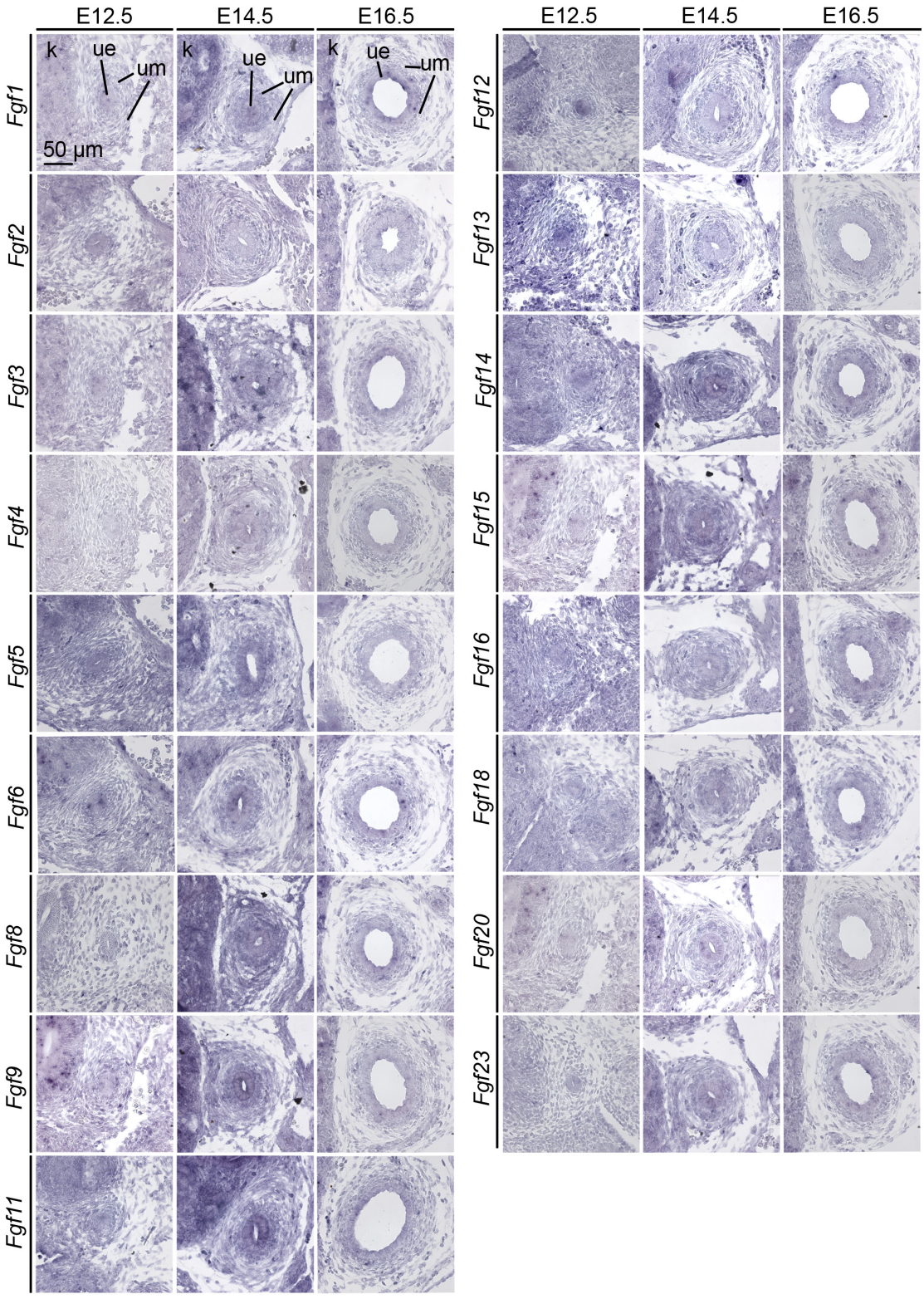

Fig. S1. FGF ligand genes do not show specific expression in the mesenchymal or the epithelial compartment of the early ureter. RNA *in situ* hybridization analysis on transverse sections through the posterior trunk region at the proximal (kidney) level of the ureter for expression of genes encoding FGF ligand genes in wildtype embryos from E12.5 to E16.5. Due to expanded colorimetric detection a homogenous bluish background developed in most cases, but no specific signal was detected in the UE or the UM for any of the probes. n>=3 for all probes and stages. k, kidney; ue, ureteric epithelium; um, ureteric mesenchyme.

