CORRIGENDUM

The Effect of A1 Adenosine Receptor in Diabetic Megalin Loss with Caspase-1/IL18 Signaling [Corrigendum]


The authors of this paper have advised that Figures 5 and 6 are incorrect. The authors provided only one actin control for each treatment. However, their different target proteins had their own matched actin from the same experiment. This does not change the conclusion of the paper. The authors apologize for this error. The correct Figures 5 and 6 are as follows:

Figure 5 A1 adenosine receptor (A1AR) deletion aggravated diabetes-induced renal inflammation by activation caspase-1 and interleukin (IL)-18. (A) A1AR activation was observed in WT-DN mice, compared to WT-control mice by Western blotting. (E) Showed its quantitative analysis. (B1–F1) Showed caspase-1 protein expression by Western blotting and its quantitative analysis. (C) Compared to WT-control, analyzed protein expression increased in WT-DN mice, while further more increasing in A1AR knockout (KO)-DN mice than in WT-DN mice. (B2–F2) Showed IL-18 protein expression by Western blotting and its quantitative analysis. (D) Compared to WT-control, analyzed protein expression increased in WT-DN mice, while further more increasing in A1AR knockout (KO)-DN mice than in WT-DN mice. Data are shown as Mean ± SEM; n=6 per group.

Figure 6 A1 adenosine receptor (A1AR) deletion aggravated diabetes-induced renal inflammation by activation caspase-1 and interleukin (IL)-18. (A) A1AR activation was observed in WT-DN mice, compared to WT-control mice by Western blotting. (E) Showed its quantitative analysis. (B1–F1) Showed caspase-1 protein expression by Western blotting and its quantitative analysis. (C) Compared to WT-control, analyzed protein expression increased in WT-DN mice, while further more increasing in A1AR knockout (KO)-DN mice than in WT-DN mice. (B2–F2) Showed IL-18 protein expression by Western blotting and its quantitative analysis. (D) Compared to WT-control, analyzed protein expression increased in WT-DN mice, while further more increasing in A1AR knockout (KO)-DN mice than in WT-DN mice. Data are shown as Mean ± SEM; n=6 per group.
**Figure 6** The role of A1AR antagonist DPCPX and agonist CCPA in caspase-1/IL18 activation and megalin loss in HK2 cell cocultured with high glucose. (A, B) Protein expression of caspase-1 (A1, B1), IL-18 (A2, B2), and megalin (A3, B3) in high glucose, high glucose with DPCPX and CCPA by Western blotting. (C, D, E) Quantitative analysis of caspase-1 (C), IL-18 (D), and megalin (E) showed the upregulation of caspase-1/IL-18 and downregulation of megalin expression in high glucose. DPCPX further increased caspase-1/IL-18 expression and decreased megalin expression. However, CCPA inhibited caspase-1/IL-18 expression and improved megalin loss obviously. Data are shown as Mean ± SEM; *P<0.05 HG +DPCPX vs HG, #P<0.01 HG vs NG, **P<0.05 HG+CCPA vs HG. n=4 per group.

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