Errata

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Estrogen receptor α mediates the nongenomic activation of endothelial nitric oxide synthase by estrogen


During the production process, panels a and b of Figure 2 were mistakenly repeated as panels c and d. The correct display of the figure and accompanying legend is reproduced here. We regret the error and have provided corrected reprints to the corresponding author: Philip W. Shaul, Department of Pediatrics, University of Texas Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, Texas 75235-9063, USA. Phone: (214) 648-2015; Fax: (214) 648-2481; E-mail: pshaul@mednet.swmed.edu.

Figure 1
Rapid activation of eNOS in endothelial cells. (a) Effect of E2 on eNOS activity in intact PAEC. [3H]arginine conversion to [3H]citrulline was measured over 5–15 min in the presence of 10⁻⁸ M E₂. (b) Effect of actinomycin D (Act D) on the rapid activation of eNOS. After 120 min incubation, Cer content, nuclear fragmentation, mitochondrial transmembrane potential and cell viability were determined in parallel samples as described in the Methods section. The results are representative of two independent experiments.

Table 1
Jurkat cells (J16) were preincubated for 2 h with zVAD-fmk (50 μM), DEVD-CHO (100 μM) or left untreated and then exposed to etoposide (10 μg/ml) or IR (30 Gy). After 16 h incubation, Cer content, nuclear fragmentation, mitochondrial transmembrane potential and cell viability were determined in parallel samples as described in the Methods section. The results are representative of two independent experiments.

Ordering of ceramide formation, caspase activation, and mitochondrial changes during CD95- and DNA damage–induced apoptosis
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During the production process, the word caspase was misspelled in the title; the correct title appears above. Also, in the legend for Table 1 the μ symbol (μ) was formatted incorrectly; the correct legend appears below. We regret the error.

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