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Bacteria and Bacterial Diseases

# Prevalence and risk factors for *Staphylococcus aureus* colonisation among healthy individuals in low- and middle-income countries: A systematic review and meta-analysis

Thomas E. Locke [a,b,*], Alexander J. Keeley [a,b,c,d], Nicholas Laundy [e], Christopher Keil [f], Jean Hamilton [g], Abdullah Pandor [g], Thushan I de Silva [a,b,c], Thomas C. Darton [a,b]

[a] Division of Clinical Medicine, School of Medicine and Population Health, The University of Sheffield, UK
[b] The Florey Institute of Infection, The University of Sheffield, UK
[c] Vaccines and Immunity Theme, MRC Unit The Gambia at the London School of Hygiene and Tropical Medicine, The Gambia
[d] Clinical Research Department, London School of Hygiene and Tropical Medicine, London, UK
[e] The Royal Hobart Hospital and University of Tasmania, Tasmania, Australia
[f] Department of Medical Microbiology, Sheffield Teaching Hospitals NHS Foundation Trust, Sheffield, UK
[g] Sheffield Centre for Health and Related Research (SCHARR), School of Medicine and Population Health, The University of Sheffield, UK

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SUMMARY

*Background:* *Staphylococcus aureus* is capable of asymptomatic colonisation, which can progress to opportunistic and potentially life-threatening infection. The data on *S. aureus* colonisation in low- and middle-income countries (LMIC) are limited. This systematic review and meta-analysis estimates the prevalence of *S. aureus* colonisation in asymptomatic individuals in LMIC, with secondary objectives of assessing antimicrobial resistance, colonisation risk factors, and the molecular epidemiology of colonising strains.

*Methods:* Articles published up to July 2023 were identified by searching four electronic databases. Studies that presented *S. aureus* colonisation prevalence in healthy individuals from a community setting in LMIC were included. Data extraction was performed independently by two reviewers with disagreement resolved through consensus. Studies were critically appraised using the Joanna Briggs Institute Prevalence tool. Random effects meta-analysis was conducted where appropriate. This study was registered in advance with PROSPERO (CRD42019147780).

*Findings:* A total of 16 610 citations were identified of which 138 studies (59 732 participants) met the eligibility criteria. The majority of studies had a low risk of bias. The pooled prevalence of *S. aureus* colonisation at nose and/or throat sites was 26·4% (95% CI 23·8 - 29·1%). The prevalence of methicillin-resistance in colonising *S. aureus* strains was 15·0% (95% CI: 11·8 to 18·6%), with a higher prevalence observed in Africa compared to Asia and South America (22·5% vs. 13·1% vs. 5·4% respectively). Panton-Valentine leukocidin genes were present in 26·4% (95% CI: 17·1% to 32·8%) of 2531 isolates.

*Interpretation:* While the prevalence of asymptomatic *S. aureus* colonisation in LMIC mirrors that found in high-income countries, there was a higher prevalence of antimicrobial resistance and other virulence factors. Variability in study methods and sparsity of data from many LMIC, underscore the need for a global approach to *S. aureus* surveillance. This will be critical for informing effective infection prevention strategies.



* Correspondence to: Division of Clinical Medicine, School of Medicine and Population Health, The University of Sheffield, Sheffield S10 2RX, UK.
*E-mail address:* mda05tel@sheffield.ac.uk (T.E. Locke).

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**Impact statement**

*Staphylococcus aureus* is the leading cause of bacterial infection related deaths worldwide, with treatment often complicated by antimicrobial resistance (AMR). Asymptomatic colonisation increases the risk of infection, often with an individual's own colonising strain. Community-based transmission perpetuates cycles of colonisation and infection, which intersect with healthcare settings. Targeted interventions, such as topical decolonisation therapies and future vaccines, will play a key role in addressing the global burden of *S. aureus* infection but require robust surveillance data to maximise their efficacy. While epidemiological data of this nature is readily available from high-income countries (HIC), there is a lack of equivalent data from low- and middle-income countries (LMIC). Existing LMIC studies are often limited by a narrow geographical scope or a focus on high-risk populations, such as healthcare workers or those with active infection. The lack of comprehensive global data on *S. aureus* colonisation in healthy individuals from community settings is a critical gap that limits our understanding of this pathobiont and our ability to address its burden of disease.

This systematic review and meta-analysis provides the most comprehensive overview to date of *S. aureus* colonisation in healthy individuals from LMIC, including over 50 000 participants from 40 countries. Our data reveals that *S. aureus* colonisation is a global phenomenon, with similar prevalence in LMIC and HIC. However, colonising strains in LMIC show higher levels of AMR and putative virulence factors, raising concerns about their potential to cause more severe infections. This review identifies significant disparities in the quality and quantity of *S. aureus* colonisation data between LMIC and HIC, highlighting critical gaps in global surveillance.

By mapping *S. aureus* colonisation epidemiology across LMIC, this study provides essential insights into this pathobiont, laying the groundwork for strategies to reduce the global burden of *S. aureus* infection. Future efforts should focus on establishing a standardised, collaborative approach to global *S. aureus* surveillance. This approach will be crucial for capacity building and informing localised infection prevention strategies that ultimately help to address the morbidity and mortality associated with *S. aureus*.

## Introduction

*Staphylococcus aureus* is a human pathobiont which frequently colonises the anterior nares and predisposes to infection, ranging from localised skin infection to systemic, life-threatening complications of bloodstream infection. Host- and pathogen-related factors affecting human colonisation dynamics have been reviewed recently[1] and are key to developing interventions for minimising or preventing *S. aureus* infection risk, especially in higher-risk groups or populations.

The prevalence of human colonisation by *S. aureus* is not well known. Whilst robust *S. aureus* colonisation surveillance data and multinational publications are available, there is bias towards high-income countries (HIC); a more comprehensive description of *S. aureus* colonisation prevalence within and across low- and middle-income countries (LMIC) is required. These data could highlight important epidemiological trends and also pave the way to localised infection transmission and prevention strategies. For example, *S. aureus* is highly clonal with certain strains exhibiting distinct virulence profiles (including antimicrobial resistance [AMR]) and becoming established in specific geographic or environmental niches.[2] Furthermore, there has been a focus on *S. aureus* colonisation related to healthcare exposure, but networks of community transmission in healthy populations likely also play a key role in maintaining circulation.[3] Finally, *S. aureus* colonisation is associated with a number of chronic non-communicable diseases, and the prevalence of these varies but is known to be increasing globally.[4,5]

The primary objective of this systematic review and meta-analysis was to determine the pooled prevalence of *S. aureus* colonisation among healthy individuals in community settings by LMIC. Secondary objectives included determining the prevalence of AMR, identifying risk factors for colonisation and characterising the molecular epidemiology of colonising strains.

