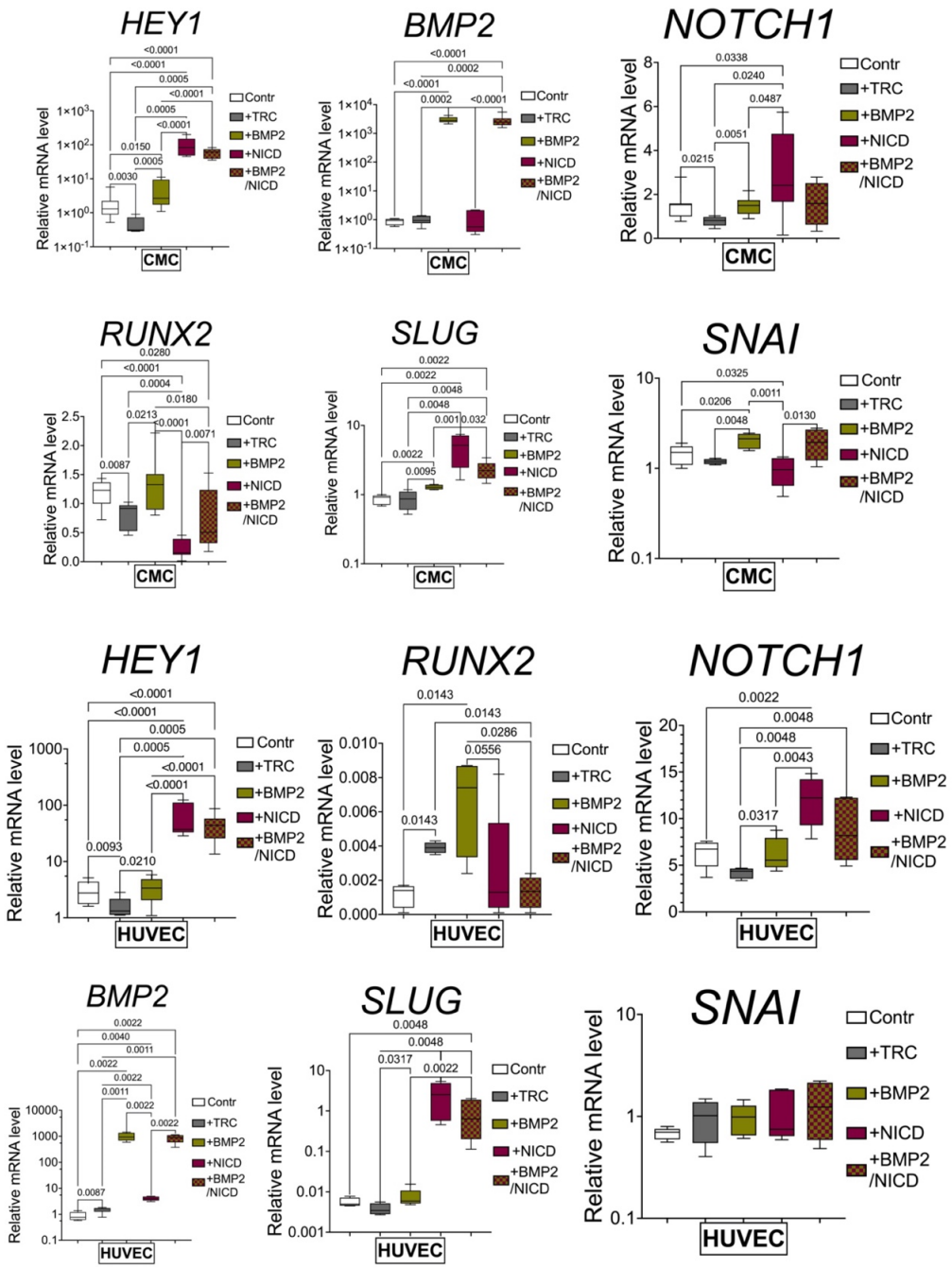

Figure S1 Exogenous activation of Notch signaling by the introduction of a lentiviral construct carrying *NICD* or/and *BMP2* sequences resulted in increased expression of signaling pathway components. Cells without induction were used as a negative control and the virus TRC was a vector that did not carry insert genes. The y-axis represents the relative amount of mRNA in each group, measured by the $2^{-\Delta\Delta Ct}$ method; box plots with whiskers at minimum to the maximum are presented. P values (numbers) and brackets show significant differences between groups at $P<0.05$ (unpaired nonparametric Mann-Whitney test). The experiment was repeated three times. CMC, cardiac mesenchymal cell; HUVEC, human umbilical vein endothelial cell; Contr, control; TRC, transduction control; BMP2, bone morphogenetic protein 2; NICD, NOTCH1 intracellular domain.

