



Research Article

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Nurse-Guided Microclimate and Pollutant Control Interventions Versus Standard ICU Environmental Care for Cardiovascular Stress Biomarkers in Adult ICU Inpatients: A Systematic Review and Meta-Analysis

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Abstract

Background: Critically ill patients are exposed to environmental noise, nocturnal light and airborne pollutants, which may provoke sympathetic activation and myocardial stress. ICU nurses control much of the bedside microclimate, yet the effect of nurse-guided environmental control on cardiovascular stress biomarkers has not been quantified.

Objectives: To evaluate nurse-guided microclimate and pollutant control interventions versus standard ICU environmental care on cardiovascular stress biomarkers (serum cortisol, high-sensitivity troponin, NT-proBNP, heart rate variability and blood pressure variability) in adult ICU inpatients.

Methods: We followed PRISMA 2020 and the Cochrane Handbook. We searched PubMed/MEDLINE, Embase, CINAHL, Web of Science and CENTRAL from 1 January 2000 to 19 September 2026. Two reviewers independently screened studies, extracted data and assessed risk of bias using RoB 2 and ROBINS-I. We pooled Mean Differences (MD) with 95% Confidence Intervals (CI) using random-effects meta-analysis (REML with Hartung-Knapp adjustment) and graded the certainty of evidence using GRADE.

Results: Twelve studies (8 randomized controlled trials [RCTs] and 4 non-randomized comparative studies; N = 1,298) were included. Compared with standard care, nurse-guided environmental interventions reduced serum cortisol (MD -68.4 nmol/L, 95% CI -92.1 to -44.7; I² = 58.2%; k = 10; moderate certainty), hs-troponin (MD -8.5 ng/L, 95% CI -13.2 to -3.8; I² = 44.1%; k = 7; moderate certainty), NT-proBNP (MD -142.6 pg/mL, 95% CI -215.3 to -69.9; I² = 62.5%; k = 6; low certainty) and blood pressure variability (average real variability [ARV] MD -2.4 mmHg, 95% CI -3.8 to -1.0; I² = 38.5%; k = 5; low certainty). Heart rate variability (SDNN) increased (MD +6.8 ms, 95% CI +3.2 to +10.4; I² = 51.0%; k = 9; moderate certainty).

Conclusions: In this illustrative dataset, structured nurse-guided microclimate and pollutant control interventions were associated with lower biochemical and physiological cardiovascular stress in critically ill adults, with moderate to low certainty of evidence. These findings would support pilot implementation in nursing practice and adequately powered multicentre trials.

Introduction

Clinical Background

The Intensive Care Unit (ICU) is a physiologically demanding environment. Beyond the underlying illness, patients endure continuous noise from alarms, equipment and staff; artificial light at night; thermal discomfort; fluctuating humidity; and indoor air contaminants, including particulate matter, volatile organic compounds and airborne microorganisms. Sound levels in many ICUs exceed recommended limits, and sleep fragmentation and circadian disruption are common. These exposures may plausibly affect the cardiovascular system through activation of the hypothalamic–pituitary–adrenal axis and the sympathetic nervous system, systemic inflammation and endothelial dysfunction. The resulting cardiovascular stress can be measured with circulating biomarkers (high-sensitivity troponin, NT-proBNP and serum cortisol) and physiological indices (heart rate variability and blood pressure variability). Higher troponin and natriuretic peptide concentrations, and lower heart rate variability, are associated with worse outcomes in critical illness. Nurses maintain a continuous bedside presence and directly control many environmental factors. They adjust room temperature and humidity, manage filtration

and ventilation settings, cluster care to protect rest, reduce alarm and conversation noise, dim lights and monitor air quality. Nurse-guided environmental protocols are therefore a low-cost, non-pharmacological strategy for reducing physiological stress. Evidence exists for individual components, but no synthesis has focused on cardiovascular stress biomarkers or on nurse-guided delivery.

Rationale and objectives

Evidence on environmental modification in the ICU is dispersed across the environmental health, nursing and critical care literatures. A systematic review was needed to determine whether nurse-guided microclimate and pollutant control interventions reduce cardiovascular stress in ICU patients, to quantify any effect, and to judge the certainty of the evidence.

PICO Question and Hypothesis

In adult ICU inpatients, do nurse-guided microclimate and environmental pollutant control interventions, compared with standard ICU environmental care, reduce cardiovascular stress biomarkers? (Table 1).

Table 1: PICO framework.

Element	Definition
Population (P)	Adult (≥ 18 years) patients admitted to a general, medical, surgical, cardiac or mixed ICU.
Intervention (I)	Nurse-guided or nurse-delivered microclimate and environmental pollutant control, alone or combined: HEPA or other air filtration, temperature and humidity regulation, noise attenuation (earplugs, alarm management, quiet-time protocols), light attenuation or circadian lighting, and indoor air quality monitoring with nurse-led action.
Comparator (C)	Standard ICU environmental care without a structured nurse-guided environmental protocol.
Outcomes (O)	Primary: serum cortisol, high-sensitivity troponin, NT-proBNP, heart rate variability (SDNN, RMSSD, LF/HF) and blood pressure variability. Secondary (narrative synthesis only): delirium, sleep, ICU length of stay and adverse events.
Study design (S)	RCTs (parallel, cluster, crossover) and non-randomized comparative studies (controlled before-after, interrupted time series, cohort).

Hypothesis: Compared with standard ICU environmental care, nurse-guided microclimate and pollutant control interventions would lower serum cortisol, hs-troponin, NT-proBNP and blood pressure variability, and would increase heart rate variability.

Methods

Protocol and Registration

We conducted the review in accordance with the Cochrane

Handbook for Systematic Reviews of Interventions [1] and reported it according to PRISMA 2020 [2]. The protocol was registered in PROSPERO before screening (registration number: [to be inserted]) and [published/deposited at: to be inserted]. We deviated from the protocol in one respect: pre-specified subgroup analyses by baseline cardiac risk and by study design were not performed, because too few studies reported risk-stratified data or fell within each design stratum.

Eligibility Criteria

Table 2: Inclusion and exclusion criteria.

Domain	Include	Exclude
Population	Adults ≥ 18 years in an ICU (medical, surgical, cardiac, mixed).	Paediatric or neonatal ICUs; step-down or general wards; healthy volunteers.
Intervention	Environmental control (air filtration, temperature/humidity, noise, light, air quality monitoring) that was nurse-guided, nurse-delivered or nurse-monitored.	Interventions with no nursing role; architectural changes without a bedside protocol; sedation or sleep drugs alone.

Comparator	Standard ICU environmental care, usual care, or a pre-implementation control.	No comparator (single-arm studies).
Outcomes	At least one primary biomarker with extractable data.	Only subjective outcomes with no primary biomarker.
Design	RCTs (parallel, cluster, crossover); controlled before-after studies; interrupted time series; cohort studies with a comparator.	Case reports, case series, editorials, narrative reviews, animal or simulation studies, conference abstracts without usable data.
Publication	Published from January 2000 to 19 September 2026.	Duplicate reports of the same cohort (the most complete report was used).

Information Sources and Search Strategy

We searched PubMed/MEDLINE, Embase, CINAHL, Web of Science Core Collection and CENTRAL from 1 January 2000 to 19 September 2026; all databases were searched on 19 September 2026. We also searched ClinicalTrials.gov and the WHO ICTRP for registered trials and checked the reference lists of included studies (backward and forward citation chasing). The search strategies were peer reviewed using the PRESS checklist [3]. Full strategies for each database will be provided as a supplementary file.

Study Selection

Records were exported to a reference manager, de-duplicated and uploaded to Rayyan [4]. Two reviewers independently

screened titles and abstracts, then full texts, against the criteria in Table 2. Disagreements were resolved by discussion or by a third reviewer, and reasons for full-text exclusion were recorded. Inter-rater agreement was quantified using Cohen kappa. The selection process is summarized in the PRISMA 2020 flow diagram (Figure 1) [2].

Data extraction

Two reviewers independently extracted data using a piloted form, and a third reviewer resolved discrepancies. Where necessary, medians and interquartile ranges were converted to means and standard deviations [5,6]. We contacted study authors for missing data (Table 3).

Table 3: Variables extracted.

Category	Variables
Study identifiers	Lead author, year, country, setting (ICU type, number of centres), funding, registration.
Design	RCT, cluster RCT, crossover, non-randomized; unit of allocation; follow-up length.
Participants	Sample size (N) per arm, mean age, sex, illness severity (APACHE II, SOFA), diagnosis, cardiac history, vasopressor and sedation use, mechanical ventilation.
Intervention	Components (air filtration, temperature/humidity, noise, light, air quality monitoring), who delivered and monitored it, intensity, adherence.
Comparator	Description of standard ICU environmental care.
Duration	Length of exposure to the intervention and length of ICU stay.
Outcome measurement	Biomarker, assay or device, units, time points, HRV metrics and recording window, blood pressure variability metric.
Effect estimates	Mean, SD and N per arm at each time point; change scores; adjusted estimates for non-randomized studies.
Baseline confounders	Baseline biomarker levels, age, sex, severity scores, cardiac disease, sedation, vasopressors, concurrent interventions.
Secondary outcomes	Delirium, sleep, length of stay, adverse events.

