CORRIGENDUM

DOI: 10.3892/ijmm.2021.4843

Sevoflurane post-conditioning reduces rat myocardial ischemia reperfusion injury through an increase in NOS and a decrease in phosphorylated NHE1 levels

JIANFANG CAO, HONG XIE, YING SUN, JIANG ZHU, MING YING, SHIGANG QIAO, QIN SHAO, HAORONG WU and CHEN WANG

Int J Mol Med 36: 1529‑1537, 2015; doi: 10.3892/ijmm.2015.2366

Following the publication of the above article, an interested reader drew to the authors' attention that the data shown for the I/R and L‑NAME experiments in Fig. 2A appeared to be strikingly similar. After having re-examined their raw data, the authors realized that the data panel of the L‑NAME group was inadvertently loaded incorrectly, resulting in a duplication of the I/R data in the Figure.

The revised version of Fig. 2, containing the correct data for the L‑NAME group in Fig. 2A, is shown below. The authors are grateful to the Editor of International Journal of Molecular Medicine for granting them the opportunity to publish this Corrigendum, and stress that this error did not significantly affect either the results or the conclusions of the paper. All the authors agree with the publication of this Corrigendum, and apologize to the readership for any inconvenience caused.

A one‑way ANOVA followed by Tukey's post-hoc test was used to determine statistical significance. *P<0.05 vs. I/R. Sham, sham-operated group; SEVO, sevoflurane group (no I/R injury); I/R, ischemia/reperfusion injury group; SEVOP, sevoflurane post-conditioning group; L‑NAME, group treated with NG‑nitro‑L‑arginine methyl ester (NOS inhibitor); TTC, triphenyltetrazolium chloride.

Figure 2. Sevoflurane post-conditioning decreases infarct size which is increased by myocardial ischemia/reperfusion injury (MIRI) in rats. (A) Representative TTC-stained images and (B) quantification of the percentage of the injured area. The columns and error bars represent the means ± SD; n=6 experiments. This work is licensed under a Creative Commons Attribution 4.0 International (CC BY 4.0) License.