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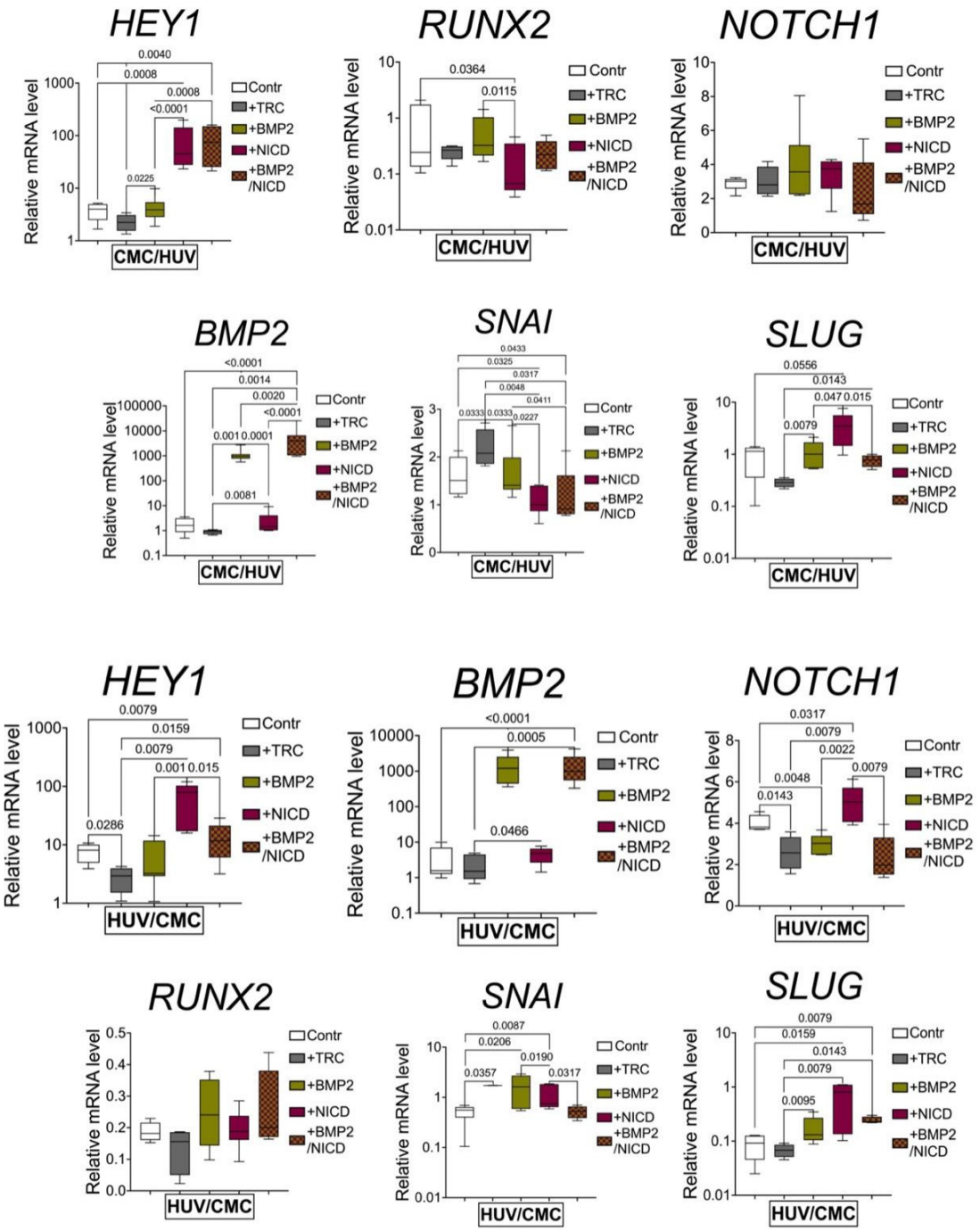

Figure S2 Activation of Notch and BMP2 by co-cultivation of CMCs and HUVECs in response to the introduction of a lentiviral construct carrying the *NICD* or/and *BMP2* sequences led to a decrease in the expression of target genes compared to monocultures. Cells without induction were used as a negative control, and the virus TRC was a vector that did not carry insert genes. The y-axis represents the relative amount of mRNA in each group, measured by the $2^{-\Delta\Delta Ct}$ method; box plots with whiskers at minimum to the maximum are presented. P values (numbers) and brackets show significant differences between groups at $P<0.05$ (unpaired nonparametric Mann-Whitney test). The experiment was repeated three times. CMC, cardiac mesenchymal cell; HUV, human umbilical vein endothelial cell; Contr, control; TRC, transduction control; BMP2, bone morphogenetic protein 2; NICD, NOTCH1 intracellular domain

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