Response to Nongenomic Response to Aldosterone

We appreciate the supportive and thoughtful comments of Dr Osanai and colleagues1 and are mindful of their contributions to our understanding of the rapid effects of aldosterone on vascular contractility. We also found it curious that aldosterone is significantly less potent for stimulation of myosin light chain phosphorylation, as well for mediating contraction as assessed at a single cell level as compared with its effects on extracellular signal–regulated kinase activation/apoptosis. Notably, for extracellular signal–regulated kinase phosphorylation, NO synthase activation, vascular reactivity,2,3 and apoptosis,3 we have consistently seen aldosterone EC50s in the picomolar range. For myosin light chain phosphorylation3 and measures of single cell contraction4 our reported aldosterone EC50s have been significantly higher, in the nanomolar range. Notably, for all of these responses, common effects of both steroid agonists (like aldosterone and G1) and antagonists (like spironolactone, G15, and eplerenone) have been apparent. The reason for the reduced potency of aldosterone in regulating single cell parameters of contractility (versus other measures of regulation of cell growth) is unclear to us. However, given the greater potency of aldosterone in mediating regulation of vascular reactivity in intact vessels (with EC50S in the picomolar range) than in single cells from the same tissue (with EC50S in the nanomolar range), we have can only conclude that this relates to less efficient coupling between the receptor and effector in these in vitro systems.

Disclosures

None.

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Hypertension. 2011;58:e4; originally published online June 6, 2011;
doi: 10.1161/HYPERTENSIONAHA.111.174847

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

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