Supplemental Table Legends

Supplemental Table1: Demographic and pathologic data of human NSCLC samples analyzed for TGFβRII immunostaining and expression. Unless otherwise noted data are expressed as raw numbers with percentages shown in parenthesis; percentages may not total 100 due to rounding. As expected with resected lung cancer samples there is a preponderance of early stage disease. Because banking protocols at both institutions (OHSU and UCD) included patients from the Veterans Administration, there is also a preponderance of male subjects and squamous cell carcinoma histology. NOTES: (1) Age was unavailable on 22 samples; (2) Gender was unavailable on 1 sample; (3) Never smokers were defined as having smoked fewer than 10 pack-years in their lifetime; (4) Includes 2 large cell carcinomas, 2 adenosquamous carcinomas, 2 pleomorphic carcinomas, and 1 mucoepitheliod carcinoma; these tumors were excluded from analyses comparing adenocarcinomas and squamous cell carcinomas; (5) Two samples clinically treated as synchronous primaries were scored as the T stage of the highest tumor; (6) Twenty two resected cancers were assumed to be M0 although this could not be independently confirmed in the medical record; (7) Clinical stage was determined according to the 2009 consensus guidelines (42) although many subjects had initial clinical staging under the previous staging guidelines (43); (8) Since survival was determined using the social security death index (SSDI), this could only be assessed on subjects with available social security numbers; if a death record could not be located in the SSDI after searching by social security number and name/date of birth, subjects were assumed to be alive.

Supplemental Table 2: TGFβRII deletion promotes Kras-initiated tumor formation. Animals harboring the noted genetic alterations were treated with tracheal RU486 as shown and monitored as described in methods. Age (in weeks) is when animals were euthanized (>80% of animals were euthanized between 40-62 weeks of age); N is the number of animals of each genotype analyzed; tracheal RU486 dose is in μg (the 1000μg dose was administered in two

500μg doses delivered 7d apart); penetrance is the percent of animals that developed any grossly visible lung tumors; tumor number is shown as mean ± SEM and range for animals that developed tumors. *p<0.05 vs. K5Cre*PR.Kras.

Supplemental Figure Legends

Supplemental Figure 1: Reduced TGFβRII expression correlates with smoking exposure. (PKY = pack years or one pack per day for one year).

Supplemental Figure 2: TGFβRII deletion increases the size of Kras-initiated lung tumors. Symbols represent individual tumor sizes with mean shown in red. On average Kras turmors were 0.93 ± 0.09 mm, Kras.TGFβRII+/- tumors were 1.22 ± 0.04 mm, and Kras.TGFβRII-/- tumors were 1.07 ± 0.02 mm, both of which are significantly larger than Kras tumors (p<0.05).

Supplemental Figure 3: Kras.TGFβRII-/- mice develop TTF positive, K5 negative adenocarcinomas and K5 positive, TTF negative squamous cell carcinomas. The scale bar is 50μm.

Supplemental Figure 4: TGFβRII deletion increases proliferation, TGFβ1 ligand elaboration, and inflammation *in vivo*: (A) Kras.TGFβRII-/- tumors have increased proliferation (PCNA staining) compared to Kras tumors. Scale bar is 100μm; quantification is shown in Fig. 5A. (B) Increased tumor burden in Kras.TGFβRII-/- mice correlates with increased BAL TGFβ1. (C) Increasing BAL macrophage count correlates with increasing tumor burden in Kras.TGFβRII-/- mice. (D) TGFβRII deletion increases recruitment of CD3+ lymphocytes to lung tumors. Scale bar is 50μm; quantification is shown in Fig. 5D.

Supplemental Figure 5: Stable TGFβRII knockdown in Beas2B cells increases TGFβ1 ligand elaboration. (A) Reduced TGFβRII expression at the mRNA and protein levels. (B-C) TGFβRII knockdown increases TGFβ1 mRNA and protein. (D) TGFβRII knockdown does not increase proliferation of Beas2B cells in MTT assay.

Supplemental Figure 6: Reduced TGFβRII expression in human lung cancer samples is associated with increased CD3+ lymphocyte infiltration. (A) Human lung cancer samples with preserved and reduced TGFβRII expression were immunostained for lymphocytes and macrophages with anti-CD3 and anti-MAC387 antibody. (B-C) Human lung cancer samples with reduced TGFβRII immunostaining have increased infiltration of CD3+ lymphocytes (brown) but not macrophages (brown). Immunostaining was quantified in four 10X fields of 24-30 human NSCLC samples. The scale bar is 100μm.