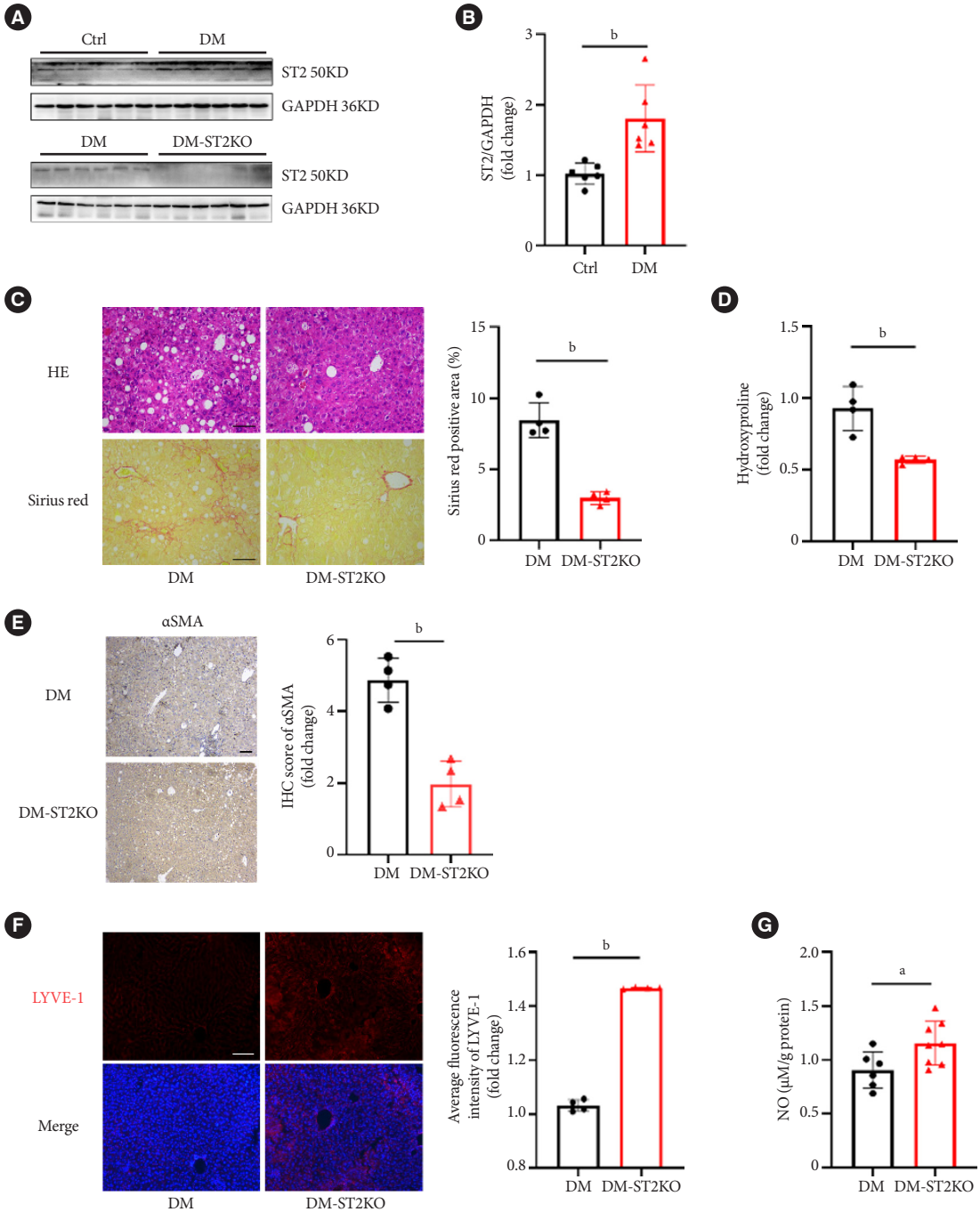

Supplementary Fig. 2. Liver sinusoidal endothelial cell (LSEC) dysfunction and liver fibrosis are reduced in diabetic mice in the absence of suppression of tumorigenicity 2 (ST2). (A, B) Western blot and measurement for ST2 of the whole liver ($n=6$). (C) Representative images of H&E and Sirius Red staining of liver sections from wild-type diabetic (DM) mice and ST2 knockout (KO) DM mice (left). Positive area of Sirius Red staining is used to quantified liver fibrosis (right) ($n=4$) (scale bar, 100 μm). (D) Hepatic hydroxyproline levels in mice ($n=4$). (E) Representative images and quantify of immunohistochemical (IHC) staining of alpha smooth muscle actin (αSMA) in liver sections ($n=4$) (scale bar, 100 μm). (F) Representative immunofluorescent images (left) and analysis (right) of vessel endothelial hyaluronan receptor 1 (LYVE-1) of liver sections ($n=4$) (scale bar, 100 μm). (G) Hepatic nitric oxide (NO) levels ($n=6$). Data was shown as mean±standard error of the mean. Ctrl, control; GAPDH, glyceraldehyde-3-phosphate dehydrogenase. [a]$P<0.05$, [b]$P<0.01$.