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Socioeconomic status and the risk for colonisation or infection with priority bacterial pathogens: a global evidence map

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MLN, CWC, and SD conceptualised the study. MLN, NN, and SD acquired the funding. RAM, SD, NN, and CWC performed the literature search. SBl, EEA, NN, CWC, SBa, LM, SAA, SD, and MLN reviewed the titles and abstracts, reviewed full texts, designed the data extraction template, and extracted the data. SBl and SBa prepared the table and figures. SBl, RAM, and MLN wrote the first draft of the manuscript. All authors reviewed the manuscript. All authors had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Declaration of interests

We declare no competing interests.

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Abstract

Low socioeconomic status likely exacerbates risks for bacterial infections; however, global evidence for this relationship has not been synthesised. We systematically reviewed the existing literature for studies detailing the socioeconomic status of participants and their risk for colonisation or community-acquired infection with priority bacterial pathogens that are increasingly becoming antibiotic resistant. 50 studies from 14 countries reported outcomes by the participants' educational attainment, access to health care, income level, residential crowding status, socioeconomic status deprivation score, community setting, or access to clean water, sanitation, and hygiene. Low educational attainment, lower than average income levels, inadequate access to health care, presence of residential crowding, and high socioeconomic status deprivation scores were generally associated with elevated risks of colonisation or infection. Limited research has been conducted on these outcomes in low-income and middle-income countries, and findings regarding the effects of community settings (eg, urban *vs* rural) on these outcomes have been conflicting. Only a proportion of studies focused on pathogen colonisation and infection yielded data stratified by the socioeconomic status of participants. Stratified data should be included in future research to enhance understanding of the complex relationship between socioeconomic status and health, particularly in low-income and middle-income countries.

Introduction

Antimicrobial resistance poses a substantial threat to global public health, as emphasised by WHO.[1] In 2019, nearly five million deaths worldwide were associated with bacterial antibiotic resistance, primarily caused by the pathogens *Escherichia coli, Staphylococcus aureus, Klebsiella pneumoniae, Streptococcus pneumoniae, Acinetobacter baumannii,* and *Pseudomonas aeruginosa.*[2]

Antibiotic resistance is exacerbated when antibiotics are misused owing to inadequacies in terms of access to clean water, sanitation, and hygiene (WASH) for humans and animals; infection control in health-care facilities and farms; access to quality, affordable medicines, vaccines, and diagnostics; awareness and knowledge about antibiotic resistance; and regulations on the purchase and use of antibiotics.[3] Conceivably, the conditions that affect where people are born, live, learn, work, play, worship, and age[4] could exacerbate the risk of acquiring antibiotic-resistant bacterial pathogens. Poverty exacerbates the risk of colonisation or infection with community-acquired antibiotic-resistant bacteria.[5] However, other indicators of socioeconomic status related to the social determinants of health, such as educational attainment or access to health care, require further investigation.

The overarching goal of this scoping review was to compile evidence for the association between the socioeconomic status of individuals and their risk for colonisation or community-acquired infection with priority bacterial pathogens that are increasingly

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becoming antibiotic resistant. We broadly defined socioeconomic status and included relevant literature from any country, provided the research included data on colonisation or infection with one or more bacteria commonly associated with antibiotic resistance. These bacteria include *Enterococcus faecium, S aureus, K pneumoniae, A baumannii, P aeruginosa*, and *Enterobacter* species (collectively known as ESKAPE pathogens) and *E coli*.

Methods

Search strategy and selection criteria

Our search strategy was constructed to support the current study as well as a scoping review of the evidence for racial and ethnic disparities in community-acquired colonisation or infection with the bacteria of interest.[6] We focused on ESKAPE pathogens, as these are often multidrug resistant (MDR); and *E coli*, as these are common causes of community-acquired infections such as urinary tract infections (UTIs). A comprehensive search of the scientific literature was conducted in MEDLINE (Ovid), MEDLINE Epub Ahead of Print, In-Process, In-Data-Review & Other Non-Indexed Citations, and Daily (Ovid), Global Health (Ovid), Embase (Elsevier), Cochrane Database of Systematic Reviews (Wiley), Cochrane Central Register of Controlled Trials (Wiley), and Web of Science Core Collection for eligible studies that reported race, ethnicity, or socioeconomic status for populations with a pathogen of interest.

Search strategies were designed using a combination of controlled vocabulary and free-text keywords. All searches were based on an initial MEDLINE search developed by means of collaboration among the authors using Medical Subject Heading terminology and related keywords for the following concepts: community-acquired infections, outpatients, ambulatory care, socioeconomic factors, health status disparities, disparities in health care, continental population groups, ethnic groups, Gram-negative bacteria, and individual ESKAPE pathogens. The MEDLINE search strategy (appendix p 2) was translated to each of the listed databases by RAM, and all databases were searched from inception up to January, 2022, except for MEDLINE Epub Ahead of Print, In-Process, In-Data-Review & Other Non-Indexed Citations and Daily (Ovid), which was searched from 2017 up to Jan 10, 2021. References were collected and deduplicated using Endnote X9 before being exported to Covidence for screening and management.

