional studies were excluded unless a pre-intervention stress–HRV association was explicitly analyzed and extractable.
- c. **Outcomes:** Time-domain (e.g., SDNN, RMSSD, pNN50), frequency-domain (e.g., LF, HF, LF/HF in absolute or normalized units), and non-linear HRV metrics (e.g., Poincaré SD1/SD2, entropy) derived from ECG or



validated heart-rate devices. Eligible studies reported an association (correlation or regression) between stress and ≥ 1 HRV metric, or within-subject HRV change under an acute stressor with stress ratings.

- d. **Study design and limits:** Observational cross-sectional, cohort, or quasi-experimental stress-reactivity studies in healthy adults. Inclusion limits were English language, India-only samples or India-specific strata, and publication dates from 1 January 2015 to 5 November 2025 (Asia/Kolkata). Exclusions were pediatric samples (<18 years) unless adult strata were separable; patient cohorts; non-Indian samples; case reports, editorials, reviews; conference abstracts without full data; non-English publications; and studies reporting HRV without a stress measure or stress contrast.

Information sources and search strategy: We searched PubMed/MEDLINE, Scopus (Elsevier), and Embase (Elsevier) through 5 November 2025 (Asia/Kolkata). Search strategies combined controlled vocabulary and free-text terms for stress, HRV, and India, with adult/English/date limits applied at retrieval. Database-specific syntax (e.g., MeSH in PubMed; Emtree in Embase; TITLE-ABS-KEY in Scopus) was used without restrictive study-design filters to preserve sensitivity. Reference lists of included articles were hand-searched for additional studies. Grey literature beyond indexed journals was not systematically searched.

Study selection: Records were exported to EndNote 20.2.1 for de-duplication and screened in two stages (titles/abstracts, then full text) by two reviewers independently using prespecified eligibility criteria. Disagreements were resolved by consensus or third-reviewer adjudication. Reasons for full-text exclusion were documented. The selection process was summarized in a PRISMA 2020 flow diagram.

Data extraction: Two reviewers independently extracted data using a standardized, pilot-tested form with cross-checks for accuracy. We captured study characteristics (first author, year, setting/state, design, sampling frame), population details (sample size, age, sex, inclusion/exclusion, health status verification), stress measurement (instrument, scoring, domains, timing; or stress-paradigm type/duration/timing), HRV acquisition

(device/lead configuration, duration, posture, breathing conditions, time of day/fasting, preprocessing/artifact handling, indices and transformations), statistical methods (effect measure, covariates/confounders, handling of non-normality), results (direction/magnitude of association, precision), and funding/conflicts. Corresponding authors were to be contacted up to two times if critical items were missing or unclear.

Risk of bias assessment: Risk of bias was appraised using the NIH Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies, applied independently by two reviewers with consensus ratings of Good/Fair/Poor. Domains included clarity of research question, defined population and participation rate, sample-size justification, validity and reliability of exposure (stress) and outcome (HRV) measurements – including HRV recording conditions and artifact handling – blinding (where applicable), control for key confounders (e.g., age, sex, BMI, physical activity, smoking, menstrual phase, medications, sleep), and appropriateness of statistical analyses.

Effect measures: The primary effect measure was the association between stress and HRV captured as correlation coefficients (Pearson r or Spearman ρ) or regression coefficients (β) for continuous stress scores versus HRV metrics. For acute stress paradigms, within-subject HRV change from rest to stress and correlations between stress ratings and HRV reactivity were extracted when available. When multiple HRV indices/time points were reported, all prespecified outcomes were extracted; if authors identified a primary HRV endpoint, this was flagged.

Synthesis methods: Meta-analysis was not planned due to expected heterogeneity in populations (students vs community adults), stress instruments (PSS vs MSSQ vs task stress), HRV protocols (recording duration/posture/breathing), and reporting formats. We conducted a structured narrative synthesis.

Results

The database search yielded 462 records (PubMed, 124; Embase, 156; Scopus, 182). After automated de-duplication in EndNote (162 records), 300 unique records underwent title and abstract screening, of which 244 were excluded. Fifty-six full-text articles were assessed for eligibility; fifty were excluded for the



following primary reasons: non-Indian population ($n = 9$), not healthy adults or patient cohorts ($n = 8$), no stress–HRV association reported ($n = 14$), interventional studies without extractable baseline association ($n = 7$), conference abstracts only ($n = 5$), outside the 2015–2025 window ($n = 4$), and non-English language ($n = 3$). Six studies were included in the qualitative synthesis; the PRISMA 2020 flow diagram is provided in the Figure 1.