## Methods

### *Search strategy and search criteria*

We performed a systematic review and meta-analysis following a protocol registered with PROSPERO (CRD42019147780)[6] in accordance with PRISMA guidelines.[7] We systematically searched electronic databases, including Medline, Scopus, Web of Science, and the Cochrane Library until 31 July 2023 [AJK], for free text, thesaurus terms and combined synonyms relating to *Staphylococcus aureus*, colonisation, and LMIC as defined by the OECD (appendix I).[8] No language or date restrictions were used. All records were downloaded to EndNote (Clarivate Analytics, Philadelphia) for deduplication and initial screening [TL or AJK], with any citations clearly not meeting eligibility criteria excluded.

Eligible studies: 1) presented extractable data relating to the prevalence of *S. aureus* colonisation in healthy individuals in a community setting (i.e. non-healthcare related) in a LMIC, 2), where relevant, presented colonisation data collected before any intervention was performed, 3) were available in the English language, and 4) had the full text available.

Studies were excluded if they: 1) reported data for subjects who were hospitalised, were attending healthcare facilities (unless as part of a healthy review, for example, pregnant women attending antenatal care or people living with HIV attending for antiretroviral medication review), were healthcare workers; 2) did not make it clear that the subjects were asymptomatic; 3) did not report original data; 4) reported data from a single case; 5) did not describe the body site sampled to assess for colonisation; 6) did not use at least one of microbiological culture, targeted antigen or PCR testing to detect *S. aureus*; or 7) only reported methicillin-resistant *S. aureus* (MRSA) prevalence.

Eligible studies were imported to Covidence systematic review software (Veritas Health Innovation, Melbourne,) which was used for all subsequent selection and extraction steps. Eligibility was confirmed by full text review [TL, AJK, NL, or CK]. Studies were included if only a subpopulation met the eligibility criteria, but only subpopulation data was extracted.

### *Data extraction*

Data extraction was performed independently by two reviewers [TL, AJK, NL, CK], with disagreement resolved through consensus or arbitration [TD]. Only original data presented in the publicly available publication or associated supplementary materials were extracted (appendix II). Missing data for binary measures (e.g. sex) were calculated and missing values for the Human Development Index (HDI) were imputed using data from the nearest available year. Otherwise, values were marked as missing. Electronic tools such as digitisers were not used. For longitudinal studies, data from the earliest time-point was extracted. Reviewers did not evaluate their own publications.

*Risk of bias assessment*

We assessed full texts for methodological quality and risk of bias using the Joanna Briggs Institute (JBI) Prevalence Critical Appraisal Tool.[9] Assessments were performed at the time of data extraction. Scoring of nine criteria, for responses including "yes" (one point), "no or unclear" (zero points) and "not applicable" (denominator reduced by one), produced a total score for each study. These are presented as percentages and categorised into three arbitrary levels: high risk of bias as ≤33%, moderate risk 34–66% and low risk > 66%.[10]

*Data synthesis and analysis*

Sampled body sites were categorised as nose and/or throat (NT, see appendix III for a list of NT sites), skin, other single site, or a combination where >1 site sampled without site-specific data reported separately. A single colonisation prevalence figure was included from each eligible study. If a study reported data from multiple distinct body sites, then data relating to NT sites, and specifically the anterior nares were preferentially selected, being the most high-yield site for *S. aureus* colonisation. The compiled dataset including all body sites is termed "all sites". To minimise heterogeneity, a subset of the data were created including only studies reporting colonisation of ≥1 NT site. This "NT only" dataset was used to calculate pooled *S. aureus* colonisation prevalence and associated subgroup, meta-regression, and risk factor analyses. The "all sites" dataset was used to assess risk of bias, AMR and typing analyses as the specific body site sampled was deemed less important for these purposes.

Analysis was performed using R version 4.3.1.[11] Prevalence data (*S. aureus* colonisation, AMR, *pvl* gene presence, and *tst* gene presence) was transformed using the Freeman-Tukey double arcsine method and synthesised using a random effects model with the DerSimonian estimator and associated 95% confidence intervals calculated by the Clopper-Pearson method ('meta'[12] package). Methods for exploring heterogeneity between studies included the $I^2$ statistic, prediction intervals, leave-one-out analysis and externally studentised residuals ('meta'[12] and 'dmetar'[13] packages). Where appropriate, between study heterogeneity in colonisation prevalence was explored through subgroup analysis and meta-regression. Risk factors for *S. aureus* colonisation included variables reported at the individual participant level from more than one study. A pooled prevalence ratio (PR) for each risk factor was calculated using a meta-analysis of binary data ('meta'[12] package). A random effects model was used unless fewer than five studies reported data for a risk factor, in which case a fixed model (Mantel-Haenszel method without continuity correction) was used. MLST typing data were grouped by continent and the proportion of each MLST type was calculated. Strain diversity was quantified using Simpson's Diversity Index (SDI).[14]

*Role of the funding source*

The funder had no role in the study design, data collection, analysis, nor in the writing of the manuscript or the decision to submit for publication.

## Results

We identified 16 610 citations of which 138 studies met the eligibility criteria and were included in further analysis (Fig. 1 and appendix IV).[15–152] The "all sites" dataset included 138 studies with 59 732 participants (appendix V). Most studies assessed *S. aureus* colonisation at a single body site (128 studies, 93%) with NT sites being the most commonly sampled (121 studies, 88%).

The "NT only" dataset included 121 studies comprising 54 527 participants (91% of participants represented in the "all sites" dataset). Publication year ranged from 1975 to 2023, with 114 studies (94%) performed since 2005. Most studies used a cross-sectional design (117 studies, 97%). Studies were conducted in four continents, with 68 studies (56%) from Asia, 41 studies (34%) from Africa, 11 studies (9·1%) from South America and one study (0·8%) from Oceania. All publications reported data from a single country only, comprising 40 distinct countries. Five countries (India, Nigeria, Iran, China, and Brazil) accounted for 56 publications (46%) and 59% of participants (32 361 of 54 527).

The median percentage of male participants was 52% although sex was only reported in 84 studies (69%). Studies described colonisation by age group including 44 adult only studies (36%), 41 children only (34%), 29 adult and children (24%), and seven in which age-related data were not reported (5·8%). Study populations were diverse in setting and demography, but most commonly included: being resident within a defined geographical region (32 studies, 26%), attending healthcare facilities as part of a health review (22, 18%), school children (17, 14%), university students (14, 12%), specific occupations (13, 11%), and childcare attendees (10, 8%).

