Prostaglandin E\(_2\) Mediates Connecting Tubule Glomerular Feedback

To the Editor:

We read with interest the publication of Ren et al\(^1\) about prostaglandin E\(_2\) being the major mediator of the connecting tubule glomerular feedback (CTGF) response (afferent arteriolar vasodilation in response to an increase in distal Na tubular delivery). The same group of authors has previously shown that an impairment of macula densa TGF (afferent arteriolar constriction in response to the same stimulus) is responsible for the increased intraglomerular pressure in Dahl-S rats and that this is, in part, because of its resetting by enhanced counterregulatory CTGF. CTGF was found to be increased in Dahl-S rats, consistent with this interpretation.\(^2\) The observations are important because they could account for the renal injury of salt-sensitive hypertension. However, increased CTGF in Dahl-S rats is difficult to reconcile with old data about prostaglandin E\(_2\) in this strain. These rodents have increases in Cox-2 activity in the renal cortex (probably mediated by angiotensin II and reactive oxygen species).\(^3\) However, the production of prostaglandin E\(_2\) by their cultured renal papillary collecting tubule cells is markedly decreased, whether in the prehypertensive or hypertensive stages, whether on low- or high-salt diet, and also after stimulation with a calcium ionophore or addition of arachidonic substrate.\(^4\) Therefore, the authors should at least speculate how they think that CTGF, a mechanism putatively enhanced in Dahl-S rats, can have as a major mediator a substance (prostaglandin E\(_2\)) that is deficient in this strain, particularly in the cells that are the site of this renal autoregulatory mechanism.

Disclosures

None.

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