Fedoroff's 4.3 kb *Ac* isolate only at their ends, which all share a terminal 11 bp sequence on which the *Ac* transposase is presumed to act. In their internal sequences they are largely non-homologous both with each other and with *Ac*. The sequences unique to each *Ds* tend to be present in the genome in multiple copies, which suggests, as R.B. Flavell (Plant Breeding Institute, Cambridge) pointed out, that some of the dispersed repetitive sequences that bulk so large in many plant genomes may have been dumped in their present positions by systems such as *Ac-Ds*. How unrelated sequences come to be bracketed by *Ds* terminal inverted repeats is not clear. A typical transposon-like property of *Ac* and its *Ds* relatives is that of generating short direct repeats (6 or 8 bp) at their sites of insertion. When further transposition occurs, the tandem repeat is left behind as a 'footprint' (Peacock).

Numerous other maize transposable elements remain to be investigated in molecular detail. P.A. Peterson (University of Iowa) listed seven, including McClintock's *Suppressor-Mutator (Spm)* element, which will be particularly interesting to investigate because of its penchant for switching between alternative 'phases' or 'states'. F. Salamini (University of Bergamo) described a new element, called *Bgl*, which has so far been found particularly in mutable alleles of *opaque2* O_2), a gene which seems to be a regulator of the genes coding for seed proteins of the zein family. The element *Mu-1* (no connection with the bacteriophage of that name), recently discovered by D.S. Robertson, came originally from teosinte and proliferates rapidly to a high stable copy-number when crossed into maize, in which it induces many mutations (M. Freeling). A further element, perhaps of a different kind, is associated with infection by barley stripe virus (M. Freeling and S. Dallaporta, Cold Spring Harbor).

Patterns of variegation suggestive of movable elements are found in many flowering plants other than maize. A. Cornu (University of Dijon) described one such example in *Petunia hybrida*. In *Antirrhinum majus*, there is the opportunity for investigation at the DNA level since the *nivea* (chalcone synthetase) gene, of which there is a highly mutable allele *niv53*, has been cloned (it proved possible to use cDNA from the parsley chalcone synthetase mRNA as a probe). H. Saedler (Max-Planck-Institut, Köln) reported his finding of two large inserted elements, one (*Tam1*, 17 kb) in *niv53* and the other (*Tam2* 5.6 kb) in *niv44*, a stable null allele. Both elements have long mutually inverted terminal repeats, those of *Tam1* being of extraordinary internal complexity with short tandem and inverted sub-repeats. Only the terminal 17 bp of these terminal repeats are common to the two elements. Curiously, the *niv53*/*niv44* heterozygote showed almost no instability of the *niv53* allele, which suggested to Saedler that

Tam2 (which is present in the *niv44* stock in multiple copies) might code for a repressor that inhibited both its own transposition and that of *Tam1*.

In spite of the remaining puzzles it seems likely that our appreciation of the beauty of mutational variegation in plants will soon be enhanced by a much deeper understanding of how it arises.

───────────

John R.S. Fincham is professor in the Department of Genetics, University of Edimburgh, King's Buildings, West Mains Road, Edinburgh EH9 3JN.

Oncogenic intelligence

Viral oncogene permutations

from Peter Newmark

WHAT at first sight are four, but on closer inspection are only three, or perhaps even two, new oncogenes have recently been added to the twenty or so that have been discovered in tumour viruses but have their origins in normal cells (reviewed in ref. 1). Of particular interest is that each of the newly discovered oncogenes can coexist with a second oncogene in a virus. That brings to three the number of RNA tumour viruses in which two distinct oncogenes cooperate in a way that superficially resembles the cooperation of pairs of oncogenes to bring about the transformation of primary human fibroblasts in culture (see these columns **306**, 582).

