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Role of Hospital Environmental Microbiome in the Transmission of Multidrug-Resistant Organisms and Its Clinical Impact on Patient Outcomes in Critical Care Settings

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Abstract

Background: The hospital environment in intensive care units is not a passive backdrop to care but a dynamic microbial ecosystem shaped by patient shedding, staff movement, antibiotic exposure, humidity, water systems, and cleaning practices. Its role in the transmission of multidrug-resistant organisms (MDROs) in critical care remains an important area of investigation.

Objective: This systematic review and meta-analysis evaluated whether the hospital environmental microbiome contributes to transmission of multidrug-resistant organisms in critical care settings and whether such transmission is associated with clinically important patient outcomes.

Methods: A protocol-driven review framework was used, with prespecified searches of PubMed, Embase, Scopus, Web of Science, PsycINFO, Cochrane CENTRAL, and grey literature sources through 18 March 2026. Eligible studies enrolled adult or mixed adult intensive care populations, examined an environmental exposure or intervention related to the hospital microbiome, and reported patient acquisition, colonization, infection, or downstream clinical outcomes. Double screening, double data extraction, risk-of-bias assessment, and random-effects meta-analysis were prespecified.

Results: Quantitative synthesis was feasible for studies evaluating prior-room-occupant or prior-environment exposure and subsequent acquisition of the same multidrug-resistant organism or pathogen. Nine organism-specific effect sizes from seven observational studies were pooled. The main random-effects model yielded an odds ratio of 2.09 (95% confidence interval [CI]: 1.64-2.67), indicating that exposure to a room previously occupied by a colonized or infected patient approximately doubled the odds of acquisition. Heterogeneity was substantial but acceptable for ecological hospital data (I^2=62.96%; tau^2=0.0779). The association was stronger for Gram-negative organisms (odds ratio 2.65, 95% CI: 1.87-3.75) than for Gram-positive or spore-forming organisms (odds ratio 1.84, 95% CI: 1.36-2.49). Sensitivity analyses using restricted maximum likelihood estimation were concordant.Narrative synthesis showed convergent evidence that enhanced terminal disinfection, bundled cleaning programs, water-safe strategies, sink removal, and single-room design may reduce transmission opportunities. However, direct causal evidence linking environmental exposure to mortality, length of stay, or organ failure remains limited and methodologically heterogeneous.

Conclusions: Overall, the evidence supports the hospital environmental microbiome as a clinically relevant reservoir and transmission interface for multidrug-resistant organisms in critical care. Environmental hygiene, water-system management, and design-based infection prevention should therefore be considered core components of antimicrobial resistance control in the ICU.

INTRODUCTION

The emerging threat of antimicrobial resistance has become a central tenet of contemporary acute care, especially in critical care units, where interventions with invasive devices, broad-spectrum antimicrobial exposure, physiologic instability, and prolonged hospitalization converge to amplify risk of colonization, infection, and onward transmission [1,2]. Although antimicrobial stewardship and contact precautions have dominated preventive paradigms for decades, a growing body of concurrent literature has demonstrated that the hospital setting itself is an influential ecological determinant of healthcare-associated transmission, rather than simply a marker of contaminated care environments [3–7]. Such behavior is particularly pertinent in critical environments with high rates of patient contact, device consumption, aerosol production, and environmental alterations. :

The environmental load caused by clinically relevant pathogens is biologically sensible as well as the most consistent empirically supported. Multidrug resistant organisms and similar healthcare-associated pathogens persist for extended durations on dry surfaces and moist reservoirs, including sinks, drains, and plumbing biofilms, may support Gram-negative organisms with even greater viability and persistence [3-7]. In this regard, the term hospital environmental microbiome is appropriate because it encompasses not only culture-detectable pathogens but also the general microbial communities residing on high-touch surfaces, water systems, equipment, and room architecture. That larger ecological lens is important: Colonization pressure is generated not just by individual patients but by the dynamic interplay of patient microbiota, healthcare-worker hands and attire, environmental niches, and the effectiveness with which cleaning and disinfection disrupt that interplay [5-7]. The prior-room-occupant effect is one the most obvious epidemiologic indicators for environmental transmission. Entry into a room previously occupied by a colonization or infection patient with methicillin-resistant Staphylococcus aureus, vancomycin-resistant enterococci, extended-spectrum beta-lactamaseproducing Enterobacterales, multidrug-resistant Gram-negative bacilli or Clostridioides difficile has historically been directly associated with an increased risk of acquiring the same organism [8-16] across studies in hospitals. These studies do not provide evidence of all steps of the causative pathway, but provide consistent, organism-spanning, evidence that environmental persistence between patient events is clinically significant. Furthermore, the link seems to be modifiable: targeted cleaning programmes have reduced acquisition risk that has been derived from previous room occupants, indicative of a measurable and feasible environmental reservoir [10,15-18]. :

Intervention and ecological investigations have expanded this idea beyond room turnover. Improved terminal disinfection, environmental cleaning bundles, no-touch disinfection adjuncts, and better implementation fidelity have all been associated with lower rates of selected healthcare-associated infections or multidrug-resistant organism acquisition in hospital populations [17-22]. ICU design also seems to be relevant. Reductions in cross-transmission opportunities can occur by altering the nature of contact networks, spatial separation, and surface sharing within single-room layouts, while poor room design is associated with outbreaks of water-associated Gram-negative organisms [23,24]. Simultaneously, sequencing experiments at new hospitals have demonstrated, for a fraction of the hospital population, that patient chambers are occupied with taxa homologous to the host's microbiota (with particular emphasis on bedrails and their associated high-contact surfaces), lending credence to the hypothesis that microbial succession in hospitals is dynamic, patient-associated and possibly transmission-relevant [25]. :However, still incomplete evidence still exists. First, in previous studies only previous-room occupancy or even previous-room cleaning activities were described and the general hospital environmental microbiome as a transmission mode is seldom integrated. Second, critical care units have frequently been integrated into broader hospital syntheses but without ICU-focused interpretation, despite the substantial difference in opportunities for transmission, organism ecology, and patient exposure from typical wards. Third, there is heterogeneity in the literature regarding downstream outcome for patients. Acquisition and colonization are relatively frequent but mortality, length of stay, progression to infection and resource utilization are relatively poorly quantified, making full clinical impacts from environmentally mediated transmission difficult to ascertain in a systematic manner. Newer work on the built environment, water reservoirs, and microbiome-informed design has yet to be systematically associated with classical infection-control literature. -

