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Molecular Mechanism of Multi-Drug Resistance in *Mycobacterium tuberculosis* †

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Abstract: In this study, we demonstrate the clinical applicability of molecular assays for the differential identification of *M. tuberculosis* isolates by Double Repetitive Element-PCR (DRE-PCR), Duplex PCR (DPCR), Random Amplified Polymorphic DNA-PCR (RAPD-PCR) and IS*6110* flanking PCR and for the detection of specific codon mutations in antibiotic-resistant genes, *rpoB* and *katG* in 55 MDR-TB and 25 drug-susceptible clinical isolates by Multiplex PCR assays. The MAS-PCR assay was identified as the most prevalent *rpoB* gene mutations at codon 531 (83.6%), followed by codon 526 (12.7%), and no mutation was found in codon 516. Among the 55 MDR-TB isolates, 49 (89%) isolates had S315T, 5 (9%) had S315N mutations. DRE-PCR and RAPD-PCR generated similar banding of cluster III strains and suggested that MDR-TB strain genotype C may be responsible for the transmission of TB infection among the study population. PCR based differential identification of *mtp*40 and *rpoB* DPCR procedures identified two NTM strains among the isolates studied. Genotypic method DRE-PCR was found highly reproducible, followed by RAPD-PCR and *mtp*40, and *rpoB* DPCR methods effectively-identified NTM infection in this region. The presence of S315T mutation in *katG* gene and S531L, H526Y mutations in *rpoB* gene in MDR-TB isolates proved resistant phenotype. The simplicity of the MAS-PCR assay permits its implementation for the detection of resistance to INH and RIF in clinical laboratories in regions where this mutation is predominant among MDR-TB strains.

Keywords: *Mycobacterium tuberculosis*; Multidrug resistance; *katG* gene; *rpoB* gene; MAS-PCR; PCR-RFLP; RAPD-PCR; DRE-PCR.

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Conflicts of Interest

The authors declare no conflict of interest.