

Patterns of First-Line Anti-Tuberculosis Drug Resistance and Multidrug-Resistant Tuberculosis and Their Association with Diabetes Co-Morbidity: A Cross-Sectional Study at the NTBLTC, Saye, Zaria, Northern Nigeria

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Cite this article as: Victor Edet Essien, Abiodun Olaiya Paul, Francisca Oby Nwaokorie. Patterns of first-line anti-tuberculosis drug resistance and multidrug-resistant tuberculosis and their association with diabetes co-morbidity: a cross-sectional study at the NTBLTC, Saye, Zaria, Northern Nigeria. Texila Int J Acad Res. 2026;13(4). <https://doi.org/10.21522/TIJAR.2014.13.04.Art021>

Received: 24.08.2026

Accepted: 08.10.2026

Published: 30.10.2026

Article Type: Original Research

Abstract Drug-resistant tuberculosis threatens global tuberculosis control, and diabetes mellitus is a plausible but incompletely characterised driver of both active and resistant disease in high-burden settings. This study characterised first-line anti-tuberculosis drug-resistance patterns, including multidrug-resistant tuberculosis, and examined their association with diabetes co-morbidity among presumptive tuberculosis patients at a national reference centre in northern Nigeria. A facility-based, descriptive cross-sectional study enrolled 200 consecutive presumptive-TB adults at the National Tuberculosis and Leprosy Training Centre, Saye, Zaria. Tuberculosis was diagnosed using Ziehl–Neelsen smear microscopy, the Xpert MTB/RIF assay, and Löwenstein–Jensen culture with first-line drug-susceptibility testing by the proportion method; a single fasting plasma glucose measurement classified diabetes status. Associations were assessed using chi-square or Fisher exact tests and logistic regression at a significance threshold of 0.05. Bacteriologically confirmed tuberculosis was identified in 26.0% of participants and diabetes in 17.0%. Among 48 culture-positive isolates with valid drug-susceptibility testing, isoniazid resistance was most frequent (22.9%), followed by rifampicin (16.7%), streptomycin (14.6%), and ethambutol (10.4%); across all 52 confirmed cases, rifampicin resistance was 17.3% (9/52) and multidrug-resistant tuberculosis 11.5% (6/52). Any first-line resistance and multidrug resistance were more than twice as frequent among the 13 tuberculosis patients with diabetes than among the 39 without (46.2% versus 20.5% and 23.1% versus 7.7%, respectively), although these differences did not reach statistical significance (Fisher exact $p = 0.086$ and $p = 0.157$). Diabetes independently predicted confirmed tuberculosis (adjusted odds ratio 2.34, 95% CI 1.08–5.05), alongside prior tuberculosis treatment and low body-mass index. Because diabetes was based on a single fasting plasma glucose measurement obtained during acute illness, stress hyperglycaemia cannot be excluded. These findings reveal a substantial resistance burden at referral level and a consistent but statistically unconfirmed resistance signal associated with diabetes. Routine drug-susceptibility testing and bidirectional tuberculosis–diabetes screening are warranted, and larger studies are needed to confirm the resistance association.

Keywords Diabetes Mellitus, Drug-Susceptibility Testing, Multidrug-Resistant Tuberculosis, Nigeria, Rifampicin Resistance, Tuberculosis.

Introduction

Tuberculosis (TB) remains among the leading causes of death from a single infectious agent, and the emergence of drug-resistant disease is one of the most serious obstacles to its control worldwide [1]. Each year, hundreds of thousands of people develop rifampicin-resistant or multidrug-resistant tuberculosis (MDR-TB), defined as resistance to at least rifampicin and isoniazid, and only a minority are diagnosed and successfully treated [1, 2]. Drug-resistant tuberculosis requires longer, more toxic and more expensive treatment, and is associated with poorer outcomes, so that understanding its distribution and drivers is central to national control programmes [23, 24].

In parallel with the persistence of tuberculosis, the global prevalence of diabetes mellitus (DM) has risen sharply, most rapidly in low- and middle-income countries undergoing epidemiological transition [3, 4]. The convergence of the two conditions has revived interest in a long-recognised clinical association [4]. Diabetes roughly triples the risk of active tuberculosis and is associated with more severe disease, delayed sputum conversion, higher relapse and increased mortality [3, 5, 6, 28]. A meta-analysis has confirmed the association between diabetes and active tuberculosis across diverse settings [30].

