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Primary Drug-Resistant Mycobacterium Tuberculosis in Yaoundé, Cameroon: Microbiological and Molecular Characterization

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Abstract

Background: Drug-resistant tuberculosis threatens global TB control, yet molecular resistance patterns remain poorly characterized in Central Africa. We investigated the prevalence and molecular characteristics of primary drug resistance among newly diagnosed pulmonary TB patients in Yaoundé, Cameroon. **Methods:** We conducted a prospective study at Jamot Hospital, Yaoundé (March 2023-September 2025). Of 147 screened patients, 105 treatment-naïve, HIV-negative, smear-positive pulmonary TB patients aged ≥ 21 years residing within 50 km of Yaoundé were enrolled. MGIT liquid culture and GenoType® MTBDRplus line-probe assay were used to characterize resistance to rifampicin and isoniazid and to identify specific mutations in *rpoB*, *katG*, and *inhA* genes. Of 100 culture-positive isolates, 98 were successfully genotyped. **Results:** Among 98 genotyped isolates, 32.7% (32/98) exhibited drug resistance: 14.3% rifampicin mono-resistance, 11.2% isoniazid mono-resistance, and 7.1% MDR-TB, substantially exceeding national estimates (2.4%). Molecular characterization revealed a distinctive regional resistance profile: *rpoB* H526Y/D mutations predominated (76.2% of rifampicin-resistant isolates) over the globally common S531L (23.8%), while *katG* S315T accounted for 77.8% of isoniazid resistance. Among the 66 fully susceptible patients who completed standard 2HRZE/4HR therapy, 96.2% achieved sputum conversion by month 6; outcome data for resistant patients referred to MDR-TB programmes were not available within the study timeframe. The el-

evated mono-resistance rates warrant cautious interpretation, as undisclosed prior treatment cannot be entirely excluded. **Conclusion:** Yaoundé exhibits unexpectedly high primary drug resistance with a distinctive H526-predominant molecular signature. Whether this pattern reflects clonal transmission of specific lineages requires confirmation by whole-genome sequencing and strain typing, which are urgently needed. Universal molecular drug susceptibility testing at diagnosis is a priority. Findings are applicable to smear-positive, treatment-naïve adults ≥ 21 years near Yaoundé and should not be extrapolated to paediatric, smear-negative, or HIV-co-infected populations.

Keywords

Line-Probe Assay, *rpoB* Mutations, *katG* Mutations, Molecular Epidemiology, Drug-Resistant Tuberculosis, Cameroon

1. Introduction

Tuberculosis (TB) remains among the leading infectious causes of mortality globally, with the African region disproportionately affected. In 2023, the WHO documented 8.2 million new TB cases and 1.25 million deaths worldwide, yet these figures likely underestimate the true burden in resource-limited settings where diagnostic capacity remains constrained. Cameroon, classified as a high TB burden country with 174 cases per 100,000 population and 29% HIV co-infection, exemplifies these diagnostic and surveillance challenges [1] [2].

The emergence of drug-resistant TB, particularly multidrug-resistant TB (MDR-TB, defined as resistance to at least rifampicin and isoniazid), threatens to reverse decades of TB control progress. WHO estimates suggest that 2% - 3% of new TB cases in Cameroon harbour drug resistance, yet national programme data indicate substantial underdiagnosis due to limited access to Drug Susceptibility Testing (DST), available in fewer than 10% of diagnosed cases [1] [3].

Molecular diagnostics, particularly the GenoType MTBDRplus line-probe assay, have revolutionized rapid resistance detection by identifying specific mutations in resistance-determining regions [4] [5]. For rifampicin, mutations within codons 507 - 533 of the *rpoB* gene account for >95% of resistance, with S531L predominating globally (50% - 70%) [6] [7]. For isoniazid, mutations in *katG* codon 315 and the *inhA* promoter confer high-level and low-level resistance, respectively [8]-[10].

Despite bearing a substantial tuberculosis burden, Cameroon lacks comprehensive data on the molecular epidemiology of primary drug resistance. Published evidence on resistance-associated mutation patterns among treatment-naïve tuberculosis patients in Yaoundé, the country's capital and largest metropolitan area, remains unavailable [2] [11] [12]. Elucidating whether the molecular resistance profiles circulating in Cameroon mirror globally established patterns or exhibit unique regional signatures is of paramount importance for refining mo-

lecular diagnostic algorithms, informing therapeutic decision-making, and enhancing the control of drug-resistant tuberculosis transmission.

