

Erratum

In the original article by Panico, et al., entitled „Cardiac Inflammation after Ischemia-Reperfusion of the Kidney: Role of the Sympathetic Nervous System and the Renin-Angiotensin System“ [Cell Physiol Biochem 2019;53(4):587-605, DOI: 10.33594/00000159], there has been a mistake in Fig. 7:

In Figure 7B, the representative blot of Figure 7A, which corresponds to Angiotensin II type 1 receptor (AT1R), has been repeated. The authors have corrected the figure, which now shows a proper representative blot of Angiotensinogen/Angiotensin II (AGT/Ang II). The correct Fig. 7 is shown below.

The authors confirm that all of the results and conclusions of the article remain unchanged, as well as the figure legend.

The authors sincerely apologize for this mistake.

Fig. 7. Ang II type 1 receptor density and angiotensinogen/Ang II level in heart tissue after renal I/R. Data are expressed as mean ± SD. The number of experiments using different preparations is indicated within or above each bar. Mice from Sham (empty bars) and I/R (filled bars) groups were treated with or without different drugs, as indicated on the abscissae. (A) AT1R abundance. Upper panel: Representative immunodetection for AT1R and glyceraldehyde-3-phosphate dehydrogenase (GAPDH) as protein loading control. Lower panel: Graphical representation. (B) Angiotensinogen (AGT)/Ang II abundance and GAPDH as protein loading control. Upper panel: Representative immunodetection for AGT/Ang II and GAPDH as protein loading control. Lower panel: Graphical representation. **p<0.01, ***p<0.001, ****p<0.0001, as assessed using onefactor ANOVA followed by Bonferroni’s test for selected pairs. Upper bands in A (a, b, c) and B (g, h, i) show for comparison 3 different representative immunodetections developed in separate experiments. Lower bands in A and B (d, e, f) show loading controls, which were the same for A and B.