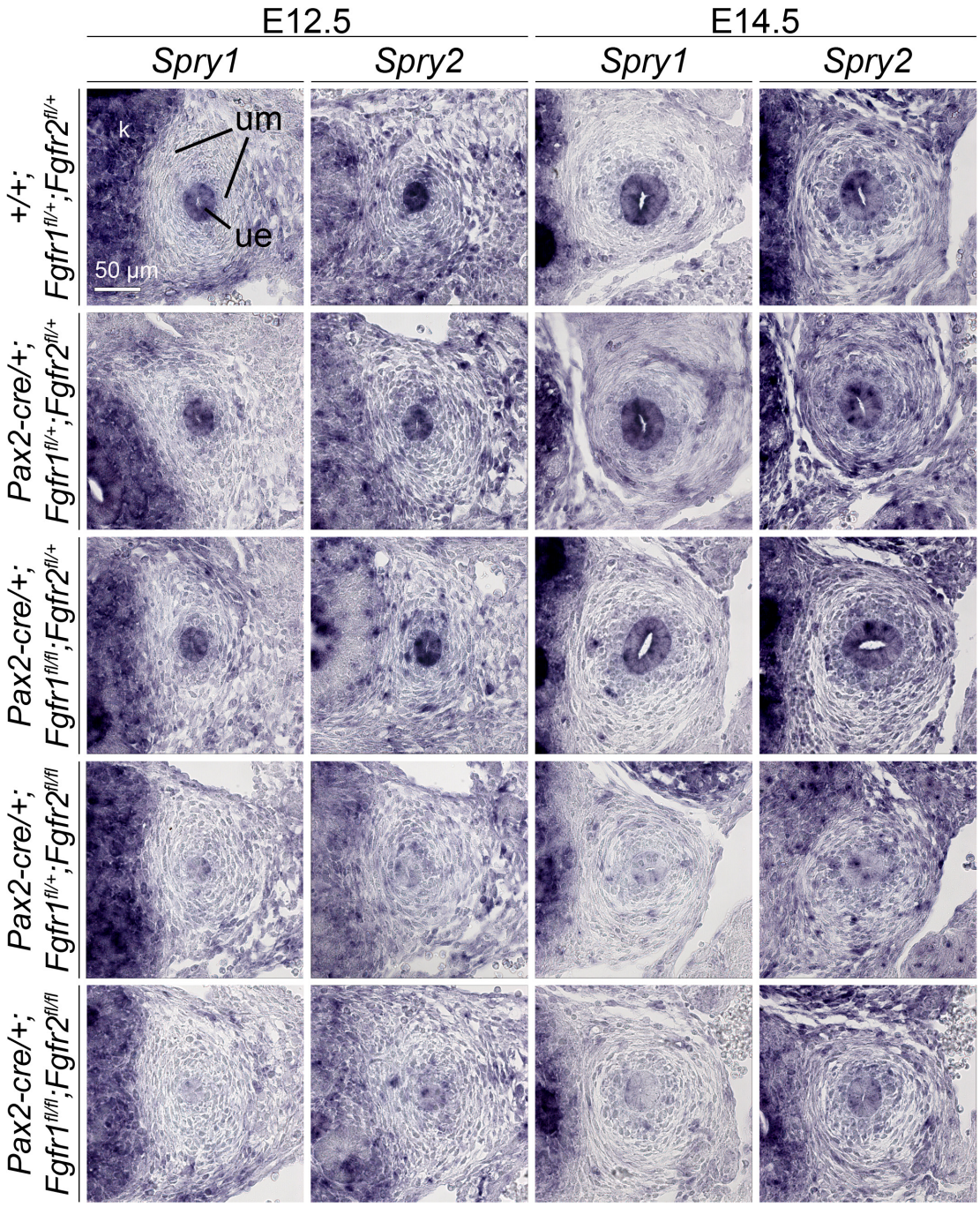

Fig. S2. FGF signaling is specifically diminished in the UE of embryos with conditional loss of *Fgfr2* in this tissue. RNA *in situ* hybridization analysis of expression of transcriptional targets of FGF signaling (*Spry1*, *Spry2*) on transverse sections through the posterior trunk region at the proximal level of the ureter of E12.5 and E14.5 embryos with conditional loss of *Fgfr1* and/or *Fgfr2*. n>=3 for all probes, stages and genotypes. k, kidney; ue, ureteric epithelium; um, ureteric mesenchyme.

