Corrections

H-Ras Regulates ERCC1

In the article on how H-Ras regulates ERCC1 in the July 15, 2004 issue of *Cancer Research* (1), Figures 4 and 8 were incorrect. The correct figures appear below.

1. Youn CK, Kim MH, Cho HJ, Kim HB, Chang IY, Chung MH, You HJ. Oncogenic H-Ras up-regulates expression of ERCC1 to protect cells from platinum-based anticancer agents. Cancer Res 2004;64:4849–57.

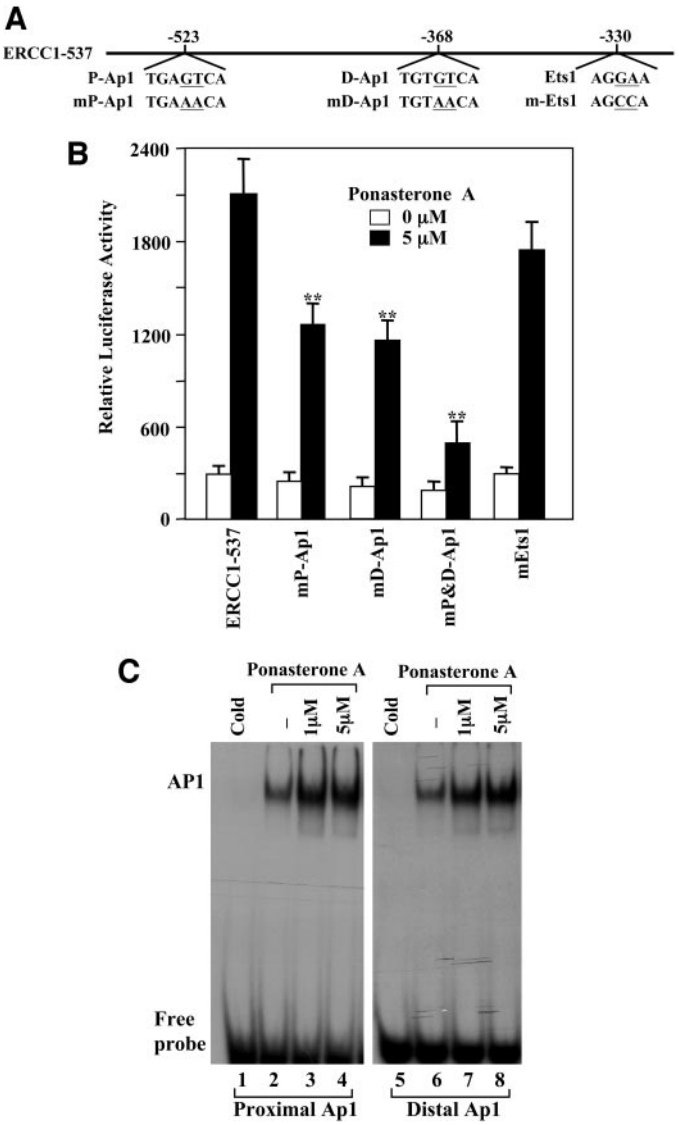

Fig. 4. Oncogenic H-Ras-triggered increase in the DNA binding activity of Ap1 to the ERCC1 promoter. *A,* ERCC1–537 promoter has two putative Ap1 binding sites and one putative Ets1 binding site. The *underlined GT* in Ap1 and *GA* in Ets1 were mutated to AA (*mP-Ap1* and *mD-Ap1*) and CC (*m-Ets*), respectively. *B,* the ERCC1–537 reporter constructs containing the indicated mutations, in the proximal Ap1 site (*mP-Ap1*), in the distal Ap1 site (*mD-Ap1*), in the proximal and distal Ap1 sites (*mP&D-Ap1*), or in the Ets1 site (*mEts1*) were transfected together with pRL-CMV into the NIH3T3 clone-1 cells. The cells were then treated with or without 5 μM ponasterone A for 24 h, and the luciferase activities were measured. The graph shows the luciferase activity (relative to that in the cells transfected with pGL3-Basic) in the cells treated with or without 5 μM ponasterone A. The values represent the means from six separate experiments; *bars,* ±SD. ** denotes $P < 0.01$. *C,* electrophoretic mobility-shift assays were performed using a radio-labeled probe from the ERCC1 promoter region containing the proximal or distal Ap1 sites and the nuclear extracts from the ponasterone A-treated or untreated (−) NIH3T3 clone-1 cells. *Lane 1*, NIH3T3 clone-1 nuclear extract treated with a 50-fold excess of the unlabeled consensus Ap1 oligonucleotide; *lane 2*, untreated NIH3T3 clone-1 cells; *lane 3*, NIH3T3 clone-1 cells treated with 1 μM ponasterone A for 24 h; *lane 