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The aetiology and antimicrobial resistance of bacterial maternal infections in Sub-Saharan Africa—a systematic review and meta-analysis

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Abstract

Background Understanding the aetiological organisms causing maternal infections is crucial to inform antibiotic treatment guidelines, but such data are scarce from Sub-Saharan Africa (SSA). We performed this systematic review and meta-analysis to address this gap.

Methods Microbiologically confirmed maternal infection data were collected from PubMed, Embase, and African Journals online databases. The search strategy combined terms related to bacterial infection, pregnancy, postnatal period, observational studies, SSA. Exclusion criteria included colonization, asymptomatic infection, and screening studies. Pooled proportions for bacterial isolates and antimicrobial resistance (AMR) were calculated. Quality and completeness of reporting were assessed using the Newcastle–Ottawa and STROBE checklists.

Findings We included 14 papers comprising data from 2,575 women from four sources (blood, urine, surgical wound and endocervical). Mixed-growth was commonly reported at 17% (95% CI: 12%-23%), *E. coli* from 11%(CI:10%-12%), *S. aureus* from 5%(CI: 5%-6%), *Klebsiella* spp. at 5%(CI: 4%- 5%) and *Streptococcus* spp. at 2%(CI: 1%-2%). We observed intra-sample and inter-sample heterogeneity between 88–92% in all meta-analyses. AMR rates were between 19% -77%, the highest with first-line beta-lactam antibiotics. Convenience sampling, and limited reporting of laboratory techniques were areas of concern.

Interpretation We provide a comprehensive summary of microbial aetiology of maternal infections in SSA and demonstrate the paucity of data available for this region. We flag the need to review the current local and international empirical treatment guidelines for maternal bacterial infections in SSA because there is high prevalence of AMR among common causative bacteria.

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Trial registration Prospero ID CRD42021238515.

Keywords Maternal infections, Bacterial, Aetiology, Antimicrobial resistance, Sub-Saharan Africa

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Evidence before this study

- Global frequency for infection-related severe maternal outcomes is 70.4 per 1000 livebirths in Low and Middle-Income Countries (LMIC) and 10.9 per 1000 livebirths in High-Income countries (HIC).
- The World Health Organisation (WHO) international empiric clinical treatment guidelines for maternal infection in LMICs recommend ampicillin (a penicillin) and once-daily gentamicin as first-line antibiotics for treating maternal peripartum infections, and a combination of clindamycin and gentamicin for postpartum endometritis. The WHO also recommends amoxicillin for lower urinary tract infections and a medical review for other causes, such as soft tissue infections.
- There is still no global data on the microbial aetiology of maternal infections, which is needed to guide management of maternal infections in LMIC, where the burden of disease is the highest

Added value of the study

This study summarizes the available microbial aetiology and the antibiotic susceptibility profiles for the causative bacterial agents for maternal infection in Sub-Saharan Africa (SSA). It also estimates the prevalence, diagnostic yield and antimicrobial drug resistance patterns of symptomatic and clinically relevant maternal bacteraemia, bacteriuria, endometritis, and soft tissue infection. This study also demonstrates the limited and scanty evidence available for the microbiology of maternal infection.

Implications of all the available evidence

- There is a need for high-quality surveillance of maternal microbiological data in this context.
- This study provides the best evidence to inform empirical treatment guidelines for local and international maternal infections.
- This study also flags the need to review the current local and international treatment guidelines for maternal bacterial infections in SSA because the results of this study indicate a high prevalence of AMR in common causative events of maternal infections to commonly used antimicrobial drugs in SSA.

Introduction

Over the past 25 years, there has been a 44% decrease in Maternal Mortality Ratio (MMR) globally [1], which falls well short of the 2015 Millennium Development Goal target of a 75% reduction in MMR [2]. There is therefore an urgent need to strengthen efforts to reduce maternal mortality if the Sustainable Development Goals target, to reduce MMR to less than 70 per 100,000 live births by 2030 is to be met [3].

The global burden of maternal deaths is highest in Sub-Saharan Africa (SSA) [4, 5]. Over two-thirds of global maternal deaths occur in SSA, with at least 200,000 deaths per year [6, 1]. Of these deaths, up to 10% are due to infection which is twice the proportion observed in high income countries [7]. The problem's origins lie in the low quality of care areas, including inconsistent infection prevention, poor infection treatment, delayed sepsis diagnosis, and inadequate sepsis management. In addition, limited availability of validated diagnostics (culture and sensitivity) poses difficulties in prompt identification and management of maternal infection in SSA [8]. Therefore, prioritizing surveillance in this arena to describe the existing landscape of maternal infections in this region is essential to guide simple strategies to prevent morbidity and mortality from maternal infections. It also has the potential to reduce maternal death rates globally.

Data on the microbiologic causes of bacterial maternal infections can inform policies (programme strategies and treatment guidelines) and identify antimicrobial resistance (AMR) threats in SSA. However, there are challenges in estimating maternal sepsis burden due to differences in its definitions [9, 10]. Notably, within obstetrics there are a broad range of clinically relevant infections and infection sources that are associated with maternal sepsis. These include infections from the urogenital tract, but also infections associated with other organ systems, such as pneumonia. The World Health Organization (WHO) has therefore proposed a broad definition for maternal sepsis which we used for this systematic review to describe any "symptomatic maternal bacterial infection". It spans across the pregnancy period spectrum, including the antenatal, peripartum, postpartum, and post-abortion periods (up to 42 days of pregnancy termination). It also incorporates an extensive host of infectious morbidities, including genital tract infections (e.g., chorioamnionitis and endometritis), extra-genital infections (e.g., surgical site infections), as well as other maternal bacterial infections complicating pregnancy, childbirth, and the puerperium (e.g., sexually transmitted infections (STIs), urinary tract infections (UTIs) [10].

Despite significantly higher maternal infection-related mortality in SSA, to date there has been no systematic review that summarizes the underlying microbiological agents causing maternal bacterial infection in SSA, nor their resistance patterns. Therefore, using the WHO definition of maternal infection and sepsis, this systematic review will summarise the available data on the main bacterial agents causing maternal infection and their susceptibility to antibiotics to inform the international and local current empiric antibiotic treatment guidelines. It will also assess completeness and quality of the available data.

Methods

Registration

This systematic review is registered with the International Prospective Register of Systematic Reviews (PROSPERO) on March 2021 (Registration number: CRD42021238515).

Search strategy and selection criteria

We searched PUBMED, Embase and African Journals online databases using a search strategy that combined terms relating to laboratory-confirmed bacterial infection, pregnancy, postnatal period, observational studies, and SSA (Supplementary I). A comprehensive literature search (last search 29th March 2023) was performed through the three databases with the support of a clinical librarian. The search did not contain letters or editorials. We used the "humans", "female", and "age" filters. We translated non-English articles. We also searched the systematic review registries for ongoing reviews.

We included any observational study (cohort, casecontrol, and cross-sectional studies) describing the aetiology and antimicrobial resistance (AMR) patterns of maternal bacterial infection in SSA. This review considered any studies that evaluated symptomatic laboratoryconfirmed bacterial infection in keeping with the WHO definition of maternal infection/sepsis among pregnant and postpartum women (up to 42 days after birth). We excluded papers that contained incomplete/internally inconsistent data, that assessed the diagnostic accuracy of any test using only positive samples and that were not in the clinical context of suspected maternal infection. We excluded studies that only described maternal colonization of bacteria rather than infection; for example, maternal colonization of Group B streptococci (GBS) in neonatal infection. Screening studies that did not include women who fulfil the WHO definition were also excluded, for example, studies that evaluated the prevalence of sexually transmitted infections among women attending routine clinical check-ups. Finally, we excluded studies reporting on only one type of bacteria as this may skew proportional estimates.

Two independent authors (CC and CVD) screened titles and abstracts with aid from a librarian (AS) in Rayyan. Duplicates were removed and reviewed individually by CC and CVD. If at least one of the authors approved the study, we obtained the full-text report. In both stages, we compared the results against eligibility criteria. CC and CVD resolved disagreements through discussion; if they could not reach an agreement, a third author (DL) resolved the disagreement.

