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Antimicrobial resistance of bacterial pathogens isolated from cancer patients: a systematic review and meta-analysis

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Abstract

Background Antimicrobial resistance (AMR) is a major threat to global public health, limiting treatment options for infections. AMR is particularly life-threatening for cancer patients, who are at increased risk of antibiotic-resistant infections. This review presents the first comprehensive data on the prevalence of AMR in major bacterial pathogens isolated from cancer patients.

Method An extensive search was conducted in PubMed, Scopus, and Web of Science, focusing on studies published in English from 2000 to 2024. A single-group meta-analysis was performed to determine the resistance prevalence of major bacterial species.

Results One hundred thirty-two full-text articles were included in the systematic review, and studies on haematological cancer patients were the most common (36.4%). The major bacterial pathogens reported were *Escherichia coli*, *Klebsiella pneumoniae*, *Staphylococcus aureus*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa, Enterococcus faecium, Streptococcus pneumoniae* and *Enterobacter* spp. For *E. coli*, resistance prevalence was highest for penicillins (81.84%), followed by cotrimoxazole (65.79%) and monobactams (61.61%). For *K. pneumoniae*, the highest prevalence of resistance was observed for penicillins (98.99%), followed by cotrimoxazole (70.92%). *Acinetobacter baumannii* had high resistance prevalence to multiple antimicrobial classes, including third-generation cephalosporins (84.10%), fourth-generation cephalosporins (80.75%), carbapenems (82.58%), fluoroquinolones (80.37%), beta-lactam-beta-lactamase inhibitors (79.15%), cotrimoxazole (75.77%), and aminoglycosides (64.05%). *Enterobacter* spp. and *Enterococcus faecium* showed high resistance prevalence to penicillins at 91.77% and 90.64% respectively. *P. aeruginosa* had a high prevalence of resistance to third-generation cephalosporins (49.41%) while *S. aureus* showed high prevalence to macrolides (55.63%) and methicillin (45.29%).

Conclusion This review indicated a high prevalence of antimicrobial resistance in bacterial pathogens isolated from cancer patients worldwide. The pronounced resistance prevalence observed, especially among ESKAPE pathogens, underscores the urgent need to improve infection prevention and antimicrobial stewardship in cancer care globally.

Keywords Antimicrobial resistance, Cancer patients, Antimicrobial classes, Prevalence, Haematological, Bacteria, Infection

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Introduction

The emergence and dissemination of antimicrobial-resistant pathogens are increasingly limiting the available treatments for infection [1]. Antimicrobial resistance (AMR) has gained much attention in recent decades and has been recognized as a major public health threat by the World Health Organization (WHO). From 2019 to 2022, the estimated number of deaths associated with AMR exceeded 5 million [2, 3]. In the absence of significant effort and proactive strategies to combat this global health crisis, the projected number of deaths is estimated to reach 10 million per year by 2050 [3]. While antimicrobial resistance is a natural survival phenomenon exhibited by bacteria, several factors, such as the inappropriate use of antibiotics in humans and livestock and the presence of antibiotics in the environment, contribute to the increase in AMR [4, 5].

Like AMR, cancer is also a major public health threat, with a global incidence of 19 million new cases and 9 million deaths in 2022 [6]. The growing global cancer burden is driven by several factors, including the aging and growth of the population, genetic predispositions, environmental factors and personal lifestyle choices [6, 7]. Patients with cancer face a high risk of acquiring antibiotic-resistant infections due to frequent hospital visits and admissions, prior antibiotic exposures, surgeries, the use of urinary catheters, and comorbidities [8]. The nature of the disease and its treatments, such as chemotherapy and bone marrow transplantation, leave cancer patients with compromised immune systems, making them less likely to survive fatal infections compared to healthy individuals [9]. The growing concern of AMR is significantly compromising cancer care, resulting in increased morbidity, mortality, and healthcare costs [8, 10, 11]. Multiple studies have shown that a wide range of resistant pathogens are associated with poorer outcomes in cancer patients [12, 13]. According to a systematic review of clinical outcomes of antimicrobial resistance in cancer patients, mortality was the most frequent outcome, occurring in 47% of the studies included, highlighting the significant impact of AMR on the survival of cancer patients [14].

A significant proportion of cancer patients are immunocompromised, making them more prone to bacterial infections, which further exacerbates their already fragile health. The rise of AMR further complicates treatment, creating a perfect storm of vulnerability and limited options that put cancer patients at risk of fatal outcomes. Notably, despite the plethora of research data on AMR in cancer patients, a systematic review of AMR patterns in this population is still lacking. To address this gap, our study aims to provide a comprehensive analysis of the global prevalence of AMR in common pathogenic bacteria causing infections in cancer patients.

Methods

Search strategy

A comprehensive database search for eligible publications on antimicrobial resistance in pathogens isolated from cancer patients was conducted in PubMed, Scopus, and the Web of Science. The search was conducted using the keywords described in Table 1. The search was restricted to full-text research articles published in English from 2000 to 2024. The review adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [15]. Additionally, the review protocol was registered on the Open Science Framework (OSF) website, available at https://doi.org/10.17605/OSF.IO/SNPUV.

Table 1 Keywords used in the various databases

PubMed	
Keywords	(Drug Resistance, Bacterial [Mesh] OR Antibacterial resistan* OR Antimicrobial resistan* OR Antibiotic Resistan* OR Drug Resistance, Multiple, Bacterial [Mesh] OR Multidrug resistan* OR Multidrug resistan* OR Multiple drug resistan* OR AMR) AND (Neoplasms [Mesh] OR Cancer* OR Malignan*) AND (Bacterial Infections [Mesh] OR Staphylococcus aureus OR Streptococcus pneumoniae OR Enterococcus faecium OR Escherichia coli OR Klebsiella pneumoniae OR Pseudomonas aeruginosa OR Acinetobacter baumannii OR Enterobacter spp.)
Limiters	Text availability – Free full-text; Language – English
Scopus	
Keywords	("antibacterial resistance" OR "antimicrobial resistance" OR "antibiotic resistance" OR "multidrug resistance" OR "multidrug resistance") AND ("cancer*" OR "malignan*") AND ("bacteria" OR "staphylococcus aureus" OR "streptococcus pneumoniae" OR "enterococcus faecium" OR "escherichia coli" OR "klebsiella pneumoniae" OR "pseudomonas aeruginosa" OR "acinetobacter baumannii" OR "enterobacter spp") AND ("infection*")
Limiters	Document type – Articles; Language – English
Web of Science	
Keywords	("antibacterial resistance" OR "antimicrobial resistance" OR "antibiotic resistance" OR "multidrug resistance" OR "multidrug resistance") AND ("cancer*" OR "malignan*") AND ("bacteria" OR "staphylococcus aureus" OR "streptococcus pneumoniae" OR "enterococcus faecium" OR "escherichia coli" OR "klebsiella pneumoniae" OR "pseudomonas aeruginosa" OR "acinetobacter baumannii" OR "enterobacter spp") AND ("infection*")
Limiters	Document type – Articles; Language – English

Study eligibility and selection criteria

This systematic review included studies that reported antimicrobial resistance in pathogenic bacteria infecting cancer patients and presented data based on the specific bacterial species and antimicrobial drugs tested. The pathogenic bacteria included in this study were *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Enterococcus faecium*, *Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, and *Enterobacter* spp. Additionally, studies reporting resistant isolates such as methicillin-resistant *S. aureus* (MRSA) and extended-spectrum beta-lactamase (ESBL)-producing *K. pneumoniae* or *E. coli* were also included in the study.

Reviews, case series or reports, commentaries, and editorials were excluded in this systematic review. Studies that did not differentiate resistance by specific bacterial species and instead grouped related bacterial species (such as *E. coli* and *K. pneumoniae* from the *Enterobacteriaceae* family) or categorized them based on their Gram reaction (gram-negative or gram-positive organisms) were excluded. Likewise, studies that reported resistance in bacterial species not listed in the inclusion criteria were also excluded from the analysis. Two investigators (O.K.N. and A. A.-D.) independently screened and assessed the eligibility of the studies. Duplicates were first identified and removed, followed by titles and abstracts screening, and a full-text assessment to identify eligible studies. The results of the two investigators were carefully compared, with disagreements resolved through consensus discussion.

