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Increased IL-12 and Interferon-gamma, But Not IL-18 Production, After *In Vitro* Stimulation with a 30-kDa Mycobacterial Antigen in Patients with Tuberculous Pleurisy

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Tuberculous pleurisy is a good model for resolution of local cellular immunity. However, little is known about the role of mycobacterial antigen (Ag) in pleural inflammation through its release of Th1 regulatory cytokines. We investigated the cytokine profiles attributable to Th1 elevation associated with tuberculous pleurisy, interleukin (IL)-12, IL-18 and IL-10 productions after *in vitro* stimulation with a 30-kDa or purified protein derivatives (PPD) Ag in 12 patients with pleural effusion, using by enzyme-linked immunosorbent assay (ELISA). The 30-kDa or PPD Ag stimulated pleural mesothelial cells (PMC) from 8 patients with tuberculosis (TB) contained significantly ($p<.001$) more IL-12 productions, highly correlated with interferon-g (IFN-g), than did effusions from 4 patients with malignant effusions. Unexpectedly, IL-18 production by Ag-stimulated PMC from TB patients was significantly ($p<.001$) decreased, compared with those from malignant patients, although pleural fluids had higher levels of IL-18 ($p<.005$) than those with malignant effusions or autologous serums. In IL-10 production, PMC showed a significantly ($p<.05$) decreased level after stimulation with the 30-kDa, compared with those by PBMC from the TB patients. Our findings provide evidence that increased IL-12, but not IL-18, in addition to the decreased IL-10 is contributed to the protective immunity in human tuberculous pleurisy through 30-kDa-mediated Th1 elevation.