

Correction for: Silencing of long non-coding RNA H19 downregulates CTCF to protect against atherosclerosis by upregulating PKD1 expression in ApoE knockout mice

Yongyao Yang^{1,*}, Feng Tang^{1,*}, Fang Wei¹, Long Yang¹, Chunyan Kuang¹, Hongming Zhang², Jiusheng Deng³, Qiang Wu¹

¹Department of Cardiology, Guizhou Provincial People's Hospital, Guiyang 550002, P. R. China

²Department of Cardiology, The General Hospital of Ji'nan Military Region, Ji'nan 250031, P. R. China

³Department of Pathology and Laboratory Medicine, Emory University School of Medicine, Atlanta, GA 30322, USA

*Equal contribution

Correspondence to: Qiang Wu; email: 729142921@qq.com

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This article has been corrected: The authors replaced the “oe-H19 + oe-CTCF + oe-PKD1” panel of the HE staining in **Figure 5C**, which was accidentally mislabeled and partially duplicated with the “oe-NC + oe-NC + oe-NC” image. Replacement was done using representative images from the original sets of experiments. This alteration does not affect the results or conclusions of this work.

The new **Figure 5** is presented below.

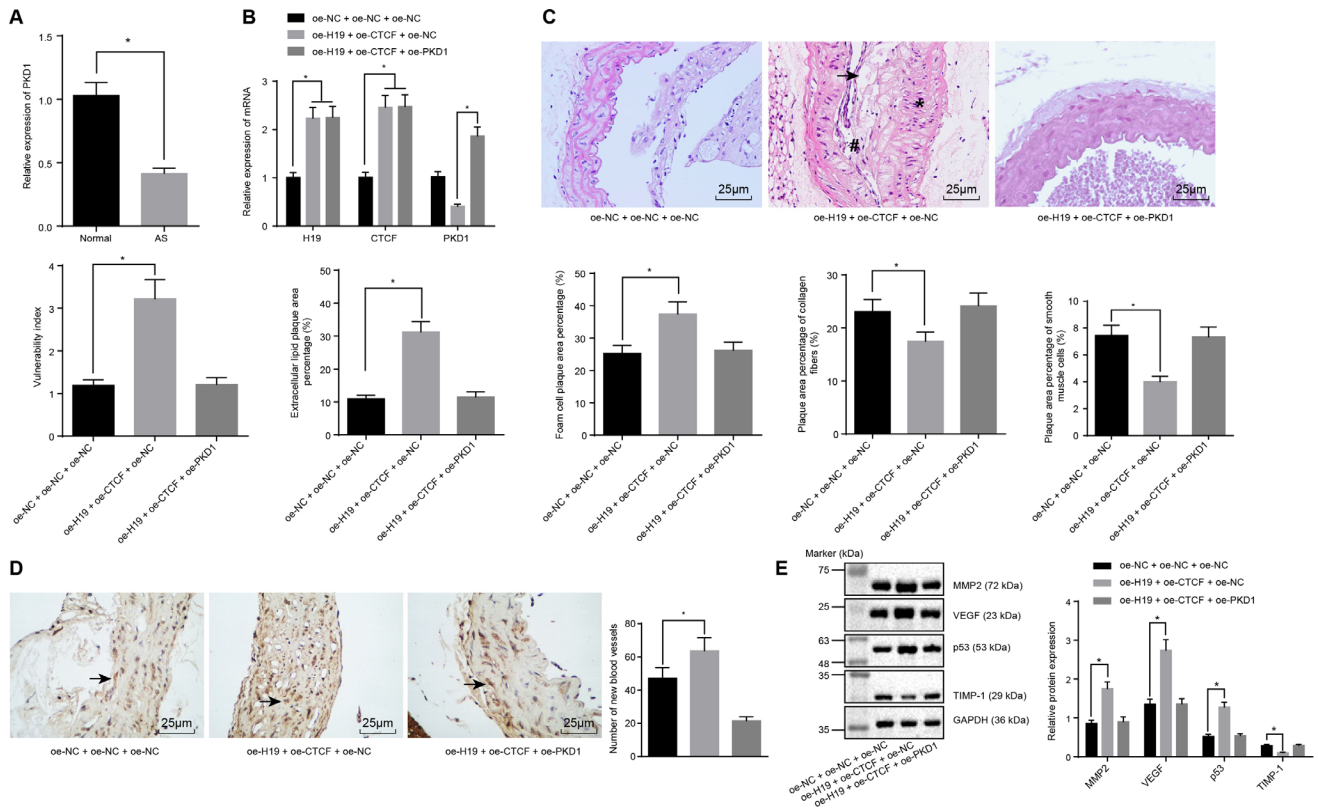


Figure 5. H19 is involved in atherosclerotic vulnerable plaque formation and intraplaque angiogenesis through down-regulating PKD1 by recruiting CTCF in ApoE knockout mice with AS. (A) The expression pattern of PKD1 in the aortic tissues of normal and AS mice determined by RT-qPCR. * $p < 0.05$ vs. the control group. (B) The overexpressing efficiency of H19, CTCF and PKD1 assessed by RT-qPCR. * $p < 0.05$ vs. the oe-NC + oe-NC + oe-NC group; # $p < 0.05$ vs. the oe-H19 + oe-CTCF + oe-NC group. (C) The atherosclerotic vulnerable plaque formation evaluated by HE staining ($\times 400$) (The arrow referred to lipid vacuoles, * represented inflammatory cells and # indicated fractured smooth muscle). (D) The number of new blood vessels measured by Immunohistochemical staining ($\times 400$) (The arrow referred to CD34-positive cells). (E) The protein levels of MMP-2, VEGF, p53 and TIMP-1 in atherosclerotic plaques normalized to GAPDH after transfection determined by Western blot analysis. * $p < 0.05$ vs. the oe-NC + oe-NC + oe-NC group. The data were measurement data and expressed by mean \pm standard deviation. Data differences between two groups were analyzed by unpaired t -test; comparisons made among multiple groups were analyzed by one-way ANOVA. The experiments were repeated three times independently.