Runyon (Tubercle 55[1974] 235-240), rather than specifying M. scrofulaceum, used the terms "scrofulaceum complex" or "M. avium-scrofulaceum complex" and suggested that a fluorescent antibody "may be useful for diagnosis of leprosy in the future."

Olaf K. Skinsnes
ALM Leprosy Atelier
Department of Pathology
University of Hawaii School of Medicine
Honolulu, Hawaii

Study of Subcutaneous Fat in Leprosy Patients

To The Editor:

A. Carayon in a recent paper (1) pointed out the high proportion of serious and contagious forms of leprosy, serious and widespread nerve damage, a very high percentage of serious and bilateral facial paralysis, and the high proportion of an inhabitual type of ulnar palsy at the wrist without nerve damage at the elbow in leprosy patients in Iran. He stated that the cold weather and nutrition are responsible for such epidemiologic and clinical findings.

I also examined many of these patients and fully agree with the concepts of A. Carayon who believes that, "the lipid balance seems to be an active factor to prevent the transmission of leprosy and that malnutrition interferes in the spread of leprosy." He also quoted that, "the various lipid diets in the northern and southern provinces of Iran may explain the differences in the spread of leprosy" and the "hypothesis is advanced that more than cold, variations in lipid diet in the north and south may explain the high prevalence in the north and the low in the south. Chemical studies of subcutaneous fat of the two groups are necessary to bring scientific proof and this study would be a good goal for the next year's program."

In regards to the last point, i.e., the study of subcutaneous fat, I indicated the importance of this work in many of my publications which I summarized recently (2). In this paper I discussed my concept on the relationship between pathogenesis of leprosy and autooxidation of lipids, peroxidation, antioxidants, tocopherols, unsaturated fatty acids, subcutaneous fat, as well as the biological antioxidant activity of diamino- diphenylsulfone, and the growth of M. leprae in animals with prooxidant diets (low in vitamin E and high in unsaturated fatty acids).

I believe that the study of subcutaneous fat recognized by Carayon and myself should be done in order to complete the study of the pathogenesis of leprosy.

Meny Bergel
Medical Director
Instituto de Investigaciones Leprologicas
Rosario, Argentina

REFERENCES

Leprous Myositis

To The Editor:

This is with reference to the paper entitled "A Histopathologic Study of Striated Muscle Biopsies in Leprosy" by J. C. Gupta et al., published in the JFL 43 (1975) 348-355.

I am a little disturbed by this paper which includes unclear and incorrect muscle pathology, and fails to take note of one earlier paper on this subject which described most of the changes that occur in the muscle and the neuromuscular endings in both tuberculoid and lepromatous leprosy.

While the authors have described and tabulated a lot of histopathological "changes," many of these are nonspecific and in fact frequent end-results of myopathies and denervation atrophies. Thus, in Figure 1, "Intramysial granulomas extending along
sarcoplasm" are mentioned, while all that we see is a poorly photographed and badly shredded section of a longitudinally cut muscle, bearing large and small mononuclear cells among the shreds of muscle tissue. These cellular clusters could be shown in a better section to be amongst the muscle fibers, probably constituting an endomysial exudate.

Figure 3 attempting to show "Extensive foam cell leproma" actually shows nothing but a few remaining atrophic muscle fibers amidst fatty tissue, which is the usual end-stage of any myopathy or atrophy. One fails to see how the diagnosis of a "leproma" can be accepted on mere hematoxylin and eosin staining. Similarly, Figure 5 shows groups of atrophic fibers (if this is a muscle of an adult subject), amidst fatty tissue, representing the late stage of an atrophic process.

Figure 4 probably represents the most glaring error of all, as we see clearly that an oval structure under the surface of a muscle fascicle is either a nerve twig or a muscle spindle, probably the former though this is not certain on account of the poor quality of the picture. The capsule bounding the oval entity is either the perineurium or the capsule of the spindle, probably the former. In any event this is certainly not a "tuberculoid granuloma" or a granuloma of any sort.

