A genetic variation may increase tuberculosis susceptibility

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Researchers have shown that a single nucleotide change in a gene that affects production of hepcidin—a peptide involved in inflammation, immunity, and control of iron levels—is associated with greater susceptibility to extrapulmonary tuberculosis. Individuals with this single nucleotide polymorphism (SNP) make significantly less hepcidin in response to infection by Mycobacterium tuberculosis, as reported in *Genetic Testing and Molecular Biomarkers*. Li Liang, Jun Yue, Li-rong Liu, Min Han, Liu-lin Luo, and Heping Xiao, Tongji University School of Medicine, Shanghai; Huijuan Liu, Chinese Academy of Medical Sciences and Peking Union Medical College, TianJin; and Yan-lin Zhao, Chinese Center for Disease Control and Prevention, Beijing, China, concluded that the SNP in the hepcidin promoter gene may play a critical role in susceptibility to tuberculosis affecting organs other than the lungs, but not pulmonary tuberculosis. The researchers propose that decreased hepcidin production in response to infection and inflammation reduces the ability of macrophages to destroy M. tuberculosis, which then enter the circulation and spread the infection from the lungs to other areas of the body. They present their findings in the article entitled "Association of Single Nucleotide Polymorphism in the Hepcidin Promoter Gene with Susceptibility to Extrapulmonary Tuberculosis."

"This is a potentially important finding in the realm of human susceptibility to infectious disease, which is a greatly under-studied area of research and one that GTMB has chosen to highlight," says *Genetic Testing and Molecular Biomarkers* Editor-in-Chief Garth D. Ehrlich, PhD, FAAAS, Professor of Microbiology and Immunology, Executive Director, Center for Genomic Sciences and Center for Advanced Microbial Processing, Institute for Molecular Medicine and Infectious Disease, Drexel College of Medicine (Philadelphia, PA).


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