We therefore conducted a prospective molecular epidemiological study to: 1) determine the prevalence of primary drug resistance among newly diagnosed pulmonary TB patients in Yaoundé; 2) characterize the molecular mutations conferring rifampicin and isoniazid resistance; and 3) assess treatment response in relation to baseline resistance patterns. We hypothesized that urban Yaoundé would exhibit higher resistance prevalence than national estimates and potentially distinctive mutation patterns.

2. Methods

2.1. Study Setting and Design

This prospective longitudinal study was conducted from March 2023 to September 2025 at Jamot Hospital in Yaoundé, Cameroon's national reference centre for tuberculosis management. The study adhered to STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines.

2.2. Ethical Considerations

Ethical approval was obtained from the Regional Ethics Committee for Human Health Research, Central Region (CERSHC), under approval number CE N˚1297/CRERSHC/2022, dated 19 September 2022. The study was conducted in strict accordance with the Declaration of Helsinki and Good Clinical Practice guidelines. Written informed consent was obtained from all participants. Confidentiality was maintained through numerical coding of all data.

2.3. Sample Size

The target sample size was calculated *a priori* based on the nationally representative estimate of rifampicin resistance (RR-TB, proxy for MDR-TB) among new bacteriologically confirmed pulmonary TB cases in Cameroon reported by Noeske *et al.* (2018): a RR-TB prevalence of 1.6% (95% CI: 0.8% - 2.3%) [13]. This figure was adopted as the reference prevalence for the present sentinel-site study. Using the standard single-proportion formula $n = z^2 \times p\,(1 - p)/e^2$, with $z = 1.96$ (two-sided 95% CI), p = 0.016, and a desired absolute precision of $e = \pm 2.6$ percentage points, the minimum evaluable sample size was: $n = (1.96)^2 \times 0.016 \times 0.984/(0.026)^2 \approx 90$ patients.

2.4. Sampling and Eligibility

Consecutive non-probability sampling of eligible treatment-naïve TB patients presenting at Jamot Hospital was used. All eligible patients were approached sequentially from March 2023 until the target was reached. The extended recruitment period captured seasonal and temporal variations in TB epidemiology.

Inclusion criteria: Age ≥ 21 years; sputum smear-positive by auramine or Ziehl-Neelsen microscopy; no documented prior anti-TB treatment (verified

through patient interview, medical records review, and national TB programme registry check); residence within 50 km of Yaoundé to facilitate follow-up.

These criteria were selected to characterise primary resistance in the highest-burden diagnostic subgroup accessible at this referral centre. The lower age limit of 21 years reflects minimum legal consent age under Cameroonian regulations and the dominant TB demographic in urban Yaoundé. Smear-positivity was required to ensure sufficient bacillary load for reliable MGIT culture and MTBDR-plus genotyping. Residence within 50 km was necessary to achieve 6-month follow-up completeness. Accordingly, the findings are generalisable to smear-positive, treatment-naïve adults ≥ 21 years residing near Yaoundé and should not be extrapolated to paediatric cases, smear-negative disease, or patients residing in rural or remote areas.

Exclusion criteria: Extrapulmonary TB only; HIV, hepatitis B, or hepatitis C co-infection (verified by rapid diagnostic testing); ongoing immunosuppressive therapy; baseline serum creatinine > 120 μmol/L or ALT/AST > 2 × upper limit of normal; inability or unwillingness to provide informed consent or complete 6-month follow-up.

The exclusion of HIV, HBV, and HCV co-infected patients was motivated by the differential influence of co-infection on resistance rates and treatment outcomes, which would have introduced important confounding. These immunocompromised patients represent a distinct subgroup warranting dedicated investigation. Their exclusion limits the generalizability of resistance estimates given Cameroon's high HIV-TB co-infection rate (29%) and should be acknowledged as a key limitation.

2.5. Participant Flow

Figure 1 presents the complete participant flow. Briefly, 147 patients were screened for eligibility during the study period. Of these, 42 were excluded: 12 reported prior anti-TB treatment, 9 were HIV-positive, 7 had HBV or HCV co-infection, 6 resided > 50 km from Yaoundé, 4 had extra pulmonary TB only, 2 had abnormal renal/hepatic function, and 2 refused consent. The remaining 105 patients were enrolled. Of these, 100 (95.2%) yielded positive MGIT cultures; 5 remained culture-negative and were excluded from resistance analyses. Among the 100 culture-positive isolates, 98 (98.0%) were successfully genotyped by MTBDR-plus (2 yielded indeterminate results due to insufficient DNA). All 105 enrolled patients completed the 6-month treatment follow-up.