Eligibility criteria

We included studies that reported data on socioeconomic status for at least one pathogen of interest irrespective of study design (except case reports, case studies, case series, or narrative reviews), age group, or country. We also included studies that reported both infection and colonisation, as long as the data were reported separately.

We excluded studies presenting mixed pathogens when more than half of the pathogens were not of interest unless such studies included subgroup data for at least one pathogen of interest. Only studies providing clear identification of the pathogen as community-acquired or presenting exclusively outpatient or community-based data were included. Studies

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without outpatient or community data and those defining community acquisition solely through phenotype or sequence type were excluded. Of studies that included both hospital-acquired and community-acquired data, we excluded those lacking subgroup data for the community-acquired pathogens. Additionally, we excluded those that compared persistent colonisation with cleared colonisation, as well as those presenting comparisons between countries, regions, or hospitals instead of individual data.

Screening, data extraction, and data synthesis

Abstracts and full-text articles that met predefined inclusion criteria (table) were screened independently by at least two reviewers from a team that included SBl, EEA, NN, CWC, SBa, LM, SAA, and MLN, and any conflicts were resolved during weekly team meetings. A customised extraction form was created in Covidence to capture relevant study data from eligible studies, including country, study design, study definition of community-acquired infection, characteristics of the study population, exposure and comparator, outcomes of interest, and directionality of results.

For each paper, we extracted all possible comparisons between a socioeconomic status exposure (eg, access to health care) and an outcome (eg, *S aureus* infection), regardless of whether a statistical test was conducted in the original study to assess the significance of that association or not. Each of these extracted exposure-outcome pairs is referred to as a comparison here. When a study had data stratified by more than one socioeconomic status exposure or outcome of interest, or both, then multiple comparisons were extracted from that study.

The extraction form was piloted by the author team for a subset of studies and then revised to ensure that all relevant data were captured. Data from each study were extracted independently by at least two team members among SBl, EEA, NN, SBa, LM, SAA, and MLN. A third team member then compared the data entries of the two extractors and resolved any discrepancies. The extracted data from all the included studies are summarised in the table and figures using R version 4.3.1 and the ggplot2 package. This study followed the PRISMA checklist for scoping reviews (appendix pp 4–5).

Results

The literature search yielded 1039 unique papers, 388 of which met the preliminary inclusion criteria for both the scoping reviews. Upon screening of the full texts, 85 papers met the inclusion criteria (figure 1). Two additional articles were identified by searching the references of the 85 papers, resulting in the inclusion of a total of 87 papers.

50 articles from 14 countries reported colonisation or infection with one or more of the bacteria of interest by the socioeconomic status of the participants and were included in this scoping review (appendix pp 6–12 and figure 2). 37 papers provided data from high-income countries, two from upper-middle-income countries, eight from low-income and middle-income countries (LMICs), and three from low-income countries, as per the definitions of country income status provided by the World Bank in 2023. The studies included in this review were published from 1992 onwards (figure 3).

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We organised our findings into seven socioeconomic status categories, which were chosen post hoc based on the exposures reported in the included studies: educational attainment (13 studies), access to health care (n=14), income level (n=18), residential crowding status (n=16), socioeconomic status deprivation score (n=14), community setting (n=12), and WASH access (n=4). Most studies (n=66) involved participants with *S aureus* colonisation or infection (figure 4).

Educational attainment

13 studies presented outcomes of interest by the educational attainment of the participants (appendix p 13);[7–19] we extracted one to five comparisons from each study (total n=31). Among 25 comparisons involving educational attainment and outcomes of interest, 13 showed an association between lower educational attainment and elevated risks for colonisation or infection.

A shorter duration of education was significantly associated with a greater risk of *K pneumoniae* oropharyngeal tract colonisation among community-dwelling adults and children in Viet Nam[9] and a higher incidence of *S aureus* bacteraemia, *E coli* bacteraemia, and community-acquired bacteraemia among Danish participants.[13] Compared with a college-level education or higher, having no formal education presented a greater risk of bacteriuria among adults who visited an outpatient department in Ethiopia.[15] Compared with individuals who had completed at least high school in Ecuador, those with lower educational attainment were more likely to have children whose intestines were colonised with MDR and extensively drug-resistant *E coli*.[14] Additionally, lower educational attainment was associated with higher incidences of meticillin-resistant *S aureus* (MRSA) infections among patients in the USA[8,18,19] and nitrofurantoin-resistant *E coli* infections among patients in the UK.[16] Pregnant women whose head of the family was unemployed, unskilled, semi-skilled, or a non-manual employee had a higher risk of intestinal colonisation with extended-spectrum betalactamase (ESBL)-producing Enterobacterales than those whose head of the family was a manager.[12] 12 comparisons from eight studies did not indicate any statistically significant associations in this context.[7,8,10,12–14,16,17]

