

## Correction

**Correction: Cepharranthine hydrochloride reverses the mdr1 (P-glycoprotein)-mediated esophageal squamous cell carcinoma cell cisplatin resistance through JNK and p53 signals****Pengjun Zhou<sup>1,2,\*</sup>, Rong Zhang<sup>3,\*</sup>, Ying Wang<sup>1</sup>, Dandan Xu<sup>4</sup>, Li Zhang<sup>5</sup>, Jinhong Qin<sup>1</sup>, Guifeng Su<sup>1</sup>, Yue Feng<sup>1</sup>, Hongce Chen<sup>2</sup>, Siyuan You<sup>2</sup>, Wen Rui<sup>2</sup>, Huizhong Liu<sup>6</sup>, Suhong Chen<sup>4</sup>, Hongyuan Chen<sup>2,7</sup> and Yifei Wang<sup>1</sup>**

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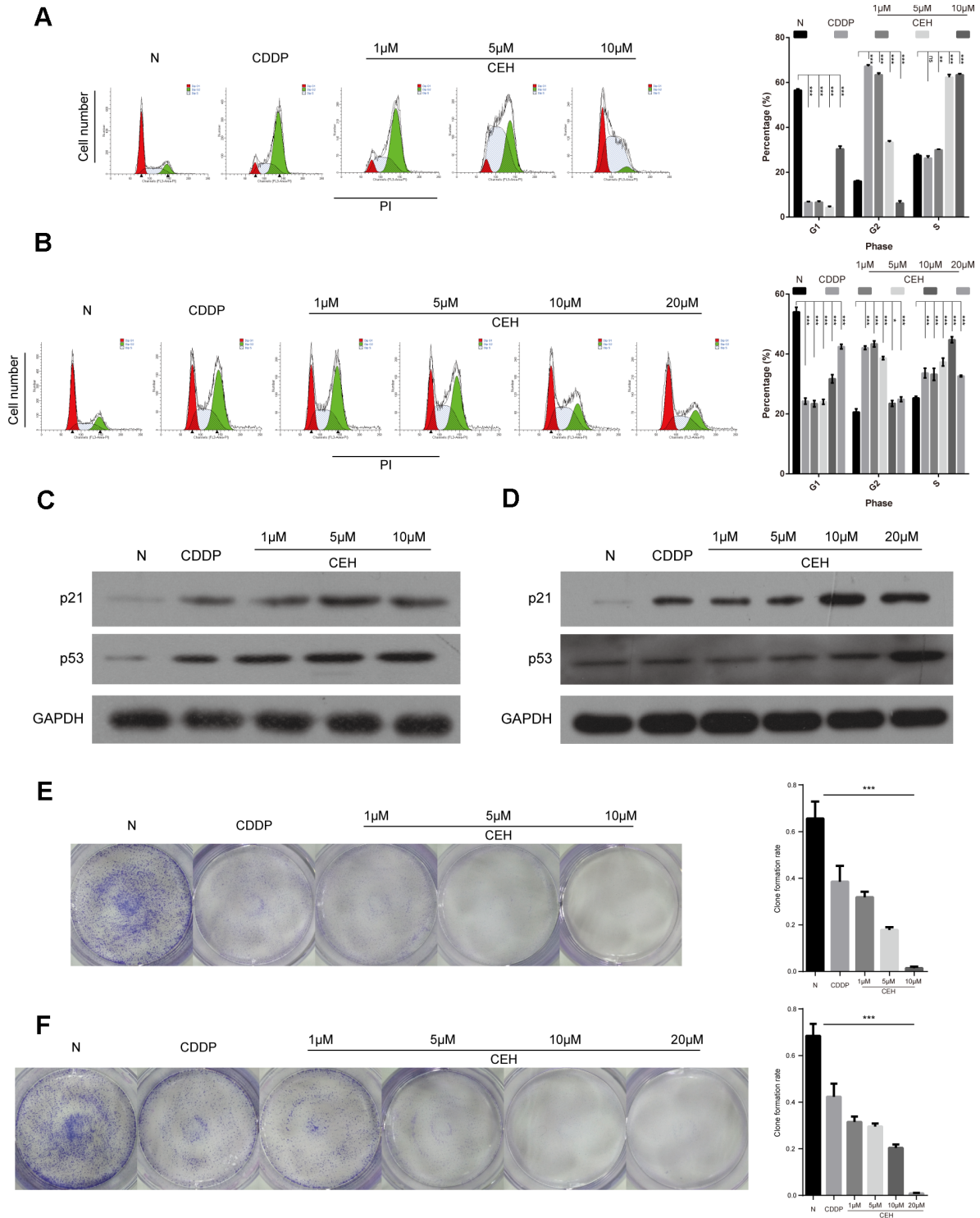
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**Published:** January 05, 2021

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**This article has been corrected:** Due to errors during image assembly, the images for Figure 2, panels E and F, were accidentally switched. The corrected Figure 2 is shown below. The authors declare that these corrections do not change the results or conclusions of this paper.

Original article: Oncotarget. 2017; 8:111144–111160. <https://doi.org/10.18632/oncotarget.22676>



**Figure 2: Induction of cell cycle arrest and inhibition of cell proliferation by cisplatin (cDDP) alone and combined with cepharanthine hydrochloride (CEH) in esophageal cancer cell lines.** (A and B) Cell cycle analysis. Percentages of Eca109 and Eca109/CDDP cells in the G1, S, and G2/M phases are presented respectively. Effects of cDDP and combined with various concentrations of CEH medication on cell cycle distribution. Eca109 (A) and Eca109/CDDP (B) cells were treated with 0, 1, 5, 10 and 20  $\mu$ M CEH combined with cDDP for 48 h, and cell cycle distribution was measured by flow cytometry after PI staining. (C and D) p21 and p53 protein levels were determined by western blot analyses. GAPDH was used as the loading control. (E and F) Cells were treated with 0, 1, 5, 10 and 20  $\mu$ M CEH combined with cDDP for 48 h; representative images of Eca109 (E) and Eca109/CDDP (F) clone formation are shown. \*\*,  $P < 0.001$  compared with the control.