2.6. Data Collection and Specimen Processing

Sociodemographic, clinical, and microbiological data were collected prospectively using a standardised case report form. An early-morning sputum specimen (~2 mL) was collected from each enrolled patient and transported within 4 hours at 4˚C to the TB Research Laboratory, Biotechnology Centre of Nkolbisson, University of Yaoundé I.

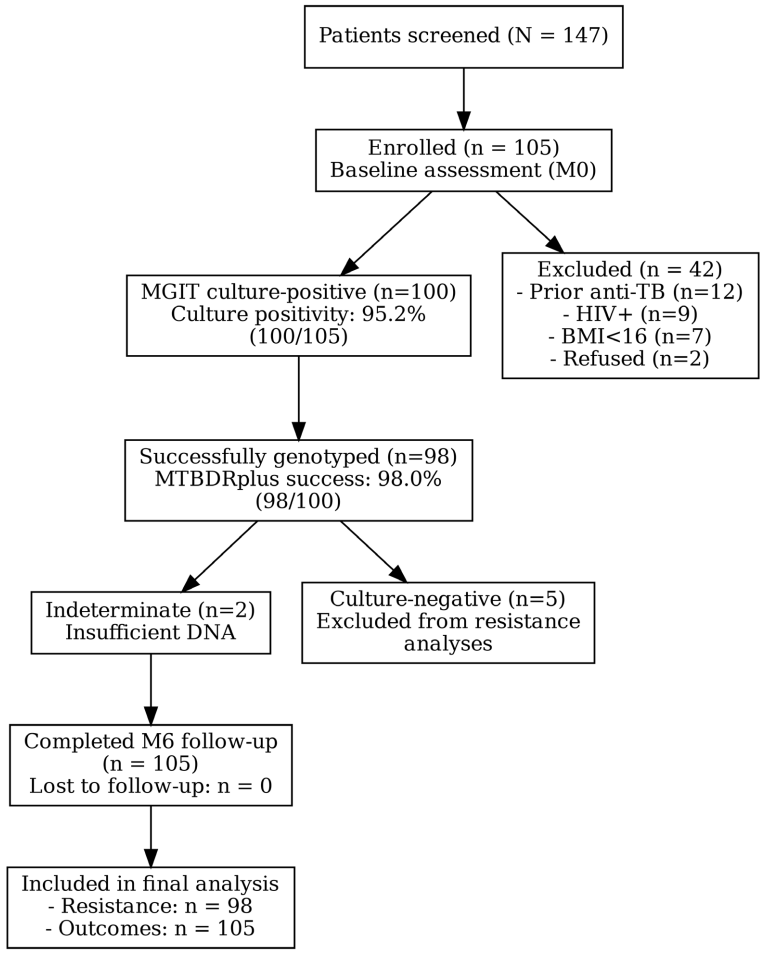


Figure 1. Participant flow. Of 147 patients screened, 42 were excluded (prior treatment, HIV infection, HBV/HCV co-infection, distant residence, extrapulmonary TB, renal/hepatic impairment, or refusal). A total of 105 patients were enrolled; 100 were culture-positive and 98 successfully genotyped. All enrolled patients completed 6-month follow-up.

Auramine O Fluorescence Microscopy: Sputum smears were stained using the standard auramine O fluorochrome method and examined at 400× magnification. Smear positivity was graded according to WHO standards: scanty (1 - 19 AFB per slide length), 1+ (20 - 199 AFB), 2+ (5 - 50 AFB per field), or 3+ (>50 AFB per field) [14] [15].

MGIT Culture: Mycobacterial culture was performed using the BACTEC™ MGIT™ 960 automated liquid culture system (Becton Dickinson). Sputum samples were decontaminated using the NaCl-NaOH method, neutralized with 67 mM phosphate buffer, and centrifuged at 3000 – 4000 × g for 15 minutes at 4˚C. The pellet (0.5 mL) was inoculated into MGIT tubes with Middlebrook 7H9 broth, OADC enrichment, and PANTA antibiotic mixture. Automated fluorescence monitoring was performed from day 14 through day 42. Positive cultures were confirmed as *M. tuberculosis* complex by colony morphology, acid-fast smear, and MTBDRplus [16].

GenoType® MTBDRplus Assay: DNA was extracted from MGIT-positive cul-

tures by standardised thermal lysis. Multiplex PCR targeted *rpoB*, *katG*, and the *inhA* promoter [13] [17]. Amplicons were hybridized to nitrocellulose strips containing 27 oligonucleotide probes. Strip interpretation followed the manufacturer's reading guide and was cross-validated by two independent observers.

