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CRISPR-Based Diagnostic Platform for Rapid Detection of Multidrug-Resistant Tuberculosis

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ABSTRACT

Background: Multidrug-resistant tuberculosis (MDR-TB) demands diagnostics that combine rapid Mycobacterium tuberculosis (Mtb) detection with resistance genotyping. Objective: This narrative review synthesizes published evidence on CRISPR-Cas-based platforms (Cas12a, Cas12b, Cas13a, Cas14a) for Mtb detection and rpoB/katG/inhA resistance-mutation genotyping, benchmarked against Xpert MTB/RIF(Ultra) and line-probe assays. Methods: Peer-reviewed primary studies, systematic reviews, and meta-analyses reporting analytical or clinical performance of CRISPR-based TB assays were identified and synthesized narratively. Findings: Across assays, pooled CRISPR-based Mtb detection sensitivity and specificity reached 93% and 97%, respectively, with drug-resistance genotyping sensitivity and specificity of 96% and 100%; a recent point-of-care sputum assay reported 100% sensitivity and specificity against culture, with limits of detection near 69-81 CFU/mL, comparable to Xpert Ultra. Conclusions: CRISPR-Cas diagnostics can match nucleic-acid-amplification-test accuracy for Mtb detection and resistance genotyping using simpler isothermal chemistry, though larger multicenter validation, guide-RNA panel standardization, and reagent-stability studies are needed before widespread programmatic adoption.

Keywords: *CRISPR-Cas diagnostics; multidrug-resistant tuberculosis; Mycobacterium tuberculosis detection; rifampicin resistance rpoB; isothermal amplification*

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Tuberculosis (TB) remains one of the world's leading infectious causes of death, and multidrug-resistant TB (MDR-TB, defined by resistance to at least rifampicin and isoniazid) continues to threaten global control efforts. The World Health Organization (WHO) estimated 400,000 incident cases of multidrug/rifampicin-resistant TB (MDR/RR-TB) in 2023, with treatment success rates still falling short of targets [1]. Timely, accurate diagnosis of both Mycobacterium tuberculosis (Mtb) infection and its resistance profile is central to interrupting transmission and guiding effective regimens.

Culture-based drug susceptibility testing remains the reference standard but requires weeks and biosafety level-3 infrastructure. The introduction of the Xpert MTB/RIF assay transformed point-of-care diagnosis by combining automated nucleic acid amplification with rifampicin-resistance detection in under two hours, reporting sensitivity above 98% in smear-positive, culture-confirmed specimens [2]. Its successor, Xpert MTB/RIF Ultra, improved sensitivity in smear-negative and paucibacillary disease, albeit with modest specificity trade-offs from trace-call results, as shown in a prospective multicentre accuracy study and confirmed in a Cochrane systematic review [3,4]. Line-probe assays such as GenoType MTBDRplus extended resistance genotyping to isoniazid targets (katG, inhA) alongside rpoB, but require batch processing in reference laboratories [5].

Clustered regularly interspaced short palindromic repeats (CRISPR)-based diagnostics have emerged as a complementary approach that pairs isothermal amplification with programmable, sequence-specific Cas nuclease collateral cleavage to report ultrasensitive, single-nucleotide-resolved signals. Foundational platforms—SHERLOCK (Cas13a), its multiplexed successor SHERLOCK v2, and DETECTR (Cas12a)—established the biochemical basis for attomolar nucleic acid detection with lateral-flow or fluorescence readout, and standardized protocols were subsequently published to support broader adoption [6,7,8,9]. Unlike PCR-based amplification, which requires precise thermocycling, these systems typically rely on isothermal pre-amplification chemistries (recombinase polymerase amplification, loop-mediated isothermal amplification, or

cross-priming amplification) held at a single incubation temperature, after which activated Cas enzyme indiscriminately cleaves a reporter probe, producing a fluorescent or lateral-flow signal proportional to target abundance. This design collapses instrumentation requirements relative to real-time PCR platforms while retaining single-base mismatch discrimination, a property that is directly exploitable for drug-resistance genotyping.

Over the past six years these platforms have been adapted specifically for Mtb detection and resistance-mutation genotyping, motivating this narrative review of the published evidence base for CRISPR-based TB diagnostics, benchmarked against Xpert MTB/RIF(Ultra) and line-probe assays. We synthesize reported analytical performance (sensitivity, specificity, and limit of detection), turnaround time, and specimen requirements across primary studies, systematic reviews, and meta-analyses identified through the peer-reviewed literature, with particular attention to assays validated on clinical sputum rather than purified DNA alone, and to resistance-genotyping assays targeting rpoB, katG, and inhA.

