Transcription Factors, Oncogenes, and Apoptosis

Four papers [A. A. Beg and D. Baltimore, Reports, 1 Nov. 1996, p. 782; C.-Y. Wang *et al.*, *ibid.*, p. 784; D. J. Van Antwerp *et al.*, *ibid.*, p. 787 and (*1*)] have recently described a role for nuclear factor kappa B (NF-κB), a cellular transcription factor, in blocking apoptosis that is induced by tumor necrosis factor. These studies suggest that agents which inhibit Rel transcription factors may increase the sensitivity of tumor cells to therapeutics that induce apoptosis, as discussed in the Research News article "Life-death balance within the cell" by Marcia Barinaga (1 Nov., p. 724). Earlier studies demonstrated that the retroviral oncoprotein v-Rel can also block apoptosis in vivo (*2*) and in vitro (*3*). Although there was initially some controversy, it is now clear that v-Rel malignantly transforms and immortalizes cells by inducing expression of specific genes. Collectively, these observations suggest that some of the target genes for Rel-mediated oncogenesis and for the Rel/NF-κB–mediated blockage of apoptosis may be identical. Furthermore, the likely involvement of Rel transcription factors in many human lymphoid cancers [for example, (*4*)] suggests that agents that inhibit Rel transcription factors may have direct anti-cancer effects in some situations, as well as effects that facilitate apoptosis.

David W. White
Millennium Pharmaceuticals, 640 Memorial Drive, Cambridge, MA 02139, USA
Thomas D. Gilmore
Biology Department, Boston University, Boston, MA 02215, USA
E-mail: gilmore@bio.bu.edu

References

1. Z.-G. Liu, H. Hsu, D. V. Goeddel, M. Karin, *Cell* **87**, 565 (1996).
2. P. E. Neiman, S. J. Thomas, G. Loring, *Proc. Natl. Acad. Sci. U.S.A.* **88**, 5857 (1991).
3. D. W. White, A. Roy, T. D. Gilmore, *Oncogene* **10**, 857 (1995); D. W. White and T. D. Gilmore, *ibid.* **13**, 891 (1996).
4. C.-C. Chang, J. Zhang, L. Lombardi, A. Neri, R. Dalla-Favera, *Mol. Cell. Biol.* **15**, 5180 (1995).

■

"Note Added in Proof"

The following "Note added in proof" should have been added at the end of our report "Formation of actin stress fibers and focal adhesions enhanced by Rho-kinase" (28 Feb., p. 1308) (*1*).

After we submitted this report, similar observations were reported [T. Leung *et al.*, *Mol. Cell. Biol.* **16**, 5313 (1996)].

Kozo Kaibuchi
Division of Signal Transduction, Nara Institute of Science and Technology, 8916-5 Takayama, Ikoma, 630-01, Japan
E-mail: kaibuchi@bs.aist-nara.ac.jp

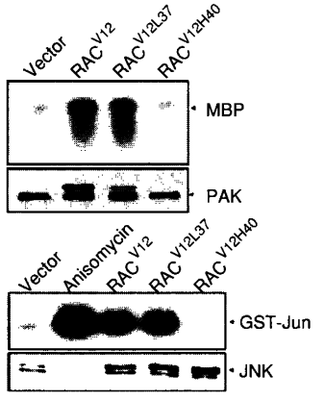
References

1. M. Amano, K. Chihara, K. Kimura, Y. Fukata, N. Nakamura, Y. Matsuura, K. Kaibuchi, *Science* **275**, 1308 (1997).

Corrections and Clarifications

In the article "Panel approves gene trial for 'normals'" by Eliot Marshall (News & Comment, 14 Mar., p. 1561), R. Scott McIvor was incorrectly identified as director of the Gene Therapy Program at the University of Minnesota. He is Director of the Gene Therapy Program at the University of Minnesota's Institute of Human Genetics, which is directed by Anthony Faras.

In figure 1 on page 1375 of the report "RAC regulation of actin polymerization and proliferation by a pathway distinct from Jun kinase" by T. Joneson *et al.* (22 Nov., p. 1374), several of the labels were incorrect. The correct figure appears below.



In "The Genome Maps 1996" (25 Oct., p. 547), the DNA content of chromosome 1 should have read, "263 Mb."

Letters to the Editor

Letters may be submitted by e-mail (at science_letters@aaas.org), fax (202-789-4669), or regular mail (*Science*, 1200 New York Avenue, NW, Washington, DC 20005, USA). Letters are not routinely acknowledged. Full addresses, signatures, and daytime phone numbers should be included. Letters should be brief (300 words or less) and may be edited for reasons of clarity or space. They may appear in print and/or on the World Wide Web. Letter writers are not consulted before publication.