



Corrigendum: Lipoxin A4 Inhibits NLRP3 Inflammasome Activation in Rats With Non-compressive Disc Herniation Through the JNK1/Beclin-1/PI3KC3 Pathway

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A Corrigendum on

Lipoxin A4 Inhibits NLRP3 Inflammasome Activation in Rats With Non-compressive Disc Herniation Through the JNK1/Beclin-1/PI3KC3 Pathway

by Jin, J., Xie, Y., Shi, C., Ma, J., Wang, Y., Qiao, L., et al. (2020). Front. Neurosci. 14:799. doi: 10.3389/fnins.2020.00799

In the original article, there was a mistake in the legend for **Figure 6** as published. **The results were error in Figure 6A, so the legend of Figure 6 was revised**. The correct legend appears below.

**Figure 6. The expression of autophagy- and apoptosis-related protein expression in spinal neuron cells by western blot. *p < 0.05 compared with the control group; *p < 0.05 compared with the model group; $^{\land}p < 0.05$ compared with the LXA4 group. Experiments were repeated three times for each group. Data are presented as means \pm SD. One-way analysis of variance (ANOVA) was used for comparisons among groups followed by Dunnett's t-test. **

In the original article, there was a mistake in **Figure 6** as published. **The results of Figure 6A were error, so the results of Figure 6A were deleted in Figure 6 **. The corrected *Figure 6** appears below.

In the original article, there was an error. **The levels of TNF (orb79138-480), IL-1 β (orb79117), IL-18 (orb107403), IL-4 (orb303658), IL-10 (orb76364), and TGF- β 1 (orb7087) (all from Biorbyt, Cambridge, United Kingdom) in the spinal dorsal horn, dorsal root ganglion, and spinal neurons were measured following the instructions of the respective ELISA kits.**

A correction has been made to **Materials and Methods**, **ELISA**, **Page 3**:

1

**The levels of TNF- α (orb452907), IL-1 β (orb453587), IL-18 (orb107403), IL-4 (orb303658), IL-10 (orb76364), and TGF- β 1 (orb7087) (all from Biorbyt, Cambridge, United Kingdom) in

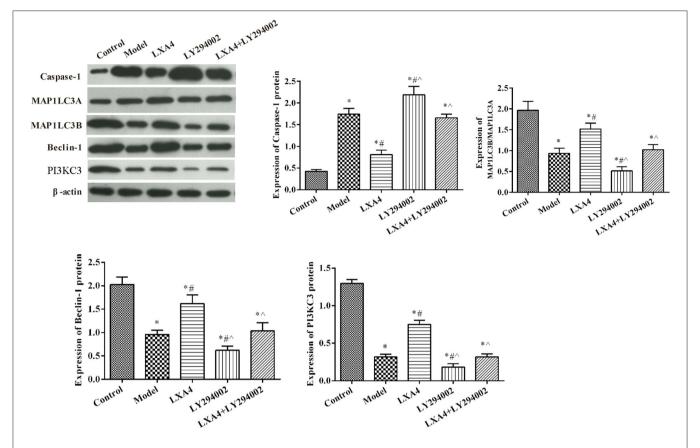


FIGURE 6 | The expression of autophagy- and apoptosis-related protein expression in spinal neuron cells by western blot. $^{\circ}P < 0.05$ compared with the control group; $^{\circ}P < 0.05$ compared with the model group; $^{\circ}P < 0.05$ compared with the LXA4 group. Experiments were repeated three times for each group. Data are presented as means \pm SD. One-way analysis of variance (ANOVA) was used for comparisons among groups followed by Dunnett's t-test.

the spinal dorsal horn and dorsal root ganglion were measured following the instructions of the respective ELISA kits. **

In the original article, there was an error. **The Effect of LXA4 on the Expression of the NLRP3 Inflammasome and Autophagy-Related Proteins in TNF- α -Induced Neuronal Cells *in vitro***

A correction has been made to **Results**, **The Effect of LXA4 on the Expression of the NLRP3 Inflammasome and Autophagy-Related Proteins in TNF- α -Induced Neuronal Cells in vitro**, **Page 9**:

The Effect of LXA4 on the Expression of Autophagy-Related Proteins in TNF- α -Induced Neuronal Cells *in vitro*

In the original article, there was an error. **The levels of proinflammatory (TNF- α , IL-1 β , and IL-18) and anti-inflammatory (IL-4, IL-10, TGF- β) mediators are shown in **Figure 6A**. Compared with control group, the levels of TNF- α , IL-1 β , and IL-18 clearly increased in other groups (p < 0.05). Administration of LXA4 led to a marked reduction in the expression levels of TNF- α , IL-1 β , and IL-18 compared with the model group, while the effect of LXA4 was weakened by LY294002 (p < 0.05). Compared with control group, the levels of TNF- α , IL-1 β , and IL-18 clearly obviously decreased in other groups (p < 0.05). Meanwhile, the expression of IL-4, IL-10, and TGF- β was significantly increased in the LXA4

group compared with the model group (p < 0.05). Similar to the *in vivo* results, LAX4 treatment markedly upregulated the contents of anti-inflammatory factors and weakened the effect of LY294002. The expression of autophagy-related proteins were also measured *in vitro* (**Figure 6B**). Compared with the control group, the expression of MAP1LC3B/MAP1LC3A, Beclin-1, and PI3KC3 was significantly decreased in other groups (p < 0.05). The expression of caspase-1 was significantly increased after TNF- α stimulated, compared with control group (p < 0.05). LY294002 administration further decreased the expression levels of these proteins. Meanwhile, treatment with LXA4 significantly increased the expression of autophagy-related proteins and weakened the effect of LY294002 (p < 0.05).**

A correction has been made to **Results**, **The Effect of LXA4 on the Expression of Autophagy-Related Proteins in TNF- α -Induced Neuronal Cells in vitro**, **Page 10**:

The expression of autophagy-related proteins were also measured *in vitro* (Figure 6**). Compared with the control group, the expression of MAP1LC3B/MAP1LC3A, Beclin-1, and PI3KC3 was significantly decreased in other groups (p < 0.05). The expression of caspase-1 was significantly increased after TNF- α stimulated, compared with control group (p < 0.05). LY294002 administration further decreased the expression levels

of these proteins. Meanwhile, treatment with LXA4 significantly increased the expression of autophagy-related proteins and weakened the effect of LY294002 (p < 0.05). **

The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

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