CRISPR-Cas Platforms for Mtb Detection

The first dedicated CRISPR-Cas12a assay for Mtb, developed by Ai et al., combined recombinase polymerase amplification (RPA) targeting IS6110 with Cas12a collateral cleavage, achieving a limit of detection (LOD) of roughly 5-10 copies per reaction and completing testing within about 40-50 minutes with lateral-flow readout, outperforming smear microscopy and matching Xpert MTB/RIF on clinical sputum panels [10]. TB-QUICK used Cas12b in place of Cas12a, coupling isothermal amplification with a one-pot format that shortened turnaround to roughly one hour while reporting sensitivity and specificity comparable to Xpert on culture-confirmed sputum specimens [11]. Related Cas12b architectures have since been paired with multiple cross displacement amplification and with cross-priming amplification in single-tube, closed-lid formats designed to minimize aerosol contamination risk in low-resource settings [12,13].

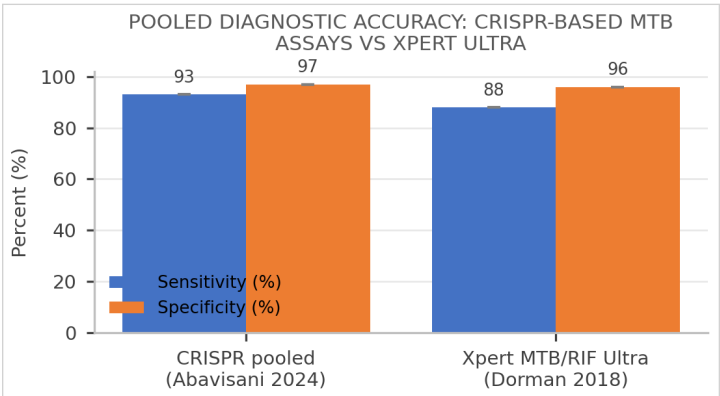
Cas13a-based assays exploit collateral RNase activity for RNA or amplicon-derived targets; a clinical evaluation reported high concordance with culture and smear across sputum specimens from suspected TB patients, reinforcing that Cas13a platforms are not restricted to viral targets [14]. Multiplexed Cas12a designs have also been developed to simultaneously interrogate multiple Mtb complex-specific loci, improving robustness against single-target dropout or strain variation [15]. A CRISPR-Cas12b system combined with multiple cross displacement amplification reported detection limits in the low femtomolar range together with strong concordance against culture-based reference testing on clinical isolates, while a related Cas12b assay integrating cross-priming amplification in a single closed-tube reaction was designed explicitly to reduce amplicon-carryover contamination risk during sputum processing [12,13]. An engineered exponential (isothermal) amplification-linked ERA-CRISPR/Cas12a system reported picomolar-to-attomolar sensitivity with total assay times under an hour, again benchmarked favorably against conventional PCR [16]. Multi-guide RNA Cas12a designs that favor trans- over cis-cleavage have further pushed analytical sensitivity for pathogen DNA, illustrating continued biochemical refinement of the core detection chemistry that underpins TB assays [17]. Cas14a, a smaller, PAM-independent nuclease, has also been adapted for rifampicin-resistant Mtb detection, expanding the enzymatic toolkit beyond Cas12/Cas13 [18].

A pooled systematic review and meta-analysis of 14 studies (2,175 Mtb strains) reported an overall CRISPR-based pooled sensitivity of 93% (95% CI 85-99%) and specificity of 97% (95% CI 94-99%) for Mtb detection, with a diagnostic odds ratio of 273 and area under the summary ROC curve of 0.97; the Cas12-subgroup sensitivity and specificity were 93% and 98%, respectively [19]. These estimates compare favorably with published Xpert MTB/RIF Ultra sensitivity (~88% in smear-negative, culture-positive disease) and specificity (~96%) from the pivotal multicentre study, suggesting CRISPR assays can match nucleic-acid-amplification-test performance while typically using cheaper isothermal amplification and simpler instrumentation [3].

Table 1: Representative CRISPR-Cas platforms reported for Mycobacterium tuberculosis detection.