Data collection and extraction

Using a Microsoft Excel database, CC and CVD extracted the following data from the eligible full-text studies; identification details of the study, including the title, language, authors, year of publication, country, region, setting (urban or rural), study design, study inclusion and exclusion criteria; participant characteristics, including participants' age (median and range), gestation period, co-morbidities (for example, HIV status) and study sample size; exposure of interest, such as type of bacterial infection (invasive or non-invasive), source of infection, when the invasive bacterial infection occurred (antenatal/ postnatal); and outcomes of interest, namely the number and type of samples taken, number of positive samples; the diagnostic method used, antimicrobial susceptibility testing results and the methodology used, and maternal outcomes (if reported).

We contacted the study correspondence authors for further information for studies published only as abstracts. For study reports containing little details on methods and results, we also contacted the authors to obtain additional information on these elements. Disagreements regarding the data extracted were resolved by discussion and, if necessary, by consulting a third review author (DL).

Data analysis

The analysis was conducted in R (R Core Team, 2022), using RStudio [11]. We applied the *metafor* package [12] to perform a meta-analysis of; i prevalence (number of causative agent of interest/total number of samples assessed) and, ii diagnostic yield (number of causative agent of interest/total number of positive isolates; and their 95% confidence intervals) [12]. Pooled estimates from the eligible studies on each causative agents of maternal bacterial infections were estimated using random-effects models (REM) and the DerSimonian-Laird method [13]. We also performed sub-group meta-analyses according to the source of infection; and estimated pooled proportions of antimicrobial resistance for the commonest bacteria for the papers that reported on antibiotic susceptibility.

Completeness and quality assessments of the papers

To assess reporting completeness and quality of included studies, two authors (CVD with either EJMM or EB) independently assessed included studies according to Strengthening the Reporting of Observational Studies in Epidemiology [14–16] and the Newcastle–Ottawa Scale [17, 18], respectively. They then independently applied the checklists to each included study. Both checklists were adopted to better serve reporting of microbiological data.

Results

This systematic review and meta-analysis database search identified 3108 papers for abstract screening after removing duplicates (Fig. 1). Fifty-eight papers were eligible for full-text review; 12 articles were excluded because of no correspondence from authors in studies that reported little detail on results; three papers were on premature rupture of membranes (PROM) screening, six papers had no microbiological data, two were microbiome papers, 20 articles targeted the wrong population, and one article had incoherent results. Therefore, 14 articles were eligible for data extraction. All 14 articles reported data on aetiology, and ten reported on AMR. Two articles were in French [19, 20] and translated by EM and CVD prior to analysis.

The main characteristics of the papers included in this systematic review and meta-analysis are summarised in Table 1 and the geographical distribution in Fig. 2. Eight studies were from East Africa (five from Ethiopia), two from Southern Africa and three from West Africa (Fig. 2). The proportion of study participants with suspected maternal infection who had laboratory confirmed diagnosis ranged from 20.4% to 100%.

Figure 3 shows a forest plot of the pooled proportion of the reported causative agents of bacterial maternal infection for all sample types combined. Overall, mixed growth was reported most at 17% (CI: 12%—23%), followed by *E. coli* at 11% (CI:10%—12%), *S. aureus* at 5% (CI: 5% -6%), *S. epidermis* at 7% (CI: 2%, 16%), *Klebsiella* spp. at 5% (CI: 4%-5%), *L. lactis* at 5% (CI:1%-15%) and Coagulase negative Staphylococci (CONS) was 3% (CI:2%-4%).

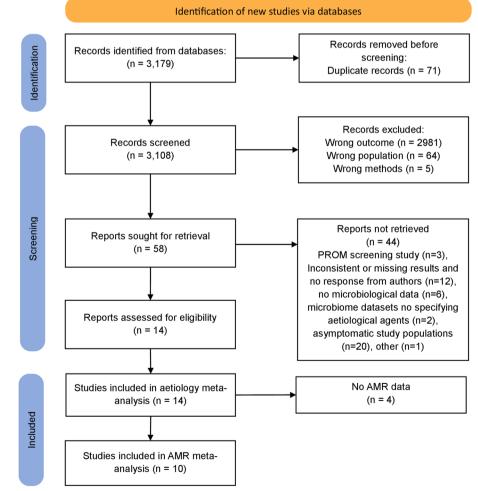


Fig. 1 PRISMA Flow diagram

Study ID	Country	Study design	Sample type	Number of positive samples	Total number of samples tested	Proportion laboratory confirmed maternal infection (95% Cl)	Most common causative agent
Kiponza 2019 [21]	Tanzania	Cross-sectional study	Blood and endocervical discharge	107	197	54.3 (47.4 – 61.3)	<i>K. pneumoniae</i> (blood), <i>E. coli</i> (endocervical discharge)
Biset 2020 [22]	Ethiopia	Cross-sectional study	Urine	38	122	31.1 (22.9 – 39.4)	E. coli
Admas 2020 [23]	Ethiopia	Cross-sectional study	Blood	56	166	33.7 (26.5 – 40.9)	E. coli
Bako 2012 [<mark>24</mark>]	Nigeria	Retrospective study	Endocervical discharge	158	191	82.7 (77.4 – 88.1)	S. aureus
Bebell 2017 [25]	Uganda	Prospective cohort study	Urine and blood	28	360	7.8 (5 – 10.5)	<i>Acinetobacter</i> (urine), no common culture (blood)
Belete 2020 [26]	Ethiopia	Cross-sectional study	Urine	21	79	26.6 (16.8 - 36.3)	E. coli
Majangara 2019 [27]	Zimbabwe	Prospective cohort study	Blood and endocervical discharge	138	301	45.8 (40.2 – 51.5)	<i>Bacillus spp</i> . (blood), <i>E. coli</i> in endocervi- cal discharge
Ouedraogo 2016 [20]	Burkina Faso	Cross-sectional study	Endocervical discharge	61	102	59.8 (50.3 – 69.3)	E. coli
Sawadongo 2019 [19]	Burkina Faso	Cross-sectional study	Wound discharge	45	70	64.3 (53.1 – 75.5)	S. aureus
Gessese 2017 [28]	Ethiopia	Cross-sectional study	Urine	21	103	20.4 (12.6 – 28.2)	E. coli
Kifilie 2018 [<mark>29</mark>]	Ethiopia	Cross-sectional study	Wound discharge	101	107	94.4 (90 – 98.8)	S. aureus
Johnson 2021 [30]	Uganda	Cross-sectional study	Urine	140	400	35 (30.3 – 39.7)	K. pneumoniae
Velin 2021 [<mark>31</mark>]	Rwanda	Prospective cohort study	Wound discharge	44	44	100 (92.0 – 100.0)	Coagulase negative Staphylococci
Orji 2022 [<mark>32</mark>]	South Africa	Retrospective study	Urine	333	1984	16.8 (15.2 – 18.5)	E. coli

Table 1 Description of	f studies reporting aet	iological data for laborato	rv-confirmed bacterial materna	l infection in Sub-Sahara Africa

When we stratified the data by sample type (Fig. 4); blood culture was rarely positive at 2% (CI: 2%-3%), of which growth for *Klebsiella* spp. at 4% (CI: 2%-6%), S. aureus at 4% (CI: 2%-5%) and E. coli at 4% (CI: 3%-6%) was most reported. Around 4% (CI: 2% -6%) of endocervical discharge samples yielded positive cultures of which the majority yielded E. coli at 25% (CI:22%-29%), mixed growth at 17% (CI: 12%-23%) and S. aureus at 14% (CI: 11%-17%). The urine cultures were positive in 1% of cases (CI: 1%-2%). The most common pathogens identified were Klebsiella spp at 4% (CI: 3%-5%), S. aureus at 2% (CI: 2%-3%), and E. coli at 9% (CI: 8%-10%). Approximately 8% (CI: 5-12%) of wound samples generated positive cultures of which the majority showed growth for S. aureus at 30% (CI: 24%-37%) followed by Acinetobacter (16%; CI: 7%-28%). Notably, we observed both intra-sample and inter-sample

heterogeneity in both meta-analyses ranging from 79–96%.