Access to health care

14 studies from the USA reported outcomes of interest by access to health care among participants[11,19–31] (appendix p 14); we extracted up to eight comparisons from each study (n=32). In 21 comparisons of the association between access to health care and outcomes of interest, more than 75% (18 comparisons) showed a statistically significant association. Using Medicaid, Medicare, or no health insurance was associated with a significantly elevated risk of *S aureus* colonisation than using private or military insurance.[31] A study of medical records from two health-care facilities on the west coast of the USA indicated that Medicaid use was associated with a 0·6% and 0·9% reduction in the risk of being diagnosed with a UTI. In addition, Medicaid use was associated with an 8–9% increase in the risk of UTI caused by MDR *E coli*.[26] Findings from two studies showed that children with Medicaid coverage or no insurance were more likely to be colonised[22] or infected with MRSA[24] than those with private insurance or commercial plans.

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Similar trends have been reported in adults; five reports indicated that adults who rely onpublicinsurance,[20,23,29] self-pay,[28,29] or other forms of insurance[28] had a significantly higher risk of MRSA infection than those with private insurance. The rate of MRSA infection among individuals living in medically underserved areas was higher (rate ratio: 2·4 [95% CI2·16–2·68]) than among those not living in such areas.[19] Additionally, the risk of MRSA infection was 92% lower for individuals with health-care coverage than for those without health-care coverage.[19] Furthermore, requiring an interpreter was associated with a 28–36% increase in the risk of UTI caused by MDR *E coli*.[26] Research from the midwest USA showed that uninsured patients of a county hospital system were at a higher risk of infections with ceftriaxone-resistant Enterobacteriaceae and ceftriaxone-resistant *E coli* than those with insurance.[21] Three comparisons from three studies did not indicate any statistically significant associations in this context.[25,27,30]

Income level

18 studies reported outcomes of interest by the income level of the participants (appendix p 15).[7–10,13,14,16,18–21,23,29,32–36] Among the 36 comparisons of the association between the income of the participants and an outcome of interest, nine comparisons indicated that low-income levels were associated with an elevated risk of colonisation or infection with priority pathogens, regardless of their susceptibility to antibiotics. Four of those comparisons, which were carried out as part of a Danish study,[13] indicated that low personal annual income was associated with a significantly elevated risk for community-acquired bacteraemia and bacteraemia caused by *S aureus*, *Enterococcus* spp, or *E coli* compared with that for high personal annual income. Research from Ethiopia showed that children with caretakers earning a low monthly income were more likely to be gut-colonised with diarrhoeagenic *E coli* than those with caretakers earning a high monthly income. On the west coast of the USA, patients residing in neighbourhoods within the lowest income quintile had 17% higher odds of *S aureus* skin and soft tissue infection (SSTI) versus other causes of SSTI than those in neighbourhoods with the highest income quintile.[36] An analysis from India revealed that pregnant women in the lower half of the household income distribution were at higher risk of bacteriuria than those in the higher half.[7] In addition, women in the lower half of the household income distribution had a greater risk of having bacteriuria caused by ESBL-producing organisms and being colonised with ESBL bacteria than those in the higher half.[7]

Eleven additional comparisons revealed an association between low income and an elevated risk of colonisation or infection with antibiotic-resistant pathogens. Research from the USA indicated that low-income households had an overall increased risk of MRSA infection,[8,19,20,] as well as an increased risk of having MRSA over meticillin-sensitive *S aureus* (MSSA) than high-income households.[8] People residing in low-income neighbourhoods on the west coast of the USA had higher odds (odds ratio [OR] 1·42 [95% CI 1·33–1·51]) of having an MRSA SSTI than an MSSA SSTI.[36] Another study from the USA indicated that patients living in neighbourhoods with a high proportion of poverty over the past 12 months had increased rates (rate ratio 16·78 [95% CI 11·92–23·62]) of invasive MRSA infection.[19] In addition, patients living in neighbourhoods with a higher percentage of homes valued at 400% or more of the median home value (ie, US$750

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000) had reduced rates (rate ratio 0·46 [95% CI0·31–0·68]; n=2521) of MRSA infection.[19] Further, patients living in neighbourhoods with extreme income inequality had significantly greater risk (rate ratio 12·99 [95% CI 6·54–25·82]; n=2521) of MRSA infection than those living in neighbourhoods with less income inequality.[19] Two studies from the southeast USA indicated that children living in block groups where most households were below the the poverty level had higher odds of community-onset MRSA SSTI[23] and were at a higher risk of developing MRSA SSTI than MSSA SSTI,[29] relative to children living in block groups where a few households were below the poverty level. Findings of a study from the midwestern region of the USA revealed that individuals with a low income had a higher risk of developing MSSA infections than non-MSSA infections.[8] Of the 36 comparisons, 16 indicated no statistically significant associations in this context.[9,13,14,16,18,21,32,34,35]

