CORRECTION

Correction to: A high-cholesterol diet promotes steatohepatitis and liver tumorigenesis in HCV core gene transgenic mice

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In the original publication of the article, there is a mistake in Fig. 2e and the author would like to correct it. It has been corrected and replaced in the HTML and PDF versions of the manuscript.

The corrected Fig. 2 is given below.

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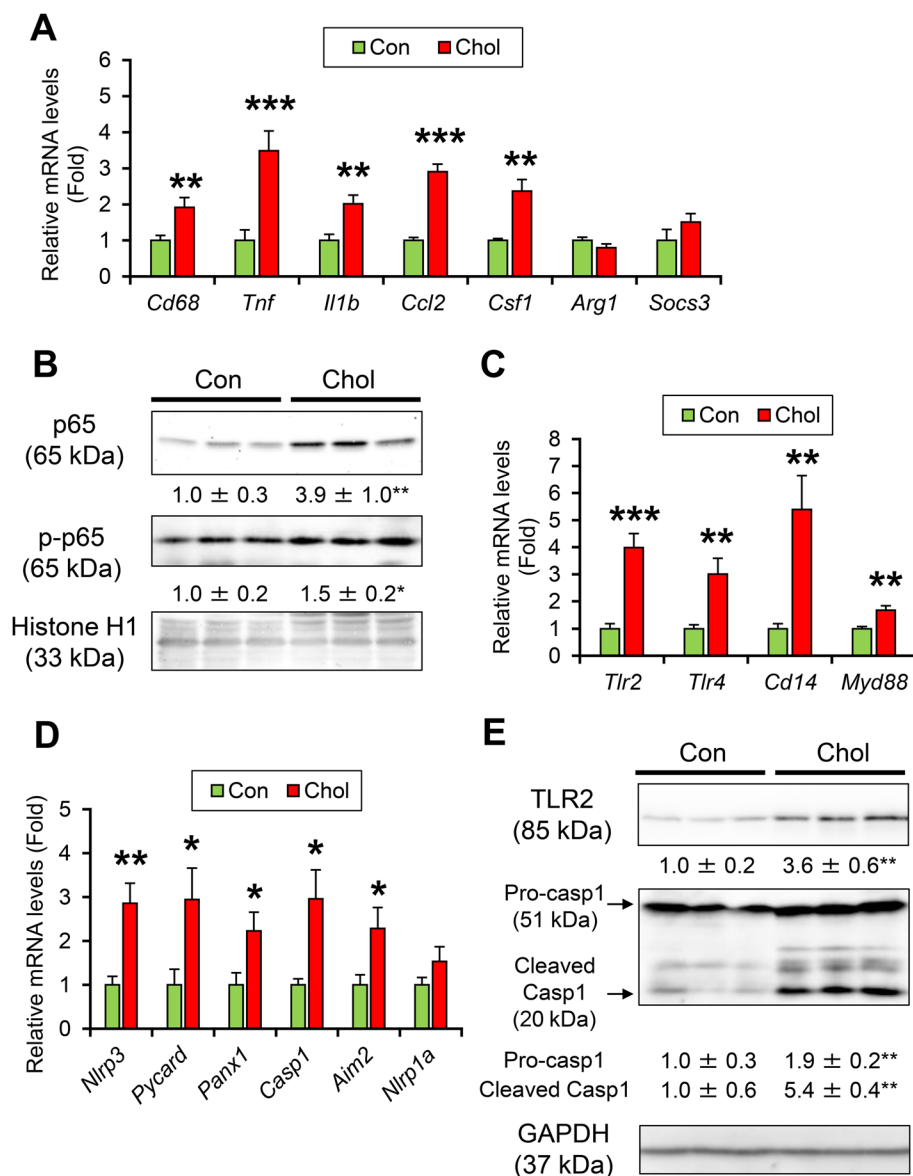8 Department of Gastroenterology, The University of Tokyo, Tokyo, Japan

Fig. 2 Aggravated hepatic inflammation in the liver of HCVcpTg mice after a 15-month high-cholesterol diet. **a** qPCR analysis of genes associated with inflammation. **b** Immunoblot analysis of NF-κB p65 and phosphorylated p65 (p-p65). Liver nuclear fractions (20 μg of protein) were loaded into each well. The band of histone H1 was used as a loading control. Band intensities were measured densitometrically, normalized to those of the loading controls, and subsequently expressed as values relative to those of control diet mice. Results were obtained from two independent immunoblot experiments. **c** qPCR analysis of genes associated with TLRs. **d** qPCR analysis of genes associated with inflammasomes. **e** Immunoblot analysis of TLR2 and caspase 1. Whole liver homogenates (45 μg of protein) were loaded into each well. The caspase 1 antibody could detect pro-caspase 1 and cleaved (activated) caspase 1. The band of GAPDH was used as a loading control. Band intensities were measured densitometrically, normalized to those of the loading controls, and subsequently expressed as values relative to those of control diet mice. Results were obtained from two independent immunoblot experiments. All mRNA levels were normalized to 18S ribosomal RNA levels and subsequently expressed as values relative to those of control diet mice. Values are expressed as the mean ± SEM. *$P<0.05$, **$P<0.01$, ***$P<0.001$ between control diet and high-cholesterol diet HCVcpTg mice