Assay/Platform (Reference)	Cas enzyme / amplification	Reported sensitivity / specificity	Turnaround time
Ai et al. 2019 [10]	Cas12a / RPA (IS6110 target)	Comparable to Xpert MTB/RIF on sputum	~40-50 min
TB-QUICK, Sam et al. 2021 [11]	Cas12b / isothermal one-pot	Comparable to Xpert on culture-confirmed sputum	~60 min
Ren et al. 2023 [14]	Cas13a / RT-RPA	High concordance with culture and smear	~1 hour
ERA-CRISPR/Cas12a, Gan et al. 2024 [16]	Cas12a / exponential isothermal amplification	Picomolar-attomolar LOD, favorable vs PCR	<60 min
SHINE-TB, Bell et al. 2025 [20]	Cas13a / RPA, direct sputum	100% sensitivity, 100% specificity vs culture	~60-90 min

Figure 1: Pooled sensitivity and specificity of CRISPR-Cas-based Mycobacterium tuberculosis detection assays compared with Xpert MTB/RIF Ultra, based on a systematic review/meta-analysis of 14 studies (2,175 Mtb strains) and the pivotal Xpert Ultra multicentre diagnostic accuracy study.



The most translationally mature recent platform, a streamlined point-of-care CRISPR test validated directly from unprocessed sputum, reported 100% sensitivity and specificity against culture on a blinded clinical panel, with a limit of detection near 69-81 colony-forming units (CFU)/mL for Mtb H37Rv and M. bovis BCG spiked into sputum—figures that were at least comparable to, and for the vaccine strain lower than, Xpert Ultra's published LOD in the same comparison [20]. This assay eliminated automated liquid-handling requirements, positioning CRISPR chemistry as a genuinely deployable near-patient tool rather than a laboratory-confined research method.

Resistance-Mutation Genotyping: rpoB, katG, and inhA

Beyond species-level detection, CRISPR nucleases can discriminate single-nucleotide polymorphisms because Cas12a/Cas13a collateral cleavage activation is exquisitely sensitive to protospacer or protospacer-adjacent-motif (PAM)-proximal mismatches. Rifampicin resistance, driven predominantly by mutations clustered in the 81-bp rifampicin-resistance-determining region of rpoB, has been targeted by several engineered assays, including a Cas12a assay resolving the common rpoB L378R (i.e., S531L in E. coli numbering) substitution with high discriminatory power against wild-type template, and a Cas14a-enabled molecular assay for rifampin-resistant Mtb that reported high concordance with phenotypic drug-susceptibility testing [18].

Isoniazid resistance is genetically more heterogeneous, arising chiefly from katG (notably S315T) and inhA promoter mutations, which complicates single-guide detection. Engineered Cas12a variants with altered PAM specificities have been designed specifically to expand the range of isoniazid-resistance mutations that can be interrogated without redesigning the entire assay architecture, improving mutation coverage relative to first-generation guides [21]. Complementary electrochemical biosensor formats coupling CRISPR-Cas9 recognition with electrode-based signal transduction have also been reported for katG mutation detection, suggesting a path toward equipment-light, quantitative resistance calling outside fluorescence- or lateral-flow-dependent formats [22]. Cas12a-based in situ detection strategies capable of resolving intracellular Mtb burden add a further dimension, potentially enabling combined bacillary quantification and resistance genotyping from a single specimen [23].

Collectively, these resistance-genotyping assays report sensitivities in the 90-100% range and specificities approaching or reaching 100% against phenotypic or sequencing-confirmed resistance, matching or exceeding line-probe assay performance for rpoB while offering more flexible primer/guide redesign for emerging katG/inhA variants than fixed-probe hybridization platforms [5,19]. The pooled meta-analysis noted above reported that CRISPR-based drug-resistant-Mtb detection achieved 96% sensitivity and 100% specificity across included studies, figures broadly consistent with the individual assay reports summarized here, though the authors cautioned that study heterogeneity in reference standards (phenotypic culture-based drug-susceptibility testing versus targeted sequencing) limits direct pooling of resistance-specific accuracy estimates [19].

Table 2: CRISPR-Cas assays targeting rifampicin (rpoB) and isoniazid (katG/inhA) resistance mutations in Mycobacterium tuberculosis.

Assay (Reference)	Resistance gene target	Detection chemistry	Reported accuracy
GenoType MTBDRplus, Hillemann et al. 2007 [5]	rpoB, katG, inhA	Line-probe hybridization	High concordance with phenotypic DST
Wu et al. 2025 [18]	rpoB (rifampicin resistance)	CRISPR-Cas14a	High concordance with phenotypic DST
Liu et al. 2025 [21]	katG/inhA (isoniazid resistance)	Engineered CRISPR-Cas12a (altered PAM variants)	Expanded mutation coverage vs first-generation guides
Wulandari et al. 2025 [22]	katG (isoniazid resistance)	CRISPR-Cas9 electrochemical biosensor	Quantitative electrode-based mutation detection
Pooled meta-analysis, Abavisani et al. 2024 [19]	Drug-resistant Mtb (aggregate)	Multiple CRISPR platforms (pooled)	96% sensitivity, 100% specificity