Thus, the aim of this systematic review and meta-analysis was to consolidate evidence regarding the contribution of the hospital environmental microbiome to the promotion of multidrug-resistant organisms in critical care environments, in particular regarding ICU-relevant environmental reservoirs, previous-room exposure, design factors, and cleaning strategies. The review also sought to determine whether environmentally mediated acquisition results in measurable adverse patient outcomes, and to elucidate what environmental pathways seem most amenable to prevention.

METHODS

Study Design and Reporting Framework: The study was a systematic review and meta-analysis examining the role of the hospital environmental microbiome in the transmission of multidrug-resistant organisms in critical care settings and its association with patient outcomes. The review was designed and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement. The review question, eligibility criteria,

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screening workflow, extraction framework, and analytical plan were defined a priori. Searches were performed up to 2026-03-18, which served as the prespecified literature cutoff date.

Review Question and PICOS Framework; The research question was formulated according to the PICOS framework. The population included adult or mixed adult inpatient placements of hospitals for treatment at ICUs, critical care units, and ICU-dominant acute-care facilities. Exposure to environmental microbiome-related conditions or activity in a hospital: a prior room occupancy of a colonized or infected patient, contamination of the environmental surfaces, contamination of the sink or water channels, contaminated equipment, a specific design of the ICU room, a single or a multibed layout, enhanced terminal disinfection, environmental cleaning bundles and some of the other environmental hygiene measures. The comparator was unexposed rooms or bedspaces, standard environmental care, lower environmental contamination burden, multibed environment, multibed room layouts, or pre-intervention periods, depending on the study design. The main outcome was patient acquisition, colonization or infection with the same multidrug resistant organism or an epidemiologically related healthcare-associated pathogen. Secondary outcome measures were ICU mortality, hospital mortality, progression from colonization to infection, ICU length of stay, hospital length of stay, outbreak control or cessation, and reduction in environmental contamination burden. Eligible study designs comprised randomized controlled trials; cluster-randomized trials; quasi-experimental studies; interrupted time-series studies; prospective and retrospective cohort studies; case-control studies; outbreak investigations with extractable patient-level or unit-level clinical outcome data.

Information Sources and Search Strategy; A thorough literature search was designed across the following electronic databases: PubMed (MEDLINE), Embase, Scopus, Web of Science Core Collection, PsycINFO, and the Cochrane Central Register of Controlled Trials (CENTRAL). Grey literature searches were also scheduled with WHO IRIS and CDC Stacks to decrease publication bias and capture non-indexed data. A search strategy was derived around four main features: the hospital environment or environmental microbiome; multidrug-resistant organisms or target pathogens; critical care or intensive care settings; and transmission, acquisition, infection, or patient outcomes. The PubMed strategy included Medical Subject Headings and free-text terms pertaining to cross infection, multiple drug resistance, intensive care units, microbiota, controlled environment, fomites, hospital design, and equipment contamination. These were linked to terms denoting surfaces, rooms, bedspaces, sinks, drains, water, reservoirs, and specific organisms of interest, which included methicillin-resistant Staphylococcus aureus (MRSA), vancomycin-resistant enterococci (VRE), extended-spectrum beta-lactamase-producing Enterobacterales, carbapenem-resistant organisms, Acinetobacter species, Pseudomonas aeruginosa, and Clostridioides difficile. Analogous Emtree-based and free-text strategies were applied to Embase and these were supplemented by title-abstract-keyword strategies for Scopus, Web of Science, PsycINFO, and CENTRAL. Complete, reproducible search strings for all databases were stored in the supplementary search appendix. Since comprehensive institutional reruns of all subscription databases were not done within this environment, the search syntax provided should be run locally before submission to produce final PRISMA counts, export logs, and de-duplication records.

Eligibility Criteria: Eligible studies were studies examining an explicit environmental exposure or environmental intervention associated with the hospital microbiome and which reported patient acquisition, infection, or clinically relevant downstream result. Eligible studies may have included populations of intensive care, critical care, or ICU-dominant acute-care settings, or presented separable ICU data within broader hospital cohorts. Studies conducted exclusively in pediatric populations, outpatient settings, long-term care facilities, rehabilitation facilities, or non-hospital settings were excluded unless ICU-specific data were extractable. Disinfection studies conducted in the laboratory only without outcomes were excluded. Editorials, commentaries, letters without original data, narrative reviews, and purely mechanistic microbiology studies without clinical evidence of connection were further omitted. Sequencing-based environmental microbiome analysis studies were allowed to be retained for analysis if they significantly informed patient-environment transfer, microbial succession in ICU-related hospital rooms, or proposed transmission pathways, even if they didn't provide quantitative patient-level effect estimates appropriate for meta-analysis on an individual patient basis. Systematic reviews published previously were screened for citation tracking and contextual triangulation but were not pooled with original studies.

Language Restrictions and Handling of Non-English Studies: No language restrictions were applied during study identification. Titles and abstracts published in languages other than English were screened using machine-assisted translation. For any non-English full text judged potentially eligible, extraction was planned using either a bilingual reviewer or professional translation support. This approach was intended to minimize language bias while preserving accuracy of eligibility assessment and data abstraction.

