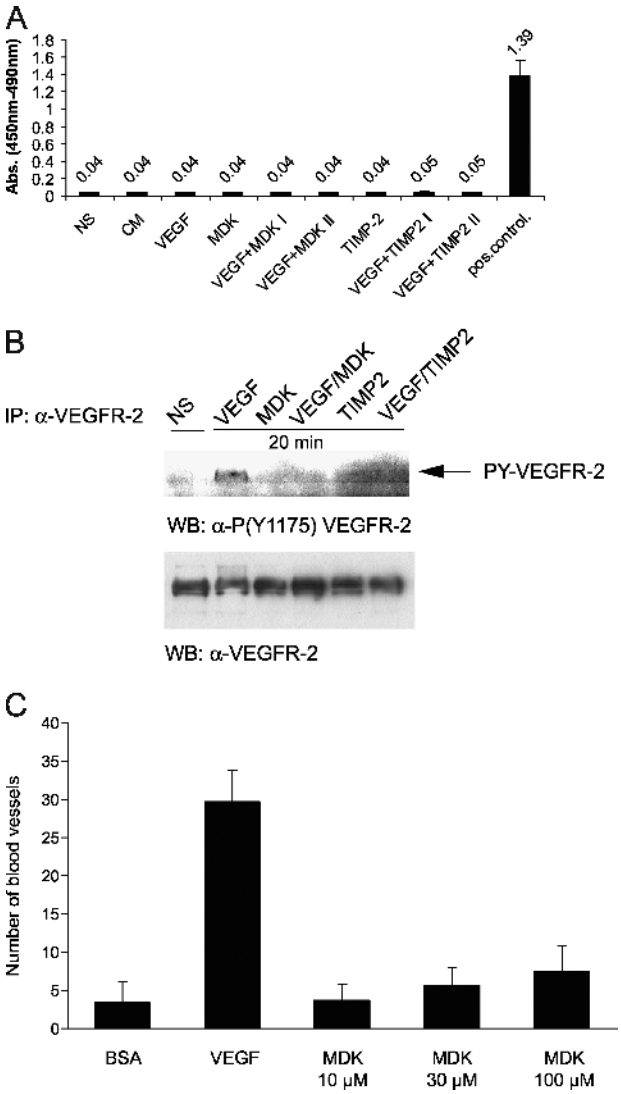

Figure W1. MDK has no influence on survival of HMVECs. (A) DNA fragmentation-assays with HMVECs was performed similarly in Figure 1*A* for 48 hours in hexaduplicates. Midkine and TIMP-2 were used in two different concentrations as indicated. Please note that the absorbance values above the individual bars in the apoptosis assay display no difference in apoptotic events in all treatment groups. *Symbols* indicate mean; *bars*, SD. Midkine inhibits VEGF-A–mediated VEGFR-2 phosphorylation in HMVECs. (B) Cells were stimulated and lysates prepared similarly in Figure 2*A*. Cell lysats were subjected to immunoprecipitation using antibodies against VEGFR-2 (VEGFR-2). Tyrosine phosphorylation was analyzed by Western blot analysis (WB) with phospho-specific anti–phopspho-VEGFR-2 (Y1175) antibody (B, top panel). Protein levels were checked by reblotting with antibodies against VEGFR-2 (B, lower panel). Midkine does not promote significant angiogenesis in the rat cornea angiogenesis model. (C) Corneal angiogenesis was induced in female CD rats (eight animals per group) with either 10 µM VEGF or the indicated amounts of MDK. After 7 days, the corneas were photographed, and for each image, the number of blood vessels intersecting the midpoint between the disc and the limbus was measured. Values represent the group mean ± standard error of the mean. Statistical significance was assessed by analysis of variance followed by Fisher's post hoc test.