atomic and free radical species which are directly and indirectly responsible for the formation of nitrogen oxides. Detailed investigations of the mechanism of catalytic action are in progress.

Flames in several commercial firing systems examined have also been found to respond to catalysts. Thus carbon formation can be inhibited or promoted, and nitrogen oxides reduced, by suitably positioning catalysts in the flames. The magnitude of these effects, however, is somewhat less than in the laboratory flames. We are currently developing suitable catalyst systems for field applications.

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BIOLOGICAL SCIENCES

Transmission of Kuru from Man to Rhesus Monkey (*Macaca mulatta*) 8½ Years after Inoculation

Neurological disease has appeared in a female rhesus monkey (*Macaca mulatta*) following an asymptomatic incubation period of 8 years and 5 months after inoculation intracerebrally (i.c.) and intravenously (i.v.) with a 10% suspension of brain tissue from a human kuru patient. Clinical signs were remarkably similar to those observed in patients naturally affected with kuru and in sub-human primates in which the disease has been experimentally induced by inoculation of the kuru virus. Histopathological examination of the brain of this rhesus monkey has confirmed this first successful transmission of kuru to an old-world monkey. Previously the chimpanzee and four species of new-world monkeys (spider, *Ateles*; capuchin, *Cebus*; squirrel, *Saimiri*; and woolly, *Lagothrix*) have been found to be susceptible to kuru.

In August 1963, one chimpanzee (A2), 5 rhesus, 4 cynomolgus and 3 African green monkeys were inoculated i.c. (0.2 ml.) and i.v. (0.3 ml.) with a 10% suspension of brain tissue from kuru patient Enage. Thirty months after inoculation chimpanzee A2 developed experimental kuru and was killed in the terminal stages of disease 4 months after onset. During subsequent months (between 1 month and 62 months after inoculation) 1 rhesus (16L), the 4 cynomolgus and the 3 African green monkeys died of intercurrent infections without signs of neurological disease; histological examinations by light and electron microscopy revealed no pathological lesions in the central nervous system. The remaining 4 rhesus monkeys remained clinically well until January 1972 (101 months after inoculation) when one (11L) was noted to have occasional tremors and locomotor ataxia. The animal climbed with reluctance and caution and became withdrawn and docile. Her hair coat became rough and there was piloerection over the entire body. Neurological signs became progressively worse and one and a half months after onset she developed clonic jerks of all four limbs and trunk and almost continuous coarse generalized tremors. Two and a half months after onset, although alert to her surroundings, she lay down on her side and had to be fed by hand. She was killed at this advanced stage of disease. At no time during clinical illness was there any fever nor were significant changes noted in haematological and serum chemistry values.

At necropsy the animal was thin with scant subcutaneous and omental fat. The brain was firm and, on cutting, a blanching of the grey matter was noted. There were no other gross pathological changes.

Preliminary histological examination of the brain by light and electron microscopy revealed extensive neuropathological lesions restricted to the grey matter. There was moderate to severe status spongiosus of the cerebral cortex and basal ganglia, most severe in the deeper layers of the cortical mantle and less extensive in the dentate nucleus of the cerebellum (Fig. 1). In all areas examined there was marked intraneuronal vacuolation, loss of neurones and astroglial proliferation and hypertrophy.

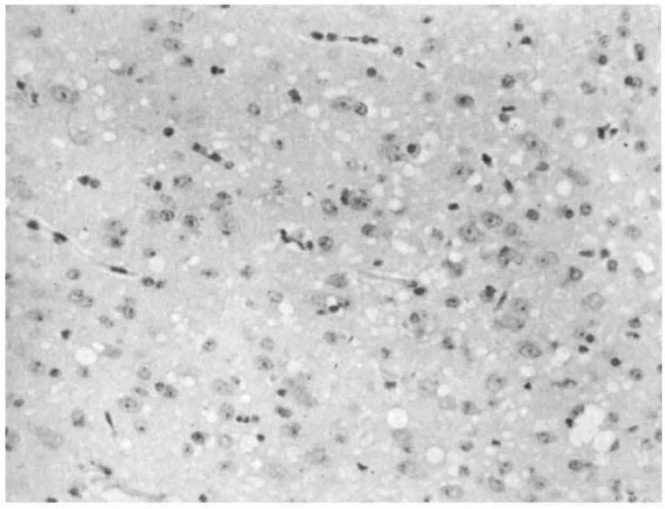

Fig. 1 Status spongiosis in the cerebral cortex of a rhesus monkey (11L) dying with kuru. In the areas of spongiform alteration there is neuronal loss and gliosis. Haematoxylin and eosin stain.

To date Creutzfeldt-Jakob disease, which resembles kuru in both the neuropathology of its cellular lesion and in many properties of its virus, has shown the same species specificity as kuru except for its failure thus far to cause disease in any of the old-world monkeys inoculated. However, none of the animals inoculated with brain suspensions from Creutzfeldt-Jakob disease has yet been observed over asymptomatic incubation periods as long as the 8.5 years required for disease to appear in this kuru-affected rhesus monkey. Rhesus monkeys inoculated with C-J disease virus 42 months ago are still under observation and remain well.

It is of interest that the two spongiform encephalopathies of animals have been transmitted recently to old-world monkeys: scrapie to the cynomolgus monkey[1] and mink encephalopathy to the rhesus monkey[2]. In view of the rapid decrease in the incubation period of kuru in the chimpanzee and some new-world monkeys on serial passage, it seems likely that the host range may be altered by serial passage in different hosts, even perhaps on blind passages in hosts not yet demonstrated to be susceptible.

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