SUPPLEMENTAL FIGURE LEGEND

Supplemental Figure 1. *(A)* NF-κB activity in LLC cells is induced by TNF-α, and inhibited by a proteasome inhibitor and sTNFR:Fc. NF-κB reporter (*pNGL*) LLC cells were plated in 12-well culture plates at equal densities (~20-30%), and incubated with TNF-α (1 nM throughout the experiment), sTNFR:Fc (1 nM throughout the experiment), or the proteasome inhibitor MG-132 (20 μM pretreatment for 2 h). NF-κB-dependent luciferase activity was determined 4, 24, and 48 h later. (n = 3/data-point, $*P < 0.001$ and $^{\#}P < 0.05$ compared with PBS-treated cells in neighboring wells). *(B)* Time-course of NF-κB activity in LLC cells after transient Ad-κB infection. *pNGL* LLC cells were serially assessed for NF-κB-dependent luciferase activity during and after 24 h of incubation with Ad-GFP (control), Ad-*Rel*A (NF-κB activator) or Ad-IκBα-DN (NF-κB inhibitor) at MOI = 500. NF-κB was significantly activated by Ad-*Rel*A and inhibited by Ad-IκBα-DN (n = 3/data-point, $**P < 0.001$ and $^{\#}P < 0.01$ compared to Ad-GFP). *(C)* TNF-α elaboration by LLC cells is NF-κB-dependent. LLC cells were treated as in *(A)*, and TNF-α was serially determined in cell-free supernatants by ELISA (n = 4/data-point, $*P = 0.002$ and $**P < 0.001$ compared with other treatments). *(D)* VEGF elaboration by LLC cells is not influenced by NF-κB activity. LLC cells were treated with Ad-κB vectors as in *(B)*, and VEGF was serially determined in cell-free supernatants by ELISA (n = 4/data-point, P = not significant). All cell experiments were done in triplicate; data are presented as mean ± SEM; A-B: numbers in parentheses represent maximal induction or inhibition and the time-point at which it was observed; C: numbers in columns represent percent change compared with baseline.