For samples yielding positive cultures, we pooled the proportion of each causative agent (Fig. 5). Overall, the predominant causative agents among positive cultures were *E. coli* at 36% (CI: 33%-38%), mixed growth at 20% (CI: 14%-27%), *Veillonella* spp. at 20% (CI: 1%-72%), *S. aureus* at 18% (CI: 16%-20%), and *Klebsiella spp.* at 15% (CI: 13%-17%). The forest plot in Fig. 6 shows the pooled proportion stratified by sample type. Positive samples from cervicovaginal and urine most commonly yielded E coli, at 38% (CI: 33%-43%) and 43% (CI:38%-47%), respectively; while wound swabs yielded S aureus at 28% (CI: 22%-34%). We also observed both intra-sample and inter-sample heterogeneity in both meta-analyses ranging from 80–96%. Similarly, Fig. 7 presents a stratified bar graph based on sample sources, namely blood,

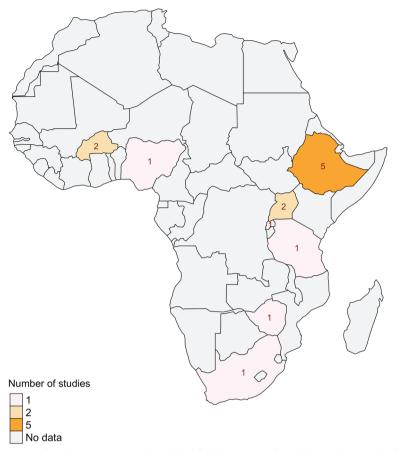


Fig. 2 Geographical representation of studies reporting aetiological data for laboratory-confirmed bacterial maternal infection in Sub-Sahara Africa

endocervical discharge, urine, and wound discharge. The graph illustrates the composition of resistant samples for each bacterium. The data reveals that among all resistant bacteria, E. coli is the most frequently isolated bacterium from blood (n=27/ 96, 28%), urine (n=247/ 581, 43%), and endocervical samples (n=163/ 416, 39%). In contrast, S. aureus (61/ 203, 30%) is predominantly isolated from surgical wound sites.

We summarized pooled antibiotic susceptibility testing (AST) profiles *E. coli, Klebsiella spp.* and *S aureus* (reported from the studies) to commonly used WHO classes of antibiotics in SSA; penicillin (amoxicillin and ampicillin), gentamicin, ciprofloxacin, and ceftriaxone (Fig. 8) as these were the most common clinically relevant antibiotics retrieved from this meta-analysis. Notably, there was limited data on third-line treatments carbapenems, and co-trimoxazole. The pooled resistance of *E. coli* to Penicillin (amoxicillin and ampicillin), gentamicin, ciprofloxacin, and ceftriaxone was 65%, 22%, 24% and 36%, respectively. The pooled resistance of *Klebsiella* spp. to penicillin (amoxicillin and ampicillin), gentamicin, ciprofloxacin, and ceftriaxone was 77%, 33%, 19% and 40%, respectively. The pooled resistance of *S. aureus* to penicillin (amoxicillin and ampicillin), gentamicin, ciprofloxacin, and ceftriaxone was 68%, 30%, 24%, and 32%, respectively.

The assessment of strength of reporting within each included study, according to the STROBE criteria, is summarized in Fig. 9. We assessed completeness of reporting using the STROBE checklist, including the title, introduction, methodology, results, discussion, and funding. Of the criteria assessed, bias reporting was the least well-reported methods criteria (only 5/14 [35.7%] addressed bias clearly). Other criteria that should have been reported more comprehensively were the limitations of the studies and funding sources. The main study results of interest for this review, however, were clear and detailed for all included studies.

Supplementary II summarises the quality of the 14 papers included in the study using the Newcastle– Ottawa score. We determined the risk of bias in each article by assessing its ability to represent the exposed group, ascertain the exposed group, control for confounding, and make an outcome assessment. Notably, all studies

Ormaniam	6	Total	Descertis	0.5% 01	Weight	Weight
Organism	Cases s	amples	Proportio	n 95%-CI	(common)	(random)
Corynebacterium pseudomonas	7	1984 +	0.0	0 [0.00; 0.01]	0.6%	3.4%
Staphylococcus sp.	8	1984 +	0.0	0 [0.00; 0.01]	0.7%	3.4%
Veillonella sp.	1	185 +	- 0.0	1 [0.00; 0.03]	0.1%	1.8%
Bulkholderia cepacia	1	175 +		1 [0.00; 0.03]	0.1%	1.8%
E. faecium	1	175 +	- 0.0	1 [0.00; 0.03]	0.1%	1.8%
S. typhi	3	511 +	0.0	1 [0.00; 0.02]	0.2%	2.8%
Kluyvera sp.	1	151 +	- 0.0	1 [0.00; 0.04]		1.8%
Moraxella	1	150 +	0.0	1 [0.00; 0.04]		1.8%
Enterobacter	24	2589 +		1 [0.01; 0.01]		3.8%
Enterococci	2	197 -+	- 0.0	1 [0.00; 0.04]		2.5%
Proteus	30	2810 +		1 [0.01; 0.02]		3.8%
Acinetobacter	31	2656 +	0.0	1 [0.01; 0.02]		3.8%
R. orinthinolytica	2	166 -	•	1 [0.00; 0.04]		2.5%
Morganella morganii	2	151 -		1 [0.00; 0.05]		2.5%
Providencia sp.	2	151 -		1 [0.00; 0.05]		2.5%
Shigella	2	151 -		1 [0.00; 0.05]		2.5%
Yersinia sp.	2	151 -		1 [0.00; 0.05]		2.5%
Alcaligenes	4	301 -	0.0	1 [0.00; 0.03]		3.0%
Citrobacter	12	761 -		2 [0.01; 0.03]		3.6%
Streptococcus sp.	53	2787		2 [0.01; 0.02]		3.9%
P. aeruginosa	26			2 [0.01; 0.03]		3.8%
E. faecalis	44	2159		2 [0.01; 0.03]		3.9%
K. rhizophilia	1	44 —	0.0	2 [0.00; 0.12]		1.8%
S. paucimobilis	1	44 —		2 [0.00; 0.12]		1.8%
S. pseudointermedius	1	44 —		2 [0.00; 0.12]		1.8%
Bacillus	7	301 -		2 [0.01; 0.05]		3.4%
CoNS	93	3183		3 [0.02; 0.04]		3.9%
Corynebacterium	5	151 -		3 [0.01; 0.08]		3.2%
L. lactis	2	44 -		5 [0.01; 0.15]		2.4%
Klebsiella	190	3985		5 [0.04; 0.05]		3.9%
S. aureus	233	4248		5 [0.05; 0.06]		4.0%
S. epidermis	5	70	0.0	7 [0.02; 0.16]		3.1%
E. coli	467	4423		1 [0.10; 0.12]		4.0%
Mixed growth	32	191	0.1	7 [0.12; 0.23]	2.2%	3.8%
Common effect model		38408		5 [0.05; 0.05]		
Random effects model		_	> 0.0	2 [0.01; 0.03]		100.0%
Heterogeneity: $I^2 = 96\%$, $\tau^2 = 0.82$	10, <i>p</i> < 0.0		0.05 0.1 0.15 0.2 0.25 0.3			
		0	0.05 0.1 0.15 0.2 0.25 0.3			

Fig. 3 Pooled proportion of bacterial aetiological agents isolated from patients with suspected maternal infection in Sub-Saharan Africa

dropped at least three points, spread across 2–3 assessment categories, with 8/14 (57.1%) dropping two points in a single category and at particular risk of introducing bias. The main reasons for introducing bias were use of convenience sampling rather than random sampling of included patients, lack of laboratory facilities to perform anaerobic culture, minimal to no control of confounding (for e.g., antibiotic exposure, HIV status), no or insufficient differentiation in outcome assessment regarding commensal contamination versus pathogen.

