

CORRIGENDUM

In Chi et al.,¹ the published article contains errors in [Figures 3](#) and [8](#). The bar-graph ([Figure 3F](#)) was accidentally misused as [Figure 3G](#). The immunofluorescence picture of the control group ([Figure 2G](#)) was accidentally misused in shHOTAIR group ([Figure 8B](#)). The corrected figures and their legends are shown below. The authors confirmed all results and conclusions of this article remain unchanged.

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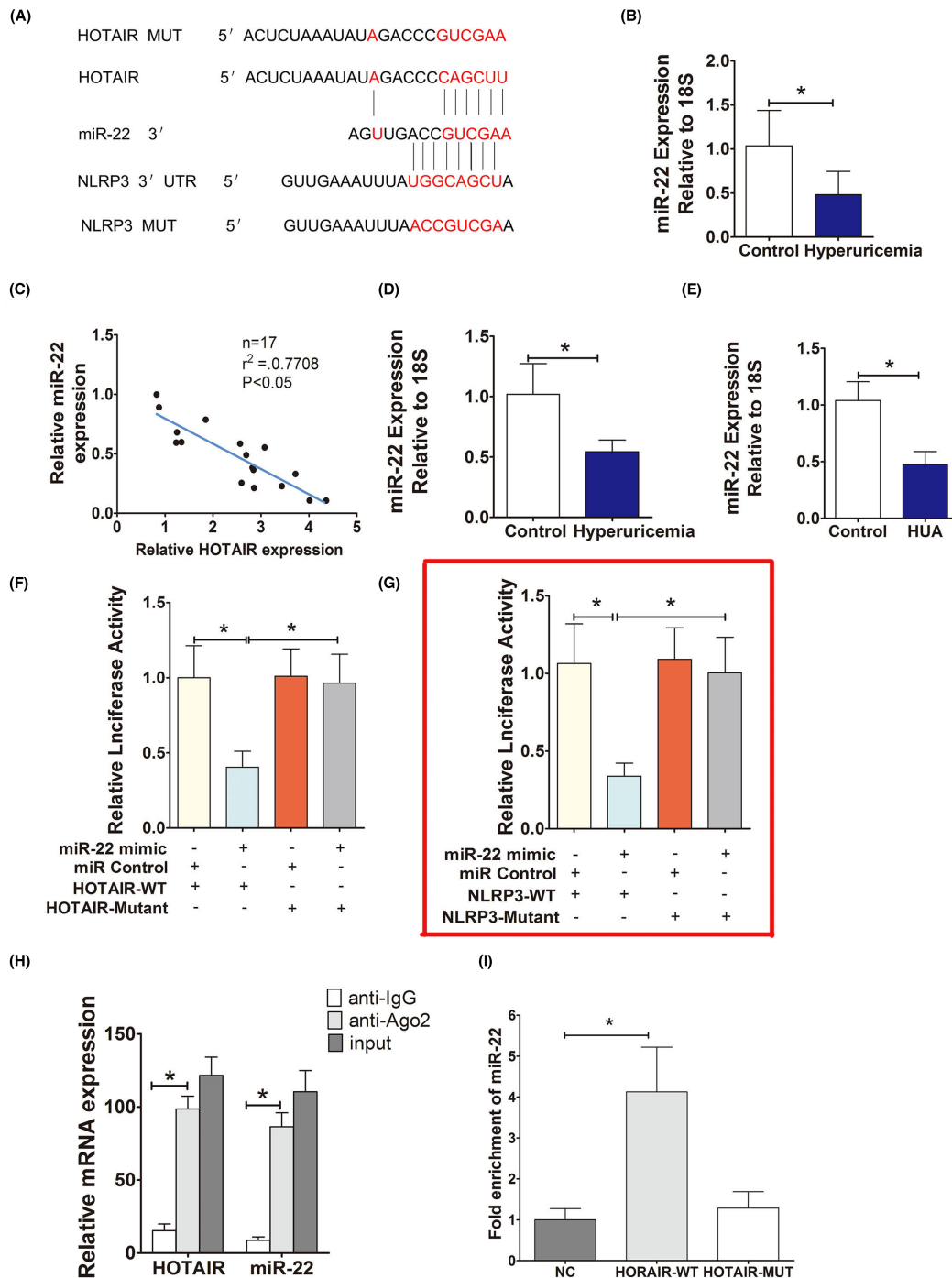


