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Effect of a Muscarinic Antagonist Drug on Extracellular Lipase Activityof Pseudomonas aeruginosa

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Abstract : Pseudomonas aeruginosa is a Gram-negative, rode shape and aerobic bacterium that has shown to be resistance to many antibiotics. This resistance makes the bacterium very harmful in some diseases. It can also generate diseases in any part of the gastrointestinal tract from oropharynx to rectum. P. aeruginosa has become an important cause of infection, especially in patients with compromised host defense mechanisms. One of the most important reasons that make P. aeruginosa an emerging opportunistic pathogen in patients is its ability to use various compounds as carbon sources. Lipase is an enzyme that catalyzes the hydrolysis of lipids. Most lipases act at a specific position on the glycerol backbone of lipid substrate. Some lipases are expressed and secreted by pathogenic organisms during the infection. Muscarinic antagonist used as an antispasmodic and in urinary incontinence. The drug has little effect on glandular secretion or the cardiovascular system. It does have some local anesthetic properties and is used in gastrointestinal, biliary, and urinary tract spasms. Aim: In this study the inhibitory effect of a muscarinic antagonist on lipase of P. aeruginosa was investigated. Methods: P. aeruginosa was cultured in minimal salt medium with 1% olive oil as carbon source. The cells were harvested and the supernatant, which contained lipase, was used for enzyme assay. Results: Our results showed that the drug can inhibit P. aeruginosa lipase by competitive manner. In the presence of different concentrations of the drug, the Vmax (2 mmol/min/mg protein) of enzyme did not change, while the Km raised by increasing the drug concentration. The Ki (inhibition constant) and IC50 (the half maximal inhibitory concentration) value of drug was estimated to be about 30 uM and 60 uM which determined that the drug binds to enzyme with high affinity. Maximum activity of the enzyme was observed at pH 8 in the absence and presence of muscarinic antagonist, respectively. The maximum activity of lipase was observed at 600C and the enzyme became inactive at 900C. Conclusion: The muscarinic antagonist drug could inhibit lipase of P. aeruginosa and changed the kinetic parameters of the enzyme. The drug binded to enzyme with high affinity and did not chang the optimum pH of the enzyme. Temperature did not affect the binding of drug to musmuscarinic antagonist.

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