Potassium Ions as Vasodilators
Role of Inward Rectifier Potassium Channels

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Much of the research on potassium-induced dilations has focused on the cerebral and coronary circulation. Small increases in circulating potassium ions in vivo dilate and increase cerebral flow. Recently, Chritsalis et al. demonstrated that cerebral artery dilations in vivo to elevated K+ in cerebral spinal fluid were Ba2+-sensitive and insensitive to ouabain, strongly supporting a role for inward rectifier potassium channels. In the cerebral vasculature, elevations in K+ increase with neuronal activity and during stresses such as cerebral hypoxia, ischemia, and hypoglycemia. K+-induced dilations have also been reported in coronary arteries. K+ ions are normally released from cardiac cells during increased workload and particularly under ischemia. In the kidney, elevated potassium (>10 mmol/L) or acute hyperkalemia have been shown to increase renal blood flow and glomerular filtration rate.

In a study in this issue of Circulation Research, Chilton and Loutzenhiser have explored the role of inward rectifier potassium channels in K+-induced dilations of rat renal afferent arterioles, using the hydrencephalic kidney model. This model permits visualization of the renal microvasculature under normal flow and pressure conditions. Loutzenhiser et al. have taken this model one step further and developed a method for measuring stable membrane potentials while simultaneously measuring diameter of intact afferent arterioles in the intact kidney. In pressurized afferent arterioles, increasing [K+]e from 5 to 15 mmol/L caused dilation. In the presence of the α-adrenoceptor blockers, K+-induced dilations were also abolished by chloroethylclonidine (CEC). CEC has been shown to inhibit native inwardly rectifying potassium channels (Kir) in skeletal muscle (rat flexor digitorium brevis) as well as Kir2.1 channels expressed in the MEL cell line. Neither the KATP channel inhibitor glibenclamide nor ouabain inhibited K+-induced dilations of rat renal afferent arterioles, suggesting a role for Kir channels in regulating membrane potential. The Chilton and Loutzenhiser study, along with studies on the cerebral and coronary circulation, strongly supports the idea that the inward rectifier potassium channel, in particular the Kir2.1 subtype, is a molecular target for external potassium-induced vasodilation.

The opinions expressed in this editorial are not necessarily those of the editors or of the American Heart Association.

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References

See related article, pages 152–158


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