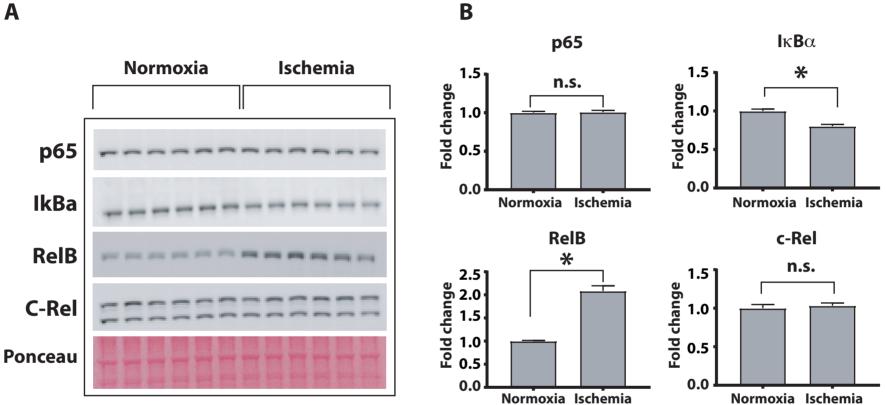
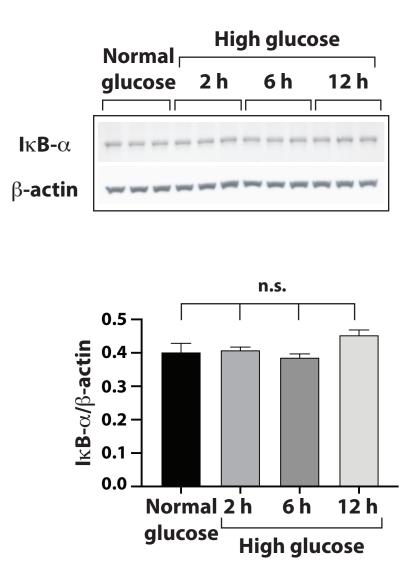
## Suppl. Figure 1.





Ischemia induced NF-κB activation in HUVEC cell lysates. Cells were exposed to ischemia (2% O<sub>2</sub> and nutrient deprivation) for 24 hours and lysates were used for Western blots for NF- $\kappa$ B subunits (p65, RelB, c-Rel) and I $\kappa$ B $\alpha$ . The levels of p65 subunit were unchanged. However,  $I\kappa B\alpha$  protein was decreased. In addition, the RelB subunit of the non-canonical NF- $\kappa$ B pathway was increased, while levels of c-Rel, another subunit of the NF- $\kappa$ B pathway, did not change. N> 6, asterisks denote  $p \le 0.05$ , n.s.= not significant.

## Suppl. Figure 2.



Change in  $I\kappa B\alpha$  abundance in lysate of HUVEC grown in normal (5 mM) or high D-glucose (25 mM). Cells were exposed to high D-glucose for 2, 6 or 12 h and lysates were analyzed by Western blotting. The protein band signals were quantified by densitometry using the signals from  $\beta$ -actin bands as loading control, and are presented in the bar graph. Compared to the normal glucose grown cells, there was no change in  $I\kappa B\alpha$  levels in high D-glucose grown cells after 2 h. One-way ANOVA was used to compare results with Normal glucose and Dunnett's post-hoc test was applied. n.s.= no significant change.