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Aqp2$^+$ progenitors are the principal source of connecting tubule

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Connecting tubule (CNT) interconnects nephron and collecting duct (CD), which arise from kidney mesenchyme and the branching ureteric epithelium, respectively, to generate the functional tubular networks. CNT is thought as a product of reciprocal induction between the adjoining segments. The identity of the ureteric progenitors contributing to CNT remains virtually unknown. Recently, we reported that Aqp2-expressing cells with disrupted *Dot1l* give rise to principal cells (PC) and intercalated cells (IC). However, whether such derivation occurs naturally and whether Aqp2$^+$ progenitors contribute to CNT has never been addressed. Here, we generate a new mouse model (R^{AC}) in which Aqp2 lineage is genetically traced by red fluorescence protein. With high-resolution image analysis, we demonstrate that Aqp2$^+$ progenitors naturally give rise to not only PC and IC, but also several types of CNT cells. CNT can be divided into three molecularly distinct segments. These segments contain CNT/DCT transitional cells, which originate from Aqp2+ progenitors, but gain expression of NCC, a well-established DCT marker. Our study highlights the molecular identity and the origin of novel and distinct CNT segments and discovers Aqp2$^+$ progenitors as one of the origins of various types of cells not only in the CD but also in the CNT. Therefore, our study reports a novel mouse model that faithfully traces Aqp2 lineage and demonstrates a novel function of Aqp2$^+$ progenitors in CNT formation. The discovery of the CNT segments and Aqp2$^+$ progenitors may facilitate their isolation and functional evaluation.

Biography

Wenzheng Zhang is an Associate Professor in the University of Texas Medical School at Houston. He received his PhD. from MD. Anderson Cancer Center and postdoctoral training from Howard Hughes Medical Institute, Baylor College of Medicine. His research focuses on epigenetic mechanism of Na+ and water homeostasis, with a special emphasis on histone H3 K79 methyltransferase Dot1l and the epithelial Na+ channel. He created Af17 knockout and Dot1l conditional knockout mice. Recently, he began to study biomarkers and stem cells in kidney injury and repair. He has published >40 peer-reviewed papers and received funding from NIH, AHA and ASN.

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