Across the six India-based observational studies (2016–2025; total $N = 550$), four recruited first-year MBBS students in physiology laboratory settings in Tamil Nadu, Kerala, and Karnataka; one enrolled healthy young adults for an acute mental stress protocol in Kerala; and one community-based study evaluated middle-aged women across menopausal stages in Gujarat. Stress exposure was measured psychometrically using the Medical Student Stressor Questionnaire (MSSQ; three studies) or the Perceived Stress Scale (PSS; one study) and physiologically using acute or real-life stress paradigms (mental arithmetic or composite task batteries; two studies). HRV was uniformly assessed as short-term supine recordings. Devices and analysis pipelines included BIOPAC with AcqKnowledge, ADInstruments PowerLab, HRV Soft (AIIMS), Kubios HRV (version 3.3.1), and Variowin HR, with generally standardized recording conditions (supine posture, five-minute epochs) and, in several studies, pre-test restrictions such as abstinence from caffeine or alcohol and follicular-phase scheduling for women.

Among studies using psychometric stress measures at rest, findings were heterogeneous. One laboratory-based student study (Punita et al.(12) 2016) demonstrated a strong inverse association between cumulative stress and vagal HRV; higher stress correlated with lower SDNN ($\rho = -0.715$), RMSSD ($\rho = -0.662$), HF power ($\rho = -0.681$), and total power ($\rho = -0.733$), along with a modest increase in LF/HF ($\rho = +0.253$). HRV medians for SDNN, RMSSD, and HF declined stepwise from moderate to high to severe stress with highly significant omnibus tests, and cumulative stress correlated positively with systolic and diastolic blood pressure, heart rate, pulse pressure, and rate-pressure product. By contrast, two student cohorts using MSSQ (Thomas et al.(13) 2021; Vageesh et al.(14) 2022) did not detect statistically significant differences in time- or frequency-domain HRV across stress grades or domains, although

the directions of change generally favoured higher LF, LFnu, and LF/HF and lower HFnu in stressed groups. The community study of middle-aged women (Solanki et al.(15) 2025) found no significant correlations between PSS scores and HRV parameters within pre-, peri-, or postmenopausal strata; patterns suggested modest modulation by diabetes and/or hypertension (for example, higher LF/HF in postmenopausal women with comorbidity), but these differences were not consistent across indices. Overall, approximately 56% of women had any PSS-defined stress, while in the student cohort of Thomas et al.(13) the prevalence of any MSSQ-defined stress was 97.2%, suggesting possible ceiling effects that may have limited between-group separation.

The two acute-stress studies provided convergent evidence of autonomic reactivity. In the serial-sevens task (Zachariah et al.(16) 2019), there were no sex differences in resting HRV; however, during stress, males exhibited significantly lower RMSSD than females (21.02 ± 10.87 vs 33.73 ± 15.33 ms; $p = 0.01$), indicating greater vagal withdrawal. In a male-only task-battery study (Pagadala et al.(17) 2019), acute stress produced marked tachycardia and repolarization changes, with heart rate increasing from 79.98 ± 13.07 to 100.37 ± 16.49 bpm ($p < 0.001$), QT shortening from 353.08 ± 21.57 to 324.57 ± 25.69 ms ($p < 0.001$), and QTc prolongation from 396.78 ± 21.18 to 418.31 ± 16.93 ms ($p < 0.001$); PR and QRS intervals were unchanged. HRV indices were not tabulated in this study, but the ECG interval behaviour and heart-rate responses are physiologically coherent with sympathetic activation and vagal withdrawal under acute stress.

Sex-stratified results, where reported, suggested that males generally showed higher resting vagal HRV and lower LF/HF than females in student samples, and a greater degree of vagal withdrawal during acute stress. In higher stress strata, males also demonstrated higher SDNN, RMSSD, HF power, and total power than females, while sex differences in cardiovascular variables diminished. The community sample of women did not permit sex comparisons but underscored the influence of comorbidity on blood pressure and, to a lesser extent, on HRV patterns.

