Rajalakshmi, K. & Srikantia, S. G. (1980). Cu, Zn and Mg content of breast milk of Indian women. *American Journal of Clinical Nutrition*, **33**, 664-669.
Underwood, E. J. (1977). Trace elements in human and animal nutrition. (4th edit.) New York: Academic Press.
WHO (1973). *Trace elements in human nutrition.* Report of WHO expert committee. WHO technical report series no. 532, Geneva.

Accepted for publication 6th August, 1982.

TRANSACTIONS OF THE ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE, VOL. 77, NO. 2, 1983

Joint Meeting of the Transvaal Section of the Royal Society of Tropical Medicine and Hygiene and the Parasitological Society of Southern Africa, Johannesburg, July 1982.

Summaries of Papers

Tick-bite fever—severe cases

J. H. S. GEAR
State Health Department of Southern African Institute for Medical Research

Tick-bite fever, the form of tick typhus occurring in southern Africa caused by *Rickettsia conori* var. *pipjeri* transmitted by ixodid ticks, is usually a mild disease in children and young adults, but in middle-aged and elderly patients it may follow a severe course and if not treated early may have serious complications and occasionally may be fatal.

Three cases illustrative of the severe form of tick-bite fever were described. All began with a flu-like illness, with a feeling of tiredness, muscle pain and headache which became severe. All developed a maculopapular rash on about the fourth day of illness which became profuse and haemorrhagic with petechiae in the skin and oedema and, in two of the patients, gangrene of the fingers and toes. All had leucocytosis with, in two of the patients, a marked neutrophilia and a marked thrombocytopenia and the presence of fibrin degradation products in the blood. All developed signs of marked involvement of the brain and marked disorder of liver and kidney function leading to kidney failure and the need for treatment in the Intensive Care Unit. One of the patients died but the other two responded to tetracycline treatment and slowly recovered and were discharged from hospital relatively well. In each case the infection was presumably transmitted to the patients by ticks from their dogs which were allowed inside and to sleep on the patient's bed.

From these and other instances it is clear that if tick-infested dogs are allowed indoors, one or more members of the family will, sooner or later, suffer from an attack of tick-bite fever. Infections transmitted by dog ticks tend to be more severe than those acquired in the bushveld and recent serological tests have indicated that there are antigenic differences between suburban and bushveld strains.

Rift Valley fever virus in southern Africa

B. M. MCINTOSH
Arbovirus Research Unit, National Institute for Virology, Sandringham, Transvaal, South Africa

Rift Valley fever virus is widespread in southern Africa where it is apparently enzootic on the temperate plateau as well as the subtropical coastal region. On the plateau it has caused severe, widespread, but infrequent, epizootics and some human infection. During outbreaks on the plateau the virus has been isolated from three *Aedes* species and 22 times from *Culex theileri*. The latter mosquito is very prevalent, feeds readily on domestic ungulates and proved to be the most efficient experimental vector from among the plateau species. For many years it has been difficult to identify the main vector in the coastal region of Natal because of the sporadic nature of bovine infection. However, an opportunity arose in 1981 when a localized outbreak occurred in a herd of 250 cattle, during which four deaths and 13 abortions occurred over a period of three months. Attempts to isolate virus from mosquitoes resulted in one isolation from *Cx neavei* and seven from *Cx zombaensis*, the most prevalent species. laboratory tests showed *Cx zombaensis* to be an efficient vector. Because Rift Valley fever is rarely isolated from mosquitoes in the absence of domestic ungulate infection, the possibility of phlebotomines as maintenance hosts was investigated. Attempts to isolate the virus from 8575 wild-caught phlebotomines were unsuccessful, and 252 sentinel hamsters exposed for 103 days in animal burrows harbouring phlebotomines, as well as in other phlebotomine habitats, failed to become infected. Antibody surveys among burrow-dwelling mammal species revealed positively reacting sera in porcupines, hyrax and meercats, but not in ground squirrels and springhares.

