# ANTIBIOTIC RESISTANCE PROFILE OF BACTERIA ISOLATED FROM CLINICAL LUNG SAMPLES IN NIAMEY

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## Abstract

***Background:*** *Since their discovery, the efficacy of antibiotics has been compromised by bacterial resistance, leading to increased morbidity, mortality, and healthcare costs globally. This issue is particularly critical in regions with limited surveillance data, such as sub-Saharan Africa.* ***Objectives:*** *This study aimed to determine the antibiotic resistance profile of bacterial strains isolated from clinical lung samples in Niamey, Niger, to inform local treatment guidelines.* ***Methods:*** *A prospective study was conducted from October 2013 to September 2016 in major hospitals in Niamey. Bacterial strains were isolated from 247 pulmonary samples. A total of 91 identified strains were tested for susceptibility to 23 antibiotics using the agar diffusion method, with interpretation based on the recommendations of the Antibiogram Committee of the French Microbiology Society (CASFM).* ***Results:*** *Of the 91 isolates, 53.86% were Gram-negative rods and 46.14% were Gram-positive cocci. Overall, 56.45% of tested isolates exhibited resistance. High resistance rates were observed, notably in Staphylococcus aureus against teicoplanin (90.47%), Stenotrophomonas maltophilia against colistin (69.24%), and Pseudomonas aeruginosa against cotrimoxazole (83.33%). Streptococcus pneumoniae showed 62.5–100% resistance to β-lactams. Enterobacteriaceae displayed resistance rates of 71.43% to 92.86% against several antibiotics, including amoxicillin, ticarcillin, and tetracycline.* ***Conclusion:*** *Bacteria responsible for lung infections in Niamey demonstrate alarming levels of resistance to commonly prescribed antibiotics. This poses a significant challenge to effective empirical treatment. The findings underscore an urgent need for implementing molecular assays to identify resistance genes, which would improve diagnostic accuracy and guide more effective therapeutic strategies.*

***Keywords:*** *Antibiotic resistance, Bacteria, Lung disease, Niamey, Niger.*

## 1. INTRODUCTION

The discovery of antibiotics marked an extraordinary advance in medicine, fundamentally improving the prognosis for infectious diseases [1]. However, resistance to these drugs emerged quickly and has since evolved into a major global health problem [2], [3]. The consequences are numerous, including increased morbidity and mortality, as well as higher healthcare costs due to prolonged hospital stays and the necessity of using more expensive and often more toxic alternative drugs [4], [5]. Some infections have become resistant to all antibiotics currently available [6].

The timeline of resistance development is stark: resistance to penicillin emerged in the 1950s [4], to first-generation cephalosporins in the 1970s, and to third-generation cephalosporins in the 1990s. In recent years, the frequency and scope of infections caused by resistant bacteria have expanded in both hospital and community settings [7]. It is estimated that 60% of nosocomial infections worldwide are caused by resistant bacteria, with patients in intensive care units being particularly vulnerable [8].

The overuse and misuse of antibiotics are primary drivers of resistance. In North America, approximately one-third of hospitalized patients receive an antibiotic, with inappropriate prophylactic use estimated at 40% to 75% [9], [10], [11]. Despite the significant burden of infectious diseases in sub-Saharan Africa, research on antibiotic resistance in the region remains limited [12]. In Niger, very few studies have specifically addressed the resistance patterns of bacteria isolated from pulmonary samples [13], highlighting a critical knowledge gap. This study aims to address this gap by characterizing the antibiotic resistance profiles of bacteria causing lung infections in Niamey.

## 2. MATERIALS AND METHODS

This prospective study was conducted in Niamey, the capital of Niger, from October 1, 2013, to September 30, 2016. The study sites included the two national reference hospitals—the National Hospital of Niamey and the Lamordé National Hospital—as well as a private clinic known for managing respiratory diseases. Samples from other public and private health centers were also included. Microbiological analyses were performed at the biology department of the Lamordé National Hospital and the Center for Research in Food and Nutritional Biological Sciences (CRSBAN) at the University of Ouaga Pr Joseph Ki-Zerbo in Burkina Faso.

