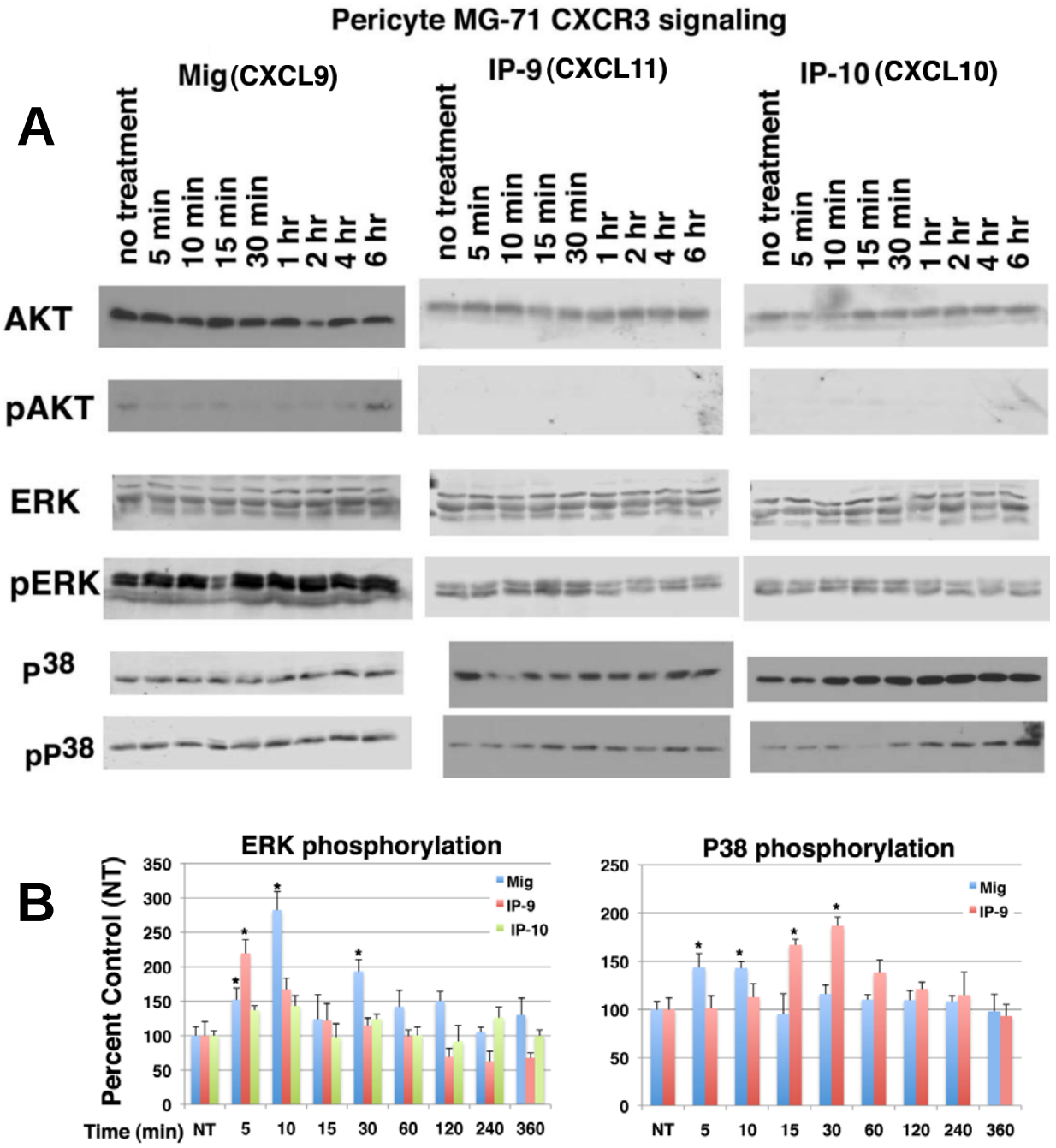