Whether diabetes also contributes to drug-resistant tuberculosis is more contested. Several meta-analyses report an association between diabetes and MDR-TB [7, 8], and pharmacokinetic studies show that diabetic patients achieve lower plasma concentrations of rifampicin and other first-line drugs, which could favour the selection of resistant organisms [9, 10]. Other analyses attribute resistance chiefly to prior treatment, incomplete adherence and programmatic factors rather than to diabetes itself [26, 27]. Reviews of risk factors for drug-resistant tuberculosis consistently identify previous treatment as the dominant determinant [25, 27].

Policy responses recognise the need to address comorbidity. The WHO/Union Collaborative Framework for Care and Control of Tuberculosis and Diabetes recommends bidirectional screening and integrated management [11], and subsequent guidance has reinforced the integration of tuberculosis and non-communicable disease services [12]. Systematic reviews from sub-Saharan Africa report a substantial but heterogeneous burden of diabetes among tuberculosis patients [13-15]. In Nigeria, among the highest-burden tuberculosis countries and with a rapidly rising diabetes prevalence [19], studies have documented elevated diabetes among tuberculosis patients and the feasibility of bidirectional screening [16-18], yet local data specifically linking diabetes to drug-resistant disease remain scarce.

The advent of rapid molecular diagnostics, particularly the Xpert MTB/RIF assay, has transformed the simultaneous detection of tuberculosis and rifampicin resistance [20-22]. Deployed alongside culture and phenotypic drug-susceptibility testing, these tools permit accurate characterisation of resistance patterns at reference level. Against this background, the present study characterised first-line drug-resistance patterns, including MDR-TB, among presumptive-TB patients at a national reference centre in northern Nigeria, and examined whether diabetes co-morbidity was associated with resistant disease, in support of the WHO End TB Strategy and the USAID Global TB Strategy 2023–2030 [29].

Materials and Methods

Study Design and Setting

A quantitative, facility-based, descriptive cross-sectional study was conducted at the National Tuberculosis and Leprosy Training Centre (NTBLTC), Saye, Zaria, Kaduna State, a national reference centre for tuberculosis diagnosis, care and training in north-western Nigeria that receives presumptive-TB patients referred from across the country. Data

collection extended over an approximately two-year period (January 2023 - December 2024), a window adopted to accommodate the long incubation and processing time of mycobacterial culture and variable patient flow.

Study Population, Recruitment and Sample Size

The study population comprised consecutive new adult patients aged 18 years and above presenting with presumptive pulmonary tuberculosis, suggestive clinical features such as chronic cough, fever, night sweats and weight loss, at the outpatient tuberculosis clinics of the NTBLTC. Eligible, consenting patients were enrolled by consecutive sampling until the target sample was reached. Patients younger than 18 years, those with extra-pulmonary tuberculosis, those co-infected with HIV, those already receiving treatment for both tuberculosis and diabetes, and women with gestational diabetes were excluded, as were patients unable to produce an adequate sputum specimen or unwilling to give informed consent.

The minimum sample size was estimated using the Cochran formula for a single proportion, $n = Z^2P(1-P)/d^2$, where $Z = 1.96$ (95% confidence level), P is the estimated prevalence of the outcome and d is the desired absolute precision (0.05). Taking an estimated tuberculosis prevalence of approximately 10% in Nigeria, the calculation yielded a minimum of 138 participants. To allow for non-response and incomplete records and to strengthen the precision of the estimates, the target was increased, and a working sample of 200 presumptive-TB patients was enrolled and analysed.

Specimen Collection and Laboratory Procedures

Two sputum specimens (a spot and an early-morning sample) were collected from each participant into sterile containers and processed at the NTBLTC reference laboratory following

national tuberculosis reference-laboratory standard operating procedures and WHO consolidated guidelines [20, 24]. Direct smears were prepared and examined by Ziehl–Neelsen (ZN) microscopy and graded according to the national scale. The Xpert MTB/RIF assay was used for the molecular detection of the *Mycobacterium tuberculosis* complex and *rpoB* (rifampicin) resistance according to the manufacturer's instructions [21].