Data extraction and analysis

Microsoft Excel 365 was used to manage and visualize all the data extracted from studies included in the systematic review. The data extracted included the geographical region, author and year of publication, year of study, study design, type of cancer, number of cancer patients, isolate type, isolate site, and method of sensitivity testing. The antibiotics selected for each organism were based on the lists of antibiotics recommended by the Clinical Laboratory Standard Institute (CLSI). Two reviewers (O.K.N. and E.S.D.) extracted the relevant data from the studies.

The meta-analysis was conducted using the meta package in RStudio version 4.3.3. The Freeman-Tukey double arcsine transformation was used to stabilize variances among studies, and the DerSimonian–Laird method was employed to determine the pooled resistance. Heterogeneity in the extracted data was assessed using I-squared statistic, which can be interpreted as low, moderate, or high heterogeneity using values $\leq 25\%$, 50%, and $\geq 75\%$, respectively. The confidence intervals for heterogeneity were generated using the Jackson method. A funnel plot and Egger's test were used to visualize and statistically assess publication bias, respectively. A p-value of < 0.05 was considered to indicate statistical significance.

Sensitivity analysis (excluding low-quality studies) and meta-regression were conducted to assess the robustness of synthesized results and sources of heterogeneity respectively. An alpha value of < 0.05 was considered statistically significant.

Quality assessment

The quality of the included studies was assessed by two investigators (O.K.N., and A.-H. O.) using the STROBE checklist for reporting observational studies. The checklist was modified to include 15 items, where each item was either answered 'YES' if the study provided adequate information or 'NO' if the study had no information or unclear description. Studies were graded into three categories: high quality (yes for 11–15 items), moderate quality (yes for 6–10 items), and low quality (yes for 1–5 items).

Results

Study selection and overview of included studies

The search identified 6,602 records, of which 5,647 unique records were screened after removing 955 duplicates. After systematically screening the 5,647 records, 132 full-text articles were ultimately included in the analysis (Fig. 1). The included studies were from thirty-nine (39) countries on six continents (Asia, $n = 73$; Africa, $n = 22$; Europe, $n = 18$; North America, $n = 12$; South America, $n = 6$; Oceania, $n = 1$). Most studies were conducted in India ($n = 25$, 18.9%), followed by China ($n = 17$, 12.9%) and Egypt ($n = 10$, 7.6%) (Fig. 2).

Most studies reported antimicrobial resistance in multiple pathogens ($n = 89$, 67%), while 43 (33%) focused on single pathogens (Table S1). Resistance in *E. coli* was reported in 96 studies, *K. pneumoniae* in 66 studies, *S. aureus* in 65 studies, and *P. aeruginosa* in 60 studies. Additionally, 29 studies described resistance in *Enterobacter* spp., 24 studies in *A. baumannii*, 15 studies in *E. faecium*, and 4 studies in *S. pneumoniae*.

Additionally, a total of 1622 resistance data points for selected pathogenic bacteria were extracted for the meta-analysis. This dataset included the total number of isolates tested and the number of resistant isolates identified.

Description of cancer patients

The included studies identified 49,638 cancer patients. Most of the studies described resistance in pathogens from adults ($n = 58$, 43.9%), while 21 (15.9%) studies only described resistance in children. Additionally, 33 (25%) studies reported AMR in both children and adults, and 20 (15.1%) studies did not specify the age of the participants. Patients with haematological malignancies were

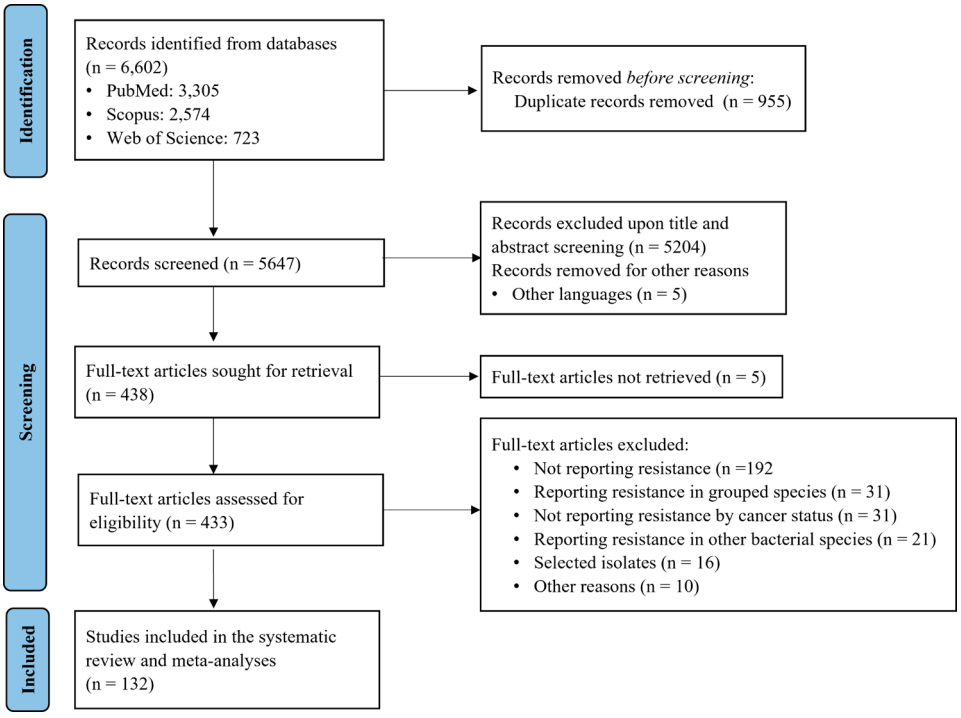

Fig. 1 Flow diagram of the study selection process

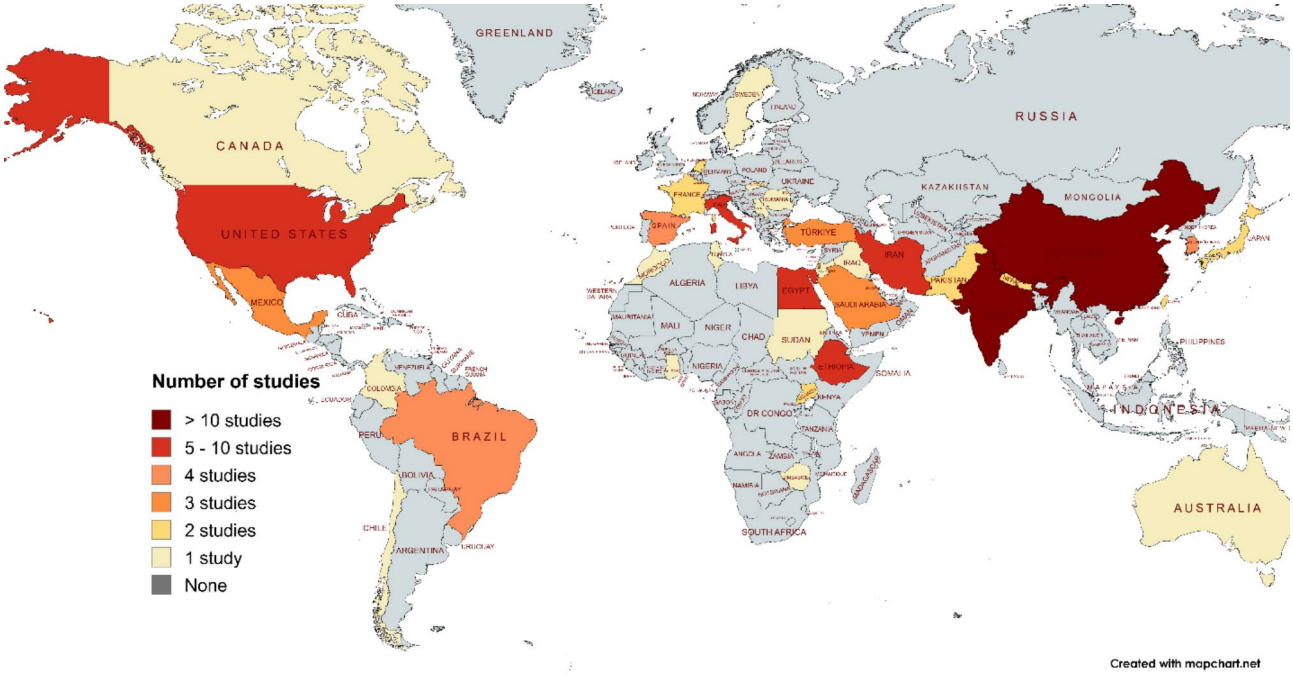

Fig. 2 Geographical distributions of studies reporting AMR in cancer patients worldwide. Countries were shaded if at least one study on AMR in cancer patients was identified in the review. Each colour represents the number of studies from the respective country