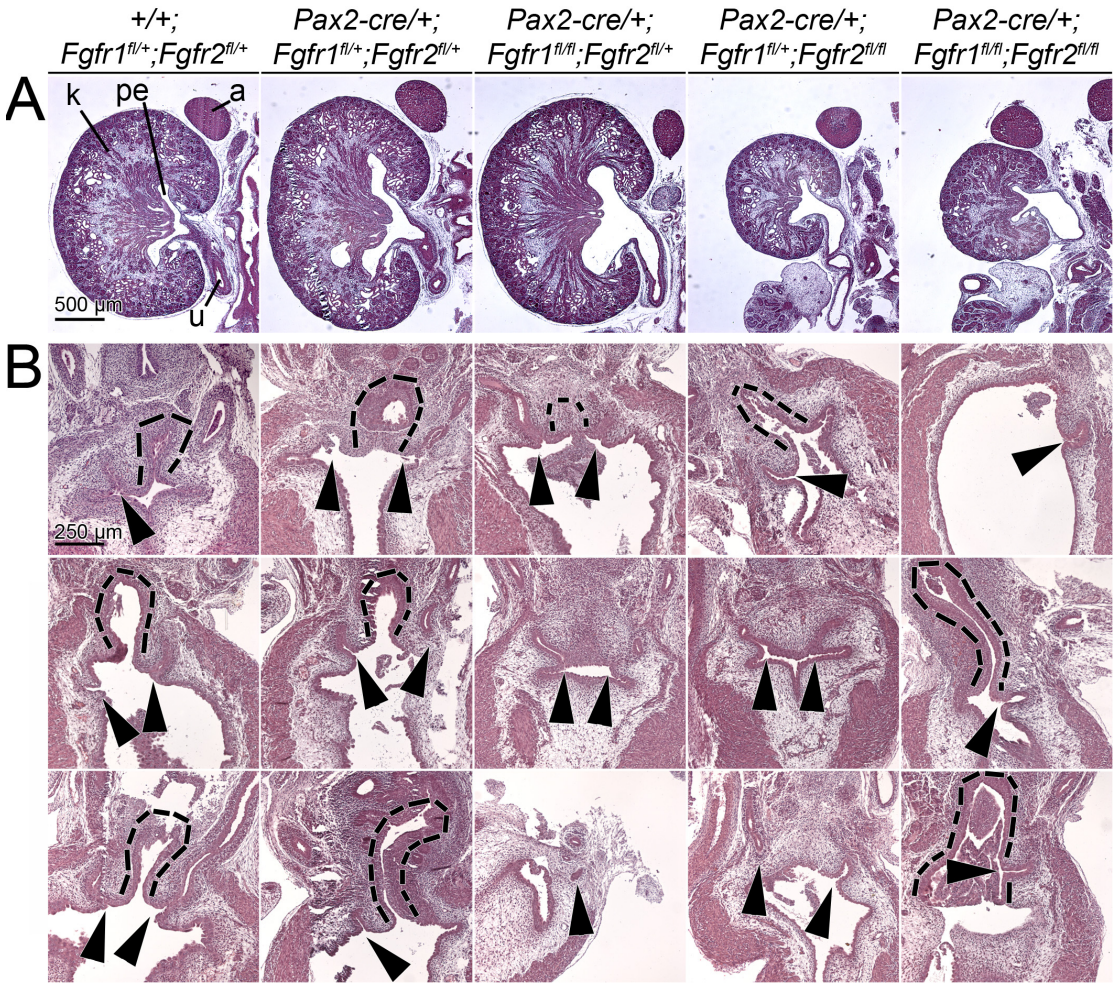

Fig. S3. Histological analysis of the urogenital system of mice with conditional loss of *Fgfr1* and/or *Fgfr2* in the UE. HE staining of sagittal sections of the kidney (A) and the ureter insertion into the bladder (B) at E18.5. (B) Dotted lines mark the urethra; arrowheads mark the ureter insertion. Note the blind ending ureter in the third *Pax2-cre/+;Fgfr1*$^{fl/fl}$*;Fgfr2*$^{fl/+}$ specimen, and the urethral insertion in the third *Pax2-cre/+;Fgfr1*$^{fl/+}$*;Fgfr2*$^{fl/fl}$ specimen. n>=3 for all genotypes. a, adrenal; k, kidney; pe, pelvis; u, ureter.

