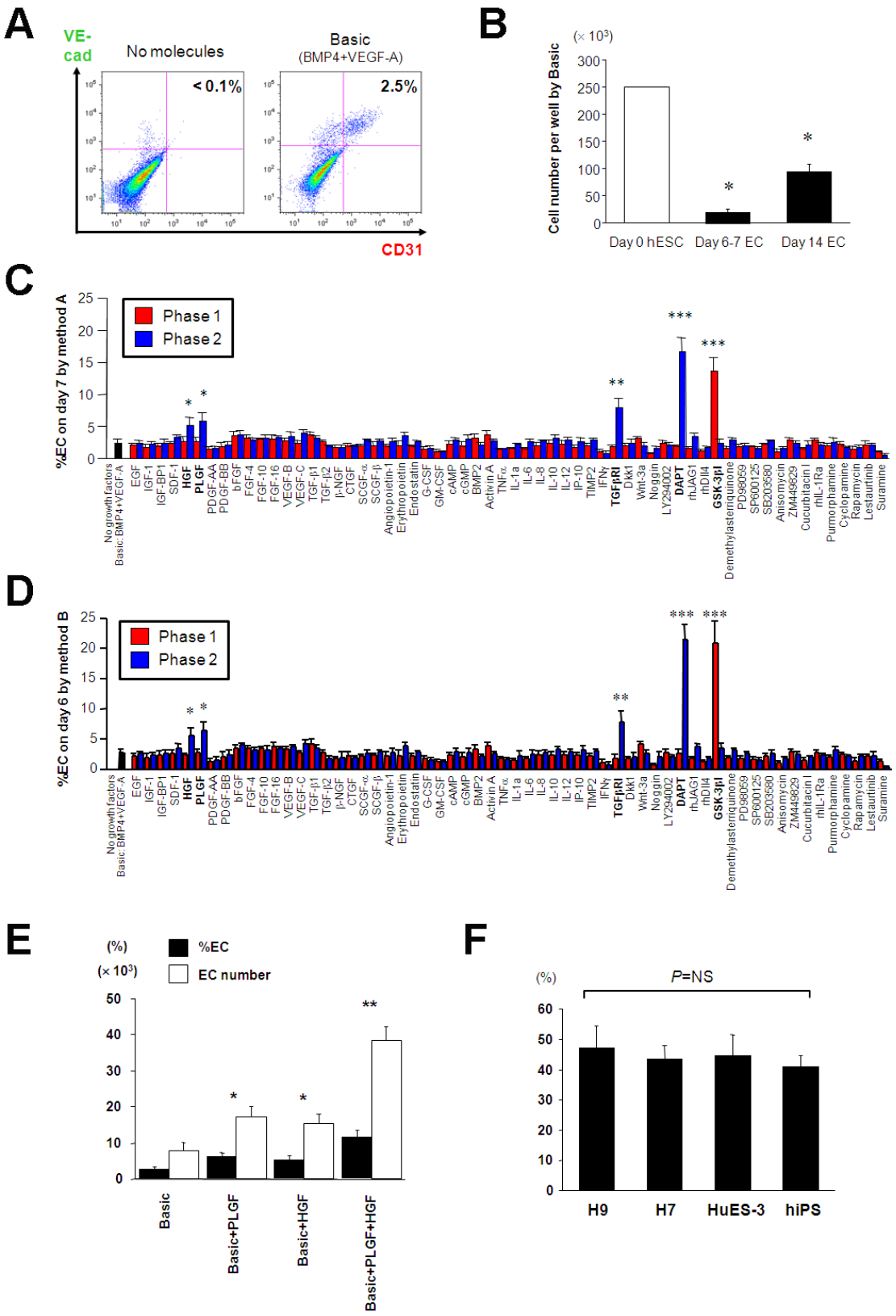

Supplementary information, Figure S2 Screening of bioactive small molecules for endothelial differentiation.

(**A**) Representative FACS results detecting the VEC-EGFP^{+}CD31^{+} ECs at the end of phase 2 when no molecules (*left*) or basic molecules (BMP4 plus VEGF-A; *right*) were administered in the culture of the endothelial differentiation. The induction ratio of VEC^{+}CD31^{+} ECs by BMP4 with VEGF-A was only 1–3%. (**B**) The yields of VEC^{+}CD31^{+} ECs generated with BMP4 plus VEGF-A at the end of phase 2 (day 6–7) and phase 3 (day 14) were less than the initial number of hESCs before differentiation. **P*<0.01 vs day 0 hESC. (**C** and **D**) The ratios of VEC^{+}CD31^{+} ECs at the end of phase 2, induced by all the bioactive chemical molecules tested in the method A (**C**) and method B (**D**). Among them, a γ-secretase inhibitor DAPT in phase 2 and a GSK-3β inhibitor (GSK-3βI; BIO or CP21R7) in phase 1 proved the significantly-enhanced endothelial differentiation. A TGFβ inhibitor (SB431542), HGF and PLGF in phase 2 also showed their modest efficacies, respectively. **P*<0.05, ***P*<0.01, and ****P*<0.0001 vs Basic (BMP4 plus VEGF-A). (**E**) Averaged percent EC (VEC^{+}CD31^{+}) and sorted EC number at the end of phase 2 by basic treatment (Basic; BMP4 plus VEGF-A) with or without PLGF and/or HGF in phase 2. **P*<0.05 and ***P*<0.01 vs Basic. (**F**) Averaged percent EC (VEC^{+}CD31^{+}) of each hESC (WA09[H9], H7, HuES-3) and hiPS line, induced by the combination treatment with BMP4/GSK-3βI in phase 1 and VEGF-A/DAPT in phase 2. NS, not significant.