KNOWN MECHANISMS OF M. TUBERCULOSIS DRUG RESISTANCE TO MAJOR FIRST AND SECOND LINE ANTIMYCOBACTERIAL DRUGS

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Abstract

The continued rise in the prevalence of multidrug-resistant and extensively drug-resistant tuberculosis in the era of human immunodeficiency virus infection poses a serious threat to effective tuberculosis control. Drug resistance in M. tuberculosis occurs due to low-frequency spontaneous chromosomal mutations. The clinical form of drug-resistant tuberculosis occurs mainly as a result of human selection during the treatment period of these genetic rearrangements due to indiscriminate drug provision, suboptimal treatment regimens prescribed by physicians, and poor adherence to treatment by patients.

In the review article, the molecular genetic mechanisms of the development of drug resistance were thoroughly elucidated in relation to the main antimycobacterial drugs of the 1st and 2nd line, namely: isoniazid, rifampicin, pyrazinamide, ethambutol, streptomycin, amikacin, kanamycin, capreomycin and ethionamide/prothionamide. Knowledge of the relationship between drug-resistant strains of M. tuberculosis and their virulence/transmissibility, understanding of the mechanisms of drug resistance formation in M. tuberculosis will allow developing new, even more advanced accelerated methods for molecular genetic diagnosis of tuberculosis and better understanding the specifics of creating new drugs for the treatment of tuberculosis.

Key words: tuberculosis, drug resistance, mechanisms.

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