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The recovery mechanism from alkalosis in mesenteric arteriole of rat

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Basically all cells have the recovery mechanisms from the shift of intracellular pH (pHi). Many mechanisms were found and characterized. Generally the recovery mechanisms from acidosis are Na^+-dependent, such as Na^+-H^+ exchange and Na^+-HCO_3- symport. The recovery mechanism from alkalosis are Cl^--dependent, such as $Cl^-HCO_3^-$ exchange and Cal^--OH^- exchange. In the previous report, we showed two Na+ dependent mechanisms were working in the recovery process from acidosis, such as Na^+-H^+ exchange and Na^+-HCO_3^-symport. In this report, we would like to characterize the alkaline recovery mechanism in vascular smooth muscle. We used mesenteric arteriole (<150 um) and loaded carboxy SNARF-1 to measure pHi change. To induce alkalosis, we used acetate pre-pulse technique. In HCO_3^--free HEPES buffered conditions or CO_2/HCO_3^- buffered conditions, the pHi was recovered from alkalosis. The calculated proton flux in the CO_2/HCO_3^- buffered conditions was larger than that in HCO_3^--free HEPES buffered conditions. This recovery was completely inhibited by the removal of extracellular Cl^- (Cl^-_o) which was replaced by glucuronic acid. DIDS (4-acetamido-4'-isothiocyanatostilbene -2,2'-disulfonic acid, 500 uM), a classical blocker of Cl^-- HCO_3^- exchanger, did not inhibit the alkaline recovery in HCO3--free HEPES buffered conditions or CO_2/HCO_3^- buffered conditions. The other stilbene drugs such as SITS (4-Acetamido-4'-isothio-cyanatostilbene-2,2'-disulfonic acid) or DBDS (dibenzamidostilbene-disulphonic acid) also had no effect on the recovery. In CO_2/HCO_3^- buffered conditions, the removal of extracellular Na^+ (Na^+_o) which was replaced by NMDG (N-methyl-d-glucamine) accelerated the recovery. When K^+ or Cs^+ were substituted for Na+o, the recovery was slightly accelerated but was greatly attenuated compared to NMDG substitution. These results suggested that in

arteriolar smooth muscle, a novel Cl^--depedent and HCO_3^- dependent mechanism was responsible for the recovery from alkalosis. This mechanism was not sensitive stilbene derivates and affected by monovalent cations such as Na^+, K^+ or Cs^+. Still we did not know the exact stoichiometry of this mechanism and it is necessary to do further study to identify the mechanism.

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Reference

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