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NOVEL ACTIVATORY MECHANISM OF ACTIN-GATED SODIUM CHANNELS IN K562 CELLS

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The work is aimed to search for extracellular regulatory pathways of actin-gated sodium channel activation in human K562 leukemia cells. The characteristics of sodium channels are close to the ENaC family except the sensitivity to amiloride and its derivatives. To study the effect of potential channel activators, we used the opportunity of recording and analyzing unitary currents in plasma membrane of K562 cells by whole-cell patch-clamp technique. The development of single channel activity induced by the disassembly of the actin cytoskeleton in response to application of cytochalasin D was shown in whole-cell experiments; biophysical properties of the channels were analyzed on unitary currents level. Activation of the channels was observed in response to the application of serine protease trypsin, a known activator of the ENaC channels in the renal epithelium, to the extracellular solution. The functional characteristics of the channels activated by the disruption of the cytoskeleton or by trypsin protease were identical. The unique feature of the cell model allowed us to analyze single channel currents in whole-cell membrane and to reveal a novel pathway of extracellular activation of actin-gated channels.

K e y w o r d s: plasma membrane, single sodium channels, whole-cell, actin cytoskeleton, proteolytic activation