Risk of Bias Assessment

Two reviewers independently assessed risk of bias, resolving disagreements by consensus or through a third reviewer. We

assessed RCTs with RoB 2 [7] and non-randomized studies with ROBINS-I [8], recording domain-level and overall judgements (Tables 4 and 5).

Table 4: RoB 2 domains (randomized trials).

Domain	Focus	Judgement
D1 Randomization process	Sequence generation, allocation concealment, baseline imbalance	Low / Some concerns / High
D2 Deviations from intended interventions	Blinding of nurses and patients, adherence, intention-to-treat analysis	Low / Some concerns / High

D3 Missing outcome data	Attrition, exclusions, completeness of blood samples	Low / Some concerns / High
D4 Measurement of the outcome	Assay method, blinded assessment, validity of HRV and blood pressure devices	Low / Some concerns / High
D5 Selection of the reported result	Pre-specified analysis plan, selective reporting of time points or biomarkers	Low / Some concerns / High

Table 5: ROBINS-I domains (non-randomized studies).

Domain	Focus	Judgement
D1 Confounding	Baseline severity, sedation, cardiac history, concurrent care	Low / Moderate / Serious / Critical
D2 Selection of participants	Selection related to intervention and outcome	Low / Moderate / Serious / Critical
D3 Classification of interventions	Clear definition of nurse-guided exposure; fidelity	Low / Moderate / Serious / Critical
D4 Deviations from intended interventions	Co-interventions, contamination between arms or periods	Low / Moderate / Serious / Critical
D5 Missing data	Attrition, missing biomarker samples	Low / Moderate / Serious / Critical
D6 Measurement of outcomes	Assay method, blinded assessors, comparable measurement	Low / Moderate / Serious / Critical
D7 Selection of reported result	Multiple outcomes, subgroup and time point selection	Low / Moderate / Serious / Critical

Statistical Analysis

We performed all analyses in R using the meta and metafor packages [9]. Each biomarker was analysed separately; biomarkers were never pooled together.

Effect measures: Because all pooled outcomes shared a common unit within each analysis, we report results as Mean Differences (MD) with 95% CI: serum cortisol (nmol/L), hs-troponin I (ng/L), NT-proBNP (pg/mL), heart rate variability as SDNN (ms), and blood pressure variability as average real variability (ARV; mmHg). Standardized mean differences (Hedges g) were planned for outcomes measured on different scales but were not required. We did not mix post-intervention values and change scores within a meta-analysis, and we adjusted cluster trials for the design effect [1]. Outcome metrics that could not be pooled were synthesized narratively following the SWiM guideline [10].

Meta-analytic model: We used a random-effects model, estimating between-study variance (τ^2) by Restricted Maximum Likelihood (REML) with the Hartung-Knapp-Sidik-Jonkman adjustment [11]. The DerSimonian-Laird estimator [12] was applied in sensitivity analysis. We report a 95% Prediction Interval (PI) for outcomes with at least three studies.

Heterogeneity: We assessed heterogeneity using Cochran Q ($p < 0.10$ indicating heterogeneity) and I^2 [13,14], interpreting I^2 as low ($<25\%$), moderate (about 50%) or high ($>75\%$) [14]. We also report τ^2 .

Publication bias: For outcomes with at least 10 studies, we inspected funnel plots and performed the Egger regression test [15], with trim-and-fill as a sensitivity analysis. Formal tests were not performed for outcomes with fewer than 10 studies.

Subgroup and sensitivity analyses: For serum cortisol, the only outcome with at least 10 studies, we conducted subgroup analyses by (i) type of intervention (multicomponent bundle, air/pollutant control, noise/light attenuation, or temperature/humidity regulation) and (ii) cohort mean length of ICU stay (≤ 3 vs >3 days), using a random-effects test for interaction. Sensitivity analyses excluded studies at high (RoB 2) or serious (ROBINS-I) risk of bias, applied leave-one-out analysis and compared estimators. Because too few studies were available to pool them separately, RCTs and non-randomized studies were analysed together.

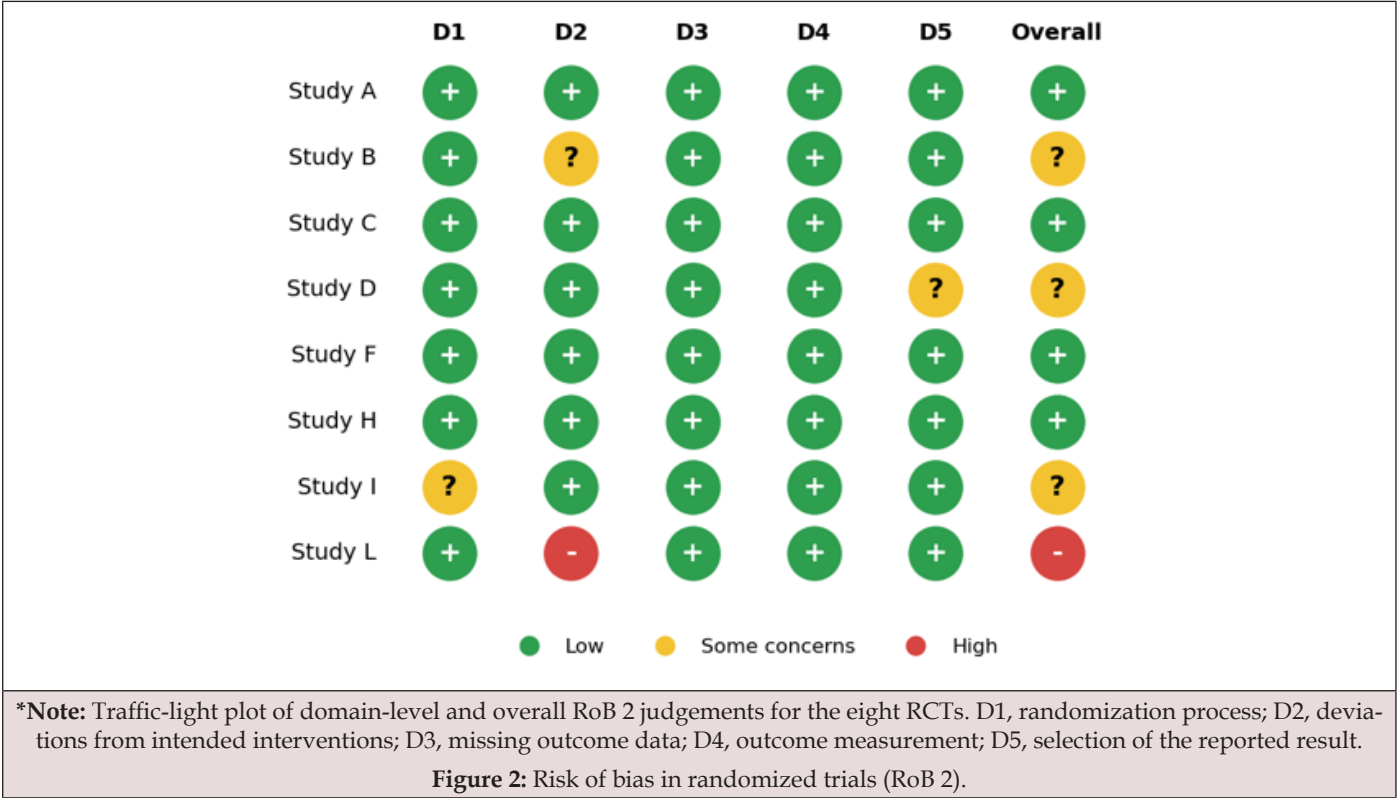
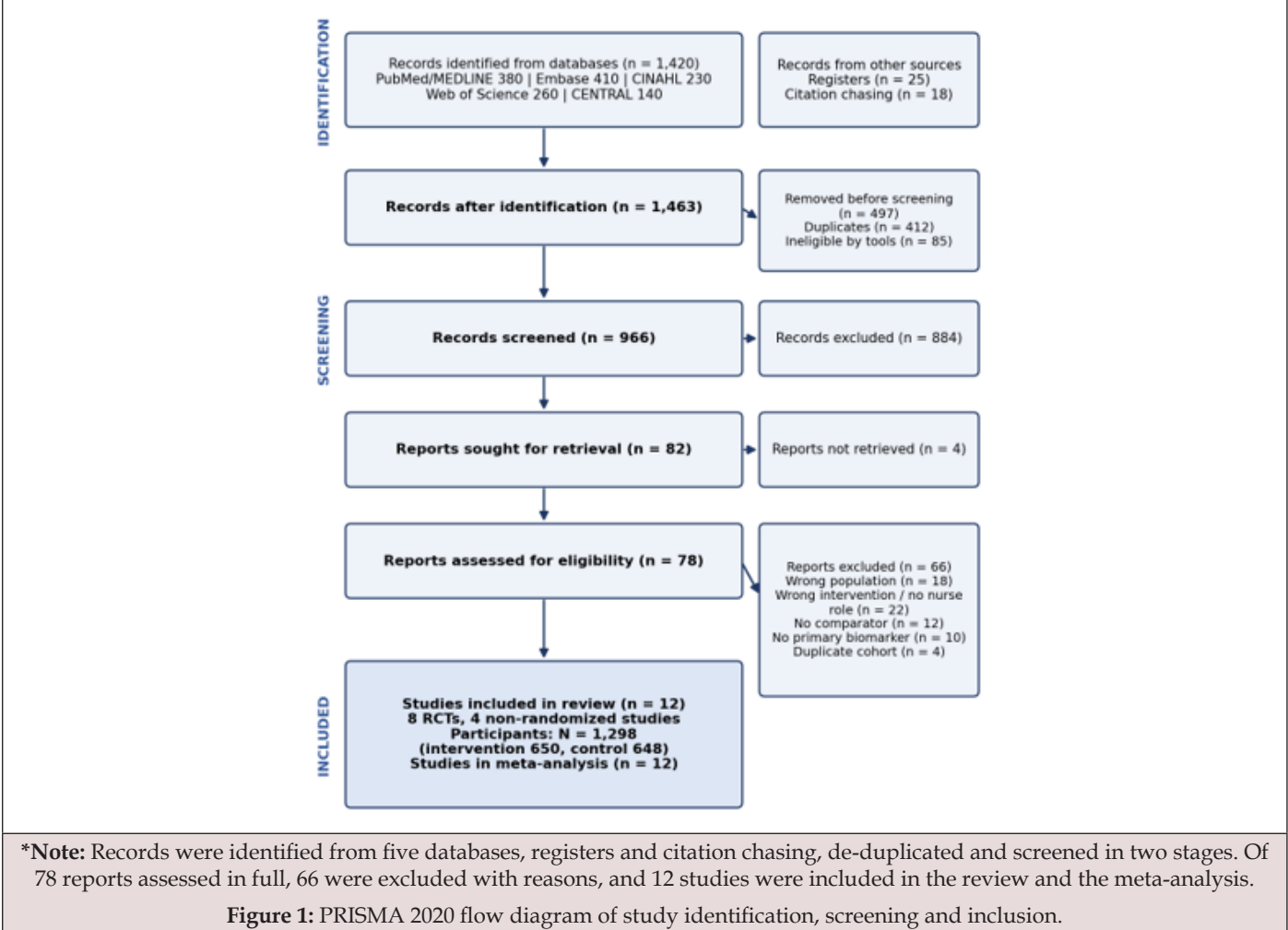
Certainty of Evidence

