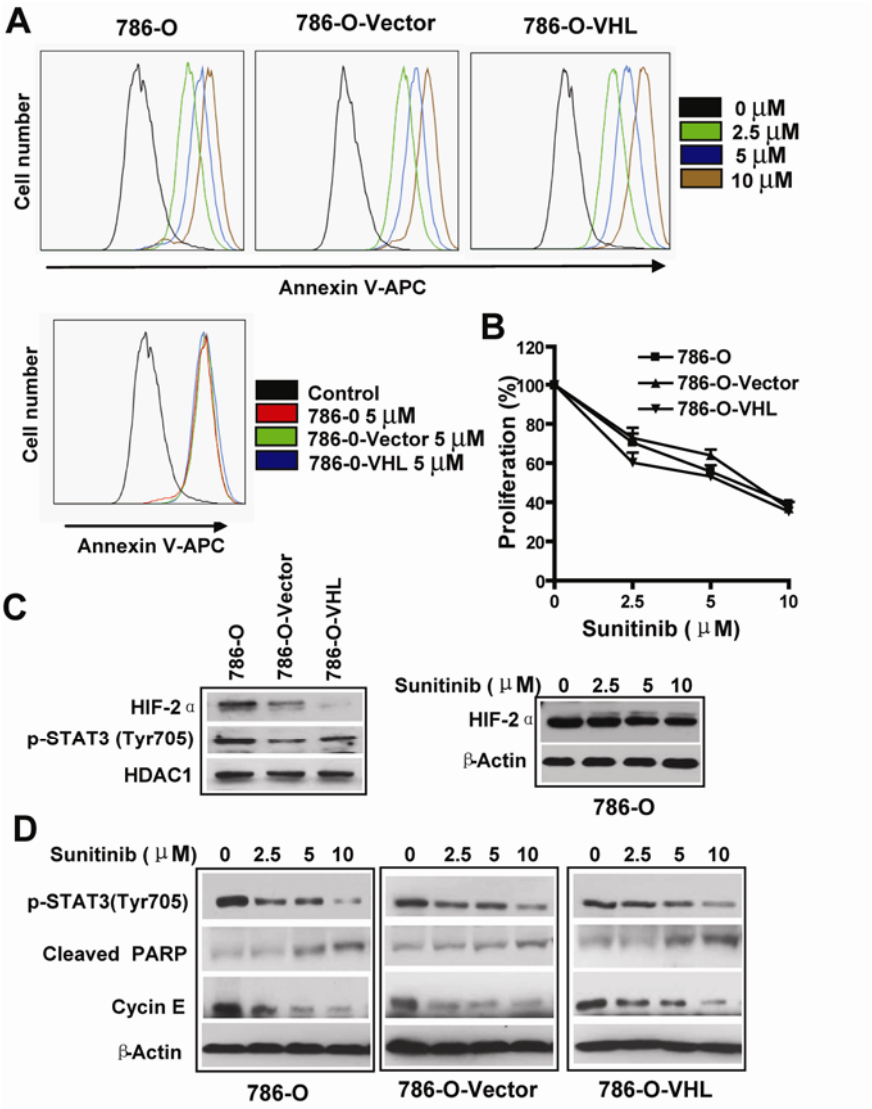

Supplementary Figure 1. Sunitinib effects on RCC tumor cell apoptosis and growth arrest are independent of VHL and HIF-2α. (A) Apoptosis induced by sunitinib in 786-O and its derivatives, 786-O-vector and 786-O-VHL, tumor cells. Tumor cells were treated with sunitinib at the indicated concentrations, followed by Annexin-V antibody staining and flow cytometry analyses. The lower panel represents superimposed data from the upper three panels. (B) Growth arrest induced by sunitinib in 786-O and derivatives. Experiments done at least in duplicate. (C) Sunitinib has no effects on HIF-2α levels in RCC tumor cells. Left panel: VHL status is important for HIF-2α levels, but not Stat3 activity. Nuclear extracts of 786-O and the derived 786-O-vector and 786-O-VHL cells were prepared; HIF-2α and p-Stat3 were detected by western blot analyses. Right panel: sunitinib has no effects on HIF-2α levels. 786-O tumor cells were treated (24 h) with indicated doses of sunitinib and HIF-2α detected by western blotting. (D) Effects of sunitinib on levels of phospho-STAT3, Cyclin E and cleaved PARP in 786-O, 786-O-vector and 786-O-VHL cells. Cells were treated (24 h) with sunitinib at indicated concentrations; total cell lysates were prepared and western blot analyses performed using relevant antibodies.