For culture, specimens were decontaminated by Petroff's method (4% sodium hydroxide), neutralised, centrifuged to deposit the bacilli, and the sediment (about 200 μ L) inoculated onto two glycerol-enriched Löwenstein–Jensen (LJ) slopes per specimen. Slopes were incubated at 37°C and examined regularly for up to eight weeks; those showing no growth by week eight were recorded as negative, and growth was confirmed as acid-fast by ZN staining. Contaminated cultures were noted and, where a specimen remained available, reprocessed; otherwise they were excluded from phenotypic testing. First-line drug-susceptibility testing (DST) against isoniazid, rifampicin, streptomycin and ethambutol was performed on LJ medium by the proportion method at the standard critical concentrations recommended for that method, an isolate being classified resistant when the proportion of colonies growing on drug-containing medium exceeded 1% of that on the drug-free control.

All assays followed standard operating procedures with positive and negative controls and the routine quality-assurance practices of the national reference laboratory, and were performed by trained personnel; the use of three complementary tuberculosis modalities provided internal corroboration of case status. Of the 52 bacteriologically confirmed cases, 48 yielded culture growth with valid first-line DST; the remaining four were Xpert MTB/RIF-positive but culture-negative, showed no growth, or were contaminated without a repeat specimen, and were therefore excluded from

the phenotypic DST denominator (see Participant Flow Section).

Diabetes Ascertainment

After an overnight fast, venous blood was collected into a fluoride-oxalate tube (or, where necessary, a capillary finger-prick sample), and plasma glucose was measured by the glucose-oxidase–peroxidase colorimetric method; plasma glucose, rather than whole-blood glucose, was used for classification. A history of previously diagnosed diabetes and of diabetes symptoms and treatment was recorded through the structured, interviewer-administered questionnaire. Because of resource constraints, elevated values were not confirmed with a repeat fasting measurement or glycated haemoglobin (HbA1c); diabetes status therefore rested on a single measurement obtained during acute illness, so transient (stress) hyperglycaemia cannot be excluded — a limitation carried through to the Discussion.

Operational Definitions

Bacteriologically confirmed tuberculosis was defined as a positive result on Xpert MTB/RIF and/or LJ culture. Rifampicin resistance among confirmed cases was defined by a combined criterion — resistance detected by Xpert MTB/RIF (rpoB) and/or by phenotypic DST; where both were available, results were concordant in the great majority of isolates, and any discordant isolate was classified as resistant if either test indicated resistance, with both results reported. Multidrug-resistant tuberculosis (MDR-TB) was defined as phenotypic resistance to at least both rifampicin and isoniazid, with rpoB molecular results used to corroborate rifampicin status, consistent with WHO definitions [24]. Diabetes mellitus was defined as fasting plasma glucose ≥ 7.0 mmol/L and impaired fasting glucose as 6.1–6.9 mmol/L, in line with WHO criteria. Prior tuberculosis treatment was defined as a history of at least one month of anti-tuberculosis therapy before

enrolment (WHO “previously treated”), and low body-mass index as BMI < 18.5 kg/m² (underweight). Age was analysed dichotomously (≥ 45 versus < 45 years).

Data Analysis

Data were analysed using the Statistical Package for the Social Sciences (SPSS V29). Categorical variables were summarised as frequencies and percentages and continuous variables as means with standard deviations. Associations between tuberculosis status, diabetes status and drug resistance were examined using the chi-square test, with the Fisher exact test substituted where any expected cell count was below five, as was the case for the resistance-by-diabetes comparisons, given the small comorbid subgroup.

Multivariable logistic regression was used to identify independent predictors of the primary outcome, bacteriologically confirmed tuberculosis (analysed across all 200 participants); it was not used to model drug resistance, which was assessed descriptively because of the small number of resistant, comorbid cases. Candidate covariates namely sex, age (≥ 45 years), low BMI (< 18.5 kg/m²), prior tuberculosis treatment and diabetes, were specified a priori on the basis of clinical and biological plausibility and prior literature rather than by stepwise selection; continuous variables were dichotomised at the clinically meaningful thresholds stated above. Crude (unadjusted) odds ratios were obtained from the bivariate cross-tabulations, and adjusted odds ratios (aOR) with 95% confidence intervals from the multivariable model; reference categories are reported in Table 3. Questionnaires were checked for completeness at the point of administration and inconsistencies resolved before participants left the clinic, so the analysis was effectively complete-case across all 200 participants with negligible missing data. Formal goodness-of-fit testing (for example, the Hosmer–Lemeshow test) was not undertaken, a point noted among

the limitations. A p-value below 0.05 was considered statistically significant.