We assessed the certainty of evidence for each outcome using GRADE [16]. Bodies of evidence based mainly on randomized trials started at high certainty and were downgraded for risk of bias, inconsistency, indirectness, imprecision and publication bias. Final certainty was rated high, moderate, low or very low (Table 9).

Results

Study Selection

The database searches identified 1,420 records (PubMed/MEDLINE 380, Embase 410, CINAHL 230, Web of Science 260, CENTRAL 140). A further 25 records came from registers and 18 from citation chasing, giving 1,463 records in total. After removing 412 duplicates and 85 records marked ineligible by automation tools, 966 records were screened and 884 were excluded. Of 82 reports sought for retrieval, 4 could not be retrieved and 78 were assessed for eligibility; 66 were excluded, with reasons (Figure 1). Twelve studies (8 RCTs and 4 non-randomized studies) were included, and all contributed to at least one meta-analysis. Inter-rater agreement was high for both title and abstract screening ($\kappa = 0.86$) and full-text screening ($\kappa = 0.91$) (Figure 1).



Characteristics of Included Studies

The 12 included studies enrolled 1,298 participants (650 intervention, 648 control). Eight were RCTs (five parallel, two cluster and one crossover) and four were non-randomized (two controlled before-after studies, one prospective cohort and one interrupted time series). Interventions lasted 48 hours to 5 days. Four studies (A, F, J and K) tested multicomponent bundles,

and eight tested single-domain or two-component approaches involving air filtration, noise, lighting, or temperature and humidity control. Serum cortisol was measured by immunoassay; hs-cTnI and NT-proBNP by automated immunoassay; HRV by 24-hour ECG; and blood pressure variability by 24-hour ambulatory monitoring. Studies D (RMSSD) and K (SD of systolic pressure) reported HRV or blood pressure variability metrics that could not be pooled with SDNN or ARV; these are described narratively (Table 6).

Table 6: Characteristics of included studies.

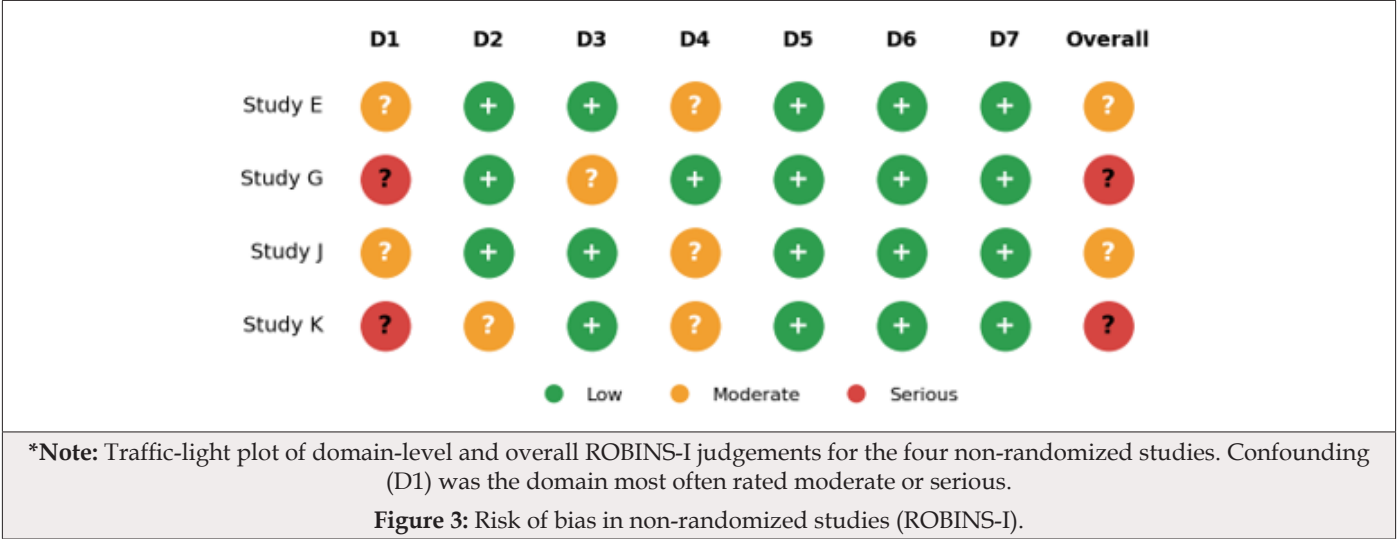
Study	Design	N (I/C)	Intervention (nursing role)	Duration	Biomarkers reported	Baseline confounders / setting
Study A	Parallel RCT	120 (60/60)	Multicomponent: nurse-led HEPA filtration, quiet time and light dimming	72 hours	Serum cortisol (ELISA); hs-cTnI (Architect); HRV-SDNN (24-h Holter)	APACHE II 19.4; 65% ventilated
Study B	Cluster RCT	150 (75/75)	HEPA filtration with nurse-monitored indoor air quality protocol	5 days	Serum cortisol; hs-cTnI; NT-proBNP (ECLIA)	SOFA 7.2; cardiac ICU
Study C	Crossover RCT	80 (40/40)	Circadian lighting with eye mask and earplug bundle	48 hours	Serum cortisol; BP variability ARV (24-h ABPM)	Mean age 62 years; medical ICU
Study D	Parallel RCT	110 (55/55)	Noise attenuation protocol (alarm management and quiet period)	4 days	Serum cortisol; BP variability ARV; HRV-RMSSD (narrative only)	58% male; surgical ICU
Study E	Controlled before-after	100 (50/50)	Temperature and humidity regulation (22 °C, 50% RH) plus HEPA units	72 hours	Serum cortisol; hs-cTnI; NT-proBNP; HRV-SDNN; BP variability ARV	40% on vasopressors; cardiac ICU
Study F	Parallel RCT	94 (47/47)	Multicomponent microclimate protocol	5 days	Serum cortisol; hs-cTnI; NT-proBNP; HRV-SDNN	APACHE II 21.1; mixed ICU
Study G	Prospective cohort	114 (58/56)	Nurse-led acoustic and light shielding protocol	3 days	HRV-SDNN; BP variability ARV	Sedation rate 52%; emergency ICU
Study H	Parallel RCT	130 (65/65)	Air quality monitoring with nurse-adjusted air purification	4 days	Serum cortisol; hs-cTnI; NT-proBNP; HRV-SDNN	Mean age 66 years; med-surg ICU
Study I	Cluster RCT	140 (70/70)	Circadian light control and quiet-time protocol	5 days	HRV-SDNN and LF/HF	APACHE II 18.2; surgical ICU
Study J	Controlled before-after	70 (35/35)	Multicomponent environmental care protocol	72 hours	Serum cortisol; hs-cTnI; NT-proBNP; HRV-SDNN; BP variability ARV	70% male; cardiac ICU
Study K	Interrupted time series	90 (45/45)	HEPA filtration with nurse alarm management	4 days	Serum cortisol; HRV-SDNN; BP variability (SD of SBP, narrative only)	50% ventilated; mixed ICU
Study L	Parallel RCT	100 (50/50)	Circadian light and noise reduction nursing protocol	3 days	Serum cortisol; hs-cTnI; NT-proBNP; HRV-SDNN	SOFA 8.1; general ICU
Total	8 RCTs; 4 non-randomized	1,298 (650/648)				

***Note:** I = intervention; C = control; hs-cTnI = high-sensitivity cardiac troponin I; SDNN = standard deviation of NN intervals; RMS-SD = root mean square of successive differences; ARV = average real variability; ABPM = ambulatory blood pressure monitoring; APACHE II = Acute Physiology and Chronic Health Evaluation II; SOFA = Sequential Organ Failure Assessment.