included in 48 (36.4%) studies, while 18 (13.6%) studies included patients with solid malignancies, and 56 (42.4%) studies included patients with both types of cancer. For ten studies, the type of cancer was not specified. Most patients had bacteraemia ($n = 78$, 59.0%), followed by surgical site infection ($n = 39$, 29.5%). Six studies reported on urinary tract infection, 4 studies on skin and soft tissue infections, 3 studies on respiratory infection and 2 studies on biliary tract infection (Table S1).

AMR in *Escherichia coli*

Antimicrobial resistance in *E. coli* was reported in 96 studies. Studies reported *E. coli* infections in several sites including bloodstream: $n = 60$, urinary tract: $n = 6$, respiratory tract: $n = 3$, wound: $n = 2$, biliary tract: $n = 2$, multiple sites: $n = 23$. The majority of studies reported resistance to fluoroquinolones ($n = 75$), followed by resistance to aminoglycosides ($n = 71$), BLBLIs ($n = 67$), third-generation cephalosporins ($n = 66$) and carbapenems ($n = 56$). The prevalence of antibiotic resistance was highest for penicillins (81.84%, 95% CI [71.79; 90.37]), cotrimoxazole (65.79%, 95% CI [57.31; 73.86]), and monobactams (61.61%, 95% CI [49.79; 72.82]), with lower resistance prevalence observed for carbapenems (20.30%, 95% CI [13.85; 27.51]) and polymyxins (20.75%, 95% CI [4.14; 43.58]). The resistance prevalence among third-generation cephalosporins, fluoroquinolones, and fourth-generation cephalosporins was 56.06% (95% CI [48.15; 63.82]), 53.24% (95% CI [45.93; 60.50]), and 51.08% (95% CI [40.83; 61.30]), respectively (Figure S2). Of the 96 studies describing resistance in *E. coli*, 27 studies (27.2%) identified extended-spectrum beta-lactamase-producing isolates that cause infection, with a resistance prevalence of 49.87% (95% CI [39.29; 60.46]) (Fig. 3). Heterogeneity was high (I^2 89–98%) across studies for almost all the antibiotic classes except for chloramphenicol, which was moderate (I^2 56%) (Table 2).

AMR in *Klebsiella pneumoniae*

Sixty-six studies described antibiotic resistance in *K. pneumoniae* infections (bloodstream: $n = 42$, urinary tract: $n = 2$, respiratory tract: $n = 3$, wound: $n = 1$, biliary tract: $n = 1$, multiple sites: $n = 17$). Most studies reported resistance to fluoroquinolones (51 studies), followed by resistance to aminoglycosides (49 studies), third-generation cephalosporins (47 studies), BLBLIs (44 studies), and carbapenems (44 studies) (Table 2). Resistance to chloramphenicol was described in only 5 studies, as shown in Table 2. Resistance to penicillin had the highest resistance prevalence of 98.99% [95.11; 100], while a lower prevalence of antibiotic resistance was observed among polymyxins (16.29%, 95% CI [7.46; 27.45]). The resistance prevalence to cotrimoxazole, third-generation cephalosporins, BLBLIs, monobactam, fourth-generation cephalosporins, and fluoroquinolones were 70.92% (95% CI [55.95; 84.19]), 65.95% (95% CI [56.03; 75.29]), 65.45% (95% CI [56.27; 74.14]), 64.64% (95% CI [47.45; 80.22]), 60.85% (95% CI [48.75; 72.37]), and 59.23% (95% CI [49.93; 68.26], respectively (Figure S3). Additionally, the resistance prevalence of extended-spectrum beta-lactamase-producing *K. pneumoniae* infection was 41.06% (95% CI [27.09; 55.67]) (Fig. 3). High heterogeneity was shown across studies reporting resistance to almost all antibiotic classes except chloramphenicol (Table 2).

AMR in *Staphylococcus aureus*

The prevalence of antimicrobial resistance in *S. aureus* infection was described in 65 studies. Isolates were from blood ($n = 33$), wounds ($n = 3$), respiratory samples ($n = 2$), urine ($n = 1$), pus ($n = 1$), or multiple sources ($n = 25$). Of the 65 studies focusing on infection, 42 studies identified methicillin-resistant *Staphylococcus aureus* (MRSA), with a pooled prevalence of 45.29% (95% CI [33.95;56.86]). Resistance to macrolides had the highest prevalence (55.63%, 95% CI [46.93; 64.19]), followed by fluoroquinolones (40.73%, 95% CI [28.19; 53.81]). Rifampin resistance, described in 6 studies, had a lower resistance prevalence of 18.19% (95% CI [3.38; 38.76]). The heterogeneity of studies was high for all the antibiotic classes ($I^2 = 80–97\%$) (Table 2).

AMR in *Pseudomonas aeruginosa*

AMR in *P. aeruginosa* was described in 60 studies. Isolates associated with infections were from blood ($n = 36$), wounds ($n = 3$), urine ($n = 2$), respiratory samples ($n = 2$), bile ($n = 1$), pus ($n = 1$), and multiple sources ($n = 15$).

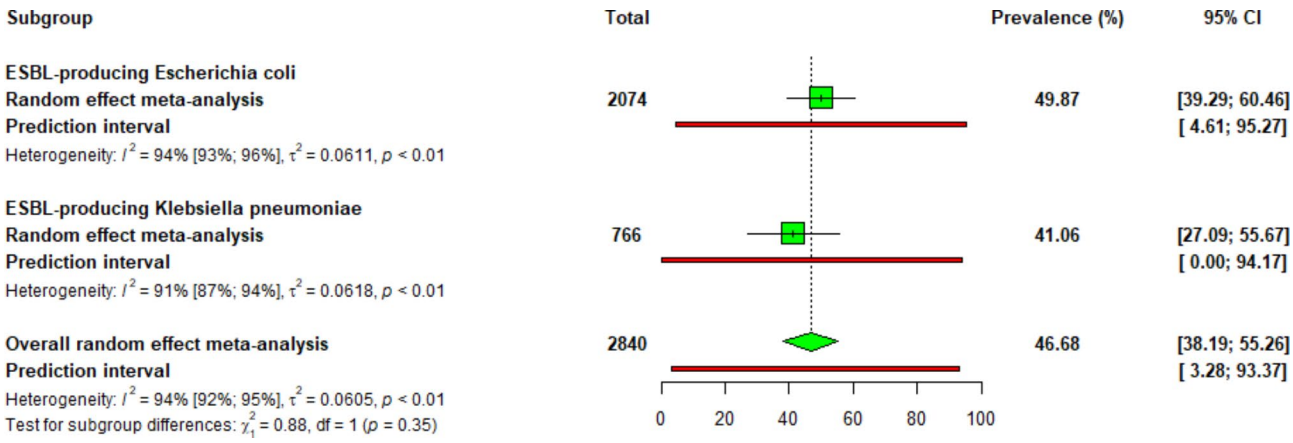

Fig. 3 Pooled prevalence of ESBL-producing *Escherichia coli* and *Klebsiella pneumoniae* isolated from cancer patients

Table 2 Pooled resistance to selected antibiotics among bacterial pathogens isolated from cancer patients