Across the 121 studies in the "NT only" dataset there were 124 NT sites sampled, as three studies sampled both anterior nares and oropharynx. A single NT site was selected from each publication which resulted in the following distribution of sites sampled: anterior nares (104 sites, 86%), nasopharynx (9, 7%), oropharynx (3, 2·5%), oral rinse (2, 1·7%), and combinations of NT sites (3, 2·5%). Bacterial culture was the most common method used for the detection of *S. aureus* colonisation (92 studies, 76%), followed by a combination of culture and molecular (28, 23%) or molecular methods alone (one study, 0·8%). Skin sites were sampled in 12 studies including 2264 participants (appendix VI).

Methodological quality assessment of the 138 included studies ("all sites") demonstrated a low risk of bias (120 studies, 87%) with the remaining 18 studies (13%) categorised as moderate risk (appendix VII). Performance tended to be poorer in domains related to participant selection. Specifically, 43 studies (31%) scored zero points for lower quality recruitment methods such as convenience sampling or due to insufficient methodological detail. Similarly, sample size was deemed inadequate in 30 studies (22%), with limited description of the population studied in 52 studies (38%).

From 121 studies included in the "NT only" dataset, 12 933 of 54 527 subjects were colonised with *S. aureus* in the nose and/or throat. The pooled prevalence was estimated at 26·4% (95% CI 23·8 – 29·1%) (Fig. 2). Between study heterogeneity in *S. aureus* colonisation prevalence was high ($I^2$=98·0%, 95%CI 97·8 – 98·1%, prediction interval of 4·3–58·2%). However, sensitivity analysis yielded consistent results with no notable change in the pooled *S. aureus* colonisation prevalence estimate (appendix VIII).

Factors explored in subgroup analysis of NT *S. aureus* colonisation prevalence included continent the study was performed in, specific NT site sampled, participant age group, HDI category, laboratory methods for *S. aureus* detection, and type of population sampled. Significant differences were observed for continent ($p < 0{\cdot}001$), NT site sampled ($p=0{\cdot}036$) and laboratory method ($p < 0{\cdot}001$; appendix IX). The highest prevalence was found in South America (36·7%, 95% CI: 24·6–49·8%), followed by Africa (31·0%, 95% CI: 25·7–36·7%), while studies from Asia (22·4%, 95% CI: 19·5–25·4%) reported lower prevalence rates (Fig. 3). Combined culture and molecular methods detected a higher prevalence (27·2%, 95%CI: 23·2–31·4%) of *S. aureus* colonisation compared to culture alone (26·4%, 95% CI: 23·2–29·7%). A lower prevalence of *S. aureus* colonisation was detected from nasopharyngeal sampling (14·1%, 95% CI: 6·0–24·9%) compared to the anterior nares (27·0%, 95% CI: 24·3–29·8%), although the number of participants in the nasopharyngeal sampling group was limited.

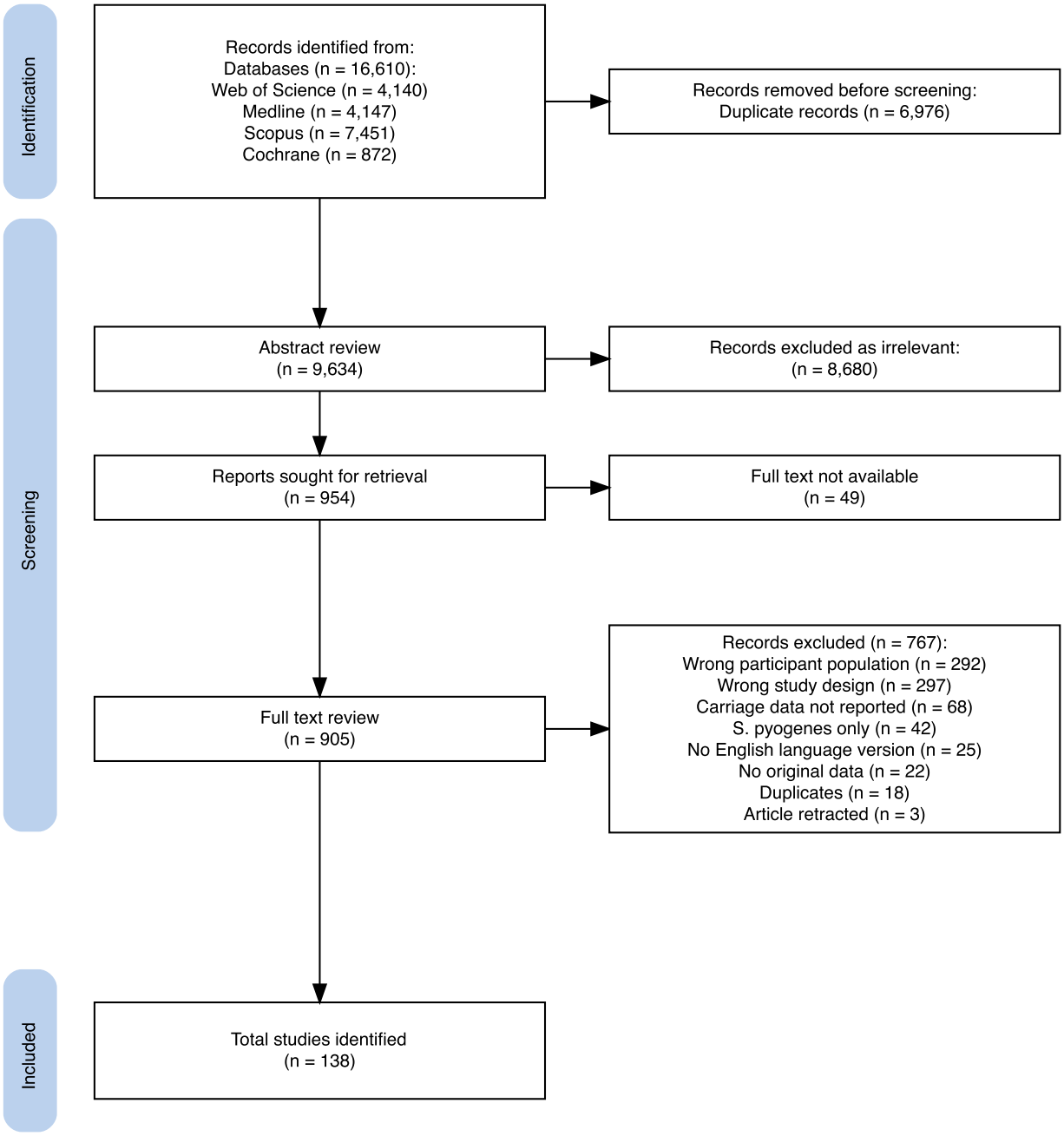


**Fig. 1.** PRISMA flowchart (aside from the 'Included' step, all numbers reported here include studies relating to *S. aureus* and *S. pyogenes).*