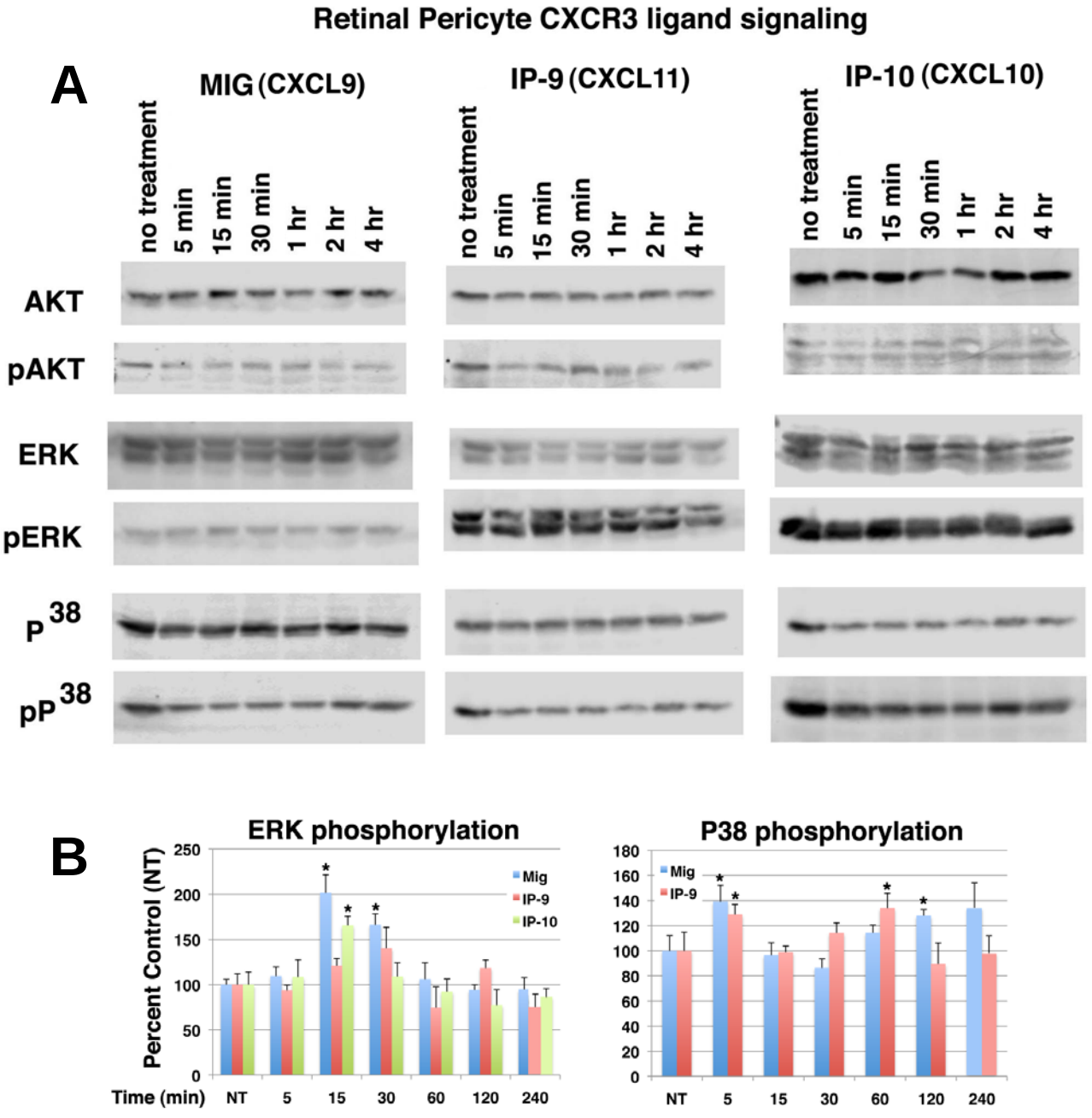
Supplemental Figure 1