Organism	Antimicrobial class	Studies pooled	No. cancer patients (No. isolates)	Pooled resistance (%) [95% CI]	Heterogeneity I^2 (%), *p*-value
Escherichia coli	3rd gen. cephalosporins	66	32,266 (6725)	56.06 [48.15; 63.82]	97, $p=0$
	4th gen. cephalosporins	44	12,244 (2790)	51.08 [40.83; 61.30]	96, $p<0.01$
	Carbapenems	56	15,097 (3793)	20.30 [13.85; 27.51]	96, $p<0.01$
	Fluoroquinolones	75	32,773 (7222)	53.24 [45.93; 60.50]	97, 96, $p=0$
	Penicillins	36	12,695 (3410)	81.84 [71.79; 90.37]	97, $p<0.01$
	Beta-lactam-beta-lactamase inhibitors (BLBLIs)	67	20,858 (5147)	42.76 [36.29; 49.35]	94, $p<0.01$
	Aminoglycosides	71	17,150 (4299)	31.47 [26.53; 36.59]	89, $p<0.01$
	Cotrimoxazole	38	13,181 (4010)	65.79 [57.31; 73.86]	95, $p<0.01$
	Tetracyclines	20	5240 (1119)	43.23 [21.59; 66.06]	98, $p<0.01$
	Monobactams	19	6077 (1759)	61.61 [49.79; 72.82]	95, $p<0.01$
	Polymyxins	13	2695 (643)	20.75 [4.14; 43.58]	97, $p<0.01$
	Chloramphenicol	7	897 (153)	32.40 [17.31; 49.02]	56, $p=0.03$
Klebsiella pneumoniae	3rd gen. cephalosporins	47	15,627 (2107)	65.95 [56.03; 75.29]	94, $p<0.01$
	4th gen. cephalosporins	34	10,365 (1755)	60.85 [48.75; 72.37]	96, $p<0.01$
	Carbapenems	44	11,603 (2431)	49.89 [38.25; 61.53]	97, $p<0.01$
	Fluoroquinolones	51	13,248 (2331)	59.23 [49.93; 68.26]	93, $p<0.01$
	Penicillins	20	4342 (477)	98.99 [95.11; 100.00]	59, $p<0.01$
	Beta-lactam-beta-lactamase inhibitors (BLBLIs)	44	11,639 (2107)	65.45 [56.27; 74.14]	93, $p<0.01$
	Aminoglycosides	49	12,291 (2340)	46.13 [38.79; 53.54]	90, $p<0.01$
	Cotrimoxazole	24	5449 (1098)	70.92 [55.93; 84.19]	95, $p<0.01$
	Tetracyclines	19	5003 (1371)	36.32 [17.21; 57.71]	98, $p<0.01$
	Monobactams	18	5382 (924)	64.64 [47.45; 80.22]	96, $p<0.01$
	Polymyxins	14	2627 (1180)	16.29 [7.46; 27.45]	95, $p<0.01$
	Chloramphenicol	5	524 (55)	43.09 [20.84; 66.57]	46, $p<0.01$
Staphylococcus aureus	MRSA	42	26,002 (2995)	45.29 [33.95; 56.86]	97, $p<0.01$
	Fluoroquinolones	28	8396 (909)	40.73 [28.19; 53.81]	91, $p<0.01$
	Aminoglycosides	23	7626 (810)	26.25 [16.87; 36.69]	88, $p<0.01$
	Cotrimoxazole	17	4519 (602)	36.72 [23.11; 51.35]	90, $p<0.01$
	Tetracyclines	14	4246 (517)	29.70 [17.98; 42.69]	83, $p<0.01$
	Lincosamides	25	6085 (1021)	31.40 [21.93; 41.60]	88, $p<0.01$
	Macrolides	26	7727 (1059)	55.63 [46.93; 64.19]	82, $p<0.01$
	Vancomycin	8	1607 (184)	28.82 [5.33; 59.12]	92, $p<0.01$
	Rifampin	6	1348 (140)	18.19 [3.38; 38.76]	80, $p<0.01$
Pseudomonas aeruginosa	3rd gen. cephalosporins	46	15,168 (1389)	49.41 [39.75; 59.09]	88, $p<0.01$
	4th gen. cephalosporins	27	8087 (879)	32.74 [21.87; 44.50]	90, $p<0.01$
	Carbapenems	45	20,108 (2095)	37.73 [29.35; 46.43]	91, $p<0.01$
	Fluoroquinolones	44	11,191 (1398)	40.07 [29.33; 51.25]	93, $p<0.01$
	Beta-lactam-beta-lactamase inhibitors (BLBLIs)	44	10,192 (1241)	43.81 [33.34; 54.51]	90, $p<0.01$
	Aminoglycosides	41	8726 (1260)	38.96 [27.12; 51.32]	93, $p<0.01$

Table 2 (continued)

Organism	Antimicrobial class	Studies pooled	No. cancer patients (No. isolates)	Pooled resistance (%) [95% CI]	Heterogeneity I^2 (%), *p*-value
	Polymyxins	8	1707 (298)	13.22 [1.35; 31.10]	85, $p < 0.01$
Enterobacter spp.	3rd gen. cephalosporins	23	9833 (413)	65.68 [48.74; 81.14]	87, $p < 0.01$
	4th gen. cephalosporins	10	3336 (256)	43.21 [24.32; 62.95]	82, $p < 0.01$
	Carbapenems	15	5364 (346)	39.43 [18.38; 62.33]	92, $p < 0.01$
	Fluoroquinolones	20	5597 (380)	48.22 [31.42; 65.19]	86, $p < 0.01$
	Penicillins	11	2483 (123)	91.77 [53.88; 100.00]	85, $p < 0.01$
	Beta-lactam-beta-lactamase inhibitors (BLBLIs)	21	5559 (352)	60.22 [46.63; 73.22]	70, $p < 0.01$
	Aminoglycosides	19	5206 (342)	48.84 [36.27; 61.48]	70, $p < 0.01$
	Cotrimoxazole	10	2780 (221)	67.11 [43.14; 90.77]	88, $p < 0.01$
	Monobactams	6	2490 (208)	50.22 [24.41; 75.97]	89, $p < 0.01$
Acinetobacter baumannii	3rd gen. cephalosporins	20	4713 (789)	84.10 [67.85; 96.20]	93, $p < 0.01$
	4th gen. cephalosporins	14	3765 (744)	80.75 [59.01; 96.44]	96, $p < 0.01$
	Carbapenems	19	4590 (830)	82.58 [68.37; 93.83]	92, $p < 0.01$
	Fluoroquinolones	21	5069 (790)	80.37 [64.64; 93.05]	91, $p < 0.01$
	Beta-lactam-beta-lactamase inhibitors (BLBLIs)	20	4540 (883)	79.15 [65.70; 90.50]	89, $p < 0.01$
	Aminoglycosides	22	4834 (907)	64.05 [47.12; 79.64]	94, $p < 0.01$
	Cotrimoxazole	9	2731 (335)	75.77 [57.34; 90.95]	81, $p < 0.01$
	Tetracyclines	8	2546 (441)	44.28 [13.93; 76.80]	97, $p < 0.01$
	Polymyxins	7	884 (183)	18.97 [3.01; 41.87]	88, $p < 0.01$
Enterococcus faecium	Vancomycin	9	7637 (397)	21.07 [8.53; 36.56]	87, $p < 0.01$
	Penicillins	7	1864 (112)	90.64 [68.00; 100.00]	74, $p < 0.01$
	Tetracyclines	6	1898 (106)	28.26 [1.58; 65.18]	85, $p < 0.01$
Streptococcus pneumoniae	Penicillins	3	252 (9)	84.15 [45.92; 100.00]	0, $p = 0.45$
	Lincosamides	3	426 (9)	70.78 [31.00; 99.13]	0, $p = 0.50$

Resistance to third-generation cephalosporins, carbapenems, BLBLIs, fluoroquinolones, and aminoglycosides was widely reported in most of the studies (Table 2). Resistance to third-generation cephalosporins had the highest resistance prevalence (49.41%, 95% CI [39.75; 59.09]), followed by BLBLIs (43.81%, 95% CI [33.34; 54.51]), and fluoroquinolones (40.07%, 95% CI [29.33; 51.25]), while the lowest prevalence of resistance was observed for polymyxins (13.22%, 95% CI [1.35; 31.10]). Heterogeneity was high across studies for all antibiotic classes (I^2 = 85–93%) (Table 2).

