

COMMENTARY

I read with interest the paper by Winter and colleagues describing the expression of the human erythropoietin receptor (EpoR) gene in TF-1 cells (1). Unfortunately, their data, while intriguing, are not necessarily relevant to the physiological regulation of the EpoR gene.

We have previously shown that one copy of the EpoR gene is rearranged in TF-1 cells due to a chromosome translocation affecting chromosome 19 (2). Furthermore, the large majority of the EpoR mRNA in TF-1 cells is produced by the translocated gene, whereas the normal EpoR gene is expressed only minimally (3). The very high level of expression of the rearranged gene appears, therefore, to be the result of deregulated transcriptional control produced by the translocation. Consequently, the fluctuations in EpoR mRNA observed by Winter almost certainly result from modulating the translocated EpoR. Unless they show data that the normal EpoR gene is regulated in TF-1 cells, or in another system, I remain

unconvinced that their conclusions apply to physiological erythro-poiesis.

REFERENCES

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John C. Winkelmann, M.D.
Director, Division of Hematology-Oncology
Department of Internal Medicine
University of Cincinnati College of Medicine
231 Bethesda Avenue
Cincinnati, OH 45267-0508
winkeljc@uc.edu
phone 513-558-2195
fax 513-558-6703