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MOLEKULARNE MECHANIZMY ODPORNOŚCI NA GRUŹLICĘ

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Molecular mechanisms of resistance to tuberculosis

Abstract: Tuberculosis is a major infectious disease with up to a third of the world's population infected, 8-10 million people developing the active disease and about 3 million dying of tuberculosis each year. One key to the pathogenic potential of tubercule bacilli lies in their capacity to resist destruction by macrophages. The development of genomics, proteomics and transcriptomics have shed light on the exploitation of macrophages by mycobacteria. The initial interaction between mycobacterial surface components and numerous macrophage receptors is critical in determining the fate of the bacteria in a phagocyte. The inhibition of fagosom-lysosome fusion by pathogenic mycobacteria promote their intracellular persistence and growth. Dendritic cells which exhibit the unique ability to activate naive T lymphocytes are critical for triggering innate and acquired anti-mycobacterial immunity. A large body of data show the regulatory role of numerous cytokines and chemokines in the outcome of mycobacterial infection. The recognizing of the cytokines which up-regulate and down-regulate protective immunity to tuberule bacilli could lead the way to innovative therapeutic approaches. The effectiveness of the currently used tuberculosis vaccine, Bacillus Calmette-Guerin (BCG) has been highly variable. Recombinant BCG bacilli with an over production of some mycobacterial antigens or cytokines have been shown to be more effective in generating anti-mycobacterial immunity in mice and guinea pigs than the parental BCG bacilli.

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