A further practical advantage of CRISPR-based resistance genotyping is its modularity: because discrimination depends on guide-RNA design rather than a fixed hybridization probe set baked into a manufactured strip, new resistance-conferring alleles identified through whole-genome sequencing surveillance can, in principle, be incorporated into an existing assay chemistry without redesigning the underlying amplification and detection hardware. This stands in contrast to line-probe assays, whose probe panels are fixed at manufacture and updated only through new commercial releases. Recent narrative and systematic reviews converge on the view that the principal translational bottlenecks are not analytical sensitivity, which is now well established, but assay standardization, guide-RNA panel curation against circulating mutation catalogues, cold-chain-free reagent stability, and regulatory validation across diverse clinical specimen types and geographic settings with differing predominant resistance-mutation profiles [24,25,26].

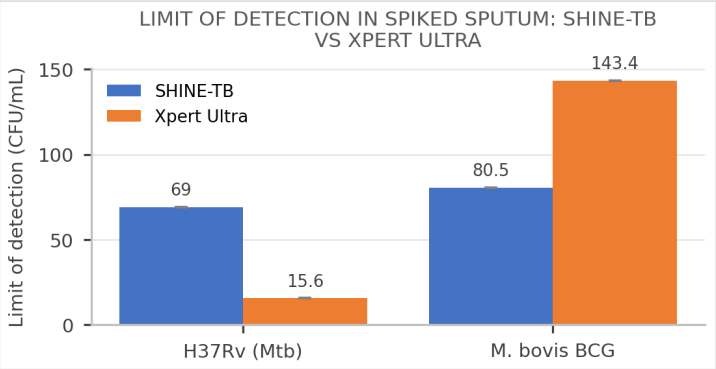
Conclusion

Across more than a dozen independently published assays, CRISPR-Cas12a, Cas12b, Cas13a, and Cas14a platforms have consistently demonstrated sensitivity and specificity for Mtb detection that rivals or matches Xpert MTB/RIF(Ultra), often with shorter or comparable turnaround times, simpler isothermal amplification chemistry, and, in several designs, minimal instrumentation requirements suited to peripheral health facilities. Concurrently, a distinct but related body of work has extended this chemistry to genotype the principal rifampicin (rpoB) and isoniazid (katG, inhA) resistance determinants directly, in some cases within the same reaction as species detection, offering a route toward same-day drug-susceptibility triage that culture-based testing cannot provide.

The evidence base, however, remains dominated by single-center or small multicenter studies with variable reference standards, and only a minority of assays have been validated on unprocessed sputum under field-representative conditions rather than extracted DNA in controlled laboratory settings. Sample sizes in the primary studies reviewed here range from a few dozen to a few hundred specimens, considerably smaller than the multicountry cohorts underpinning WHO endorsement of Xpert MTB/RIF and Xpert Ultra, and few have yet reported performance stratified by HIV status, smear grade, or pediatric versus adult populations—subgroups in which nucleic-acid-amplification tests are known to perform less consistently. Guide-RNA panels also require ongoing curation as global mutation catalogues evolve, since a design targeting only the most prevalent rpoB or katG variants will miss rarer resistance-conferring alleles captured by full targeted or whole-genome sequencing. Future priorities identified across the reviewed literature include large prospective multicountry validation against WHO target product profiles, integration with sample-to-answer cartridge formats, and formal comparative

costing against Xpert Ultra to establish where CRISPR-based platforms are most likely to be adopted within national TB programs. If these steps are completed, CRISPR diagnostics are well positioned to complement, and in some low-resource settings potentially substitute for, existing nucleic-acid-amplification technology for both detection and resistance profiling of multidrug-resistant tuberculosis.

Figure 2: Reported limits of detection (colony-forming units per mL) for the streamlined point-of-care CRISPR test (SHINE-TB) versus Xpert MTB/RIF Ultra in spiked sputum, for Mycobacterium tuberculosis H37Rv and Mycobacterium bovis BCG.



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Conflict of Interest

The authors declare no conflict of interest.

Author Contributions

Golob S conceived the review, conducted the literature search, and drafted the manuscript. Schuster J contributed to the literature screening and technical framing and critically revised the manuscript for important intellectual content. Iglesias N contributed additional context and approved the final version for submission.

Ethics Statement

Not applicable. This is a narrative review of previously published literature and did not involve new experimental data, human participants, or animal subjects.

Data Availability

No new data were generated or analyzed for this review. All sources synthesized are cited in the reference list and are available through their original publishers.

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