Using multiple meta-regression we explored the relationship between *S. aureus* colonisation prevalence at NT sites and study world sub-region, year of study, laboratory methods for *S. aureus* detection, HDI score, and specific NT site sampled (Table 1). Heterogeneity between studies remained high ($I^2$ 97·6%), although variance in colonisation prevalence was comparably low ($tau^2$ 0·02). East Asian countries, namely China, exhibited significantly lower *S. aureus* colonisation prevalence compared to Sub-Saharan African countries (coefficient −0·2341, p=0·003). Lower prevalence was also estimated when sampling the nasopharynx compared to the anterior nares (coefficient −0·2105, p=0·0019). Both findings remained significant after adjusting for study year, HDI, and laboratory method of *S. aureus* detection. Missing data necessitated the exclusion of sex and average age as covariates in the meta-regression model, with data only available from 84 studies (69%) and 50 studies (41%), respectively. However, no significant relationship between either variable and colonisation was found in univariate analysis (average age: coefficient −0·0017, p=0·31; proportion of male participants: coefficient 0·53, p=0·96).

Most studies (85%) described AST data, but these were not extractable from eight of 117 studies due to limitations in reporting, including the absence of raw data. Testing methods included phenotypic techniques such as disc diffusion and gradient strip testing (52 studies, 47%), molecular methods (4 studies, 3·7%), or both (52 studies, 47%). Overall, 56 studies (51%) included molecular testing, with all testing for MRSA by PCR amplification of the *mecA* gene. Additionally, six studies (11%) tested for *mecC*, and one study (1·8%) for VRSA using *vanA* and *vanB* gene amplification. Among the 104 studies using phenotypic methods, 79% (82 studies) cited Clinical and Laboratory Standards Institute guidance.

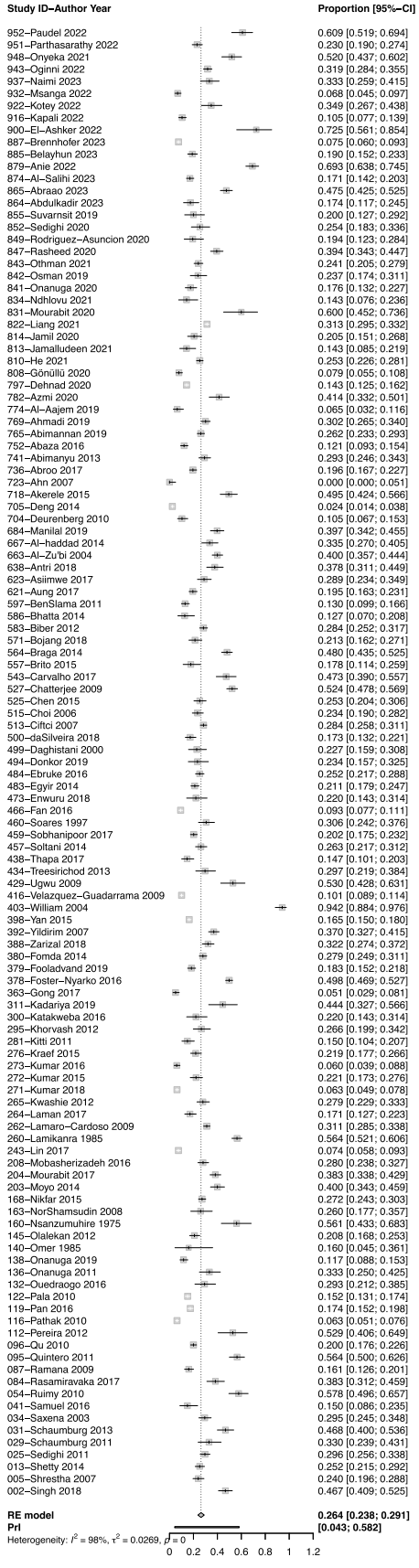


**Fig. 2.** Forest plot showing the pooled prevalence of *S. aureus* colonisation from nose and/or throat sites.

Pooled prevalence of *S. aureus* resistance to 14 antimicrobials is detailed in Table 2; the majority of *S. aureus* isolates were resistant to penicillin (90%), whereas resistance to gentamicin, mupirocin, rifampicin, or linezolid was uncommon (from 0·8 to 9·4%).

Nearly all studies where AST data were reported (103 studies, 95%) included testing for methicillin resistance, using phenotypic methods (47 studies, 46%), molecular methods (four studies, 4%), or both (52 studies, 50%). Among studies using molecular methods, only 25 (45%) tested all isolates while the remainder used it for confirmatory testing. Overall, 12 234 isolates were tested for MRSA, yielding a pooled prevalence of 15·0% (95% CI: 11·8 to 18·6%). Subgroup analysis and meta-regression were used to further explore the relationship between MRSA prevalence and continent the study was performed in, AST methodology, and study end year. MRSA prevalence varied by continent with higher prevalence in Africa (22·5%, 95%CI: 13·3–33·3%), compared to Asia (13·1%, 95%CI: 9·9%–16·8%), and South America (5·4%, 95%CI: 2·9–8·4%) (appendix X). Subgroup analysis of MRSA prevalence by study country is presented in Fig. 4. The lower prevalence of methicillin resistance in Asian countries remained statistically significant in a meta-regression model that included AST testing methodology and study end year (appendix X). Since 2000, African and Asian studies have shown an increasing trend in MRSA prevalence (Africa: R=0·39, p=0·026, Asia: R=0·31, p=0·018) although this was not seen in studies from South America (R=0·041, p=0·92; appendix X).

Vancomycin resistance was assessed in 4 385 isolates from 43 of 109 studies. All studies used phenotypic methods, and one also used molecular techniques. The pooled prevalence estimate for VRSA was 0·5% (95%CI 0 to 1·8%). The prevalence of VRSA was highest in African countries (4·1%) although this was not statistically significant (appendix XI).

We performed a meta-analysis to explore potential risk factors for *S. aureus* colonisation in LMIC using the "NT only" dataset (Table 3). Aside from participant sex, this analysis was hindered by the limited data available for each risk factor resulting in small sample sizes. Household animal contact (PR 1·35, p=0·03, fixed effects model) and attendance at childcare facilities (PR 1·38, p < 0·001, fixed effects model) were significantly associated with *S. aureus* colonisation at NT sites. There was a non-significant trend towards less colonisation among smokers (PR 0·73, p=0·14, random effects model). No difference in colonisation was observed between males and females (PR 1·03, p=0·58, random effects model; appendix XII).