Discussion

Here we performed a systematic review of the literature summarizing available aetiological and antimicrobial resistance data on bacterial maternal infections in SSA. Our study shows that *E. coli, S. aureus, and Klebsiella spp.* are the most common pathogens associated with maternal infections. Notably, *E. coli and Klebsiella spp.* are commonly and intrinsically resistant to penicillin, respectively. At least 21.5% of these isolates (*E. coli, S. aureus, and Klebsiella spp.*) exhibited resistance to other first-line and second-line antibiotics, including ciprofloxacin and gentamicin. Additionally, up to 39.6% of these isolates were resistant to ceftriaxone, a second-line treatment.

Our findings are consistent with the 2022 Global Antimicrobial Resistance and use Surveillance System (GLASS) [33]. In their report, the WHO reported third-generation cephalosporin- resistance for *E. coli* at 42%, *K. pneumoniae* between (59% – 65%) and

subgroups = Blood		
Acinetobacter		0.02 [0.01; 0.04]
Alcaligenes	* :	0.01 [0.00; 0.04]
Bacillus		0.03 [0.01; 0.08]
CoNS E. coli		0.02 [0.01; 0.05] 0.04 [0.03; 0.06]
Enterococci		0.01 [0.00; 0.04]
Klebsiella		0.04 [0.02; 0.06]
Moraxella		0.01 [0.00; 0.04]
P. aeruginosa	***	0.01 [0.00; 0.04]
R. orinthinolytica		0.01 [0.00; 0.04]
S. aureus	_ 	0.04 [0.02; 0.05]
S. typhi		0.01 [0.00; 0.03]
Veillonella sp. Random effects model		0.01 [0.00; 0.03] 0.02 [0.02; 0.03]
$l^2 = 48\% [1\%; 73\%], \chi^2_{12} = 23.06 (p = 0.03)$		0.02 [0.02, 0.03]
subgroups = Endocervical discharge		
Acinetobacter	- <u>-</u>	0.02 [0.00; 0.07]
Alcaligenes	-	0.02 [0.00; 0.06]
Bacillus		0.01 [0.00; 0.05]
Citrobacter	- Inter-	0.03 [0.01; 0.07]
CoNS		0.04 [0.02; 0.07] 0.03 [0.01; 0.08]
Corynebacterium E. coli		0.25 [0.22; 0.29]
Enterobacter	÷	0.03 [0.01; 0.06]
Klebsiella		0.07 [0.05; 0.09]
Kluyvera sp.	* i	0.01 [0.00; 0.04]
Mixed growth		0.17 [0.12; 0.23]
Morganella morganii		0.01 [0.00; 0.05]
P. aeruginosa	-	0.03 [0.02; 0.06]
Proteus Providencia sp.		0.03 [0.01; 0.08]
S. aureus		0.01 [0.00; 0.05] 0.14 [0.11; 0.17]
S. typhi	-	0.01 [0.00; 0.04]
Shigella	-	0.01 [0.00; 0.05]
Streptococcus sp.		0.07 [0.04; 0.09]
Yersinia sp.		0.01 [0.00; 0.05]
Random effects model	^	0.04 [0.02; 0.06]
$l^{2} = 94\% [92\%; 95\%], \chi_{19}^{2} = 302.1 (p < 0.01)$		
subaroups = Urine		
subgroups = Urine Acinetobacter		0.01 [0.00: 0.01]
Acinetobacter		0.01 [0.00; 0.01]
		0.01 [0.00; 0.01] 0.01 [0.00; 0.03] 0.01 [0.00; 0.02]
Acinetobacter Bulkholderia cepacia		0.01 [0.00; 0.03]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03]
Acinetobacter Bulkholderia cepacia Citrobacter CONS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.01] 0.00 [0.00; 0.01] 0.04 [0.03; 0.05]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus		$\begin{array}{cccc} 0.01 & [0.00; 0.03] \\ 0.01 & [0.00; 0.02] \\ 0.02 & [0.02; 0.03] \\ 0.00 & [0.00; 0.01] \\ 0.09 & [0.08; 0.10] \\ 0.02 & [0.01; 0.03] \\ 0.01 & [0.00; 0.03] \\ 0.00 & [0.00; 0.01] \\ 0.04 & [0.03; 0.05] \\ 0.01 & [0.01; 0.03] \\ 0.01 & [0.02; 0.01] \\ 0.02 & [0.02; 0.03] \\ 0.02 & [0.02$
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. typhi		$\begin{array}{cccc} 0.01 & [0.00; 0.03] \\ 0.01 & [0.00; 0.02] \\ 0.02 & [0.02; 0.03] \\ 0.00 & [0.00; 0.01] \\ 0.09 & [0.08; 0.10] \\ 0.02 & [0.01; 0.03] \\ 0.01 & [0.00; 0.03] \\ 0.00 & [0.00; 0.01] \\ 0.04 & [0.03; 0.05] \\ 0.01 & [0.01; 0.03] \\ 0.01 & [0.02; 0.03] \\ 0.02 & [0.02; 0.03] \\ 0.01 & [0.00; 0.03] \\ \end{array}$
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecialis E. faecialis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. surpui Staphylococcus sp.		$\begin{array}{cccccccccccccccccccccccccccccccccccc$
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. sureus S. typhi Staphylococcus sp. Streptococcus sp.		$\begin{array}{cccc} 0.01 & [0.00; 0.03] \\ 0.01 & [0.00; 0.02] \\ 0.02 & [0.02; 0.03] \\ 0.00 & [0.00; 0.01] \\ 0.09 & [0.08; 0.10] \\ 0.02 & [0.01; 0.03] \\ 0.01 & [0.00; 0.03] \\ 0.00 & [0.00; 0.01] \\ 0.04 & [0.03; 0.05] \\ 0.01 & [0.01; 0.03] \\ 0.01 & [0.00; 0.03] \\ 0.01 & [0.00; 0.03] \\ 0.01 & [0.00; 0.03] \\ 0.01 & [0.00; 0.01] \\ 0.00 & [0.00; 0.01] \\ 0.00 & [0.00; 0.01] \\ 0.01 & [0.00$
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Random effects model		$\begin{array}{cccccccccccccccccccccccccccccccccccc$
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. sureus S. typhi Staphylococcus sp. Streptococcus sp.		$\begin{array}{ccccc} 0.01 & [0.00; 0.03] \\ 0.01 & [0.00; 0.02] \\ 0.02 & [0.02; 0.03] \\ 0.00 & [0.00; 0.01] \\ 0.09 & [0.08; 0.10] \\ 0.02 & [0.01; 0.03] \\ 0.01 & [0.00; 0.03] \\ 0.00 & [0.00; 0.01] \\ 0.04 & [0.03; 0.05] \\ 0.01 & [0.01; 0.03] \\ 0.01 & [0.00; 0.03] \\ 0.01 & [0.00; 0.03] \\ 0.01 & [0.00; 0.03] \\ 0.01 & [0.00; 0.01] \\ 0.00 & [0.00; 0.01] \\ 0.00 & [0.00; 0.01] \\ 0.01 & [0.0$
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Random effects model		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.09 [0.08; 0.10] 0.09 [0.08; 0.10] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Kiebsiella P. aeruginosa Proteus S. aureus S. sureus S. typhi Staphylococcus sp. Streptococcus sp. Streptococcus sp. Random effects model $r^2 = 97\%$ (96%; 98%), $\chi^2_{15} = 520.72$ ($p < 0.01$ subgroups = Wound discharge Acinetobacter		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.02 [0.10; 0.35]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecialis E. faecialis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Streptococcus sp. Random effects model $t^2 = 97\%$ [96%; 98%], $\chi_{15}^2 = 520.72$ ($p < 0.01$ subgroups = Wound discharge Acinetobacter Citrobacter		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.20 [0.10; 0.35] 0.44 [0.01; 0.09]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Random effects model I [°] = 97% [96%; 98%], χ_{15}^2 = 520.72 (p < 0.01 subgroups = Wound discharge Acinetobacter Citrobacter Citrobacter CoNS		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.00 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.20 [0.10; 0.35] 0.44 [0.01; 0.03] 0.17 [0.11; 0.23]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. sureus S. sureus S. typhi Staphylococcus sp. Random effects model $r^2 = 97\% [96\%; 98\%], \chi^2_{15} = 520.72 (p < 0.01$ subgroups = Wound discharge Acinetobacter Citrobacter CoNS E. coli		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.20 [0.10; 0.35] 0.44 [0.01; 0.09] 0.14 [0.09; 0.19] 0.14 [0.09; 0.19]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. supti Staphylococcus sp. Streptococcus sp. Streptoccus sp. Streptococcus sp. Strept		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.20 [0.10; 0.35] 0.04 [0.01; 0.09] 0.17 [0.11; 0.23] 0.14 [0.09; 0.19] 0.18 [0.04; 0.13] 0.18 [0.04; 0.13]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. sureus S. sureus S. typhi Staphylococcus sp. Random effects model $r^2 = 97\% [96\%; 98\%], \chi^2_{15} = 520.72 (p < 0.01$ subgroups = Wound discharge Acinetobacter Citrobacter CoNS E. coli		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.01; 0.03] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.20 [0.10; 0.35] 0.44 [0.01; 0.09] 0.17 [0.11; 0.23] 0.44 [0.09; 0.19] 0.02 [0.00; 0.12]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. surpti Staphylococcus sp. Streptococcus sp. Str		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.20 [0.10; 0.35] 0.04 [0.01; 0.09] 0.17 [0.11; 0.23] 0.14 [0.09; 0.19] 0.18 [0.04; 0.13] 0.18 [0.04; 0.13]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecialis E. faecialis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Carbon effects model $t^2 = 97\%$ (96%; 98%), $\chi_{15}^2 = 520.72$ ($p < 0.01$ subgroups = Wound discharge Acinetobacter Citrobacter Citrobacter CoNS E. coli Enterobacter K. rhizophilia Klebsiella L. lactis P. aeruginosa		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.01; 0.03] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.04 [0.01; 0.03] 0.14 [0.01; 0.02] 0.08 [0.04; 0.13] 0.02 [0.00; 0.12] 0.16 [0.02; 0.16] 0.05 [0.01; 0.05]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Random effects model $I^2 = 97\%$ [96%; 98%], $\chi_{15}^2 = 520.72$ ($p < 0.01$ subgroups = Wound discharge Acinetobacter Citrobacter Citrobacter CoNS E. coli Enterobacter K. rhizophilia Klebsiella L. lactis P. aeruginosa Proteus		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.03] 0.04 [0.03; 0.05] 0.04 [0.03; 0.05] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.20 [0.10; 0.35] 0.04 [0.01; 0.09] 0.17 [0.11; 0.23] 0.14 [0.09; 0.19] 0.28 [0.04; 0.13] 0.02 [0.01; 0.15] 0.04 [0.01; 0.05] 0.05 [0.01; 0.05] 0.06 [0.04; 0.10]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Streptococcus sp. Random effects model $r^2 = 97\%$ (96%; 98%), $\chi^2_{15} = 520.72$ ($p < 0.01$ subgroups = Wound discharge Acinetobacter Citrobacter CoNS E. coli Enterobacter K. rhizophilia Klebsiella L. lactis P. aeruginosa Proteus S. aureus		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.00 [0.01; 0.035] 0.04 [0.01; 0.09] 0.17 [0.11; 0.23] 0.14 [0.09; 0.19] 0.08 [0.04; 0.13] 0.02 [0.01; 0.15] 0.05 [0.01; 0.15] 0.28 [0.22; 0.34]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Streptoccus sp. Streptococcus sp. Strept		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.03] 0.04 [0.03; 0.05] 0.04 [0.01; 0.03] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.04 [0.01; 0.02] 0.04 [0.01; 0.09] 0.17 [0.11; 0.23] 0.14 [0.09; 0.19] 0.05 [0.01; 0.15] 0.05 [0.01; 0.15] 0.05 [0.01; 0.15] 0.06 [0.04; 0.10] 0.28 [0.22; 0.34] 0.70 [0.02; 0.16]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Random effects model $I' = 97\%$ [96%; 98%], $\chi_{15}^2 = 520.72$ ($p < 0.01$ subgroups = Wound discharge Acinetobacter Citrobacter CoNS E. coli Enterobacter Citr		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.02 [0.10; 0.35] 0.04 [0.01; 0.09] 0.17 [0.11; 0.23] 0.14 [0.09; 0.19] 0.08 [0.04; 0.13] 0.04 [0.00; 0.12] 0.11 [0.07; 0.16] 0.02 [0.01; 0.05] 0.06 [0.04; 0.10] 0.22 [0.22; 0.34] 0.07 [0.02; 0.16] 0.02 [0.00; 0.12]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Streptococcus sp. Random effects model $r^2 = 97\%$ [96%; 98%], $\chi^2_{15} = 520.72 (p < 0.01$ subgroups = Wound discharge Acinetobacter Citrobacter CoNS E. coli Enterobacter K. rhizophilia Klebsiella L. lactis P. aeruginosa Proteus S. aureus S. aureus S. paucimobilis S. pseudointermedius		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.00 [0.00; 0.03] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.02 [0.10; 0.35] 0.04 [0.01; 0.09] 0.17 [0.11; 0.23] 0.14 [0.09; 0.19] 0.08 [0.04; 0.13] 0.02 [0.01; 0.15] 0.02 [0.01; 0.15] 0.02 [0.01; 0.15] 0.02 [0.01; 0.15] 0.02 [0.01; 0.15] 0.02 [0.01; 0.15] 0.02 [0.00; 0.12] 0.02 [0.00; 0
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Random effects model $I' = 97\%$ [96%; 98%], $\chi_{15}^2 = 520.72$ ($p < 0.01$ subgroups = Wound discharge Acinetobacter Citrobacter CoNS E. coli Enterobacter Citr		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.00 [0.00; 0.01] 0.04 [0.03; 0.05] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.01; 0.02] 0.02 [0.10; 0.35] 0.04 [0.01; 0.09] 0.17 [0.11; 0.23] 0.14 [0.09; 0.19] 0.08 [0.04; 0.13] 0.04 [0.00; 0.12] 0.11 [0.07; 0.16] 0.02 [0.01; 0.05] 0.06 [0.04; 0.10] 0.22 [0.22; 0.34] 0.07 [0.02; 0.16] 0.02 [0.00; 0.12]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Subgroups = Wound discharge Acinetobacter Citrobacter Citrobacter Citrobacter CoNS E. coli Enterobacter K. rhizophilia Klebsiella L. lactis P. aeruginosa Proteus S. aureus S. epidermis S. paucimobilis S. pseudointermedius		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.11] 0.02 [0.01; 0.05] 0.08 [0.04; 0.13] 0.02 [0.01; 0.15] 0.02 [0.01; 0.15] 0.02 [0.01; 0.05] 0.06 [0.24; 0.34] 0.07 [0.22; 0.34] 0.07 [0.02; 0.12] 0.25 [0.02; 0.09]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Streptococcus sp. Constructional discharge Acinetobacter Citrobacter CoNS E. coli Enterobacter CoNS E. coli Enterobacter Citrobacter CoNS E. coli Enterobacter Constructional Enterobacter S. aureus S. aureus S. aureus S. epidermis S. paucimobilis S. pseudointermedius Streptococcus sp. Random effects model $I^2 = 86\%$ (79%; 91%), $\chi_{14}^2 = 103.48$ ($p < 0.01$		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.02 [0.01; 0.05] 0.04 [0.01; 0.05] 0.05 [0.02; 0.09] 0.02 [0.00; 0.12] 0.05 [0.02; 0.09] 0.08 [0.05; 0.12] 0.05 [0.02; 0.09] 0.08 [0.05; 0.12]
Acinetobacter Bulkholderia cepacia Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. typhi Staphylococcus sp. Random effects model $I' = 97\%$ [96%; 98%], $\chi_{15} = 520.72$ ($p < 0.01$ subgroups = Wound discharge Acinetobacter Citrobacter CoNS E. coli Enterobacter Citrobacter CoNS E. coli Enterobacter K. rhizophilia Klebsiella L. lactis P. aeruginosa Proteus S. aureus S. aureus S. paucimobilis S. pseudointermedius Streptococcus sp.		0.01 [0.00; 0.03] 0.01 [0.00; 0.02] 0.02 [0.02; 0.03] 0.00 [0.00; 0.01] 0.09 [0.08; 0.10] 0.02 [0.01; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.03] 0.01 [0.00; 0.01] 0.02 [0.02; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.01] 0.01 [0.00; 0.03] 0.01 [0.00; 0.01] 0.01 [0.00; 0.11] 0.02 [0.01; 0.05] 0.08 [0.04; 0.13] 0.02 [0.01; 0.15] 0.02 [0.01; 0.15] 0.02 [0.01; 0.05] 0.06 [0.24; 0.34] 0.07 [0.22; 0.34] 0.07 [0.02; 0.12] 0.25 [0.02; 0.09]