Congo virus infection in southern Africa

R. SWANEPOEL, J. K. STRUTHERS AND G. M. MCGILLIVRAY
National Institute for Virology, Sandringham, Transvaal, South Africa

Crimean-Congo haemorrhagic fever had not been recognized in southern Africa until February 1981. On the 14th of that month, a 13-year-old schoolboy, after attending a nature camp near Bloemhof in the Transvaal, was taken ill with severe headache and chills and a tick later identified as a species of *Hyalomma* was found attached to his scalp. On 17th February he developed a haemorrhagic condition and in spite of treatment to combat this, died on 19th

Continued on page 207

Marr, J. J. (1977). Antileishmanial effect of allopurinol. II. Relationship of adenine metabolism in *Leishmania* species to the action of allopurinol. *Journal of Infectious Diseases*, **136**, 724-
Nelson,

Pfaller, M. A. & Marr, J. J. (1974). Antileishmanial effect of allopurinol. *Antimicrobial Agents and Chemotherapy*, 5, 469-472.

Accepted for publication 20th August, 1982.

Continued from p. 203—South African Meeting

February from profuse gastro-intestinal bleeding. Virus was isolated from his blood and identified as Congo virus. In follow-up studies, antibodies to Congo virus were detected in 5/74 sera from the staff and their families living at the nature reserve. Antibodies were also detected in the sera of 109/170 cattle and 74/270 sheep from surrounding farms and 4/10 sera from hares trapped in the nature reserve. Most of the adult ticks (2964/3037) collected at the veld school and nature reserve were species of *Hyalomma* with *H. truncatum* and *H. marginatum rufipes* occurring in a ratio of 3:1. Congo virus was isolated from 21/120 of the tick pools, the infection rate being similar in the two *Hyalomma* species. A number of eland antelope in the reserve died in the following September; antibodies were detected in their sera and virus was isolated from the ticks collected from them but their deaths could not be definitely ascribed to Congo virus infection.

Serological studies of sera collected between 1977 and 1979 from various sites in South Africa revealed that 28/200 sera collected from five species of hare were positive for Congo virus antibodies, thus confirming the presence of this virus in all four provinces.

Haemorrhagic fevers of Central Africa

D. I. H. SIMPSON
PHLS Centre for Applied Microbiology and Research, Porton Down, Salisbury, Wiltshire, UK

Haemorrhagic fevers of viral origin, notably smallpox and yellow fever, have been recognized for centuries. During the past 40 years a number of other viruses causing haemorrhagic disease have come to light, notably Rift Valley fever, Congo-Crimean haemorrhagic fevers, Argentinian and Bolivian haemorrhagic fevers, Lassa, Marburg, Ebola and Korean haemorrhagic fevers.

Yellow fever maintained in a sylvan cycle involving monkeys and forest-dwelling mosquitoes remains an important cause of haemorrhagic disease in Africa.

Rift Valley fever was recognized as causing large epizootics involving sheep and cattle associated with epidemics in humans. These were reported from Kenya and regularly from South Africa. More recently it has caused devastating epizootics in Sudan and Egypt, involving cattle, sheep, camels and water buffaloes and was associated with an estimated 20,000 human infections with at least 90 deaths.

Crimean haemorrhagic fever, first recognized in the Crimea and transmitted by the tick *Hyalomma marginatum marginatum*, is now known to occur throughout East and West Africa and recently has been responsible for outbreaks inolving man with a high fatality rate in Iraq and the United Arab Emirates.

Lassa fever, first recognized in Lassa in Nigeria where it was responsible for an outbreak with a high death rate, is now known to be widespread in West Africa. The virus appears to have a natural cycle in rodents and, in particular, *Mastomys natalensis* which, when infected at birth, remains infective during its lifetime, excreting virus in urine. Transmission to man occurs from close contact with rodents.

Marburg virus disease, first recognized in an outbreak in Germany in 1967, was traced to direct contact with the blood and tissues of vervet monkeys imported from Uganda. Marburg was next reported in February 1975 when a young Australian student contracted the disease while hiking in southern Africa. He died shortly after his admission to the Johannesburg Hospital. His companion and a nursing sister who attended them both also contracted the disease but both recovered. An outbreak also occurred in Kenya in January 1980.

Between June and November 1976 outbreaks of severe haemorrhagic disease occurred in southern Sudan and north-eastern Zaire, with a very high death rate. The aetiological agent was found to be morphologically identical to Marburg virus but antigenically distinct and was named Ebola virus. The second outbreak occurred in the same area of southern Sudan in August and September 1979.

The ecology of both Marburg and Ebola virus still remains to be elucidated.

The use of specific therapy in the form of convalescent plasma has been successful in some cases.