The crucial areas of health education in leprosy are: 1) ulcers, 2) patches, 3) appropriate footwear, 4) reactions, and 5) treatment. The game's simple questions have been formulated for each of these areas and may be answered with either "Yes" or "No." Twenty or more such questions are printed one on each card which measures typically 4 × 5 cm. Answers, incentives, and disincentives are printed on the same card. An incentive for the correct answer could be "Move forward five spaces" or "Collect 25 points." A disincentive for the wrong answer could be "Miss a turn" or "Pay a penalty of 20 points," or "Go directly to hospital."

One of the players is appointed "Doctor" and reads the contents of the cards and asks the questions of the other players. Guessing is allowed since this would serve to reinforce the correct answers.

To start, the players have to throw a "six" on a single, six-sided die. Players must progress along the numbered segments of the board corresponding to the throw of the die. They must answer the questions pertaining to the segments which are so marked. Some questions pertaining to "Patch" may read: 1) A leprosy patch can appear anywhere on the skin of the body. 2) A leprosy patch often shows a loss of sweating. 3) A leprosy patch is caused by God's curse. 4) All patches on the skin are due to leprosy.

Players must throw a "six" and pay a penalty to get out of the hospital since it is not desirable for patients to go to the hospital in the first place. The space labeled "Choice" enables the players to choose whichever topic they feel is their strong point. They may choose the topic which they know has more incentives per card, also.

A points system consisting of various colored cards, representing various values, is incorporated into the game in order to authenticate the incentive/disincentive scheme of things. The person with the maximum number of points after a fixed time is declared the "Winner."

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Reactive Oxygen Intermediates in the Phagocytes from Leprosy Patients: Correlation with Reactional States and Variation During Treatment

TO THE EDITOR:

It is well known that lepromatous leprosy patients' lymphocytes are unresponsive to *Mycobacterium leprae in vitro*. Theoretically, this defect in cell-mediated immunity toward *M. leprae* could be due to a macrophage defect in these individuals. Monocytes from lepromatous leprosy patients secrete subnormal quantities of H_2O_2 after stimulation with the potent secretagogue, phorbol myristrate acetate (PMA) ([4]). *M. leprae* fails to stimulate significant O_2^- release from human monocytes, human neutrophils, or mouse peritoneal macrophages ([1, 3]). On the other hand, monocyte activation does occur during reactional episodes in leprosy patients, implying that some other pathway must be operating during reaction which does not operate in the absence of reactions in the patients ([5]). To further

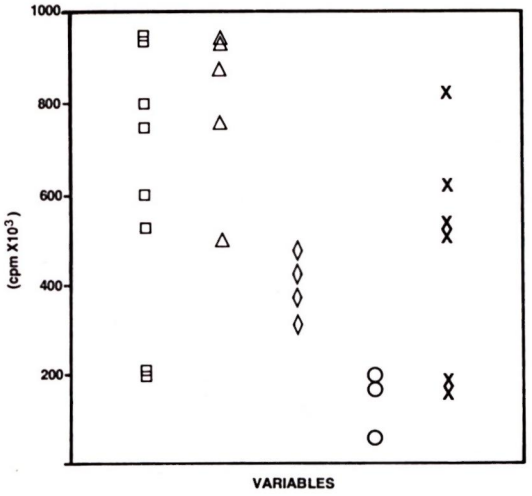

FIG. 1. Enhancement of chemiluminescence response by monocytes from laboratory controls (□), BT patients (△), BL patients (◇), LL patients (○), and lepromatous patients with ENL (×). Amount of chemiluminescence is expressed as Δcpm = PMA stimulated count − PMA free count. Monocytes were cultured for 1 day, washed, and transferred to chemiluminescence reaction medium, and then stimulated with PMA (0.5 μg/ml).