Fig. 4 Pooled prevalence of bacterial causative agents- sub-group analysis by sample type

Organism	Cases orç	Total janisms		Proportion	95%-CI	Weight (common)	Weight (random)
Kluyvera sp.	1	124 +		0.01	[0.00; 0.04]	0.1%	1.8%
Morganella morganii	2	124 +			[0.00; 0.06]	0.2%	2.5%
Providencia sp.	2	124 🕂		0.02	[0.00; 0.06]	0.2%	2.5%
Shigella	2	124 +		0.02	[0.00; 0.06]	0.2%	2.5%
Yersinia sp.	2	124 +		0.02	[0.00; 0.06]	0.2%	2.5%
K. rhizophilia	1	57 🕂		0.02	[0.00; 0.09]	0.1%	1.8%
S. paucimobilis	1	57 🕂			[0.00; 0.09]	0.1%	1.8%
S. pseudointermedius	1	57 🕂			[0.00; 0.09]	0.1%	1.8%
S. typhi	3	157 +			[0.00; 0.05]	0.3%	2.8%
Corynebacterium pseudomonas		333 +		0.02	[0.01; 0.04]	0.7%	3.4%
Staphylococcus sp.	8	333 +			[0.01; 0.05]	0.8%	3.5%
Alcaligenes	4	138 🕂			[0.01; 0.07]	0.4%	3.0%
Citrobacter	12	386 +			[0.02; 0.05]	1.1%	3.6%
Enterobacter	24	735 +			[0.02; 0.05]	2.3%	3.8%
L. lactis	2	57 🕂			[0.00; 0.12]	0.2%	2.4%
Bulkholderia cepacia	1	28			[0.00; 0.18]	0.1%	1.8%
E. faecium	1	28 + +			[0.00; 0.18]	0.1%	1.8%
R. orinthinolytica	2	56 🕂			[0.00; 0.12]	0.2%	2.4%
P. aeruginosa	26	675 +			[0.03; 0.06]	2.4%	3.8%
Proteus	30	758 +			[0.03; 0.06]	2.8%	3.8%
Corynebacterium	5	124 🕂			[0.01; 0.09]	0.5%	3.2%
Bacillus	7	138 🕂			[0.02; 0.10]	0.6%	3.4%
Acinetobacter	31	540 +			[0.04; 0.08]	2.8%	3.8%
Streptococcus sp.	53	864 +			[0.05; 0.08]	4.9%	3.9%
Moraxella	1	14	-		[0.00; 0.34]	0.1%	1.7%
Enterococci	2	22			[0.01; 0.29]	0.2%	2.4%
CoNS	93	854 =			[0.09; 0.13]	8.1%	4.0%
S. epidermis	5	45			[0.04; 0.24]	0.4%	3.1%
E. faecalis	44	361 +			[0.09; 0.16]	3.8%	3.9%
Klebsiella	190	1269			[0.13; 0.17]	15.7%	4.0%
S. aureus	233	1281			[0.16; 0.20]	18.6%	4.0%
Veillonella sp.	1	5			[0.01; 0.72]	0.1%	1.6%
Mixed growth	32	158	+-		[0.14; 0.27]	2.5%	3.8%
E. coli	467	1309	+	0.36	[0.33; 0.38]	29.3%	4.0%
Common effect model		11459		0.16	[0.15; 0.16]	100.0%	
Random effects model		<u> </u>			[0.04; 0.07]		100.0%
Heterogeneity: $I^2 = 96\%$, $\tau^2 = 0.81$	11, <i>p</i> < 0.01						
		0 0.2	0.4 0.6 0	0.8 1			

Fig. 5 Pooled proportions of bacterial causative agents amongst the positive isolates

methicillin-resistant S. aureus (MRSA) at 35% causing bloodstream infections [33]. In addition, resistance rates of E. coli to first-line antibiotics (penicillins) and second-line drugs (ciprofloxacin) were both > 20% and of great concern. A notable limitation of GLASS data is the lack of stratified data on age and gender, and it did not specifically look at the pregnant/post-partum population to inform the global picture of bacterial infection and AMR. This challenge limits the data interpretation in the context of maternal infections. For this population analysing AMR surveillance data by age groups and infection types are crucial to informing and directing mitigation strategies and interventions to control the mechanisms of the spread of the causative agents and AMR. In addition, some antibiotics used in the general population cannot be used in pregnancy due to adverse effects on the unborn infant. Therefore, these factors need to be accounted for in the surveillance systems.

Our systematic review and meta-analysis provide stratified maternal infection data, but it has some limitations. For example, the reporting AST of *K. pneumoniae* to co-trimoxazole and carbapenems is of public health relevance because resistance genes in *Enterobacterales* to these antibiotics are frequently associated with mobile genetic elements that increase the likelihood of pan-drug-resistant and extreme-drug-resistant isolates. Our study could not assess the resistance of carbapenems and co-trimoxazole due to limited data to make such pooled estimates. This likely reflects the local prescribing behaviour as third-generation drugs such as carbapenems are rarely available. Nonetheless, rising ESBL rates may incentivize the use of carbapenems and co-trimoxazole, making urgent surveillance of resistance to these