All studies relied on short-term HRV with standard time- and frequency-domain metrics; one also reported non-linear Poincaré indices. Despite broadly similar



recording postures and epoch lengths, methodological non-uniformity remained in breathing control, artifact handling, reporting units (absolute versus normalized power), and software, which constrains direct quantitative pooling and likely contributes to between-study variability. Risk of bias was appraised with the NIH Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies. Overall quality was mostly rated as fair. Typical strengths included clearly stated research questions, standardized laboratory acquisition of HRV, and appropriate basic statistical testing. The principal limitations were the reliance on convenience student samples, limited sample-size justification and statistical power, incomplete control for key confounders relevant to HRV (including physical activity, sleep, menstrual cycle beyond scheduling, medications, caffeine or nicotine exposure, and respiration), variability in HRV processing and software with inconsistent documentation of artifact correction, and the cross-sectional nature of most psychometric studies.

Discussion

This systematic review synthesised evidence on the association between stress and heart-rate variability in healthy Indian adults identified through a comprehensive search across PubMed, Embase, and Scopus, and screened according to PRISMA 2020 standards. The search yielded 462 unique records, of which six studies met the inclusion criteria following dual-review screening and full-text assessment, providing a focused yet heterogeneous dataset spanning student, young-adult laboratory, and community settings from 2016 to 2025. Across the four psychometric studies evaluating resting HRV, results ranged from strong, graded inverse associations to null findings, underscoring how sampling frame, stress burden, and analytic choices can shape observed effects. The clearest signal arose in a physiology-laboratory study of first-year medical students, where higher cumulative stress was associated with lower vagally mediated HRV (SDNN, RMSSD) and HF power, together with a modest rise in LF/HF; these associations co-occurred with higher systolic and diastolic blood pressure, heart rate, pulse pressure, and rate-pressure product, presenting a coherent picture of autonomic imbalance with haemodynamic correlates.(12) By contrast, two other student cohorts using the MSSQ found no statistically significant HRV

differences across stress strata or domains, although the directional pattern – higher LF, LFnu, and LF/HF with lower HFnu – was congruent with sympathetic predominance under stress.(13, 14) A community-based study of middle-aged women similarly reported no correlation between PSS scores and HRV within pre-, peri-, or postmenopausal strata, while suggesting that cardiometabolic comorbidity (diabetes and/or hypertension) may shift LF/HF upward in post menopause.(15)

Several plausible explanations reconcile these discrepant findings. First, the magnitude and distribution of perceived stress differed substantially across samples. In one student cohort, 97.2% screened positive for at least mild stress, a ceiling effect that compresses variance and reduces statistical separability across MSSQ grades.(13) In contrast, the community sample showed 56% prevalence of any PSS-defined stress with stage-specific variations, potentially introducing greater heterogeneity in both exposure and unmeasured confounders.(15) In a meta-analysis of 37 human studies by Kim et al. (2018), stress exposure was consistently linked to reduced parasympathetic HRV, most often reflected as a decrease in HF power with a reciprocal increase in LF power, supporting HRV as an objective indicator of psychological stress.(6) The findings also corroborate with that reported by Thielmann et al. (2022) and Tiwari et al. (2021).(3, 18) Second, psychometric instruments capture different constructs – academic stressors in MSSQ versus general perceived stress in PSS – so construct alignment with autonomic outputs may vary across populations and contexts.(13-15) The findings were reinforced by Hachenberger et al. (2023), Immanuel et al. (2023) and Schneider et al. (2025).(19-21) Third, HRV acquisition and processing pipelines differed (BIOPAC/AcqKnowledge, PowerLab, HRV Soft, Kubios, Variowin), as did artefact handling and the balance of absolute versus normalised spectral indices; such methodological non-uniformity can attenuate between-study comparability and dilute pooled inferences even when posture and epoch length are standardised. Finally, confounding by respiration, sleep, physical activity, stimulant exposure, and menstrual phase – addressed only partially through pre-test restrictions or follicular-phase scheduling – could bias associations toward the null or inflate variance, as noted by Brar et al. (2015) and Damoun et al. (2024).(10, 22)