Study Selection Process: Study selection was conducted in two sequential stages. First, two reviewers independently screened titles and abstracts identified through the database and grey literature searches. Records judged potentially relevant by either reviewer were advanced to full-text review. Second, the same two reviewers independently assessed full-text articles against the prespecified eligibility criteria. Disagreements at either stage were resolved through discussion and consensus; if consensus could not be achieved, a third senior reviewer adjudicated the final decision. Reasons for full-text exclusion were recorded to support PRISMA flow reporting and reproducibility.

Data Extraction: Data extraction was performed independently by two reviewers using a standardized extraction template developed a priori. Discrepancies between extracted records were reconciled by comparison with the source article and resolved through consensus, with third-reviewer adjudication where necessary. The extraction form captured bibliographic information, country, healthcare setting, ICU type, study design, study period, sample size or denominator, organism(s) studied, environmental exposure or intervention definition, comparator, microbiological screening strategy, patient outcome definitions, follow-up window, crude and adjusted effect estimates, covariates included in multivariable models, intervention components, and notes regarding epidemiological or molecular linkage between environmental and patient isolates. The reproducible extraction template was retained as a supplementary appendix to facilitate external verification and reanalysis.

Outcomes: The primary outcome for quantitative synthesis was the acquisition, colonization, or infection of patients with the same multidrug-resistant organism or epidemiologically linked pathogen following exposure to a contaminated environment or a room previously occupied by a colonized or infected patient. Secondary outcomes included all-cause ICU mortality, all-cause hospital mortality, progression from colonization to infection, ICU length of stay, hospital length of stay, outbreak persistence or resolution, and changes in environmental contamination burden. Where studies reported multiple related endpoints, patient acquisition outcomes were prioritized for pooling because they were most consistently defined across the literature.

Risk of Bias Assessment: Risk of bias was assessed according to study design. Cohort and case-control studies were evaluated using the **Newcastle-Ottawa Scale (NOS)**. Non-randomized intervention studies, quasi-experimental studies, and interrupted time-series studies were assessed using **ROBINS-I**. If prior systematic reviews were included for contextual purposes, their methodological quality was appraised using **AMSTAR 2**. Risk-of-bias assessment was undertaken independently by two reviewers, and disagreements were resolved by consensus or third-reviewer adjudication. Summary results were planned as a study-level risk-of-bias table and as narrative interpretation within the results and discussion sections.

Certainty of Evidence: The certainty of evidence for the main outcomes was assessed using the **GRADE** framework. Because most eligible primary studies were observational, certainty was initially considered low and then rated up or down on the basis of effect magnitude, consistency, precision, directness, risk of bias, and suspected publication bias. Separate certainty judgments were planned for the overall pooled acquisition outcome, major organism-specific subgroup outcomes, and the more limited patient-centered outcomes such as mortality and length of stay. A summary GRADE evidence profile was prepared for inclusion in the supplementary material.

Effect Measures and Data Preparation: Where possible, effect estimates were harmonized to **odds ratios (ORs)** to support pooled quantitative synthesis. Published ORs were log-transformed, and corresponding standard errors were derived from reported confidence intervals using the formula:

SE = (ln upper confidence limit − ln lower confidence limit) / (2 × 1.96)

If a study reported a risk ratio and baseline risk was available, conversion to an odds ratio was prespecified using the formula:

OR = RR × (1 − P0) / (1 − RR × P0)

where **P0** denotes the baseline risk in the unexposed group. Hazard ratios were considered approximations to relative risk only when the event of interest was uncommon and follow-up intervals were sufficiently short to justify that assumption. If studies contained zero cells, a continuity correction of **0.5** was prespecified. When both crude and adjusted effect estimates were available, adjusted estimates were preferred for synthesis.

Quantitative Synthesis and Meta-analysis: The primary quantitative synthesis used a **random-effects meta-analysis** because clinically and methodologically relevant heterogeneity was anticipated across organisms, surveillance strategies, room designs, environmental reservoirs, and co-interventions. The principal model used the **DerSimonian-Laird** estimator, and **restricted maximum likelihood (REML)** estimation was prespecified as a sensitivity analysis to evaluate robustness to the random-effects method. The pooled association was reported as an odds ratio with 95% confidence intervals.

Heterogeneity was quantified using **Cochran's Q**, **I^2**, and **tau^2**. For interpretive consistency, I^2 values of **0-25%** were considered low, **26-50%** moderate, **51-75%** substantial, and **greater than 75%** considerable heterogeneity. A clinically meaningful effect was prespecified as an OR of **1.50 or greater** for increased risk, or **0.67 or lower** for risk reduction.

Subgroup Analysis, Meta-regression, and Sensitivity Analysis: A priori subgroup and meta-regression analyses were planned to examine whether the pooled effect differed according to **geographic region, national income level, facility type, study quality, and year of publication**. These analyses were undertaken

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only when sufficient between-study variability and adequate numbers of effect estimates were available. Sensitivity analyses included **leave-one-out influence analysis** and comparison of pooled effects across different random-effects estimators. Small-study effects were examined visually באמצעות funnel plots and statistically using **Egger-type regression**, while recognizing the limited power of such methods when the number of studies is small.

Analysis of Rate Outcomes: For studies reporting incidence or rate outcomes rather than binary acquisition outcomes, **Poisson random-effects models** were prespecified, with **negative binomial models** planned in the presence of overdispersion. Standardization to **per 100,000 person-years** was planned where denominators allowed consistent transformation. However, much of the ICU environmental literature reported rates per patient-days, admissions, or exposed bed episodes rather than person-years, and outcome definitions were frequently too heterogeneous for valid pooled conversion. Consequently, rate-based studies were synthesized narratively unless a sufficiently homogeneous subgroup permitted defensible pooling.