FIGURE 3 HOTAIR, as a ceRNA, regulates the expression of miR-22. (A) The sequences of HOTAIR and NLRP3 aligned with miR-22, including the wildtype (WT) and a mutant. Schematic illustration of the presumed target site for HOTAIR and NLRP3 in miR-22. (B) Serum levels of miR-22 in normal controls and hyperuricemia patients, measured by qPCR. The expression of miR-22 in hyperuricemia group was significantly downregulated and negatively correlated with the expression of HOTAIR; $*p < 0.05$ compared to the control group; $n = 17$ in each group. (C) Correlation analysis between HOTAIR and miR-22 levels in normal controls and hyperuricemia patients. miR-22 was negatively correlated with HOTAIR ($r^2 = -0.77$); $*p < 0.05$ compared to the normal controls; $n = 17$ in each group. (D, E) The levels of miR-22 in hyperuricemia mice ($n = 6$) and HUVECs ($n = 3$), measured by qPCR. The expression of miR-22 in HUA mice and HUA-stimulated HUVECs was significantly downregulated; $*p < 0.05$ compared to the control group; $n = 6$ in each group. (F) Luciferase activity results. There is direct binding between HOTAIR and miR-22; $*p < 0.05$ versus the mimic NC+HOTAIR-WT group; $n = 3$ in each group. (G) Luciferase activity results. miR-22 directly regulates the expression of NLRP3, $*p < 0.05$ compared to the mimic NC+NLRP3-WT group; $n = 3$ in each group. (H) RIP assays using cell lysate IgG or anti-Ago2 as the input. Relative expression levels of HOTAIR and miR-22 in HUVECs were detected by qPCR and normalized to 18s. The results indicated higher HOTAIR and miR-22 RNA levels in Ago2 immunoprecipitates relative to control IgG immunoprecipitates; $*p < 0.05$ compared to the anti-IgG group; $n = 3$ in each group. (I) Detections of miR-22 using qRT-PCR in the same sample pulled down by biotinylated HOTAIR and NC probe. miR-22 expression was significantly higher in the HOTAIR-WT group; $*p < 0.05$ compared to NC group; $n = 3$ in each group.

