multiply in the face of contact inhibition at 32° C but not at 39° C.

Having isolated several such temperature sensitive cells they assayed other parameters at 32° C and 39° C. As expected, the cells, which have SV40 T antigen at both temperatures, grow to high saturation densities at 32° C but only to low saturation densities typical of untransformed cells at 39° C. At 32° C the temperature sensitive cells are able to form colonies when plated on top of confluent monolayers of untransformed 3T3 cells, but at 39° C colony formation in these conditions is severely impaired and little greater than that observed with untransformed cells. Moreover, although the temperature sensitive transformants at 32° C and 39° C require for growth much less serum than do untransformed cells, they stop making DNA in response to cell with cell contact at the higher temperature but continue to make DNA when in equally dense cultures at 32° C. Preliminary experiments also apparently indicate that the ease with which these cells can be agglutinated by wheat germ agglutinin is temperature sensitive; at 39° C they are less susceptible to agglutination than at 32° C.

One way to account for all these properties is to suggest that the transforming gene of the resident SV40 genome(s) carries a temperature sensitive mutation. This is unlikely to be the case, however, for the SV40 virus rescued when the temperature sensitive transformants are fused with permissive BSC-1 monkey cells is apparently wild type, not mutant. The temperature sensitive mutation in these cells which affects the transformed cell phenotype seems therefore to be in a cellular gene(s) and the finding that these cells cannot be retransformed at 39° C by infection with multiplicities of up to 200 SV40 particles per cell supports this view.

In the same issue of the *Proceedings* (*ibid.*, 152), Smith, Gelb and Martin report a most unexpected finding with BALB/C cells abortively transformed with SV40 virus. From DNA hybridization experiments they conclude that cells of two of three lines of abortive transformants, cells which after infection by SV40 had only transiently behaved as if transformed and had then resumed the untransformed cell phenotype, contain about five copies of the complete SV40 genome per diploid cell genome; for comparison stable SV40 BALB/C 3T3 transformants contain only two copies of SV40 DNA per cell. To convince any sceptics of the validity of their conclusion Smith *et al.* could always fuse cells of these three lines with monkey cells and show that SV40 virus can only be rescued from the two lines which contain SV40 DNA.

LYMPHOMAS

Monkey Models

from our Medical Virology Correspondent

A RECENT report from the University of California National Centre for Primate Biology (Stowell *et al.*, *Lab. Invest.*, **25**, 476; 1971) records the largest ever outbreak of simian malignant solid lymphomas in twenty-four adult rhesus monkeys. This outbreak occurred over a period of 25 months and the attack rate was eighteen cases per 1,000 adult rhesus monkeys. This astonishing incidence—the reported occurrence of malignant lymphomas in non-human primates is very rare—together with the remarkable similarity to Burkitt lymphoma in man, may provide a useful model for the experimental study of human lymphoma. There are also several other intriguing features. No single potential or actual carcinogenic factor was identified in this outbreak although three herpes virus-like agents have been isolated. Ten of the twenty-four affected monkeys, however, had been involved in experiments with malarial parasites and subjected to exposure to X-irradiation.

The possible relation between hepatitis associated with Australia antigen and the pathogenesis of primary liver cell cancer in man has been previously discussed (see *Nature New Biology*, **232**, 100; 1971). More recent information is now available. Persistent carriage of Australia antigen is common in patients with a defective immune response, for example, lymphocytic

leukaemia, chronic renal failure and lepromatous leprosy. There is also epidemiological and experimental evidence for immunological changes, including depression of the immune response in malaria.

A. J. Zuckerman (*Brit. Med. J.*, **1**, 49; 1972) has proposed that the high frequency of carriage of Australia antigen in the serum of apparently healthy people living in the tropics may be related to this immunological phenomenon coupled with repeated biting by blood-sucking insects. This hypothesis may be extended further because an altered immune reactivity in malaria may also affect the outcome of chronic infection with a virus by allowing the virus to become frankly oncogenic and possible by interfering with the immune reaction to neoplastic cells. Indeed, Salaman *et al.* (*J. Gen. Microbiol.*, **59**, 383; 1969) and Wedderburn (*Lancet*, **ii**, 1114; 1970) demonstrated an increased incidence of malignant lymphoma caused by murine oncogenic viruses in mice infected with *Plasmodium berghei yoelli*.

A similar relation might exist between chronic infection with the serum hepatitis virus and the subsequent development of hepatoma (A. J. Zuckerman, *loc. cit.*), and it is of interest that an association of Burkitt's tumour and holoendemic malaria has also been suggested by Kafuko *et al.* (*East Afr. Med. J.*, **46**, 414; 1969). The possibility therefore that sub-human primates might prove a useful model for the study of lymphomas, hepatoma and other malignant tumours presents an intriguing and exciting challenge.

Radio Observations of Cygnus X-1

CONTINUING observations of Cyg X-1 at radio frequencies are being carried out with the Westerbork array. The figure here shows results obtained by L. L. E. Braes and G. K. Miley during 1971, which are reported in next Monday's issue of *Nature Physical Science* (February 21).

The sudden appearance of the source in April 1971, and its subsequent

roughly constant flux, are confirmed by independent observations carried out by the NRAO group at 2,695 MHz (the Westerbork results were obtained at 1,415 MHz). The accumulation of data has also enabled Braes and Miley to obtain an improved estimate of the position of this radio source, which agrees well with those of the X-ray source and the star HDE 226868.