Response to Prostaglandin E₂ Mediates Connecting Tubule Glomerular Feedback

We thank Elijo vich and Laffer¹ for their interest in our article. First, we clarify that prostaglandin E₂ is not the only mediator of connecting tubule glomerular feedback (CTGF). We had previously reported that about half of the CTGF response is mediated by epoxyeicosatrienoic acids, with the other half attributable to a prostaglandin,² which we have now identified as prostaglandin E₂.³ Second, it is important to note that the decrease in prostaglandin E₂ production reported in Dahl salt-sensitive rats was noted in cultured papillary cells, whereas our experiments dealt with a cortical segment of the nephron, namely the connecting tubule. The regulation of COX isoforms in Dahl salt-sensitive rats is known to be different in the cortex versus the medulla. For example, COX-1 expression is increased in these rats by high salt in the cortex but decreased in the medulla.⁴ This is especially relevant for CTGF because the connecting tubule only expresses COX-1,³ whereas the papilla expresses significant amounts of COX-2.⁴ Finally, the relative contribution of different eicosanoids to the CTGF response may be different in this report when compared with our previously published studies in Dahl salt-sensitive rats because of differences in pathophysiological status (normotensive versus hypertensive) or species (rabbit versus rat). Thus, it is our opinion that our current results are not in conflict with our previously published data; however, we acknowledge that further studies are necessary to elucidate the mechanisms involved in CTGF enhancement in Dahl salt-sensitive rats.

Disclosures

None.

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