Ethical Considerations

Ethical approval was obtained before data collection from the institutional ethics committee overseeing the NTBLTC and its partner institutions. Written informed consent (or a witnessed thumbprint for those unable to write) was obtained from every participant after the purpose, procedures, risks and benefits had been explained in a language they understood; participation was voluntary, participants could withdraw at any time without affecting their care, and all data were anonymised using unique study identifiers and handled confidentially in accordance with the

Declaration of Helsinki. Participants found to have tuberculosis or diabetes were referred for management at the facility.

Results

Participant Characteristics

Two hundred presumptive-TB patients were enrolled and analysed. Males constituted 58.0% of the sample, and most participants fell within the economically active 26–45-year age band. The mean age was 41.6 ± 14.8 years, mean body-mass index 20.5 ± 3.4 kg/m², and mean fasting plasma glucose 6.1 ± 1.9 mmol/L. Selected characteristics are summarised in Table 1.

Table 1. Selected Characteristics of Study Participants (n = 200)

Variable	Category	Frequency (n)	Percentage (%)
Sex	Male	116	58.0
	Female	84	42.0
Age group (years)	18–25	24	12.0
	26–45	98	49.0
	≥46	78	39.0
Fasting glucose	Normoglycaemic	132	66.0
	Impaired fasting glucose	34	17.0
	Diabetes	34	17.0

Source: Field data, 2026.

Participant Flow

Of the 200 presumptive-TB adults enrolled and analysed, 52 (26.0%) had bacteriologically confirmed pulmonary tuberculosis (Xpert MTB/RIF and/or LJ culture positive): 40 were ZN smear-positive, 52 Xpert MTB/RIF-positive and 48 LJ culture-positive, while 148 were not bacteriologically confirmed. Valid

first-line phenotypic DST was obtained for the 48 culture-positive isolates; the four remaining confirmed cases were Xpert-positive but culture-negative, showed no growth, or were contaminated, and had no phenotypic DST. Among the 52 confirmed cases, 13 had diabetes and 39 did not. The flow of participants through screening, diagnosis and analysis is shown in Figure 1.

Participant flow through screening, diagnosis, and analysis

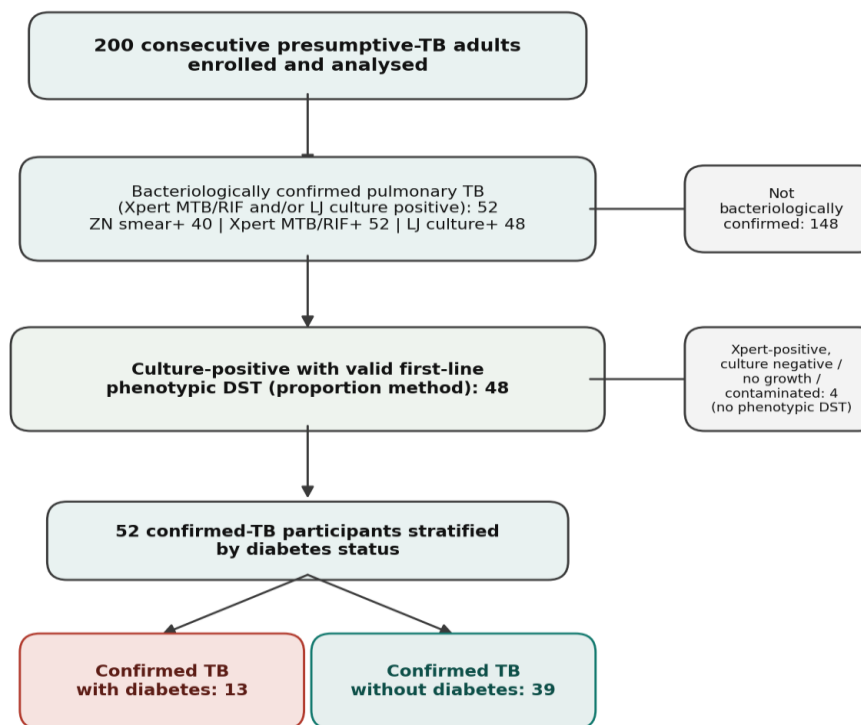


Figure 1. Participant Flow Through Screening, Diagnosis, and Analysis

Tuberculosis and Diabetes Prevalence

Bacteriologically confirmed pulmonary tuberculosis was identified in 52 of 200 patients (26.0%) and diabetes in 34 of 200 (17.0%). The Xpert MTB/RIF assay detected the largest

number of cases (52), followed by culture (48) and smear microscopy (40) (Figure 2), reaffirming the higher sensitivity of molecular and culture-based methods relative to smear microscopy [20, 22].