Panton-Valentine leukocidin is a *S. aureus* toxin putatively associated with virulence and transmission. In 37 studies comprising 2 531 isolates, the pooled prevalence of *pvl* genes was 24·6% (95% CI: 17·1% to 32·8%). Additionally, 13 studies comprising 1 181 isolates assessed the presence of toxic shock syndrome toxin (*tst*) gene, with a pooled prevalence of 10·8% (95% CI: 4·4% to 19·1%).

Multi-locus sequence typing (MLST) was the most frequently reported *S. aureus* typing method. A total of 129 unique MLST types were identified from 911 bacterial isolates across 20 studies. Only 14 countries reported MLST data, with China accounting for nearly a third (279 of 911 isolates, 31%). The top 20 most common MLST types from each continent are presented in Fig. 5. Diversity was higher in studies performed in Africa and Asia compared to South America, although this difference was not statistically significant (SDI: 0·92, 0·93, and 0·88, respectively, p=0·36). Although there was strain diversity within each continent, types ST1, ST5, ST15, ST188, and ST30 were among the most commonly encountered in all three continents. In contrast, several types were only associated with a single continent, such as ST22 and ST398 in Asia and ST1223 in South America.

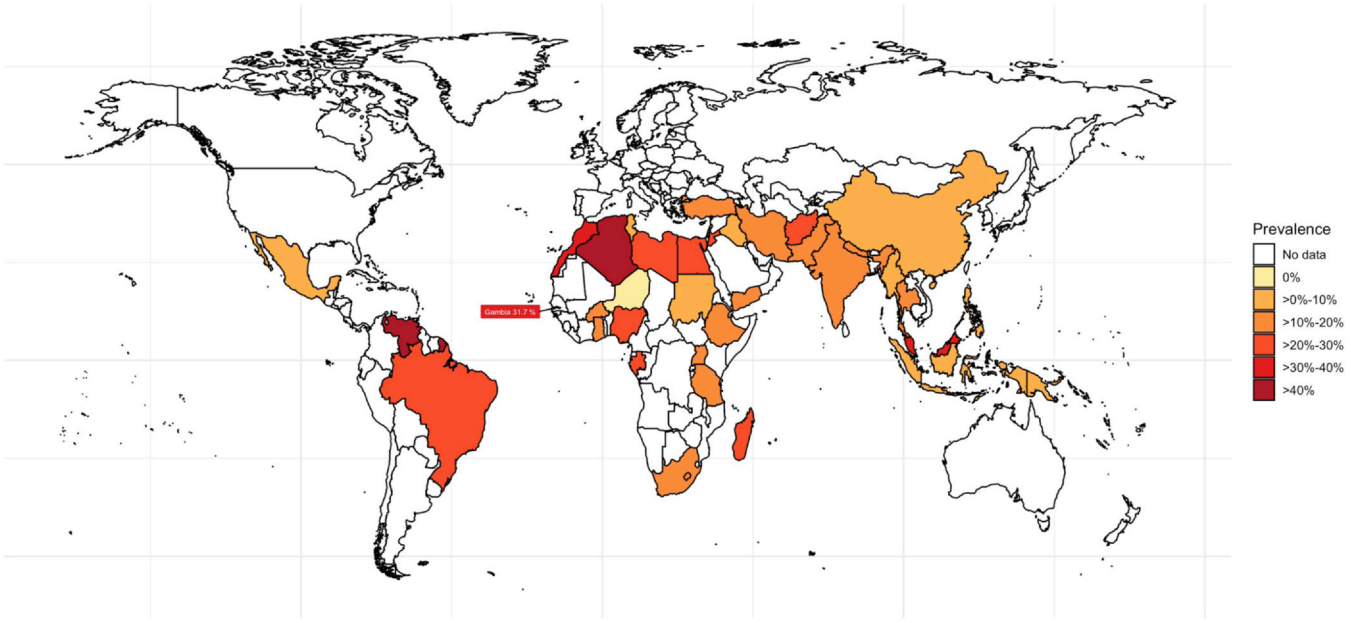


**Fig. 3.** World map with pooled prevalence of *S. aureus* colonisation by country from nose and/or throat sites.

## Discussion

In this systematic review of healthy community populations living in LMIC, including over 50 000 participants from 40 LMIC, we estimate the prevalence of *S. aureus* colonisation at nose and/or throat sites to be 26·4%. Notably, we found a high prevalence of bacterial factors putatively associated with virulence, including methicillin-resistance and PVL-toxin presence. Colonisation is a major risk factor for invasive infection, and with *S. aureus* being a leading cause of death due to bacterial infection frequently complicated by AMR[153,154], the data presented in this review highlight the critical need for public health interventions suitable to LMIC community settings.

Our estimation of *S. aureus* colonisation prevalence specifically addresses healthy individuals in community settings. Despite the ubiquity of *S. aureus*, global data from these settings are scarce. Instead, most studies focus on those with infection, or populations at higher risk of exposure such as healthcare contact. Previous meta-analyses from individual LMIC report *S. aureus* colonisation prevalence between 21·2% and 30·9%.[155,156] While multi-country estimates have been generated, these are limited by methodological weaknesses that make direct comparison difficult. These include a focus on MRSA only, limited patient populations, uncertainty regarding sites sampled, incorporating estimates from studies with less strict inclusion criteria, and the inclusion of infection cases or non-LMIC settings.[157–160] In contrast, large-scale *S. aureus* prevalence studies are more common in HIC. In Europe, the pooled nasal colonisation prevalence from nine countries was 21·6% (range 12·7–29·4%, 32 206 samples).[161] Similarly, a nationwide study in the US estimated nasal colonisation prevalence at 28·6% (9 004

