CORRESPONDENCE

To the Editor

LATENCY IN _PLASMODIUM VIVAX_ INFECTION

Sir,—Professor Sh. D. Moshkovsky has published recently a theory for the explanation of the appearance of short-term latency and long-term latency in _Plasmodium vivax_ malaria (Meditsinskaya Parasitologiya, 42, 393)*, and he has invited discussion.

In essence, Prof. Moshkovsky postulates the existence of 2 types of _P. vivax_ sporozoites, one which causes short-term latency infections and one which causes long-term latency infections. The normal course of events in infection with most strains of _P. vivax_ would result from an inoculation of a mixture of these 2 types of sporozoites. That is a short-term latency with symptoms at about 15 days due to the Type 1 sporozoites, followed 7–9 months later by a new invasion of the blood and symptoms due to Type 2 sporozoites which had up until then been latent. Thus the late invasion of the blood is due to a new type, not due to a relapse, even when preceded by a primary short-term attack.

Prof. Moshkovsky's views always command respect and attention but in my view this theory is not tenable. According to this theory, in strains of _P. vivax_ such as St. Elizabeth or Madagascar, infections in mosquitoes derived from the 7–9 month blood invasion would give rise only to long term latency infection and those derived from the early blood invasion would give rise only to short-term latency infections with no 7–9 month activity as the pure types would be selected.

But as we all know this does not happen, though it is somewhat embarrassing to have to admit that the fact that it does not happen is not recorded in the literature. Mr. P. G. Shute, however, has written to me to say that he has infected mosquitoes on numerous occasions from both relapses and long-term latency primary attacks and obtained normal short-term primary infections on feeding back to non-immunes. The fact that infections in mosquitoes derived from short-term primaries do in fact give rise to infections with both primary attacks and relapses is well known (e.g. Coatney, et al., 1950).

Also in nature the 7–9 month invasion postulated by Prof. Moshkovsky to be due to Type 2 sporozoites is the infection which maintains _P. vivax_ from year to year where transmission is seasonal. If Prof. Moshkovsky's theory were true, then only his Type 2 parasite would be transmitted as a pure infection and no short-term infections of the blood would occur. We know that this does not occur and that early primary attacks do occur in areas of seasonal transmission of _P. vivax_.

Prof. Moshkovsky’s theory is only tenable if it is also postulated that infection of mosquitoes with gametocytes of either type gives rise to sporozoites of both types. However, even then problems arise as it is known that long-term latency can be achieved by lowering critically the sporozoite inoculum (Garnham et al., work in progress), that is, according to Prof. Moshkovsky's theory only Type 2 sporozoites are injected in sufficient numbers.

Modifications of Prof. Moshkovsky’s theory are possible to cope with both these problems though quite what the pressure brought to bear to achieve the appearance of a new type in the mosquito would be, is difficult to envisage. Prof. Moshkovsky's paper, however, does not plead any such modifications and without them I do not believe the theory can withstand critical evaluation. Even with suitable modifications it seems to me very dubious, even though I have nothing better to put in its place.

*I am indebted to Prof. L. Bruce-Chwatt for a translation of this paper.

I am, etc.,

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REFERENCE


FURUNCULAR MYIASIS CAUSED BY LARVA OF _CORDYLOBIA ANTHROPOPHAGA_ IN IBADAN, NIGERIA

Sir,—In June 1974, a 4th year student of veterinary medicine at the University of Ibadan complained of a painful swelling on the lateral side of the right shoulder joint. He had felt intermittent intense itching pain on the swollen area 2 days previously. By the 3rd day he had noticed a discrete zone of inflammation around the painful area. The pain had also become continuous. By the 4th day, the pain had extended to most of the shoulder muscles and his axillary lymph nodes were enlarged. He had also observed a whitish speck of about 2 mm. in diameter in the centre of the inflamed zone.
The affected area revealed a discrete lesion of about 3 mm. in diameter situated on the lateral side of the right shoulder joint. In the centre was a whitish speck; when this was carefully removed with a sterile cotton wool, a small aperture was seen, through which protruded the last segment of a larva, which was expressed and later identified as the second stage larva of the adult fly Cordylobia anthropophaga. The wound was subsequently dressed and it healed 3 days later.

