Role of Direct Renin Inhibitor in Renoprotection

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The renin-angiotensin-aldosterone system (RAAS) plays a fundamental role in many of the pathophysiologic changes that lead to progression of renal disease. Traditionally RAAS was considered as an endocrine system and its principal role was to maintain blood pressure. In recent years local RAAS has been described to operate independently from systemic and local angiotensin II in the kidney to contribute in hypertension and renal damage. Renin is the rate-limiting step in the RAAS cascade, which makes direct renin inhibitors an attractive target for RAAS suppression and treatment of hypertension. Current regimens using either angiotensin-converting enzyme inhibitor or angiotensin receptor blocker result in feedback upregulation of renin and aldosterone escape or breakthrough, which contribute to incomplete suppression of the RAAS. Aliskiren, the first direct renin inhibitor approved for the treatment of hypertension, blocks the RAAS at its point of activation. As renin inhibition acts at the top of the RAAS cascade, this mechanism has been proposed to offer advantages over existing modes of RAAS blockade. The RAAS is also considered to be a major factor in the pathogenesis of many renal diseases, especially diabetic nephropathy, the main cause of end-stage renal disease in Taiwan.