Residential crowding status

16 studies reported outcomes of interest by the residential crowding conditions of the participants (appendix p 16);[8,12,14,19,21–24,29,37–44] between one and three comparisons were extracted from each study (n=29). Among the 27 comparisons evaluating the association between the residential crowding status of the participants and an outcome of interest, two indicated that having more than two household occupants in a bedroom was associated with an increased risk of *S aureus* colonisation in children living in New Zealand.[40] Another study from Argentina revealed that living in homes with greater than three individuals per room was associated with an increased risk of *S aureus* SSTI.[37] Four comparisons indicated that having more than one person per room was associated with an increased risk of infection with MRSA,[29] SSTI (including MRSA, MSSA, and other pathogens),[38] or ceftriaxone-resistant *E coli*.[21] Having more than two individuals per bedroom was associated with an increased risk of MRSA colonisation in children from Madagascar.[22] Further, an increasing ratio of inhabitants per room was associated with an increased risk of ESBL carriage in adults from Madagascar.[12]

Three comparisons from the USA indicated that repeat,[44] recent,[41] or history of incarceration[42] was associated with an increased risk of MRSA colonisation and MRSA SSTI in adults. Compared with those living in stable housing, those living in temporary housing were at an increased risk of MRSA colonisation.[41] One comparison revealed that people currently residing in public housing had increased odds of having an MRSA infection (21 of 35 individuals) compared with those not residing in public housing (497 of 1187 individuals; adjusted OR 2·5 [95% CI 1·25–4·98]; n=1222).[41] Another comparison indicated that the odds of MRSA infection for individuals who had lived in a group setting (six of ten individuals) were higher than that of those who had never lived in a group setting (69 of 291 individuals; OR 3·9; n=301). 11 of the 29 comparisons did not indicate any statistically significant associations in this context.[8,14,21,39–43]

Socioeconomic status deprivation score

13 studies provided data on outcomes of interest by socioeconomic status deprivation scores (appendix p 17); [16,18,26,37,40,42,45–51] between one and eight comparisons were extracted from each study (n=27). Within these, the association between the socioeconomic status deprivation score and an outcome of interest was statistically evaluated in 25 comparisons.

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Notably, four comparisons from New Zealand indicated that medium to high deprivation deciles were associated with a higher risk of paediatric *S aureus* or MRSA infections than low deprivation deciles.[40,49,51] In the USA, individuals residing in communities characterized by higher than median socioeconomic status deprivation scores (determined by six indicators from US 2000 Census data)[45] showed a greater risk of *S aureus* SSTI than those in areas with lower than median scores. Similarly, in Argentina, residents of more deprived neighbourhoods faced a heightened risk of *S aureus* SSTI than those living in less deprived neighbourhoods.[37]

Using the 2016 Index of Relative Social Economic Disadvantage to assess socioeconomic status deprivation and ceftriaxone-resistant *E coli* uropathogens in Victoria, Australia, Chua and Stewardson found that living in a community ranked in the first decile (most deprived) was associated with an increased risk of this outcome compared with living in communities ranked in any other decile.[46] Eight comparisons from studies conducted in the USA and the UK indicated that, compared with individuals living in low-deprivation neighbourhoods, those living in neighbourhoods with high socioeconomic status deprivation scores had an increased risk of infection with MRSA[20,45] and MDR,[26] trimethoprim-resistant,[16] ampicillin-resistant,[16] ciprofloxacin-resistant,[16] and nitrofurantoin-resistant *E coli*.[16] Ten of the 27 comparisons did not reveal any statistically significant associations in this context.[16,18,40,42,47,48,50]

Community setting

12 studies from eight countries provided data on outcomes of interest based on the community type or setting of participants (appendix p 18);[9,16,19,20,26,40,45,46,52–55] between one and five comparisons were extracted from each study (n=33). Among the 21 comparisons evaluating the association between the community setting of the participants and an outcome of interest, seven indicated that living in a non-urban setting was associated with an increased risk of colonisation or infection with an outcome of interest compared with living in an urban setting.[9,20,54] For example, two comparisons revealed that individuals living in a rural location had an increased risk of oropharyngeal *K pneumoniae* carriage[9] and *S aureus* colonisation compared with those living in an urban location.[54] Community members working as farmers or hired labourers in Viet Nam had a greater risk of oropharyngeal *K pneumoniae* carriage than those in other occupations.[9] In the northwest USA, residents with greater proximity to swine farms had a greater probability of acquiring an MRSA SSTI than that of not acquiring it.[20] Findings from a study conducted in Austria revealed that living in rural areas (defined as small cities, villages, or the countryside) was associated with a greater risk of being colonised with resistant *S aureus* than living in urban areas (ie, big and intermediate cities).[54] However, the opposite relationship was also reported.[19,45] For example, city-dwelling individuals from central and northeastern Pennsylvania, USA had increased odds of *S aureus* SSTI than township-dwelling ones.[45] Meanwhile, two comparisons indicated that living in a US city[45] or a US community with high urbanicity[19] was associated with an increased risk of MRSA infection. 11 of the 33 comparisons did not reveal any statistically significant associations in this context.[16,40,46,54]