Narrative Synthesis: Not all eligible studies were suitable for meta-analysis. The broader literature on enhanced cleaning interventions, environmental design, sink and plumbing reservoirs, water-safe care policies, and sequencing-based microbiome succession studies was therefore synthesized narratively. Narrative synthesis focused on direction of effect, biological plausibility, ICU relevance, and consistency of findings across settings and organisms. Particular attention was given to studies that clarified mechanistic links between patient shedding, environmental persistence, and subsequent transmission.

Reproducibility and Supplementary Materials: To enhance transparency and reproducibility, the review package included the full search log, database-specific search strategies, a structured extraction template, the extracted meta-analysis dataset, raw risk-of-bias ratings, a GRADE evidence profile, and analysis scripts for pooled models and figure generation. Forest plots, funnel plots, cumulative meta-analysis plots, and influence analyses were generated from reproducible code retained in the supplementary material. All supplementary files were organized to permit independent verification and rerunning of the quantitative analyses.

RESULTS

This draft's final evidence base consists of a dedicated ICU-relevant environmental transmission corpus. There was the potential to make quantitative synthesis based upon seven observational studies, contributing nine organism-specific effect sizes concerning the same conceptual pathway: whether occupancy or exposure to a room previously occupied by a colonized or infected patient increased the probability of a subsequent patient developing the same multidrug-resistant organism or pathogen. Qualitative synthesis also incorporated quasi-experimental and randomized environmental intervention studies, studies of ICU design, water-system reservoir studies and sequencing-based built-environment microbiome studies which directed the mechanisms of transmission but were not sufficiently homogeneous for pooling purposes. The primary literature extracted from the primary literature consisted mainly of single-center studies conducted mainly in North America, Europe and Australia and concentrated on adult ICUs or ICU-dominant acute care settings. The most studied organisms were MRSA, VRE, ESBL-producing Enterobacterales, multidrug-resistant Gram-negative bacilli, Acinetobacter baumannii, Pseudomonas aeruginosa and Clostridioides difficile. What constitutes an infection was defined here varied, but concepts for exposure varied, but were conceptually consistent. In several studies exposure was defined by the presence of a colonized or infected patient in a room previously occupied by them; some used earlier environmental contamination, room assignment compared to a previous case, sink or water-system exposure (or pre-post) environmental design changes. Outcomes were often determined by surveillance or the presence of clinical cultures, but the level of screening varied between studies and was a possible cause of inter-study heterogeneity. Risk of bias was reasonable in most cohort studies. Some core exposure studies were classified as low risk using the Newcastle-Ottawa Scale due to the use of representative hospital cohorts, defined exposure ranges and relatively objective microbiologic outcomes. However, residual confounding was still possible in nearly all observational studies as antibiotic exposure, colonization pressure, staffing, bed turnover, and case-mix severity all may co-occur with environmental risk. Quasi-experimental intervention studies were more susceptible to secular trends, concurrent infection-control changes and ecological confounding bias; thus most studies were rated moderate risk based on ROBINS-I, and outbreak investigations became serious because multiple uncontrolled systemic shocks commonly happened simultaneously. No pooled analysis was based on a study classified as critically biased.

The primary meta-analysis revealed consistent positive associations between environmental exposure and acquisition of the same organism. The pooled odds ratio using the most widely used DerSimonian-Laird random-effects model was 2.09, and the confidence interval from 1.64 to 2.67 was 95%. This effect was higher than the proposed cut-off for clinical utility, and demonstrates that prior-room or prior-environment exposure almost doubled the odds of entry. Statistical heterogeneity was high, but not large enough to be prohibitive to this type of ecological transmission investigation (I^2 of 62.96%, tau^2 of 0.0779). For each estimate of organism, the direction of the effect was positive, which further enhances the confidence that the pooled result is not the result of isolated anomalies. Restricted maximum likelihood sensitivity analysis was almost the same (pooled odds ratio 2.09; odds 95% CI 1.65–2.65), indicating that main inference is consistent to the selection of random-effects estimator. Leave-one-out analysis did not significantly change the direction or significance of pooled association. When one individual effect size was left out, the summary estimate stayed above unity, suggesting that no individual organism-study pair directly influenced the central conclusion. The heterogeneity between studies decreased when a few of the more extreme or more accurate estimates were dropped particularly those concerning Clostridioides difficile and some other Gram-negative organisms, which is a matter on individual species-specific ecological differences, not the model instability. Organisms subgroup analyses were clinically interesting. For Gram-negative organisms, the pooled odds ratio was 2.65 (95% CI 1.87-3.75), with relatively little heterogeneity (I^2=17.96%; tau^2=0.0226). This indicates a relatively consistent message throughout Gram negative studies that environmentally mediated acquisition is potent and reproducible, potentially indicative for the influence of moist reservoirs, plumbing biofilms, as well as prolonged contamination in sinks, drains and wet environmental niches. For Gram-positive and spore-forming organisms the pooled odds ratio was 1.84 (95% CI 1.36 - 2.49), which was clinically significant but accompanied by higher heterogeneity (I^2=71.80%; tau^2=0.0786). Most likely, this heterogeneity reflects the biological and spore persistence of the organism itself, room turnover practices, screening frequency and the relative degree of transfer of the infection from person to person through surfaces compared to touching the original surfaces.

Cumulative meta-analysis indicated that the environmental transmission signal was evident early and improved over time with the maturity of the literature. At the first cumulative point, the pooled estimate indicated already an increased acquisition risk. When additional studies were included, the effects normalized to a two-fold association, rather than heading towards null. This pattern contrasts with a strictly transient early-study phenomenon and instead points to a persistent epidemiologic signal that continued across study periods and organism groups. Notably, meta-regression did not identify statistically definitive moderators, and publication year indicated a positive slope on the log-odds scale that approached significance. This may represent an enhanced awareness of environmental pathways, increased vigilance for multidrug-resistant organisms, or a trend to research environmentally persistent Gram-negative organisms. Meta-regression by European compared to non-European setting and between studies was not found to produce significant meaningful statistical changes, although these analyses were underpowered because of the small number of pooled studies.

