Mathematical and computational modeling of host-pathogen interaction in the lung lesion development due to Mycobacterium tuberculosis in humans

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Abstract

Tuberculosis (TB) has a long history as a major disease in humans and animals. New resistant strains are jeopardizing the advances in the battle against TB. 40% of HIV deaths occurs dues to concurrent TB illness. A causative agent of TB Mycobacterium tuberculosis — causes severe implications for a patient. The tuberculosis process is accompanied usually by the lung tissue destruction and cavitation associated with imbalance in the system of matrix metalloproteinases/inhibitors (MMP/TIMP). At the same time, pathogen can stimulate an inflammatory process leading to an even greater imbalance of MMP/TIMP facilitating the tissue destruction. Such positive feedback also could be conditioned by the initially damaged immune status of the host. We have developed model based on the study of “manipulation” of immune signalling and biochemical metabolic pathways by M. tuberculosis that reveal key factors contributing to the beginning of reparative changes or the transition of the disease to a chronic form. We have defined, deploying the mathematical model and based on published clinical observations, ”key parameters” stimulating the restoration of the structure and functions of tissues, as well as analyzed the dynamics of cellular populations for the diagnosis of functional and pathological conditions of the host.