# Correction to "Rimonabant, a Cannabinoid Receptor Type 1 Inverse Agonist, Inhibits Hepatocyte Lipogenesis by Activating Liver Kinase B1 and AMP-Activated Protein Kinase Axis Downstream of $G\alpha_{i/o}$ Inhibition"

In the above article [Wu HM, Yang, YM, and Kim SG (2011) *Mol Pharmacol* **80:**859–869], several figures contained errors that require correction and explanation.

The IP/IB Western blots shown in Figs. 4A, left, and 6D, right, were done using the same LXR$\alpha$ immunoprecipitates; likewise, those in 6D, left and middle, were done using the same LXR$\alpha$ immunoprecipitates. However, the first lane of the LXR$\alpha$ band in Fig. 4A, right, was accidentally overlapped with the fourth lane of the LXR$\alpha$ band in Fig. 6D, left. Corrected versions of Figs. 4A and 6D, along with the related raw blots and corrected legends, are provided below.

In addition, two lanes loaded for positive control were mistakenly included in the p-ACC band in Fig. 6A, right. The corrected band and the related raw blot are provided below.

Finally, in the scheme in Fig. 7, "AMP" should be "ATP."

The online version of this article has been updated in departure from the print version.

The authors regret these errors and apologize for any confusion or inconvenience they may have caused.

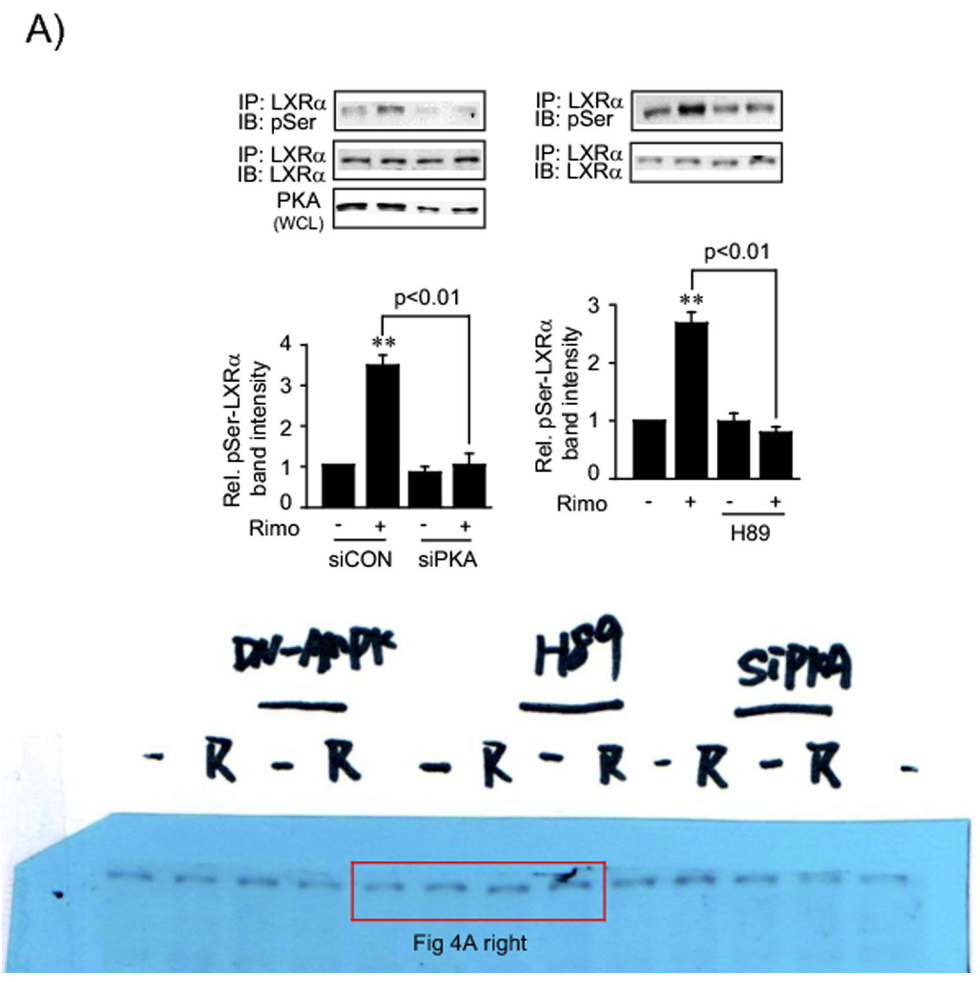


**Fig. 4.** A, effect of PKA on serine phosphorylation of LXR$\alpha$ by rimonabant. HepG2 cells were treated with vehicle or 10 $\mu$M rimonabant after siPKA transfection (100 pmol/ml) for 48 h or 1 $\mu$M H89 treatment for 1 h. LXR$\alpha$ immunoprecipitates (IP) were immunoblotted (IB) with anti-phosphorylated serine antibody (pSer). After verifying equal loading of proteins in each experiment by immunoblotting of LXR$\alpha$ immunoprecipitates for LXR$\alpha$, the relative protein levels of pSer-LXR$\alpha$ from at least three separate experiments were compared among four treatment groups in each experimental set (i.e., siCON + vehicle, siCON + rimonabant, siPKA + vehicle, and siPKA + rimonabant, or vehicle, vehicle + rimonabant, H89 + vehicle, and H89 + rimonabant) by analysis of variance and multiple comparisons (**, $p < 0.01$; compared from siCON + vehicle or vehicle-treated group). Left LXR$\alpha$ control blot is also the control blot for pThr-LXR$\alpha$ (Fig 6D, right). WCL, whole cell lysate. (Legend continues as in original.)