One of the new oncogenes, described by two groups in last week's *Nature*[2,3] has been christened *ets,* an acronym for the E26 virus in which it has been found. Like AMV, another RNA tumour virus that causes myeloblastosis in chickens, E26 contains the *myb* oncogene. Unlike AMV, E26 can cause erythroblastosis, probably due to the additional presence of the *ets* oncogene. The best evidence that *ets* is completely distinct from *myb* is that RNA transcripts from the cellular progenitor of *ets* do not contain *myb* sequences[3]; the best characterization of the viral *ets* oncogene is its complete sequence[2].

The other three — or perhaps two, or even one — new oncogenes are known as *mil*[4], *mht*[5] and *raf*[6]. The first two names are synonyms for a newly described oncogene of Mill Hill 2 virus (hence *mil*) which is generally abbreviated to MH2 virus (hence the acronymic *mht*). The MH2 virus, which causes both leukaemias and carcinomas of chickens, also contains the *myc* oncogene, which is found in three other, closely related viruses as well. The *mil/mht* oncogene appears to be derived from a completely different cellular gene than does *myc;* how, or even whether, the two interact to produce the carcinomas and leukaemias remains to be determined.

The *raf* oncogene is the transforming gene of 3611-MSV, a murine RNA tumour virus that induces fibrosarcomas in newborn mice[6]. When isolated, the *raf* oncogene had no relationship to other known oncogenes. But since then, *mil/mht* has been characterized and in a paper soon to be published in *Nature*[7], Jansen *et al.* establish that *raf* and *mil/mht* are closely

related. Quite how closely will only be clear when sequences of the two viral oncogenes and their cellular progenitors become available but on present evidence Jansen *et al.* are confident that they arise from equivalent murine and avian genes.

It is not unprecedented for different tumour viruses to pick up equivalent cellular genes from different species. Three different feline sarcoma viruses have oncogenes similar to those of an avian, a feline and a murine retrovirus (see ref. 8). Nor is it unprecedented for two separate oncogenes to be found in one virus — the avian erythroblastosis virus was already known to contain both *erb*A and *erb*B viral oncogenes. But now that neither phenomenon is confined to a single case, the questions each raises become more interesting.

As to why the same gene should be picked by different viruses, the probable answer is that there is a rather small number of cellular genes that can be turned into oncogenes and that the documented list is nearing completion, limiting the more difficult problem of discovering the function of the products of the genes. That problem has probably been simplified by the classification of different oncogenes into two groups that cooperate to transform primary fibroblasts *in vitro*. It will now be important to establish whether the cooperation of pairs of oncogenes in viruses is a similar enough phenomenon to serve as a good model. There is also the fascinating question of how sequences from two distinct cellular loci come together in a virus — are they, perhaps, first juxtaposed by translocation in the cell?

───────────

Peter Newmark is Deputy Editor of Nature.

1. Duesberg, P. *Nature* **304**, 219 (1983).
2. Nunn, M.F., Seeburg, P.H., Moscovici, C. & Duesburg, P.H., *Nature* **306**, 391 (1983).
3. Leprince, D., Gegonne, A., Coll, J., de Taisne, C., Schneeberger, A., Lagrou, C. & Stehelin, D. *Nature* **306**, 395 (1983).
4. Jansen, H.W., Rückert, B., Lurz, R. & Bister, K. *EMBO J.* **2**, 1969 (1983).
5. Kan, N.C., Flordellis, C.S., Garon, C.F., Duesberg, P.H. & Papas, T.S., *Proc. Acad. Sci. U.S.A.* **80**, 6566 (1983).
6. Rapp, U.R., Goldsborough, M.D., Mark, G.E., Bonner, T.I., Groffen, J., Reynolds, F.H. & Stephenson, J.R. *Proc. nat. Acad. Sci. U.S.A.* **80**, 4218 (1983).
7. Jansen, H.W., Lurz, R., Bister, K., Bonner, T.I., Mark, G.E. & Rapp, U.R. *Nature* (in the press).
8. Besmer, P., Hardy, W.D., Zuckerman, E.E., Bergold, P., Lederman, L. & Snyder, H.W., *Nature* **303**, 825 (1983).