

We study how renin cells acquire their identity and how changes in their fate result in disease. Renin cells respond to changes in perfusion pressure and the volume and composition of the extracellular fluid. Normally, a few JG cells secrete renin and maintain homeostasis, but if there is a homeostatic threat, smooth muscle cells along the kidney arterioles have the plasticity to synthesize and release renin. This ability so important to sustain well-being may lead to pathology if homeostasis is not regained resulting in concentric arteriolar hypertrophy and decreased glomerular filtration. Given that 1.3 billion people are hypertensive and are frequently treated with renin-angiotensin inhibitors, it is urgent to uncover mechanism and develop strategies to preserve kidney health while preventing the deleterious effects of hypertension.

## Renin cells, from vascular development to blood pressure sensingMaria Luisa MDUniversity of Virginia, Harrison Distinguished Prof

In the embryo, renin cells are widely distributed throughout the renal vasculature and contribute to the development of the renal arterial tree. With maturation, they differentiate into other cell types and become confined to the tips of the arterioles, thus their name juxtaglomerular cells. Juxtaglomerular cells are sensors that release renin to control blood pressure and fluid-electrolyte homeostasis. Three major mechanisms control renin release:  $\beta$ -adrenergic stimulation, macula densa signaling, and the baroreceptor, whereby a decrease in arterial pressure leads to increased renin release and viceversa. The renin cell baroreceptor transmits external forces via integrin  $\beta$ 1 to the chromatin to regulate *Ren1* expression. We will discuss the origins of renin cells, their role in kidney vascular development and current understanding of the blood pressure sensing mechanism.

※総合医療センターにてTV会議システムを介して参加をご希望される方は<u>7月8日(火)までに</u>医学会講演会担当までご相談ください。 ※大学院生で講義として受講する場合は必ず教務課(大学院)へ問い合わせ下さい。

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《皆様のご来聴をお待ちしております》