Evidence from acute/real-life stress paradigms was more consistent and physiologically persuasive. In a serial-sevens task with short-term HRV acquisition, males exhibited significantly lower RMSSD than females during stress despite no sex difference at rest, indicating greater vagal withdrawal in men under cognitive load.(16) In a male-only task battery emulating evaluative or performance stressors, heart rate rose markedly and repolarisation indices shifted – QT shortened and QTc prolonged – without changes in PR or QRS, a pattern that aligns with sympathetic activation and reduced vagal restraint during acute stress.(17) Although HRV indices per se were not tabulated in the latter, the combination of tachycardia with QT/QTc dynamics is mechanistically concordant with autonomic reactivity and complements the HRV-based sex-difference findings from the former study.(16, 17)

Sex-stratified analyses, where available, suggest a nuanced pattern. In resting states within student cohorts, males tended to exhibit higher vagally mediated HRV and lower LF/HF than females, whereas under acute stress males showed greater reductions in RMSSD, implying larger vagal withdrawal.(12, 16) These findings corroborate with Li et al. (2009) and Zeng et al. (2023).(23, 24) In higher stress strata, males displayed higher SDNN, RMSSD, HF power, and total power than females with attenuation of sex differences in blood pressure and heart rate, suggesting that autonomic flexibility may differ by sex and stress load in young adult populations.(12) The community cohort, restricted to women, did not permit sex comparisons but highlighted that coexisting diabetes or hypertension can raise blood pressure consistently and modestly alter HRV patterns across menopausal stages, emphasising the importance of clinical stratification in community samples.(15) In Martinez & Okoshi (2018), patients with type 2 diabetes plus hypertension showed shorter mean RR intervals (801.1 vs 871.5 ms) and lower Shannon entropy (3.0 vs 3.2) than comparison diabetics, indicating reduced HRV and aggravated autonomic dysfunction; similarly, Bassi et al. (2018) reiterates that lower HRV reflects reduced parasympathetic and relatively increased sympathetic tone, a pattern associated with worse cardiovascular prognosis.(25, 26)

An additional insight from this corpus concerns construct validity at the interface of stress measurement and HRV

endpoints. The strongest resting associations were observed when stress was summarised as a cumulative burden and analysed against multiple vagal and global HRV metrics with robust, non-parametric statistics and complementary regression, while also demonstrating parallel cardiovascular changes.(12) Where stress was categorised into coarse strata with highly skewed distributions, between-group contrasts were less likely to reach significance, particularly in modest samples, even when directional trends suggested sympathetic shift.(13, 14) Meanwhile, acute paradigms that directly elicit cognitive or evaluative stress produced reproducible autonomic signatures within individuals, reinforcing the sensitivity of HRV to state-level stressors in controlled laboratory conditions.(16, 17)

These findings have practical implications for future Indian HRV–stress research. First, sampling frames should move beyond convenience student cohorts to include community and occupational populations spanning wider age, socioeconomic, and behavioural spectra, thereby improving generalisability and stress variance. Second, studies should prespecify and standardise HRV acquisition (posture, epoch length, breathing control), artefact detection and correction, and the choice and units of spectral indices, ideally reporting both absolute and normalised values; convergence on a common processing platform (e.g., Kubios) with transparent parameter settings would facilitate cross-study synthesis. Third, stress measurement should pair validated psychometrics with ecologically valid stressors or ambulatory assessments where feasible, while incorporating core confounders such as habitual activity, sleep, stimulant intake, menstrual phase/stage, and medications into design or analysis. Fourth, sex- and stage-specific analyses – particularly across the menopausal transition and in relation to cardiometabolic comorbidity – are warranted to delineate moderators of autonomic stress responses suggested by the present evidence.

Limitations

This review has several limitations that temper inference. The evidence base was small (six studies; total N=550) and skewed toward convenience samples of first-year medical students, limiting generalizability beyond young, academically stressed populations and under-representing community and occupational cohorts.



Designs were predominantly cross-sectional, precluding causal interpretation and making results vulnerable to residual confounding. Methodological heterogeneity was substantial across stress constructs (MSSQ vs PSS vs acute tasks), HRV acquisition and processing (devices, software, artefact handling, breathing control, epoch length, absolute vs normalized spectral units), and reporting practices, which prevented meta-analysis and likely contributed to between-study inconsistency.

Conclusion

In this systematic review of six India-based studies (N=550), acute cognitive or evaluative stress reliably elicited autonomic reactivity – manifested as vagal withdrawal and, when reported, concordant ECG interval changes – supporting the physiological sensitivity of HRV to state stress in healthy adults. By contrast, cross-sectional associations between perceived/academic stress and resting HRV were heterogeneous; one student-laboratory study demonstrated large, graded inverse relationships between stress and vagal HRV with parallel haemodynamic elevations, while three others (including a community cohort) reported null effects despite directional trends toward sympathetic predominance. Taken together, these findings suggest that HRV is a robust marker of acute stress responses but a context-dependent and method-sensitive indicator of resting stress burden.