2.7. Patient Management after Baseline DST

Baseline DST results were communicated to the treating clinician within 48 hours of genotyping. Patients with fully susceptible isolates were maintained on the standard WHO 2HRZE/4HR regimen. Patients with rifampicin mono-resistance were referred for individualised regimen adjustment in accordance with national MDR-TB guidelines; patients meeting MDR-TB criteria received WHO-recommended MDR-TB regimens. Because resistant cases were moved to alternative regimens, sputum conversion data are reported separately by baseline resistance status to avoid confounding treatment-outcome comparisons.

2.8. Statistical Analysis

Data were analysed using IBM SPSS 23.0 and R 4.2.0. Categorical variables are reported as frequencies and percentages with 95% Wilson score CIs; continuous variables as medians with IQRs. Drug resistance prevalence was estimated with exact binomial 95% CIs. Between-group comparisons used Fisher's exact test and Mann-Whitney U test. Sputum conversion rates were compared by Kaplan-Meier analysis with log-rank tests. All tests were two-tailed ($p < 0.05$).

3. Results

3.1. Baseline Characteristics

A total of 105 treatment-naïve pulmonary tuberculosis patients were enrolled at Jamot Hospital between March 2023 and September 2025 and were completed the 6-month treatment follow-up.

The cohort was predominantly male (73.3%, n = 77) with a median age of 34 years (range: 21 - 65 years). Baseline bacillary burden was substantial, with 70.5% (74/105) exhibiting high bacterial loads: 42.9% (n = 45) graded 2+ and 25.7% (n = 27) graded 3+. Lower-grade positivity accounted for the remainder: 17.1% (n = 18) scanty and 14.3% (n = 15) graded 1+ (Table 1).

Table 1. Distribution of baseline sputum smear grades.

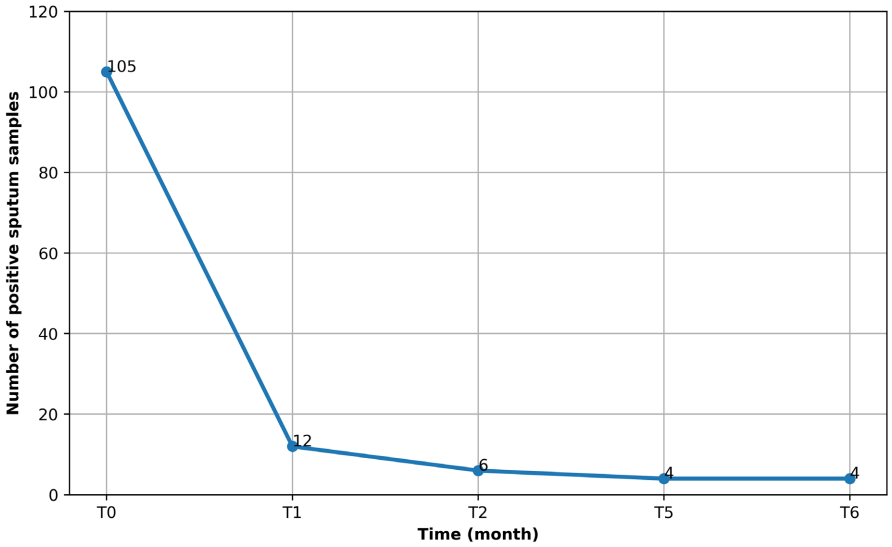
Smear Grade	n	%
Scanty	18	17.15
1+	15	14.29
2+	45	42.86
3+	27	25.70
Total	**105**	**100**

3.2. MGIT Culture Results

Viable mycobacterial growth was detected in 95.2% (100/105) of baseline specimens. The five culture-negative cases (4.8%) likely represented non-viable bacilli, pre-analytical errors, or contamination during processing, as all were smear-positive.

3.3. Treatment Response and Sputum Conversion

Sputum conversion data are presented separately by baseline drug susceptibility status, since patients with drug-resistant isolates were switched to alternative regimens (see Section 2.7). Among the 66 fully susceptible patients who continued standard 2HRZE/4HR therapy, rapid bacteriological clearance was observed: 88.6% converted to smear-negative by month 1, increasing to 94.3% by month 2, and 96.2% (63/66) achieved sustained sputum conversion at treatment completion (month 6). Three patients (4.5% among susceptible cases) remained persistently smear-positive at month 6, raising concern for undetected resistance to pyrazinamide or ethambutol, poor adherence, or pharmacokinetic variability. For the 32 patients with resistant isolates, treatment outcomes following regimen adjustment are reported descriptively: all 14 rifampicin mono-resistant and all 7 MDR-TB patients were referred to specialised MDR-TB management; follow-up data from those programmes were not available within the timeframe of this study and are therefore not included in the overall cure-rate estimate (Figure 2).



