

Hypothesis: Solar Ultraviolet Radiation and the Initial Skin Lesion of Leprosy

TO THE EDITOR:

We wish to put forward a hypothesis attributing the pathogenesis of the initial skin lesion of leprosy to solar ultraviolet radiation. Even though the importance of nasal discharge from lepromatous patients in the dissemination of *Mycobacterium leprae* is well known (^{4, 11}), the portal of entry of the organism has been a matter of debate. The two portals of entry seriously considered are the skin and the upper respiratory tract (¹⁰). Since it is improbable that an inert, non-motile and relatively nontoxic organism such as *M. leprae* can invade the intact epidermal barrier, the entry could only occur through the epidermal barrier broken as a result of injuries, insect bites, or scabetic lesions (⁷). The proposition that the skin is a portal of entry is also supported by several reported cases of inoculation leprosy which have been listed by Machin (⁹). The epidemiological and pathological data to be discussed here are also in favor of such a proposition.

At the time of entry through the skin, the first immunocompetent cell that *M. leprae* would encounter is the Langerhans' cell which forms a network in the midepidermis. These cells, after ingesting the antigen, migrate to the regional lymph nodes and present the processed antigen to T lymphocytes. It is known that antigen presentation by the Langerhans' cells is impaired by ultraviolet-B radiation (UV-B; wavelength 280 to 320 nm) both in mice and in humans (^{1, 5, 14}). In UV-B-treated human subjects, there is a 50% decrease in ATPase-stained Langerhans' cells (⁵).

Three features of the epidemiology of leprosy are important in support of the hypothesis to be presented: a) Geographic dis-

tribution—Today leprosy is mainly a disease seen in the tropics and subtropics, both of which receive a maximum quantity of solar radiation which includes ultraviolet-A (wavelength 320 to 400 nm) and UV-B radiation.

b) Distribution of the first skin lesion of leprosy—The first skin lesion of leprosy in adults is most often present on the exposed (unclothed) parts of the body (^{3, 6}). In children, the distribution of the first skin lesion is random (⁶). These observations are explained by the fact that the uncovered parts of the body are often exposed to minor injuries and insect bites through which *M. leprae* could gain entry. Children in warm climates are scantily clad; hence, the distribution of the first skin lesion is random (⁶). Also, in South India, when the distribution of a single lesion of tuberculoid leprosy was studied, it was found that females had significantly fewer lesions on the trunk and lower extremities, areas which are covered by a saree (³). The results pooled from various studies also support the observation that the initial skin lesion of leprosy in adults occurs on body parts which are exposed to the environment (⁹) and, hence, to solar radiation.

c) Pathological and experimental data—Histopathological and bacteriological studies of early lesions of leprosy tend to favor the skin as one of the portals of entry (^{12, 13}). If immunity is maximum, the bacilli are arrested in the epidermis. If immunity is partial, the bacilli reach the subepidermal zone. Intra-epidermal leprosy lesions have also been observed (¹³).

Doses of UV-B which correspond to the exposure levels encountered by humans cause local immunological unresponsive-

ness in mice⁽⁵⁾. In humans, UV radiation impairs the expression of MHC class II antigens by the Langerhans' cells and also abrogates their alloantigen-presenting function⁽²⁾. Finally, Liu, *et al.*⁽⁸⁾ have shown a significant decrease in the number of Langerhans' cells in the lesional skin of TT and BT leprosy.

Hypothesis. We suggest that abrogation of the antigen-presenting function of epidermal Langerhans' cells (at the site of entry of *M. leprae*) by solar UV-B radiation is involved in the pathogenesis of the initial skin lesion of leprosy. It is likely that *M. leprae*, after gaining entry through the epidermis, bypass the network of epidermal Langerhans' cells whose function is impaired by the solar UV-B radiation. This leads to failure of the initiation of the cell-mediated immune response at the level of the epidermis. The organisms then enter the dermis where they are phagocytosed by dermal macrophages or Schwann cells of unmyelinated nerve fibers. The further course of the infection depends upon the immunological status of the host. If the bacilli are destroyed as a result of the cell-mediated immune response, no disease occurs. If the bacilli multiply, there may be an indeterminate lesion which may progress to a classifiable lesion if the immune response fails to eradicate the infection.

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