Prostaglandin E₂ Mediates Connecting Tubule Glomerular Feedback

To the Editor:

We read with interest the publication of Ren et al.¹ about prostaglandin E₂ 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 E₂ 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 E₂ 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 E₂) that is deficient in this strain, particularly in the cells that are the site of this renal autoregulatory mechanism.

Disclosures

None.

Fernando Elijovich
Cheryl L. Laffer
Division of Clinical Pharmacology
Department of Medicine
Vanderbilt University School of Medicine
Nashville, TN

Prostaglandin E$_2$ Mediates Connecting Tubule Glomerular Feedback
Fernando Elijovich and Cheryl L. Laffer

*Hypertension*. 2014;63:e19; originally published online January 20, 2014;
doi: 10.1161/HYPERTENSIONAHA.113.02900

*Hypertension* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2014 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://hyper.ahajournals.org/content/63/3/e19

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Hypertension* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the *Permissions and Rights Question and Answer* document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Hypertension* is online at:
http://hyper.ahajournals.org/subscriptions/