AMR in *Enterobacter* spp

A total of 29 studies evaluated AMR in *Enterobacter* spp. isolated from infections (Table 2). Resistance to third-generation cephalosporins, BLBLIs, and fluoroquinolones was widely reported in isolates causing infection. The highest prevalence of AMR was observed for penicillin (91.77%, 95% CI [53.88; 100]), followed by cotrimoxazole (69.11%, 95% CI [43.14; 90.77]) and BLBLIs (60.22%, 95% CI [46.63; 73.22]. The lowest resistance prevalence was for carbapenem, at 39.43% (95% CI [18.38; 62.33]). Heterogeneity was moderate across studies for both BLBLIs and aminoglycosides (I^2 = 70%) (Table 2).

AMR in *Acinetobacter baumannii*

Twenty-four studies described AMR in *A. baumannii* isolates from infected patients. The resistance rate was highest for third-generation cephalosporins (84.10%, 95% CI [67.85; 96.20]), followed by carbapenems (82.58%, 95% CI [68.37; 93.83]), fourth-generation cephalosporins (80.75%, 95% CI [59.01; 96.44]), and fluoroquinolones (80.37%, 95% CI [64.64; 93.05]). Resistance to polymyxins had the lowest resistance prevalence of 18.97% (95% CI [3.01; 41.87]). High heterogeneity was shown across all studies (Table 2).

AMR in *Enterococcus faecium*

Fifteen studies reported AMR in *E. faecium* infections (Table 2). Penicillin resistance had the highest resistance prevalence of 90.64% (95% CI [68.00; 100]), while the lowest resistance prevalence was observed for vancomycin (21.07%, 95% CI [8.53; 36.56]). The prevalence of resistance to tetracyclines was 28.26% (95% CI [1.58; 65.30]) (Table 2).

AMR in *Streptococcus pneumoniae*

Four studies reported AMR in *S. pneumoniae*. All the studies described invasive pneumococcal disease, where isolates were obtained from the blood. Penicillin and lincosamide resistance were evaluated in 3 studies, with pooled resistance prevalence of 84.15% (95% CI [45.92; 100]) and 70.78% (95% CI [31.00; 99.13]), respectively (Table 2).

AMR by cancer types and study designs

A subgroup analysis was performed to estimate the pooled resistance to the various antibiotic classes based on the type of cancer patients included in the studies and study designs. Isolates from patients with haematological cancer exhibited higher rates of resistance to fourth-generation cephalosporins, carbapenems, fluoroquinolones, beta-lactamases inhibitors, aminoglycosides, monobactams, polymyxins, and vancomycin. Higher rates of resistance to penicillins, co-trimoxazole, and tetracyclines were observed among isolates from solid cancer patients. The pooled resistance to methicillin, lincosamides, and macrolides was higher among isolates reported in studies describing patients of both cancer types. Overall, high rates of resistance to penicillins and low polymyxin resistance were observed among all cancer types. For study design, isolates reported in prospective studies exhibited relatively higher resistance to almost all the antibiotic classes. Resistance to penicillins and polymyxins had relatively higher rates among isolates reported in retrospective studies except penicillins and polymyxins. Chloramphenicol resistance was only tested in prospective studies. Table 3 summarizes the results for the subgroup analysis.

Quality of the included studies

Most of the studies included (n = 70, 53%) were of moderate quality according to the modified STROBE checklist. Only two studies were of low quality because they did not discuss most of the items on the checklist. The following items were not often discussed: how the study sample size was derived, patient outcomes, source of data, and variable definitions. The reporting completeness was high in 60 studies (46%) (Table S2).

Meta-regression

A meta-regression was performed to identify the sources of heterogeneity observed in our meta-analysis. The regression model incorporated four study characteristics: cancer type, bacterial isolates, class of antibiotics, and study design. For cancer type, only heamatological and solid cancers exhibited a significant positive association with the observed heterogeneity. Regarding bacterial isolates, all showed a significant negative association with the heterogeneity, except for *Streptococcus pneumoniae*, which displayed a non-significant negative association. In terms of antibiotic class, 4th-generation cephalosporins, aminoglycosides, carbapenems, polymyxins, and tetracyclines were significantly negatively associated with the observed heterogeneity, while penicillins had a significant positive association. Additionally, retrospective studies demonstrated a significant negative association with heterogeneity. However, the model could not generate

Table 3 Pooled resistance to the various antibiotic classes according to cancer patient types and study designs

Antibiotic class	Bacterial pathogens tested	Pooled resistance (%) [95% CI]				
		Cancer types			Study design	
		Haematological	Solid	Both	Prospective	Retrospective
3rd gen. cephalosporins	EC, KP, PA, EB, AB	60.09 [52.01; 67.93]	60.13 [48.60; 71.19]	60.79 [51.39; 69.89]	64.67 [55.70; 73.22]	58.28 [51.68; 64.76]
4th gen. cephalosporins	EC, KP, PA, EB, AB	53.79 [42.14; 65.26]	50.85 [37.55; 64.09]	50.42 [37.47; 63.35]	58.72 [48.12; 68.99]	48.52 [39.81; 57.27]
Carbapenems	EC, KP, PA, EB, AB	40.49 [30.65; 50.68]	33.15 [20.96; 46.43]	39.99 [28.40; 52.08]	44.70 [35.00; 54.57]	35.97 [27.74; 44.58]
Fluoroquinolones	EC, KP, PA, EB, AB, SA	55.23 [47.24; 63.10]	50.85 [39.20; 62.46]	52.25 [44.43; 60.03]	53.30 [46.10; 60.45]	51.67 [45.29; 58.02]
Penicillin/penicillin derivatives	EC, KP, EF, EB, SP	85.14 [65.60; 98.02]	93.41 [85.88; 98.67]	89.90 [78.34; 98.08]	87.93 [75.55; 97.18]	90.55 [84.17; 95.72]
Beta-lactamase inhibitors	EC, KP, PA, EB., AB	54.47 [46.19; 62.65]	52.35 [41.32; 63.29]	53.57 [45.65; 61.42]	61.04 [52.27; 69.53]	48.08 [42.24; 53.94]
Aminoglycosides	EC, KP, PA, EB, AB, SA	43 [35.39; 50.76]	39.15 [31.67; 46.87]	39.11 [32.48; 45.92]	46.76 [39.96; 53.61]	34.62 [29.80; 39.57]
Cotrimoxazole	EC, KP, SA, EB, AB	60 [50.43; 69.24]	67.95 [52.31; 81.98]	60.92 [50.58; 70.87]	69.41 [59.96; 78.24]	55.97 [47.03; 64.74]
Tetracyclines	EC, KP, SA, EB, AB	29.32 [17.26; 42.94]	44.58 [17.84; 73.01]	30.77 [23.08; 55.44]	43.49 [29.15; 58.29]	32.89 [19.42;47.78]
Monobactams	EC, KB, EB	62.26 [45.85; 77.44]	58.41 [45.76; 70.50]	54.89 [41.23; 68.22]	74.07 [61.53; 85.06]	50.08 [37.88; 62.26]
Polymyxins	EC, KP, PA, AB	26.81 [12.41; 44.06]	13.67 [0; 46.45]	13.06 [3.49; 26.01]	10.67 [2.76; 21.75]	24.18 [13.20; 36.84]
Chloramphenicol	EC, KP	29 [21.74; 36.82]	41.39 [12.96; 72.57]	48.39 [20.08; 77.13]	36.04 [23.66; 49.19]	
Vancomycin	EF	34.31 [12.07; 60.15]	16.13 [0; 77.72]	23.13 [6.36; 44.72]	27.28 [9.58; 49.13]	20.11 [6.04; 38.12]
Methicillin	SA	52.24 [26.09; 77.85]	39.63 [25.32; 39.60]	45.23 [27.18; 63.86]	45.05 [26.33; 64.44]	45.58 [30.77; 60.73]
Lincosamides	SA, SP	27.48 [3.32; 60.33]	32.74 [26.19; 39.60]	35.67 [21.46; 51.02]	41.05 [21.30; 62.12]	27.50 [17.00; 39.19]
Macrolides	SA	37.44 [24.45; 51.23]	51.15 [35.32; 66.88]	59.44 [45.77; 72.52]	61.11 [52.97; 68.99]	51.83 [39.06; 64.50]
Rifampin	SA			31.81 [10.79; 56.54]	31.87 [8.30; 60.67]	4.8 [0.00; 14.52]

EC: Escherichia coli, KP: Klebsiella pneumoniae, SA: Staphylococcus aureus, PA: Pseudomonas aeruginosa, EB: Enterobacter spp., AB: Acinetobacter baumannii, EF: Enterococcus faecium, and SP: Streptococcus pneumoniae

results for *A. baumannii*, 3rd-generation cephalosporins, or prospective studies due to redundancy (Table 4).

