Astrocytes need to preserve constant volume in the face of osmolarity perturbations to function properly. To regain their original volume after hyposmotically induced swelling, they extrude intracellular electrolytes and organic osmolytes, such as inorganic ions, excitative amino acids or polyols, accompanied by osmotically driven water. This process is termed regulatory volume decrease and is ensured by various ion channels and transporters. Recently, much attention has been focused on the ubiquitous volume-regulated anion channels activated by cell swelling. VRACs are moderately outwardly rectifying with intermediary conductance, permeable to inorganic anions and organic osmolytes and sensitive to broad-spectrum anion channels blockers.

Using patch-clamp technique we aimed to characterize VRACs in cultured cortical astrocytes isolated from neonatal Wistar rats and to elucidate the effect of intracellular Na^+ on VRAC activity. In addition, we also intended to characterize these channels *in situ* in brain slices of 10 – 12 days old rats, focusing mainly on hippocampal astrocytes. To induce astrocytic swelling, we exposed astrocytes to hypotonic solution (250 mOsm). In agreement with previous findings, we showed that cultured cortical astrocytes activate VRAC currents upon exposure to hypotonic stress, which are inhibited by DCPIB, a specific VRAC blocker. Moreover, we found that VRAC activity *in vitro* is strongly dependent on intracellular Na^+ – 50 mM Na^+ completely abolished it. *In situ* we were able to detect VRAC activity in a small subpopulation of recorded astrocytes. The observed current was sensitive to DCPIB and tamoxifen.

In summary, elucidating functional properties of VRACs in astrocytes *in situ* is particularly interesting, because many brain pathologies, such as ischemia, traumatic brain injury or hyponatremia, are associated with marked astrocytic swelling and VRACs could thus constitute a target for therapy of cerebral edema.