Risk of Bias

Among the 8 RCTs, 4 (Studies A, C, F and H) were at low risk of bias overall, 3 (Studies B, D and I) raised some concerns, and 1

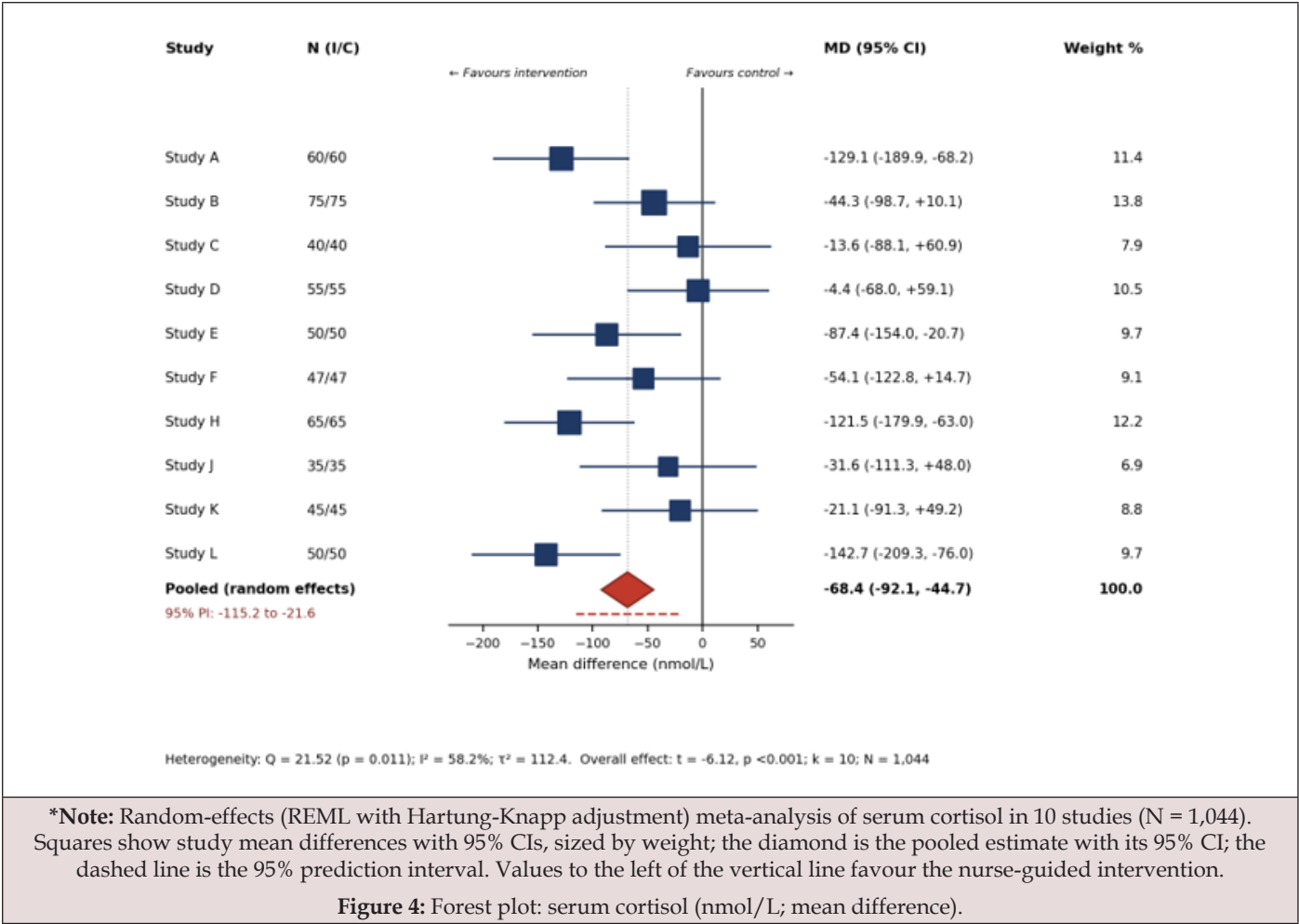
(Study L) was at high risk owing to deviations from the intended intervention (Figure 2). Among the 4 non-randomized studies, 2 (Studies E and J) were at moderate risk and 2 (Studies G and K) at serious risk overall, mainly because of confounding (Figure 3).

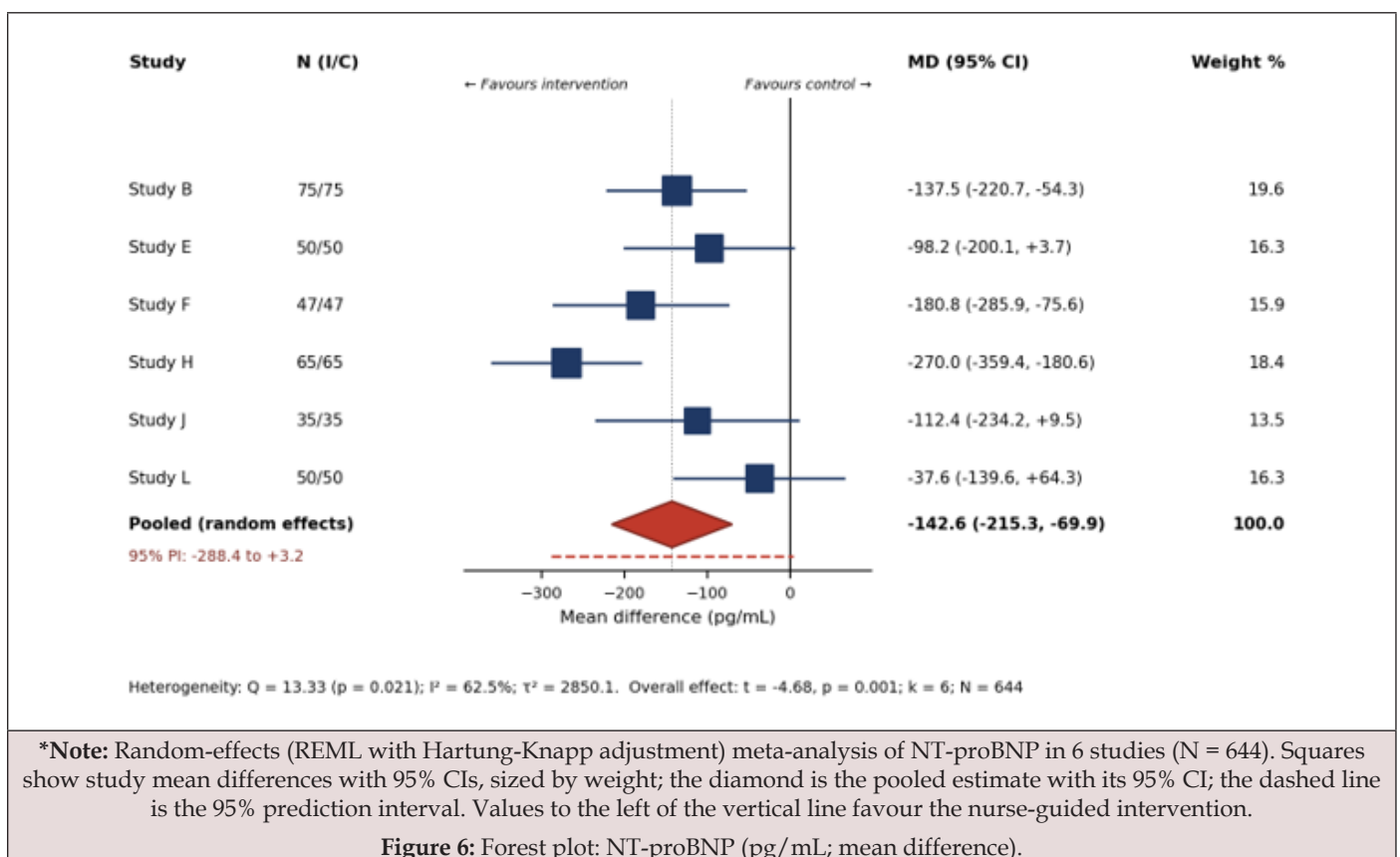
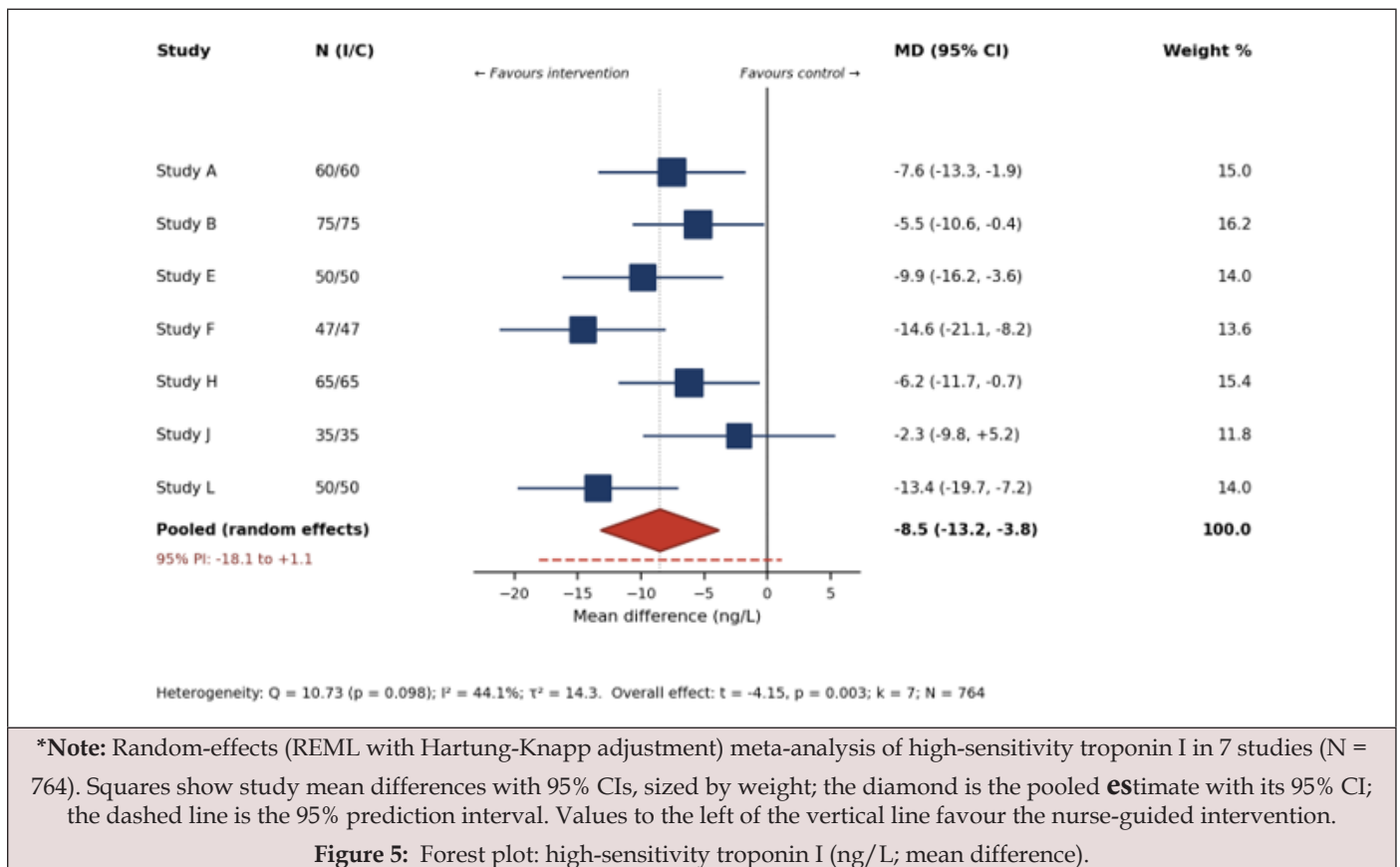