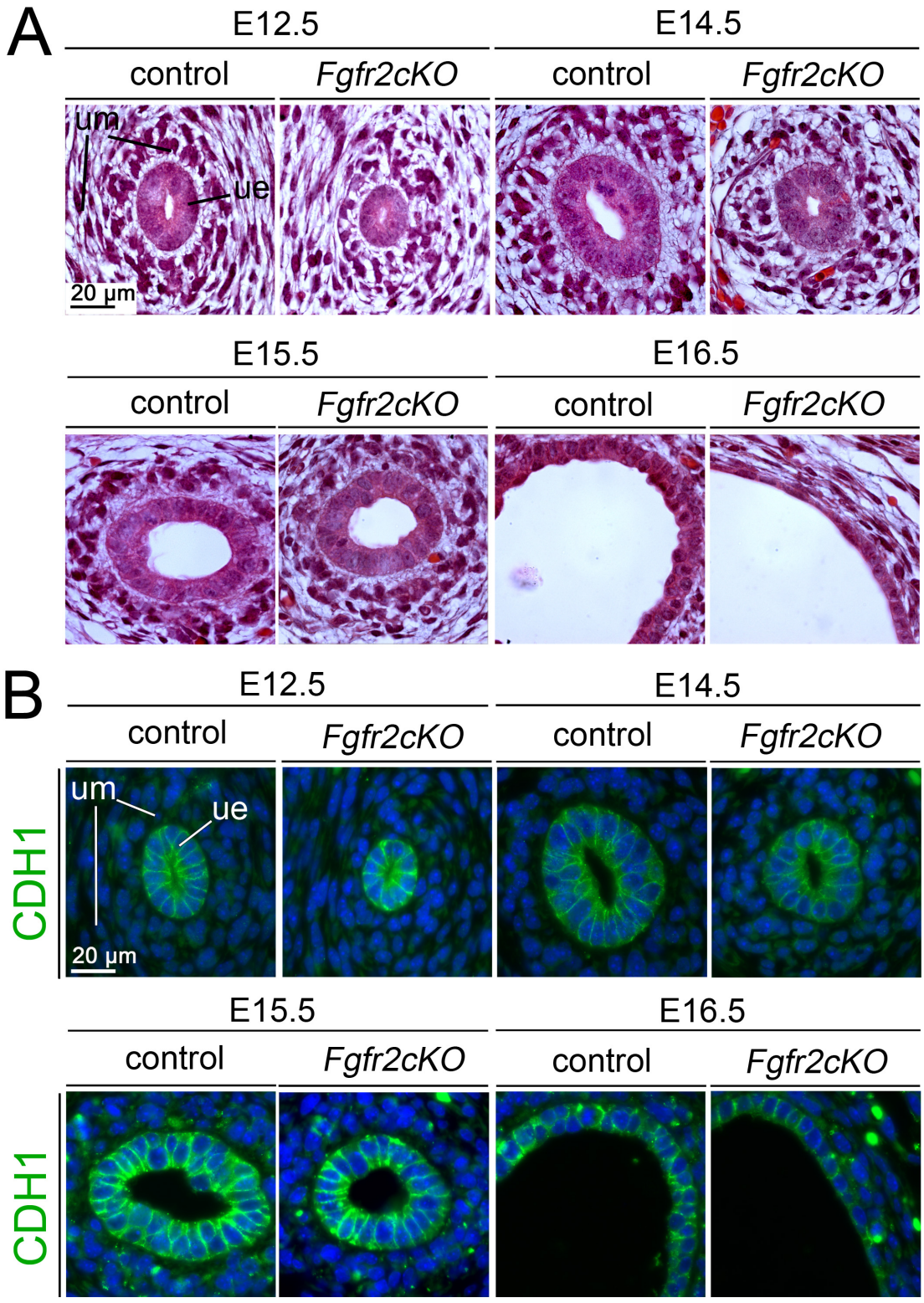

Fig. S4. *Fgfr2cKO* ureters are hypoplastic and exhibit reduced stratification in development. (Higher magnification of images shown in Fig. 3A,B). (A) Hematoxylin and Eosin (HE) staining of transverse sections of the proximal region of the developing ureter at the indicated stages. (B) Analysis of CDH1 expression by immunofluorescence on adjacent sections. Nuclei are counter-stained with DAPI (blue). n>=3, for all probes, assays and genotypes. ue, ureteric epithelium; um, ureteric mesenchyme.

