Preventing tuberculosis reactivation

October 18 2007

Tuberculosis (TB) is the leading cause of death due to infectious disease in the world today. It is estimated that 2 billion people are currently infected, and although most people have latent infection, reactivation can occur. This paper by Denise Kirschner and colleagues, publishing in PLoS Computational Biology, conducts virtual clinical trials to examine the causes of reactivation.

Tumor necrosis factor alpha (TNF) is a protein that facilitates cell–cell communication during an inflammatory immune response. Animal models have shown that TNF is vital for control of TB infection. However, anti-TNF treatments are common therapies for treating autoimmune diseases, and this can cause an unwanted side effect of reactivating latent TB. Kirschner has developed a computational model that can quickly perform virtual clinical trials to predict why reactivation occurs and why it happens differently with different drugs.

Their results suggest that anti-TNF therapy is highly likely to lead to many incidents of TB if used in areas where exposure to the TB pathogen is probable. However, they also propose that a TNF-modulating agent could be developed that could balance the requirement for reduction of inflammation with the necessity to maintain resistance to infection and microbial disease. In the mean time, modifying the dosage and timing of anti-TNF treatment could prevent reactivation, as could a complete regimen of antibiotic treatment for TB prior to anti-TNF treatment.

Source: Public Library of Science