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Leprosy and Nutrition

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Of the many pathological sections of leprosy the most neglected are the nutritional. It is proposed to devote some attention to them in this paper.

Leprosy and Nutrition from the Epidemiology Point of View

According to Munro and Newman quoted by Rogers and Muir[1] the great prevalence of leprosy in the Middle Ages was based on a feeding system poor in the ingestion of meat and fresh vegetables. Also Keil[2] pointed out in Dutch Surinam the relationship between a diet of salted meat and fish and a predisposition to leprosy. Lampe[3] in Java found strong correlation between dried salted fish eating and leprosy. Varriel[4] noted that the eating of a large quantity of raw vegetables in Syria reduced the incidence and morbidity of leprosy. Hasselmann[5] in the Philippines noted a direct relationship between leprosy and the diet of fermented and dried fish, also in Burma Badger[6] pointed a probable relationship between nutrition and leprosy. Hutchinson[7] asserted all his lifetime leprosy depended on feeding with fish and affirmed that leprosy was a 'fish-eater's gout,' and ascribes the improvement in leprosy in the 16th Century to the increased consumption of green vegetables and the decrease of salty fish and meat. Bergel[8] interprets Hutchinson's theory nowadays as follows:— the ingestion of an excessive quantity of unsaturated fatty acids accompanied by a deficiency of tocopherol causes *in vivo* an increase of the auto-oxidative process which would provide a favourable ground for the growth of *M. leprae*.

Oberdörffer[9] assigns to the ingestion of some vegetables a definite role in the infection of leprosy, such as colacassia and agrostemona, foods which contain sapotoxins which have a deleterious action on the adrenals. Bergel[10] showed the inoculation of *M. leprae* in rats was helped by dietetic changes. These have assisted in other infections as Dubós[11] and Hedgecock[12] have shown the importance of lipids in the development of the experimental tuberculous infection. Scrimshaw, Taylor and Gordon[13] mentioned the nutritional factors as perhaps involved in the formation of antibodies, in the phagocytic activity, non-specific resistance factors, tissue integrity, state of the intestinal flora, balance of endocrines, interference with non-specific protective substances, destruction of bacterial toxins, etc., Niemeyer[14] pointed out that the diet could cause vitamin and proteinic deficiencies, disturbance of the electrolytic balance, anaemia, modify enzyme activity and the correlation between enzymes and cells.

Relationship between Diets and Chemotherapy

The author has used pro-oxidant diets in animal experimentation. Bergel[15], Eisman[16], Moore[17] found that the administration of DDS, the thiosemicarbazones, the isoniazids, and thiambutosine, in various amounts to a pro-oxidant diet (a semi-synthetic diet composed of casein, yeast powder, mineral salts, starch, and cod-

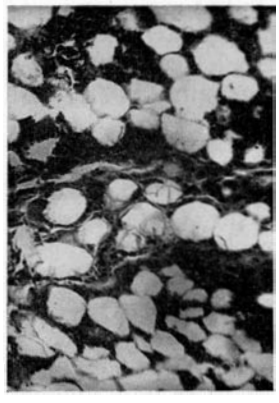

FIG. 1.
Perirenal fat of rat fed 4 months on pro-oxidant diet (Mag. 80 x)

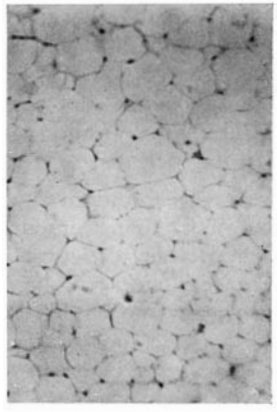

FIG. 2.
Perirenal fat of rat fed 4 months pro-oxidant diet with addition of 4–4[1] DDS at 0.5 per 1,000 (Mag. 80 x)

liver oil) avoids the formation of fuchsinophil ceroid pigment. This means an anti-oxidant biological activity of the anti-mycobacterial chemotherapies. Figs. 1 and 2 show the perirenal fat of a rat fed with a pro-oxidant diet. The therapy has avoided peroxidation of the deposit fat.

Bergel insists that the anti-oxidant diet should have a very low concentration of tocopherols (Vitamin E) and a more or less high quantity of unsaturated fatty acids. *M. leprae* is capable of reproducing itself actively in rats fed with pro-oxidant diets. Bergel verified the effect of a pro-oxidant diet, 10 of which were fed with semi-synthetic diet to which 15% of cod liver oil was added, 10 of which were fed on the same diet, but the 15% oil was rancid industrial coconut oil. The semi synthetic diet was composed of:

industrial casein	23.8 g.
mineral salts	3.0 g.
yeast powder	8.9 g.
corn starch	48.9 g.