Note: Analysis limited to patients on standard 2HRZE/4HR; drug-resistant cases (n = 32) were excluded.

Figure 2. Sputum smear conversion during standard therapy. In fully drug-susceptible pulmonary TB patients (n = 66), smear conversion reached 88.6% (M1), 94.3% (M2), and 96.2% (M6); 4.5% remained positive at treatment completion.

3.4. Drug Resistance Patterns

Among the 100 culture-positive isolates, 98 were successfully genotyped using the GenoType MTBDRplus assay. Drug resistance was detected in 32.7% (32/98) of

isolates: 14 isolates (14.3%) exhibited rifampicin mono-resistance, 11 (11.2%) showed isoniazid mono-resistance, and 7 (7.1%) met criteria for MDR-TB. The remaining 67.3% (66/98) were fully susceptible (Table 2).

Table 2. Drug resistance profiles among genotyped *M. tuberculosis* isolates.

Resistance Profile	n	% of Total (n = 98)	% of Resistant (n = 32)
Fully susceptible (RIF-S, INH-S)	66	67.3	—
Rifampicin mono-resistant (RIF-R)	14	14.3	43.8
Isoniazid mono-resistant (INH-R)	11	11.2	34.4
Multidrug-resistant (RIF-R + INH-R)	7	7.1	21.9
Total	**98**	**100**	**100**

RIF = rifampicin; INH = isoniazid; -S = susceptible; -R = resistant.

3.5. Molecular Characterisation of Rifampicin Resistance

Twenty-one rifampicin-resistant isolates were analyzed for *rpoB* gene mutations within the RRDR (codons 507 - 533). Three mutations accounted for all detected resistance: *rpoB* H526Y and H526D (probe MUT2B) were equally prevalent at 38.1% (8/21) each, while *rpoB* S531L (MUT3) was identified in 23.8% (5/21) of cases (Table 3). All three mutations confer high-level rifampicin resistance by disrupting rifampicin binding to the β-subunit of RNA polymerase.

3.6. Molecular Characterisation of Isoniazid Resistance

Eighteen isoniazid-resistant isolates harboured mutations in *katG* or the *inhA* promoter. The *katG* S315T mutation predominated: S315T2 was detected in 55.6% (10/18) and S315T1 in 22.2% (4/18), collectively accounting for 77.8% of isoniazid resistance. The *inhA* promoter mutation C-15T was identified in 22.2% (4/18) of isolates (Table 4).

Table 3. Distribution of *rpoB* mutations conferring rifampicin resistance.

Mutation	Codon (Probe)	n	%	Resistance Level
rpoB S531L	531 (MUT3)	5	23.8	High
rpoB H526Y	526 (MUT2B)	8	38.1	High
rpoB H526D	526 (MUT2B)	8	38.1	High
Total		**21**	**100**	

Table 4. Distribution of *katG* and *inhA* mutations conferring isoniazid resistance.

Mutation	Gene/Region	n	%	Resistance Level
inhA C-15T	Promoter region	4	22.2	Low
katG S315T1	Codon 315	4	22.2	High
katG S315T2	Codon 315	10	55.6	High
Total		**18**	**100**	

4. Discussion

This prospective molecular epidemiological study reveals three key findings. First, primary drug resistance in Yaoundé (32.7%) substantially exceeds both national surveillance estimates (2.4%) and rates reported in neighbouring Central African countries (3% - 5%) [3]. Second, the molecular signature of rifampicin resistance differs markedly from global patterns, with H526 mutations predominating (76.2%) over S531L (23.8%). Third, among patients with susceptible isolates who received standard therapy, 96.2% achieved microbiological cure, validating regimen efficacy when drug susceptibility is preserved.

The predominance of high bacillary loads at presentation (70.5% with smear grades 2+ or 3+) reflects diagnostic delays characteristic of resource-limited settings. These patients represent highly efficient transmitters; published models estimate that high-grade smear-positive cases generate substantially more secondary infections than low-grade cases, though precise transmission ratios are model-dependent and setting-specific [18] [19]. The concurrent presence of drug-resistant strains within this high-burden subgroup substantially amplifies public health risk.

Treatment-outcome interpretation is restricted to the 66 fully susceptible patients who received standard 2HRZE/4HR therapy throughout follow-up, as resistant-case patients were switched to alternative regimens. The 96.2% sputum conversion rate among susceptible patients aligns with international benchmarks and is consistent with prior regional data [11]. Persistent smear positivity in three susceptible patients (4.5%) may reflect undetected resistance to pyrazinamide or ethambutol, pharmacokinetic variability, or paradoxical upgrading. We did not link the overall cure estimate to baseline susceptibility status for the full cohort, as data on resistant-case outcomes following regimen change were unavailable.

