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.


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.


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 (m-Ets1) 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 unlabelled 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 unlabelled 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.
Corrections

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