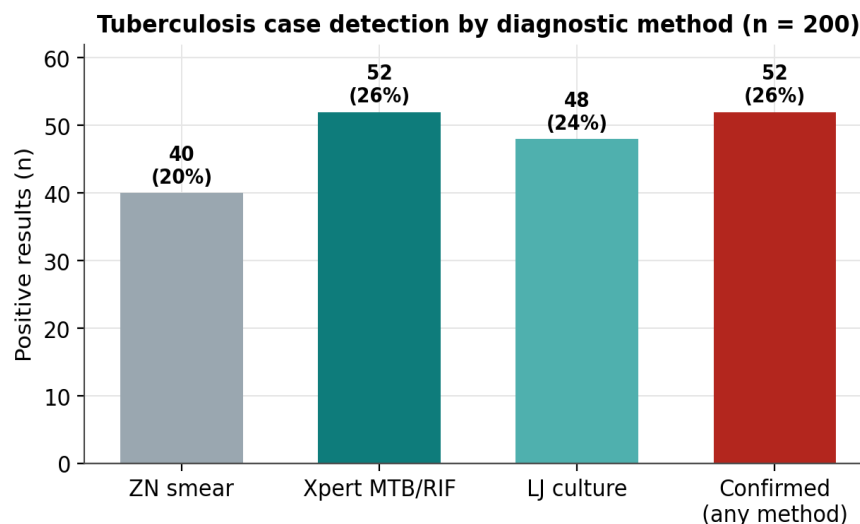


Figure 2. Tuberculosis Case Detection by Diagnostic Method (n = 200)

First-Line Drug-Resistance Patterns

Among the 48 culture-positive isolates with valid phenotypic DST, isoniazid resistance was the most frequent (11/48; 22.9%), followed by rifampicin (8/48; 16.7%), streptomycin (7/48; 14.6%) and ethambutol (5/48; 10.4%) (Figure 3). Combining phenotypic DST with Xpert MTB/RIF results across all 52 confirmed

cases, rifampicin resistance was 17.3% (9/52) and multidrug-resistant tuberculosis 11.5% (6/52); the single additional rifampicin-resistant case beyond the eight identified phenotypically was Xpert-positive without a valid culture. The predominance of isoniazid resistance is consistent with global surveillance of first-line resistance [23].

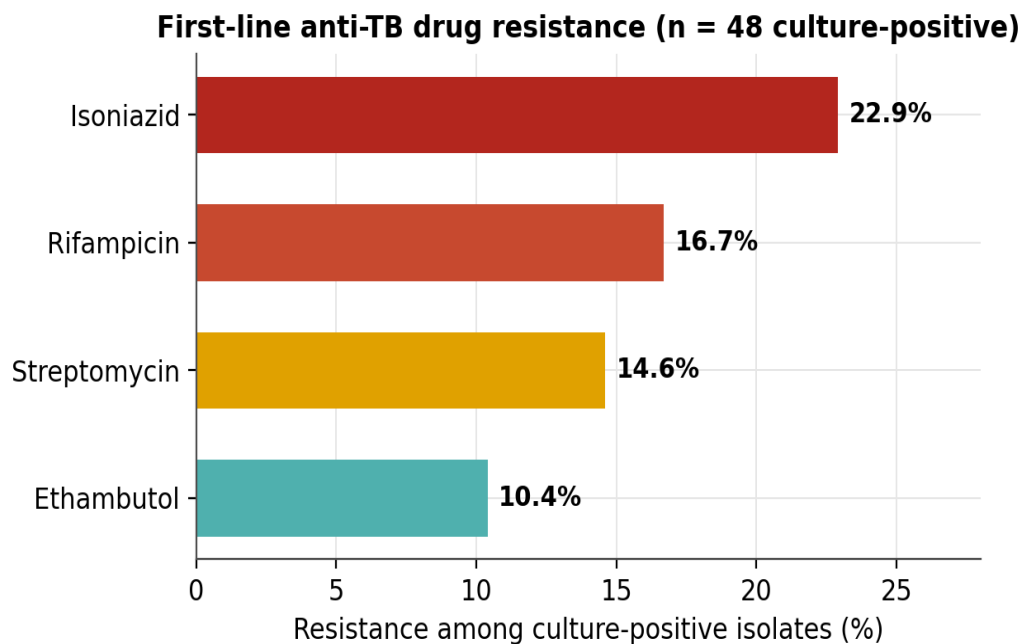


Figure 3. First-Line Anti-TB Drug Resistance Among Culture-Positive Isolates (n = 48)