The distribution of *rpoB* mutations in our cohort differed from global patterns, where S531L is the predominant rifampicin-resistance mutation (60% - 70% of cases). In contrast, H526 mutations (H526Y/H526D) accounted for 76.2% (16/21) of rifampicin-resistant isolates, while S531L represented only 23.8% (5/21). This finding may indicate a distinct local resistance profile in Yaoundé. However, MTBDRplus mutation data alone cannot determine whether this predominance reflects clonal transmission, founder effects, or convergent evolution. Further studies using whole-genome sequencing or strain-typing methods are needed to clarify the molecular epidemiology of rifampicin-resistant *M. tuberculosis* in this setting [20] [21].

The predominance of *katG* S315T mutations (77.8%) aligns with global surveillance data where this mutation accounts for 60% - 80% of isoniazid resistance [22]. The *inhA* promoter C-15T mutation (22.2%) confers low-level isoniazid resistance with important implications for cross-resistance to ethionamide, a key second-line MDR-TB drug [23]. MDR-TB patients harbouring *inhA* mutations may therefore require alternative regimens incorporating linezolid, bedaquiline, or delamanid [24].

The high prevalence of rifampicin mono-resistance (14.3%) and isoniazid mono-resistance (11.2%) observed in this treatment-naïve cohort was unexpected, as mono-resistance rates are generally reported to be below 2% in well-characterized treatment-naïve populations [25]. Although treatment-naïve status was verified through patient interviews and review of hospital and national TB programme records, undisclosed prior treatment cannot be entirely excluded. Consequently, some resistance may represent acquired rather than primary resistance. Alternative explanations include transmission of mono-resistant strains with preserved fitness, heteroresistance below the detection threshold of the MTBDRplus assay [26], or independent acquisition of resistance-conferring mutations [27]. Further studies incorporating whole-genome sequencing and contact tracing are required to clarify the origin and transmission dynamics of these resistance profiles.

5. Study Limitations

Several limitations must be acknowledged. The eligibility criteria, including age ≥ 21 years, smear-positivity, and exclusion of HIV/HBV/HCV co-infected patients, restrict generalisability to the population actually studied: smear-positive, treatment-naïve adults ≥ 21 years near Yaoundé without immunocompromising comorbidities. The MTBDRplus assay detects only rifampicin and isoniazid resistance, missing resistance to pyrazinamide, ethambutol, and fluoroquinolones. The relatively small sample size (n = 105) from a single national reference hospital may not reflect nationwide community-based patterns. Lack of whole-genome sequencing prevented detailed lineage identification and transmission network analysis. Treatment outcome data were unavailable for patients with resistant isolates who were transferred to MDR-TB programmes, limiting our ability to compare outcomes comprehensively across resistance categories.

6. Conclusion

This study documents alarmingly high primary drug resistance in Yaoundé, Cameroon: 32.7% among treatment-naïve TB patients, including 7.1% MDR-TB, far exceeding national estimates. Molecular profiling reveals a distinctive H526Y/D-predominant rifampicin resistance profile and *katG* S315T-driven isoniazid resistance. Whether this pattern reflects clonal transmission of specific lineages requires confirmation by whole-genome sequencing and strain typing, which are urgently needed. Universal rapid molecular DST at diagnosis, extended to pyrazinamide, ethambutol, and second-line drugs, intensified contact tracing, enhanced adherence support, and strengthened infection control are immediate priorities for TB elimination in this high-burden setting.

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Author Contributions

A.E.M. and J.N.B. designed the study protocol and secured ethical approval. A.E.M. and A.U.N.B. recruited participants and collected clinical data. J.P.A.A., A.E.M., and E.D.F.M.N. performed sputum microscopy, MGIT culture, and MTBDRplus molecular testing. E.N.M. conducted statistical analyses. A.E.M. drafted the manuscript. S.H.R. and J.N.B. provided critical revisions. All authors approved the final version.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] World Health Organization (2024) Global Tuberculosis Report 2024. World Health Organization.
https://www.who.int/teams/global-tuberculosis-programme/tb-reports

[2] Ministry of Public Health, Cameroon (2023) National Tuberculosis Control Programme Annual Report 2022. National Tuberculosis Control Programme, Ministry of Public Health.
https://fiscameroun.org/wp-content/uploads/2023/06/2022-FIS-Activity-report.pdf