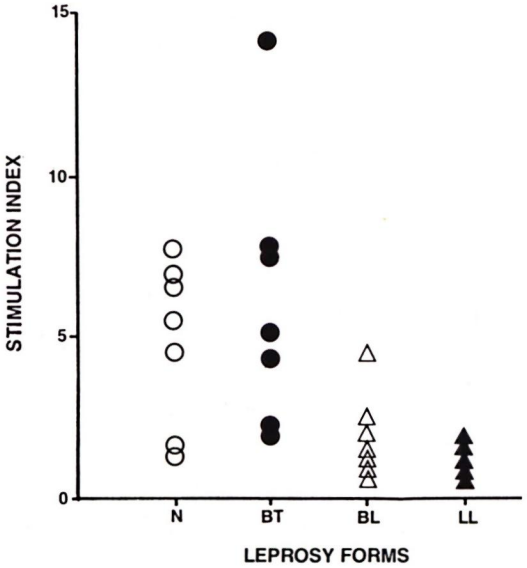

FIG. 2. Lymphocyte transformation test (LTT) measured by [³H] TdR incorporation for laboratory controls (N) and leprosy forms (BT, BL, LL). Amount of LTT response is expressed as Stimulation Index = antigen-stimulated culture count/antigen-free culture count. Mononuclear cells were cultured for 5 days in presence or absence of *M. leprae*. [³H] TdR was added and after 18 hr of labeling, thymidine uptake was assayed with a liquid scintillation counter.

study monocyte function in reactional and nonreactional leprosy patients we measured monocyte activation by luminol-dependent chemiluminescence and lymphocyte proliferation in response to *M. leprae*.

Subjects. The leprosy patients were attending the outpatient leprosy unit of the Oswaldo Cruz Foundation, Rio de Janeiro, Brazil. The clinical diagnosis was confirmed by skin biopsy in each case. We studied 8 patients with polar lepromatous (LL), 8 with borderline lepromatous (BL), 8 with borderline tuberculoid (BT) disease, and 8 healthy laboratory controls. Five of the 8 LL patients were experiencing erythema nodosum leprosum (ENL). Three of the 8 BL patients had ENL and 1 had reversal reaction. Two of the 8 BT patients had neuritis. Two of the 5 LL patients were not being treated with anti-reactional drugs. All of the other patients with reactions were receiving treatment with clofazimine and/or thalidomide.

Peripheral blood mononuclear cells were prepared with Ficol-Hypaque gradient centrifugation ([2]). After 24-hr incubation on glass coverslips, the adherent cells were transferred to liquid scintillation vials containing RPMI 1640 medium supplemented with 10% v/v fetal calf serum and luminol (5-amino-2,3-dihydro-1,4-phthalazinedione) at a final concentration of 5×10^{-5} M. Upon oxidation, luminol exhibits striking chemiluminescence. This was quantitated using a liquid scintillation counter, measuring chemiluminscence for 1 min after stimulation of the monocytes with PMA at a concentration of 0.5 μg/ml and subtracting the background chemiluminescence of the monocyte preparation before the addition of PMA.

Resting monocytes from nonreactional BL and LL patients showed lower background chemiluminesence than monocytes from healthy laboratory controls (an average of 22% of control values). Monocytes from BL and LL patients with reactions, on the other hand, showed higher resting chemiluminescence than cells from healthy controls (228% of control values). Upon stimulation with PMA there was an increase in chemiluminescence with monocytes from the healthy

controls and from BT patients (Fig. 1). In nonreactional LL patients there was no chemiluminescence with PMA stimulation. In contrast, monocytes from LL patients with ENL showed activation with PMA. In patients with ENL reactions being treated with thalidomide, chemiluminescence induced by PMA was lower than that seen in patients with untreated ENL.

As expected, lymphocyte proliferation *in vitro* in response to *M. leprae* was positive in some of the healthy controls and BT patients, but was essentially negative in BL and LL patients (Fig. 2).

Although the number of patients studied in each group is small, the results suggest that during ENL the monocytes of LL patients can respond to PMA although their lymphocytes remain unresponsive to *M. leprae.*

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Can Sandflies Be the Vector for Leprosy?

To the Editor:

There are several biting arthropods existing in leprosy-endemic areas of which any one could theoretically act as a vector for the transmission of leprosy. Arthropods such as mosquitoes, bed bugs and a few others take up *Mycobacterium leprae* while feeding on lepromatous leprosy patients ([1, 3, 7]). Bacilli remain viable for many days in the body of these arthropods ([5, 6, 8]). In tropical and subtropical countries where leprosy is endemic, sandflies are also commonly found. However, their role as a vector in the transmission of leprosy has so far not been investigated.

Acid-fast bacilli in field-collected sandflies. As a preliminary approach to screen for acid-fast bacilli (AFB), sandflies were periodically collected from the mud houses of known leprosy patients as well as from houses where there were no cases of leprosy. The sandflies were identified and classified ([2, 9]). There are eight species of sandflies belonging to the genera *Phlebotomus* and *Sergentomyia* existing in these areas of Agra, India. *P. papatasi* is found to be the most