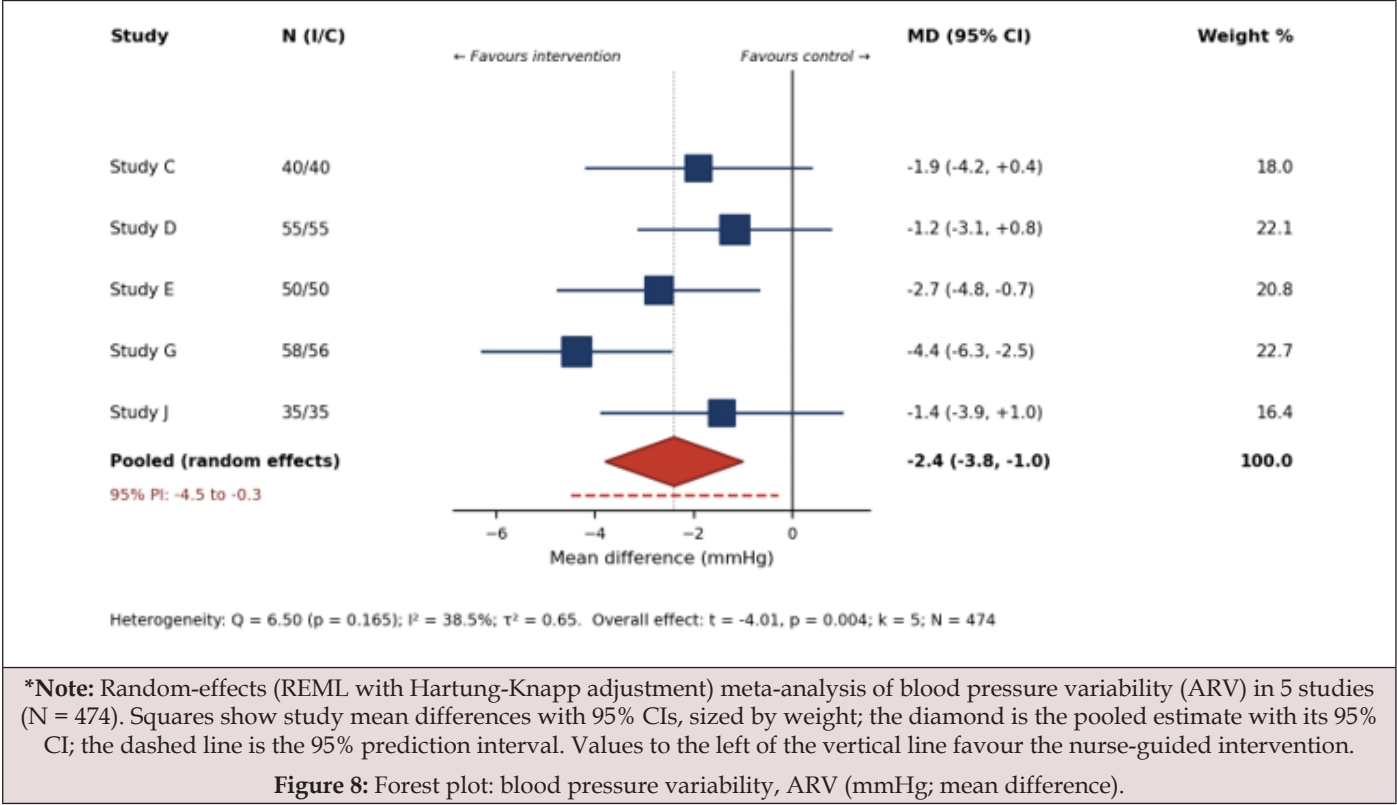
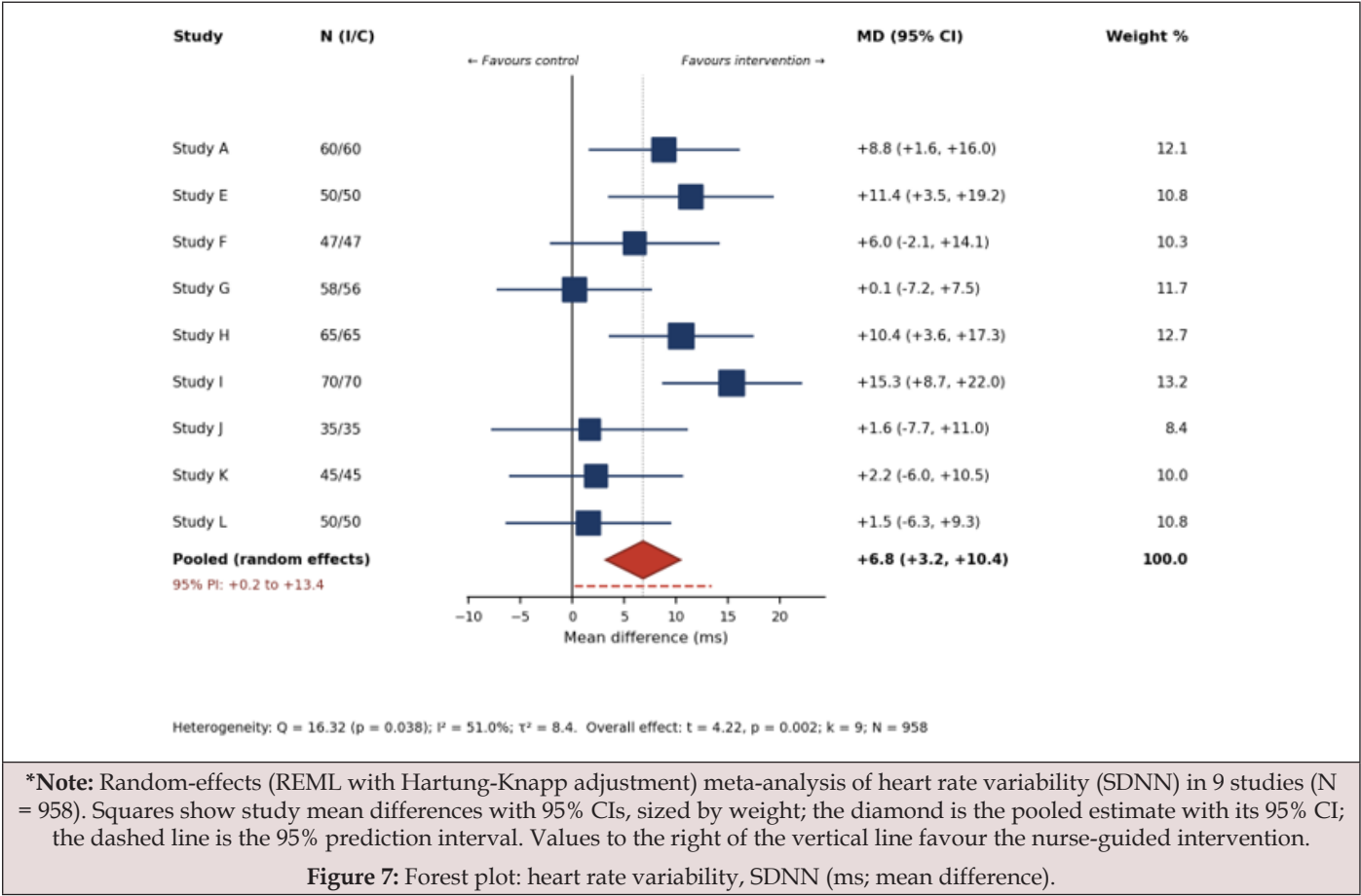
Meta-Analysis by Biomarker