Resistance by Diabetes Co-Morbidity

Among the 52 confirmed-TB patients, drug resistance was more frequent in the 13 with diabetes than in the 39 without. Any first-line resistance occurred in 46.2% (6/13) of comorbid patients versus 20.5% (8/39) of those without diabetes, and MDR-TB in 23.1% (3/13) versus 7.7% (3/39) (Table 2, Figure 4).

Although these differences were sizeable and directionally consistent with the hypothesised association [7, 8], the small number of comorbid cases limited statistical power, and neither difference reached conventional significance (Fisher exact $p = 0.086$ for any first-line resistance and $p = 0.157$ for MDR-TB). These estimates should therefore be regarded as hypothesis-generating

Table 2. Drug Resistance among Confirmed-TB Patients by Diabetes Status

Resistance Category	TB with Diabetes (n = 13)	TB without Diabetes (n = 39)
Any first-line resistance	6/13 (46.2%)	8/39 (20.5%)
Multidrug-resistant TB	3/13 (23.1%)	3/39 (7.7%)

Source: Field data, 2026. Fisher exact test: $p = 0.086$ (any first-line resistance); $p = 0.157$ (MDR-TB).

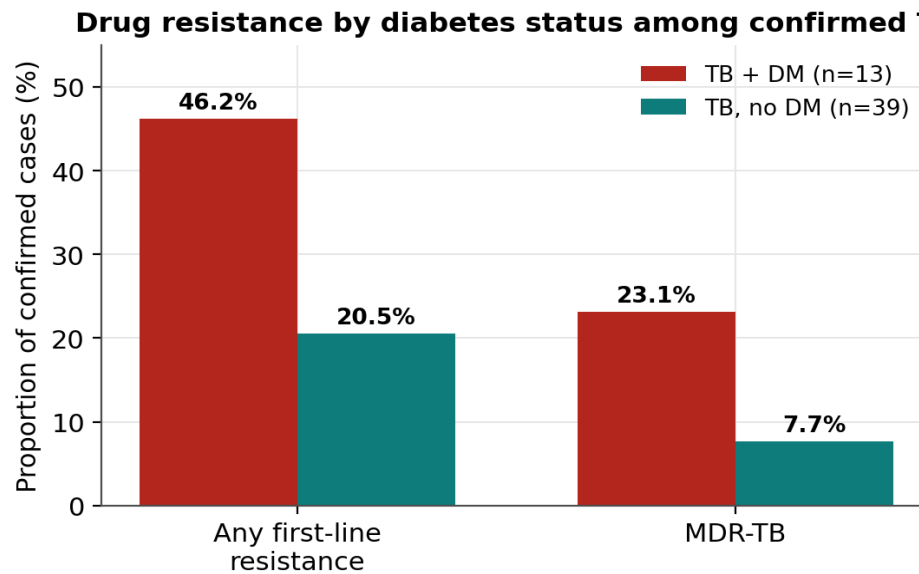


Figure 4. Drug Resistance by Diabetes Co-Morbidity Among Confirmed-TB Patients

Predictors of Confirmed Tuberculosis

In multivariable logistic regression, the outcome was bacteriologically confirmed tuberculosis (not drug resistance). Diabetes independently predicted confirmed tuberculosis (aOR 2.34, 95% CI 1.08–5.05, $p = 0.031$), as did prior tuberculosis treatment (aOR 3.10, 95% CI 1.36–7.06, $p = 0.007$) and low body-mass index (aOR 1.95, 95% CI 1.02–3.73, $p = 0.043$); male sex (aOR 1.42, 95% CI 0.71–2.84, $p = 0.320$) and age ≥ 45 years (aOR 1.85, 95% CI 0.92–3.71, $p = 0.084$) were not

independently associated (Table 3, Figure 5). Diabetes prevalence was significantly higher among confirmed-TB patients than among those without confirmed disease (26.9% versus 13.5%, $\chi^2 p = 0.03$). This regression addresses predictors of confirmed disease and should be distinguished from the study's resistance question, which, owing to the small numbers involved, was examined descriptively rather than modelled. The prominence of prior treatment as a predictor accords with established evidence on drivers of resistant disease [25, 27].

