Roles of Ca^{2+} and Cl^- in Na^+ transport and clearance of lung fluid in fetal lung

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Summary

To clarify roles of Ca^{2+} and Cl^- in regulation of amiloride-sensitive Na^+ transport in rat fetal distal lung epithelial (FDLE) cells, we measured single channel currents from cell-attached and inside-out patches formed on the apical membrane of FDLE. FDLE had two types of amiloride-sensitive Na^+-permeable cation channels: nonselelctive cation (NSC) and Na^+ channels. Only the NSC channel responded to a beta adrenoceptor agonist (beta agonist), but the Na^+ channel did not. Therefore, we focused our study on the NSC channel. A beta agonist increased the cytosolic Ca^{2+} concentration ($[Ca^{2+}]_c$) and decreased cytosolic Cl^- concentration ($[Cl^-]_c$). The NSC channel was activated by cytosolic Ca^{2+}, while the channel was inhibited by cytosolic Cl^-. Therefore, we studied which factor, an increase in $[Ca^{2+}]_c$ or a decrease in $[Cl^-]_c$ caused by a beta agonist, played an essential role in stimulation of the channel leading to an increase in the Na^+ transport and clearance of lung fluid. Our study indicates that the increase in $[Ca^{2+}]_c$ plays an important role in decreasing the $[Cl^-]_c$, however the beta-agonist-caused decrease in $[Cl^-]_c$ essentially activates the NSC channel. Based on these results, we conclude that that the extracellular Ca^{2+} plays an important role in the stimulatory action of beta agonist on the NSC channel and Na^+ reabsorption leading to fetal lung fluid clearance via reduction of $[Cl^-]_c$.