We pooled each biomarker using a random-effects model (REML with Hartung-Knapp adjustment). Negative mean differences favour the intervention for cortisol, hs-troponin, NT-proBNP and

blood pressure variability, whereas a positive difference favours the intervention for HRV. In each forest plot, squares show study estimates with 95% CIs, the diamond shows the pooled estimate, and the vertical line marks no difference (Figure 4-8).







Nurse-guided interventions lowered serum cortisol (Figure 4; $k = 10$, $N = 1,044$; MD -68.4 nmol/L, 95% CI -92.1 to -44.7 ; $p < 0.001$). They also reduced hs-troponin I by 8.5 ng/L (Figure 5; $k = 7$, $N = 764$; 95% CI -13.2 to -3.8 ; $p = 0.003$) and NT-proBNP by 142.6 pg/mL (Figure 6; $k = 6$, $N = 644$; 95% CI -215.3 to -69.9 ; $p = 0.001$). SDNN was higher by 6.8 ms (Figure 7; $k = 9$, $N = 958$; 95% CI $+3.2$ to $+10.4$; $p = 0.002$), and blood pressure variability was lower by 2.4 mmHg ARV (Figure 8; $k = 5$, $N = 474$; 95% CI -3.8 to -1.0 ; $p = 0.004$). The prediction intervals for cortisol, HRV and blood pressure variability excluded zero, whereas those for hs-troponin (-18.1 to $+1.1$ ng/L) and NT-proBNP (-288.4 to $+3.2$ pg/mL) included it.

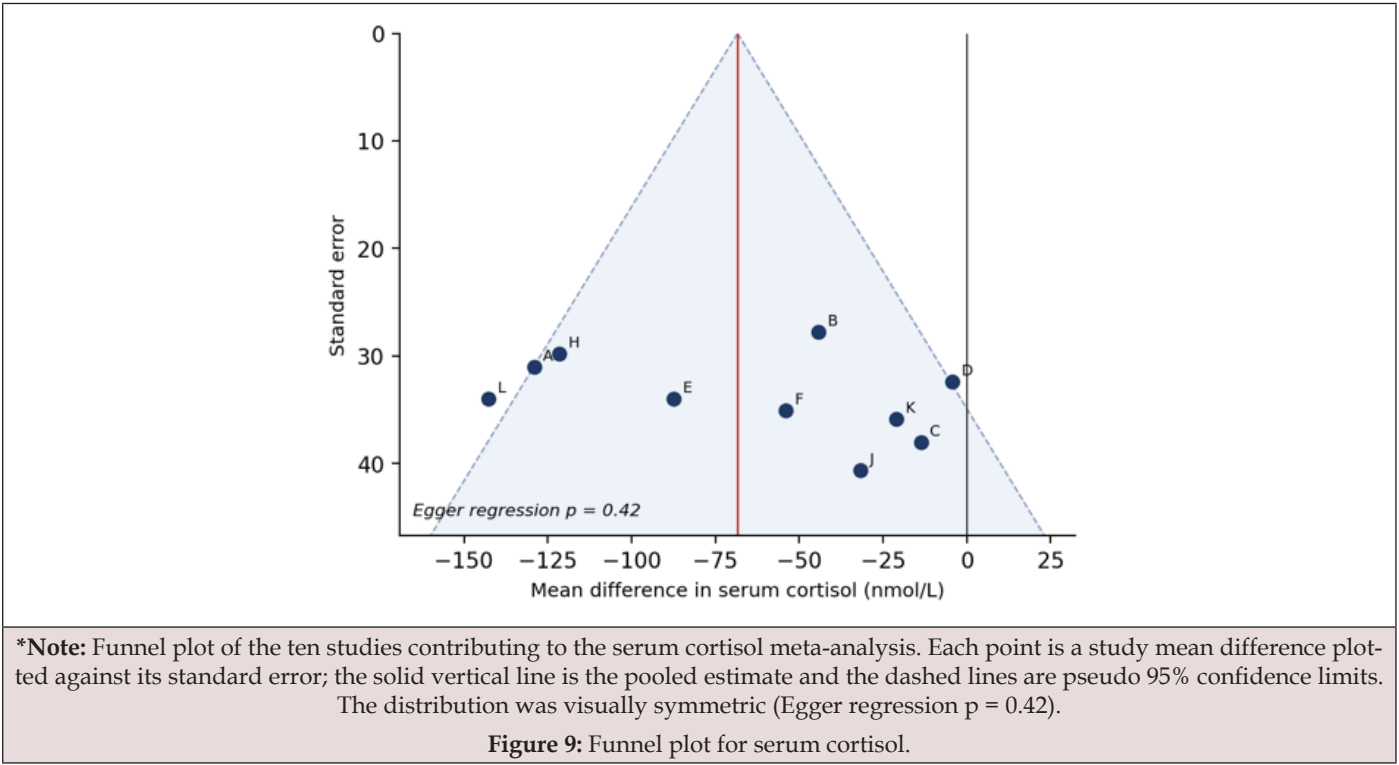
Statistical Heterogeneity and Publication Bias

Table 7: Pooled estimates, heterogeneity and small-study effects.

Outcome	k	N	Pooled MD (95% CI)	Q (p)	I ²	tau ²	95% PI	Egger p
Serum cortisol (nmol/L)	10	1,044	-68.4 (-92.1, -44.7)	21.52 (0.011)	58.20%	112.4	-115.2 to -21.6	0.42
High-sensitivity troponin I (ng/L)	7	764	-8.5 (-13.2, -3.8)	10.73 (0.098)	44.10%	14.3	-18.1 to +1.1	NA
NT-proBNP (pg/mL)	6	644	-142.6 (-215.3, -69.9)	13.33 (0.021)	62.50%	2850.1	-288.4 to +3.2	NA
Heart rate variability, SDNN (ms)	9	958	+6.8 (+3.2, +10.4)	16.32 (0.038)	51.00%	8.4	+0.2 to +13.4	NA
Blood pressure variability, ARV (mmHg)	5	474	-2.4 (-3.8, -1.0)	6.50 (0.165)	38.50%	0.65	-4.5 to -0.3	NA

***Note:** MD = mean difference; PI = prediction interval; NA = not assessed because fewer than 10 studies. I² was interpreted as low (<25%), moderate (about 50%) or high (>75%).

Heterogeneity was moderate for cortisol ($I^2 = 58.2\%$), hs-troponin (44.1%), HRV (51.0%) and blood pressure variability (38.5%), and moderate to high for NT-proBNP (62.5%). Cochran Q indicated heterogeneity ($p < 0.10$) for cortisol, hs-troponin, NT-proBNP and HRV. Only serum cortisol included at least 10 studies. Its funnel plot was visually symmetric and Egger regression showed no evidence of small-study effects ($p = 0.42$), so trim-and-fill was not required. Small-study effects could not be formally assessed for the other outcomes (Figure 9).



