



Figure 4. *Co-culture with PPD of the normal positive PPD control cells and patient's adherent cells. *Gradual increase in the percentage of adherent cells per total patient's cells resulted in a progressive decrease of the proliferative response.

as suppressor cells in human tuberculosis,³ as well as in leprosy.⁵ Prostaglandin-mediated adherent cells¹⁶ have been found to suppress cellular immunity in both sarcoidosis and Hodgkin's Disease.^{8,9}

This particular patient has, in addition to anergy and increased suppressor cell activity, common variable hypogammaglobulinemia. It has been shown by Waldmann et al¹¹ that this disease is also associated with an immunoregulatory imbalance, namely abnormal suppressor cell activity. We postulate that the association of common variable hypogammaglobulinemia and *M. avium-intracellulare* pulmonary infection in this patient may be explained as due to a generalized suppressor hyperreactivity which may have been first associated with the hypogammaglobulinemia. Increased pulmonary infections ensued, leading to bronchiectasis. Due to the deformed structure and impaired clearance secondary to bronchiectasis, colonization of an opportunistic organism such as *M. avium-intracellulare* was facilitated. Because the increased suppressor activity resulted in depressed cellular immunity, the colonization eventually resulted in tissue invasion by the organisms with progressive destruction of lung parenchyma.

Summary

Immunologic studies were done in a patient with common variable hypogammaglobulinemia, bronchiectasis and *M. avium intracellulare* pulmonary infection.

Adherent cells from the patient were found to suppress the proliferative response of normal control cells to PPD antigen. The suppression seemed to be mediated by prostaglandin as it was reversed by indomethacin.

Increased suppressor cell activity may play an important role in the pathogenesis of *Mycobacterium avium-intracellulare* infection by permitting the transformation of a status of colonization by the organism in pre-existing pulmonary conditions into an invasive, progressive disease.

The role of prostaglandin in mediating this suppression merits further consideration as reversal of the suppression by indocin may favorably influence the outcome of this difficult-to-treat disease. Immunologic studies of a large group of patients with *M. avium-intracellulare* infection would be required to clarify this possibility.

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