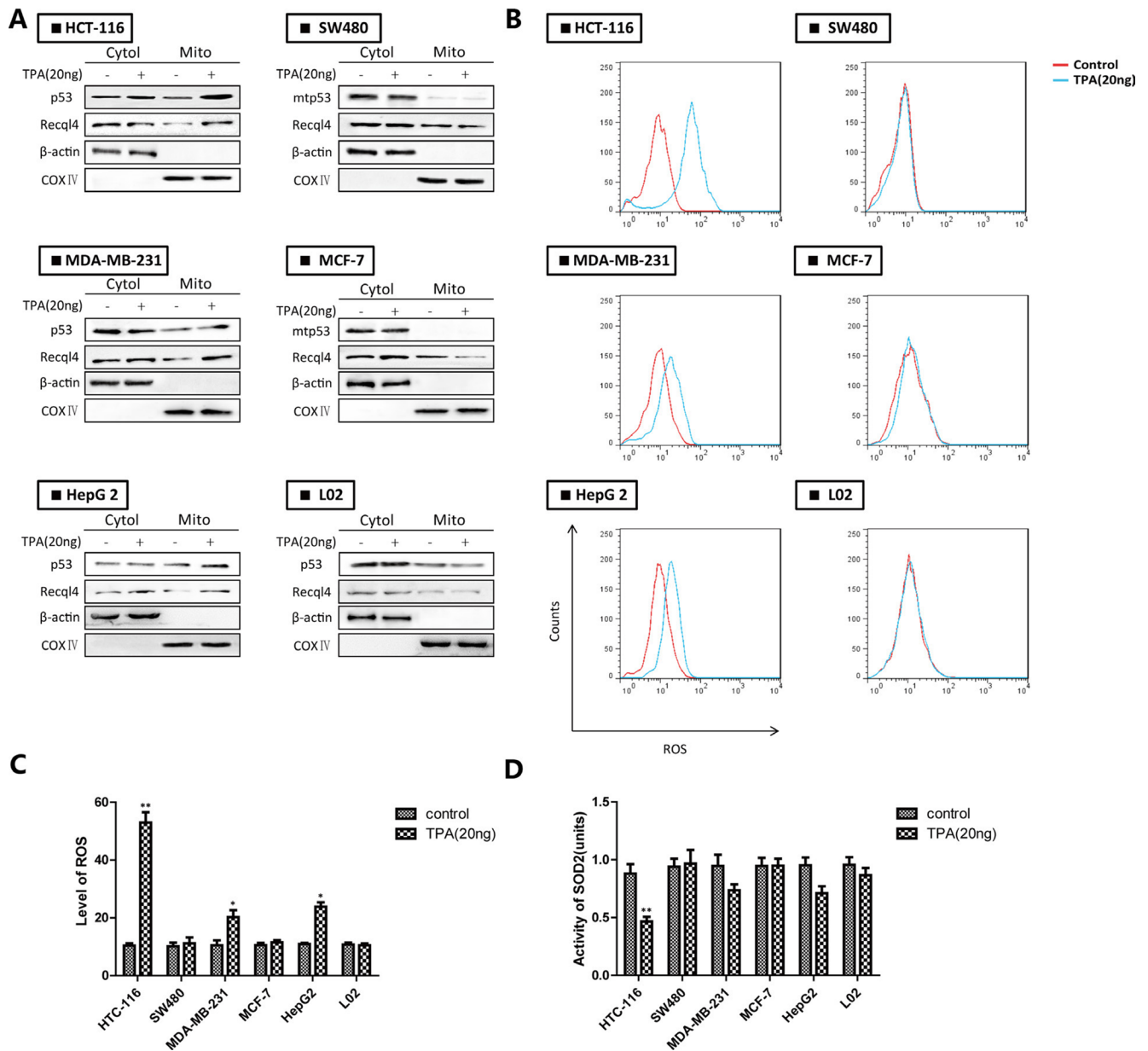


## Correction

**Correction: Oroxylin A modulates mitochondrial function and apoptosis in human colon cancer cells by inducing mitochondrial translocation of wild-type p53****Chen Qiao<sup>1,\*</sup>, Na Lu<sup>1,\*</sup>, Yuxin Zhou<sup>1</sup>, Ting Ni<sup>1</sup>, Yuanyuan Dai<sup>1</sup>, Zhiyu Li<sup>2</sup>, Qinglong Guo<sup>1</sup>, Libin Wei<sup>1</sup>**<sup>1</sup>State Key Laboratory of Natural Medicines, Jiangsu Key Laboratory of Carcinogenesis and Intervention, China Pharmaceutical University, Nanjing 210009, People's Republic of China<sup>2</sup>Department of Medicinal Chemistry, China Pharmaceutical University, Nanjing 210009, People's Republic of China

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**Published:****Copyright:** Qiao et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License 3.0 (CC BY 3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.**This article has been corrected:** Due to errors during image assembly, the Western Blots of  $\beta$ -actin of HepG 2 group and COX IV of L02 group in Fig. 1 were accidentally misplaced. The corrected Figure 1 is shown below. The authors declare that these corrections do not change the results or conclusions of this paper.Original article: Oncotarget. 2016; 7:17009–17020. <https://doi.org/10.18632/oncotarget.7927>



**Figure 1: The accumulation of p53 in mitochondria leads to inhibition of SOD2 and increased ROS generation in wt-p53 cancer cells. (A) Western blotting analysis of mitochondrial p53. (B) ROS production was monitored using 10  $\mu$ M DCFH-DA and detected by flow cytometry. (C) Quantification of ROS levels. (D) Evaluation of SOD2 activity. Bar, SD. \* $P < 0.05$  or \*\* $P < 0.01$  versus the untreated control.**