CORRESPONDENCE

To the Editor

POTENTIATION OF PROGUANIL RESISTANCE BY SULPHONAMIDES

SIR,—Among the considerable literature which has accumulated in recent years on antimalarial sulphonamide treatment (cf. Richards, 1970) and on drug resistance (Peters, 1969), I have yet to find any reference to the significant observations of Bishop and McConnachie (1948, 1950), that treatment of Plasmodium gallinaceum with representative sulphonamides, such as sulphadiazine and sulphanilamide, rapidly potentiated the development of proguanil resistance in the absence of exposure to proguanil and before the appearance of sulphonamide resistance. Thurston (1953) using P. berghei, found that a sulphadiazine-resistant strain was cross-resistant to pyrimethamine, but showed no early resistance to proguanil. As pyrimethamine and proguanil show some cross-resistance, a more extended experimental investigation of the various sulphonamide derivatives now in antimalarial use, for possible ability to potentiate resistance to pyrimethamine and proguanil, would seem to be not only desirable but essential. Bishop (1962) has tested one such compound (Dapsone) and found no enhanced resistance to pyrimethamine or to proguanil, but a considerable number of other sulphonamides remain to be examined similarly. Experiments of this type were said to be under way, according to the WHO Technical Report Series No. 375 (1967), but I have seen no report of the results; the WHO Expert Committee on Malaria in their 14th Report (1968) recommended against sulphonamide combinations for mass treatment, presumably on the basis of toxicity, and therapy of individual cases only was advised.

The sulphonamide potentiation of resistance is probably a reflection of the considerable potentiative therapeutic synergism (Richards, 1970) which makes the sulphonamide—pyrimethamine combination so useful in the field.

I am, etc.,

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16 October, 1971

REFERENCES


SOUR MILK AS A PROTEIN SUPPLEMENT

SIR,—I was interested to read the letter of Sister Gowenlock et al. (Transactions, 65, 68) on their observation of the beneficial value of a lactose-free diet of eggs, sugar, oil and water. Lactose deficiency is frequently found amongst Rhodesian Africans. The administration of large amounts of fresh milk to these patients often precipitates diarrhoea which may lead to severe dehydration. Most of these patients can tolerate a diet containing eggs, glucose, oil and a protein supplement (casilan). We found that the administration of lactose to patients who had had an ileal resection resulted in severe diarrhoea, an increase in the amount of reducing substances in the stools and a significant lowering of the pH. We observed that a change to sour milk enabled these patients to tolerate larger quantities
of milk. The substitution of sour milk for fresh milk was also of value in providing adequate calories and protein for other malnourished patients.

I am, etc.,

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15 November, 1971

TRYpanosomal meningoencephalomyelitis with localization of T. BRUCEI IN THE Brain of a dog

SIR,—A 3-year-old dog was admitted to the veterinary clinic of the University of Ibadan, Nigeria, 4 months after importation from Britain. The owner stated that the animal had been progressively weak and lethargic for several weeks. On examination it was found to be severely emaciated and anaemic (packed cell volume 10.5%, haemoglobin 3.1 g.), but no parasites were found in blood smears. There was also depression, incoordination, circling and apparent blindness. The condition of the dog deteriorated and it was killed 24 hours later.

Examination of smears of the brain stained with Giemsa revealed large numbers of trypanosomes which were identified as T. brucei on the basis of their morphology. Trypanosomes were not found in smears made from the blood and other organs.

Microscopic lesions were confined mainly to the brain and spinal cord. There was a diffuse meningo-encephalomyelitis involving all sections of the brain and cord but most severe in the thalamic region. The inflammatory cells consisted of dense perivascular accumulations of lymphocytes, large reticuloendothelial cells, some plasma cells and occasional morular cells. In addition, there was marked proliferation of microglial cells and swelling of astrocytes. Neuronal reaction was minimal.

Numerous trypanosomes were present in sections of the brain. The parasites were present in the subarachnoid space and around blood vessels. They could be seen in routine haematoxylin and eosin sections, but their detailed morphology was best demonstrated in Giemsa-stained sections. They were indistinguishable from the blood forms of T. brucei.

Extravascular localization of T. brucei following systemic infections in some domestic animals and monkeys was well documented in the early part of the century (YORK, 1910; WOLBACH and BINGER, 1912 and PERUZZI, 1928). Later, and for almost three decades, this fact appeared to have been ignored. Recently, LOSOS and IKEDE (1970) and GOODWIN (1971) re-emphasized this important feature of trypanosomes of the brucei group. Further evidence of tissue localization of T. brucei (this time in the canine brain), is provided in the present communication.

We are, etc.,

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26 November, 1971

REFERENCES


DEVELOPMENTAL SITES OF Trypanosoma (Herpetosoma) SPP. IN THEIR FLEA VECTORS

SIR,—It has been stated (MOLYNEUX, 1970) that stercorarian trypanosomes of the subgenus Herpetosoma have developmental sites in their flea vectors in either the (1) rectum or (2) pylorus and small intestine. This dichotomy is not absolute and further examination of the references listed by MOLYNEUX (1970) (in addition to several others) reveals that, in all but 4 instances (Table), development takes place throughout much of the proctodaeum.