Microbiological methods involved the isolation, identification, and antibiotic susceptibility testing of bacterial strains. Antibiotic sensitivity was determined on Mueller-Hinton agar using the disk diffusion method, following the manual technique recommended by the Antibiogram Committee of the French Microbiology Society (CASFM) [14]. This method was chosen over microdilution techniques to avoid issues with the growth of strict aerobic bacteria and bacterial aggregation that can interfere with photometric readings.

The interpretation of results was based on the diameter of the inhibition zone (Ø) around each antibiotic disc. Strains were categorized as Sensitive (S), Intermediate (I), or Resistant (R) based on critical diameters (d and D) defined by CASFM standards:

- If $Ø \geq D$, the strain is considered Sensitive (S).
- If $Ø < d$, the strain is considered Resistant (R).
- If $d \leq Ø < D$, the strain has Intermediate sensitivity (I).

A panel of nineteen (19) antibiotics (Bio-Mérieux, France) was used: Ticarcillin (75 µg), Imipenem (10 µg), Ceftazidime (30 µg), Gentamicin (15 µg), Netilmicin (30 µg), Tetracycline (30 µg), Colistin (50 µg), Rifampicin (30 µg), Ciprofloxacin (5 µg), Cotrimoxazole (1.25/23.75 µg), Amikacin (30 µg), Amoxicillin (25 µg), Amoxicillin/clavulanic acid (20/10 µg), Ceftriaxone (30 µg), Cefalotin (30 µg), Kanamycin (30 µg), Nalidixic acid (30 µg), Cefoxitin (30 µg), and Pefloxacin (5 µg).

## 3. RESULTS

From 247 pulmonary samples, a total of 91 bacterial strains were isolated and identified. The overall resistance rate of these strains to the tested antibiotics was 56.45% (Table 1). The isolates comprised 46.14% Gram-positive cocci and 53.86% Gram-negative rods.

**Table1**: frequencies and resistance pattern of bacteria isolated from pulmonary infections

| **Overall susceptibility pattern of bacteria isolated from pulmonary infection** | | | |
|---|---|---|---|
| **Bacterial Strain** | **Number of isolates** | **Susceptible** | **Resistant** |
| *S. aureus* | 252 | 121 (48.01%) | 131 (51.99%) |
| *S. pneumoniae* | 246 | 60 (24.39%) | 186 (75.61%) |
| *S. maltophilia* | 131 | 69 (52.67%) | 62 (47.33%) |
| *P. aeruginosa* | 130 | 71 (54.61%) | 59 (45.39%) |
| *H. influenzae* | 45 | 19 (42.22%) | 26 (57.78%) |
| *A. baumannii* | 44 | 24 (54.54%) | 20 (45.46%) |
| Enterobacteriaceae | 238 | 109 (45.79%) | 129 (54.21%) |
| **Total** | **1086** | **473 (43.55%)** | **613 (56.45%)** |
| **Antibiotics susceptibility profile of Staphylococcus Strains** | | | |
| **Antibiotic** | **Susceptible** | **Intermediate** | **Resistant** |
| Oxacillin 5µg | 5 (23.80%) | 1 (4.76%) | 15 (71.44%) |
| Ceftriaxone 30µg | 9 (42.85%) | 0 | 12 (57.15%) |
| Lincomycin 15µg | 14 (66.67%) | 0 | 7 (33.33%) |

| | | | |
|---|---|---|---|
| Erythromycin 15UI | 15 (71.44%) | 1 (4.76%) | 5 (23.80%) |
| Gentamicin 15µg | 17 (80.96%) | 1 (4.76%) | 3 (14.28%) |
| Fusidic acid 10µg | 16 (76.20%) | 0 | 5 (23.80%) |
| Tetracycline 30UI | 8 (38.09%) | 0 | 13 (61.91%) |
| Vancomycin 30µg | 5 (23.80%) | 2 (9.53%) | 14 (66.67%) |
| Rifampicin 30µg | 7 (33.33%) | 0 | 14 (66.67%) |
| Teicoplanin 30µg | 2 (9.53%) | 0 | 19 (90.47%) |
| Cotrimoxazole 1.25/23.75µg | 7 (33.33%) | 0 | 14 (66.67%) |
| Kanamycin 30UI | 16 (76.20%) | 0 | 5 (23.80%) |