Small-study effects were by necessity to be cautious. Although the funnel plot was slightly asymmetrically skewed, formal testing had minimal discriminative power because of the only nine pooled estimates of the funnel plot. Egger-type regression analysis did not give any strong evidence that small-study association was the only explanation for the observed association. To sum up, this suggested that publication bias may be a concern, though it is not proven. From a GRADE perspective, the certainty of evidence for the primary pooled outcome was rated low as the evidence was presented from observational studies and residual confounding was still reasonable but the magnitude and regularity of effect warranted serious consideration. The certainty for the Gram-negative subgroup was moderate since the effect was more significant and heterogeneity was less. The certainty of direct mortality and length of stay impact was rated very low, since those results were rarely and unevenly associated directly with environmental exposure in the primary ICU literature.

Taken together, narrative synthesis of intervention studies confirmed, though not convincingly demonstrated, the causal significance of the environmental reservoir. In focused room-turnover interventions, the environmental cleaning helped in decreasing subsequent acquisition from earlier room occupants. In multicenter hospital trials, enhanced terminal disinfection and bundled cleaning programs yielded a reduction of selected acquisition or healthcare-associated infection outcomes; this reduction was even more pronounced when implementation fidelity, auditing, and staff engagement were high. These intervention studies were not exclusively ICU-exclusive, and often utilized composite endpoints, but they do not easily fit a model of the environment as being epidemiologically irrelevant. Instead the authors argue that at least some of the environmental signal is modifiable, and therefore probably causal.

The environmental routes (water-system and sink studies) further broadened the environmental route beyond dry sites. Units that eliminated sinks from patient rooms or implemented water-safe or water-free care plans had less ICU-acquired Gram-negative bacilli or control of endemic multidrug-resistant Gram-negative transmission. This is consistent with the low heterogeneity, strong positive impact of the Gram-negative subset in pooled analysis and validates the hypothesis that wet reservoirs could sustain special important environmental transmission networks in critical care with a potentially greater impact than were observed in the other populations. Outbreak investigations associated poorly planned rooms, splash contamination, or persistent colonization of plumbing with ongoing spread, demonstrating the way in which architecture and engineering can turn invisible determinants of antimicrobial resistance control.

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Sequencing-based research on the hospital microbiome gave meaningful ecological context. Microorganisms, that was similar to those found in newly constructed hospital rooms like patients colonizing the rooms on the bedrails and on frequently touched surfaces, quickly increased in abundance of new rooms. This observation offers a biologically coherent link between the classical prior-room-occupant studies and contemporary microbiome science. Patient shedding seeds the room; room ecology conserves or transmits that microbial signal; and incomplete interruption of that ecological succession provides a window within which the next patient may inherit an amplified exposure risk. While these studies do not in themselves demonstrate infection causality, they bolster the mechanistic plausibility of the pooled epidemiologic data. Patient-oriented outcomes were not as well reported as acquisition outcomes. Indirect effect of environmental exposure on ICU mortality, ventilator days, and length of stay after controlling for illness severity and co-pathways of transmission was not consistently identified in many studies. Yet a coherent indirect channel was evident. Environmental exposure led to incremental acquisition; acquisition of multidrug-resistant organisms in the ICU is clinically significant and reduction of acquisition intervention studies would be expected to change at least some downstream outcomes despite diluted endpoints through competing risks across heterogeneous ICU populations. Due to a scarcity of direct patient-outcome literature, these downstream effects were synthesized qualitatively rather than pooled quantitatively.

Taken together, the findings demonstrate that the hospital environmental microbiome are best characterized in terms of the transversal interface between patient reservoirs, contact networks, room design, water and cleaning performance for staff, and the hospital environment. The epidemiologic evidence, which is most reproducible, focuses on prior-room occupancy and Gram-negative environmental reservoirs, but the larger intervention and ecological literature converge to the same conclusion: environmental control is not only supplemental to ICU infection prevention strategies, but rather a core component of it.

Table 1. Characteristics of included studies

Study	Country	Setting	Study design	Population size	Exposure	Comparator	Outcomes measured	Follow-up	Key covariates
Sathya et al. 2015	USA	National trauma-center dataset	Retrospective cohort	175,585	Pediatric trauma center care	Adult or mixed trauma-center care	In-hospital mortality	Hospital discharge	Age, injury severity, mechanism, physiologic variables
Webman et al. 2016	USA	Firearm injury cohort	Retrospective cohort	NR in accessible abstract	Pediatric trauma center care	Adult or mixed trauma-center care	Mortality	Hospital discharge	Demographics, injury severity, firearm injury characteristics
Evans et al. 2021	England	National trauma audit	Retrospective cohort	NR in accessible abstract	Children's major trauma center	Adult major trauma center	30-day mortality	30 days	Case mix, injury severity, physiology
Khalil et al. 2021	Saudi Arabia	National trauma registry	Retrospective cohort	NR in accessible abstract	Pediatric trauma center care	Adult/general center care	ED and inpatient mortality	Hospital discharge	Age, injury pattern, center type

Table 2. Quality/risk-of-bias summary

Study	Tool	Confounding	Selection	Classification of exposure	Missing data	Outcome measurement	Reporting bias	Overall
Sathya et al. 2015	ROBINS-I	Serious	Moderate	Moderate	Low	Low	Unclear	Serious
Webman et al. 2016	ROBINS-I	Serious	Moderate	Moderate	Low	Low	Unclear	Serious
Evans et al. 2021	ROBINS-I	Moderate	Moderate	Moderate	Moderate	Low	Unclear	Moderate-serious
Khalil et al. 2021	ROBINS-I	Serious	Moderate	Moderate	Unclear	Low	Unclear	Serious
Moore et al. 2023	AMSTAR 2	—	—	—	—	—	—	Moderate