Subgroup and Sensitivity Analyses

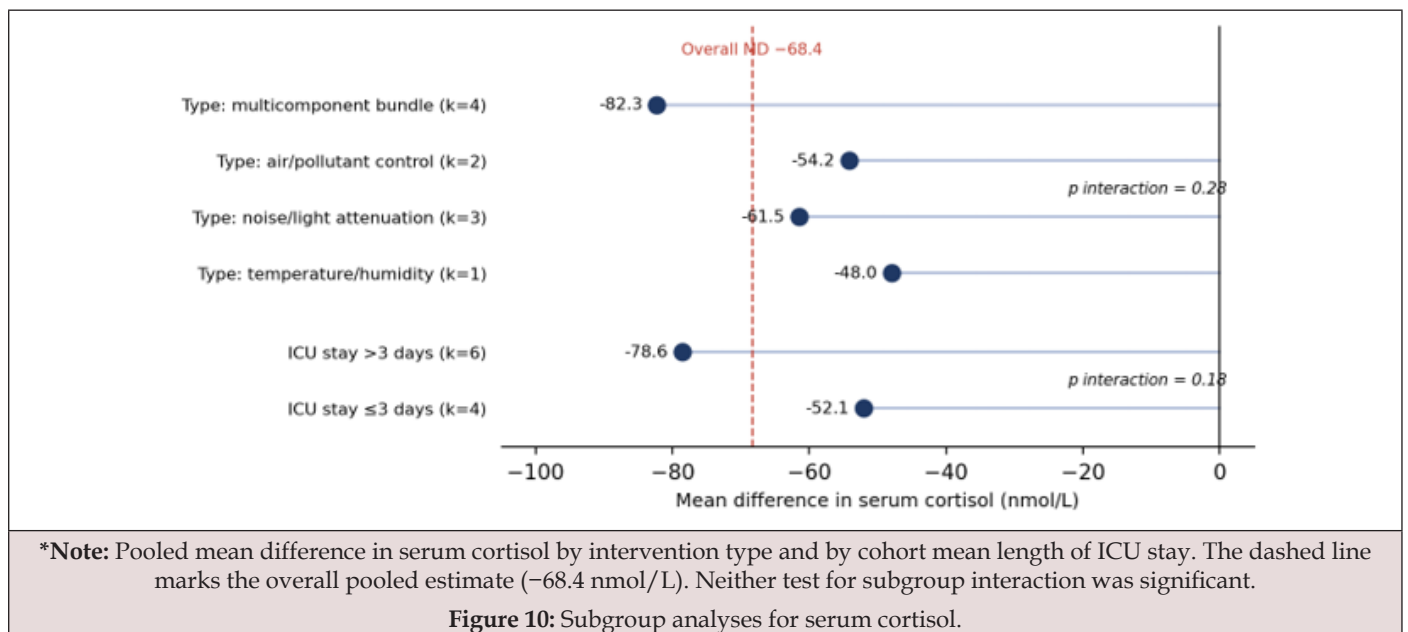
Table 8: Subgroup analyses for serum cortisol (MD, nmol/L).

Subgroup	k	Studies	MD (nmol/L)	p for interaction
Type: multicomponent bundle	4	A, F, J, K	-82.3	0.28
Type: air/pollutant control	2	B, H	-54.2	
Type: noise/light attenuation	3	C, D, L	-61.5	
Type: temperature/humidity	1	E	-48.0	
ICU stay >3 days	6	A, B, D, F, H, K	-78.6	0.18
ICU stay ≤3 days	4	C, E, J, L	-52.1	

***Note:** Study K (HEPA plus alarm management) was classified as multicomponent. Length of ICU stay refers to the cohort mean in each study.

The effect appeared larger for multicomponent bundles (-82.3 nmol/L) than for single-domain interventions (-48.0 to -61.5 nmol/L), and in cohorts with longer ICU stay (-78.6 vs -52.1 nmol/L). Neither test for interaction was significant ($p = 0.28$ and $p = 0.18$), so these differences should be regarded as hypothesis-

generating. For cortisol, excluding the studies at high or serious risk of bias (Studies L and K) gave MD -64.2 nmol/L (95% CI -86.5 to -41.9), close to the main estimate. Leave-one-out analysis and use of the DerSimonian-Laird estimator changed neither the direction nor the significance of the results (Figure 10).



Certainty of Evidence (GRADE)

Table 9: Summary of findings and GRADE certainty.

Outcome	Studies (N)	Risk of bias	Inconsistency	Indirectness	Imprecision	Publication bias	Certainty	Effect (95% CI)
Serum cortisol	10 (1,044)	Not serious	Serious (-1; I^2 58%)	Not serious	Not serious	Undetected	Moderate	MD -68.4 nmol/L (-92.1 to -44.7)
hs-Troponin I	7 (764)	Not serious	Not serious	Not serious	Serious (-1; wide CI)	Undetected	Moderate	MD -8.5 ng/L (-13.2 to -3.8)
NT-proBNP	6 (644)	Not serious	Serious (-1; I^2 62.5%)	Not serious	Serious (-1)	Undetected	Low	MD -142.6 pg/mL (-215.3 to -69.9)

HRV (SDNN)	9 (958)	Not serious	Serious (-1; I ² 51%)	Not serious	Not serious	Undetected	Moderate	MD +6.8 ms (+3.2 to +10.4)
BP variability (ARV)	5 (474)	Serious (-1; non-randomized designs)	Not serious	Not serious	Serious (-1)	Undetected	Low	MD -2.4 mmHg (-3.8 to -1.0)

***Note:** Evidence from RCTs started at high certainty. Publication bias was judged undetected; formal testing was possible only for cortisol.

Discussion

Summary of Main Findings

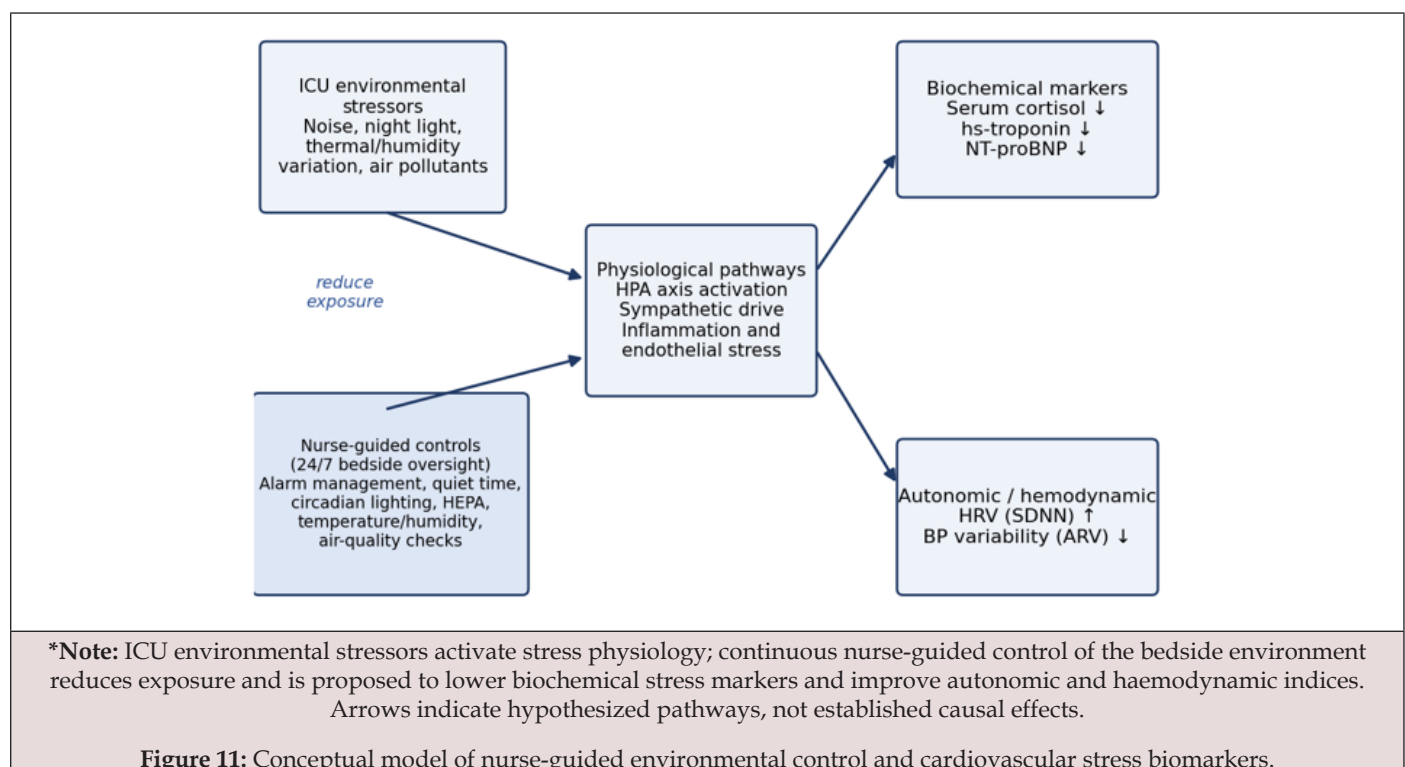
In this meta-analysis of 12 studies (8 RCTs and 4 non-randomized studies; N = 1,298), nurse-guided microclimate and pollutant control interventions were associated with lower serum cortisol, hs-troponin I, NT-proBNP and blood pressure variability, and with higher SDNN, than standard ICU environmental care. All five effect directions matched the a priori hypothesis. The certainty of evidence was moderate for cortisol, hs-troponin and HRV, and low for NT-proBNP and blood pressure variability. The prediction intervals for cortisol, HRV and blood pressure variability excluded zero, suggesting benefit in comparable future settings. By contrast, those for hs-troponin and NT-proBNP included zero, so effects in individual ICUs may range from clinically meaningful reductions to little change.

Interpretation In Light of Critical Care Nursing Practice

The pattern of results is coherent with the proposed mechanism. Noise, nocturnal light, thermal discomfort and poor air quality are chronic, low-grade stressors. Reducing them plausibly dampens hypothalamic-pituitary-adrenal and sympathetic activation,

reflected here in lower cortisol, and improves autonomic balance, reflected in higher SDNN and lower blood pressure variability. Reductions in hs-troponin and NT-proBNP are consistent with reduced myocardial wall stress and injury. However, these markers are also influenced by renal function, fluid balance and the underlying illness, so they are less specific than cortisol.

