pneumonitis, although the data are inadequate for probit analysis of relative potencies.

In previous studies (see Florey, 1952), as also in this, penicillin seemed to limit the multiplication of the organisms rather than to destroy them. When the lungs of apparently healthy mice at the end of the period of treatment were examined no lesions could be seen, but when they were homogenized and passed undiluted to untreated mice, the latter died with typical signs of mouse pneumonitis. Only one mouse in the whole series died after cessation of treatment, however, so the mice had clearly developed resistance (presumably mediated by antibody) against the organisms harboured in their lungs.

CONCLUSION

Mouse pneumonitis, a typical organism of the psittacosis-lymphogranuloma group, was more sensitive to ampicillin than to benzylpenicillin. Since ampicillin is non-toxic, acid-stable, and suitable for oral administration, it may be of value in treatment of human infections with organisms of the same group.

We are indebted to Beecham Research Laboratories for a sample of ampicillin and to Miss M. Mussett for statistical advice.

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Phlebotomus martini—a Probable Vector of Kala-azar in Kenya

The finding of a sandfly, probably *Phlebotomus martini*, naturally infected with a leishmania was reported by Heisch, Wijers, and Minter (1962). The strain has now been proved pathogenic to man and is therefore considered to be *Leishmania donovani*.

Further evidence incriminating *P. martini* as a vector of kala-azar has been obtained recently. Batches of wild-caught *P. martini* were fed on three kala-azar patients with positive blood cultures. Of the 58 sandflies which survived 72 hours after the infective feed, 28 were found to have leptomons in the anterior part of the midgut. A hamster inoculated intrasplenically with leptomons from the positive sandflies subsequently developed visceral leishmaniasis. Seventeen *P. schwetzi*, which also fed on the patients, remained negative.

During these experiments other specimens of *P. martini* were collected in the same area and examined for leptomons; of these, 19 were nulliparous, 8 in an advanced stage of the gonotrophic cycle, and 26 parous. All were negative, indicating that the infections in the flies used in the feeding experiments were derived from the patients and not previously acquired in nature.

These results show that *P. martini* is an efficient host for *L. donovani*, and that *P. schwetzi*, one of the few other species in the area that bites man, is not.

There seems little doubt that *P. martini* is an important vector of kala-azar in Kenya; transmission experiments will be attempted in due course.

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Medical Memoranda

Association of Peripheral Neuritis with "Autoimmune" Disease

There is some evidence to suggest that acute peripheral neuritis is due to an antigen-antibody disorder, particularly from the experimental work of Waksman and Adams (1956), and there is also some support for this view from clinical observations—for example, the response of the condition to steroid treatment. In the case of chronic peripheral neuritis, however, when diabetes mellitus, chronic alcoholism, toxic causes, and malignant disease have been excluded there remain a large proportion of cases of chronic polyneuritis for which no adequate cause can be found. In view of this diagnostic difficulty a report of two cases of chronic peripheral neuritis associated with autoimmune disease might be of interest.

One of these cases was associated with Hashimoto's disease, the other with polyarteritis nodosa and acute haemolytic anaemia. Peripheral neuritis as a feature of polyarteritis nodosa is well known, but the present report is believed to be the first of an associated haemolytic anaemia. Although polyneuritis has recently been described in association with carcinoma of the thyroid (Beardwell, 1961) and occasionally occurs in myxoedema, I have not been able to find an account of it associated with Hashimoto's disease.

CASE 1

A widow of 70 and mother of nine children had never been ill until March, 1960, when she noticed weakness of the legs, followed by pins-and-needles in her hands, which was progressive.

Examination showed bilateral foot-drop with slight wasting of the anterior tibial muscles. The marked weakness of the legs and slight weakness of the arms were both more severe distally. The tendon reflexes in the upper limbs were present and equal. The knee- and ankle-jerks were absent; plantar responses could not be elicited. There was severe impairment to all forms of sensation up to the knees, and slight impairment in the hands up to the wrists. The thyroid gland was diffusely enlarged and firm. There were no palpable lymph nodes in her neck.

Investigations revealed a normal blood count and a sedimentation rate of 40 mm. in one hour. Liver-function tests, plasma proteins, and electrophoretic strip were normal.