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WASH

Four studies provided outcomes of interest by the WASH access of the participants (appendix p 19);[7,10,14,56] up to eight comparisons were extracted from each study (n=13). Six of these comparisons did not investigate the association between WASH and an outcome of interest.[7] Crum-Cianflone and colleagues found that among HIV-infected US adults, using a public bath in the past 6 months was associated with higher odds (OR 6·91 [95% CI 2·62–18·27]) of MRSA colonization compared with those who did not use a public bath.[56] No other comparisons had any statistically significant associations in this context.[7,10,14]

Discussion

The global burden of bacterial infections differs among countries. However, whether individual susceptibility varies within countries, particularly as a function of the social determinants of health, has not been comprehensively examined. Establishing such risk factors is crucial, as many bacterial infections are becoming increasingly difficult to treat. Here, we systematically reviewed the literature to determine whether socioeconomic status of an individual could be associated with their risk for colonisation or infection with bacterial pathogens, including the ESKAPE pathogens and *E coli*, which are frequently antibiotic resistant. Among the 50 studies included, the risk of colonisation or infection was generally associated with low educational attainment, low income levels, inadequate access to health care, residential crowding, and high socioeconomic status deprivation scores. Evidence for associations with community setting showed mixed results. Notably, we identified only a few studies originating from LMICs, despite their high burden of antibiotic resistance. Collectively, our findings from this scoping review suggest that social determinants of health are important yet understudied factors that shape the risk of colonisation or infection with bacterial pathogens that are increasingly becoming antibiotic resistant.

Income level was one of the most reported socioeconomic characteristics. Of the 36 comparisons analysed, 20 revealed that low income (whether at the individual, household, or neighbourhood level) was significantly associated with a higher risk of colonization or infection with priority bacterial pathogens, including drug-resistant *S aureus*, *K pneumoniae*, *E coli*, and *Enterobacter* spp. The relationship between income level and antibiotic resistance is well-established at the country level;[57] however, we observed associations between the incomes of individuals and bacterial colonisation or infection risks in countries with varying gross national incomes. Regardless of the gross national income of a country, individuals with low income might have reduced access to timely medical care;[28] be more likely to use unprescribed antibiotics (ie, from friends, relatives, online, or from abroad);[58] reside in overcrowded homes with inferior infrastructure;[5] or have limited access to hygiene, among other factors exacerbating their susceptibility to bacterial infections compared with individuals with high income.

The USA is the only country where access to health care was identified as an indicator of socioeconomic status; in the USA, insurance is typically provided through employers, resulting in profound disparities in health and access to health care.[59] Among these studies, poor access to health care was defined as insufficient health insurance, use of public

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insurance, the need for a medical interpreter, or residence in a medically underserved area. Most comparisons indicated that poor access to health care was associated with a higher risk of infection or colonization with a bacterial pathogen of interest, including MRSA and drug-resistant *E coli* which cause UTIs. However, two of 21 comparisons indicated an inverse correlation, where Medicaid use (ie, public insurance for individuals with low income) was associated with a reduced risk of infection.[26] These conflicting findings are challenging to interpret; although they could reflect true differences in susceptibility to infection, they could also reflect the fact that people with low access to health care could be less likely to seek treatment and be diagnosed with infections.[60,61] Previous studies involving paediatric patients have suggested that inadequate access to timely care for infections among uninsured or Medicaid patients is a highly likely cause of elevated risk of infections.[23,27]

Our findings that lower educational attainment was typically associated with an increased risk of colonisation or infection with priority bacterial pathogens contrast with those from a previous analysis of anthropological factors related to global antibiotic resistance.[57] In this country-level analysis, educational attainment may have been correlated with general access to resources such as hospitals and antibiotics,[62] which might increase exposure to antibiotic-resistant bacteria in populations. At an individual level, people with less education often have inadequate access to information necessary for making health decisions, leading to behaviours that might increase the risk of bacterial infections. Our findings align with general trends observed between educational attainment and various poor health outcomes.[63]

Conflicting trends of association were observed between community settings and individuals' risk of colonisation or infection with priority pathogens. Of the 21 comparisons extracted from eight studies, seven comparisons from three studies indicated that residing in a rural setting was associated with increased risks of infection or colonisation compared with living in an urban setting, whereas three comparisons from two studies indicated the opposite relationship. These conflicting patterns could be partly explained by important differences in the types of exposures present in some rural settings versus others. Specifically, individuals in some rural settings might be exposed to livestock or intensive animal farming, which are known sources of exposure to drug-resistant *S aureus* and *E coli*,[64] whereas those in rural settings without frequent livestock exposure might face a lower risk of bacterial disease compared with their urban counterparts, possibly owing to the lower population density in rural areas. We noted that these studies rarely provided definitions of urban versus rural; current literature suggests that the relative position of individuals within an urban versus rural environment might be a less important determinant of health than broader aspects of urbanisation and urban life.[65–67] Further research is required to distinguish the effects of population density, access to health care, and exposure to livestock relative to the community setting of an individual on the outcomes of interest.