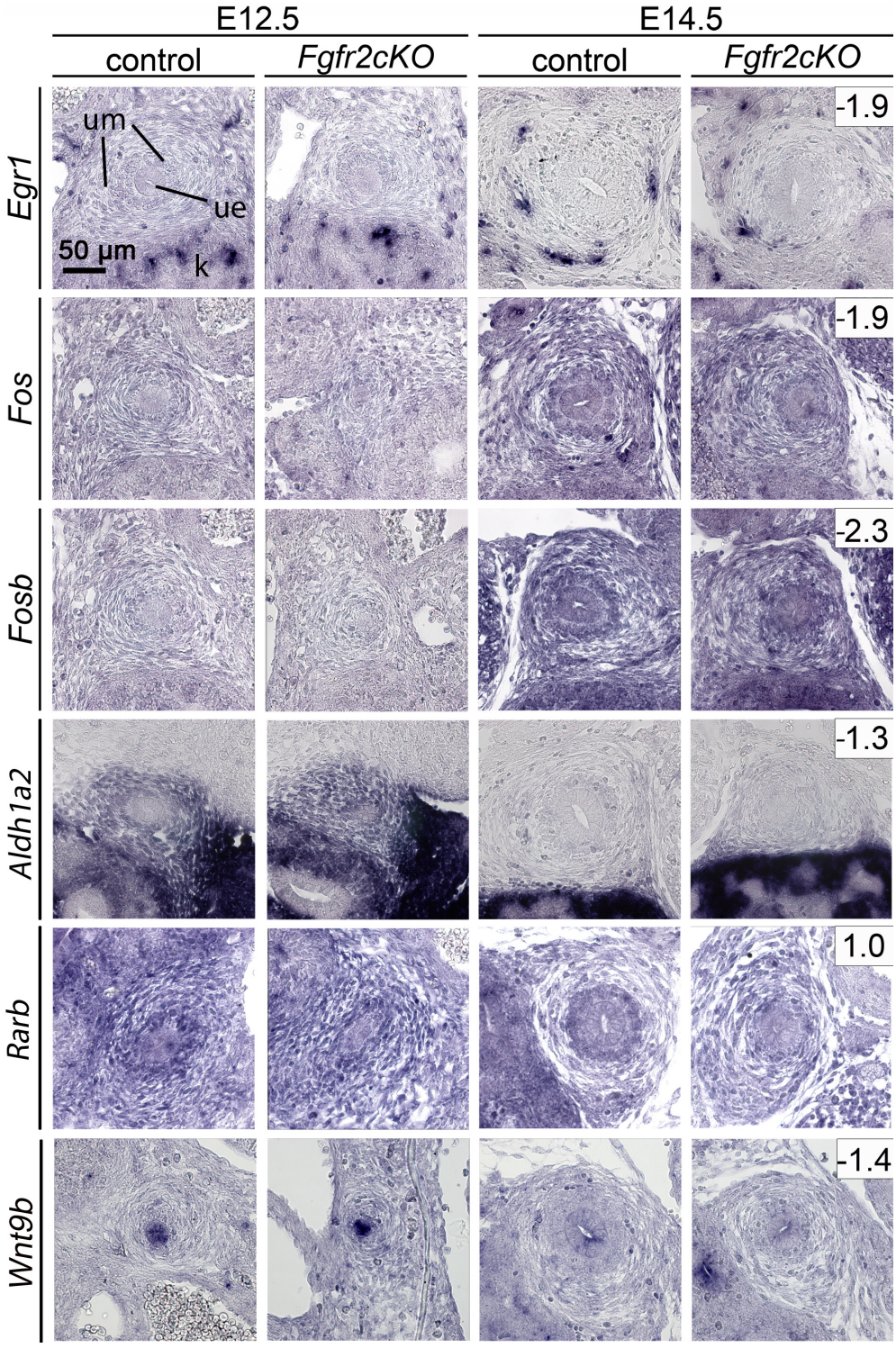

Fig. S5. Expression of immediate early genes, of mesenchymal RA signaling components and of *Wnt9b* is unchanged in *Fgfr2cKO* ureters at E12.5 and E14.5. *In situ* hybridization analysis on transverse sections of the proximal ureter of E12.5 and E14.5 control and *Fgfr2cKO* embryos for expression of immediate early genes (*Egr1, Fos, Fosb)*, of RA signaling components in the UM (*Aldh1a2*, *Rarb*), and of *Wnt9b*. Numbers indicate fold change in the E13.5 microarray analysis. n>=3 for all probes and genotypes k, kidney; ue, ureteric epithelium; um, ureteric mesenchyme.