Sensitivity analysis

A sensitivity analysis was performed by excluding low-quality studies identified during our quality assessment. The comparison of pooled resistance rates for bacterial isolates before and after the exclusion showed general stability between the resistance rates, indicating that the findings of our meta-analysis are robust (Table 5).

Discussion

The predisposition of cancer patients to infections is exacerbated by their usual administration of immunosuppressive drugs in an attempt to reduce the proliferation of cancer cells [16–19]. Consequently, the compromised immune system of these individuals necessitates concomitant antibiotic usage to clear bacterial cells in infections [20–22]. Unfortunately, this frequent exposure to antibiotics can contribute to the development of antibiotic resistance. As a result, cancer patients have the potential to be significant sources of antibiotic-resistant bacteria, as bacteria tend to develop resistance when exposed to high levels of antibiotics [23–25]. We conducted a pioneering systematic review and meta-analysis to consolidate and present data on antibiotic resistance among major bacterial pathogens isolated from cancer patients.

The wide scope of studies identified in this review reflects the heightened concern and extensive research devoted to understanding and combating AMR in cancer patients worldwide. The focus on patients with haematological cancers is clearly justified, given their greater risk of developing infections due to the nature of the underlying disease and its treatment [9]. Notably, infections account for 60% of fatalities in haematological cancer patients.

Our current study revealed a strong association between antibiotic resistance and cancer patients. We observed a consistently high prevalence of antibiotic resistance among the included pathogens in cases of infection. The resistance patterns exhibited variations across different antibiotics and to varying degrees. In infection cases, *E. coli* showed high resistance, ranging from 20.30% for carbapenem to 81.84% for penicillin. This was followed by *K. pneumoniae*, with a resistance range of 16.29% for polymyxins to 98.99% for penicillin, while *S. aureus* exhibited resistance ranging from 18.19% for rifampin to 55.63% for macrolides. These findings reinforce the concerning rise of carbapenemases, which are enzymes responsible for carbapenem resistance. This resistance mechanism involves the production of

Table 4 Factors affecting heterogeneity in the study

Covariates	Estimate	Standard Error	z-value	*p*-value	95% CI	
					ci.lb	ci.ub
Type of Cancer						
Haematological and solid cancer (Intercept)	1.1415	0.0371	30.8009	< 0.0001	1.0689	1.2141
Haematological cancer	0.0321	0.0189	1.6963	0.0898	-0.0050	0.0692
Solid cancer	0.0192	0.0230	0.8335	0.4046	-0.0260	0.0644
Unspecified	0.0147	0.0307	0.4785	0.6323	-0.0455	0.0749
Bacterial Isolates						
Enterobacter spp.	-0.2297	0.0407	-5.6405	< 0.0001	-0.3095	-0.1499
Enterococcus faecium	-0.3196	0.0889	-3.6195	0.0003	-0.4926	-0.1465
Escherichia coli	-0.2884	0.0312	-9.2375	< 0.0001	-0.3496	-0.2272
Klebsiella pneumoniae	-0.1652	0.0323	-5.1101	< 0.0001	-0.2286	-0.1018
Pseudomonas aeruginosa	-0.3035	0.0345	-8.7992	< 0.0001	-0.3711	-0.2359
Staphylococcus aureus	-0.3800	0.0452	-8.3989	< 0.0001	-0.4687	-0.2913
Streptococcus pneumoniae	-0.1838	0.1705	-1.0784	0.2809	-0.5180	0.1503
Class of Antibiotics						
4th generation cephalosporins	-0.0756	0.0353	-2.1404	0.0323	-0.1447	-0.0064
Aminoglycosides	-0.1752	0.0309	-5.6621	< 0.0001	-0.2358	-0.1145
Beta-lactamase inhibitors	-0.0628	0.0319	-1.9712	0.0487	-0.1253	-0.0004
Carbapenems	-0.2006	0.0323	-6.2162	< 0.0001	-0.2638	-0.1373
Chloramphenicol	-0.2111	0.0999	-2.1137	0.0345	-0.4068	-0.0154
Co-trimoxazole	0.0343	0.0396	0.8654	0.3868	0.0434	0.1120
Fluoroquinolones	-0.0587	0.0306	-1.9218	0.0546	-0.1186	0.0012
Lincosamides	-0.1042	0.0743	-1.4019	0.1610	-0.2498	0.0415
Macrolides	0.1045	0.0750	1.3927	0.1637	-0.0426	0.2516
Methicillin	0.0155	0.0647	0.2389	0.8112	-0.1114	0.1423
Monobactams	0.0096	0.0520	0.1841	0.8539	-0.0923	0.1114
Penicillins	0.2902	0.0449	6.4569	< 0.0001	0.2021	0.3783
Polymyxins	-0.4526	0.0529	-8.5529	< 0.0001	-0.5564	-0.3489
Rifampin	-0.2435	0.1380	-1.7645	0.0777	-0.5140	0.0270
Tetracyclines	-0.2077	0.0460	-4.5159	< 0.0001	-0.2979	-0.1176
Vancomycin	-0.2302	0.0953	-2.4146	0.0158	-0.4170	-0.0433
Study Design						
Retrospective	-0.0811	0.0166	-4.9008	< 0.0001	-0.1136	-0.0487

Table 5 Sensitivity analysis of the overall pooled resistance among each organism

Organism	Before excluding low-quality studies			After excluding low-quality studies		
	Overall pooled resistance (%)	L. CI	U. CI	Overall pooled resistance (%)	L. CI	U. CI
Escherichia coli	47.39	44.43	50.36	46.73	43.73	49.73
Klebsiella pneumoniae	58.24	54.14	62.29	58.18	54.00	62.31
Staphylococcus aureus	38.19	33.60	42.88	38.23	33.59	43.04
Pseudomonas aeruginosa	40.00	35.69	44.38	39.23	34.95	43.59
Enterobacter spp.	55.83	49.44	62.14	55.83	49.44	62.14
Acinetobacter baumannii	73.00	66.36	79.26	73.00	66.36	79.26
Enterococcus faecium	44.57	23.66	66.30	44.57	23.66	66.30
Streptococcus pneumoniae	77.71	50.17	97.63	77.71	50.17	97.63

L. CI: lower confidence interval; U. CI: upper confidence interval

beta-lactamases that can deactivate carbapenems and other beta-lactam antibiotics [27, 28]. The resistance to penicillin was generally high across all the bacteria evaluated, ranging from 84.15% in *S. pneumoniae* to 98.99% in *K. pneumoniae*. This prevalence of penicillin resistance is greater than the 23% recorded among human subjects in the WHO African regions [29]. The high resistance observed in polymyxins, known as last-resort antibiotics, is a cause for the alarm. Importantly, pathogens resistant to this vital antibiotic are not bacteria that naturally possess intrinsic resistance to polymyxins [30]. This finding underscores that the bacteria isolated from

cancer patients have acquired resistance to these antibiotics, emphasizing the alarming development of acquired resistance. Possible underlying factors for this high antibiotic resistance among cancer patients are immunosuppression, prolonged antibiotic exposure, invasive medical devices such as central venous lines, urinary catheters, and intensive treatments such as chemotherapy and radiation therapy. These procedures and treatments can weaken their immune system, increasing their susceptibility to infections. Moreover, the acquisition of resistant bacteria from healthcare settings is significant, considering that cancer patients spend a substantial amount of time in these facilities. Healthcare settings have an elevated prevalence of resistant strains [31, 32] and may play a crucial role in the high prevalence of antibiotic-resistant bacterial isolates among cancer patients.

