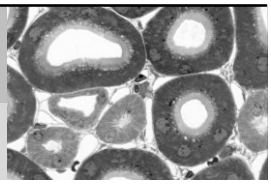
SFB 1350

Tubular system and interstitium of the kidney:
(Patho-) physiology and crosstalk

Seminar



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Prolyl-4-hydroxylases 2 and 3 stabilize endocrine function of renin expressing cells

Recent data suggest that activation of the hypoxia-signaling pathway induced by deletion of the ubiquitin ligase von Hippel-Lindau causes an endocrine shift of renin expressing cells to erythropoietin expressing cells. The mechanisms underlying this striking phenotype change are not yet understood. Since oxygen regulated stabilization of hypoxia-inducible transcription factors relevant for erythropoietin expression involves the prolyl-4-hydroxylases (PHD) 2 and 3, this study aimed to determine the relevance of these PHD isoforms for the endocrine function of renin expressing cells in vivo.

Therefore, deletions of PHD2 and/or PHD3 driven by the renin gene promotor were induced in mice with normal and with hyperplastic renin expression.

Surprisingly, deletion of PHD2 in renin expressing cells induced EPO expression in a subset of interstitial cells but not in the typical juxtaglomerular renin cells. Deletion of PHD3 had no effect on EPO expression. Codeletion of PHD2 and PHD3 stimulated EPO expression and suppressed renin expression in juxtaglomerular and hyperplastic renin cells.

These findings suggest tubular interstitial cells as a novel site of renal renin expression. Moreover, PHD2 and PHD3 seem to play an important role for the stabilization of the endocrine phenotype of juxtaglomerular and hyperplastic renin cells.

Time: **Monday 21th June 2021, 17:15h**
Location: **zoom**

To get the zoom link please contact:
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