LETTERS TO THE EDITOR

(*Dr. Chaussinand's letter is repeated from July* Leprosy Review *so as to present the author's own corrected version of the English text of his letter; the original was in French*)

Dr. R. CHAUSSINAND of Institut Pasteur, Paris, writes about the article "Is Leprosy Transmitted by Arthropods?" (by Prof. NIELS DUNGAL of Reyjavik, Iceland, in *Leprosy Review* **32,** 1, pp.28–35). CHAUSSINAND says, "In this article Prof. DUNGAL declares concerning the routes of penetration of the Hansen bacillus that 'CHAUSSINAND and many others with him have incriminated the inhalation of nasal droplets of mucus from infective patients, as in tuberculosis' " (p.29). However, I have always affirmed the contrary, both in my articles and in my books. So in the paragraph in the two editions of *La Lèpre*, to which DUNGAL refers, my opinion is expressed in the following terms: "Most leprologists consider at the present time that the penetration of the Hansen bacillus through the mucosae is exceptional. They base themselves on the fact that mucosal lesions are never observed at the beginning of the disease. Furthermore, leprosy patients with the benign type of leprosy only infrequently show changes in the pituitary and buccopharyngeal mucosae, and just as in leprosy patients with the malign type of leprosy, the appearance of these lesions always occurs after that of skin and nerve lesions. On the other hand, there is no hint that the Hansen bacillus may enter into the body through the pituitary, buccopharyngeal and laryngeal mucosae or through the mucosa of the stomach, intestine, and lungs".

As for the various arguments presented in this article, I only agree with NIELS DUNGAL when he states in connection with my theory on the antagonism between tuberculosis and leprosy: "this theory would explain much, but is difficult to prove".

The phenomenon of crossed premunition between two infections relatively akin in nature is determined by the pathogenic agent which infected the body in the first place. This contamination thus renders the body ready to defend itself, in certain measure, against a later attack by the second pathogenic germ. To obtain clinical observations which are conclusive, it is then indispensable in each case to know the primary contaminating agent. There is no room for doubt in this matter, if one is presented with a leprosy patient in whom the tuberculin reactions are negative. On the other hand, the problem will be practically insoluble when the leprosy patient reacts to tuberculin. It is then generally impossible to be certain of the nature of the initial bacillary infection.

It is however evident that this crossed premunition is only relative and that its intensity differs from one subject to the other. The degree of para-immunity of the body against the second infection depends on

the degree of acquired immunity against the first infection. A bacillary impregnation which has not provoked any phenomenon of specific immunity cannot produce a para-immunity. So the organism of a lepromatous case of leprosy, anergic to leprolin, which presents no immunity to the Hansen bacillus, will never achieve premunition by means of its leprosy against a later infection due to the Koch bacillus. Now the degree and the very existence of the specific anti-leprosy immunity or especially the antituberculosis immunity, which can benefit the body at the moment of its contamination by the second germ, are very often impossible to determine retrospectively. Doubtless this antagonism between tuberculosis and leprosy is not the sole cause for the progressive evolution of leprosy. Other factors, varying from one country to another, enter in to play a role more or less important.

I think we can obtain a valuable clinical hint on the problem of relative para-immunity between leprosy and tuberculosis when one studies, in different countries where the two infections are endemic, the percentage of patients attacked by advancing pulmonary tuberculosis, on the one hand in tuberculoid leprosy patients strongly allergic to lepromin and on the other hand in lepromatous leprosy patients who are anergic to lepromin. The causes of error are considerably equalised in the two groups, if the both groups are numerically important and well matched. The percentage of advancing pulmonary tuberculosis should then be significantly higher in the group of lepromatous cases. It is especially clear that leprosy cases attacked with advancing pulmonary tuberculosis should only be taken into account. Leprosy patients which only show tuberculinic allergy or benign or regressive lesions of tuberculosis should be excluded from these statistics, since this para-immunity can only be relative. Also there should be excluded such patients who have an antileprosy therapy of the nature of streptomycin, INH, or other drugs very active against tuberculosis.

As for the para-immunity between tuberculosis and leprosy, it is very difficult to obtain a useful indication unless these researches deal with subjects having reacted to tuberculin or having been vaccinated and re-vaccinated with BCG, at least three years before the appearance of clinical lesions of leprosy. Subjects negative to tuberculin and not vaccinated with BCG should then furnish a higher percentage of leprosy cases and especially of intermediate or lepromatous leprosy. Whereas among subjects reacting to tuberculin or vaccinated by BCG since at least three years, the cases of leprosy should be rarer and mostly tuberculoid in type.

It is certain that it is difficult to prove the existence of an antagonism between tuberculosis and leprosy but the search for this proof is indeed worth trying, for it will bring, as NIELS DUNGAL justly says, valuable clarification of leprosy epidemiology.

Inst. Oswaldo Cruz,
Rio de Janeiro