**Table 1**
Multiple meta-regression of *S. aureus* colonisation prevalence at nose and/or throat sites.

| Factor | Coefficient | 95% CI (Coefficient) | OR | P-value |
|---|---|---|---|---|
| Sub-region | | | | |
| Sub-Saharan Africa (reference) | NA | NA | 1.0000 | NA |
| Northern Africa | 0.0068 | −0.1521 to 0.1657 | 1.0068 | 0.9328 |
| Southern Asia | −0.1155 | −0.2245 to −0.0066 | 0.8909 | **0.0379** |
| South-Eastern Asia | −0.1012 | −0.2517 to 0.0493 | 0.9037 | 0.1852 |
| Eastern Asia | −0.2341 | −0.3868 to −0.0813 | 0.7913 | **0.0030** |
| Western Asia | −0.1278 | −0.2706 to 0.015 | 0.8800 | 0.0788 |
| Latin America and the Caribbean | 0.0033 | −0.1571 to 0.1636 | 1.0033 | 0.9678 |
| Melanesia | −0.2165 | −0.5607 to 0.1277 | 0.8053 | 0.2151 |
| Other | | | | |
| Study end year | −0.0035 | −0.0084 to 0.0015 | 0.9965 | 0.1667 |
| HDI | 0.0165 | −0.4292 to 0.4621 | 1.0166 | 0.9417 |
| Laboratory detection method | | | | |
| Culture (reference) | NA | NA | 1.0000 | NA |
| Culture & molecular | −0.0357 | −0.1209 to 0.0496 | 0.9649 | 0.4084 |
| Molecular | −0.1298 | −0.4887 to 0.2291 | 0.8783 | 0.4749 |
| Nose/throat site | | | | |
| Anterior nares (reference) | NA | NA | 1.0000 | NA |
| Nasopharynx & throat | 0.1220 | −0.2329 to 0.477 | 1.1298 | 0.4970 |
| Anterior nares & throat | 0.1793 | −0.0696 to 0.4282 | 1.1964 | 0.1562 |
| Nasopharynx | −0.2105 | −0.3414 to −0.0797 | 0.8102 | **0.0019** |
| Oral rinse | 0.1739 | −0.0877 to 0.4356 | 1.1900 | 0.1903 |
| Oropharynx | −0.1626 | −0.3822 to 0.0569 | 0.8499 | 0.1449 |

Statistically significant results (P < 0.05) are indicated in bold.
HDI = Human Development Index.

**Table 2**
Pooled prevalence of antimicrobial resistance to 14 antimicrobials for *S. aureus* isolates from all body sites.

| Antimicrobial | Number of studies | Number of isolates | Proportion resistant | 95% CI |
|---|---|---|---|---|
| Chloramphenicol | 26 | 2752 | 0.1073 | 0.0556 - 0.1718 |
| Clindamycin | 52 | 5570 | 0.1838 | 0.1199 - 0.2567 |
| Co-trimoxazole | 60 | 5648 | 0.2237 | 0.1537 - 0.3018 |
| Doxycycline | 9 | 530 | 0.2858 | 0.0876 - 0.5367 |
| Erythromycin | 68 | 6470 | 0.2954 | 0.2328 - 0.3618 |
| Gentamicin | 58 | 4870 | 0.0938 | 0.0629 - 0.1293 |
| Linezolid | 21 | 2337 | 0.0082 | 0 - 0.034 |
| Methicillin (MRSA) | 103 | 12 234 | 0.1502 | 0.1177 - 0.1857 |
| Mupirocin | 7 | 1130 | 0.0320 | 0 - 0.1365 |
| Penicillin | 44 | 4501 | 0.9031 | 0.8245 - 0.9625 |
| Rifampicin | 27 | 2762 | 0.0310 | 0.0128 - 0.0549 |
| Teicoplanin | 13 | 1339 | 0.0013 | 0 - 0.0481 |
| Tetracycline | 42 | 4693 | 0.2400 | 0.1832 - 0.3014 |
| Vancomycin (VRSA) | 43 | 4385 | 0.0053 | 0 - 0.0181 |

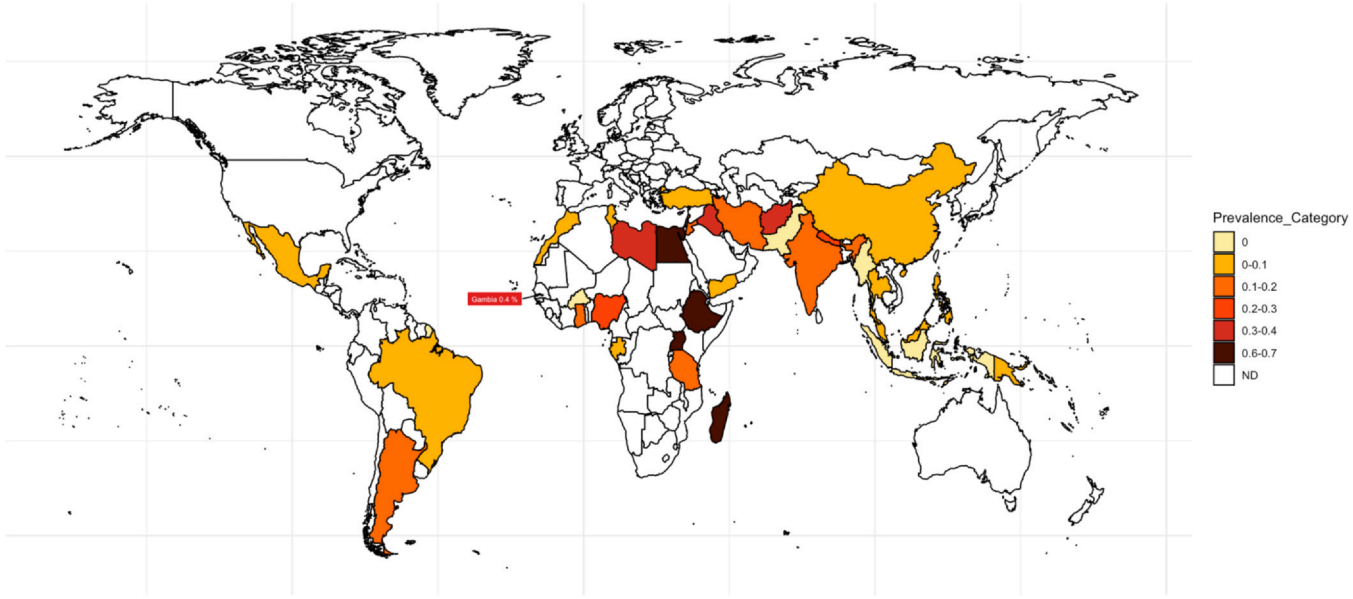


**Fig. 4.** World map with pooled prevalence of methicillin resistance in colonising *S. aureus* isolates by country from all body sites.

