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Cover picture: Fetal liver-derived erythroid precursors from Raf-1-deficient mice undergo accelerated erythroid differentiation. The cover shows Raf-1-deficient erythroblasts 24 hr after the induction of differentiation in vitro. A decrease in overall cell volume and the accumulation of hemoglobinized cells are evident, whereas the wild-type population consists mostly of blast-like cells. Caspase activation is necessary for the differentiation of murine erythroblasts, and is accelerated in erythroid progenitors lacking Raf-1. In vivo, runaway differentiation results in a depletion of erythroid progenitors likely responsible for the anemic phenotype of Raf-1-deficient embryos. These results reveal an essential function of Raf-1 in erythropoiesis and demonstrate that the ability of Raf-1 to restrict caspase activation is biologically relevant in a context distinct from apoptosis. See related article by Kolbus et al., pp. 1347–1353.

- 1373** *Marie-Claude Guillemin, Emmanuel Raffoux, Dominique Vitoux, Scott Kogan, Hassane Soilihi, Valérie Lallemand-Breitenbach, Jun Zhu, Anne Janin, Marie-Thérèse Daniel, Bernard Gourmet, Laurent Degos, Hervé Dombret, Michel Lanotte, and Hugues de Thé.* In vivo activation of cAMP signaling induces growth arrest and differentiation in acute promyelocytic leukemia

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