Abstract

Mycobacterium tuberculosis, the causative agent of human tuberculosis, is responsible for almost three million deaths annually worldwide, being at the same time one of the prevalent pathogens affecting AIDS patients. The therapeutic treatment of tuberculosis requires the use of several antimycobacterial drugs for a period of six months, leading to a high level of non-compliance. This situation favors the appearance of clinical strains resistant to one or more drugs. Studies performed over the last few years have identified the molecular targets for the currently used anti-mycobacterial drugs and the most frequent mechanisms of resistance. The information generated have pointed out a very interesting fact: the majority of the specific anti-mycobacterial drugs such as Isoniazid, Ethionamide or Pyrazinamide- affect the synthesis of fatty acids (including mycolic acids, long-chain fatty acids that are a hallmark of mycobacteria), or the synthesis of components of the cell wall such as arabinogalactan, inhibited by Ethambutol. These results have generated great interest in the study of the biosynthetic pathways of those cell components with the goal of identifying new targets suitable for the design of novel drugs, which brings renewed hope to achieve the goal of obtaining better drugs to treat this dreadful disease

Keywords

tuberculosis, drug resistance, mechanism of action, antibiotics, fatty acids