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striking aldosterone excess, low renin, and hypertension. Here, we deleted TASK-1 and TASK-3 channels selectively from zona glomerulosa cells and generated a model of mild aldosterone autonomy with attendant hypertension that is aldosterone-driven and Ang II (angiotensin II)-independent. This study shows that a zona glomerulosa–specific channel defect can produce mild autonomous hyperaldosteronism sufficient to cause chronic blood pressure elevation.

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