With respect to each pathogen, the resistance varied depending on its classification. Compared with gram-positive bacteria, gram-negative bacteria exhibited distinct interactions and resistance patterns. A wide variety of gram-negative strains, such as *E. coli* isolates from infections, were resistant to many classes of antibiotics, including third- and fourth-generation cephalosporins (51.08%-56.06%) and fluoroquinolones (53.24%). In addition to being highly resistant to penicillins and monobactams, many of these isolates (49.87%) that cause infections are ESBL-producing *E. coli*, which may account for the resistance to wide classes of antibiotics, as some ESBL-producing *Enterobacteriaceae* are resistant to nearly all antibiotics [33]. Similarly, *K. pneumoniae* strains exhibited a wide range of resistance to various classes of antibiotics in this study. This included resistance to penicillins (98.99%) and polymyxins (16.29%), cotrimoxazole (70.92%), third- and fourth-generation cephalosporins (65.95%-60.85%), BLBLIs (65.45%), monobactams (64.64%), and fluoroquinolones (59.23%). These key findings reveal the limited effectiveness of these antibiotics against *K. pneumoniae*, presenting significant challenges in the clinical management of their infections as the available antibiotic options become increasingly limited. The resistance prevalence of ESBL-producing *K. pneumoniae* was 41.06%. This highlights that ESBL-producing *E. coli* and *K. pneumoniae* are globally emerging pathogens that pose significant challenges, especially among cancer patients. These key findings highlight rapid evolutionary resistance and the need for alternative therapeutic options and emphasize the importance of antibiotic stewardship programmes to minimize the emergence of further resistance. Like the other gram-negative strains, *P. aeruginosa* was also associated with resistance to many of the antibiotic classes under consideration, including third-generation cephalosporins (49.41%), carbapenems (37.73%), BLBLIs (43.81%), fluoroquinolones (40.07%), polymyxins (13.22%), and aminoglycosides (38.96%). The repeatedly high resistance of the Gram-negative organisms could be due to several factors, including the ready availability of antibiotics (e.g., penicillins and cotrimoxazole) and the fact that antibiotics are inexpensive and easily acquired to treat infections caused by a wide variety of organisms, resulting in widespread use [34].

A. baumannii, an already established and dangerously resistant bacterium, has progressively evolved into a pan drug-resistant bacterial infection in recent years due to its strong biofilm formation ability and other resistance mechanisms. Unsurprisingly, our study revealed significant resistance to multiple classes of antibiotics, including third- and fourth-generation cephalosporins (84.10-80.75%), carbapenems (82.58%), fluoroquinolones (80.37%), and polymyxins (18.97%). *A. baumannii* is recognized as a major pathogen with widespread resistance and is responsible for severe invasive infections such as bacteraemia. In the context of cancer patients, this poses a grave concern because it can lead to therapeutic failure due to the high levels of antibiotic resistance. Its ability to overcome the action of numerous antibiotics stems from various adaptive features, modifications of target sites, aminoglycoside-modifying enzymes, efflux pumps, including β-lactamases, and strong biofilm formation [35]. These adaptive mechanisms grant *A. baumannii* superior resistance ability, making it highly challenging to treat effectively with existing antibiotics [36]. Moreover, *Enterobacter* spp., known for causing a range of infections, such as bloodstream infection, respiratory infection, urinary tract infection, and intra-abdominal infections, exhibited high resistance rates to penicillin (91.77%), cotrimoxazole (67.11%), and carbapenem (39.43%). This resistance pattern observed in *Enterobacteriaceae* may be attributed to the expression of single genes that encode potent drug-modifying enzymes [37].

Consistent with previous research by Montassier et al. [38] on bacteraemia among cancer patients, we also observed a predominance of gram-negative bacteria compared to gram-positive bacteria. Among the gram-positive isolates, only a few, including *S. aureus*, *S. pneumoniae*, and *E. faecium*, were detected. *S. pneumoniae* is known to play a significant role in invasive bacterial infections such as pneumonia and meningitis, especially in individuals with compromised immune systems. Consequently, cancer patients are particularly susceptible to severe infections caused by these pathogens [39]. Notably, *S. pneumoniae* exhibited high resistance rates to penicillin (84.15%) and lincosamide (70.78%). The significant resistance observed in *S. pneumoniae* is a cause for concern, particularly because β-lactam drugs are the primary treatment for pneumococcal disease [40]. This limited availability of effective therapeutic options, coupled with high resistance rates, raises serious concerns about the

potential for treatment failure. *S. pneumoniae* resists the action of these antibiotics through genetic mutations that alter the structure of penicillin-binding proteins, leading to a decreased affinity for β-lactam antibiotics [41]. *E. faecium* also exhibited resistance to several antibiotics, including penicillin (90.64%), vancomycin (21.07%), and tetracyclines (28.26%). Although intrinsic resistance is observed in some Enterococcus strains, additional resistance mechanisms have been identified. These mechanisms involve the acquisition of β-lactamases, the accumulation of point mutations in the penicillin-binding region of PBP5 [42], the acquisition of broad-host-range plasmids, and the exchange of resistance-encoding genes [43].

Furthermore, *S. aureus*, a bacterium widely acknowledged for its high resistance, can cause a diverse range of serious infections. We determined that the pooled prevalence of MRSA infection among cancer patients was 45.29%. *S. aureus* exhibited resistance to a wide variety of antibiotics, such as macrolides (55.63%), methicillin (45.29%), fluoroquinolones (40.73%), and rifampin (18.19%). The high resistance of *S. aureus* indicates that it is still a significant burden among cancer patients. In addition to biofilm formation, *S. aureus* becomes resistant to beta-lactamase through the acquisition of a genomic island, *SCC mec*, which carries the *mecA* gene (methicillin resistance determinant) [44]. Additionally, the enzymatic inactivation of antibiotics, such as through penicillinase and aminoglycoside-modification enzymes, as well as antibiotic entrapment and efflux pumps, contributes to antibiotic resistance [45].

Our sensitivity analysis confirmed the reliability of the results from the meta-analysis. However, this study has several limitations. While most of the observational studies were conducted among cancer patients, many lacked control groups of non-cancer patients, making it impossible to compare the association between antimicrobial resistance (AMR) in cancer patients versus non-cancer patients. By focusing primarily on pathogens that are significant public health threats, this review may have overlooked valuable data on emerging pathogens and opportunistic infections in cancer patients. Additionally, high heterogeneity was observed across the included studies. A meta-regression was performed to identify the sources of the observed heterogeneity. Furthermore, the wide prediction interval could be attributed to the high variability in the extracted resistance data.

Conclusion

The review highlighted high prevalence of antimicrobial resistance in bacterial pathogens isolated from cancer patients. Resistance to key antibiotics, such as fluoroquinolones, aminoglycosides, and third-generation cephalosporins, was frequently observed in multiple pathogens. The rapid emergence of extended-spectrum beta-lactamase-producing isolates poses an additional challenge for effective treatment. Methicillin-resistant *Staphylococcus aureus* remains a significant concern, with a considerably high resistance prevalence reported. Carbapenem resistance in *Acinetobacter baumannii* and *Klebsiella pneumoniae* underscores the urgent need for infection control measures. We therefore recommend that the prophylactic use of antibiotics in cancer patients be effectively monitored to reduce the development rate of antibiotic resistance.

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Author contributions

Conceptualization: E.S.D. and O. K. N.; Data extraction: O.K.N. and E.S.D.; Quality assessment: O.K.N. and E.S.D.; Meta-analysis: O.K.N. and A. A.-D; Project administration: O.K.N. and E.S.D; Validation: O.K.N., E.S.D., A.A.-D., and A.-H.O.; Funding acquisition: E.S.D.; Writing – original draft: O.K.N., E.S.D. and A.-H. O.; Writing – review & editing: O.K.N., E.S.D., A.A.-D., and A.-H.O.

