**Supplemental Figure Legend**

**Supplemental Figure 1. (A)**  Calu-1 cells were treated with VEGF (50 ng/ml) for 15 minutes and phospho-p38 levels were evaluated by Western blotting. VEGF treatment resulted in activation of p38, and this was blocked with the addition of VEGFR TKIs axitinib or sorafenib. (**B**) A549, H23, and Calu-1 cells express minimal levels of VEGFR1 as determined by ELISA assay. SKNAS neuroblastoma cells serve as a positive control.

**Supplemental Figure 2.** VEGFR TKIs inhibit the migration of NSCLC cells with *KDR* CNGs. H1993 (*KDR* amplified), H23 (*KDR* amplified) and A549 (*KDR* normal) NSCLC cells were plated in Boyden chambers in media containing 1M sunitinib (TKI targeting VEGFR, PDGFR, and KIT),sorafenib (TKI targeting VEGFR-2,-3, KIT, PDGFR), axitinib (TKI targeting VEGFR-1,-2,-3, PDGFR, and KIT), or imatinib (TKI targeting KIT, PDGFR, but not VEGFR) for 24 hours. Number of migrating cells were counted under 100x magnification. Data is graphed as mean + standard error. \*P<0.05 versus control; #P<0.05 versus VEGF only.

**Supplemental Table 1.** In the TCGA NSCLC dataset there was a significantly greater prevalence of p53 mutations in patients with *KDR* CNG compared to *KDR* CNG negative patients (p = 0.001).