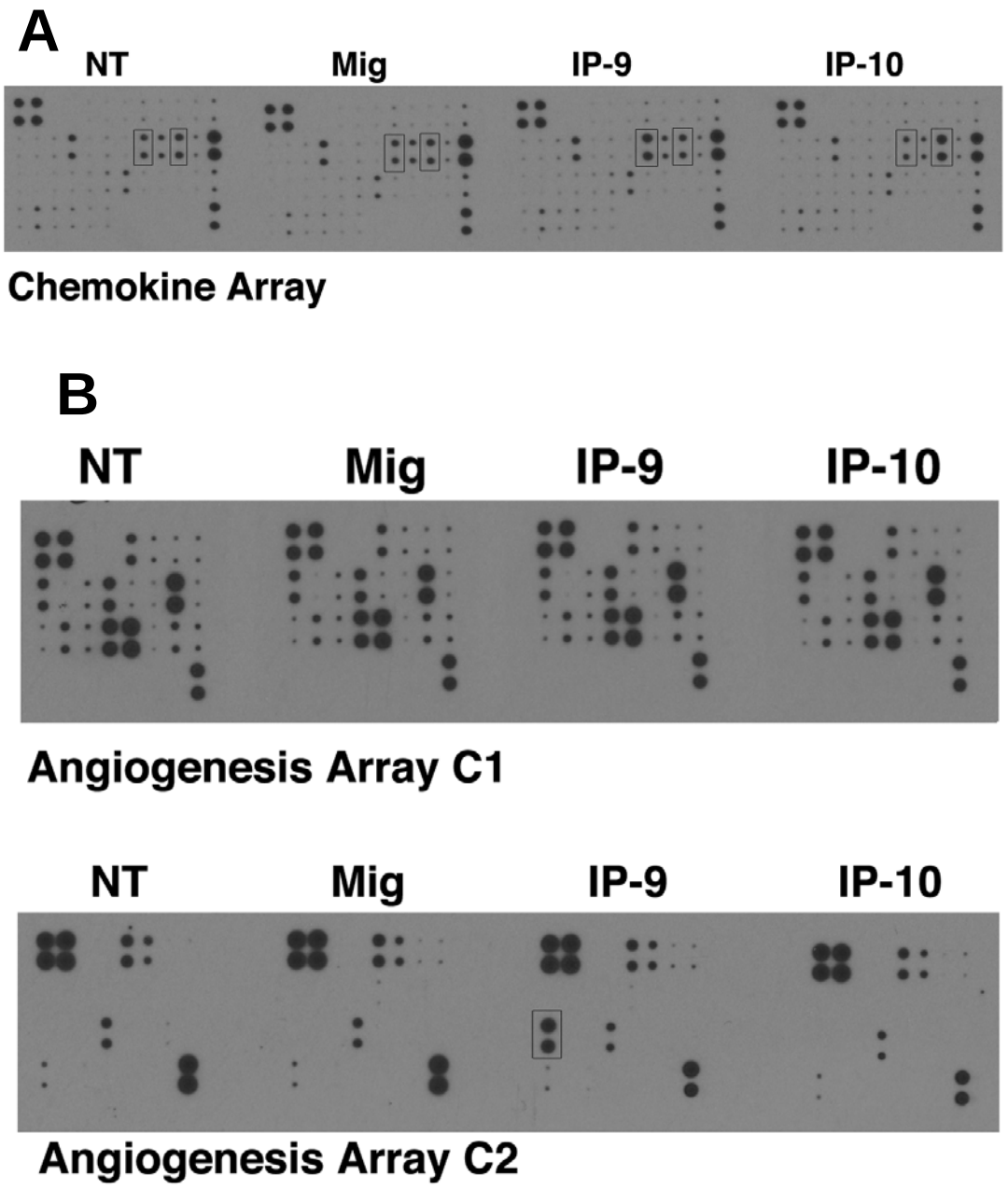
Supplemental Figure 1: Pericytes (muscle, MG-71) were incubated with Mig, IP-9 or IP-10 at 100 ng/ml for various times. The cells were lysed with RIAP buffer containing phosphatase inhibitors cocktail. The lysate was run on SDS-PAGE. The proteins were transferred to PVDF membrane and stained for AKT, ERK, P38^{MAK} and phosphorylated AKT, ERK and P38^{MAK}. B.) Quantification of ERK and P38^{MAK}. *P< .05 (n=3)We shown the CXCR3 ligands mediated the phosphorylation of ERK and P38^{MAK} but not AKT

Supplemental Figure 2



Supplemental Figure 1: A.) Retinal pericytes were incubated with Mig, IP-9 or IP-10 at 100 ng/ml for various times. The cells were lysed with RIAP buffer containing phosphatase inhibitors cocktail. The lysate was run on SDS-PAGE. The proteins were transferred to PVDF membrane and stained for AKT, ERK, $P38^{MAK}$ and phosphorylated AKT, ERK and $P38^{MAK}$. B.) Quantification of ERK and $P38^{MAK}$. *P< .05 (n=3)

Supplemental Figure 3



Supplemental Figure 3: Pericytes secrete basal levels of CXCR3 ligands.
Using protein arrays, we analyzed conditioned media from pericytes (muscle, MG-136) treated with CXCR3 ligands to identify whether activation of CXCR3 promoted protein expression of A.) Chemokines or B.) Angiogenic factors. Pericytes stimulated with CXCR3 ligands did not up regulate any of the chemokine or angiogenic proteins. Of interest, are the high basal levels of CXCR3 expression observed from the diluent (NT) treated cells. Shown are representative of two experiments with similar results (n=2).