PCR-RFLP Analysis of a Point Mutation in Codons 315 and 463 of the katG Gene of Mycobacterium tuberculosis Isolated from Patients in Silesia, Poland

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Abstract

Resistance to antituberculous agents is an important cause of ineffectiveness of antimicrobial therapy. The resistance of M. tuberculosis to antituberculous agents is a result of mutations in genes participating in those agent’s action. The antituberculous drug - isoniazid can be activated by Mycobacterium tuberculosis either through a hydroperoxidase I/II or a superoxide-dependent oxyferrous pathway. The present study analyzed the frequency of the mutations occurring in codons 315 and 463 in katG gene of Mycobacterium tuberculosis strains, isolated from patients with pulmonary tuberculosis from Silesia, Poland. In this study 23 isoniazid-resistant Mycobacterium tuberculosis strains were analyzed. For RFLP analysis, a 620 bp amplified fragment of katG gene was digested with restriction endonuclease MspI. Among 24 isoniazid-resistant strains, isolated from patients between 2000-2001, point mutations were found in 30% of analyzed isoniazid-resistant strains in codons 315 or 463 (7 strains). In contrast, no mutations in codons 315 and/or 463 katG gene were found in 16 strains (70%). Obtained results suggests that point mutations S315T (AGC—>ACC) and R463L in katG gene are infrequent in the analyzed population.

Key words: M. tuberculosis resistance, isoniazid activation, katG mutations

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