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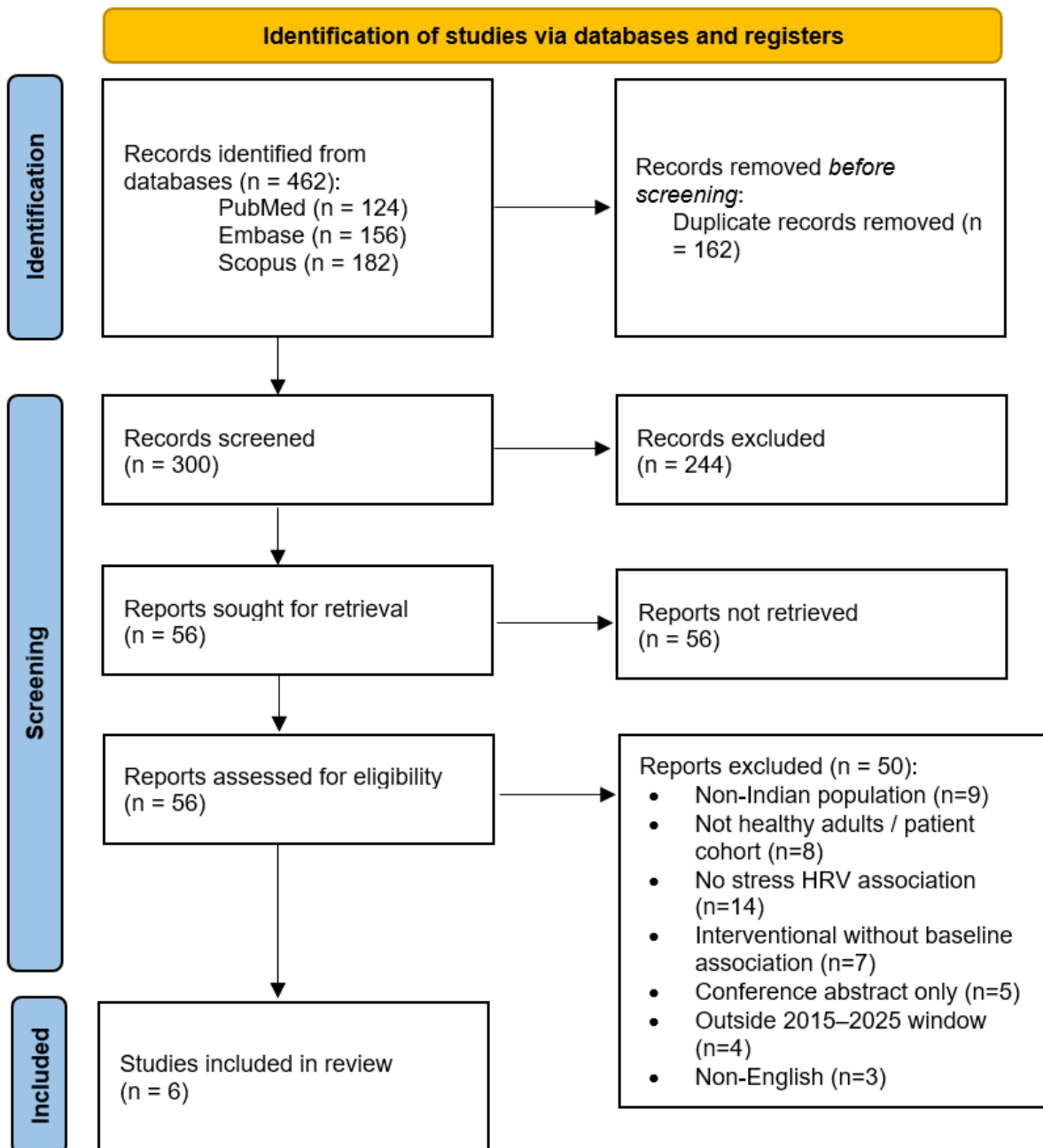


Figure 1: PRISMA flow diagram of study selection for the systematic review on stress–HRV association in healthy Indian adults (2015–2025)



