rectified. An impressive new investigation by two Japanese industrial scientists (K. Nakagawa and T. Izumitani, *J. Non-Crystall. Solids*, **7**, 168; 1972) has served to determine the form of part of the miscibility gap in the Li_2O-Al_2O_3-SiO_2 system, which is of great importance for the production of glass ceramics with minimum thermal expansion. This study is at the same time an illustration of the power of that very simple technique, the determination of lattice parameters from X-ray diffraction patterns. Nakagawa and Izumitani prepared two series of glasses along ternary sections of constant Li_2O/Al_2O_3 ratio, and heat-treated them for different times at several temperatures to crystallize them (with the help of dissolved TiO_2 or ZrO_2 catalyst). The crystallized phase was always a ternary solid solution: the *a* and *c* lattice parameters varied as the phase ranged from nearly pure β-quartz to β-eucryptite, $Li_2O.Al_2O_3.2SiO_2$.

Isothermal heat treatments of a range of glasses at different temperatures produced classical plots of lattice parameter versus silica content, with horizontal plateaux identifying the region in which the crystalline phase is of constant composition. Such fields lie inside the miscibility gap. The crystalline phase was taken to crystallize at the same composition as one of the coexistent glassy phases (an assumption which strictly requires confirmation).

Examination of the lattice parameters of the phase at a range of temperatures proved that it had crystallized out of the silica-poor phase, and accordingly the form of the miscibility gap on the silica-poor side was accurately determined. (The peak of the gap lies at 800–820° C and about 85 wt per cent silica.) The silica-rich glassy phase does not crystallize and remains in the final glass ceramic. The fraction of residual glassy phase was determined as a function of temperature and could have been (but was not) used to determine the silica-rich side of the miscibility gap. The conclusions from X-ray study were tested and confirmed by electron-micrographic observations.

Another original approach to the problem of determining some features of a miscibility gap was recently described by C. T. Moynihan, P. B. Macedo, I. D. Aggarwal and W. E. Schnaus of the Catholic University of America at Washington (*ibid.*, **6**, 322; 1971). They performed differential calorimetric scans on cooling various glasses in the Pb-Ge-As-Se system. The scans showed two discontinuities which they were able to identify with the glass-transition temperatures of the two separated glassy phases. A critical analysis of the magnitudes of the specific heat discontinuities for glasses of various macroscopic compositions allowed some points on the miscibility gap to be located. This arises because the equilibrium begins to be "frozen" as soon as one of the two glassy phases has been cooled to its transition temperature.

TUMOUR VIRUSES

Transforming Subunit

from our Cell Biology Correspondent

THE isolation of mutant strains of Rous sarcoma virus (RSV) which carry temperature sensitive lesions in a gene or genes the expression of which is required to transform chick fibroblasts rules out the possibility that viral transformation is a hit and run affair. Indeed, quite the reverse is the case, for to keep a fibroblast transformed not only must the viral genome or part of it be maintained in the cell but also it must be continuously expressed; when, for example, a temperature sensitive transforming gene(s) is switched off by raising the temperature the phenotype of the transformed fibroblast reverts to that of an untransformed fibroblast.

Precisely what proteins are specified by the transforming genes of Rous sarcoma virus, and for that matter by the corresponding genes of any other oncogenic virus, is at present an unanswerable question which will probably long reside in the realms of speculation, for there are no obvious short cuts to an unequivocal answer. Looking for viral specified transforming proteins in a morass of cell proteins is, if not hopeless, daunting enough to dissuade most experimenters. If, however, what Duesberg and Martin have to say in the current issue of *Virology* (**47**, 494; 1972) can be taken at face value, and there is no good reason for doing otherwise, it should not be long before that part of the Rous sarcoma virus genome which contains at least some of the transforming genes is, in useful amounts, separated from the part carrying genes not involved in transformation of fibroblasts.

As Duesberg has shown, the subunit RNA molecules obtained when the 60–70S single-stranded genomic RNA of avian sarcoma viruses is denatured either by heat or by DMSO can be separated into two size classes by polyacrylamide gel electrophoresis—so-called *a* subunits with a larger molecular weight than *b* subunits. When, by contrast, genomic RNA of avian leukosis virus, which cannot transform fibroblasts, or of a non-transforming variant of the B77 strain of Rous sarcoma virus is similarly denatured and analysed, Duesberg finds only *b* subunits. One attractive way to account for these observations is to suggest that *a* subunits contain the genes that are essential for the transformation of fibroblasts; but one might equally suggest that *a* subunit RNA is only made in transformed cells and therefore only appears in those viruses which have replicated in transformed cells.

It is this possibility which Martin and Duesberg have now eliminated by analysing the subunit composition of the RNA of a temperature sensitive mutant, T5, of Schmidt Ruppin RSV isolated by Martin, and of stocks of Bryan high titre RSV. Martin's mutant replicates in chick fibroblasts at 41° C but cannot transform cells at that temperature although virus produced at 41° C can transform fibroblasts at 37° C. In other words, T5 carries a temperature sensitive mutation in a transforming gene(s) and if the *a* subunit of RSV RNA carries this gene(s) then *a* subunits should be present in the genomic RNA of T5 virus particles grown in a cell which, at 41° C, has the untransformed-cell phenotype. If, by contrast, the *a* subunit is only found in viruses grown on cells with the transformed-cell phenotype, T5 virus grown in fibroblasts at 41° C should lack *a* subunits. In fact, of course, the T5 virus has *a* subunit RNA irrespective of the temperature at which the virus is grown. What is more, stocks of Bryan high titre sarcoma virus which are known to contain an excess of leukosis virus mixed with a small proportion of sarcoma genomes yield very few *a* subunit RNA molecules, and when by repeated passage non-transforming variants of RSV are produced these virus particles are found to lack *a* subunits.

All these data are consistent with the notion that *a* subunits carry the genes necessary for the transformation of fibroblasts; they also are consistent with the suggestion that the subunits of the sarcoma virus genome may replicate independently as do the RNA subunits of the genomes of flu virus and reovirus.

Duesberg and his colleagues would no doubt predict that FU 19, the temperature sensitive mutant RSV isolated by Biquard and Vigier (*Virology*, **47**, 444; 1972), will be found to have *a* subunits, for FU 19 behaves very like Martin's T5 strain. And it seems equally certain that Duesberg and others will soon be attempting to isolate *a* subunits and to discover what genes they carry, perhaps by using them to programme cell free systems which support protein synthesis.

CANCER RESEARCH

Immunological Factors

from a Correspondent

THE possibility of immunological regulation of the growth of malignant cells was discussed by Professor N. A. Mitchison (University College, London) and Professor G. Hamilton Fair-