**Table 3**
Pooled prevalence ratio for eight risk factors for *S. aureus* colonisation at nose and/or throat sites.

| Risk factor | Studies | Cases | Total | Model | Prevalence ratio | 95% CI | P-value |
|---|---|---|---|---|---|---|---|
| Male sex | 36 | 10 037 | 19 919 | Random | 1.0303 | 0.9254 - 1.1472 | 0.5758 |
| Hospitalisation (12 months) | 11 | 1244 | 9317 | Random | 1.1977 | 0.8498 - 1.6879 | 0.2686 |
| Antibiotics (6 months) | 9 | 2530 | 8090 | Random | 1.1988 | 0.7754 - 1.8533 | 0.3653 |
| Smoker (active) | 5 | 1173 | 5183 | Random | 0.7308 | 0.4529 - 1.1793 | 0.1430 |
| HCP (household) | 5 | 366 | 2042 | Random | 0.9175 | 0.6880 - 1.2235 | 0.4529 |
| Animal (household) | 2 | 287 | 1540 | Fixed | 1.3523 | 1.0333 - 1.7698 | **0.0279** |
| HIV | 3 | 485 | 1146 | Fixed | 0.9293 | 0.7206 - 1.1985 | 0.5722 |
| Childcare (attends) | 2 | 259 | 853 | Fixed | 1.3766 | 1.1454 - 1.6545 | **0.0007** |

Statistically significant results (P < 0.05) are indicated in bold.
HCP = Healthcare professional.
HIV = Human immunodeficiency virus.

samples).[162] These two studies underscore the stark disparity in the quantity of *S. aureus* data available from HIC and LMIC, with a combined sample size of 41 210, compared to 54 527 in the 121 studies from 40 countries included in this systematic review.[161,162]

As a pathobiont, colonising strains of *S. aureus* are often implicated in opportunistic, invasive infections, emphasising the importance of understanding resistance profiles to guide antimicrobial selection. While we found that nearly all colonising *S. aureus* isolates exhibited resistance to penicillin, the widespread and high levels of resistance to commonly used anti-staphylococcal agents such as macrolides, clindamycin, tetracyclines, and co-trimoxazole was frequent and concerning. These agents are often used first-line for managing skin and soft tissue infection (SSTI) and are widely available and affordable. In contrast, resistance rates among colonising *S. aureus* isolates in Europe are notably lower. For example, erythromycin (1·6% to 16·5%), tetracycline (1·8% to 7·2%), and co-trimoxazole (0% to 1%).[161] We also found a high prevalence of MRSA among colonising isolates in LMIC (15%), markedly surpassing that typically observed in HIC (0–2% in European and US populations).[161,162] Particularly concerning were the high MRSA prevalences found in the Africa and Asia regions, with increasing trends since 2000. This is in contrast to previous reports from the Americas,

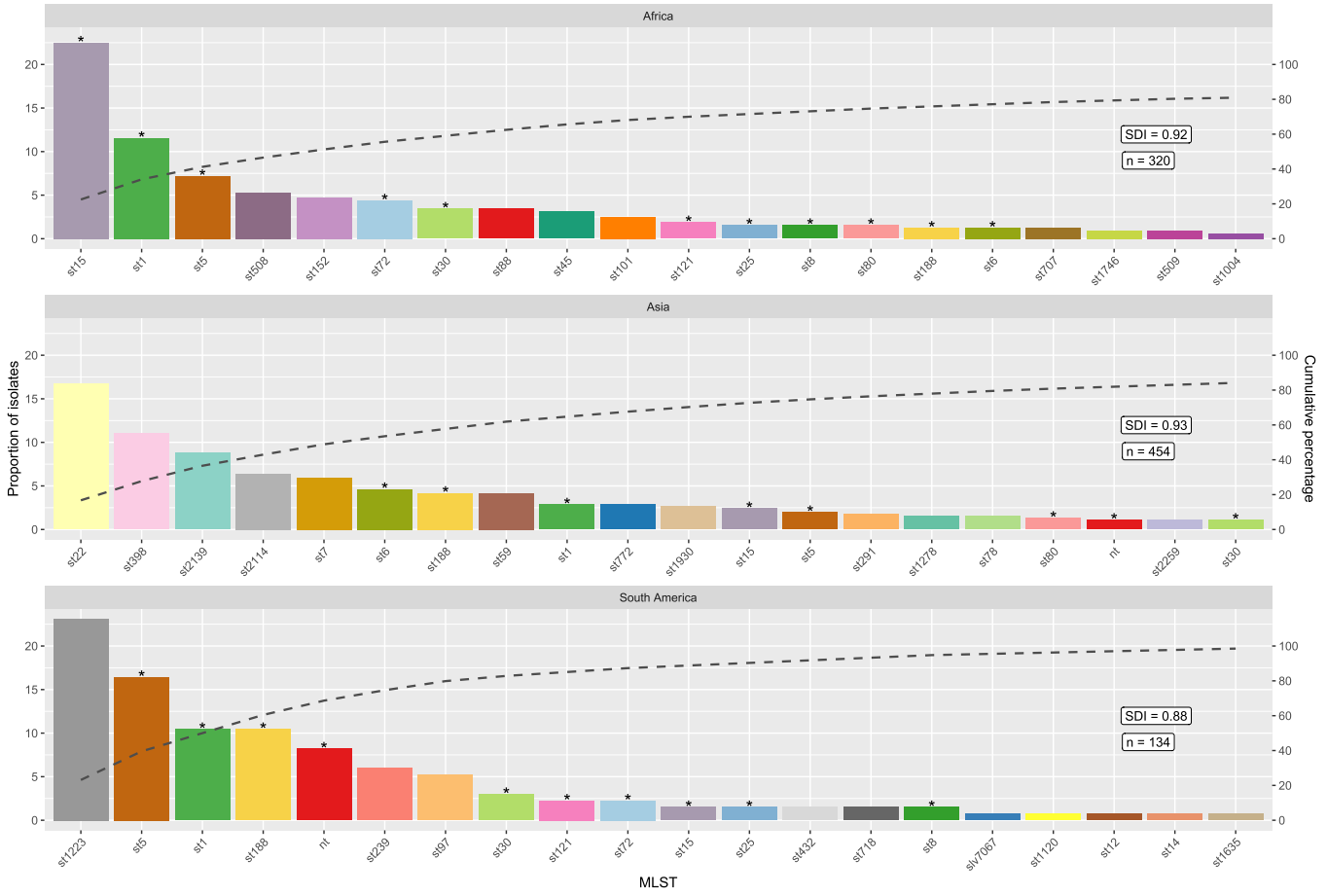


**Fig. 5.** Top 20 most common MLST as proportions of all isolates for three continents (Oceania excluded as only three isolates underwent MLST). "*" denotes an MLST identified in two or more continents. SDI = Simpson's Diversity Index.