More than half of the 25 comparisons we extracted indicated that living in a high-deprivation neighbourhood was associated with an elevated risk of colonisation or infection with priority bacterial pathogens, including MRSA and drug-resistant *E coli*, compared with living in a lower-deprivation neighbourhood. Socioeconomic status deprivation scores are composite, area-level measurements that were first developed in the 1970s and have largely been used by high-income countries to study the relationships between socioeconomic status and

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health outcomes.[68] The positive correlation between socioeconomic status deprivation and the risk of colonization or infection mirrors trends we observed for individual components of socioeconomic status deprivation scores, such as income level, educational attainment, and residential crowding. Only one middle-income and no low-income countries stratified rates of colonisation or infection with the pathogens of interest by a socioeconomic status deprivation score. Deprivation scores could be more challenging to calculate in some LMICs where the income generated through the informal economy is difficult to measure,[69] and health-care expenditures are poorly tracked.[70] Nonetheless, socioeconomic status deprivation scores can be valuable when granular data are unavailable or unreliable. Future studies on bacterial infection should consider including such data.

Despite interest in the role of improved WASH in reducing global antibiotic resistance, we identified only four studies that included colonisation or infection with priority bacterial pathogens in individuals stratified by their WASH access. The low number of studies identified could be a consequence of our search strategy, as WASH access might not always be considered a characteristic of socioeconomic status, and thus, our search terms might not have captured relevant studies. Alternatively, this low number could be attributed to recent studies focusing more on the relationship between antibiotic resistance and WASH at regional[71] or country levels[72] rather than at individual levels.

Conclusions

In this scoping review, we found sufficient evidence to support future systematic reviews and meta-analyses to evaluate the relationship between socioeconomic status and risks for colonization or infection with community-acquired bacterial pathogens, particularly *S aureus* and *E coli*.

However, this study has some limitations. First, we identified few studies from LMICs, where the burden of antibiotic resistance is the highest. Bacterial infections and their antibiotic susceptibility profiles might be poorly characterised in some LMICs due to inadequate laboratory infrastructure and diagnostic challenges.[73] However, the US Centers for Disease Control and Prevention and other public health organisations are working actively with the health ministries of LMICs to improve their capacity for timely diagnosis. Expanded laboratory infrastructure will provide new opportunities to examine intersections between the socioeconomic status of individuals and bacterial colonisation or infection risks, which is important to inform equitable interventions.

Second, although we reported findings by individual socioeconomic status characteristics, many of these are collinear by nature. Among the proportion of published studies that provided data stratified by socioeconomic status, only a few considered the collinearity between the reported socioeconomic status characteristics, thus rendering the assessment of the most important exposures driving observed associations challenging. Future studies should provide data stratified by socioeconomic status characteristics to facilitate a better understanding of the complex interplay between socioeconomic status and health, especially in LMICs.

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Third, we did not extract the bacterial detection or antibiotic resistance characterisation methods used in the studies included. Given the wide time range and diversity of countries represented, differences in the sensitivity of the laboratory methods could have affected the number of cases reported.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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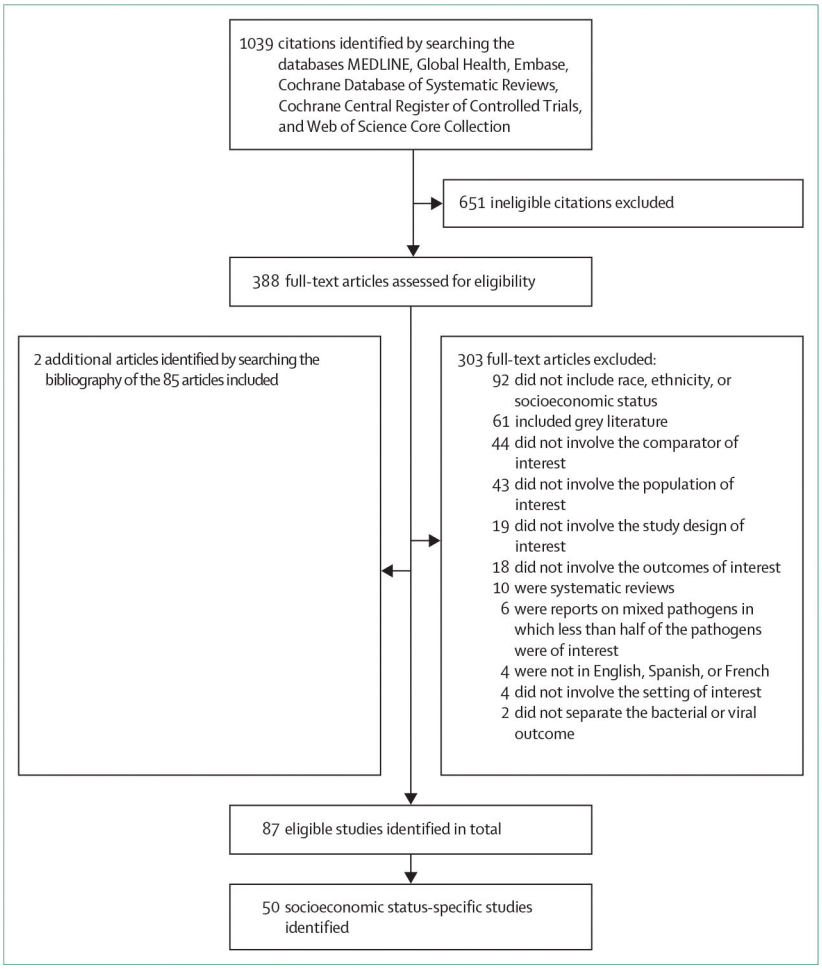

