lodged in the organs or collected from the circulating blood, so that a greater percentage is capable of reproduction. Also, the smaller number of dead cells and decreased amount of cellular debris in these cell suspensions may decrease the possibility of deleterious interaction with partially damaged cells.

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Primary Tissue Phase of *Plasmodium berghei* in Different Experimental Hosts

IN a previous communication[1], we reported the finding of pre-erythrocytic tissue schizonts of *P. berghei* in the liver of an experimentally infected young hamster. The forms observed were few in number, involving examination of many hundreds of stained liver sections. The sporozoites used in the former experiments were derived from experimentally infected *Anopheles quadrimaculatus*. In this laboratory vector only small numbers of viable sporozoites reached the salivary glands. The introduction of *Anopheles stephensi* as an experimental vector of *P. berghei*[2] has greatly facilitated and advanced our work, for in this mosquito species invasion of the salivary glands by very large numbers of sporozoites takes place with regularity after exposure to gametocyte carriers and maintenance at 21° C.

Using techniques of homogenation of infected *A. stephensi* or the trituration of infected salivary glands and midguts, we have been able, by intravenous and intraperitoneal inoculation, to inject large numbers of viable sporozoites, and we have easily found pre-erythrocytic growth forms of *P. berghei* in parenchyma cells of the liver of experimental rodent hosts.

Tissue schizonts have been found in liver sections stained in Giemsa colophonium[3] in experimentally infected laboratory-bred tree rats (*Thamnomys surdaster*), in hamsters (*Mesocricetus auratus*) in young albino rats[4,5], and in white mice.

The inoculation of 500,000–750,000 sporozoites in each of these mammalian hosts of *P. berghei* has produced four to seven tissue schizonts per histological section (1 cm × 0·5 cm in diameter and 4μ in depth). The pre-erythrocytic schizonts show similar morphological features in the different hosts. Growth forms of 18 h, 22 h, 36 h, 48 h, and mature schizonts 51–52 h old were found in the different experimentally infected animals. A search for the early stages of 3–18 h is now in progress.

The regular experimental production of pre-erythrocytic tissue stages of rodent malaria under well-defined laboratory conditions may facilitate research in the chemotherapy and chemoprophylaxis of mammalian plasmodial infections as well as in other fields of malaria research.

A detailed description of the exo-erythrocytic development of *P. berghei* and the techniques involved will be published shortly.

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Virus Aetiology for Down's Syndrome (Mongolism)

FOR many years now, we have been working on the epidemiology of Down's syndrome (mongolism) in the State of Victoria, Australia, and have charted its occurrences during 1942–64. Peaks of incidence, of 2-year duration have been recorded at 5–7 year intervals from 1942 until 1957 and, as a result of this, a further peak of occurrence for this congenital anomaly was forecast for 1962–63. This was the first time ever that such a forecast had been able to be made and, in fact, this eventuated[1,2]. On the basis of our original findings[1,2], we had postulated a hypothesis of an infective virus, of long incubation, affecting mostly, but not exclusively, the ovum of the ageing mother, either directly or through some immunity pattern. Our reasons for this were not only the perception by one of us (A. S.) of a possible clinical relationship between the exposure of the mother to infective hepatitis prior to conception, but also the epidemiological findings that cases of mongol births clustered significantly in time and place, that urban peaks of annual incidence were in every case greater than rural peaks (higher contact rates), and rural peaks followed on

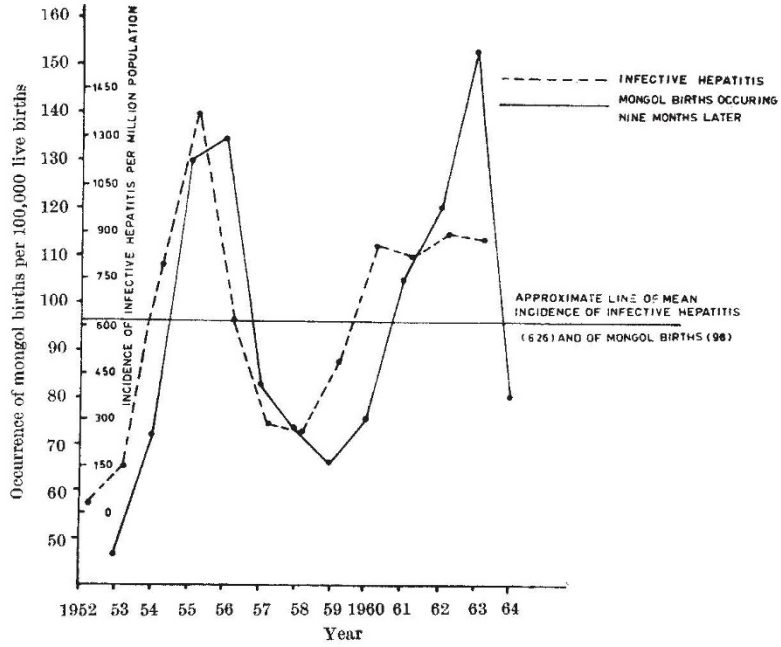

Fig. 1. Annual incidence of infective hepatitis in Melbourne, and of mongol births 9 months later for the period 1952 to 1964