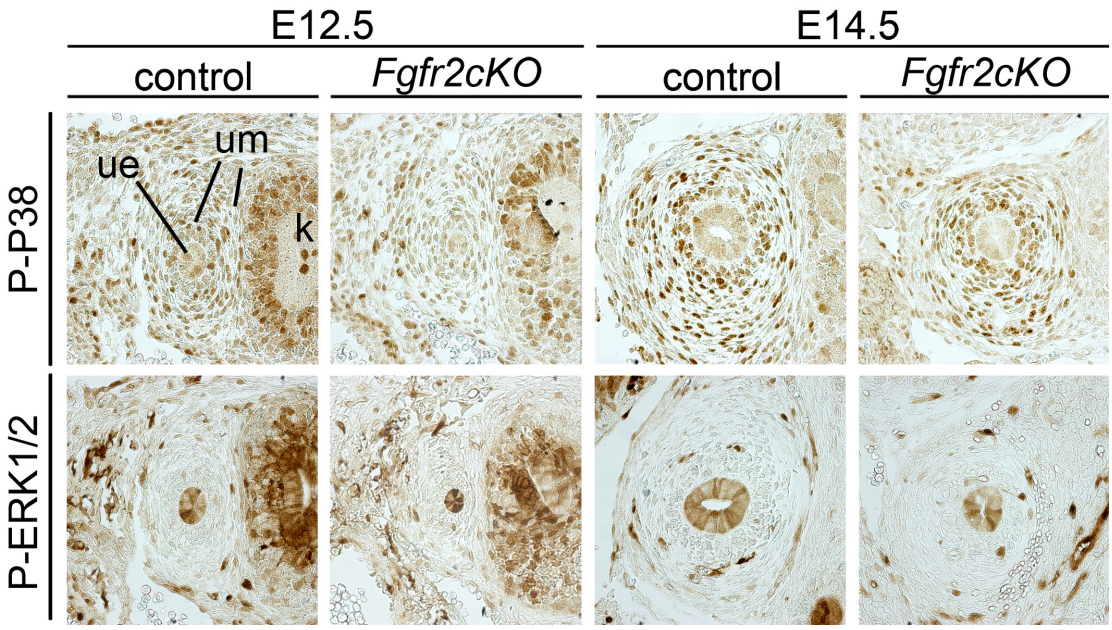

Fig. S6. Phosphorylation of P38 and of ERK1/2 is not changed in *Fgfr2cKO* ureters. Immunohistochemical detection of activated, i.e. phosphorylated forms of cytoplasmic effectors of BMP4 signaling (P-P38, P-ERK1/2) on transverse sections of the proximal ureter of E12.5 and E14.5 control and *Fgfr2cKO* embryos. n>=3 for all probes, genotypes and stages. k, kidney; ue, ureteric epithelium; um, ureteric mesenchyme.

