Oncoviruses and cell membrane antigens

from Robin Weiss

THE presence of envelope glycoproteins of oncoviruses (the new name for oncornaviruses (Fenner *Virology* **71**, 371; 1976)) on the surface of normal cells has been known since the recognition of murine G_{IX} antigen and of chick helper factor in 1969. Since then there has been much discussion of whether the expression of endogenous viral antigens has a functional role in cellular differentiation. Certainly there is differential expression of viral antigens in different tissues and at different stages of development, but Strand, August and Jaenisch in the February issue of *Virology* (**76**, 886; 1977) conclude from studies of oncovirus gene expression in embryos of different inbred mouse strains that viral antigen synthesis is incidental to differentiation. While I agree that embryos of mice or chickens that show minimal endogenous viral gene expression appear to develop quite well, it is worth noting that feral mice and chickens (jungle fowl) do express viral antigens, so one can say at least that there is no natural selection against viral antigen synthesis. Furthermore, Chen and Vogt in the same issue of *Virology* (**76**, 740; 1977) find that oncovirus envelope glycoproteins are detectable in a variety of avian species.

In this week's *Nature*, (page 23) Elder, Jensen, Bryant and Lerner describe a more detailed study of the glycoproteins (gp 70) related to C-type oncoviruses in mice. By analysis of serotypes and of tryptic peptide maps, they can divide gp70 molecules associated with normal tissues into different groups, more or less related to gp70 specificities of virus isolates. Now most mouse strains inherit several distinct oncovirus genomes and this may account for much of the polymorphism. For instance in strain 129 mice, the serum gp70 resembles that of xenotropic virus, whereas seminal fluid gp70 is more closely related to the FMR group of viruses, not previously thought to be endogenous. Some of the tissue gp70-like molecules may not be linked to oncovirus glycoproteins at all. We do not know whether oncovirus glycoproteins have evolved from cellular antigens or *vice versa*, although it is well known that oncoviruses readily recombine for this genetic marker.

If viral glycoproteins serve as differentiation markers on the surface of normal cells, one might also expect cellular membrane proteins to be assembled into virus envelopes. In fact, this has been evident since de Thé, Becker and Beard (*J. natn. Cancer Inst.* **32**, 201; 1964) demonstrated the presence of leukocyte membrane ATPase in the envelope of avian myeloblastosis virus. Indeed, Beard used this enzyme activity as a biochemical assay of virus particles. Aupoix, Huppert and Vigier (*C. r. hebd. Acad. Sci. Paris* **282**, 1379; 1976) have demonstrated chick cellular antigens in Rous sarcoma virus particles which are inactivated by treatment with anti-chick serum plus complement, and Hecht and Summers (*J. Virol.* **19**, 833; 1976) have shown that vesicular stomatitis virus picks up H-2 antigens when grown in mouse cells. What is most intriguing, however, is the report by Bubbers and Lilly last month in *Nature* (**266**, 458; 1977) that Friend virus particles selectively incorporate $H\text{–}2D^b$ antigen. It is interesting enough that one particular histocompatibility antigen is selectively assembled in the virus envelope but Bubbers and Lilly further point out that the $H\text{–}2^b$ haplotype confers a relative resistance to Friend disease mediated by T-cell cytoxicity against associative recognition of both viral and $H\text{–}2^b$ antigens on the cell surface (Blank, Freedman & Lilly *Nature* **260**, 250; 1976). Thus the association of membrane antigens which act in Zinkernagel and Doherty's (*J. exp. Med.* **141**, 1427; 1975) 'altered-self' recognition system on the cell surface may also be reflected in the virus envelope. Lindenmann (*Biochim. biophys. Acta* **355**, 49; 1974) has argued for many years that viruses could be exploited as immunological adjuvants; complexes of viral and cellular glycoproteins in virus envelopes might be most potent immunogens.

The degree of selectivity of the assembly of $H\text{–}2^b$ into Friend virus is a little surprising in view of the variety of unrelated glycoproteins that enveloped viruses can assemble. Perhaps this is most strikingly seen in phenotypic mixing between unrelated viruses, where the 'foreign' antigen can be detected by changes in the infectivity of the virus (Zavada *Methods in Biology*, **6**, 109; 1977). This is how Chen and Vogt (*op. cit.*) detected functional viral glycoproteins in various species of pheasant and quail. No less than seven other papers in the same issue of *Virology* report studies on the assembly and behaviour of glycoproteins in enveloped animal viruses.

Cathodoluminescence topography

from John Walker

DIAMONDS are reputed to look good in candelight; to the scientist they look even better in electron light (cathodoluminescence—the emission of light during bombardment by electrons, as in a TV screen). Cathodoluminescence topography is a technique which has developed relatively recently and complements the older techniques of X-ray and UV topography. Scientists at the University of Bristol have recently published (Hanley, Kiflawi & Lang *Phil. Trans. R. Soc.* **284**, 329–368; 1977) their fascinating results on topographically-identifiable sources of cathodoluminescence in natural diamonds, including some fine colour photographs.

Their photographs carry a message, which as yet cannot be entirely deciphered. For example, a greenish-yellow luminescence system denoted H3, which is well-known from previous non-topographic studies, is observed at slip traces and dislocations and at 'platelets' (planar precipitates on {100} planes—see *Nature* **266**, 209; 1977). However, the usual method of producing the H3 defect is to irradiate the diamond with electrons or neutrons and then anneal it. Now, Hanley *et al.*'s diamonds might have been irradiated by natural radioactivity —indeed some of them undoubtedly had been, as discussed below—but such damage is confined to a thin surface layer. Hence these H3 defects, lying deep within the crystal, must have been produced by other means.

It is known that the H3 defect is composed of a radiation damage product (probably a vacancy), trapped at