**Antibiotics susceptibility profile of *Streptococcus pneumoniae***

| Antibiotic | Susceptible | Intermediate | Resistant |
|---|---|---|---|
| Penicillin G 6µg | 0 | 0 | 16 (100%) |
| Amoxicillin 25µg | 4 (25%) | 2 (12.5%) | 10 (62.5%) |
| Oxacillin 5µg | 2 (12.5%) | 2 (12.5%) | 12 (75%) |
| Ceftriaxone 30µg | 6 (37.5%) | 0 | 10 (62.5%) |
| Erythromycin 15µg | 9 (56.25%) | 1 (6.25%) | 6 (37.5%) |
| Tetracycline 30UI | 9 (56.25%) | 0 | 7 (43.75%) |
| Lincomycin 15µg | 5 (31.25%) | 1 (6.25%) | 10 (62.5%) |
| Rifampicin 30µg | 11 (68.75%) | 0 | 5 (31.25%) |
| Vancomycin 5µg | 7 (43.75%) | 3 (18.75%) | 6 (37.5%) |
| Teicoplanin 30µg | 3 (18.75%) | 0 | 13 (81.25%) |
| Cotrimoxazole 1.25/23.75µg | 4 (25%) | 0 | 12 (75%) |

**Antibiotics susceptibility profile of *Stenotrophomonas maltophilia***

| Antibiotic | Susceptible | Intermediate | Resistant |
|---|---|---|---|
| Ticarcillin 75µg | 7 (53.84%) | 0 | 6 (46.16%) |
| Imipenem 10µg | 0 | 0 | 13 (100%) |
| Ceftazidime 30µg | 5 (38.46%) | 0 | 8 (61.54%) |
| Gentamicin 15µg | 10 (76.93%) | 1 (7.69%) | 2 (15.38%) |
| Netilmicin 30µg | 9 (69.25%) | 1 (7.70%) | 3 (23.05%) |
| Tetracycline 30UI | 8 (61.55%) | 1 (7.69%) | 4 (30.76%) |
| Colistin 50µg | 4 (30.76%) | 0 | 9 (69.24%) |
| Rifampicin 30µg | 10 (73.51%) | 1 (6.49%) | 3 (20%) |
| Ciprofloxacin 5µg | 11 (84.62%) | 0 | 2 (15.38%) |
| Cotrimoxazole 1.25/23.75µg | 5 (38.45%) | 0 | 8 (61.55%) |

**Antibiotics susceptibility profile of *Pseudomonas aeruginosa***

| Antibiotic | Susceptible | Intermediate | Resistant |
|---|---|---|---|
| Ticarcillin 75µg | 3 (25%) | 1 (8.33%) | 8 (66.67%) |
| Imipenem 10µg | 10 (83.33%) | 0 | 2 (16.67%) |
| Ceftazidime 30µg | 3 (25%) | 1 (8.33%) | 8 (66.67%) |
| Gentamicin 15µg | 11 (91.67%) | 0 | 1 (8.33%) |
| Amikacin 30µg | 10 (83.33%) | 0 | 2 (16.67%) |
| Netilmicin 30µg | 12 (100%) | 0 | 0 |
| Tetracycline 30UI | 6 (50%) | 2 (16.67%) | 4 (33.33%) |
| Colistin 50µg | 3 (25%) | 2 (16.67%) | 7 (58.33%) |
| Rifampicin 30µg | 2 (16.67%) | 1 (8.33%) | 9 (75%) |
| Ciprofloxacin 5µg | 9 (75%) | 1 (8.33%) | 2 (16.67%) |
| Cotrimoxazole 1.25/23.75µg | 2 (16.67%) | 0 | 10 (83.33%) |

**Antibiotics susceptibility profile of *Haemophilus influenzae***

| Antibiotic | Susceptible | Intermediate | Resistant |
|---|---|---|---|
| Amoxicillin 25µg | 2 | 0 | 3 |
| Amoxicillin/clavulanic acid 20/10µg | 2 | 0 | 3 |
| Cefalotin 30µg | 4 | 0 | 1 |
| Kanamycin 30µg | 1 | 0 | 4 |
| Gentamicin 15µg | 3 | 1 | 1 |
| Tetracycline 30µg | 2 | 0 | 3 |
| Nalidixic acid 30µg | 0 | 0 | 5 |
| Rifampicin 30µg | 5 | 0 | 0 |
| Cotrimoxazole 1.25/23.75µg | 0 | 0 | 5 |

