

potential host-susceptibility factor in tuberculosis in Chongqing, China.

METHODS: A total of 123 patients (including 91 children and 32 adults) with tuberculosis and 249 normal controls (including 167 children and 82 adults) were selected randomly. The polymorphisms at positions -607A/C and -137G/C in promoter of the *IL-18* gene were analyzed by using polymerase chain reaction with sequence-specific primers.

RESULTS: The allele and genotype frequencies of IL-18/-607 gene polymorphisms were similar in patients with tuberculosis and in controls. However, frequencies of the -137GG, GC, and CC genotypes were 67.9%, 28.5%, and 3.6%, respectively, in controls and 78.9%, 19.5%, and 1.6%, respectively, in those with tuberculosis. Frequency of the -137GG genotype in tuberculosis was significantly higher than that in controls ($\chi^2 = 4.881$; $P = .027 < .05$). The frequency of allele G at position -137 in patients with severe tuberculosis was significantly higher than that in patients with pulmonary tuberculosis ($\chi^2 = 4.336$; $P = .037 < .05$).

CONCLUSIONS: Polymorphism of the IL-18 gene promoter at position -137 is a potential host-susceptibility factor in tuberculosis in Chongqing. The people with allele C at position -137 in the promoter of the *IL-18* gene may be protected against mycobacterium tuberculosis infection. The polymorphisms at position -137 of the *IL-18* gene may be associated with a severe degree of tuberculosis.

ASSOCIATION OF POLYMORPHISMS OF THE INTERLEUKIN 18 RECEPTOR: A GENE WITH SUSCEPTIBILITY TO TUBERCULOSIS IN CHONGQING, CHINA

Submitted by Li-Ping Jiang

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INTRODUCTION: The interleukin 18 (IL-18) receptor complex is a heterodimer consisting of IL-18R α and IL-18R β . Both chains are required for IL-18 signaling transduction in T cells and natural killer cells.

OBJECTIVE: We aimed to determine whether polymorphisms of the IL-18R α gene promoter were associated with susceptibility to tuberculosis in Chongqing, China.

METHODS: In 123 patients (91 children and 32 adults) with tuberculosis and 249 normal controls (167 children and 82 adults) in Chongqing, we analyzed the polymorphisms at positions -69T/C and -638T/C in the promoter

of IL-18R α by using polymerase chain reaction with sequence-specific primers.

RESULTS: Allele and genotype frequencies at position -638 of the IL-18R α gene polymorphisms were similar in patients with tuberculosis and normal controls ($P > .05$). However, the frequency of -69/CC was significantly lower in patients with tuberculosis than in controls ($\chi^2 = 8.484$; $P = .004 < .05$). The frequency of -69/TT was significantly higher in patients with tuberculosis than in controls ($\chi^2 = 4.027$; $P = .045 < .05$). The frequency of allele C at position -69 in tuberculosis was significantly lower than that in controls ($\chi^2 = 9.816$; $P = .002 < .05$). The frequency of allele C at position -69 in patients with severe tuberculosis was significantly lower than that in patients with pulmonary tuberculosis ($\chi^2 = 4.664$; $P = .031 < .05$).

CONCLUSIONS: Polymorphisms of the IL-18R α gene at position -69 were associated with susceptibility to tuberculosis in Chongqing. The people with allele C at position -69 may be protected against mycobacterium tuberculosis infection. Moreover, the position -69 of IL-18R α may be associated with a severe degree of tuberculosis.

PLACENTAL ADENOVIRAL GENOME: POLYMERASE CHAIN REACTION DETECTION AND PLACENTAL HISTOLOGY IN PRETERM AND TERM INFANTS

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INTRODUCTION: Intrauterine infection is an important cause of spontaneous preterm birth. However, evidence-based etiology for the causative role of viral infection is still lacking. Intervillous trophoblasts express adenovirus receptor. Infection of trophoblast cells in vitro by adenovirus early in pregnancy has shown increased apoptosis. Adenovirus early in pregnancy may cause placental dysfunction. Mature syncytiotrophoblasts do not express adenovirus receptor.

OBJECTIVE: The aim of this study was to test the hypothesis that detection of adenovirus in placental tissue is associated with preterm birth and correlates with placental histopathological findings that are suggestive of infection.

METHODS: Placentas were prospectively collected from consecutive deliveries. Detection of the adenovirus genome was tested by polymerase chain reaction assay. Placental histology and immunohistochemistry studies with monoclonal antibody CD45 were evaluated for

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