

## Recent Update in Renin–angiotensin System and Diabetic Kidney Disease

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The activation of intrarenal reninangiotensin system has a crucial role in the development and the progression of diabetic kidney disease. In the kidney, angiotensin II, which is the central component of renin–angiotensin system, is produced in relatively high levels, and binding to AT1 receptors stimulates vasoconstriction, sodium and water retention, accumulation of extracellular matrix, and induces the early renal inflammation, followed by subsequent fibrosis. Other components of renin–angiotensin system contribute to the progression of chronic kidney diseases.

Aldosterone was well known to have profibrotic activity. The pathogenic role of prorenin/renin through the newly identified renin/prorenin receptor was also identified. They consist of a very complex system to understand. However, the previously unsuspected complexity of the reninangiotensin system has been unveiled in recent days. The recent discoveries, which produced remarkable conceptual changes in this field, were Ang–(17), angiotensin converting enzyme (ACE)2 as an important Ang–(17)–forming enzyme and Mas as an Ang–(17) receptor. ACE2 could metabolize angiotensin II to generate angiotensin–(1–7) and was highlighted as an attractive target for novel therapeutic approaches in a variety of kidney diseases, including diabetic nephropathy. This mini–review will summarize the ACE2–Ang–(1–7)–Mas system, the novel concept of the renin–angiotensin system and review recent understandings of their role on the pathogenesis of diabetic kidney disease.