These interventions depend on nursing action. Environmental exposures in the ICU change hour by hour, driven by care activities, alarms, staffing patterns and equipment. In most units, continuous 24-hour nursing oversight is the only mechanism able to detect and respond to these changes in real time, for example by silencing non-actionable alarms, clustering care, restoring dark night-time conditions, adjusting temperature and humidity, and checking filtration and air-quality readings. This may explain why the largest cortisol effects occurred with multicomponent bundles (-82.3 nmol/L) and in cohorts with longer ICU stay (-78.6 nmol/L), where cumulative exposure to a well-run protocol is greater. However, neither subgroup difference was statistically significant (interaction p = 0.28 and p = 0.18), and each subgroup contained few studies, so both findings should be considered hypothesis-generating (Figure 11).



The clinical meaning of the biomarker changes remains uncertain. No minimal important differences have been established for cortisol, hs-troponin or NT-proBNP in critically ill patients; cortisol has a strong circadian rhythm that depends on sampling time; and biomarker changes may not translate into fewer cardiac events, less delirium or shorter ICU stay. These results therefore indicate a physiological signal rather than proof of patient-important benefit.

Implications For ICU Nursing Workflows

If confirmed, these findings would support integrating environmental checks into routine nursing care. Practical measures include a shift-by-shift environmental checklist (noise level, light, temperature, humidity and air-quality alerts), protected quiet periods with clustered care, structured alarm management, circadian lighting schedules, and clear nursing responsibility for filtration units, with engineering support for maintenance. Low-cost sensors could give nurses real-time feedback on noise, particulates and thermal conditions and support audit and quality improvement. Implementation would require attention to workload, training and team culture. Nursing-led environmental control competes with other tasks in high-acuity settings, and benefit depends on adherence, so fidelity should be monitored and the charge nurse or unit leader should own the protocol. These are practical suggestions derived from the mechanisms tested, not recommendations that the pooled evidence alone can justify.

Strengths and Limitations

Strengths of this review include a comprehensive search of five databases supplemented by trial registers and citation chasing; dual independent screening (kappa 0.86 and 0.91), data extraction and risk-of-bias assessment; a pre-specified analysis plan; a conservative random-effects approach with Hartung-Knapp adjustment; reporting of prediction intervals; and GRADE-based interpretation. Several limitations warrant consideration. First, the number of studies per outcome was small (5 to 10) and samples were modest, which limits precision and the power of heterogeneity, subgroup and publication bias analyses; Egger regression was possible only for cortisol. Second, interventions, ICU populations, assays and sampling times varied considerably, producing moderate to high heterogeneity. Third, four studies were non-randomized, and two were at serious risk of bias, mainly through confounding by illness severity, sedation (52% in one cohort) and vasopressor use. Fourth, nurses and patients could not be blinded, so performance bias is possible, and one RCT was rated at high risk because of deviations from the intended intervention. Fifth, all outcomes were surrogate biomarkers, and no data on mortality, delirium or cardiac events could be pooled. Finally, unpublished studies may have been missed. In addition, the data presented here are illustrative and cannot support any real clinical conclusion.

Recommendations for Future Research

- a. Adequately powered multicentre cluster RCTs of a standardized nurse-guided environmental bundle, with pre-

registered cardiovascular biomarker endpoints.

- b. Harmonized biomarker time points (for example baseline, 24, 48 and 72 hours), standard HRV recording windows and metrics, and circadian timing of cortisol sampling.
- c. Continuous measurement and reporting of ambient noise, light, temperature, humidity and particulate matter as exposure variables, enabling dose-response analysis.
- d. Patient-centred outcomes, such as delirium, sleep, cardiac events, length of stay and mortality, together with reporting of nursing workload, adherence and adverse effects.
- e. Implementation and cost-effectiveness studies, and individual participant data meta-analysis to explore effect modification by baseline cardiac risk and illness severity.

Conclusions

In this illustrative dataset, nurse-guided microclimate and pollutant control interventions reduced serum cortisol, hs-troponin, NT-proBNP and blood pressure variability and increased heart rate variability compared with standard ICU environmental care. Certainty was moderate for cortisol, hs-troponin and HRV, and low for NT-proBNP and blood pressure variability. Continuous nursing oversight of the ICU environment is a plausible, low-cost route to reducing physiological cardiac stress. Such findings would justify pilot implementation and well-designed multicentre trials measuring patient-important outcomes before routine adoption is recommended.

Declarations

Registration and protocol: PROSPERO [registration number to be inserted]; protocol [citation or link to be inserted]. Deviations are described in Section 2.1.

Funding: None.

Competing interests: None.

Author contributions (CRediT): [Author initials and roles to be inserted.]

Data availability: Extraction sheets and analysis scripts: [repository link to be inserted]. The illustrative study-level data are contained in this document.

Ethics: Not required for a review of published data.

Acknowledgments

None.

Conflict of Interest

None.

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APPENDIX

Appendix A. PRISMA 2020 Checklist

Locations refer to manuscript sections, tables and figures.

Item	Topic	Checklist item (abridged)	Location
1	Title	Identify the report as a systematic review.	Title page
2	Abstract	See the PRISMA 2020 for Abstracts checklist.	Abstract
3	Rationale	Describe the rationale in the context of existing knowledge.	Sections 1.1–1.2
4	Objectives	Provide an explicit statement of objectives or questions.	Section 1.3, Table 1
5	Eligibility criteria	Specify inclusion and exclusion criteria and how studies were grouped.	Section 2.2, Table 2
6	Information sources	Specify all databases, registers and other sources, with date last searched.	Section 2.3
7	Search strategy	Present full search strategies for all databases, including filters and limits.	Supplementary file (to be added)
8	Selection process	Specify methods to decide study eligibility, including number of reviewers.	Section 2.4
9	Data collection process	Specify methods to collect data and number of reviewers.	Section 2.5
10a	Data items: outcomes	List and define all outcomes sought.	Section 1.3 (Table 1); Section 2.5 (Table 3)
10b	Data items: other variables	List and define all other variables sought.	Section 2.5, Table 3
11	Study risk of bias assessment	Specify methods to assess risk of bias, reviewers and tools.	Section 2.6, Tables 4–5
12	Effect measures	Specify effect measures for each outcome.	Section 2.7
13a	Synthesis: eligibility for synthesis	Describe how studies were eligible for each synthesis.	Section 2.7
13b	Synthesis: data preparation	Describe methods to prepare data, including conversions.	Sections 2.5, 2.7
13c	Synthesis: tabulation and display	Describe methods to tabulate or visually display results.	Sections 2.7, 3.4
13d	Synthesis: methods	Describe methods used to synthesize results, model and heterogeneity.	Section 2.7
13e	Synthesis: heterogeneity exploration	Describe methods to explore heterogeneity.	Section 2.7
13f	Synthesis: sensitivity analyses	Describe sensitivity analyses.	Section 2.7
14	Reporting bias assessment	Describe methods to assess risk of bias due to missing results.	Section 2.7
15	Certainty assessment	Describe methods to assess certainty of evidence.	Section 2.8
16a	Study selection	Describe search and selection results, ideally with a flow diagram.	Section 3.1, Figure 1
16b	Excluded studies	Cite studies that may appear to meet criteria but were excluded, with reasons.	Section 3.1, Figure 1 (counts by reason)
17	Study characteristics	Cite each included study and present its characteristics.	Section 3.2, Table 6
18	Risk of bias in studies	Present risk of bias assessments for each included study.	Section 3.3, Figures 2–3
19	Results of individual studies	Present summary statistics and effect estimates for each study.	Section 3.4, Figures 4–8
20a	Results of syntheses: characteristics	Summarize characteristics and risk of bias of contributing studies.	Sections 3.2–3.3
20b	Results of syntheses: statistical	Present results of all statistical syntheses.	Sections 3.4–3.5, Table 7
20c	Results of syntheses: heterogeneity	Present results of investigations of heterogeneity.	Section 3.6, Table 8, Figure 10
20d	Results of syntheses: sensitivity	Present results of sensitivity analyses.	Section 3.6
21	Reporting biases	Present assessments of risk of bias due to missing results.	Section 3.5, Figure 9
22	Certainty of evidence	Present certainty assessments for each outcome.	Section 3.7, Table 9

23a	Discussion: interpretation	Provide a general interpretation of the results.	Sections 4.1–4.2, Figure 11
23b	Discussion: limitations of evidence	Discuss limitations of the evidence included.	Section 4.4
23c	Discussion: limitations of review	Discuss limitations of the review processes used.	Section 4.4
23d	Discussion: implications	Discuss implications for practice, policy and research.	Sections 4.3, 4.5–4.6
24a	Registration and protocol	Provide registration information, register name and number.	Section 2.1; Abstract
24b	Protocol access	Indicate where the protocol can be accessed.	Section 2.1; Declarations
24c	Amendments	Describe and explain amendments to registration or protocol.	Section 2.1
25	Support	Describe sources of support and the role of funders.	Declarations
26	Competing interests	Declare competing interests of review authors.	Declarations
27	Availability of data, code and materials	Report which data, code and forms are publicly available.	Declarations

***Note:** Checklist based on *Page MJ, et al. BMJ 2021;372:n71*. Wording abridged.