therefore required to initiate transformation. When permissive African green monkey cells of the AH line are infected at 33° C, 39° C and 41° C with these mutants the yield of infectious SV40 particles is 10-fold less at 39° C than at 33° C and about 10^4 less at 41° C than at 33° C. Moreover, the amount of infectious DNA that can be extracted from cells at the higher temperatures is commensurately lower. The mutant virus particles are not themselves more thermolabile than wild type virus particles, and this indicates that the mutated gene probably does not specify a structural protein of the virion, and that infections with naked mutant DNA, like infections with mutant particles, are temperature sensitive. It would seem, therefore, that adsorption and uncoating of these mutant particles are not temperature sensitive events.

To investigate further the temperature sensitive function Tegtmeyer infected AH cells at the permissive temperature. After replication of the viral DNA had started he shifted the cells to 41° C and fed them 10-minute pulses of ^{3}H-thymidine. Viral DNA was then extracted from the cells and analysed by gel electrophoresis, using a technique that Tegtmeyer and Macaset (*J. Virol.*, **10**, 599 ; 1972) have devised. Tegtmeyer concludes, from the pattern of incorporation of label into progeny SV40 DNA during such shift-up experiments and during shift-down experiments, that at the non-permissive temperature DNA molecules partially replicated at 33° C can be completed but that new rounds of replication cannot be initiated. In other words, Tegtmeyer's experiments clearly indicate that the *tsA* gene of SV40 specifies a specific initiator of viral DNA replication and presumably the same is true of the *tsa* mutant of polyoma virus. Needless to say, the protein specified by this gene has not yet been isolated, but it will not be surprising if it proves to be either an endonuclease or a protein capable of denaturing double-stranded DNA because either or both of these activities may be required to initiate viral DNA replication and integration of viral DNA into host cell DNA.

In the same issue of the *Journal of Virology* (page 653) Scolnick, Stephenson and Aaronson report the isolation of mutants of murine sarcoma virus which carry a lesion(s) in a gene(s) required for the maintenance of transformation. They obtained mutagenized stocks of Kirsten mouse sarcoma virus by exposing to bromodeoxyuridine rat cells, which were supporting the replication of this virus together with murine leukaemia virus. They then infected NRK rat cells with limiting dilutions of the mutagenized KiMSV (MLV) and isolated clones of transformants using microwell plates. By replica plating these transformants at 32° C and 39–40° C Scolnick *et al.* detected three lines (out of 3,000 tested) of transformants which, as judged by colony morphology, were temperature sensitive for transformation. The transforming sarcoma virus genome was rescued from these non-producer cells by superinfecting them with murine leukaemia virus. Two of the three rescued sarcoma viruses proved to give rise to temperature sensitive transformants.

Hybridization tests proved that KiMSV RNA is synthesized in similar amounts in cells transformed by these viruses and grown either at 32° C or 39° C. Transcription is not, therefore, temperature sensitive. Apart from that, these mutants have not been further characterized, but Scolnick *et al.* say they intend to isolate a suite of such mutants and by complementation tests determine how many MSV genes are involved in maintaining transformation.

SPIN LABELS

Radical Solutions

from our Molecular Biology Correspondent

THE spin-label technique has been applied with discrimination and elegance to several systems by McConnell and others. For placing this instrument into so many hot and eager little hands, however, its inventors will have to answer to their consciences, for a rash of electron spin resonance (ESR) spectra is now spreading like a contagion through the biochemical literature. Spin labels being commercially available, sensitive to the smallest kind of environmental change, and the ESR experiments technically undemanding, it is now possible to generate mountains of data with rather little effort, and the comfortable conviction that even if it is by no means obvious what they mean, they must assuredly mean something.

Through the gathering fog one may, however, discern some new and potentially useful ramifications of the method, as regards, for example, the measurements of critical distances. Two unpaired spins sufficiently close together will interact, and the perturbation of the ESR spectrum can be analysed in terms of the distance between them; second, NMR signals are also apt to be broadened if the nuclei from which they arise are in close proximity to a paramagnetic centre. The second method has been explored by a group in Oxford, and an interesting approach to the potentialities of the first has now been made by Russian workers.

The story is to be found in two articles just out. Kokorin *et al.* (*Biophysics*, **17**, 34; 1972) have examined the effect of intermolecular distance on the ESR spectra of a series of iminoxyl derivatives in solution at low temperature. These conditions, including the use of glassy solvents, such as water-glycerol mixtures, are chosen to allow the observation of dipole–dipole interactions, in the absence of the otherwise important exchange contributions. As the concentration of the radical increases, the average intermolecular distance becomes small enough for dipole–dipole broadening and splitting to

Aminoacyl-tRNA Synthetase in RNA Tumour Viruses

RNA tumour virus particles have been shown to contain a variety of enzymatic activities and several sorts of nucleic acids, including some forms of host tRNAs, that are accumulated in a non-random way presumably as the virus particles bud from the surfaces of productively infected cells. These viruses also contain some ribosomes of the host cell.

It is not yet understood why the RNA tumour viruses should sequester parts of the protein synthesizing machinery of the host cell, nor is it known whether they function to make protein after they are incorporated into the virions, but the list of components discovered in these viruses continues to lengthen. Trávníček and Říman, for example, as they report next Wednesday in *Nature New Biology* (January 10), have now detected aminoacyl-tRNA synthetase in avian myeloblastosis virions.

In avian myeloblastosis virus particles lysine tRNA is more abundant than any other species of tRNA, although that is not the case in the cells in which this virus replicates. Trávníček and Říman therefore assayed virions disrupted by the non-ionic detergent 'Nonidet' for an aminoacyl-tRNA synthetase capable of charging lysyl-tRNA with lysine, and sure enough they found such activity. Furthermore, comparisons of the aminoacyl-tRNA synthetase activities for leucine, lysine and valine in AMV particles and in host cells indicate that both lysine tRNA and lysyl-tRNA synthetase are preferentially accumulated in the virus particles.

As Trávníček and Říman comment, the selective rather than random sequestration of particular components of the host cell's protein synthesizing machinery by these viruses suggests that the process may not be fortuitous. And if it is not fortuitous these molecules must presumably function at some stage in the life history of the virus particles; precisely when has yet to be determined.