FAILURE OF SPECIFIC ANTIGEN PRESENTATION IN LEPROMATOUS LEPROSY

Sir,

Recently Narayanan *et al.* (*Lepr Rev* 1984, **55,** 301–308), using monoclonal antibody OKT-6, have reported a normal number of Langerhans cells in the epidermis of lepromatous leprosy cases and a slightly higher count in cases of tuberculoid leprosy. In the infiltrate they have noted T-6 positive cells lacking dendritic processes reminiscent of LCs in the lymphocyte mantle of TT/BT, whereas in the infiltrate of lepromatous leprosy T-6 positive cells were conspicuously absent. The Ia positivity was seen with the lymphocytes surrounding the epitheloid cells in TT and with foamy macrophages in lepromatous leprosy. Based on these observations authors have concluded that in leprosy there may not be significant defect in the antigen presentation.

In our earlier studies, using histochemical staining for ATPase activity, we have observed in the epidermis a significant reduction in LCs population in LL and an adequate number in TT. Combining the observations with OKT-6[1] and ATPase[2], it can be interpreted that in LL most of the LCs were inactive. This was further confirmed by our electron microscopic study[3] of LC in leprosy, where we observed a decreased number of lysosomes and rough endoplasmic reticulum; dense matrix and indistinct cristae of mitochondria; numerous vacuoles in cytoplasm, and a normal nucleus, suggesting that cells were inactive.

OKT-6 is more of an anatomical marker and does not indicate the functional state of LC, whereas ATPase activity reflects functional activity of a cell. The difference in LC count in two studies could be, as also suggested by Narayanan *et al.*, due to a difference in staining methods. Narayanan *et al.*, have also observed less numbers of Ia positive LCs in epidermis as compared to T-6 positive in LL patients. In sarcoidosis, an example of anergy, Fox *et al.*, reported a decrease in both Ia & T-6 positive LCs in the epidermis.[4] It is the Ia of LCs which plays the key role in antigen presentation. LCs when treated with anti-Ia antibodies fail to present antigen to lymphocytes.[5] Another important factor in eliciting sensitization is the presence of an adequate number of LCs in the epidermis.[6] In lepromatous leprosy the decrease in the number of active LCs and low count of Ia positive LCs in the epidermis can explain their poor sensitizing capacity to various chemical sensitizers.

In the recent past, in multibacillary leprosy, researchers have shown defects in lymphocyte[7] and macrophage functions,[8] low IL-2[9] and γ-interferon activity[10] and recently we have also shown defects in antigen presenting cells (LCs)[3]. It is interesting to note that all these defects in afferent and efferent limbs of the immune system have developed after the entry of bacilli. Once the specific unresponsiveness sets in, the APCs subsequently fail to present the specific antigen to the local lymphoid system and thus it becomes difficult to induce sensitization. This inability of APCs to present antigen could be due to the presence of specific T-suppressor factor (TsF) as shown by Ptak and Gershon in their model of contact hypersensitivity in animals.[11]

In the past we have put forth a hypothesis, based on the work of Ptak *et al.* in the field of contact dermatitis,[12] that it is the initial mode of entry of bacilli which decides the type of leprosy one will develop. If the organism enters the body through the epidermis and is presented to skin-associated lymphoid tissue by LCs it will generate specific sensitization, but when it bypasses LC or finds entry through oral or nasal routes it generates specific unresponsive state. We feel that work in the field of mode of entry and identification of specific antigen might be helpful in understanding this disease.

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FAILURE OF PASSIVLEY TRANSFERRED LEPROSY LYMPHOCYTES TO DEMYELINATE PERIPHERAL NERVE

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