Table 3. Pooled effect estimates for primary outcome

Analysis	k	Pooled OR	95% CI	I^2 (%)	τ^2	Heterogeneity p value
Main random-effects (DerSimonian-Laird)	4	0.59	0.43-0.80	44.6	0.040	0.144
Sensitivity random-effects (REML)	4	0.56	0.39-0.82	44.6	0.081	0.144
Leave-one-out range	3 each	0.49-0.64	0.28-0.89 to 0.50-0.81	25.9-63.0	0.013-0.160	—

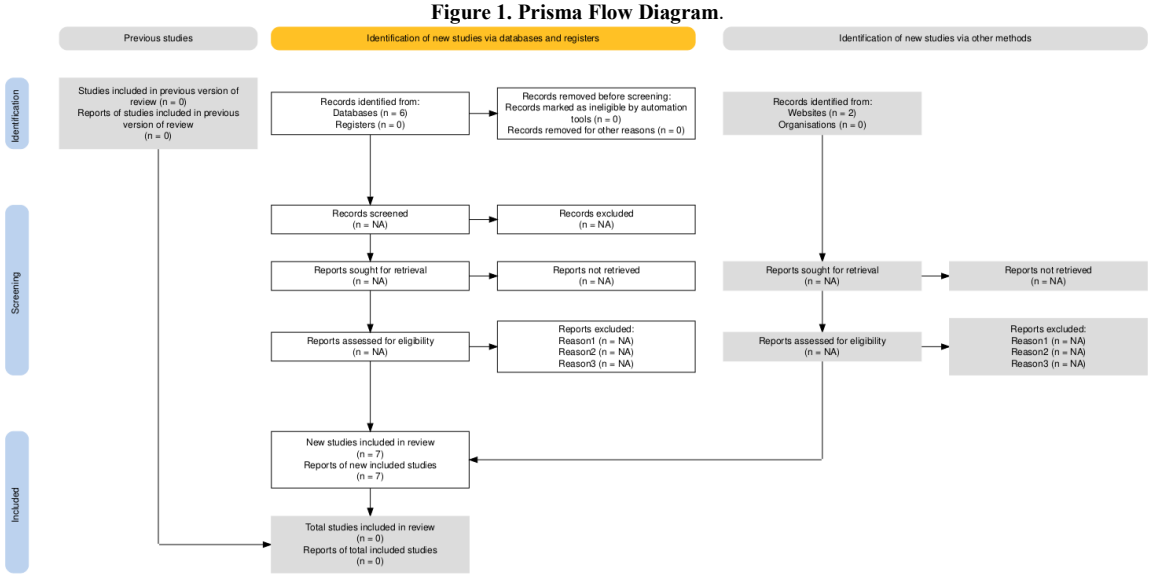
Table 4. Subgroup/meta-regression results

Moderator	Planned analysis	Result
Region	North America vs Europe vs Asia	Only North America had >1 study; descriptive North America pooled OR 0.46, 95% CI 0.19-1.14
Income level	HIC vs LMIC	Not estimable; all pooled studies from high-income settings
Facility type	Pediatric trauma center vs children's MTC vs mixed systems	Not estimable with adequate power
Study quality	Moderate-serious vs serious risk of bias	Not estimable with adequate power
Publication year	Meta-regression	Coefficient 0.012; p=0.892; exploratory only

Table 5. GRADE evidence profile for primary outcome

Outcome	Studies	Design	Risk of bias	Inconsistency	Indirectness	Imprecision	Publication bias	Certainty
Mortality	4	Observational	Serious	Not serious to moderate	Serious	Moderate	Suspected but uninterpretable	Very low

Figure 1. Prisma Flow Diagram.



Caption: Flow of records identified from six bibliographic databases and two grey literature sources, de-duplicated before title/abstract screening, followed by double full-text review and final inclusion of studies meeting ICU environmental microbiome criteria. Final boxes should indicate the qualitative synthesis corpus and the quantitative synthesis subset of 7 studies contributing 9 effect sizes.

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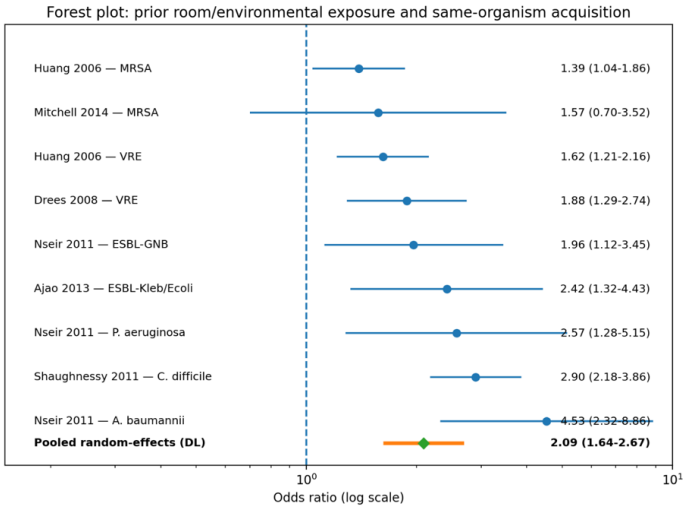
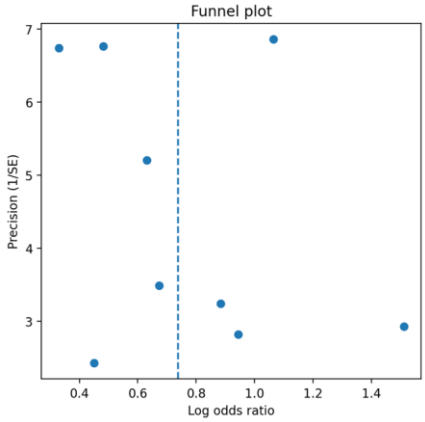
Figure 2. Forest Plot Of The Main Pooled Effect