Figure 1: Study inclusion schema

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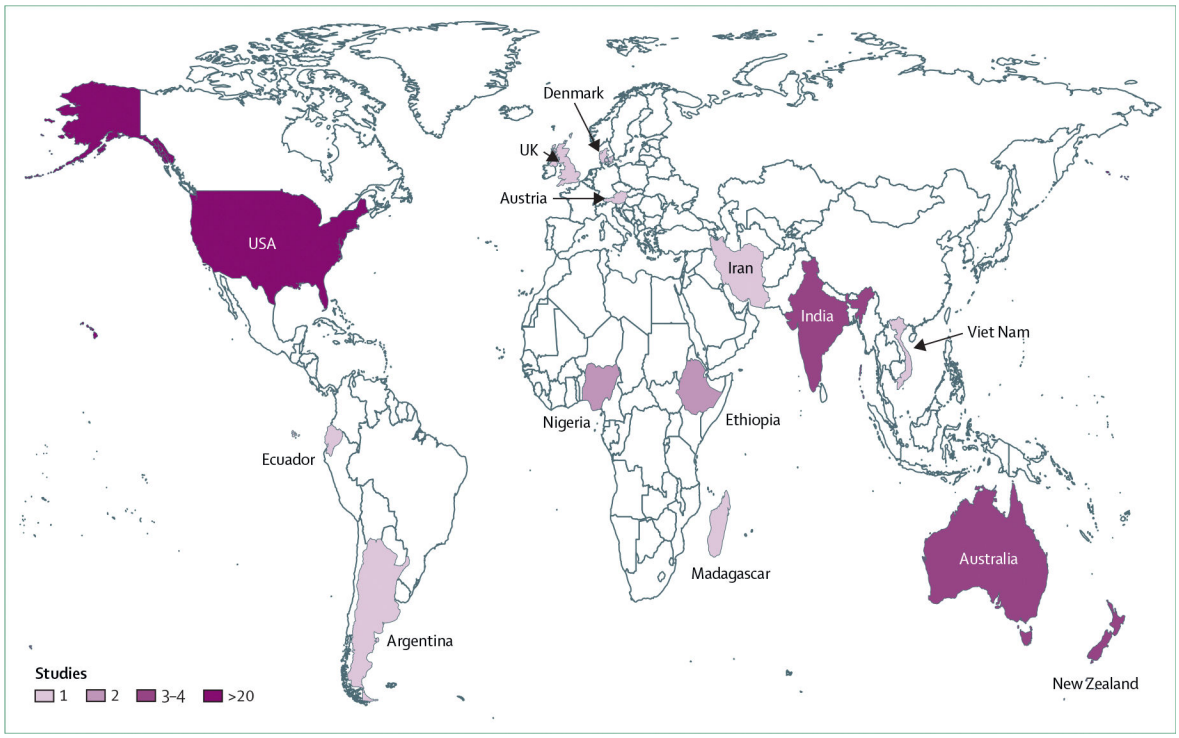

Figure 2: Study locations for 50 studies describing individuals' risk of colonisation or infection with priority pathogens by their socioeconomic status

