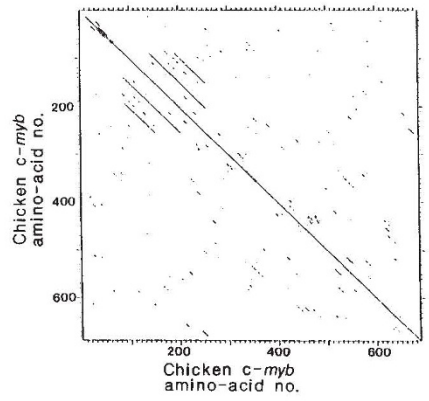

Fig. 3 Dot-matrix analysis for internal repeats within the chicken c-*myb* coding region. Segments of 25 amino acids were compared sequentially with each 25-amino-acid-long segment of the protein. A dot was placed on the matrix at the appropriate position when the total mutation data matrix score for the comparison was ⩾20.

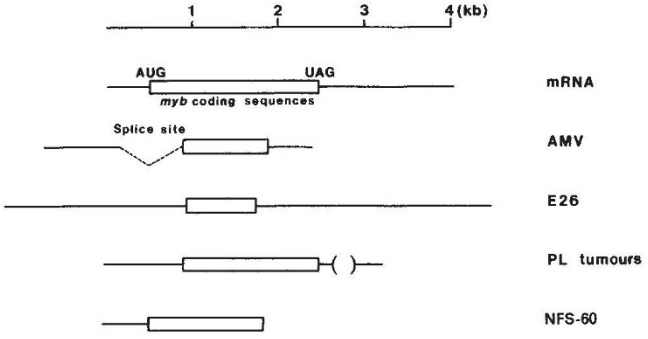

Fig. 4 Structural comparison of the *myb*-coding region of chicken c-*myb* mRNA (normal) with activated forms of *myb* mRNAs in the AMV and E26 viruses and PL and NFS-60 tumour cells. Boxes represent *myb*-coding sequences; straight lines represent flanking viral (AMV and E26) or cellular (PL tumours and NFS-60) sequences. The parentheses in PL tumour RNA indicate rearrangements at the 3′ end which have not yet been precisely defined.

of the three tandem repeats in the two v-*myb* proteins which may also be relevant for transformation.

In addition to the avian systems which suggest that deletions are important in the activation of *myb*, two other systems support this hypothesis. Analysis of the rearranged mouse c-*myb* genes in the Abelson plasmacytoid lymphosarcomas (ABPLs) induced by a mixture of Abelson murine leukaemia virus and Moloney murine leukaemia retrovirus (M-MuLV), reveals that the genomic insertion of the virus occurs in the intron immediately upstream of the sequences corresponding to nucleotide 450 in the chicken cDNA clone. This results in a deletion of the amino-terminal sequences in the aberrant c-*myb* transcripts analogous to that found in AMV (S. Lavu and E.P.R., in preparation) (Fig. 4).

A second example of activation of murine c-*myb* is seen in the NSF-60 tumour cell line induced by Cas-Br-M-MuLV[21]. In this myeloid tumour cell line the integration of the provirus has occurred at the 3′ end of the *myb* locus, which results in the synthesis of a truncated mRNA. We have recently mapped the point of integration of this virus to the point that corresponds to nucleotide 1,425 in the c-*myb* sequence presented in Fig. 2. This suggests that the *myb* protein produced in these tumour cells contains a carboxy-terminal deletion similar to that found in the AMV and E26 *myb* proteins.

Our DNA sequence comparison shows that the normal *myb* protein contains additional amino acids at the N-terminal and/or the C-terminal end when compared with any of the activated forms of this protein. The structural changes that occur as a result of these deletions probably contribute to the oncogenic potential of the proteins. The availability of chicken c-*myb* cDNA clones will allow us to make various retroviral constructs directly to test our hypothesis.

We thank Dr J. Jenson for help with computer analysis and Dr A. M. Skalka for a critical review of the manuscript.

Received 25 September; accepted 12 December 1985.

1. Baluda, M. A. & Goetz, I. E. *J. Virol.* **15**, 185–199 (1961).
2. Moscovici, C. *Curr. Topics Microbiol. Immun.* **71**, 79–96 (1975).
3. Franchini, G., Wong-Staal, F., Baluda, M. A., Lengel, C. & Tronick, S. A. *Proc. natn. Acad. Sci. U.S.A.* **80**, 7385–7389 (1983).
4. Katzen, A. L., Kornberg, T. B. & Bishop, J. M. *Cell* **41**, 449–456 (1985).
5. Mushinski, J. F., Potter, M., Bauer, S. & Reddy, E. P. *Science* **220**, 795–798 (1983).
6. Shen-Ong, G. L. C., Potter, M., Mushinski, J. F., Lavu, S. & Reddy, E. P. *Science* **226**, 1077–1080 (1984).
7. Lavu, S., Mushinski, J. F., Shen-Ong, G. L. C., Potter, M. & Reddy, E. P. *Cancer Cells* **3**, 301–306 (1985).
8. Pelicci, P.-G., Lanfrancone, L., Brathwaite, M. D., Wolman, S. R. & Dalla-Favera, R. *Science* **224**, 1117–1121 (1984).
9. Alitalo, K. *et al. Proc. natn. Acad. Sci. U.S.A.* **81**, 4534–4538 (1984).
10. Kozak, M. *Nucleic Acids Res.* **12**, 857–872 (1984).
11. Klempnauer, K. H. *et al. Cell* **33**, 345–355 (1983).
12. Maizel, J. & Leuk, R. *Proc. natn. Acad. Sci. U.S.A.* **78**, 7665–7669 (1981).
13. Klempnauer, K. H., Symonds, G., Evans, G. I. & Bishop, J. M. *Cell* **37**, 537–547 (1984).
14. Boyle, W. J., Lampert, M. A., Lipsick, J. S. & Baluda, M. A. *Proc. natn. Acad. Sci. U.S.A.* **81**, 4265–4267 (1984).
15. Moelling, K. *et al. Cell* **40**, 983–990 (1985).
16. Gonda, T. J., Gough, N. M., Dunn, A. R. & de Blaquiere, J. *EMBO J.* **4**, 2003–2008 (1985).
17. Rushlow, K. E. *et al. Science* **216**, 1421–1423 (1982).
18. Klempnauer, K. H., Gonda, T. J. & Bishop, J. M. *Cell* **31**, 453–463 (1982).
19. Nunn, M. F., Seeberg, P. H., Moscovici, C. & Duesberg, P. H. *Nature* **306**, 391–395 (1983).
20. Boyle, W. J., Lipsik, J. S., Reddy, E. P. & Baluda, M. A. *Proc. natn. Acad. Sci. U.S.A.* **80**, 2834–2839 (1983).
21. Weinstein, Y., Ihle, J. N., Lavu, S. & Reddy, E. P. *Proc. natn. Acad. Sci. U.S.A.* (submitted).
22. Gubler, U. & Hoffmann, B. J. *Gene* **5**, 263–269 (1983).
23. Maxam, A. M. & Gilbert, W. *Meth. Enzym.* **65**, 499–560 (1980).

Corrigendum

Major shear zones and autochthonous Dalradian in the north-east Scottish Caledonides

W. A. Ashcroft, B. C. Kneller, A. G. Leslie & M. Munro
Nature **310**, 760–762 (1984)

THE following reference should have appeared in support of this letter: Gallagher, J. W. thesis, Univ. Aberdeen (1983).