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Gene Technology

Commentary

Effect of Anti-Tuberculosis Drugs on *Mycobacterium Tuberculosis Rv1250* mRNA Efflux Pump Gene Expression

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DESCRIPTION

Efflux pumps are Tran's protein molecules that actively participate inside the efflux of a variety of substrates outside the bacteria, including medicines. We needed to look at RNA expression of the *Rv1250* efflux pump genes in Mycobacterium isolated from tuberculosis patients who had received drug treatment at the 1st, 3rd, and 5th months, as well as newly diagnosed tuberculosis patients who would receive drug treatment. This work used a multi cross-sectional longitudinal research design, in which 50 distinct M. *tuberculosis* isolates and a reference strain *H37Rv* was sub-cultured on LJ medium, recognized by PCR Analysis, and collected for RNA extraction. To assess mRNA quantification gene expression, total bacterial mRNA was analyzed using real-time quantitative PCR.

One of the most important milestones in the control of infectious diseases in the 20th century was the advancement in antibiotic use. Most conventional antibiotics, however, have become ineffective due to numerous types and methods of antibiotic resistance diffusion in pathogenic organisms. This rise of tuberculosis infection is causing serious public health issues and is a leading cause of death worldwide. Streptomycin is the most used short-term anti-TB medication. Isoniazid, rifampicin, and ethambutol are a powerful medication combination for infections caused by susceptible M. *tuberculosis* strains. All first- and second-line treatments are becoming resistant to M. *tuberculosis* strains, which have previously been vulnerable. *Mycobacterium tuberculosis* has the ability to change cell metabolic pathways, including anaerobic and aerobic cell respiration, into other routes.

One of M. *tuberculosis* capacities to adjust and remain inside the human body, where environment may shift from aerobic to micro aerophilic/anaerobic, is its flexibility. The presence of a permeability membrane in the cell wall against toxic substances may contribute to M. *tuberculosis* inherent resistance to various antimicrobial drugs. Mycolic acid inhibits the passage of hydrophilic and hydrophobic hydrophobic molecules into the cell, while the peptidoglycan and arabinogalactan layers hinder the transport of hydrophobic molecules into the cell. Transport of hydrophobic antibiotics like rifampicin and fluoroquinolone into the bilayer hydrophobic layer allows them to enter bacterial cells. Porin, on the other hand, allows hydrophilic antibiotics and nutrients to enter the cell that can't pass through the cell membrane. Porin is a non-specific protein channel in bacteria's outer membrane that allows hydrophilic solutions to pass through. Mutations of antibiotic target genes are recognized to be the primary source of drug resistance due to the unique structure of bacterial cells. In clinical cases, not all drug target gene alterations in various first-line drug resistances can be explained.

The efflux pumps system can produce resistance by extruding antibiotic molecules into the cell, where the intracellular concentration of antibiotics provided is determined by the cell's influx and efflux balance. To avoid antibiotic resistance, it's critical to understand the mechanisms involved influx through porin and drug outflow through efflux pumps. Genes codes for efflux pumps are involved in various physiological processes in eubacteria cells, including cell division, pH homeostasis, and intracellular metabolite secretion. The efflux pump system can be separated into two components based on bioenergetics criteria: Primary and secondary carriers. The energy sources for primary transporters include ATP hydrolysis and ATP production. The proton motive force provides energy to secondary transporters in the form of electrochemical gradients that are bound to protons being transported and distributed on the surface of bacterial cells. Transporters are ATP binding sequence, Major Facilitator Gene family, Multi-Antimicrobial Extrusion Protein, Small Multidrug Resistance, and Resistance Nodulation

New medications, pharmacological targets, and factors that drive drug resistance are all needed to treat tuberculosis infection effectively. The *H37Rv* TB genome encodes a number of uncharacterized potential efflux proteins. According to a research, mycobacteria use the active efflux system to expel a variety of medicines. Some efflux pumps are antibiotic-specific, whereas others, like multidrug-resistance efflux pumps, expel components that are physically and functionally distinct. On a laboratory scale, an experimental approach for detecting efflux

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pump expression in mutant bacteria by drug induction is limited. Clinical isolates were used in some of the studies on efflux pump overexpression. Experimental design has been used in the majority of efflux pump studies. The purpose of this study is to determine the degree of mRNA expression of the efflux pump *Rv1250* gene in an *M. tuberculosis* resistant strain isolated from patients with multi drug resistance. The level of *Rv1250* mRNA gene expression in clinical isolates acquired independently from MDR-TB patients who received antibiotic therapy during the first month, third month, and fifth month, and also newly diagnosed TB patients who would get drug therapy, was compared. The researchers also wanted to evaluate the expression of the *Rv1250* gene in resistant and susceptible groups of clinical isolates acquired from TB patients with positive lung X-ray results.