**Antibiotics susceptibility profile of *Acinetobacter baumannii***

| Antibiotic | Susceptible | Intermediate | Resistant |
|---|---|---|---|
| Ticarcillin 75µg | 1 | 0 | 4 |
| Imipenem 10µg | 4 | 0 | 1 |

| | | | |
|---|---|---|---|
| Ceftazidime 30µg | 2 | 0 | 3 |
| Gentamicin 15µg | 5 | 0 | 0 |
| Amikacin 30µg | 4 | 0 | 1 |
| Netilmicin 30µg | 5 | 0 | 0 |
| Tetracycline 30UI | 3 | 0 | 2 |
| Colistin 50µg | 2 | 2 | 1 |
| Rifampicin 30µg | 4 | 0 | 1 |
| Ciprofloxacin 5µg | 4 | 0 | 1 |
| Cotrimoxazole 1.25/23.75µg | 1 | 0 | 4 |

**Antibiotics susceptibility profile of *Enterobacteriaceae***

| Antibiotic | Susceptible | Intermediate | Resistant |
|---|---|---|---|
| Amoxicillin 25µg | 3 (21.42%) | 0 | 11 (78.58%) |
| Amoxicillin/clavulanic acid 20/10µg | 4 (28.57%) | 0 | 10 (71.43%) |
| Ticarcillin 75µg | 1 (7.14%) | 0 | 13 (92.86%) |
| Imipenem 10µg | 11 (78.57%) | 0 | 3 (21.43%) |
| Cefalotin 30µg | 8 (57.14%) | 1 (7.14%) | 5 (35.72%) |
| Cefoxitin 30µg | 8 (57.14%) | 1 (7.14%) | 5 (35.72%) |
| Ceftriaxone 30µg | 7 (50%) | 0 | 7 (50%) |
| Gentamicin 15µg | 11 (78.58%) | 1 (7.14%) | 2 (14.28%) |
| Amikacin 30µg | 9 (64.28%) | 2 (14.28%) | 3 (21.44%) |
| Kanamycin 30µg | 9 (64.28%) | 1 (7.14%) | 4 (28.58%) |
| Netilmicin 30µg | 7 (50%) | 1 (7.14%) | 6 (42.86%) |
| Tetracycline 30UI | 0 | 1 (7.14%) | 13 (92.86%) |
| Nalidixic acid 30µg | 4 (28.58%) | 1 (7.14%) | 9 (64.28%) |
| Colistin 50µg | 3 (21.43%) | 1 (7.14%) | 10 (71.43%) |
| Ciprofloxacin 5µg | 10 (71.43%) | 0 | 4 (28.57%) |
| Pefloxacin 5µg | 10 (71.43%) | 0 | 4 (28.57%) |
| Cotrimoxazole 1.25/23.75µg | 4 (28.57%) | 0 | 10 (71.43%) |

## 4. DISCUSSION

The high overall resistance rate of 56.45% observed in this study is a significant cause for concern. This finding may be linked to the uncontrolled use of antibiotics, a common practice in Niger facilitated by the informal sale of drugs on the street. Resistance was particularly high for antibiotics available in oral formulations, such as amoxicillin, tetracycline, and cotrimoxazole, suggesting that easy access contributes to self-medication and misuse. Furthermore, the medical history of patients revealed that 53.19% were on antibiotic therapy prior to sample collection, which is a known risk factor for the development of resistance [6].

*S. aureus* strains showed high resistance to teicoplanin (90.47%), vancomycin (66.67%), and oxacillin (71.44%), indicating a high prevalence of Methicillin-Resistant *S. aureus* (MRSA) and emerging resistance to glycopeptides. The prevalence of MRSA varies globally, with reports of 59% in the US and 48.8% in North Africa [15], [16]. Our finding of low sensitivity to vancomycin is particularly alarming, as vancomycin is a last-resort treatment for MRSA infections. The first case of reduced vancomycin susceptibility was reported in 1996 [17], and similar cases have since been documented worldwide [18].

For *S. pneumoniae*, we observed 100% resistance to penicillin G and high resistance to other β-lactams like oxacillin (75%) and amoxicillin (62.5%). This is consistent with global trends of increasing pneumococcal resistance. For comparison, a 2007 report from Quebec noted 16.2% of strains were non-sensitive to penicillin G [19]. Our data suggest a much more critical situation in Niamey. High resistance to cotrimoxazole (75%) and teicoplanin (81.25%) further limits treatment options.