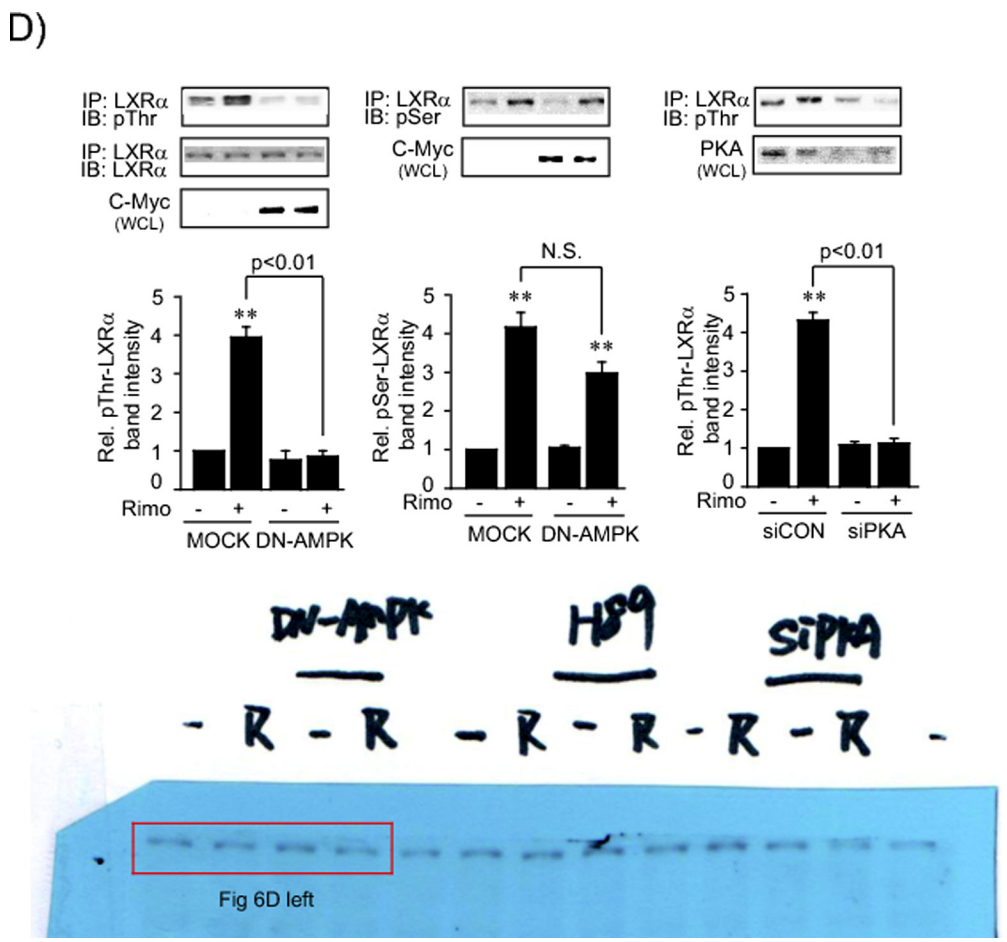


**Fig. 6.** (Legend begins as in original.) D, effect of DN-AMPK transfection or PKA knockdown (100 pmol/ml, 48 h) on LXR$\alpha$ phosphorylation by rimonabant. LXR$\alpha$ immunoprecipitates (IP) were immunoblotted (IB) with anti-phosphorylated threonine (pThr) or anti-phosphorylated serine (pSer) antibody. The LXR$\alpha$ control blot shown in D, left, is for pSer-LXR$\alpha$ (D, middle). The LXR$\alpha$ control blot shown in Fig. 4A, left, is for pThr-LXR$\alpha$ (D, right). Immunoblots for c-*myc* confirmed DN-AMPK overexpression. After verifying equal loading of proteins in each experiment by immunoblotting of LXR$\alpha$ immunoprecipitates for LXR$\alpha$, the relative protein levels of pSer-LXR$\alpha$ or pThr-LXR$\alpha$ from at least three separate experiments were compared among four treatment groups in each experimental set (i.e., MOCK + vehicle, MOCK + rimonabant, DN-AMPK + vehicle, and DN-AMPK + rimonabant; or siCON + vehicle, siCON + rimonabant, siPKA + vehicle, and siPKA + rimonabant) by analysis of variance and multiple comparisons.

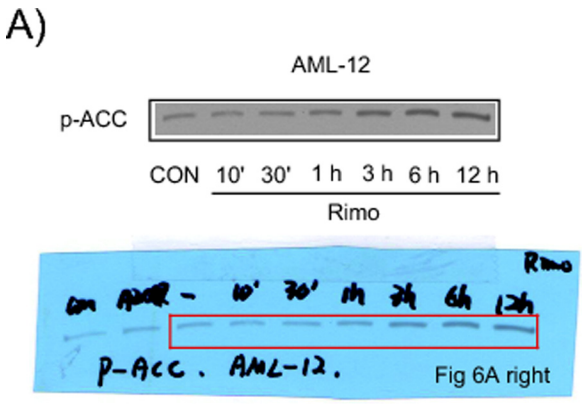


**Fig. 6A.** A, AMPK activation by rimonabant (Rimo).