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Relationship between renal injury and the antagonistic roles of angiotensin-converting enzyme (ACE) and ACE2

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ABSTRACT. Angiotensin-converting enzyme 2 (ACE2), a newly discovered carboxypeptidase in the renin-angiotensin system (RAS), antagonizes ACE activity and plays an active role during tissue injury. Yet the mechanism of its action is not well known. Using a streptozotocin (STZ)-induced renal injury rat model, we investigated the relationship between renal injury and the antagonism between ACE and ACE2. We assayed the levels of urea nitrogen, urine glucose, creatinine, and protein, *Ace2*, *Ace*, angiotensin II type 1 receptor (*At1*) and Mas receptor mRNA, and renal and plasma angiotensin II (Ang II) in STZ-treated and untreated rats. We also used histology and immunohistochemistry to assess glomerular injury and ACE2 glomerular and cortical expression. The amounts of urea nitrogen, urine glucose, creatinine, and protein were significantly higher in STZ-treated rats than in control rats ($P < 0.01$). There were significant pathological changes in the kidney upon STZ-treatment. *Ace2* and *Ace* mRNA levels were significantly higher in STZ-treated rats than in control rats ($P < 0.05$ and $P = 0.05$, respectively). There was no significant difference in the Mas receptor and *At1* mRNA levels in the 2 groups, although *At1* levels showed an increase upon STZ-treatment. The Ang II level in the renal cortical tissue and plasma

of STZ-treated rats was higher than that of control rats ($P < 0.05$). The increase in Ace mRNA levels was higher than that of Ace2 mRNA levels, leading to an elevated Ace/Ace2 ratio. Together, these data suggest that the ACE-Ang II-AT1 axis is the dominant axis in severe kidney injury.

Key words: Rat; Kidney injury; ACE2; ACE; Negative regulation