Table 3. Logistic-Regression Analysis of Factors Associated with Bacteriologically Confirmed Tuberculosis (n = 200)

Variable	Reference Category	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	p-value
Diabetes mellitus	No diabetes	2.36 (1.09–5.11)	2.34 (1.08–5.05)	0.031
Prior TB treatment	No prior treatment	2.94 (1.40–6.16)	3.10 (1.36–7.06)	0.007
Low BMI (< 18.5 kg/m ²)	BMI ≥ 18.5	2.05 (1.08–3.90)	1.95 (1.02–3.73)	0.043
Age ≥ 45 years	Age < 45	1.30 (0.69–2.44)	1.85 (0.92–3.71)	0.084
Male sex	Female	1.22 (0.64–2.33)	1.42 (0.71–2.84)	0.320

OR, odds ratio; CI, confidence interval. Adjusted odds ratios and p-values are from the multivariable logistic-regression model containing all listed covariates (n = 200). Crude odds ratios were derived from the bivariate cross-tabulations.

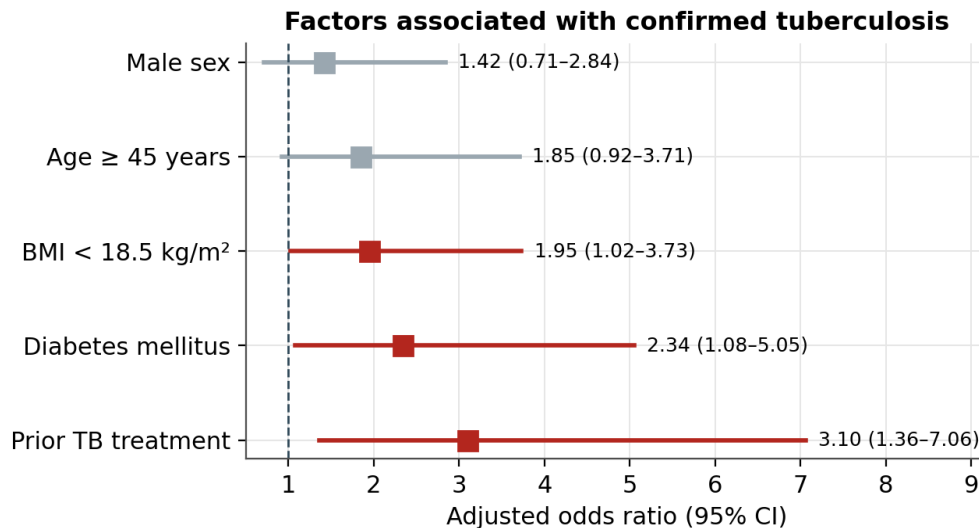


Figure 5. Adjusted Odds Ratios (95% CI) for Factors Associated with Confirmed Tuberculosis

Discussion

This study characterised first-line anti-tuberculosis drug resistance and examined its relationship with diabetes at a national reference centre in northern Nigeria. The overall rifampicin resistance of 17.3% and MDR-TB of 11.5% represent a clinically important burden. These figures are, however, considerably higher than population-level estimates for Nigeria and should be interpreted in the light of the referral setting rather than read as community prevalence [23, 25].

The elevated resistance almost certainly reflects referral bias. As a national reference centre, the NTBLTC preferentially receives patients who have failed or relapsed after first-line therapy, in whom drug resistance and prior treatment are concentrated; previously treated cases are the strongest and most consistently reported determinant of resistant disease [25, 27]. Diagnostic ascertainment is also more complete here than at peripheral facilities, so both the confirmed-TB proportion and the resistance estimates are expected to exceed those of unselected or community populations. The findings therefore describe the resistance profile arriving at referral level and are not generalisable to all presumptive-TB patients in the region.

Diabetes prevalence of 17.0% falls within the range reported for tuberculosis and presumptive-TB populations in sub-Saharan Africa [13–15] and reflects the rising diabetes burden in Nigeria [19]. The significant association between diabetes and confirmed tuberculosis, and the independent effect in multivariable analysis, are consistent with meta-analytic evidence that diabetes predisposes to active disease [3, 30] and with local Nigerian studies [16, 18].