[3] Mbuh, T.P., Mendjime, P., Goupeyou-Wandji, I., Donkeng-Donfack, V.F., Kahou, J., Endale Mangamba, L., *et al.* (2024) Trends of Drug-Resistant Tuberculosis and Risk Factors to Poor Treatment-Outcome: A Database Analysis in Littoral Region-Cameroon, 2013-2022. *BMC Public Health*, **24**, Article No. 3195.
https://doi.org/10.1186/s12889-024-20585-8

[4] Tang, P., Wang, X., Shen, X., Shi, M., Zhu, X., Yu, X., *et al.* (2017) Use of DNA Microarray Chips for the Rapid Detection of *Mycobacterium tuberculosis* Resistance to Rifampicin and Isoniazid. *Experimental and Therapeutic Medicine*, **13**, 2332-2338.
https://doi.org/10.3892/etm.2017.4250

[5] Aminu, A.I. and Tukur, A.D. (2017) Detection of Multidrug Resistant Tuberculosis (MDR-TB) among Rifampicin-Resistant TB Patients Using Line Probe Assay (LPA) in Kano, Nigeria. *Bayero Journal of Pure and Applied Sciences*, **9**, 1-8.
https://doi.org/10.4314/bajopas.v9i2.1

[6] Patel, Y., Soni, V., Rhee, K.Y. and Helmann, J.D. (2023) Mutations in *RPOB* That Confer Rifampicin Resistance Can Alter Levels of Peptidoglycan Precursors and Affect B-Lactam Susceptibility. *mBio*, **14**, e0316822.
https://doi.org/10.1128/mbio.03168-22

[7] Chikaonda, T., Ketseoglou, I., Nguluwe, N., Krysiak, R., Thengolose, I., Nyakwawa, F., *et al.* (2017) Molecular Characterisation of Rifampicin-Resistant *Mycobacterium tuberculosis* Strains from Malawi. *African Journal of Laboratory Medicine*, **6**, a463.
https://doi.org/10.4102/ajlm.v6i2.463

[8] Bollela, V.R., Namburete, E.I., Feliciano, C.S., Macheque, D., Harrison, L.H. and Caminero, J.A. (2016) Detection of *katG* and *inh*A Mutations to Guide Isoniazid and

Ethionamide Use for Drug-Resistant Tuberculosis. *The International Journal of Tuberculosis and Lung Disease*, **20**, 1099-1104. https://doi.org/10.5588/ijtld.15.0864

[9] Lale Ngema, S., Dookie, N., Perumal, R., Nandlal, L., Naicker, N., Peter Letsoalo, M., *et al.* (2023) Isoniazid Resistance-Conferring Mutations Are Associated with Highly Variable Phenotypic Resistance. *Journal of Clinical Tuberculosis and Other Mycobacterial Diseases*, **33**, Article ID: 100387. https://doi.org/10.1016/j.jctube.2023.100387

[10] Nono, V.N., Nantia, E.A., Mutshembele, A., Teagho, S.N., Simo, Y.W.K., Takong, B.S., *et al.* (2025) Prevalence of *katG* and *inhA* Mutations Associated with Isoniazid Resistance in *Mycobacterium tuberculosis* Clinical Isolates in Cameroon. *BMC Microbiology*, **25**, Article No. 127. https://doi.org/10.1186/s12866-025-03816-9

[11] Kuaban, A., Gbayanga, N.G., Kuaban, C. and Noeske, J. (2026) Effect of Extending the Intensive Phase Treatment on Treatment Outcomes in New Smear-Positive Pulmonary Tuberculosis Patients in Yaoundé, Cameroon. *Health Research in Africa*, **4**, 57-63. https://doi.org/10.5281/zenodo.18267491

[12] Mpoh, M., Deli, V., Daniel, T. and Salvo, F. (2023) Safety of Antituberculosis Agents Used for Multidrug-Resistant Tuberculosis among Patients Attending the Jamot Hospital of Yaounde, Cameroon. *International Journal of Mycobacteriology*, **12**, 168-174. https://doi.org/10.4103/ijmy.ijmy_88_23

[13] Noeske, J., Yakam, A.N., Foe, J.L.A., Nguafack, D. and Kuaban, C. (2018) Rifampicin Resistance in New Bacteriologically Confirmed Pulmonary Tuberculosis Patients in Cameroon: A Cross-Sectional Survey. *BMC Research Notes*, **11**, Article No. 580. https://doi.org/10.1186/s13104-018-3675-0

[14] Médecins Sans Frontières, & Partners in Health (2025) MSF Medical Guidelines: Tuberculosis. https://medicalguidelines.msf.org