A visit was made to the student’s house. The building was surrounded by many tall trees among which were mango, orange and oil palm trees. The windows of his room were not netted and he remarked that he was usually disturbed by flies between 1600–1800 hours. A trap net was used and within a week, 5 specimens of C. anthropophaga were caught.

It is a well known fact that C. anthropophaga is usually attracted to objects, especially clothes, which have been contaminated with sweat, urine and faeces and they are stimulated to oviposit on them. In the case of this student, it is probable that he acquired the infection through his habit of exposing his underwear to dry on a line in his room. The tall trees surrounding the building obviously provided shade for the adult C. anthropophaga. These flies are known to prefer cool, shady areas (Zumpt, 1965).

There has been no published record in Nigeria of human furuncular myiasis caused by the larva of C. anthropophaga. Between January and December, 1973, Dipeolu (unpublished observation) found larval infestation in several dogs brought for treatment in one of the veterinary clinics in Ibadan. The rôle of this animal as an important reservoir of the infection must be stressed since untreated dogs may help to spread the fly to human habitations. The fact that the lesions caused by the larva of this fly usually heal on their own after a few days is probably responsible for the scarcity of published records in Nigeria. Only a few cases complicated by bacterial invasion reach the hospitals where the cause is more likely to be attributed to the bacteria because by that time the third stage would have dropped off.

We are, etc.,
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23 August, 1974

REFERENCES


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CONCOMITANT INFECTION BY TOXOPLASMA GONDII AND TRYPANOSOMA CRUZI

Sir,—Of late the study of concomitant infections in laboratory animals has become somewhat fashionable, and in Brazil the Schistosoma mansoni-Salmonella model, believed to be responsible for “typhoid fever of long duration” has been well investigated.

An entirely different model was reported for Toxoplasma gondii, chronic infections by this organism increasing resistance to Mengo virus, Listeria monocytogenes, Salmonella typhomurium, Besnoitia jellisoni, and even fungi and tumours. These results seem to support the concept of a “cellular-based system of resistance even among phylogenetically unrelated intracellular infections” (Ruskin, McIntosh and Remington, 1969).

In the light of these data one would, therefore, be tempted to assume that the ubiquitous toxoplasmosis is not only a rather unimportant infection (with the exception of its well-known, though uncommon, complications), but that it actually might protect human populations against other facultative or obligate intracellular organisms.

Recently, however, immunodeficiency in mice infected with Toxoplasma gondii has been reported (Strickland, Pettitt and Voll, 1973), although any good immunologist will be able to establish harmony between these apparently conflicting views.

Studies under way in our own laboratory also indicate that matters are somewhat more complicated: Albino mice were infected by the intraperitoneal route with 3–5 brain cysts of the “AS-28” strain of T. gondii. This parasite is well tolerated, and we have a number of animals in our colony which are alive, and well, 15 months after infection. Intraperitoneal multiplication with this strain is scanty, but cysts abundant in the brain. The maximum number of cysts compatible with survival seems to be about 4,000, average diameter of cysts is about 50 microns, and maximum size about 120 microns.

50 days after injection with Toxoplasma a group of animals was infected, also by the intraperitoneal route, with 3,000 blood forms of T. cruzi (“Brazil” strain). This size of inoculum ordinarily is compatible with 100% survival.

Parasitaemia reached a peak at 36 days after Trypanosoma infection, when median parasite counts were 4 times as high in the concomitant infections than in the controls, and, since this parasitaemia declined very slowly, the ratio between groups increased during the following weeks. The physical condition of the animals infected by both organisms also was more severely impaired, and 35% succumbed at various intervals.

Infection by T. cruzi also seems to have some effect upon chronic toxoplasmosis, and the frequency