

Leprosy in Children One Year of Age and Under

TO THE EDITOR:

I was most interested to read the article by Brubaker, *et al.* (1) and congratulate the authors on the work involved in such a search.

There is clear and substantial evidence that *Mycobacterium leprae* antigens and also whole *M. leprae* cross the placenta. There are a few reported cases indicating that this passage has occurred early in pregnancy: Cerruti and Bechelli (2), Davison and Bernard (4). An analogous situation is seen with malaria (reviewed by Duncan (5)) where the infecting parasite is present in vast numbers in the maternal circulation, where there is clear evidence of transplacental transfer of both parasite and specific antibody, and yet where relatively few cases of congenital malaria are recorded. Similarly, with syphilis, until comparatively recently, it was thought that because evidence of transplacental infection with *Treponema pallidum* was not present before late in the second trimester of pregnancy, that that was the time when the treponeme crossed the placental barrier; it is now well recognized that *T. pallidum* crosses the placenta at an early stage, possibly as early as 14–16 weeks' gestation, but that clinical manifestations of the disease do not occur until a significant degree of immunocompetence has been achieved by the fetus.

It is well recognized that immune tolerance is induced most readily in the prenatal period by contact of antigen with immature lymphocytes, that maintenance of the antigenic challenge results in persisting immune tolerance, but when the antigenic challenge is of short duration, with a steady increase in the proportion of mature lymphocytes prenatally, immune tolerance diminishes steadily and may no longer be of significance at the time of birth. In humans,

it is recognized that small lymphocytes appear in peripheral blood at about 7 weeks' gestation, developing further in the thymus to emerge as T cells by 12 weeks, with well-developed responsiveness to PHA by 14 weeks.

It seems likely that in leprosy clinical manifestations of the disease in early childhood will depend upon several factors: a) The stage of gestation at which the fetus receives the first challenge from *M. leprae*/*M. leprae* antigens. b) The dose of the infecting organism. c) The duration of time *in utero* and postpartum during which a persistent challenge is maintained.

Clearly, considerable work must still be done on immunogenesis in the human fetus and neonate to explain these phenomena.

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