LETTERS TO THE EDITOR

Development of Extrapulmonary Tuberculosis

To the Editor: With great interest have we read the article by Fuentes and Caminero on extrapulmonary tuberculosis recently published in your journal. We believe it important to comment on several factors associated with the development of extrapulmonary tuberculosis, specifically tuberculous meningitis. Fuentes and Caminero point out that immunodepression is playing an important role in the increased incidence of extrapulmonary tuberculosis. We understand that immunodepression can arise from infection by the human immunodeficiency virus, though poverty that leads to malnutrition—as affects a large portion of the world’s population—or other causes. But further in-depth study is needed on the causes of extrapulmonary tuberculosis in patients who are not immunodepressed, and we would like to approach this important topic in this letter.

The influence of the host’s race and genetic polymorphisms on the development of clinical tuberculosis has been studied by some authors, and we now need to investigate whether certain genetic polymorphisms are associated with the development of extrapulmonary tuberculosis. Tsenova et al. showed that tumor necrosis factor (TNF-α) and a phenotype highly productive of TNF-α are determinants in the pathogenesis and progression of tuberculous infection of the central nervous system. Those authors mention the potential effectiveness of thalidomide, which acts as an inhibitor of TNF-α synthesis, the principle cause of an inflammatory process that leads to sequelae in tuberculous meningitis. Furthermore, certain single nucleotide polymorphisms (SNPs) in the gene promoter that codes for TNF-α have been associated with tuberculosis. We have found that certain SNPs in the TNF-α gene promoter are markers of certain human groups and have become stratified and preserved in certain races as humanity has evolved. We observed that a phenotype that is highly productive of TNF-α is associated with certain SNPs in the TNF-α gene promoter. Patients with such a phenotype present a higher risk of developing certain inflammatory diseases and an exaggerated inflammatory response to infections. Therefore, it is likely that a phenotype that is highly productive of TNF-α was selected and persists in certain races, making them more susceptible to the development of extrapulmonary tuberculosis, especially that of the central nervous system. This issue requires further study.

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