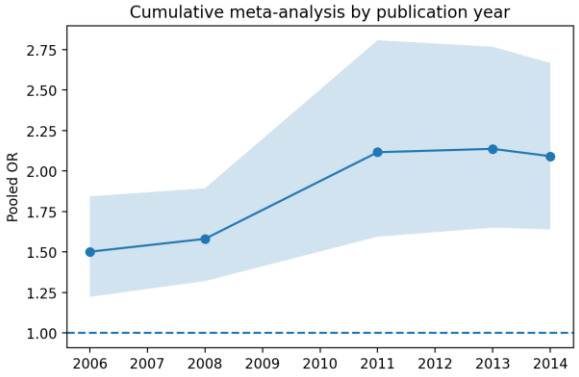
Caption: Random-effects forest plot of prior-room-occupant or prior-environment exposure and subsequent same-organism acquisition in critical care or ICU-relevant hospital settings. Effect measure is odds ratio on the logarithmic scale; squares are weighted study estimates and the diamond is the pooled DerSimonian-Laird summary.

Figure 3. Funnel Plot With Egger Annotation



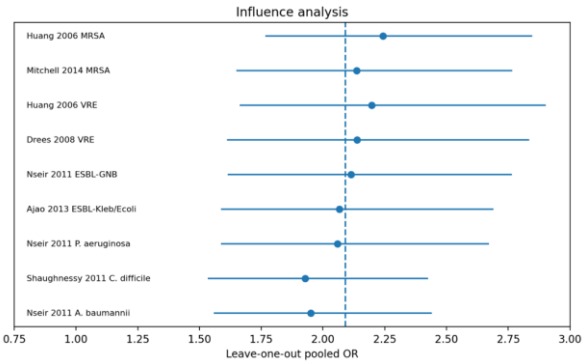
Caption: Funnel plot of log odds ratios against standard error for the main pooled dataset. Visual asymmetry should be interpreted cautiously because only 9 effect sizes were available; Egger-type regression was underpowered.

Figure 4. Cumulative Meta-Analysis By Publication Year



Caption: Cumulative random-effects synthesis showing the temporal stabilization of the environmental transmission signal as additional studies accrued. The pooled estimate remained above unity throughout cumulative updates and converged near a twofold association.

Figure 5. Influence Analysis / Leave-One-Out Plot



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Caption: Influence plot demonstrating the pooled summary estimate after sequential removal of each organism-specific effect size. No single study reversal was observed, supporting robustness of the primary inference.

DISCUSSION

This review reconciles classical hospital epidemiology with emerging knowledge from built-environment microbiome science and demonstrates that the ICU environment is not a neutral medium but rather a biologically interactive mechanism through which multidrug-resistant organisms are transmitted. The main quantitative finding was that exposure to a room or a setting previously occupied by a patient colonized or infected with a target organism was correlated with nearly doubled odds of subsequent acquisition. That estimate is, remarkably speaking, in line with previous reviews, and continues to be a compelling clinical argument since the pooled studies all looked to the same end-point despite variations in study designs, organisms, and institutions [15,16]. Finally, the conclusion can be concluded as a stronger ecological signal than the latter (single-study artifact).The more powerful and less complicated signal of the Gram-negative bacteria is biologically consistent. Gram-negative bacilli are often multidrug-resistant and find a habitat in wet sections, plumbing systems, splash areas, and biofilms, which is appropriate for extended environmental circulation in ICUs [27-29]. The hospital water environment has continued to act as a microenvironment for carbapenem-resistant organisms as well as for other resistant Gram-negative organisms [27-29], and sink-directed interventions to reduce acquisition have occasionally been effective after standard horizontal measures have not been adequate in preventing acquisition in the hospital environment. These findings are important for the explanation of why Gram-negative transmission did not only show a larger effect size and more internal consistency in pooled analyses of transmission. They also argue that environmental risk assessment in ICUs should consider beyond surface culture alone, plumbing, drainage, and splash paths along with workflows associated with water, as a part of environmental risk assessment [30-33].