[15] Gaikwad, U.N., Das, P., Bhargava, A., Behera, A.K., Jagzape, T.B. and Chandraker, S. (2025) Evaluation of Fluorescein Diacetate Viability Staining during Treatment Follow-Up Examinations of Smear Positive Pulmonary Tuberculosis Patients. *The International Journal of Mycobacteriology*, **14**, 30-35. https://doi.org/10.4103/ijmy.ijmy_220_24

[16] Hanna, B.A., Ebrahimzadeh, A., Elliott, L.B., Morgan, M.A., Novak, S.M., Rusch-Gerdes, S., *et al.* (1999) Multicenter Evaluation of the BACTEC MGIT 960 System for Recovery of Mycobacteria. *Journal of Clinical Microbiology*, **37**, 748-752. https://doi.org/10.1128/jcm.37.3.748-752.1999

[17] Djoufack, G.D., Abongwa, L.E., Campbell, A.O., Campbell, J.S.O., Gorden, K., Nkodo, J.M.M., *et al.* (2025) Molecular Characterization of *Mycobacterium tuberculosis* in HIV Patients Receiving Antiretroviral Drugs in Cameroon. *The International Journal of Mycobacteriology*, **14**, 182-190. https://doi.org/10.4103/ijmy.ijmy_47_25

[18] Beviere, M., Reissier, S., Penven, M., Dejoies, L., Guerin, F., Cattoir, V., *et al.* (2023) The Role of Next-Generation Sequencing (NGS) in the Management of Tuberculosis: Practical Review for Implementation in Routine. *Pathogens*, **12**, Article 978. https://doi.org/10.3390/pathogens12080978

[19] Storla, D.G., Yimer, S. and Bjune, G.A. (2008) A Systematic Review of Delay in the Diagnosis and Treatment of Tuberculosis. *BMC Public Health*, **8**, Article No. 15. https://doi.org/10.1186/1471-2458-8-15

[20] Soidah, S., Subroto, T., Faizal, I. and Yusuf, M. (2025) Beyond Hotspot Mutations: Diagnostic Relevance of High Frequency, Low Frequency, and Disputed *rpoB* Variants in Rifampicin-Resistant *Mycobacterium tuberculosis*. *Pathogens*, **15**, Article 16. https://doi.org/10.3390/pathogens15010016

[21] Liu, Y., Sun, Y., Zhang, X., Zhang, Z., Xing, Q., Ren, W., *et al.* (2021) Evaluation of the Frequency of Mutation Genes in Multidrug-Resistant Tuberculosis (MDR-TB) Strains in Beijing, China. *Epidemiology and Infection*, **149**, e21. https://doi.org/10.1017/s0950268820003131

[22] Seifert, M., Catanzaro, D., Catanzaro, A. and Rodwell, T.C. (2015) Genetic Mutations Associated with Isoniazid Resistance in *Mycobacterium tuberculosis*: A Systematic Review. *PLOS ONE*, **10**, e0119628. https://doi.org/10.1371/journal.pone.0119628

[23] Vilchèze, C. and Jacobs JR., W.R. (2014) Resistance to Isoniazid and Ethionamide in *Mycobacterium tuberculosis*: Genes, Mutations, and Causalities. *Microbiology Spectrum*, **2**, MGM2-0014-2013. https://doi.org/10.1128/microbiolspec.mgm2-0014-2013

[24] World Health Organization (2019) WHO Consolidated Guidelines on Drug-Resistant Tuberculosis Treatment. World Health Organization. https://www.who.int/publications/i/item/9789241550529

[25] Yu, H., Kim, T.Y., Kim, G., Shim, H.J., Kang, O.K., Kim, S., *et al.* (2023) Performance Evaluation of the BACTEC MGIT 960 System for Rifampin Drug-Susceptibility Testing of *Mycobacterium Tuberculosis* Using the Current WHO Critical Concentration. *Journal of Clinical Microbiology*, **61**, e0108622. https://doi.org/10.1128/jcm.01086-22

[26] Khabibullina, N.F., Kutuzova, D.M., Burmistrova, I.A. and Lyadova, I.V. (2022) The Biological and Clinical Aspects of a Latent Tuberculosis Infection. *Tropical Medicine and Infectious Disease*, **7**, Article 48. https://doi.org/10.3390/tropicalmed7030048

[27] Belay, G., Getachew, H., Birku, T., Tadese, A., Gashaw, Y., Getie, M., *et al.* (2025) Rifampicin-Resistant *Mycobacterium tuberculosis* and Unsuccessful Results from Xpert® MTB/Rif-Ultra Assay in Amhara Region, Ethiopia. *Journal of Clinical Tuberculosis and Other Mycobacterial Diseases*, **40**, Article ID: 100528. https://doi.org/10.1016/j.jctube.2025.100528