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Anti-inflammatory Effect of Pentoxifylline

To the Editor:

We read with great interest the recent *in vitro* study by Tong and colleagues.[1] We have some comments. First, the authors implicated the possibility of pentoxifylline as a therapeutic option for pulmonary sarcoidosis. A study[2] has described that pentoxifylline has no severe side effects as compared with dexamethasone. However, as Tong et al[1] introduced in their discussion, pentoxifylline induces a selective suppression of interleukin-2 and interferon-γ, T-helper type 1-derived cytokines.[3] T-cells play a central role in cell-mediated immune responses through the production of type 1 cytokines, such as interferon-γ and interleukin-2. It has been reported that the suppression of T-helper type 1 function damages the host response to fungi.[4] We should give careful consideration when using the agent, especially its long-term use in clinical stages, because it may lead to mycosis in patients.

In addition, we wonder if dexamethasone could not inhibit lipopolysaccharide-induced interleukin-1β expression in alveolar macrophages in the experiment by Tong et al.[1] Glucocorticoids are potent inhibitors of immune response, inflammation, and endotoxic shock. This occurs, at least partly, through an inhibition of the synthesis of proinflammatory cytokines and chemokines.[5–7] One of the target enzymes of glucocorticoids inhibition is interleukin-1β. Dexamethasone (10 nmol/L to 10 μmol/L) inhibits interleukin-1 messenger RNA in lipopolysaccharide-stimulated human monocytes in a dose-dependent fashion.[8] Also, dexamethasone suppresses interleukin-1β gene expression in lipopolysaccharide-stimulated RAW 264.7 cells through the inhibition of the activation of transcription factors related to endotoxin such as nuclear factor-κB and activator protein-1.[9] Other mechanisms may be present in lipopolysaccharide-induced interleukin-1 production in alveolar macrophages of patients with sarcoidosis.

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