The oils were kept under refrigeration and added to the dry mixture in the indicated proportion. Water was administered *ad lib.*

After 20 days under the above diet all the animals were inoculated with .05 ml. of a bacillary suspension prepared from triturated leproma of a young untreated patient. Part of the bacillary material was seeded in Lowenstein-Jensen medium at 37 °C and at room temperature, which did not produce development of bacillary colonies. The animals were maintained on the same diet for 7 months. Some died spontaneously and others were sacrificed at regular periods.

It was found in the experiment that only 1 bacillary development occurred which was a remarkable testicular atrophy in one animal. On the contrary the animals which received pro-oxidant diet did not present bacillary growth in the testes nor testicular atrophy. In previous experiments when pro-oxidant diets had been given in an early age of 20 to 22 days animal, there was a marked testicular atrophy and great bacillary development. In the present group which received coconut oil, there was a noticeable testicular atrophy in all, which presumably can be attributed to lack of Vitamin E.

The following chart (Fig. 3) shows bacillary growth in both groups of animals, the counts being made by the Hanks method.

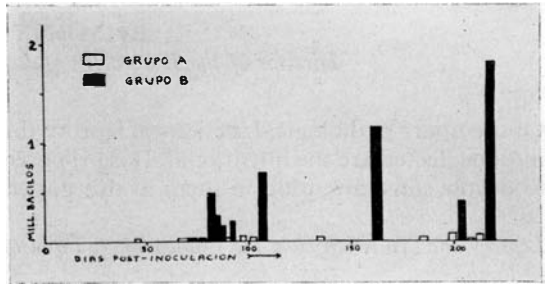

FIG. 3
Chart showing bacillary growth in both animal groups.

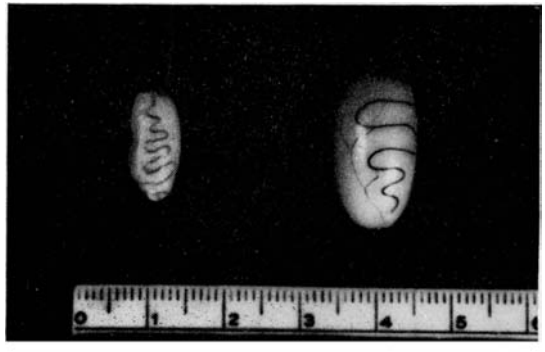

FIG. 4
This fig. shows the comparative size of the testes of both groups of animals.

Figs. 5, 6, 7 and 8 show the histology of some of the testes.

SUMMARY

From experiments with the diet of the experimental animal it appears that diets provoking Vitamin E deficiency and pro-oxidant diets given at an early stage promote the development and growth of *M. leprae* in the animals. It is suggested that the dynamics of anti-leprosy therapy could be partly explained on the basis of its relationship with nutritional factors.

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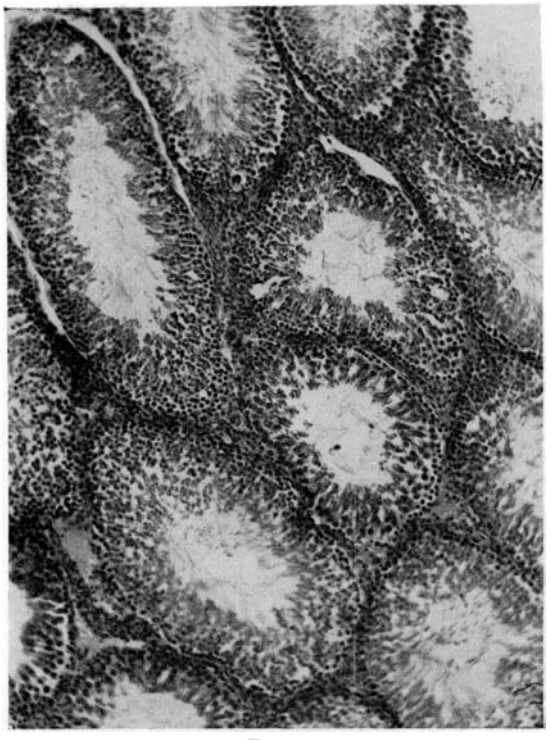

FIG. 5
Testis of 1st group at 210 days of inoculation. Normal structure (x 150).

