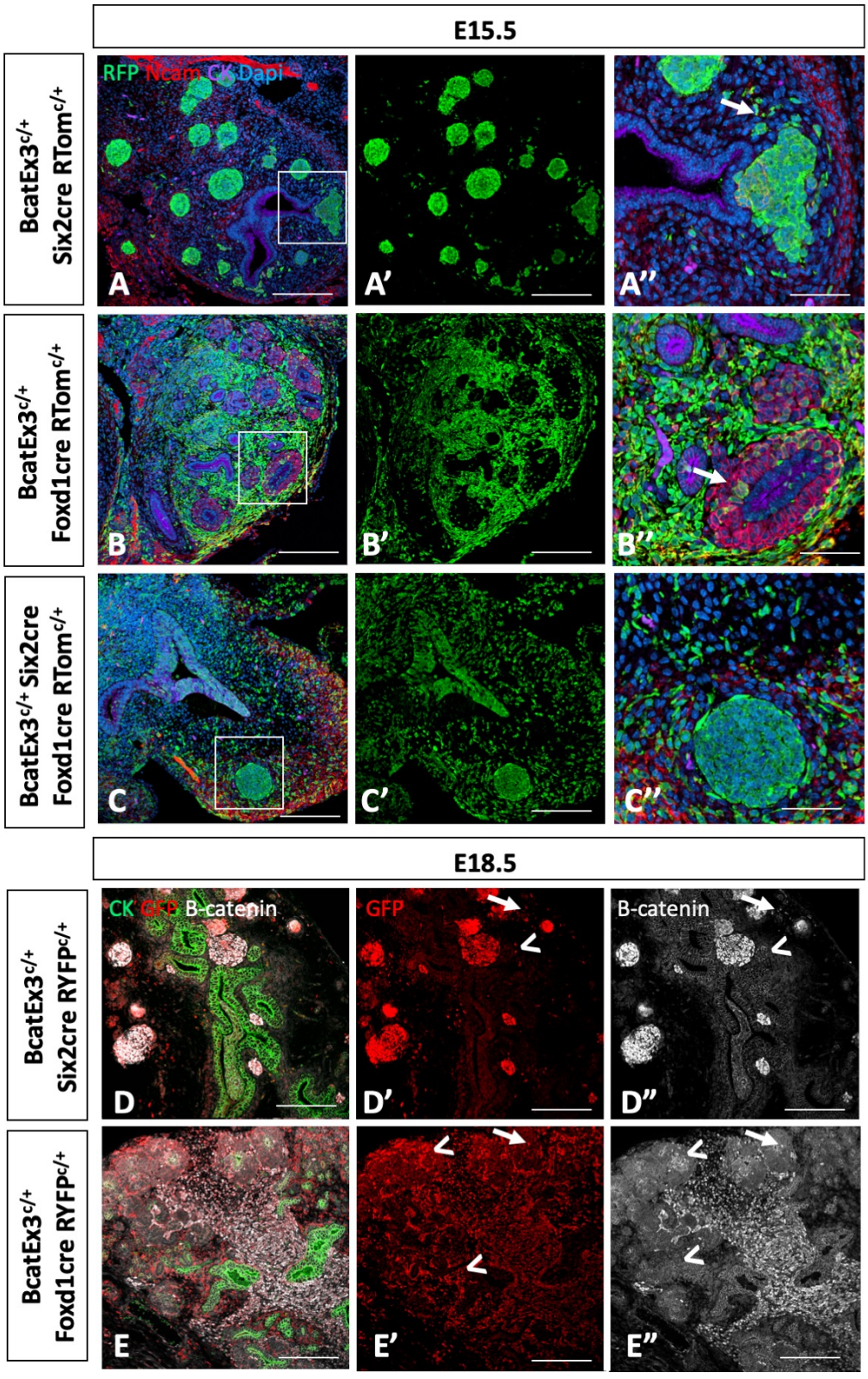

Figure S1. Lineage-tracing of beta-catenin activation in NPC and stromal cells confirm increased expression of nuclear beta-catenin in recombined cells. Use of RosaYFP and RosaRFP reporters in our mutant mouse lines show evidence of recombination in the NPC lineage (A), stromal lineage (B), and dual Six2cre and Foxd1cre mutant kidneys (C). Recombined cells in the Six2cre;Catnb$^{ex3/+}$ mutants are scattered amongst the stroma (A", arrow) with some cells expressing nuclear beta-catenin (D", arrow) while others do not (D", arrowhead). Foxd1cre;Catnb$^{ex3/+}$ mutants also show lineage infidelity, as a few scattered NPCs show evidence of recombination (E') with some cells expressing nuclear-beta-catenin (E", arrow) but others without (E", arrowhead). N = 3 for each timepoint/ genotype.