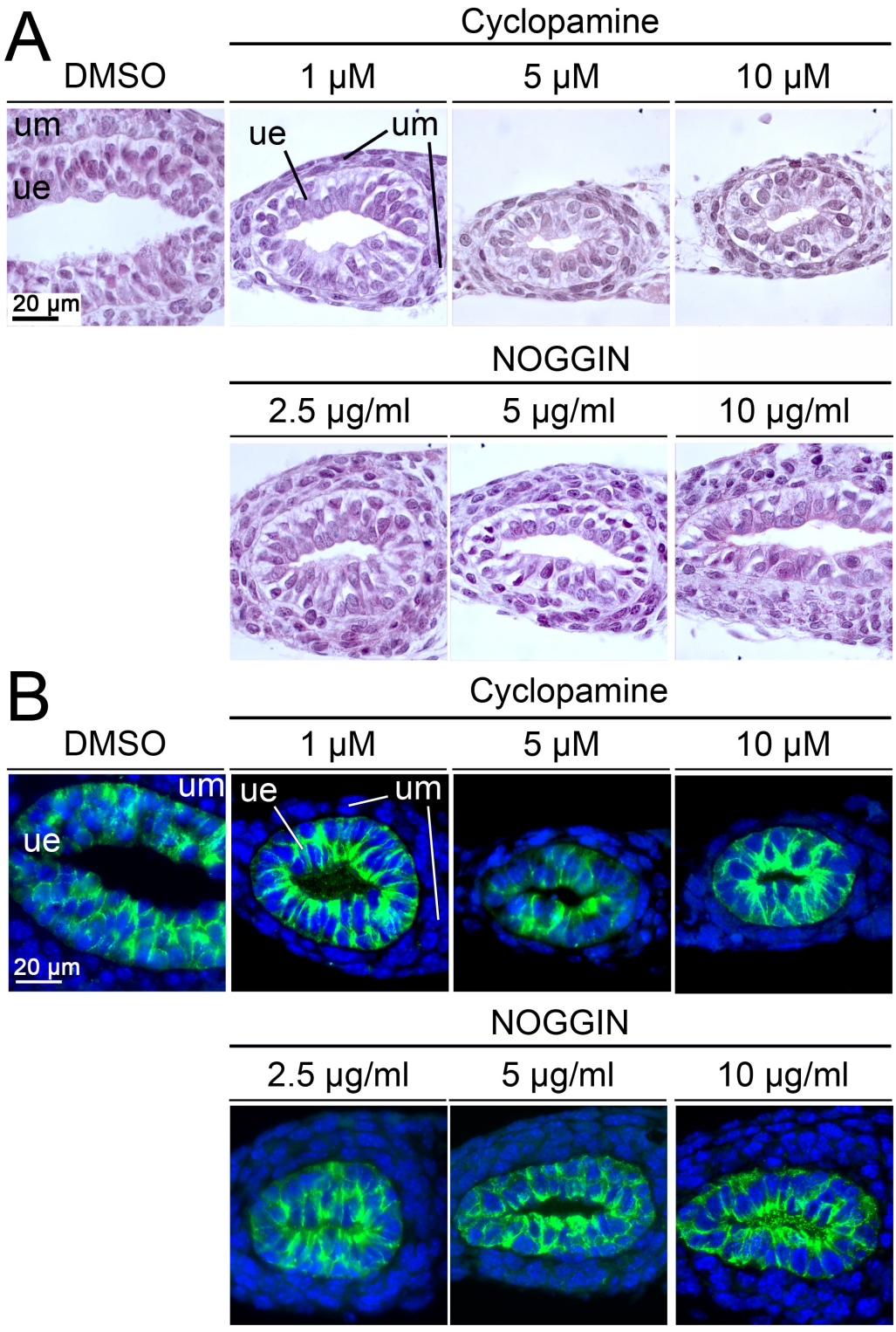

Fig. S7. Inhibition of SHH or BMP4 signaling leads dose-dependently to hypoplastic ureters with reduced stratification. (Higher magnification of images shown in Fig. 6A,B). (A,B) E13.5 wildtype ureters were cultured for 4 days with increasing concentrations of the SHH signaling inhibitor cyclopamine or the BMP4 antagonist NOGGIN, and transverse sections of the proximal region were analyzed by Hematoxylin and Eosin staining (A) and by immunofluorescence for expression of the epithelial marker CDH1. Nuclei are counter-stained with DAPI (blue). n>=3, for all probes, assays and genotypes. ue, ureteric epithelium; um, ureteric mesenchyme.

Table S1. Genotype distribution of embryos obtained from matings of *Pax2-cre/+;Fgfr1fl/;Fgfr2fl/+* males with *Fgfr1fl/fl;Fgfr2fl/fl* females at E12.5, E14.5, E16.5 and E18.5.

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Table S2. A. Distribution of phenotypic changes in urogenital systems of E18.5 embryos obtained from matings of *Pax2-cre/+; Fgfr1fl/;Fgfr2fl/+* males with *Fgfr1fl/fl;Fgfr2fl/fl* females.

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Table S3. Quantification of the BrdU incorporation assay of proximal sections of control and *Fgfr2cKO* ureters at E12.5 and E14.5.

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Table S4. List of genes with increased expression in the microarray of E13.5 ureters of *Fgfr2cKO* and control embryos. Shown are the gene names, the intensity of the two control and mutant ureter samples, the individual and the average fold change (FC).

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Table S5. List of genes with decreased expression in the microarray of E13.5 ureters of *Fgfr2cKO* and control embryos. Shown are the gene names, the intensity of the two control and mutant ureter samples, the individual and the average fold change (FC).

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Table S6. Functional annotation by DAVID for genes with increased expression in the microarray of E13.5 *Fgfr2cKO* ureters.

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Table S7. Functional annotation by DAVID for genes with decreased expression in the microarray of E13.5 *Fgfr2cKO* ureters.

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Table S8. RT-qPCR analysis of gene expression in E13.5 *Fgfr2cKO* ureters (relates to Figure 4H).

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Table S9. Pharmacological rescue experiments in explants of E13.5 *Fgfr2cKO* ureters cultured for 4 days.

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Table S10. Pharmacological inhibition of SHH and BMP4 signaling in explant cultures of E13.5 ureters. E13.5 wildtype ureters were cultured for 4 days with increasing concentrations of the SHH signaling inhibitor cyclopamine or the BMP4 antagonist NOGGIN.

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Table S11. List of primers for RT-qPCR analysis of gene expression in E13.5 *Fgfr2cKO* ureters.

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