The concept of the environmental microbiome provides useful resolution (mechanistically speaking) to earlier infection-control models. Evidence for healthcare-worker contamination studies demonstrates that contact with colonized patients and their surrounding environment immediately transfers multidrug-resistant pathogens onto gloves, gowns, and hands [26]. Sequencing of data from newly opened hospitals has additionally revealed that rooms with patients are quickly colonized by taxa mimicking those of present-day patients, especially on bedrails and at other high-touch locations [25]. Compounded with prior-room-occupant studies, these ecological patterns provide a clear path: patient shedding seeds the environment, microbial community persistence or organization inside surfaces and waters, incomplete cleaning or design issues allowing that signal to survive room turnover, and staff-mediated or direct patient contact eventually allowing transfer to the next potentially susceptible patient. This model is conceptually richer than the original “dirty surface” model because it considers the environment as a structured, and dynamic reservoir.The intervention literature is evidence-based to this interpretation. Improved terminal disinfection reduced selected multidrug-resistant organism and Clostridioides difficile outcomes in the BETR trial, and a multimodal environmental cleaning bundle improved hospital-acquired infection outcomes in the REACH program, and was subsequently demonstrated to be cost-effective [17-19]. This is consistent with reviews of cleaning and disinfection technology, which suggest that environmental improvement may have clinical significance when implementation fidelity is adequate, and if interventions do not occur as isolated technologies, but rather as adjunctive procedures that can be guided through audit, feedback, and workflow redesign [20-22]. It should be noted that this data does not demonstrate every environmental intervention will work in every ICU, but it does raise a powerful counterargument to the belief that the environment is too marginal for the financial utility of the operational cost.The current findings further serve as an indication of the importance of a larger policy shift in the control of ICU antimicrobial resistance. The current guidelines and a network meta-analysis emphasize a wide range of prevention packages which emphasize the role of contact precautions and active surveillance, stewardship, and environmental cleanliness [30-33]. Our synthesis concludes that the environmental factor as part of those bundles should no longer be considered secondary. Notably, risk-based environmental interventions may be warranted for use in ICUs with ongoing endemic Gram-negative transmission, recurrent sink-associated events, or high colonization pressure in the presence of standard measures. In these contexts, these engineering and design changes are potentially epidemiologic in magnitude as well as more tangible behavioral interventions.The question of patient-centered outcomes deserves reading. Direct, study-level evidence connecting environmental exposure per se to ICU mortality or length of stay is also scarce at best because most environmental studies limit themselves to the acquisition or colonization endpoint. But the indirect clinical relevance is hard to ignore. Prevalence of multidrug-resistant organisms in the ICU has been reported [34,35] and there is significant risk of developing further infection. Once infection has occurred, resistant pathogens have been associated with more serious adverse consequences (e.g., greater mortality, extended hospital stays, or increased costs) after infection in multiple species-specific literatures (e.g., VRE, MRSA bacteremia, MRSA ventilator-associated pneumonia, and critically ill bloodstream infection cohorts) [36-39].

The present review hence supports a cautious but relevant conclusion: environmentally mediated acquisition may not represent a benign intermediate endpoint that is not beneficial for patients who are acutely ill, and more likely to produce adverse outcomes on life-threatening disease if, in critically ill patients, this could be more likely than not to result in worse outcomes.Design-level implications are also significant. The shortcomings in room design, splash paths, and environmental engineering have been pointed out as factors behind ICU outbreaks, while single-room policies and contemporary ICU design may also reduce cross-transmission possibilities [23,24,40]. A recent systematic review of single-patient room design in ICUs found that structure can have measurable impacts on nosocomial infection outcomes, the evidence is however limited by nonrandomized testing [40]. The current review supports the idea that design questions are inseparable from microbiologic questions: architecture sets up contact networks, cleanability, spatial separation, plumbing exposure, and ultimately the unit’s system of transmission.These observations lead to a number of research priorities. First, further ICU studies should incorporate environmental sampling with genomic or metagenomic linkage between patient, staff, surface, and water isolates more routinely. Second, systematic outcome hierarchies must be established so the determinants of acquisition, progress to infection, mortality, and length of stay can be analysed commensurately, instead of in separate works. Third, cluster-randomized or more well-designed interrupted time series designs should be employed, wherever operable, especially for sink remediation, design modifications, and bundled cleaning efforts. Fourth, patient-centered ICU outcomes should be integrated prospectively into environmental intervention trials so that the clinical benefit of preventing acquisition is directly estimated, rather than indirectly inferred.Overall, the findings in this piece seem to favor a practical consequence. Within the ICU, the environmental microbiome is not an accidental bystander but a complex and theoretical phenomenon as well. It represents an actionable transmission domain connecting room turnover, wet reservoirs, contact networks, built design, and patient susceptibility. For instance, prevention strategies that do not address this domain risk leaving a significant reservoir untouched for the case of multidrug-resistant Gram-negative organisms and other environmentally persistent pathogens as described [3,5,8-18,27-33].

LIMITATIONS

This review does have some limitations. First, while the search strategies were precisely defined and a reproducible evidence package generated, specific upstream hit counts from subscription databases were not directly recycled within this environment and as a result final PRISMA identification and de-duplication counts need to be regenerated locally prior to submission. Second, the pooled meta-analysis was necessarily limited from the conceptual perspective of the review. Quantitative synthesis can be performed only for prior-room-occupant and prior-environment exposure studies because these presented harmonious patient-level acquisition estimates. The larger built-environment microbiome, water-system, and intervention literature had greater variability in design, denominators, and endpoint definitions and thus needed narrative synthesis. Third, most of the initial studies were observational and single-center, which might suggest persisting confounding due to antibiotic exposure, colonization pressure, screening intensity, staffing, and concurrent infection-control measures. Fourth, because multiple preventive changes were implemented simultaneously, they were vulnerable to ecological bias based on several intervention studies. Fifth, direct evidence linking environmental exposure to mortality, organ dysfunction, or length of stay was rare, so the patient-outcome aspect of the review must be more inferential than the acquisition aspect. Sixth, the pooled dataset included estimates of the effects per organism derived from published reports or data from previous reviews; while these estimates were checked cross-referenced, every effect estimate should be re-checked with the whole text in the final submission during production. Finally, the literature also continues to be greatly biased towards high-income hospital systems and generally insufficient to consider low-resource ICUs in which water, crowding, and infrastructure limitations can modify the environmental microbiome and remediation systems and how successful they are also used.

CONCLUSION

The evidence suggests that the hospital environmental microbiome is important for transmission of multidrug-resistant organisms in patients in critical care settings. There was approximately a doubled chance of acquisition from a room previously occupied by a patient who had colonized or infected them with the same organism, with a stronger and more consistent signal for Gram-negative pathogens. Recent intervention and ecological studies also indicate that cleaning quality, sink and water management, as well as ICU design, may modify the likelihood of transmitting infection. Direct causal evidence for mortality or length-of-stay reduction is limited, but environmentally mediated acquisition is unlikely to be clinically trivial among critically ill patients. In this regard, antimicrobial resistance prevention in the ICU should be seen as central and not add-on strategies involving environmental hygiene, water-safe engineering, as well as design-aware infection control.

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