



Corrigendum

Corrigendum to “Oroxylin A sensitizes non-small cell lung cancer cells to anoikis via glucose-deprivation-like mechanisms: c-Src and hexokinase II” [2013; 1830:2835–2845]: Clarification of figure legends, as posted by the authors



Libin Wei^a, Qinsheng Dai^a, Yuxin Zhou^a, Meijuan Zou^a, Zhiyu Li^b, Na Lu^{a,*}, Qinglong Guo^{a,*}

^a State Key Laboratory of Natural Medicines, Jiangsu Key Laboratory of Carcinogenesis and Intervention, China Pharmaceutical University, 24 Tongjiaxiang, Nanjing 210009, PR China
^b Department of Medicinal Chemistry, China Pharmaceutical University, 24 Tongjiaxiang, Nanjing 210009, PR China

It was discovered that the original online version of the above article contained errors in the legends for Figs. 3 and 5. The corrected legends appear below.

Elsevier and the authors regret and apologize for any inconvenience caused by posting a new version of this article online, but hope that the reader will understand the reasons for doing so.

Fig. 3. Oroxylin A sensitized A549 cell anoikis by inactivation of the c-Src/PI3K/AKT pathway. Detached A549 cells were treated with oroxylin A for 36 h. (A) Cell viability was determined by MTT assay. Bars, SD(±). (B) Anoikis rates were assessed as above. Bars, SD, *p < 0.05 or **p < 0.01 versus control. (C) The morphology of anoikis nuclei was stained with DAPI and observed (400×). The white arrows point to apoptotic cells. (D–E) Expression of c-Src, p-Src(Y418), procaspase 3, PARP, and proteins in the AKT/mTOR/Bad pathway. The actin controls of (C) and (D) were the same control (the same protein band) for total protein because the actual data were from the same experimental dataset. (F) Molecular structure of oroxylin A.

Fig. 5. (A–E) Oroxylin A suppressed HK II binding to VDAC, triggering mitochondrial dysfunction. Detached A549 cells were

cultured in the presence of oroxylin A for 36 h, 3-BrPA for 12 h and PP2 for 20 h, respectively. (A) Mitochondria were isolated and HK II was immunoprecipitated using VDAC antibody. Western blot assays were performed for HK II and VDAC. (B–C) Mitochondrial and cytosolic fractions were isolated after treatment and subjected to Western blot analysis for HK II, Cyt-c and AIF. (D) Mitochondria were isolated, and Western blot analysis was performed for Bax, Bid, and bcl-xl. The COXIV controls of (B), (C) and (D) were the same control (the same protein band) for mitochondrial protein because the actual data were from the same experimental dataset. As well, the GAPDH of (B) and (C) was the same control (the same protein band) for nucleus protein from the same experimental dataset. (E) The loss of MMP was analyzed by JC-1 assay using flow cytometry. The percentages of the loss of MMP are represented by histograms. **(F–G) Oroxylin A suppressed lung metastasis in vivo.** (F) Representative examples represent the metastases in the lungs of animals in each group. (G) Evaluation of macroscopically detectable lung metastases. After fixation in Bouin's solution, the number of macroscopically visible metastases on the surface was quantified. OA represents oroxylin A. Bars, SD, *p < 0.05 or **p < 0.01 versus non-treated control.

DOI of original article: <http://dx.doi.org/10.1016/j.bbagen.2013.03.009>.

* Corresponding authors. Tel./fax: +86 25 83271055.

E-mail addresses: luna555@163.com (N. Lu), anticancer_drug@yahoo.com.cn (Q. Guo).