Figure 2 is the only picture which manages to illustrate what it attempts, namely a mononuclear cell cluster replacing a muscle fiber as seen in cross section.

The authors have not only exhibited unfamiliarity with muscle pathology but have failed to consult even basic text-books of muscle disorders or pathology, such as those by Adams, Denny-Brown and Pearson, 1963; Paul B. Hoeber Inc., New York; or by Walton and several authors, 1974, Churchill Livingstone, London.

The paper to which I wish to invite the pertinent attention of the above authors (and of other interested readers) is the one by Darab K. Dastur on "The Motor Unit in Leprous Neuritis: A Clinico-Pathological Study," published in Neurology - India 4 (1956) 1-27. This paper has been quoted in literature on neuropathology and muscle pathology but has been missed by most of the few recent writers on the subject of muscle in leprosy, and has been briefly reviewed by Dastur himself in 1) Bombay University Symposium on Leprosy, held in 1965 under the chairmanship of Dr. R. G. Cochrane, edited by N. H. Antia and D. K. Dastur, Bombay University Press, 1967; 2) Pathology of the Nervous System ed. J. Minckler, New York: McGraw-Hill Book Co., 1972, vol. 3.

Almost all possible changes that beset motor and sensory nerve endings and the muscle fiber, as seen in vitally stained whole-mounts of muscle and in variously stained paraffin sections are described and illustrated in over 40 clear photomicrographs. In addition, the paper presents detailed clinical features of sensory and motor status in 69 patients with leprosy, and also gives perhaps the first account of bacilli in Schwann cells in a lepromatous nerve.

The point of relevance at the present is the so-called "myositis" of lepromatous leprosy with which Gupta et al (1975) and most of the authors quoted by them seem to be overly concerned. In the above mentioned paper by Dastur (1956), where generally the flexor carpi ulnaris was biopsied, three of the six lepromatous cases showed interfascicular inflammatory exudates as well as acid-fast bacilli. In two of these the exudate as well as the M. leprae were in intramuscular nerves (Fig. 1), and in the third a bacillus was found between two muscle fibers.
Recently, Dastur (personal communication) reexamined the muscle sections of these lepromatous cases and failed to find any organisms within muscle fibers. About a fifth of the muscles from tuberculoid cases showed inflammatory exudates made up of large and small mononuclear cells, only four of these showing a Langhan's type giant cell in the intramuscular exudate (Fig. 2), and only in these cases was the term "granuloma" used. Similarly the term "myositis" was used with reserve and restricted to cases who showed extensive inflammatory exudation amongst muscle fascicles or fibers. Denervation atrophy of the muscles was the most frequent change encountered. As stated later by Dastur (1967), the muscle in leprosy is affected generally by damage to the related motor fibers in mixed nerve trunks and, less frequently, by an extension into the muscle of the inflammatory exudate around neurovascular bundles, i.e., an intramascular neuritis.

—Daya K. Manghani
Senior Scientist in Charge
Nerve Muscle Research Cell
Medical Research Centre
of Bombay Hospital Trust
12, Marine Lines
Bombay, India

Reply: In reply to Dr. Manghani’s comments we would like to state that the material studied included biopsies from apparently normal looking biceps muscles not showing evidence of atrophy or functional impairment. Various pathologic changes observed in such biopsy tissues are reported. Nerve fibers were not involved. Hence, we do not think that the muscle changes were the end result of damage to the related motor fibers and suggested them to be the result of extension of the granulomas into muscle tissue. Regarding his comments on photographs, we beg to differ from his opinion.

We are happy that he seemed so interested in our observations but are sorry to note that they upset him. We would only wish that he would undertake a similar study and then present his own findings, contradictory or supportive, of our observations. We thank him for mentioning the names of a few books on muscle disorders or pathology which are already familiar to us.

—D. K. Gupta
Reader in Medicine and Head
Department of Skin, V.D. and Leprosy
Medical College and Associated Hospital
Jabalpur, M.P., India