The central question of this analysis — whether diabetes is associated with drug-resistant tuberculosis — yielded a consistent but statistically unconfirmed signal. Both any first-line resistance and MDR-TB were more than twice as frequent among comorbid patients, directionally consistent with meta-analyses linking diabetes to MDR-TB [7, 8] and with pharmacokinetic evidence of reduced first-line drug exposure in diabetic patients [9, 10]. However, this comparison rested on only 13 comorbid cases and six resistant events, neither difference was statistically significant (Fisher exact $p = 0.086$ and $p = 0.157$), and prior treatment remained the strongest predictor of confirmed disease [25, 27], echoing analyses that attribute resistance principally to programmatic and treatment-history factors rather than to diabetes per se [26]. The

estimates are therefore best viewed as hypothesis-generating, and no causal or policy inference about a diabetes–resistance link can be drawn from them. It should also be emphasised that the multivariable model evaluated predictors of confirmed tuberculosis, not of resistance, so it does not itself test the diabetes–resistance hypothesis.

Interpreted cautiously, these findings carry practical implications. Because much of the diabetes detected here would otherwise have gone unrecognised, the point of tuberculosis diagnosis is a reasonable opportunity for bidirectional screening, as recommended internationally [11, 12] and shown feasible in Nigeria [17]; this recommendation rests on the well-established diabetes–active-TB association rather than on the present, underpowered resistance data. Detecting and controlling diabetes in tuberculosis patients may improve treatment response and reduce relapse and death [5, 6, 28], while sustained investment in rapid molecular diagnostics and phenotypic DST is essential to detect resistance promptly and guide effective regimens [21, 22, 24]. Any conclusion that diabetes drives resistance, however, would be premature on these data and awaits confirmation in adequately powered studies, consistent with the goals of the WHO End TB Strategy and the USAID Global TB Strategy 2023–2030 [29].

Several limitations should be noted. The cross-sectional design precludes causal or temporal inference. The single-centre, referral-level setting limits generalisability and inflates the confirmed-TB and resistance estimates, as discussed above. Diabetes was classified from a single fasting plasma glucose measurement obtained during acute illness, without a repeat measurement or HbA1c confirmation, so stress hyperglycaemia may have led to some misclassification; this is a substantial limitation that should temper interpretation of all diabetes-related findings. Finally, the small number of comorbid cases limited the power to detect a diabetes–resistance association even if one

exists. Larger, multi-centre, longitudinal studies with confirmatory glycaemic testing are needed to establish this relationship [8, 27].

Conclusion

A substantial burden of first-line anti-tuberculosis drug resistance and multidrug-resistant tuberculosis was detected among confirmed cases at a national reference centre in northern Nigeria, with isoniazid resistance predominating; because of the referral setting, these figures describe patients arriving at reference level rather than the wider community. Diabetes mellitus was common and was significantly and independently associated with confirmed tuberculosis. A more than two-fold increase in drug resistance among comorbid patients was observed but did not reach statistical significance and was based on very small numbers, so it should be regarded as hypothesis-generating rather than as evidence of a causal link. The findings support routine drug-susceptibility testing, sustained investment in rapid diagnostics, and bidirectional tuberculosis–diabetes screening — the last justified by the established diabetes–active-TB association rather than by the present resistance data — while underscoring the need for larger, adequately powered studies before any firm conclusion is drawn about the relationship between diabetes and drug-resistant tuberculosis.

Ethics Approval and Consent to Participate

Ethical approval was obtained from the institutional ethics committee overseeing the NTBLTC and its partner institutions, and written informed consent was obtained from all participants. The study was conducted in accordance with the Declaration of Helsinki.

Consent for Publication

Not applicable; the manuscript contains no individually identifiable data.

Author Contributions

V.E.E. conceptualised and designed the study, conducted data collection and laboratory coordination, analysed the data and drafted the manuscript. F.O.N. supervised the study, provided methodological guidance and critically revised the manuscript. Both authors read and approved the final version.

Data Availability

The datasets generated and analysed during the current study are not publicly available in order to protect participant confidentiality, but are available from the corresponding author on reasonable request.

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Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors. It was conducted as part of the doctoral research requirements of the corresponding author.

Conflicts of Interest

The authors declare that they have no competing interests.

Acknowledgements

The authors thank the management and laboratory staff of the National Tuberculosis and Leprosy Training Centre, Saye, Zaria, and all participants who took part in the study.

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