

Editorial

SACRED COWS AND HELPFUL MICE or THE TRANSMISSION AND SPREAD OF LEPROSY

We may smile at some of the ideas and beliefs of the pre-scientific era—at the miasmas and the phlogiston, the humours and the clysters. We rather look down on the old historic battles between Danielssen and Hansen, representing respectively the champions of the traditional view that leprosy was hereditarily transmitted, and the advocates of the new-fangled theory that this disease-cum-myth was actually due to a specific micro-organism. With all our newly acquired knowledge, however, we may still be in danger of repeating some of the unverified assumptions that have been taken over uncritically from the past. Despite the continuing helpfulness of the co-operative mouse, with its footpads and its immunological apparatus—intact, or experimentally rendered deficient—“sacred cows” still exist. Some of them wax fat, get sleek and respectable, even reproduce their kind. They are referred to with fitting awe; no-one doubts their existence, or their essential rightness. They receive deferential acclaim in text-books, learned articles, lectures . . . until some bold spirit has the temerity, the effrontery, to ask a question or cast tentative doubts on their sacrosanct inviolability.

“Leprosy is spread by skin-to-skin contact”, it is said. We have all said it at some time or other. When we add “prolonged and intimate contact”, we conveniently fail to define the kind of contact. Some alternative explanation had to be sought when the hereditary theory of transmission was shown to be untenable. There were the family infections, persisting for generations, the household and community foci, and the demonstration of *Myco. leprae* in discharges from patients with multibacillary types of leprosy.

The histopathologists, however, were always insisting on the extreme rarity of organisms in

the cells of the epidermis, and also in the subepidermal clear zone. Extremely few organisms seemed to manage to scale this double barrier, and those that did so, appeared to be no longer viable. Rather more bacilli were to be found in the secretory cells and the acini of the sweat glands, and in the cells lining the hair follicles of the skin. And some could be demonstrated in the galactophorous ducts of the lactating mammary gland, and in the renal glomeruli. But the uncounted millions seemed to be safely imprisoned behind an impervious and impassable tissue barrier.

On another page (167), Pedley pursues, with the minimum of apparatus, his critical questionings of the bacterial basis for the old assumptions underlying the “skin-to-skin” theory of the transmission of *Myco. leprae*. According to his findings, acid-fast organisms are extremely rare on the surface of the intact skin, whatever may be happening in the underlying dermis. They are demonstrably numerous, of course, in the discharge from lepromatous ulcers (as distinct from neuropathic ulcerations of the extremities), and also in that from the nasal mucosa. Other observers have been similarly struck by the paucity of acid-fast organisms on the skin, and have emphasized the importance of the bacillary load in the dermis and adjacent lymphatic nodes. Concentration methods of skin scrapings have given equivocal results, of doubtful significance when the organisms have not been positively identified. In the light of present suggestive findings, then, “close contact” need no longer mean “skin-to-skin contact”, but rather proximity or propinquity, a physical nearness sufficient for air-borne infective material from the nasal mucosa or from contaminated fomites to be brought into relation with an epithelial surface of a susceptible human being. It will embrace intrafamilial proximity as well as any contact in a community where the prevalence of leprosy reaches 1%.

The importance of these observations is far-reaching. Epidemiological investigations of the kind reported recently in the pages of *Leprosy Review* should be pursued with added vigour and precision. A simple and practical method for roughly determining the infectivity of the individual patient, namely by bacteriological examination of the nasal discharge, is now placed in the hands of the field worker reluctant to make use of an instrument for obtaining material from the skin or nasal mucosa. While histological examination of skin sections is still the indispensable arbiter of the presence of *Myc. leprae*, their morphology, and the tissue response they evoke, the administrative categorization of patients into "open" and "closed" may conveniently be decided on bacteriological examination of typical examples of nasal mucus. Contaminating acid-fast organisms obtained from the vestibule are never found agglomerated in globi. This examination, or a series of such examinations, will thus be of help in establishing or confirming the diagnosis and classification, and be useful in the ascertainment of contagiousness, cure, and relapse. It may provide as good evidence as the usual scraping of the septal mucosa, and in a manner more acceptable to the patient.

For some time, nasal washings from patients with untreated lepromatous or borderline leprosy have furnished viable bacilli for mycobacteriological investigation in the experimental animal.

Just as mycotic pathogens have been isolated from the surface of the skin, fomites, and dust, and thereafter cultured and identified, so it is not too much to hope that the day may not be far distant when *Myc. leprae* may be prised from the places where it lurks—in or on human tissues, in people known to have leprosy, or

perhaps even in those not under suspicion or with inapparent infections, in contacts, in carriers, and also possibly in extra-human reservoirs—and positively identified.

The role of fomites contaminated by discharge from the nasal mucosa or from open bacilliferous ulcers needs to be reassessed, and the length of time that *Myc. leprae* remains viable after release from the body should be determined by the footpad inoculation technique.

The source of viable organisms needs also to be investigated anew, for discordant findings abound; for instance, the unduly high frequency with which patients with tuberculoid leprosy apparently act as index cases for infections among contacts; the route of exit of viable organisms during the *poussées bacillifères* of reactional tuberculoid leprosy; the possibility that non-stainable or filterable forms of *Myc. leprae*, or L-forms, exist at certain stages in the life-cycle of an organism that has diverse taxonomic affinities; the apparently sudden burgeoning of mycobacterial activity when indeterminate macules become patently lepromatous, or when bacilliferous papules arise in normal skin.

There are thus many unsolved problems surrounding the emergence of *Myc. leprae* through the intact epithelial surfaces. The upper respiratory mucosa seems to hold pride of place; the gastro-intestinal mucosa plays a negligible role; the intact skin, including mucocutaneous junctions, is of debatable importance. Once again, the clinician, the epidemiologist, and the experimental microbiologist are called to collaborate in investigating these intriguing problems, questioning the "sacred cows" and enlisting the co-operation of the humble mouse.