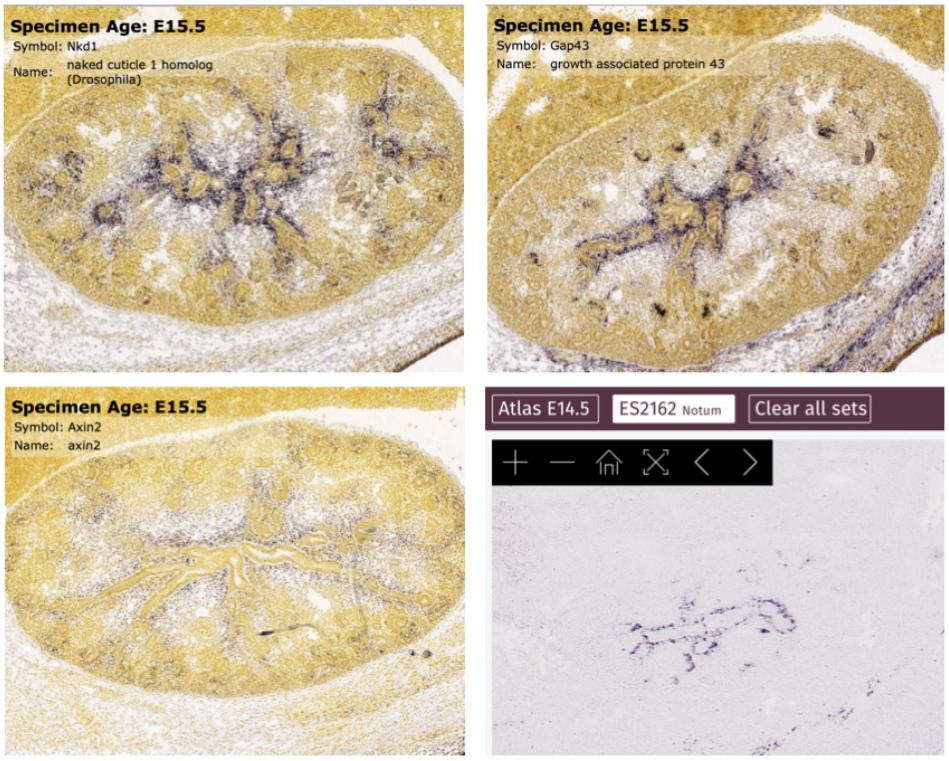

Figure S2. Beta-catenin activation drives the expression of genes normally localized to the developing renal interstitium. In-situ hybridization data from publicly available expression atlas (including Allen Brain Atlas and Genepaint) show stromal expression of Nkd1, Gap43, Axin2, and Notum – all genes also found to be up-regulated in both Six2cre;Catnb$^{ex3/+}$ mutants and Foxd1cre;Catnb$^{ex3/+}$ mutants as well at human WTs with CTNNB1 activating mutations.

Table S1. Differential gene expression from RNA-seq of E12.5 Six2cre;Catnb$^{ex3/+}$ versus Foxd1cre;Catnb$^{ex3/+}$ mutant kidneys

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Table S2. Direct β-catenin targets identified from RNA-seq of E12.5 Six2cre;Catnb$^{ex3/+}$ and Foxd1cre;Catnb$^{ex3/+}$ mutant kidneys

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Table S3. Differential gene expression in WT with CTNNB1-activating mutations versus tumors without CTNNB1-activating mutations

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Table S4. Transcriptome analysis of human WT and mutant mouse models

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Table S5. Bioinformatic algorithms and software

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