Table 1: Characteristics of included studies and key results – association between stress and heart rate variability in healthy Indian adults (2015–2025)

First author (year)	Design	Setting	Population	Sample size (N)	Sex (F/M, n)	Age (years)	Stress instrument	Stress level distribution (%)	ECG/HRV device & lead	Recording protocol	HRV indices (time)	HRV indices (frequency)	Primary association (stress ↔ HRV; effect size)
Punita (2016)	Cross-sectional analytical	Tamil Nadu	Healthy first-year MBBS students	150	78 / 72	18–24	Medical Student Stressor Questionnaire (MSSQ; 6 domains)	Mild 1 (0.7), Moderate 33 (22.0), High 94 (62.7), Severe 22 (14.7)	ECG Lead II, BIOP AC MP100; AcqKnowledge 3.8.2; 500 Hz; band-pass 2–40 Hz	Fasting; no nicotine/alcohol 48 h; no caffeine 24 h; thermoneutral room (23°C, 25–35% RH); after voiding; follicular phase for females	Mean RR, SD, RMSS, D, NN50, pNN50	LF (0.04 – 0.15 Hz), HF (0.15 – 0.40 Hz), Total Power, LFnu, HFnu, LF/HF	Higher stress associated with lower SDNN ($\rho=-0.715$), RMSSD ($\rho=-0.662$), HF power ($\rho=-0.681$), Total power ($\rho=-0.733$); higher LF/HF ($\rho=+0.253$); all $p<0.001$ except LF/HF model $F=0.084$
Zachariah (2019)	Cross-sectional (acute mental stress reactivity)	Kerala	Healthy young adults (normal BMI), 17–20 years	30	18 / 12	17–20	Acute mental arithmetic (serial 7s from 200, 3 min)	NA (reactivity paradigm)	ECG limb lead II, BPL ECG; A/D converter (National Instruments); HRV Soft	Supine; baseline 5-min ECG then 3-min stress; inclusion: normal BMI; short-	RMSSD, SD, ANF	LFnu, HFnu, LF/HF	During stress: males had lower RMSSD vs females (21.02±10.87 vs 33.73±15.33; $P=0.01$) indicating lower vagal



									1.1 (AIIMS)	term HRV; standard Task Force methods			activity; other indices NS
Pagadala (2019)	Analytical with in-subject (acute mental stress reactivity)	Karnataka	Healthy first-year MBBS students (male only)	50	0 / 50	18–22 (mean 18.27±1.28)	Acute task battery (public speaking, interviews, seminars, mental arithmetic, viva); STAI administered	NA (reactivity paradigm)	Philips C3i ECG; 25 mm/s; supine	Resting ECG day 1; immediate ECG during acute mental stress; standard clinical acquisition	Not reported beyond heart rate	Not reported	Heart rate increased with stress (100.37±16.49 vs 79.98±13.07; p<0.001) indicating vagal withdrawal/sympathetic activation
Thomas (2021)	Cross-sectional analytical	Kerala	Healthy first-year MBBS students	70	35 / 35	18–23	Medical Students Stress Questionnaire (MSSQ; Sathidevi 2009)	No stress 2 (2.9), Mild–Moderate 30 (42.9), Severe 38 (54.3)	Lead II ECG; Physio pac system	5-min HRV after 15-min supine rest; standard lab conditions	Mean RR, Mean HR, SD NN, RMSSD, pNN50	Total Power, LF, HF, LFnu, HFnu, LF/HF	Between-group differences for HRV indices across stress strata not statistically significant
Vageesh	Cross-sectional	Karnataka	Healthy first-year	58	Not rep	18–25	Medical Students	Predominantly	ADInstruments	Single HRV session	Mean NN	LF (ms ²), HF	No significant association