4*, NIH3T3 clone-1 cells treated with 5 μM ponasterone A for 24 h; *lane 5*, NIH3T3 clone-1 nuclear extract treated with a 50-fold excess of the unlabeled consensus Ap1 oligonucleotide; *lane 6*, untreated NIH3T3 clone-1 cells; *lane 7*, NIH3T3 clone-1 cells treated with 1 μM ponasterone A for 24 h; *lane 8*, NIH3T3 clone-1 cells treated with 5 μM ponasterone A for 24 h.

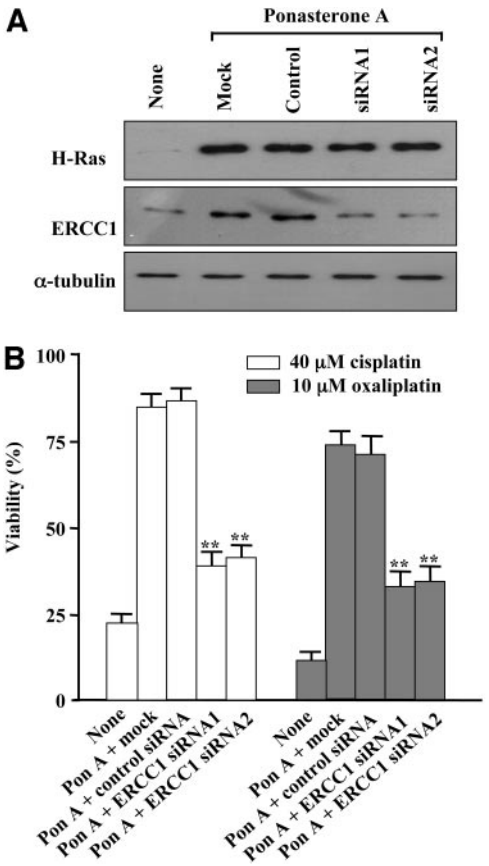

Fig. 8. Effect of small interfering (si)RNA-mediated ERCC1 depletion on the resistance to the platinum-based agents of MCF-7 cells conferred by the expression of oncogenic H-Ras. *A,* the mock-, control small interfering (si)RNA-, or ERCC1 siRNA-transfected MCF-7 clone-2 cells were treated with or without (*None*) 5 μM ponasterone A and harvested 24 h later. Immunoblots were performed with the antibodies to Ras, ERCC1, and α-tubulin, and α-tubulin expression level was used as the control experiment with an equal protein loading. *B,* the mock-, control siRNA-, or ERCC1 siRNA-transfected MCF-7 clone-2 cells were incubated with or without (*None*) 5 μM ponasterone A (*Pon A*) for 24 h. Subsequently, the cells were treated with either 40 μM cisplatin or 10 μM oxaliplatin for 1 h, and the cell viability was then determined by a clonogenic survival assay. The values represent the means from six separate experiments; *bars,* ±SD. ** denotes $P < 0.01$.

Growth Promoting Signaling by Tenascin-C

In the article on growth promoting signaling by tenascin-C in the October 15, 2004 issue of *Cancer Research* (1), there is an error in the running title. The correct running title is Growth Promoting Signaling by Tenascin-C.

1. Ruiz C, Huang W, Hegi ME, Lange K, Hamou MF, Fluri E, Oakeley EJ, Chiquet-Ehrismann R, Orend G. Differential gene expression analysis reveals activation of growth promoting signaling pathways by tenascin-C. Cancer Res 2004;64:7377–85.

Corrections

Cancer Res 2004;64:8484.