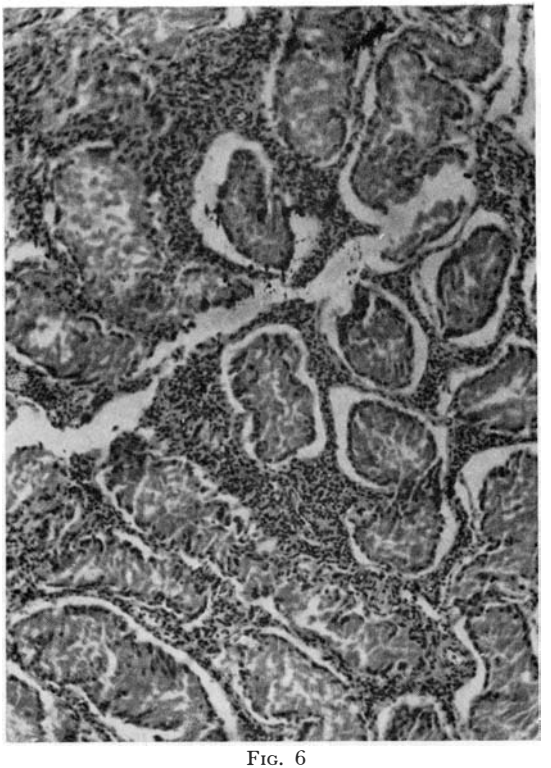

FIG. 6
Testis of 2nd group at 73 days of inoculation, with testicular degeneration and intertubular infiltration. (x 150).

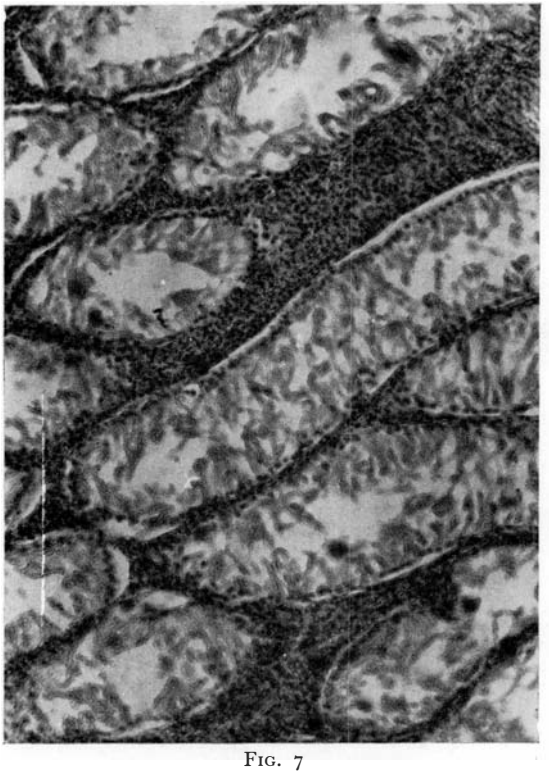

FIG. 7
Testis of 2nd group at 98 days from inoculation. Marked intertubular infiltration (x 150).

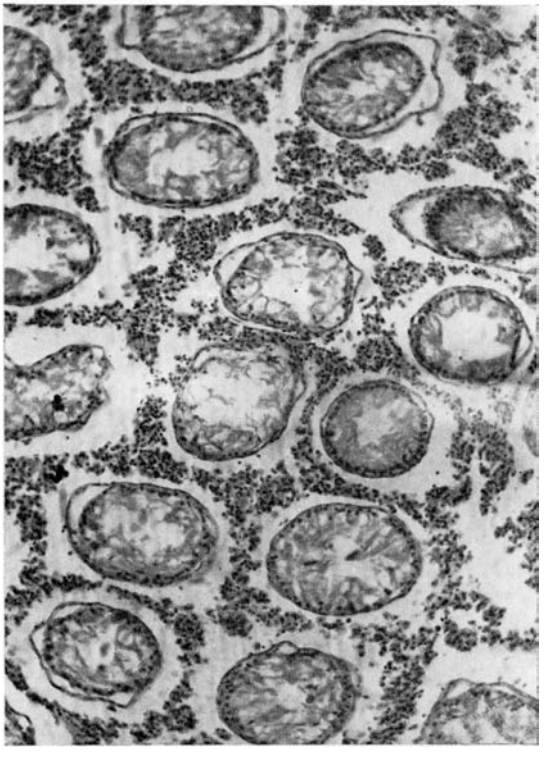

FIG. 8
Testis of 2nd group at 202 days from inoculation. Marked intertubular infiltration and testicular degeneration (x 150)

supplied the diets used in the present experiment, and also to Miss Jeanette Sperling, for her having translated his paper into English, and to Messrs. Remington Rand, Sud Americana for considerable general help.

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