(2022)	onal analytical		MBBS students with normal BMI (18.5–22.9 kg/m ²)		orted		nt Stress or Questionnaire (MSSQ; 6 domains)	mild–moderate across domains; few high/severe per domain	Power Lab, limb lead ECG; time & frequency HRV	; limb leads; HRV analysed once per participant	, Media NN, SD NN, RMSSD, pNN50	(ms ²), LF/HF	n between MSSQ domain grades and HRV indices (all p>0.05)
Solanki (2025)	Cross-sectional analytical	Gujarat (community-based recruitment (5 areas, 2 cities))	Middle-aged women, 40–55 years (pre-, peri-, postmenopausal)	192	192 / 0	40–55	Perceived Stress Scale (PSS)	Across groups: Grade 0/1/2 = 44% / 53% / 3% (no intergroup difference)	Lead II ECG; 5-min short-term HRV; 500 Hz sampling; Vario win HR software	Supine; 10-min rest; assessment 9:00–12:00; isolated room; refrain from coffee/tea/cola before test	RR interval, SD NN, RMSSD, SD NN, pNN50	VLF, LF, HF (ms ²); LFnu, HFnu; LF/HF; max LF, max HF; total power (reported narratively)	No significant correlation between PSS score and HRV parameters in pre-, peri-, or postmenopausal groups; correlations largely null



Table 1 (Continued): Characteristics of included studies and key results – association between stress and heart rate variability in healthy Indian adults (2015–2025)

First author (year)	Group comparisons / Reactivity (HRV)	Cardiovascular measures (association/reactivity)	Cardiovascular group comparisons / Reactivity	Gender comparison (overall)	High-stress subgroup (gender)	Prevalence of stress (overall)	Stress classification rule / cut-points
Punita (2016)	Medians declined from Moderate→High→Severe for SDNN (50.9→30.9→21.7 ms), RMSSD (59.5→33.6→24.6 ms), HF (1146→363→153 ms ²); all Kruskal–Wallis p<0.001	Cumulative stress positively correlated with SBP (r=0.494), DBP (r=0.468), HR (r=0.367), PP (r=0.368), RPP (r=0.515); all p<0.001	Means rose from Moderate→High→Severe for SBP (108.5→115.6→126.5 mmHg) and HR (72.9→81.4→90.6 bpm); ANOVA p<0.001	Males higher vagal HRV (e.g., RMSSD median 46.8 vs 28.4 ms) and lower LF/HF (0.691 vs 0.956), all p≤0.026; females had higher SBP/DBP/HR (all p≤0.002)	Within high stress, males showed higher SDNN/RMSSD/HF and total power; p=0.025–0.048; cardio differences attenuated		
Zachariah (2019)	No sex differences at rest (time/freq domains NS). During stress: RMSSD lower in males; frequency-domain indices (LFnu, HFnu, LF/HF) NS	Not reported	Not reported	At rest: NS differences by sex. During stress: RMSSD significantly lower in males (P=0.01)			
Pagadala (2019)	No HRV indices tabulated; see ECG intervals	SBP 114.49±9.80, DBP 70.82±6.72 (baseline descriptive); primary reactivity reported on ECG intervals and HR	HR: 79.98±13.07 → 100.37±16.49 (p<0.001); P duration: 93.71±11.37 → 100.24±15.61 (p<0.02); PR: 140.69±22.79 → 137.71±18.78 (p=0.48, NS); QRS: 87.47±6.87 → 85.73±8.20	Not applicable (male-only sample)			



			(p=0.26, NS); QT: 353.08±21.57 → 324.57±25.69 (p<0.001); QTc: 396.78±21.18 → 418.31±16.93 (p<0.001)				
Thomas (2021)	LF, LFnu, LF/HF higher and HFnu lower in stressed groups vs no-stress (direction only; NS)	Not reported beyond Mean HR	NS	Females: majority severe stress (65.7%); Males: majority mild-moderate (51.4%)		97.2% stressed (any level)	<54 no stress; 55-81 mild-moderate; 82-108 severe
Vageesh (2022)	Domain-wise Kruskal-Wallis across stress grades showed NS results (ARS/IRS/TLRS/SRS/D RS/GARS)	Not reported	Not reported	Not reported		Mild-moderate stress common in all six domains	MSSQ domain mean: 0-1 mild; 1.01-2 moderate; 2.01-3 high; 3.01-4 severe
Solanki (2025)	NDNH group: perimenopause showed lower HR and higher LF power & mode vs others; DH group: LF/HF increased from pre- to postmenopause; some subgroup differences (e.g., HF power higher peri-)	SBP/DBP compared across NDNH vs DH within each stage; DH had higher BP; HR differences minimal	Between NDNH vs DH: SBP/DBP consistently higher in DH (all p<0.001 across stages)	Not applicable (women only)		56% any PSS stress (grades 1/2)	PSS grades 0,1,2 as per article (quantitative thresholds not specified)