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Data availability

Data is available upon request.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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References

1. Dutescu IA, Hillier SA. Encouraging the development of New antibiotics: are Financial Incentives the Right Way Forward? A systematic review and case study. Infect Drug Resist. 2021;14:415–34.
2. Murray CJL, et al. Global burden of bacterial antimicrobial resistance in 2019: a systematic analysis. Lancet. 2022;399:629–55.
3. Salam MA, et al. Antimicrobial Resistance: a growing serious threat for Global Public Health. Healthcare. 2023;11:1946.

4. Donkor ES, Odoom A, Osman A-H, Darkwah S, Kotey FC. N. A systematic review on Antimicrobial Resistance in Ghana from a one health perspective. Antibiotics. 2024;13:662.
5. Giacomini E, et al. Evidence of Antibiotic Resistance from Population-Based studies: a narrative review. Infect Drug Resist. 2021;14:849–58.
6. Bray F, et al. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin. 2024;74:229–63.
7. World Health Organization. Global cancer burden growing, amidst mounting need for services. https://www.who.int/news/item/01-02-2024-global-cancer-burden-growing--amidst-mounting-need-for-services (2024).
8. Nanayakkara AK, et al. Antibiotic resistance in the patient with cancer: escalating challenges and paths forward. CA Cancer J Clin. 2021;71:488–504.
9. Zheng Y, et al. Fatal infections among Cancer patients: a Population-based study in the United States. Infect Dis Ther. 2021;10:871.
10. Kourbeti I, Kamiliou A, Samarkos M. Antibiotic stewardship in Surgical Departments. Antibiot Basel Switz. 2024;13:329.
11. Tew M, et al. Excess cost of care associated with sepsis in cancer patients: results from a population-based case-control matched cohort. PLoS ONE. 2021;16:e0255107.
12. Cornejo-Juárez P, et al. The impact of hospital-acquired infections with multidrug-resistant bacteria in an oncology intensive care unit. Int J Infect Dis. 2015;31:31–4.
13. Scheich S, et al. Bloodstream infections with gram-negative organisms and the impact of multidrug resistance in patients with hematological malignancies. Ann Hematol. 2018;97:2225–34.
14. Danielsen AS, et al. Clinical outcomes of antimicrobial resistance in cancer patients: a systematic review of multivariable models. BMC Infect Dis. 2023;23:247.
15. Page MJ, et al. PRISMA 2020 explanation and elaboration: updated guidance and exemplars for reporting systematic reviews. BMJ. 2021;372:n160.
16. Basu A, Singh R, Gupta S. Bacterial infections in cancer: a bilateral relationship. WIREs Nanomed Nanobiotechnol. 2022;14:e1771.
17. Guba M, Graeb C, Jauch K-W, Geissler EK. Pro- and anti-cancer effects of Immunosuppressive agents used in Organ Transplantation. Transplantation. 2004;77:1777.
18. Gummert JF, Ikonen T, Morris RE. Newer immunosuppressive drugs: a review. J Am Soc Nephrol. 1999;10:1366–80.
19. Wong S, Slavcev RA. Treating cancer with infection: a review on bacterial cancer therapy. Lett Appl Microbiol. 2015;61:107–12.
20. Gudiol C, Carratalà J. Antibiotic resistance in cancer patients. Expert Rev Anti Infect Ther. 2014;12:1003–16.
21. Tinsley N, et al. Cumulative antibiotic use significantly decreases efficacy of checkpoint inhibitors in patients with Advanced Cancer. Oncologist. 2020;25:55–63.
22. Yu J, et al. Effect of concomitant antibiotics use on patient outcomes and adverse effects in patients treated with ICIs. Immunopharmacol Immunotoxicol. 2023;45:386–94.
23. Cantón R, Horcajada JP, Oliver A, Garbajosa PR, Vila J. Inappropriate use of antibiotics in hospitals: the complex relationship between antibiotic use and antimicrobial resistance. Enferm Infecc Microbiol Clin. 2013;31:3–11.
24. Cantón R, Morosini M-I. Emergence and spread of antibiotic resistance following exposure to antibiotics. FEMS Microbiol Rev. 2011;35:977–91.
25. Hui C, Lin M-C, Jao M-S, Liu T-C, Wu R-G. Previous antibiotic exposure and evolution of antibiotic resistance in mechanically ventilated patients with nosocomial infections. J Crit Care. 2013;28:728–34.
26. Zembower TR. Epidemiology of infections in Cancer patients. Infect Complicat Cancer Patients. 2014;161:43–89.
27. Alfei S, Schito AM. β-Lactam antibiotics and β-Lactamase enzymes inhibitors, part 2: our Limited resources. Pharmaceuticals. 2022;15:476.
28. Nagshetty K, Shilpa BM, Patil SA, Shivannavar CT, Manjula NG. An overview of Extended Spectrum Beta lactamases and Metallo Beta Lactamases. Adv Microbiol. 2021;11:37.
29. Gahimbare L, et al. Antimicrobial Resistance in the WHO African Region: a systematic literature review 2016–2020. Antibiotics. 2024;13:659.
30. Mohapatra SS, Dwibedy SK, Padhy I. Polymyxins, the last-resort antibiotics: Mode of action, resistance emergence, and potential solutions. J Biosci. 2021;46:85.
31. Kritsotakis EI, et al. Prevalence, incidence burden, and clinical impact of healthcare-associated infections and antimicrobial resistance: a national prevalent cohort study in acute care hospitals in Greece. Infect Drug Resist. 2017;10:317–28.
32. Osman A-H, et al. Reservoirs of Nosocomial pathogens in Intensive Care units: a systematic review. Environ Health Insights. 2024;18:11786302241243239.
33. Ventola CL. The Antibiotic Resistance Crisis. Pharm Ther. 2015;40:277–83.
34. Nurjadi D, et al. Emergence of trimethoprim resistance gene dfrG in Staphylococcus aureus causing human infection and colonization in sub-saharan Africa and its import to Europe. J Antimicrob Chemother. 2014;69:2361–8.
35. Kyriakidis I, Vasileiou E, Pana ZD, Tragiannidis A. Acinetobacter baumannii Antibiotic Resistance mechanisms. Pathogens. 2021;10:373.
36. Lee C-R et al. Biology of Acinetobacter baumannii: Pathogenesis, Antibiotic Resistance mechanisms, and prospective treatment options. Front Cell Infect Microbiol 7, (2017).
37. Iredell J, Brown J, Tagg K. Antibiotic resistance in Enterobacteriaceae: mechanisms and clinical implications. BMJ. 2016;352:h6420.
38. Montassier E, Batard E, Gastinne T, Potel G, de La Cochetière MF. Recent changes in bacteremia in patients with cancer: a systematic review of epidemiology and antibiotic resistance. Eur J Clin Microbiol Infect Dis. 2013;32:841–50.
39. van Aalst M, et al. Incidence of invasive pneumococcal disease in immunocompromised patients: a systematic review and meta-analysis. Travel Med Infect Dis. 2018;24:89–100.
40. Olaru ID, et al. The association between antimicrobial resistance and HIV infection: a systematic review and meta-analysis. Clin Microbiol Infect. 2021;27:846–53.
41. Jacobs MR. Drug-resistant Streptococcus pneumoniae: rational antibiotic choices. Am J Med 106, 19S-25S; discussion 48S-52S (1999).
42. Hollenbeck BL, Rice LB. Intrinsic and acquired resistance mechanisms in enterococcus. Virulence. 2012;3:421–569.
43. Rice EW, Messer JW, Johnson CH, Reasoner DJ. Occurrence of high-level aminoglycoside resistance in environmental isolates of enterococci. Appl Environ Microbiol. 1995;61:374–6.
44. Musini A, Kandula P, Giri A. Drug resistance mechanism in Staphylococcus aureus. In: Maddela NR, García LC, editors. Innovations in Biotechnology for a sustainable future. Cham: Springer International Publishing; 2021. pp. 355–76. https://doi.org/10.1007/978-3-030-80108-3_17.
45. Pantosti A, Sanchini A, Monaco M. Mechanisms of antibiotic resistance in Staphylococcus aureus. Future Microbiol. 2007;2:323–34.

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