JCI The Journal of Clinical Investigation

Genetic inactivation of IL-1 signaling enhances atherosclerotic plaque instability and reduces outward vessel remodeling in advanced atherosclerosis in mice

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J Clin Invest. 2012;122(2):783-783. https://doi.org/10.1172/JCI62827.

Erratum

Original citation: J. Clin. Invest. 2012;122(1):70–79. doi:10.1172/JCI43713. Citation for this erratum: J. Clin. Invest. 2012;122(2):783. doi:10.1172/JCI62827. During the preparation of this manuscript, errors were inadvertently introduced into the legends for Figures 1, 2, and 3. The correct sections of the legends appear below. Figure 1: (B) Quantification of total atherosclerotic plaque area within the aortic root of Il1r1+/+Apoe–/– and Il1r1–/–Apoe–/– mice at 150-μm intervals from the aortic valve attachment site ($P < 0.001$ for difference between genotypes by Scheirer-Ray-Hare test). n = 13, Il1r1+/+Apoe–/–; n = 12, Il1r1–/–Apoe–/–. Data represent mean ± SEM. Figure 2: L-1R1 deficiency reduces compensatory outward remodeling of atherosclerotic brachiocephalic arteries. (A) Movat staining of representative brachiocephalic arteries of Il1r1–/–Apoe–/– and Il1r1+/+Apoe–/– mice. Scale bars: 200 μm. (B–D) Atherosclerotic plaque area (B), vessel area within the IEL ($P < 0.001$ for difference between genotypes by 2-way ANOVA) (C), and lumen area ($P < 0.001$ for difference between genotypes by 2-way ANOVA after square root transformation) (D) at multiple locations along the brachiocephalic arteries of Il1r1–/–Apoe–/– and Il1r1+/+Apoe–/– mice. n = 14, Il1r1+/+Apoe–/–; n = 12, Il1r1–/–Apoe–/–. Data in B–D represent mean ± SEM. Figure 3: (F–J) Quantification of (F) plaque collagen content based on picrosirius red staining, $P < 0.001$ for difference of genotypes by 2-way ANOVA, (G) plaque SMC coverage based on SM α-actin staining, […]

Erratum

Genetic inactivation of IL-1 signaling enhances atherosclerotic plaque instability and reduces outward vessel remodeling in advanced atherosclerosis in mice

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Figure 1: (**B**) Quantification of total atherosclerotic plaque area within the aortic root of *Il1r1*$^{+/+}$*Apoe*$^{-/-}$ and *Il1r1*$^{-/-}$*Apoe*$^{-/-}$ mice at 150-μm intervals from the aortic valve attachment site ($P < 0.001$ for difference between genotypes by Scheirer-Ray-Hare test). $n = 13$, *Il1r1*$^{+/+}$*Apoe*$^{-/-}$; $n = 12$, *Il1r1*$^{-/-}$*Apoe*$^{-/-}$. Data represent mean ± SEM.

Figure 2: L-1R1 deficiency reduces compensatory outward remodeling of atherosclerotic brachiocephalic arteries. (**A**) Movat staining of representative brachiocephalic arteries of *Il1r1*$^{-/-}$*Apoe*$^{-/-}$ and *Il1r1*$^{+/+}$*Apoe*$^{-/-}$ mice. Scale bars: 200 μm. (**B**–**D**) Atherosclerotic plaque area (**B**), vessel area within the IEL ($P < 0.001$ for difference between genotypes by 2-way ANOVA) (**C**), and lumen area ($P < 0.001$ for difference between genotypes by 2-way ANOVA after square root transformation) (**D**) at multiple locations along the brachiocephalic arteries of *Il1r1*$^{-/-}$*Apoe*$^{-/-}$ and *Il1r1*$^{+/+}$*Apoe*$^{-/-}$ mice. $n = 14$, *Il1r1*$^{+/+}$*Apoe*$^{-/-}$; $n = 12$, *Il1r1*$^{-/-}$*Apoe*$^{-/-}$. Data in **B**–**D** represent mean ± SEM.

Figure 3: (**F**–**J**) Quantification of (**F**) plaque collagen content based on picrosirius red staining, $P < 0.001$ for difference of genotypes by 2-way ANOVA, (**G**) plaque SMC coverage based on SM α-actin staining, $P < 0.001$ for difference of genotypes by the Scheirer-Ray-Hare test, (**H**) total plaque SMC content based on SM α-actin staining, $P < 0.001$ for difference of genotypes by the Scheirer-Ray-Hare test, (**I**) plaque macrophage content based on Mac2 staining, $P = 0.01$ for difference of genotypes by 2-way ANOVA after log transformation, and (**J**) the percentage of brachiocephalic arteries exhibiting intraplaque hemorrhage based on Movat and TER-119 staining, $^{**}P < 0.01$ by Fisher's exact test.

The *JCI* regrets the errors.