

groups HIV- and HIV+ with CD4+ >500. A significant difference in sIgE (lnIgE) distribution was also observed between the HIV+ subgroups with CD4+ >500 and 200 <CD4+ <499. Distribution of CD4+ differed between the HIV+ groups with increased and normal sIgE and the control group. In the HIV+ groups, a rising frequency of increased sIgE was observed together with CD4+ depletion. The lowest percentage – 14% in the HIV+ group with CD4+ >500 – was significantly different from the control group, the HIV+ group, and the HIV+ subgroup with CD4+ >500. In the group of HIV+ subjects in whom several parameters were determined, the analysis of CD4+ distribution showed statistically significant differences between all the determinations. sIgE distributions were different between determinations I and II and determinations I and III, and lnIgE distributions differed between determinations I and II. Fig. 1 presents the individual dynamics of sIgE. The frequency of increased sIgE was significantly lower in determination I than in determinations II and III and healthy persons.

In this study, the intensification of IgE synthesis was observed in HIV+ patients together with the progression of HIV infection. This could be explained by the qualitative and quantitative ascendancy of Th₀ and Th₂ lymphocytes (1–3) and higher levels of sCD23 (which could stimulate the IgE production) (4) during HIV infection. In the subgroup HIV+ without CD4+ depletion, the frequency of increased sIgE occurrence was statistically significantly lower than in the general population. If we assume random exposure to HIV infection, people with higher sIgE might be infected less often. The explanation of this phenomenon may be the HIV competition with β -chemokines (which augment tissue concentrations in atopic patients with increased sIgE) for the cellular receptor CCR5, which is a necessary coreceptor for

macrophage-tropic strains of HIV-1 (NSI HIV) (5, 6), which predominate in the early phase of HIV infection.

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Tuberculin-primed PBMC fail to produce TH2-type cytokines

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Key words: atopic allergy; IFN- γ ; IL-4; IL-13; Th1; Th2; tuberculin.

● In allergic rhinitis, the production of Th2 cytokines leads to IgE production, tissue eosinophilia, and allergy symptoms (1). During specific immunotherapy (SIT), the Th2-cytokine production is reconstituted by Th1 cytokines (2). Mycobacteria modify the immune response and have been proposed as adjuvants for SIT (3). Animal models suggest that mycobacteria or their components suppress the Th2 immune response (4). We investigated the allergen-induced cytokine responses of human peripheral blood mononuclear cells (PBMC) primed by tuberculin (PPD).

Ten patients with pollen allergy and seasonal rhinitis verified by skin prick test (six women and four men, 30; 22–42 years of age [median; range]) and five healthy controls with no history of allergic symptoms (three women and two men, 38; 28–43 years of age) gave a blood sample voluntarily after informed consent. Ficoll-isolated, washed PBMC were primed by incubation at 10^6 cells/ml for 3 days with 0, 1, or 10 μ g/ml of PPD (Statens Seruminstitut, Copenhagen, Denmark). The washed primed cells (2×10^6 cells/ml) were cultured with allergens (50 μ g/ml of whole extracts of birch [*Betula verrucosa*] and timothy [*Phleum pratense*], kindly provided by ALK-Abelló [Hørsholm, Denmark]) for 3 days. IL-4, IL-13, IFN- γ , and β -actin mRNA in cell pellets were analyzed by

Allergen-induced IL-4 and IL-13 are downregulated and IFN- γ upregulated.

RT-PCR and time-resolved fluorometry, as described earlier (5, 6). Cytokines IL-4 and IFN- γ released by PBMC in supernatants were measured by ELISA using antibody pairs and standard kits (Pharmingen, San Diego, CA, USA). The Wilcoxon signed-rank test was used for statistical analysis.

The PPD priming dose-dependently increased the IFN- γ production as measured by both RT-PCR ($P < 0.05$ at 1 and 10 $\mu\text{g/ml}$) and ELISA ($P < 0.01$ at 1 and 10 $\mu\text{g/ml}$) in patients and controls (Fig. 1). The PPD priming decreased the mRNA expression of the Th2 cytokines (IL-4: $P < 0.01$ and IL-13: $P < 0.05$ at 10 $\mu\text{g/ml}$) and IL-4 production by ELISA ($P < 0.05$ at 10 $\mu\text{g/ml}$) in patients.

The inverse association of mycobacterial infections and atopy has been shown in animal models (4). Our observations on decreased IL-4 and IL-13 responses and increased IFN- γ response are in line with these studies, implying that both human and mice cells, *in vitro* and *in vivo*, are applicable

to testing of Th-cell-modulating properties of microbial components. In SIT, the beneficial outcome has been associated with induction of Th1 and suppression of Th2 responses (2). The first step in development of microbial adjuvants for intensification of SIT is easily performed by the described *in vitro* method.

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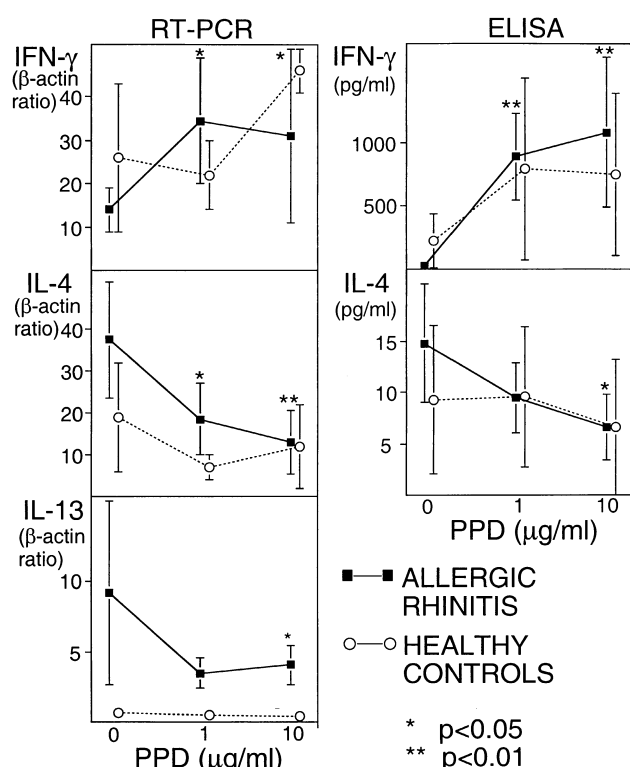


Figure 1. Effect of PPD priming on allergen-stimulated cytokine production in PBMC cultures from allergic rhinitis patients and healthy controls.

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Allergy to latex, fruit, and pollen

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Key words: IgE determination; *in vitro* diagnostic tests; natural rubber latex; profilins.

● SEROLOGIC tests have become critically important as an alternative confirmatory test in the diagnosis of natural rubber latex (NRL) allergy

(1). From a diagnostic perspective, it is

Common allergens are detected by IgE antibodies.