Effects of Cytosolic Ca^{2+} on Membrane Voltage and Conductance of Rat Renal Mesangial Cells from Stroke-Prone Spontaneously Hypertensive Rats

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Summary

Although abnormalities in renal hemodynamics may be critically involved in the pathogenesis of hypertension, intrarenal regulation of glomerular filtration dynamics in genetic hypertension is poorly understood. Cl^- was recently indicated as an important mediator of tubuloglomerular feedback (TGF) regulation of glomerular filtration by distal tubular flow, and Ca^{2+} activated Cl^- channels were detected in mesangial cells by a patch-clamp method. The purpose of this study was to characterized the effects of cytosolic Ca^{2+} on membrane voltage and conductance of MC using stroke-prone spontaneously hypertensive rats (SHRSP) and Wistar Kyoto rats (WKY).

Mesangial cells were obtained from 11-wk-old SHRSP/Izm and WKY/Izm by culturing isolated glomeruli in RPMI1640 medium containing 20% fetal calf serum (FCS), as described previously. And we used mesangial cells under the primary culture. We applied the patch-clamp technique in the whole-cell configuration to measure the membrane voltage (Vm) and conductance (gm) of two strain cells. The cytosolic Ca^{2+} increase was induced by 5 μ M ionomycine that was Ca^{2+} ionophore.

There was no significant difference in resting Vm values between MC from WKY and SHRSP. The cytosolic Ca^{2+} increase induced membrane depolarization and the increase of Cl^- currents in MC from WKY but not in MC from SHRSP. On the other hand, the Ca^{2+} increase induced membrane hyperpolarization and the increase of K^+ currents in MC from SHRSP but not in MC from WKY.

In conclusion, SHRSP-MC are under different regulations from WKY-MC. This may be related to abnormal regulation of glomerular function associated with the intrinsic renal mechanism of hypertension development in SHRSP suggesting the involvement of a similar mechanism in human hypertension.