Gram-negative bacteria also showed worrisome resistance patterns. *S. maltophilia* was 100% resistant to imipenem, a characteristic natural resistance, but also showed high acquired resistance to ceftazidime (61.54%) and cotrimoxazole (61.55%). This aligns with reports from Canada where high resistance to ceftazidime was also observed [20]. *P. aeruginosa* strains were highly resistant to cotrimoxazole (83.33%) and rifampicin (75%), though they remained largely susceptible to aminoglycosides (gentamicin, amikacin, netilmicin) and imipenem.

Enterobacteriaceae exhibited high resistance (≥71%) to amoxicillin, amoxicillin/clavulanic acid, ticarcillin, tetracycline, and colistin. The high level of resistance to β-lactams may be due to the production of cephalosporinases, which can be plasmid-mediated and spread easily among species like *E. coli* and *K. pneumoniae* [6]. Finally, *H. influenzae* showed complete resistance to nalidixic acid and cotrimoxazole, while *A. baumannii* was highly resistant to ticarcillin and cotrimoxazole but remained susceptible to gentamicin and netilmicin. The multi-drug resistance in *A. baumannii* is a known challenge globally [21].

## 5. CONCLUSION

This study reveals alarmingly high levels of antibiotic resistance among bacteria causing pulmonary infections in Niamey, Niger. The widespread resistance to first-line and even last-resort antibiotics, such as β-lactams and glycopeptides, severely complicates the empirical treatment of lung diseases and poses a grave public health threat.

Unnecessary exposure to antibiotics through self-medication and inappropriate prescription practices appears to be a major driver of this phenomenon. The findings highlight the urgent need for robust surveillance systems, antibiotic stewardship programs, and the integration of molecular diagnostics to guide therapy and preserve the effectiveness of remaining antibiotics.

## 6. STRATEGIC RECOMMENDATIONS

Based on the findings of this study, the following strategic recommendations are proposed to address the challenge of antibiotic resistance in Niamey and the broader region:

**1. Reinforce the National Antimicrobial Resistance (AMR) Surveillance Program:** A systematic, continuous surveillance system is crucial for tracking resistance trends in key pathogens. This program should integrate data from hospitals and community laboratories to create a comprehensive national antibiogram. This data will enable the development of evidence-based local and national treatment guidelines.

**2. Implement Antibiotic Stewardship Programs (ASPs) in Healthcare Facilities:** Hospitals and clinics should establish multidisciplinary ASPs focused on promoting the appropriate use of antibiotics. Key interventions should include formulary restrictions for last-resort antibiotics, prospective audit and feedback on prescriptions, and requiring justification for broad-spectrum antibiotic use.

**3. Strengthen Diagnostic Capacity with Molecular Assays:** The study highlights the need for rapid diagnostics. Investment in molecular techniques (e.g., PCR-based assays) to detect specific resistance genes (such as *mecA* for MRSA, or genes for extended-spectrum beta-lactamases) would allow for targeted therapy, reducing reliance on broad-spectrum empirical treatment and improving patient outcomes.

**4. Regulate the Sale and Distribution of Antibiotics:** Strict government regulations are needed to curb the informal sale of antibiotics without a prescription. Enforcing laws against the sale of drugs in unregulated markets ("street pharmacies") is a critical step to reduce self-medication and misuse.

**5. Public and Professional Education Campaigns:** Launch targeted awareness campaigns for the general public on the dangers of antibiotic misuse and the importance of completing prescribed courses. Concurrently, provide continuous medical education for healthcare professionals on rational prescribing practices, infection prevention and control (IPC), and the local AMR landscape.

**6. Enhance Infection Prevention and Control (IPC) Measures:** Reducing the transmission of resistant organisms is as important as controlling antibiotic use. Strengthening basic IPC practices, such as hand hygiene, environmental cleaning, and patient isolation protocols in healthcare settings, will decrease the incidence of healthcare-associated infections and limit the spread of AMR.

## ACKNOWLEDGEMENT

We express our sincere thanks to the Nigerien Agency for Scholarships and Allowances (ANAB) and the Service de Coopération et d'Action Culturelle (SCAC, French Cooperation in Niger and Burkina Faso) for financing this work.

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