Fig. 3. 6-Mercaptopurine administered 72 hr. after transfer of normal cells incubated with *B. suis* antigen *in vitro* (full line). The same cell suspension was administered, without 6-mercaptopurine, to the controls

case. If it was administered 72 hr. after transfer, the transferred cells formed antibodies, although in low titres only (Fig. 3). Using a bacterial antigen, therefore, the crucial period for sensitivity to 6-mercaptopurine is 48–72 hr. after injection of the cells mixed with antigen *in vitro* ; this corresponds exactly to the inductive period, as determined in tissue cultures[4].

It was usually found during these experiments that the dose of 0·5 mgm. 6-mercaptopurine/100 gm., which normally is tolerated well, was lethal for young animals to which cells already forming antibodies were transferred (Fig. 2). This is probably due to summation of the effect of 6-mercaptopurine and the metabolic demands of the transferred antibody-producing cells.

The results show that 6-mercaptopurine is effective during the inductive phase only ; the mechanism of its inhibitory action supports the conception that processes during the inductive phase are associated with the synthesis of nucleoprotein. These results form part of a wider series of transfer experiments in which different types of inhibitors (for example, colchicine, ethionine) are used in an attempt to define exactly the conditions and metabolic processes of the inductive phase of antibody formation.

J. ŠTERZL

Division of Immunology,
Institute of Biology,
Czechoslovak Academy of Science,
Prague.
Oct. 20.

[1] Šterzl, J., Proc. Symp. Mechanism of Antibody Formation, Prague, 1959 (in the press).
[2] Dixon, F. J., Talmage, D. W., and Maurer, P. H., *J. Immunol.*, 68, 693 (1952).
[3] Berglund, K., and Fagraeus, A., Atti VI. Congr. int. microb., 2, 231 (1953).
[4] Šterzl, J., *Experientia*, 15, 62 (1959) ; *Folia Microbiol.*, 4, 91 (1959).
[5] Šterzl, J., *Folia Biol.*, 1, 193 (1955) ; 3, 1 (1957).
[6] Šterzl, J., "Mesenchymal Tissue during Immunisation and Infection" (Praha, 1954).
[7] Šterzl, J., and Holub, M., *Cs. biol.*, 6, 75 (1957) ; *Folia Biol.*, 4, 59 (1958).
[8] Schwartz, R., Stack, J., and Dameshek, W., *Proc. Soc. Exp. Biol. Med.*, 99, 164 (1958).

BIOLOGY

Myxomatosis in Britain

IN his article, Dr. C. H. Andrewes[1] directs attention to the contrast between the host/vector relationship in Britain and Australia. When we have acquired further information about the biology of the rabbit flea (*Spilopsyllus cuniculi*) and also other possible arthropod vectors, we may find that a somewhat different set of factors comes into operation in Britain between major epizootics.

After the initial outbreak of the virulent type of myxomatosis, when the rabbit population at Ashton, Peterborough, was at its lowest ebb, the few survivors which were caught showed a greatly reduced individual flea infestation. It might have been supposed that, owing to the number of dead rabbits from which the fleas had scattered in search of living hosts, the remaining healthy rabbits, especially any immune survivors, would become progressively more heavily infested. But this was not so. All survivors examined six months after the first great epizootic harboured at the most four or five fleas each, quite frequently no fleas at all, or fleas other than rabbit fleas. In the year preceding the outbreak 500 rabbits examined from the same area showed a 100 per cent infestation-rate with a mean per rabbit of approximately 70 fleas on the small sample counted. Three years later, when rabbits were becoming more numerous at Ashton, rabbit fleas were again plentiful, although no actual counts were made at this time.

It therefore seems possible that the chief vectors during a big epidemic may be different from those between, or right at the end, of a big epizootic. Mosquitoes spread myxomatosis, at any rate to a limited extent, in Britain[1] ; and it is probable that other winged vectors such as black fly (*Simulium*) are capable of transmitting the disease. During the period of reduced rabbit and flea population the winged vectors may well play the major part as agents of dissemination, and if this were so, the avirulent strains of myxomatosis would, between big epizootics, enjoy certain advantages similar to those described for these strains in Australia. When the rabbit and flea populations build up again, which they do fairly rapidly, the rabbit flea would assume once more its role as chief vector in Britain and consequently there would again be some evolutionary pressure in favour of a virulent virus.

MIRIAM ROTHSCHILD

Elsfield Manor,
Elsfield,
Oxford.

[1] Andrewes, C. H., Thompson, H. V., and Mansi, W., *Nature*, 184, 1179 (1959).

Glucose-6-Phosphate Dehydrogenase Deficiency Trait in Nigeria

WE were led to look for the presence of this trait in Nigeria because of the demonstration that 10 per cent of American Negroes are deficient in this enzyme[1] and the discovery that hæmolytic anæmia caused by certain drugs (for example, primaquine) develops only in the presence of this sex-linked inherited deficiency of red cell glucose-6-phosphate dehydrogenase[2].

We have applied the rapid screening test as described by Motulsky[3] to 204 male Nigerians of