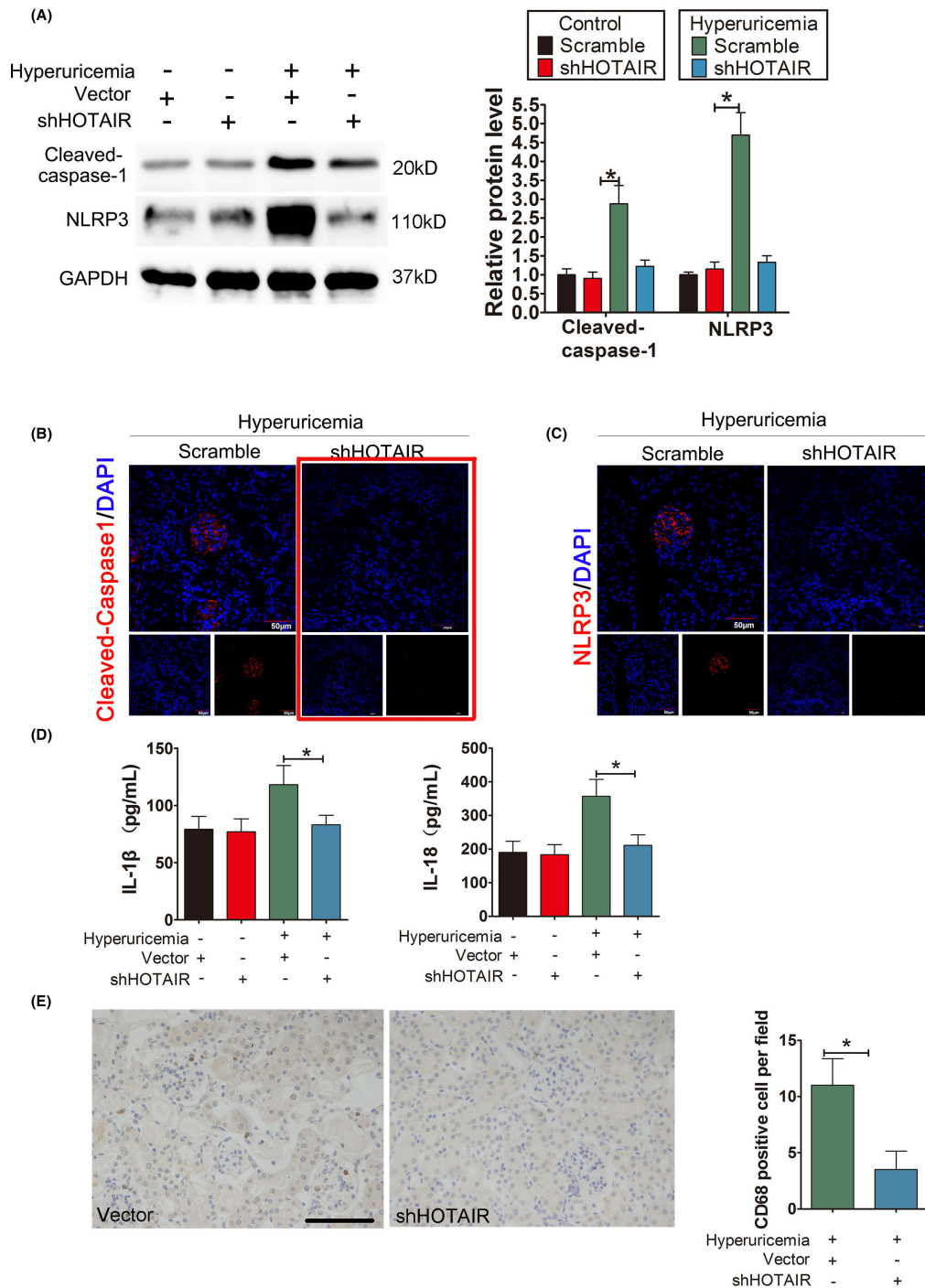


FIGURE 8 Knockdown of HOTAIR ameliorates renal inflammation in HUA mice. (A) The protein levels of caspase-1, NLRP3, GSDMD-N and GSDMD-FL, as measured by Western blot analysis; quantification normalized to GAPDH in renal tissue from hyperuricemia mice after shRNA treatment. The protein levels of caspase-1, NLRP3 decreased remarkably in hyperuricemia+shHOTAIR group; $p < 0.05$ compared to the hyperuricemia+vector group; $n = 6$ in each group. (B, C) Immunofluorescence images showing the expression of caspase-1, NLRP3 in renal tissue from hyperuricemia mice after shRNA treatment. Tissue immunofluorescence showed that the fluorescence intensity of glomerular caspase-1 and NLRP3 in the shRNA-treated group was significantly lower than that in the hyperuricemia+shHOTAIR group; $p < 0.05$ compared to the control group; $n = 6$ in each group (scale bar, 50 μm ; magnification, 400 \times); blue: nuclear staining (DAPI), red: caspase-1 and NLRP3 staining. (D) The serum levels of IL-1 β and IL-18, measured by ELISA. The serum levels of IL-1 β and IL-18 decreased remarkably in the hyperuricemia+shHOTAIR group; $p < 0.05$ compared to the in the hyperuricemia+Vector group; $n = 6$ in each group. (E) CD68 immunohistochemistry of the renal interstitium and glomerular mesangial area in the Hyperuricemia+vector group and hyperuricemia+shHOTAIR group. Immunohistochemistry of CD68 in renal tissue from hyperuricemia mice after shRNA treatment. Compared with that in the hyperuricemia+Vector group, the downregulation of HOTAIR by shHOTAIR significantly reduced the infiltration of positive CD68 macrophages in the renal interstitium and glomerular Mesangial area in hyperuricemia+shHOTAIR group; $p < 0.05$ compared to hyperuricemia+vector group; $n = 6$ in each group (scale bar, 100 μm ; magnification, 400 \times).

REFERENCE

1. Chi K, Geng X, Liu C, et al. LncRNA-HOTAIR promotes endothelial cell pyroptosis by regulating the miR-22/NLRP3 axis in hyperuricaemia. *J Cell Mol Med*. 2021;25(17):8504-8521. doi:[10.1111/jcmm.16812](https://doi.org/10.1111/jcmm.16812)