Europe and Asia-Pacific where MRSA prevalence amongst clinical isolates appears to have peaked in the mid-2000s.[163] The pooled prevalence of VRSA was 0·5%, with the highest levels in African countries. Whilst the global prevalence of VRSA is low, it remains a clinical concern, particularly for LMIC where access to alternative antimicrobials may be limited.[164] Overall, our findings demonstrate higher rates of antimicrobial resistance among colonising *S. aureus* strains in LMIC compared to HIC. Studies from HIC settings demonstrate that infection caused by drug-resistant strains is associated with increased mortality and healthcare costs[165,166], and so ongoing surveillance is crucial to systematically monitor local *S. aureus* epidemiology and to inform patient management and public health strategies.

We found that household animal exposure and attendance at childcare facilities were significantly associated with *S. aureus* colonisation, consistent with previous reports from HIC settings.[3] However, other commonly implicated factors found not to be associated in our review included male sex, recent antibiotic use or hospitalisation. This is likely due to limited data availability, substantial heterogeneity across the included studies and diversity within each risk factor. For example, differing types and duration of healthcare exposure. While we note some trend towards reduced *S. aureus* colonisation in association with smoking, the nature of this relationship remains unclear, with conflicting results from previous studies.[167,168]

PVL toxin has been associated with SSTI, particularly amongst community-acquired cases of MRSA infection.[169] In this meta-analysis, approximately a quarter of colonising *S. aureus* isolates were found to be PVL-positive. In general, there is limited data on the prevalence of PVL amongst colonising strains as most work has explored a virulence role during infection. While PVL genes appear to be more commonly found in infecting *S. aureus* strains compared to colonising isolates, the exact role in infection is still debated.[169,170] Where data is available from HIC, PVL is infrequently detected in colonising *S. aureus* isolates (< 1% in European studies).[171,172] A higher prevalence of PVL in colonising MRSA has been reported in the US likely due to the success of the PVL-positive USA300 clone, particularly in community settings. In contrast, the *tst* gene, associated with toxic shock syndrome, was identified in 10·8% of colonising isolates in this review, and a similar prevalence has been previously reported in European studies (17·5 to 32·5%).[171,172]

Multi-locus sequence typing identified 129 different sequence types from over 900 *S. aureus* isolates. Common types identified in multiple LMIC and across different continents included ST1, ST5, ST15, and ST30. However, these types are frequently reported in association with infections worldwide and are not unique to LMIC,[2,173] highlighting the capacity of *S. aureus* clones to spread and become established in diverse settings. For instance, in the current review, ST15 was frequently isolated in Africa, Asia and South America and is also associated with bacteraemia throughout Europe.[173–175] The ST22 strain was one of the most frequently identified types in this review but only in Asian studies. However, this strain is globally distributed and the observed discrepancy may be explained by the high number of isolates sequenced from Asia, specifically in China.[176] ST1223 was exclusively identified in South America but this MLST type has recently been linked with *Staphylococcus argenteus* which is a newly described member of the *S. aureus* clonal complex.[177] In general, there is a paucity of data that

characterises the molecular epidemiology of colonising *S. aureus* isolates, particularly in LMIC settings. Instead, studies have tended to focus on infecting strains and frequently those with methicillin resistance. The typing data presented here should be interpreted cautiously for three reasons. Firstly, the relevance of typing colonising *S. aureus* strains to infection is unclear. Whilst the vast majority of invasive infection is due to colonising strains, infection is relatively uncommon and therefore only a small proportion of colonising isolates will ultimately cause infection. Secondly, the establishment of dominant strains in an environmental or geographical niche is an evolving and dynamic process. Given the longitudinal nature of this meta-analysis across multiple decades, it is difficult to accurately infer the current global molecular profile of *S. aureus*. Finally, variability in typing methods and laboratory techniques can limit direct comparisons and data synthesis.

Several limitations to this study should be acknowledged. Firstly, the study populations were highly heterogeneous, encompassing a globally diverse population from multiple geographical and sociocultural regions. This heterogeneity also extended to the anatomical sites sampled. Additionally, laboratory techniques including use of molecular methods and the use of internationally-defined standards for *S. aureus* detection were variably used and described. Our aim was to capture a global snapshot of *S. aureus* in LMIC reflecting the natural diversity of colonisation while recognising that colonisation is often a transient state.[1] To mitigate this, we employed strict eligibility criteria ensuring participants were asymptomatic and not from traditionally higher risk groups. Methodologically, we used a random-effects model and meta-regression analysis to explore the high levels of heterogeneity. Secondly, although this review is broad in scope, the final dataset only includes data from ~25% of all LMIC. This may in part be due to the previously mentioned eligibility criteria but is also reflective of the gaps in *S. aureus* surveillance in many LMIC. Whilst five countries account for a large proportion of the data, they are also some of the world's most populous nations, suggesting that this analysis may be representative at a population level, albeit not geographically. Thirdly, the exclusion of non-English publications may have led to under-representation of certain regions, particularly Latin America and the Caribbean, where studies are published in relevant non-English language. Fourth, the assessment of methodological quality was limited for many studies by poor quality reporting of study methodology.

In conclusion, prevalence of asymptomatic human colonisation by *S. aureus* in LMIC is similar to that observed in HIC. In LMIC community settings however, colonising strains more often demonstrate characteristics associated with a virulent phenotype including antimicrobial resistance and presence of putative virulence-associated genes. This review highlights inequalities in the quantity and quality of *S. aureus* colonisation data from LMIC compared to HIC. Given the significance of *S. aureus* as a human pathogen, our findings underscore the need for a global approach to surveillance to effectively deploy future infection prevention strategies.

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## Author contributions

The project was developed by AJK and TL with supervision provided by TdS and TCD. The methodology was developed by TL, AJK, TdS, TCD, AP, and JH. Data extraction was performed by TL, AJK, NL, and CK. The data reported here has been independently accessed and verified by TL, AJK, NL and CK. Formal data analysis and generation of figures was performed by TL with input from JH and AP. TL wrote the first draft of the manuscript and TCD, TdS, AJK, NL, CK, AP, and JH were all involved in reviewing and editing subsequent drafts. All authors have read the final version of the manuscript, had full access to all the data in the study, and had final responsibility for the decision to submit for publication.

## Data availability

Data from this study will be made available with publication to researchers who provide a methodologically sound proposal to TL. Where appropriate this may include the raw collated data, a data dictionary, and the analysis code.

## Declaration of Competing Interest

One of the authors is an Editorial Board Member/Editor-in-Chief/Associate Editor/Guest Editor for this journal and was not involved in the editorial review or the decision to publish this article.

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## Appendices

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jinf.2025.106462.

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