

## CORRIGENDUM

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**Leptin stimulates ovarian cancer cell growth and inhibits apoptosis by increasing cyclin D1 and Mcl-1 expression via the activation of the MEK/ERK1/2 and PI3K/Akt signaling pathways**CHIACHEN CHEN, YUAN-CHING CHANG, MICHAEL S. LAN  
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Following the publication of this article, an interested reader drew to our attention an anomaly associated with the presentation of Figs. 5 and 8; essentially, the  $\beta$ -actin control bands in Fig. 8 appeared to be a mirror image of the same control bands shown in Fig. 5A.

After having re-examined the data, Fig. 5 was correctly presented as shown in the paper, although the  $\beta$ -actin blot in Fig. 8 had been inadvertently misplaced as a mirror image of the control data from Fig. 5. Two similar experiments were performed at that time, yielding similar results, and the error with the control data did not affect the second of the experiments. Therefore, we present a corrected version of Fig. 8 containing the alternative data. The findings and conclusions of this paper are still supported by our experimental data, and were not affected by this error. We sincerely apologize for this mistake, and thank the reader of our article who drew this matter to our attention. Furthermore, we regret any inconvenience this mistake has caused.

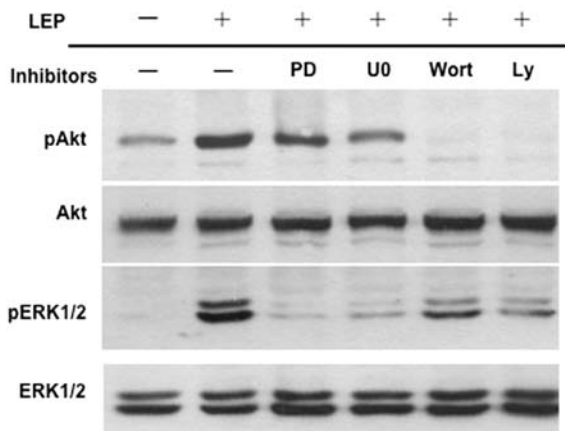


Figure 8. Leptin triggers JAK2-MEK/ERK1/2 and PI3K/Akt signaling pathways. After 24 h serum deprivation and subsequent 30 min pre-treatment with the vehicle (-), 20  $\mu$ M PD98059 (PD), 10  $\mu$ M U0126 (U0), 250 nM wortmannin (Wort) or 50 mM Ly294002 (Ly), OVCAR-3 cells were treated with vehicle (-) or 200 ng/ml leptin (+) in serum-free medium for 5 min, respectively. Subsequently, the level of the phosphorylated forms of Akt (pAkt) or ERK1/2 (pERK1/2) was detected with western blot analysis. The same blots were stripped and reprobed with antibodies specific for total protein of Akt or ERK1/2. Data represent three independent experiments.