subgroups = Blood Acinetobacter Alcaligenes Bacillus CoNS E. coli Enterococci Kiebsiella Moraxella P. aeruginosa R. orinthinolytica S. aureus S. typhi Veillonella sp. Random effects model l^2 = 57% [21%; 77%], χ^2_{12} = 28.14 (p < 0.01 subgroups = Endocervical discharge		0.11 [0.05; 0.22] 0.07 [0.00; 0.34] 0.36 [0.13; 0.65] 0.10 [0.04; 0.20] 0.28 [0.19; 0.38] 0.09 [0.01; 0.28] 0.17 [0.00; 0.28] 0.07 [0.00; 0.12] 0.48 [0.00; 0.12] 0.49 [0.00; 0.12] 0.27 [0.18; 0.37] 0.20 [0.01; 0.72] 0.20 [0.01; 0.72] 0.16 [0.11; 0.22]
Acinetobacter	-	0.03 [0.00; 0.11]
Alcaligenes		0.02 [0.01; 0.07]
Bacillus Citrobacter		0.02 [0.00; 0.06] 0.03 [0.01; 0.08]
CoNS	-	0.05 [0.03; 0.10]
Corynebacterium		0.04 [0.01; 0.09]
E. coli		0.38 [0.33; 0.43]
Enterobacter		0.04 [0.02; 0.08]
Klebsiella Kluyvera sp.	F	0.11 [0.08; 0.14] 0.01 [0.00; 0.04]
Mixed growth		0.20 [0.14; 0.27]
Morganella morganii	Ref. 1	0.02 [0.00; 0.06]
P. aeruginosa Proteus		0.04 [0.02; 0.07] 0.05 [0.01; 0.14]
Providencia sp.	E	0.02 [0.00; 0.06]
S. aureus		0.21 [0.17; 0.25]
S. typhi Shigella	180- 190-	0.01 [0.00; 0.04] 0.02 [0.00; 0.06]
Streptococcus sp.	- <u>+</u>	0.08 [0.06; 0.12]
Yersinia sp.	F	0.02 [0.00; 0.06]
Random effects model $l^2 = 95\% [93\%; 96\%], \chi^2_{19} = 351.54 (p < 0.0)$	1)	0.05 [0.03; 0.08]
$r = 35\%$ [35%, 36%], $\chi_{19} = 351.54$ ($p < 0.0$	"	
subgroups = Urine		
Acinetobacter		0.04 [0.02; 0.06]
Bulkholderia cenacia	- 192	
Bulkholderia cepacia Citrobacter		0.04 [0.00; 0.18] 0.02 [0.01; 0.06]
Citrobacter CoNS		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15]
Citrobacter CoNS Corynebacterium pseudomonas		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04]
Citrobacter CoNS		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15]
Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.12 [0.09; 0.16] 0.04 [0.00; 0.18]
Citrobacter CoNS Connebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.12 [0.09; 0.16] 0.04 [0.00; 0.18] 0.01 [0.00; 0.03]
Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.12 [0.09; 0.16] 0.04 [0.00; 0.18]
Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus		$\begin{array}{cccccccccccccccccccccccccccccccccccc$
Citrobacter CoNNS Conynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.14 [0.09; 0.16] 0.04 [0.00; 0.18] 0.01 [0.00; 0.03] 0.19 [0.16; 0.22] 0.05 [0.02; 0.09] 0.3 [0.01; 0.04] 0.10 [0.08; 0.13]
Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.12 [0.09; 0.16] 0.04 [0.00; 0.18] 0.01 [0.00; 0.03] 0.19 [0.16; 0.22] 0.05 [0.02; 0.09] 0.03 [0.01; 0.04] 0.10 [0.08; 0.13]
Citrobacter CoNNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. typhi Staphylococcus sp. Steptococcus sp.		$\begin{array}{cccccccccccccccccccccccccccccccccccc$
Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Random effects model		$\begin{array}{cccccccccccccccccccccccccccccccccccc$
Citrobacter CoNNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. taphylococcus sp. Staphylococcus sp. Staphylococcus sp. Staphylococcus sp. Random effects model $t^2 = 97\%$ [96%; 98%], $\chi^2_{15} = 540.12$ ($p < 0.0$		$\begin{array}{cccccccccccccccccccccccccccccccccccc$
Citrobacter CoNNe Conynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. taphylococcus sp. Straptococcus sp. Random effects model I ² = 97% [96%; 98%], X ₁₅ = 540.12 (p < 0.0 subgroups = Wound discharge		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.14 [0.00; 0.18] 0.04 [0.00; 0.18] 0.01 [0.00; 0.03] 0.19 [0.16; 0.22] 0.05 [0.02; 0.09] 0.03 [0.01; 0.04] 0.10 [0.08; 0.13] 0.04 [0.00; 0.18] 0.04 [0.02; 0.07] 0.04 [0.02; 0.07] 0.06 [0.03; 0.10]
Citrobacter Colvebacterium pseudomonas E. coli E. faecalis E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Random effects model $l^2 = 97\%$ [96%; 98%], $\chi^2_{15} = 540.12$ ($p < 0.0$ subgroups = Wound discharge Acinetobacter		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.12 [0.09; 0.16] 0.04 [0.00; 0.18] 0.01 [0.00; 0.03] 0.19 [0.16; 0.22] 0.05 [0.02; 0.09] 0.03 [0.01; 0.04] 0.10 [0.08; 0.13] 0.04 [0.00; 0.18] 0.02 [0.01; 0.05] 0.04 [0.02; 0.07] 0.06 [0.03; 0.10] 0.16 [0.07; 0.28]
Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. aureus S. taphylococcus sp. Staphylococcus sp. Staphylococcus sp. Random effects model $t^2 = 97\%$ [96%; 98%], $\chi_{15}^2 = 540.12$ ($p < 0.0$ subgroups = Wound discharge Acinetobacter Citrobacter CoNS		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.14 [0.00; 0.18] 0.04 [0.00; 0.18] 0.01 [0.00; 0.03] 0.19 [0.16; 0.22] 0.05 [0.02; 0.09] 0.03 [0.01; 0.04] 0.10 [0.08; 0.13] 0.04 [0.00; 0.18] 0.04 [0.02; 0.07] 0.04 [0.02; 0.07] 0.06 [0.03; 0.10]
Citrobacter CoNS Corynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Random effects model $l^2 = 97\%$ [96%; 98%], $\chi_{15}^2 = 540.12$ ($p < 0.0$ subgroups = Wound discharge Acinetobacter Citrobacter CoNS E. coli		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.12 [0.09; 0.16] 0.04 [0.00; 0.18] 0.01 [0.00; 0.03] 0.19 [0.16; 0.22] 0.05 [0.02; 0.09] 0.03 [0.01; 0.04] 0.00 [0.03; 0.13] 0.04 [0.00; 0.18] 0.02 [0.01; 0.05] 0.04 [0.02; 0.07] 0.06 [0.03; 0.10] 0.16 [0.07; 0.28] 0.16 [0.01; 0.10] 0.16 [0.11; 0.22] 0.15 [0.10; 0.20]
Citrobacter CoNNS Conynebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. typhi Staphylococcus sp. Streptococcus sp. Streptococcus sp. Random effects model I ² = 97% [96%; 98%], 2 ₁₅ = 540.12 (p < 0.0 subgroups = Wound discharge Acinetobacter Citrobacter Citrobacter CoNS E. coli Enterobacter		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.12 [0.09; 0.16] 0.04 [0.00; 0.18] 0.01 [0.00; 0.03] 0.19 [0.16; 0.22] 0.05 [0.02; 0.09] 0.03 [0.01; 0.04] 0.10 [0.08; 0.13] 0.04 [0.00; 0.18] 0.04 [0.00; 0.16] 0.04 [0.02; 0.07] 0.06 [0.03; 0.10] 0.16 [0.07; 0.28] 0.04 [0.01; 0.10] 0.15 [0.10; 0.20] 0.08 [0.04; 0.13] 0.09 [0.04] 0.10 [0.01; 0.10] 0.16 [0.11; 0.22] 0.15 [0.10; 0.20] 0.08 [0.04; 0.13]
Citrobacter CoNNe Connebacterium pseudomonas E. coli E. faecalis E. faecium Enterobacter Klebsiella P. aeruginosa Proteus S. aureus S. tarptococcus sp. Straptococcus sp. Straptococcus sp. Random effects model $l^2 = 97\%$ [96%; 98%], $\chi_{15}^2 = 540.12$ ($p < 0.0$ subgroups = Wound discharge Acinetobacter Citrobacter CoNS E. coli Enterobacter K. rhizophilia Klebsiella		0.04 [0.00; 0.18] 0.02 [0.01; 0.06] 0.12 [0.09; 0.15] 0.02 [0.01; 0.04] 0.43 [0.38; 0.47] 0.12 [0.09; 0.15] 0.04 [0.00; 0.18] 0.01 [0.00; 0.03] 0.19 [0.16; 0.22] 0.05 [0.02; 0.09] 0.03 [0.01; 0.04] 0.00 [0.03; 0.13] 0.04 [0.00; 0.18] 0.02 [0.01; 0.05] 0.04 [0.02; 0.07] 0.06 [0.03; 0.10] 0.16 [0.07; 0.28] 0.44 [0.01; 0.10] 0.16 [0.11; 0.22] 0.18 [0.04; 0.13] 0.20 [0.00; 0.09] 0.12 [0.08; 0.18]
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Fig. 6 Sub-group analysis by sample type of pooled proportions of bacterial causative agents amongst the positive isolates

