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Erratum

In the original article by Vera, et al., entitled „Antioxidant and Anti-Inflammatory Strategies Based on the Potentiation of Glutathione Peroxidase Activity Prevent Endothelial Dysfunction in Chronic Kidney Disease“ [Cell Physiol Biochem 2018;51(3):1287-1300, DOI: 10.1159/000495540], the figures Fig. 3, Fig. 4 and Fig. 5 have not been displayed correctly.
The correct figures are displayed below, and the results of this article are not affected by this.

The authors sincerely apologize for this mistake.

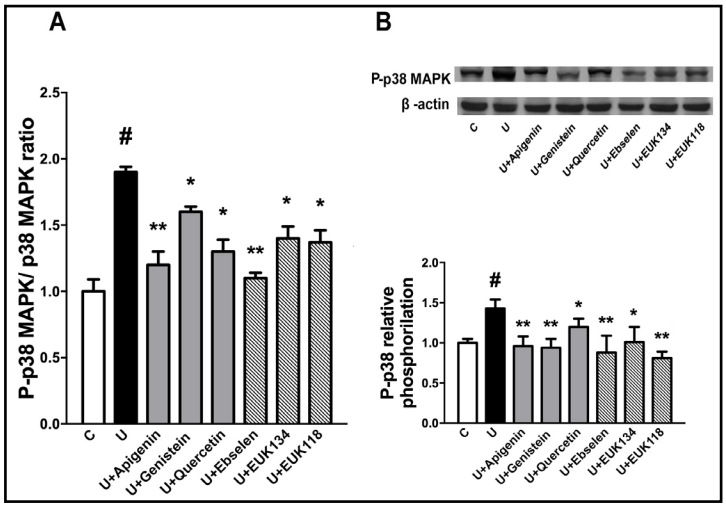

Fig. 3. Activation of p38MAPK signaling pathway by uremic sera is inhibited by flavonoids and antioxidant mimetics. ECs were incubated (15 min) with sera from control donors (C), and uremic patients (U) in the absence and presence of apigenin, genistein, quercetin, ebselen, EUK134 and EUK118 (n=5). A. Bar diagrams represent the ratios of phosphorylated p38 MAPK relative to the total protein, obtained from antibody cell-based ELISA technique. B. Phospho-p38 MAPK in ECs was also assessed by Western-Blot. Images are representative of 4 different experiments. Results were quantified and represented as Mean±SD by bar diagram. # p<0.01 vs control and *p<0.05, **p<0.01 vs uremia.

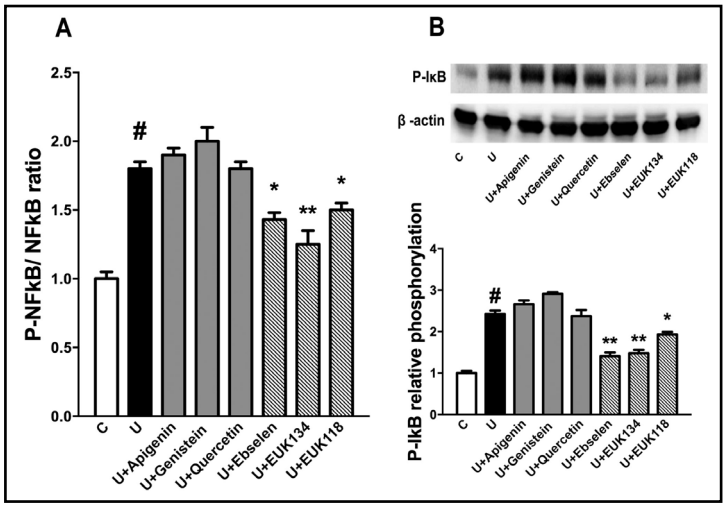

Fig. 4. Activation of NFκB (p65) signaling pathway by uremic sera is prevented by antioxidant enzyme mimetics. ECs were incubated (15 min) with sera from control donors (C), and uremic patients (U) in the absence and presence of apigenin, genistein, quercetin, ebselen, EUK134 and EUK118 (n=4). A. Ratios of phosphorylated NFκB (p65) relative to the total protein. Data are corrected for cell number and are represented as Mean±SD. B. IκB was assessed by Western-Blot in ECs previously treated with ALLN (50 μg/ml, 30 min, 37°C). Images are representative of 4 different experiments. # p<0.01 vs control and *p<0.05, **p<0.01 vs uremia.

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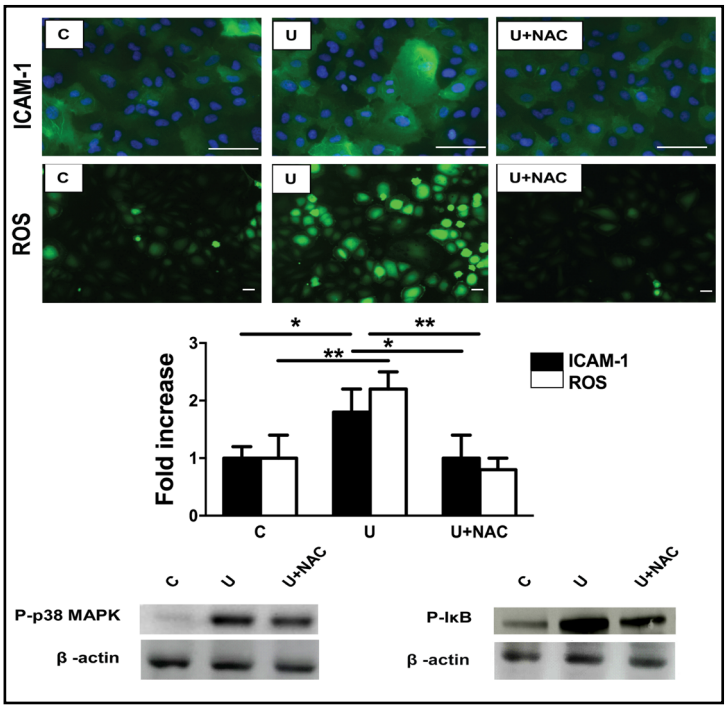

Fig. 5. Anti-inflammatory and antioxidant effects of NAC on endothelial cells exposed to uremic sera. Micrographs show ICAM-1 surface expression and ROS production in ECs exposed to control media (C), uremia (U) and uremia treated with NAC (U+NAC). Bar diagrams show the effect of the uremic sera (# $p<0.05$ vs control) and the NAC treatment (*$p<0.05$, **$p<0.01$ vs uremia) (n=4). NAC (10mM) inhibited phosphorylation of p38MAPK and activation of NFκB (n=4).