Elsevier has created a Monkeypox Information Center in response to the declared public health emergency of international concern, with free information in English on the monkeypox virus. The Monkeypox Information Center is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its monkeypox related research that is available on the Monkeypox Information Center - including this research content - immediately available in publicly funded repositories, with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the Monkeypox Information Center remains active.
Re-emergence of human monkeypox in Zaire in 1996

V B K Mukinda, G Mwema, M Kilundu, D L Heymann, A S Khan, J J Esposito, and other members of the Monkeypox Epidemiologic Working Group*

Human monkeypox is a systemic exanthem, resembling smallpox, that occurs as a sporadic zoonosis in rural rainforest villages of western and central Africa. The disease is caused by an orthopoxvirus, which is transmitted to human beings by handling infected animals; serosurveys have implicated squirrels (Funisciurus and Hylomyscus spp) as the probable reservoir. Secondary human-to-human transmission through eight members of his clan. During this time, monkeypox infections also occurred in other families living together and in a few clans in nearby villages, raising the possibility of other introductions of human monkeypox into the population.

Monkeypox was confirmed in 11 clinically suspect cases from crusted scabs, vesicular fluid, or serum collected from July to September, including three pairs of samples representing secondary contact cases in separate households. Virus-specific polymerase chain reaction (PCR) amplifications of genes for the monkeypox virus haemagglutinin (HA) and tumour necrosis factor receptor (TNFR; unpublished data) were positive for three of four available scab samples, and monkeypox virus was isolated in culture from two of the PCR-positive specimens. In addition, western blot assay showed orthopoxvirus genus-specific IgG in ten different patient sera, and an experimental enzyme-linked-indicator serum assay that used orthopoxvirus antigen peptides showed monkeypox-specific IgM in five of six sera tested.

The present cluster of cases constitutes a re-emergence of human monkeypox on a scale greater in magnitude than the approximate 65 annual cases previously indicated for Kasai Oriental, Bandundu, and Equateur regions from 1981 to 1986, and it contains a more extensive occurrence of person-to-person transmission than previously recognised. The extent of the outbreak in K atako-Kombe, which reported no cases during the previous surveillance, and the incidence of disease among household contacts, challenge previous modelling studies that suggested prolonged episodes or sustained cascades of transmission of human monkeypox would be unlikely even after smallpox vaccination, which is protective, ceased. Alternatively, the events may represent multiple introductions into the same population because of increasing encroachment of larger populations into the primary habitat of animals in this and other areas of Africa. Because sequence analyses have indicated that Zairian monkeypox strains have not diverged greatly from the first isolate from the area in 1970 (figure) and monkeypox and variola (VAR) viruses are independently evolved species, notions of monkeypox virus mutating into variola virus are unfounded.

In light of the 1996 episode, an international team coordinated by WHO, Centers for Disease Control and Prevention, and the Zairian Ministry of Health began an investigation in February, 1997, to evaluate the outbreak and determine current risk factors for infection. More specific rapid diagnostic assays should enable more precise monitoring of fluctuations in the virus and epidemiological patterns of this zoonosis as changes occur in human demographics, sanitary practices, and reservoir animal distributions.

*Member at Médecins sans Frontières Belgique, Zaire: H Koen.

** Members at Institut National de Recherche Biomédicale, Kinshasa, Zaire: M Delfi, J J Muyembe-Tamfum. Members at World Health Organization: T F K wetemina, A M oudi. Members at Centers for Disease Control and Prevention, National Center for Infectious Diseases: L M angindula, V N Loparev, J M Parsons, D L Jue, T W Crews, J C Knight.

Vol 349 • May 17, 1997 1449
Glutathione S-transferase theta 1 (GSTT1) gene defect in myelodysplasia and acute myeloid leukaemia

T Basu, R E Gale, S Langabeer, D C Linch

Approximately 30% of cases of myelodysplastic syndrome (MDS) culminate in acute myeloid leukaemia (AML). To test whether patients with MDS (46% compared with 16% in a control group) have a greater frequency of null genotypes in GSTT1, we measured frequency in a large group of controls. GSTT1 null genotype frequency (table). There was no suggestion of any difference in frequencies within the different MDS groups and our study are not clear. It is possible that there are major differences in the pathogenesis of MDS in the two countries, although this would be surprising. However, a potential problem in surveys of the frequency of the GSTT1 null genotype in a given disease is the fact that the frequency of this genotype varies markedly between racial groups, being particularly high in some Asian populations.

Chen and colleagues reported on the frequency of null genotypes for the glutathione S-transferases GSTM1 and GSTT1 genes showing a significantly increased frequency of the null genotype for the GSTT1 gene in patients with myelodysplastic syndrome (MDS). These enzymes play a role in the metabolic pathway for carcinogens and it is important for Chen et al’s findings to be confirmed. We determined the frequency of the null genotypes in a large group of controls (haematologically normal UK laboratory staff and UK general medical outpatients) and patients with primary MDS. We found no significant difference in frequency between controls and patients for either GSTM1 (odds ratio 0.89 [95% CI 0.5–1.43]) or GSTT1 (odds ratio 0.72 [95% CI 0.4–1.34]) null genotype frequency (table). There was no suggestion of any difference in frequencies within the different MDS subgroups. We also looked at eight cases of secondary MDS and found 4/8 to have the GSTM1 null genotype. The reason for the discrepancy between our findings and those of Chen are not clear. There is the possibility of racial heterogeneity in studies from the USA, causing skewed results but other possibilities include, for example, different causes of MDS in different countries.