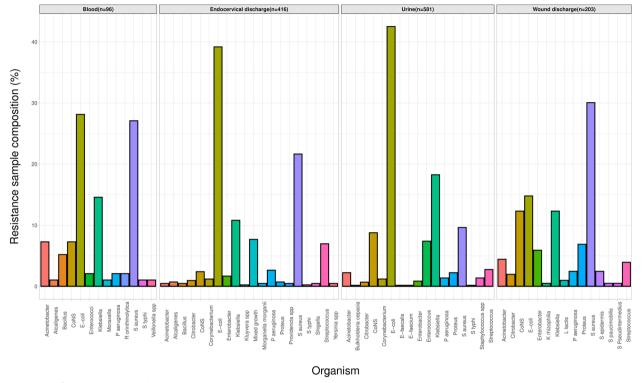


Fig. 7 Stratified resistance sample composition

antibiotics essential. Another challenge was that the studies included in our meta-analyses were performed in government and mostly tertiary hospitals and not private or primary healthcare facilities. Therefore, the data presented here may be skewed in the direction of hospitalacquired (resistant) bacterial infection as prior antibiotic exposure was not always reported and/or accounted for. Nonetheless, most patients in SSA with severe maternal infection will attend government hospitals.

Quality assessment of the papers shows some areas of concern. Most studies report convenience sampling of patients with suspected maternal sepsis and no randomized sampling was reported, which may limit the generalizability of the findings to the wider maternal population. This in addition to the lack of information on prior antibiotic exposure means there may be common infectious agents responsive to empiric treatments which were not captured within this review. Limited studies reported anaerobic culturing methods meaning important pathogens could have been missed. Only 3/7 studies reported in their methodology that they accounted for contaminants making data from non-sterile sampling sites such as wounds and cervicovaginal swabs difficult to interpret [19-21, 24, 27, 29, 31]. Also, some studies reported resistance to penicillin by Gram-negative causative agents (known to be inherently resistant to penicillin). We would have liked to stratify our analyses by important factors such as HIV status, considering that HIV increases the risk of maternal morbidity and mortality, but HIV-status data was missing for most studies. Majangara et al. [27] was the only study reviewed here that stratified data according to HIV status and reported increased length of hospitalization stay for HIV positive women with puerperal sepsis but they did not find a specific microbial agent that significantly associated with HIV status.

We expected to see high clinical and statistical heterogeneity, as demonstrated by the results (inter- and intrastudy heterogeneity 88–92% in all the meta-analyses performed). This heterogeneity can be explained by our summary table and subgroup analysis by sample type. However, pooled estimates from these papers are still clinically relevant.

In light of these findings, several crucial implications emerge. Firstly, the apparent deficiency in data underscores the pressing need for concerted efforts in systematic data collection, particularly from government laboratories. Such initiatives are essential not only for assessing sensitivities but also for obtaining accurate and comprehensive rates of AMR. The limited availability of robust data poses a significant challenge to achieving the WHO's universal health care ambition. Additionally,

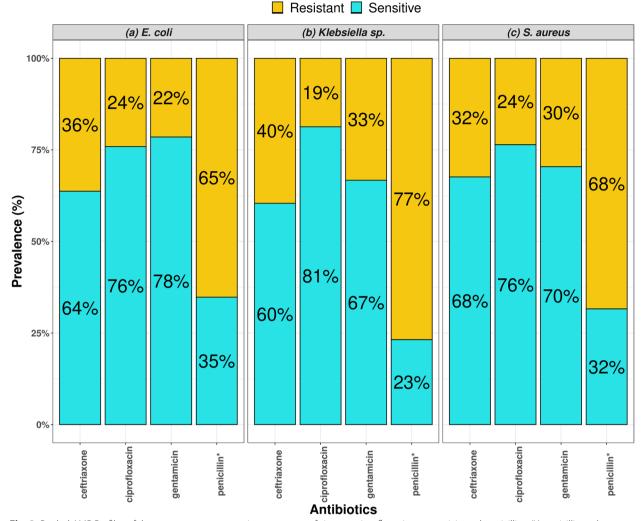


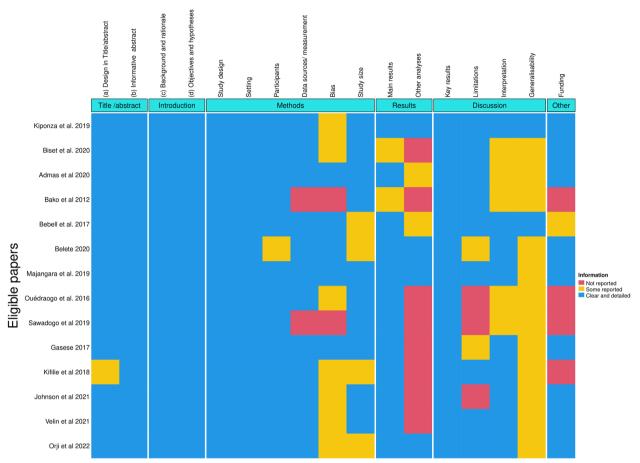
Fig. 8 Pooled AMR Profiles of the most common causative agents to ceftriaxone, ciprofloxacin, gentamicin and penicillins. *Amoxicillin and Ampicillin, (a-b) gram negative species and (c) gram positive species

the identified low rates of bacterial detection in laboratory settings raise questions about the utility of current practices. It prompts reflection on whether these detection rates are reasonable, considering their potential impact on the quality of treatment. The ability to deliver high-quality treatment is directly influenced by the accuracy and reliability of laboratory results, emphasizing the importance of addressing and rectifying potential limitations in detection methods.

Furthermore, the documented high rates of treatment failure (for invasive and non-invasive bacterial infections), particularly alarming in resource-constrained populations, emphasize the critical need for immediate attention. In economically challenged settings where financial constraints limit the purchase of various antibiotics, and follow-up is challenging in the face of detected resistance, effective treatment becomes a formidable challenge. This warrants a comprehensive re-evaluation of current guidelines and practices, especially in the context of syndromic treatment. The data strongly suggests the necessity for revisions to existing guidance to ensure that the first-line treatment is appropriately targeted and effective.

Conclusion

Our findings highlight the need for high-quality surveillance for maternal microbiological data in SSA, including stratification according to the target demographic population. The results indicate a high prevalence of resistance in common causative agents of maternal infections to essential antimicrobial drugs in empirical treatment guidelines. These include ampicillin (a penicillin) as first-line antibiotics for treating maternal peripartum infection in combination with gentamicin. AMR has also



Paper components



been observed in amoxicillin and ceftriaxone, the highest priority agent among the critically important antimicrobials for human medicine. Our findings also flag the need to review local and international treatment guidelines for maternal bacterial infections in SSA.

Supplementary Information

The online version contains supplementary material available at https://doi. org/10.1186/s12879-024-09855-3.

Supplementary 1. PUBMED search strategy

Supplementary 2. Microsoft Excel Workbook of the Newcastle Ottawa Assessment Scale

Clinical trial number Not applicable.

Authors' contributions

DL is the guarantor of this manuscript. DL, CC and CVD developed the research question. CC, CVD and AS devised the search strategy. CC, CVD, EJMM and EB performed data extraction and quality assessments of the

papers. CC, HHT and JF performed the data analysis. AW, NF, SL, DK, LG, MN, CM, TN, CD and MKM made substantial contributions to the study design. CC and CVD drafted the manuscript. All authors have read and approved the final manuscript.

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Availability of data and materials

Data are available upon reasonable request to the corresponding author.

Declarations

Ethical approval and consent to participate Not applicable.

Competing interests

The authors declare no competing interests.

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