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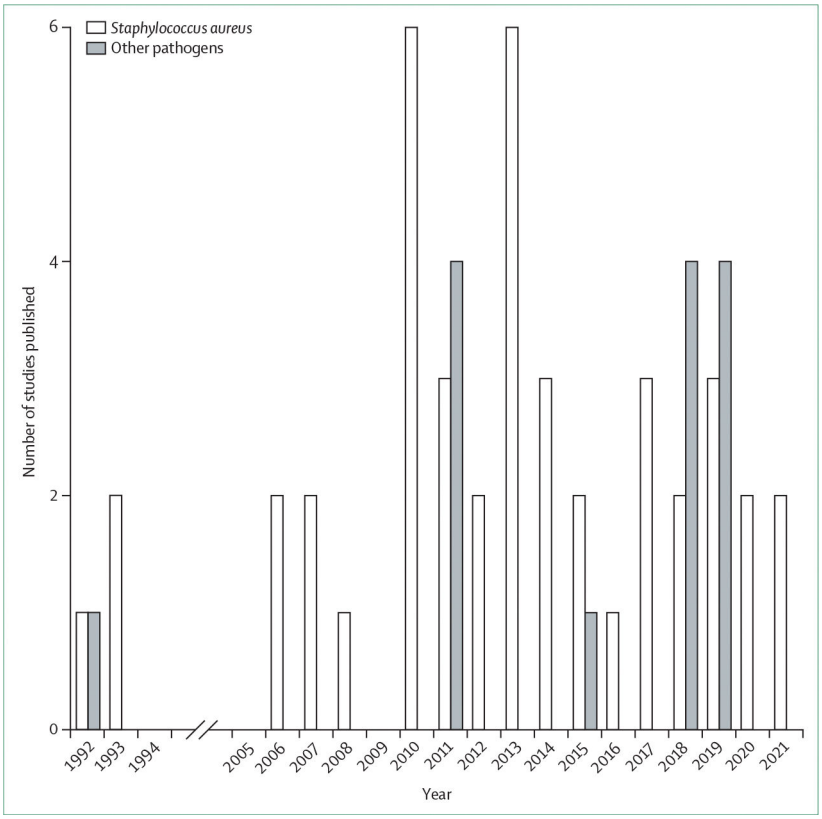

Figure 3: Year of publication for the 50 studies included

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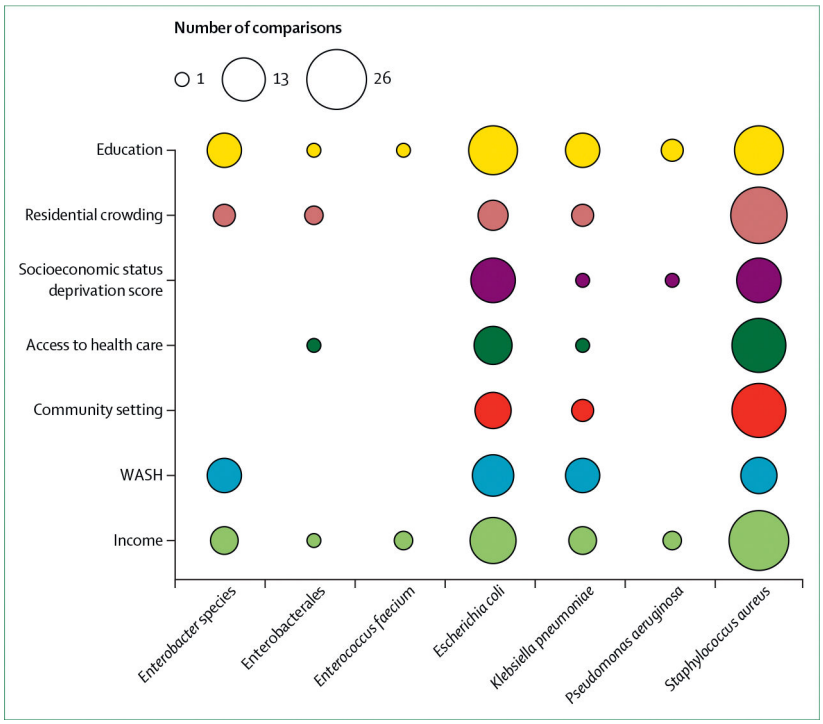

Figure 4: Distribution of indicators of socioeconomic status by priority pathogens among the 50 publications included in this scoping review

A comparison was counted more than once for visualisation purposes when the outcome reported (ie, skin and soft tissue infection, urinary tract infection, bacteraemia, bacteriuria) was caused by more than one organism of interest. WASH=water, sanitation, and hygiene access.

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Table:

Inclusion and exclusion criteria for the studies included in this scoping review

	Inclusion criteria	Exclusion criteria
Study design	Observational studies	Case reports or studies Case series Narrative reviews
Study population	Any age group, gender, country, and health status	Studies in which data for race or ethnicity or socioeconomic factors were not reported
Exposure	Any socioeconomic factor reported	Data stratified by country, region, or hospital rather than a racial or ethnic group or socioeconomic status
Comparator	Distinct socioeconomic status groups from the same country	No distinct socioeconomic metrics reported
Outcomes	Community-acquired (eg, outpatient, emergency rooms, health clinics, ambulatory care, population-level surveillance) colonisation or infection with: • *Enterococcus faecium* • *Staphylococcus aureus* • *Klebsiella pneumoniae* • *Acinetobacterbaumannii* • *Pseudomonas aeruginosa* • *Enterobacter* species • *Escherichia coli* • Enterobacteriaceae or Enterobacterales	Hospital-acquired colonisation or infection Device-associated (eg, catheter) infection Non-bacterial infection or cause of infection unclear Mixed pathogen infections, in which less than half of the pathogens were of interest Community acquisition or community association based only on phenotype or genotype